



**2nd Annual UAB Huntsville Regional Medical Campus
Research Day**

Abstract and Poster Compendium



2nd Annual UAB Huntsville Regional Campus Research Day

April 9, 2019

Monday, April 8, 2019

4:00 – 6:00pm	Poster Judging Session (3 rd Floor)
5:30 - 6:30pm	Preceptor Appreciation Cocktail Hour
6:30 – 8:30pm	45 th Anniversary Celebration & Endowment Presentations to Dr. Bill Coleman and Dr. Lourdes Corman

Tuesday, April 9, 2019

7:30 – 10:00am	Poster Judging Session (3 rd Floor)
10:00-11:00am	Tabulating Judges Scores
11:00-12:00pm	Group 1 Oral Presentations – Research Abstract
12:00pm-12:30pm	Lunch
12:30pm-1:15pm	Keynote Speaker: Carlos Estrada, MD, MS, FACP, Professor & Division Director, Department of Medicine <i>"The Importance of Research: A Tale of Two Hemispheres"</i>
1:15-1:30pm	Announcement of Poster Winners
1:30-2:30pm	Group 2 Oral Presentations – Clinical Vignette
2:30pm	Group 1 and 2 Best Oral Presentation Winners Announced

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Research Day Compendium Introduction 2019

It is once again with pride and enthusiasm that we introduce a compendium of the clinical vignettes and research projects accepted to the Huntsville Regional Medical Campus Research Day competition. Dr. Carlos Estrada, Division Chief of the General Internal Medicine at UAB was our distinguished guest this year and his presentation about his years of work and clinical research was truly inspiring to us all..

I am proud of the hard work and enthusiasm that was shown once again this year by the UAB-Huntsville faculty, residents, and students in preparing for and presenting at this conference. The participation of our community faculty and collaborations with the Auburn School of Pharmacy was also gratifying. I look forward to continuing this tradition on our campus. Please feel free to reach out to the authors of these abstracts and posters if you have questions or want to collaborate or investigate further.

This booklet contains the abstracts and posters that were accepted at the competition.

Sincerely,

Roger D. Smalligan, MD, MPH

Professor and Regional Dean

UAB Huntsville Regional Medical Campus

April 17, 2019

Huntsville campus hosts Research Day

Written by [SOM News](#)



Huntsville Regional Medical Campus Dean Roger Smalligan, M.D., MPH, (far left), other faculty, residents, and students participated in the second annual Research Day. Some winners are pictured here with Craig Hoesley, M.D., far right, senior associate dean for Medical Education in the School of Medicine.

The UAB School of Medicine Huntsville Regional Medical Campus hosted its second annual Research Day April 9. Roger Smalligan, M.D., MPH, regional dean, and Farrah Ibrahim, M.D., FACP, director of the Huntsville Internal Medicine Residency Program, developed the oral and poster competition last year, which mirrors similar endeavors held at state and national medical conferences.

Huntsville campus students, residents and faculty presented 51 posters and nine oral presentations of research abstracts and clinical vignettes. Faculty members served as judges for the event, and the winners are listed below. The keynote speaker was Carlos Estrada, M.D., M.S., professor of Medicine and director of the Division of General Internal Medicine in the UAB School of Medicine.

Research Abstract Poster Winners

- First Place—Aristotle Asis, M.D. (Internal Medicine PGY-3), Ali Hassoun, M.D., and Esmeralda Gutierrez-Asis, M.D., for “Epidemiology Of Extended-Spectrum Beta-Lactamase (ESBL) Producing Organism Infections In A Large Tertiary Medical Center.”
- Second Place—Milza Howard (MS3), Arianna Shirk, M.D., and Annalise Sorrentino, M.D., for “Effectiveness Of A Neonatal Lactation Nurse In Preventing Hypernatremic Dehydration In A Rural Kenyan Hospital.”
- Third Place—Phillip Ingram (MS3) for “Factors Influencing Specialty Choice And Rural Family Medicine.”

Clinical Vignette Poster Winners

- First Place—Forrest Gamble (MS3), Dennis Sehgal, M.D., and Roger Smalligan, M.D., MPH, for “Catch Your Breath With A Classic Crisis.”
- Second Place—Jeremy Johnson, M.D. (Family Medicine PGY-2), Randy Turner, M.D. (Family Medicine PGY-2), and Claudia Gaviria-Agudelo, M.D., for “Behcet’s Disease: A Case Presentation.”
- Third Place—Bryan Grissett, M.D. (Internal Medicine PGY-1), and Ali Hassoun, M.D., for “Diagnostic Workup Of Esophagitis In The Setting Of Immunosuppression.”

Research Abstract Oral Presentation Winner

- Meredith Lewis, LICSW, PIP, Denise Surina-Baumgartner, Ph.D., Rajalakshmi Cheerla, M.D., and Shivani Malhotra, M.D., for “Watch Your Children Grow, Learning How To Address Obesity In Underserved Populations.”

Clinical Vignette Oral Presentation Winner

- Luke Bailey (MS3) and Parekha Yedla, M.D., for “A Tumor That Would Not Be Flushed.”

Section I: Clinical Vignettes

A Case of an Isolated Increased Factor VIII Activity and its Relationship to Hypercoagulability

Syeda Sabeeka Batool, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical; Mohammed Abdulhaleem, MD, PGY3 Internal Medicine Resident; Swetha Srialluri, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical; Alan Baggett, MD, Assistant Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus, Huntsville Hospital

Learning Objectives:

Several case-control studies have shown that a high ($> \text{ or } = 150\%$) factor VIII (FVIII) level leads to an increased risk of arterial and venous thrombosis. But the routine screening of thrombosis patients for high FVIII level is not recommended owing to uncertainty about its clinical impact. Here, we present a case of recurrent ischemic stroke, with only identifiable risk factor being an elevated FVIII level.

Case Presentation:

A 28 year-old African American male with past medical history of hypertension and hyperlipidemia presented with altered mental status a few months ago. Physical exam and radiologic evaluation led to the diagnosis of acute ischemic stroke involving the left middle cerebral artery. To evaluate the cause, routine stroke workup was performed, including echocardiography and carotid Doppler imaging, which were normal. Hypercoagulability work up including Prothrombin Gene mutation (G20210A), Protein C resistance (to exclude factor V Leiden mutation), Homocysteine level, Cardiolipin antibody, ANA, Antithrombin Gene Mutation, Protein C and S levels also came negative. So FVIII level was checked which showed an elevated level of 188 % (normal range 50-150%) and repeat evaluation showed a further increase (282%). So patient was started on oral anticoagulation, but it was held secondary to hematuria.

At current hospitalization, patient again presented with altered mental status. MRI of brain showed a recurrent acute ischemic stroke involving the left middle cerebral artery. Further work up including ANA, C-ANCA, P-ANCA and syphilis serology were all negative. Anticoagulation was started which was temporarily held owing to need for an interventional procedure. Meanwhile, patient developed painful right lower extremity swelling and Doppler ultrasound revealed deep vein thrombosis involving the right popliteal vein. So, oral anticoagulation was reinstituted to be continued for an indefinite period of time. He was discharged to rehabilitation facility given residual right sided hemiparesis.

Discussion:

FVIII serves the role of activating factor X, which directly participates in the generation of thrombin. Elevated plasma factor VIII activity ($> 150\%$) is well accepted as an independent marker of increased thrombotic risk. But high FVIII levels may also persist in normal population without causing an increased incidence of hypercoagulable events. The molecular basis for the increased levels of this factor is likely a combination of inherited and acquired variables. Genetic factors such as an increased Von-Willebrand factor level and non-O blood groups have been associated with elevated FVIII level. African Americans have also shown an increased prevalence of high FVIII level. Asymptomatic subjects with elevated FVIII level, who have a positive family history of Venous Thromboembolism (VTE) or arterial thrombosis before the age of 50 years, appear to have an increased risk of first and recurrent arterial and venous

thromboembolism. But still routine screening of factor VIII levels in all patients is not indicated, but it could be expanded if it proves to have significant impact on outcome and choice of treatment.

Authors Disclosure: This is an original manuscript. Authors disclose no financial conflicts of interest.

CASE PRESENTATION

Presenting Complaint:

- Acute onset of altered mental status
- Vitals stable and normal
- Neurologic exam showed right-sided hemiparesis
- Unremarkable otherwise

Past Medical History:

- Hypertension and Hyperlipidemia
- Acute ischemic stroke involving left cerebral artery two months prior to current hospitalization
- Oral anticoagulation stopped two months ago due to development of hematuria
- Unknown family history (patient adopted)
- Smoker, no recreational drug abuse

Routine Workup:

- MRI Brain: suggestive of recurrent stroke
- ANA, c-ANCA, p-ANCA and syphilis serology negative
- On previous admission, echocardiography, carotid Doppler imaging normal

Hypercoagulability Workup:

- Prothrombin gene mutation (G20210A), Protein C resistance (to exclude factor V Leiden mutation), Homocysteine level, Cardiolipin antibody, ANA, Antithrombin Gene Mutation, Protein C and S levels negative

Significant Findings

- On admission, Factor VIII (FVIII) activity was elevated at 188% (normal 50-150%), on repeat assessment after 2 weeks showed a further increase (282%).

Treatment:

- Standard post-stroke medical and rehabilitative care
- Oral anticoagulation reinstated

Follow-up:

- Patient discharged to Rehabilitation facility due to residual right-sided hemiparesis
- Oral anticoagulation continued indefinitely

Author's Disclosure: None

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DISCUSSION

What is known?

- FVIII is a non-Vitamin K dependent factor that is produced by liver sinusoidal cells and vascular endothelial cells throughout the body. Its gene is located on long arm of Chromosome X [1]. FVIII plays an essential role in activating factor X, which directly participates in the generation of thrombin [1]. FVIII is also an acute phase reactant so elevated levels can be found in systemic inflammatory states [1].
- Since it was first reported by Gaston et al. 1971, [2] elevated plasma factor VIII activity has become a well-accepted independent risk factor for multiple thrombo-embolic states and ischemic stroke in multiple prospective and retrospective studies [1]. (see summary table). Its procoagulant role has also been indirectly demonstrated by a lower incidence of coronary artery disease and stroke in hemophilia - a blood dyscrasia due to FVIII deficiency [3].
- High FVIII levels may also persist in the normal population without causing an overt increased incidence of hypercoagulable events and inter-individual variations in blood levels also exist [4]. FVIII level and its association with stroke in children is also not well established [5].
- Increased levels of this factor are likely a combination of inherited and acquired variables. Genetic factors such as an increased Von-Willebrand factor level and non-O blood groups have also been associated with elevated FVIII activity. African Americans have also shown an increased prevalence of elevated FVIII [1].

Summary of Studies on Association Between FVIII and Ischemic Stroke in Adults

Investigators	Study Design	Study Size	Major Findings
Landolf et al. [6]	Care-control cohort	112 adults (60 adults)	Higher FVIII activity was identified in patients with stroke compared to age-matched controls. In patients with large brain infarct (infarct size > 100 ml), higher levels of FVIII were found in stroke patients (1.85 vs 1.77 U/ml, $P < 0.01$).
Pear et al. [7]	Retrospective cohort	197 adults	In a regression model adjusted for age and sex, patients in the highest quartile of elevated FVIII activity had a greater risk of cerebral thromboembolic complications compared to patients in the lowest quartile (OR 2.4, 95% CI 1.3-4.6). But there was no correlation between FVIII and stroke.
Cao et al. [8]	Retrospective cohort	185 adults	Patients with stroke had elevated FVIII activity levels (median 1.28 U/ml, and 5 months after the ischemic stroke rose to 1.80 U/ml), compared with healthy reference population (1.0 U/ml, $P < 0.001$ for both comparisons).
Fleiss et al. [9]	Prospective cohort	315 (100 adults)	Median relative risk of ischemic stroke for patients with elevated FVIII (compared to normal FVIII) is 1.8 (95% CI 1.3-2.3).
Karimian et al. [10]	Care-control cohort	190 adults	FVIII activity is higher in patients with cryptogenic ischemic stroke (1.96 vs 1.76, $P = .01$), and elevation in FVIII (highest to lowest quartile) was associated with increased risk of cryptogenic ischemic stroke in an age and sex-adjusted model (OR 2.5, 95% CI 1.1-5.2).
Karimian et al. [11]	Care-control cohort	190 adults	Poststroke plasma levels of FVIII (median 1.96 U/ml) were significantly associated with cryptogenic stroke in patients with FVIII (OR 2.5, 95% CI 1.3-4.6). However, this effect was driven mainly by the relationship between factor V Leiden and poststroke CVD (OR 2.0, 95% CI 1.3-3.1), elevated FVIII (OR 1.1, 95% CI 0.6-2.0).
Estroff et al. [12]	Retrospective cohort	148 adults	FVIII activity is not significantly associated with stroke in patients with large or small vessel atherosclerosis (OR 0.9 vs 1.0, $P = .88$).
Volkmann et al. [13]	Retrospective cohort	41 (60 adults)	The study included patients with stroke (OR 2.5, 95% CI 1.3-4.6) compared with age-matched controls (OR 1.0, 95% CI 0.6-1.6). The study included patients with stroke (OR 2.5, 95% CI 1.3-4.6) compared with age-matched controls (OR 1.0, 95% CI 0.6-1.6).
Chang et al. [14]	Retrospective cohort	110 adults	Elevated FVIII activity is related to stroke severity (stroke severity score > 10, $P = .02$) and higher risk of mortality (mortality score > 10, $P = .02$).
Tanaka et al. [15]	Retrospective cohort	140 adults	Elevated FVIII activity is independently related to general health status (OR 1.1, 95% CI 1.3-1.1), stroke (OR 1.1, 95% CI 1.3-1.1), and mortality (OR 1.1, 95% CI 1.3-1.1).

What is not known?

- The cost-effectiveness of this screening in asymptomatic patients remains to be seen in clinical studies. Moreover, the potential effect of pharmacologic therapy for primary and secondary prevention of stroke has also not yet been studied in prospective studies [1].

Should we screen?

- Current guidelines do not recommend for or against the serologic testing of FVIII levels for routine stroke risk evaluation. But in a select high-risk patient population, screening for FVIII levels may have a significant impact on outcome and choice of treatment.

LEARNING POINTS

- A high (> or = 150%) FVIII level leads to an increased risk of arterial and venous thrombosis.
- Elevated FVIII level is established risk factor for ischemic stroke in adults.
- In a select patient population, FVIII level screening may help in delineating the cause of cryptogenic stroke and can prevent recurrent ischemic events.

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A case of Idiopathic Intracranial Hypertension with loss of vision

Nancy Sharma, MD, PGY3, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Alan Reagan, MD, PGY1, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Alan Baggett, MD, Assistant Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus, Huntsville Hospital

Case report

A 27 yo CF, with a h/o cluster headaches, fibromyalgia and bipolar disorder, presented to the ED with persistent headache x 5 days, unrelieved by Fioricet. Associated symptoms included photosensitivity, bilateral diplopia, loss of vision in lower quadrants of right eye, neck stiffness, back pain, whole body tremors, nausea, vomiting and intermittent tinnitus. Home meds: Seroquel and Lamictal. PE: afebrile, BMI 26, and normal exam except for decreased vision in right-sided inferior medial and lateral fields. Labs all within the normal range. MRI brain showed evidence of right-sided papilledema along with inflammation of the optic nerve, CTV head showed patent dural venous sinuses with no filling defect, initial lumbar puncture findings of low opening pressure. On hospital day 2, patient reported complete bilateral loss of vision. IV steroids started immediately, neurology consulted. Repeat lumbar puncture performed showed increased opening pressure and patient subsequently started on Acetazolamide for idiopathic intracranial hypertension, and reported improvement in vision thereafter. Lamictal discontinued as patient was started on this recently and may have been the precipitating cause.

Discussion

Idiopathic intracranial hypertension is characterized by elevated intracranial pressure resulting most commonly in an intractable headache worsened by postural changes, intermittent pulsatile tinnitus, *symmetric* papilledema and *transient* visual obscurations, with normal CSF composition. However, in a minority of patients, it has been seen to cause *asymmetric* or *unilateral* papilledema resulting in *rapidly progressive* visual loss in a non-obese patient with no other commonly associated risk factors. Patients usually have relief of symptoms with a diagnostic lumbar puncture that releases pressure, unless inaccurately performed. Other diagnostic modalities include MRI brain and MRV head that show signs of increased intracranial pressure. Treatment is aimed at symptom reduction and visual preservation. Weight loss for obese patients is highly recommended. Pharmacologic agents such as Acetazolamide, a carbonic anhydrase inhibitor, is the mainstay of treatment as it decreases CSF production, thereby decreasing intracranial pressure. Less commonly, agents such as Topamax, Lasix and Steroids can be used if there is poor response to Acetazolamide. Of last resort, one can undergo surgical intervention including CSF shunting and/or Optic nerve sheath fenestration. Last but not least, offending agents, such as Vitamin A, tetracyclines, Lithium, should be discontinued.

Conclusion

As in our case, patient had the classic signs and symptoms of IIH, but was not easily determined due to confounding presentation, lack of risk factors and the acutely progressive nature of her vision loss. Once increased opening pressure was confirmed on repeat lumbar puncture, Acetazolamide was immediately started and symptoms improved. In a case with no risk factors, one must take a thorough history and be able to make anecdotal connections. With our patient, the temporal proximity between initiation of Lamictal and onset of IIH could not be excluded, and therefore, the medication was discontinued.



Introduction

Idiopathic intracranial hypertension, also known as "pseudotumor cerebri", is a disorder classified by signs and symptoms of increased intracranial pressure. It is typically seen in young females of childbearing age with risk factors including obesity, medication use such as Growth Hormone, Tetracyclines, Retinoids and Lithium. With an incidence of approximately 1-2 per 100,000, pseudotumor cerebri is rare and the exact pathogenesis remains unknown. There are some proposed theories including cerebral venous outflow abnormalities, obesity-related increased intracranial pressure, abnormalities of Vitamin A metabolism, or even increased CSF outflow resistance linked to the above mentioned risk factors. Aneurysmal anastomosis can also sometimes exist. This case explores disease processes in the absence of common risk factors.

Case Presentation

- A 27 year old Caucasian female presented to the ED with persistent headache 6-8 days, unrelieved by Fentanyl. Associated symptoms included photosensitivity, bilateral diplopia, loss of vision in lower quadrants of right eye, neck stiffness, back pain, whole body tremors, nausea, vomiting and intermittent tinnitus.
- Home meds: Sertraline and Lamictal.
- PE: Afebrile, BMI 26, and normal exam except for decreased vision in right-sided inferior medial and lateral fields.
- Labs all within the normal range.
- MRI brain showed evidence of right-sided papilledema along with inflammation of the optic nerve. CTV head showed patent distal venous sinuses with no filling defect, initial lumbar puncture findings of low opening pressure.
- On hospital day 2, patient reported complete bilateral loss of vision. IV steroids started immediately, neurology consulted. Repeat lumbar puncture performed showed increased opening pressure and patient subsequently started on Acetazolamide for idiopathic intracranial hypertension, and reported improvement in vision thereafter.
- Luminal discontinued as patient was started on this recently and may have been the precipitating cause.

Discussion

- Idiopathic intracranial hypertension is characterized by elevated intracranial pressure resulting most commonly in an intractable headache worsened by postural changes, intermittent papillole tinnitus, symmetric papillolema and transient visual obscurations, with normal CSF composition.
- However, in a minority of patients, it has been seen to cause asymmetric or subacute papillolema resulting in rapidly progressive visual loss in a non-obese patient with no other commonly associated risk factors.
- Patients usually have relief of symptoms with a diagnostic lumbar puncture that releases pressure, unless insufficiently performed.

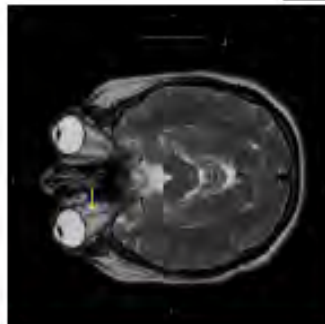


Image 1 (Left): MRI brain shows fluid collection between the right-sided optic sheath and optic nerve.

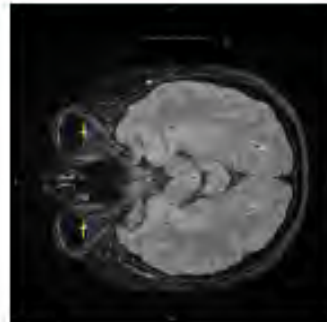


Image 2 (Right): MRI brain shows protrusion of optic disc bilaterally.

- Other diagnostic modalities include MRI brain and MRA head that show signs of increased intracranial pressure.
- Treatment is aimed at symptom reduction and visual preservation. Weight loss for obese patients is highly recommended. Pharmacologic agents such as Acetazolamide, a carbonic anhydrase inhibitor, is the mainstay of treatment as it decreases CSF production, thereby decreasing intracranial pressure. Less commonly, agents such as Topiramate, Furosemide and Steroids can be used if there is poor response to Acetazolamide. Of last resort, one can undergo surgical intervention including CSF shunting and/or Optic nerve sheath fenestration.
- Offending agents, such as Vitamin A, Tetracyclines, Lithium, should be discontinued.

Conclusion

In our case, the patient had classic signs and symptoms of ITH, but diagnosis was not readily determined due to confounding presentation, lack of risk factors and the acutely progressive nature of her vision loss. In this atypical patient, a low opening pressure on initial lumbar puncture was not expected, therefore, a repeat LP was performed. Once increased opening pressure was confirmed on repeat lumbar puncture, Acetazolamide was immediately started and symptoms improved. In a case with no risk factors, one must take a thorough history and be able to make serendipitous connections. With our patient, the temporal proximity between initiation of Lamictal and onset of ITH could not be excluded, and therefore, the medication was discontinued.

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A curious case of eosinophilia

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Learning Objectives

1. Recognise rare presentations of eosinophilia
2. Differentiate infectious, immunological and hematological causes of eosinophilia

Case Report

A 62 year old female from India on dialysis for 2 years presented with chest pain occurring in dialysis. Chest pain was midsternal, occurring with dialysis and had started the week prior. Examination revealed an alert female, comfortable on room air and afebrile. Her cardiac exam was normal and no rashes or lymphadenopathy were noted. Labs reflected a white count of $43.7 \times 10^3/\mu\text{L}$ (reference: $4.1\text{-}12.2 \times 10^3/\mu\text{L}$) and absolute eosinophil count of 37.2×10^3 (reference: $0.0\text{-}0.5 \times 10^3/\mu\text{L}$). Her cardiac enzymes were unremarkable and her kidney function was at baseline. Blood and stool cultures, ova and parasite screens and IgE levels were sent as well as a strongyloides and filarial panel given her Indian background. While cultures and parasite screen were negative, her IgE levels resulted grossly elevated. Peripheral smear and flow cytometry were inconclusive. Cardiac investigation revealed nonobstructive coronary disease. Bone marrow biopsy resulted showing eosinophilia and neutrophilia lacking malignancy. The patient attempted to continue dialysis, however again experienced chest pain. At this point a variety of differentials were considered with a discussion about recent lifestyle changes. The patient noted that she recently changed dialyzer membranes from polyarylethersulfone to polysulfone. Steroid therapy was started and her dialyzer membrane was changed back with immediate cessation of symptoms. Her eosinophilia improved and a polysulfone allergy was recorded.

Discussion

Eosinophilia can be categorised into primary/clonal, secondary/reactive or idiopathic. Diagnosis can be challenging due to the differentials present. After a detailed history is obtained, initial work up includes a complete blood count, liver and kidney function, peripheral smear, c-reactive protein, erythrocyte sedimentation rate and bone marrow biopsy. While investigating the above case, causes of secondary eosinophilia were examined while ruling out primary eosinophilia. With the patient's subtropical background, infectious and parasitic causes were initially ruled out. Her elevated IgE prompted investigation into allergic causes and drugs. Primary clonal eosinophilia and malignancies were ruled out with a bone marrow biopsy. The temporal association of symptoms with dialysis led us to question changes in dialysis causing her symptoms. Polysulfone dialyzer membranes are associated with increased incidence of hemodialysis associated eosinophilia with resolving eosinophilia noted after membrane change. The rapid response with oral steroids as well as changing the dialyzer membrane further confirmed that the patient may be having an immune mediated reaction to the Polysulfone membrane. To conclude, a meticulous history and thorough investigation is key to forming a diagnosis and carrying out appropriate treatment in unexplained eosinophilia.

*** This vignette was chosen for oral presentation on Research Day. Contact Elizabeth Thottacherry at ethottacherry@uabmc.edu for a copy of the PowerPoint.**

A rare case of infliximab induced lupus causing pericarditis

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Learning objectives

Recognize infliximab as a cause of drug-induced lupus erythematosus.

Case

A 49-year-old African American female with past medical history significant for Crohn's disease on Infliximab therapy for one year presented with one day history of sharp left sided pleuritic chest pain radiating to the back associated with shortness of breath. She denied any preceding viral prodromal symptoms. Electrocardiogram showed ST-T wave changes consistent with pericarditis and echocardiogram revealed a moderate pericardial effusion. Patient was started on Colchicine and Indomethacin. Upon further questioning, she endorsed subacute polyarthritis virtually affecting all the peripheral joints for four months prior to admission. Work up was initiated to evaluate for possible causes of pericarditis. Viral serology markers and T spot test were negative. Laboratory studies revealed elevated Erythrocyte Sedimentation Rate, C-Reactive protein, positive rheumatoid factor and Anti-Nuclear Antibody (ANA). Further workup was done which showed positive double stranded DNA (dsDNA) antibody by Crithidia and anti-histone antibodies. A diagnosis of drug-induced lupus erythematosus (DILE) due to infliximab was made in view of the temporal association of symptoms and positive clinical and serological evidence. Follow up studies showed positive anti-tumor necrosis factor alpha antibodies.

Discussion

Anti-tumor necrosis factor (anti-TNF) alpha therapies are widely used in the treatment of inflammatory bowel disease (IBD) and other rheumatological disorders. Their use has been associated with various adverse reactions including infusion reactions, reactivation of tuberculosis, serum sickness, and hematologic or bronchogenic malignancies. It may cause an autoimmune response producing symptoms similar to those of Systemic Lupus Erythematosus (SLE). This is rare with an estimated prevalence of 0.19–0.22% for infliximab.

A specific diagnostic criterion for anti-TNF alpha induced lupus has not yet been established. However, a temporal association between symptoms and therapy, at least one serologic and one non-serologic American College of Rheumatology criterion can establish diagnosis. Our patient fulfilled all three criteria and was therefore diagnosed with infliximab induced lupus.

Common presentations of infliximab induced lupus include symmetric large joint arthralgia and high titers of ANA and anti-dsDNA antibody. Pericarditis is a very rare presentation of infliximab-induced DILE. The exact mechanism of this presentation is unknown. One of the proposed mechanisms include proinflammatory activity in the pericardium and delayed reactions causing a serum sickness like reaction.

Management of infliximab-induced DILE involves immediate discontinuation of infliximab and treatment of serositis or arthritis with steroids. Vedolizumab is approved for use in moderate to severe IBD who have failed conventional and anti-TNF alpha therapy. In a small number of cases, vedolizumab has been used successfully in patients who were rechallenged after developing infliximab-induced DILE.

Conclusion

Infliximab is widely used in the treatment of inflammatory bowel disease and can rarely be associated with DILE that presents as pericarditis. Clinicians should be aware of this fact and counsel patients about this rare but clinically significant complication. Further studies are needed to establish more definitive criteria for the diagnosis of anti-TNF alpha induced lupus.

Learning Objectives:

- Recognize infliximab as a cause of drug-induced lupus erythematosus.
- A 46-year-old African American female with past medical history significant for Crohn's disease on infliximab therapy for one year presented with one-day history of sharp left-sided pleuritic chest pain radiating to the back associated with shortness of breath. She denied any preceding viral prodromal symptoms.
- Electrocardiogram showed ST-T wave changes consistent with pericarditis and echocardiogram revealed a moderate pericardial effusion. The patient was started on colchicine and indomethacin.
- Upon further questioning, she endorsed subacute polyarthralgia virtually affecting all the peripheral joints for five months prior to admission.
- Work up was initiated to evaluate for possible causes of pericarditis. Viral serology markers and T spot test were negative. Laboratory studies revealed elevated Erythrocyte Sedimentation Rate, C-reactive protein, positive rheumatoid factor and Anti-Nuclear Antibody (ANA).
- Further workup was done which showed positive double-stranded DNA (dsDNA) antibody by Crithidia and anti-binding antibodies.
- A diagnosis of drug-induced lupus erythematosus (DILE) due to infliximab was made in view of the temporal association of symptoms and positive clinical and serological evidence. Follow up studies showed positive anti-tumor necrosis factor alpha antibodies.

Discussion

- Anti-tumor necrosis factor (anti-TNF) alpha therapies are widely used in the treatment of inflammatory bowel disease (IBD) and other rheumatological disorders. Their use has been associated with various adverse reactions including infusion reactions, reactivation of tuberculosis, serum sickness, and hematologic or hematopoietic malignancies.
- Infliximab may cause an autoimmune response producing symptoms similar to those of Systemic Lupus Erythematosus (SLE). This is rare with an estimated prevalence of 0.19–0.22% for infliximab.
- A specific diagnostic criterion for anti-TNF alpha induced lupus has not yet been established. However, a temporal association between symptoms and therapy, at least one serologic and one non-serologic American College of Rheumatology criterion can establish the diagnosis. Our patient fulfilled all three criteria and was therefore diagnosed with infliximab induced lupus.



Chest X-ray showed bilateral air space opacities.

Computed tomography angiography showed moderate pericardial effusion.

Echocardiogram showed moderate pericardial effusion and no signs of cardiac tamponade.

Discussion continued

- Common presentations of infliximab induced lupus include symmetric large joint arthralgia and high titers of ANA and anti-dsDNA antibody. Pericarditis⁵ is a very rare presentation of infliximab-induced DILE (drug-induced lupus erythematosus).
- The exact mechanism of this presentation is unknown. One of the proposed mechanisms include proinflammatory activity in the pericardium and delayed reactions causing a serum sickness-like reaction⁶.
- Management of infliximab-induced DILE involves immediate discontinuation of infliximab and treatment of symptoms or arthritis with steroids.
- Methotrexate is approved for use in moderate to severe IBD who have failed conventional and anti-TNF alpha therapy. In a small number of cases, vedolizumab has been used successfully in patients who were rechallenged after developing infliximab-induced DILE.

Conclusion

- Infliximab is widely used in the treatment of inflammatory bowel disease and can rarely be associated with DILE that presents as pericarditis. Clinicians should be aware of this fact and counsel patients about this rare but clinically significant complication. Further studies are needed to establish more definitive criteria for the diagnosis of anti-TNF alpha induced lupus.

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A tumor that would not be flushed

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Learning Objectives

1. Unusual presentation of carcinoid tumor
2. Complications of carcinoid tumor

Case

The patient is a 62-year-old Caucasian female who presented complaining of occasional diarrhea, bloating, and abdominal fullness. She described these symptoms as slowly progressive and felt that something “just wasn’t right”. She denied fever, nausea, or flushing. Past medical and family histories were negative. On exam, she was afebrile with stable vital signs. Abdominal exam revealed abdominal distension and mild hypogastric tenderness to palpation; bowel sounds were positive. Laboratory studies were all within normal limits. She received an abdominal ultrasound that showed large bilateral ovarian cysts and confirmed the presence of an umbilical hernia, and she was subsequently scheduled for total abdominal hysterectomy with bilateral oophorectomy and umbilical hernia repair. Pathology of umbilical hernia contents revealed well-differentiated carcinoid tumor with positive lymphovascular invasion and positive staining for chromogranin, synaptophysin, and CD56. The patient has been followed by gastroenterology and treated with monthly Octreotide injections, which have been well-tolerated. Metastatic lesions were subsequently discovered 7 years later in the liver and abdomen, the latter necessitating a ureteral stent due to compression on the ureter. During the present admission, the patient came in with altered mental status, E. coli bacteremia, and was found to have severe right sided hydronephrosis due to migration of the ureteral stent into the renal pelvis. The patient required emergent percutaneous nephrostomy tube placement and was transferred to Vanderbilt Medical Center for further treatment.

Discussion

Well-differentiated neuroendocrine tumors, known as carcinoids, are tumors that commonly arise from the gastrointestinal tract. Carcinoid tumors are relatively rare; the annual incidence rate for Caucasians is 4.6 per 100,000. The mean age of diagnosis is 63. Though rare, the incidence of carcinoids is increasing in the United States. This increase is likely due to increased detection by imaging modalities. Carcinoids are frequently diagnosed due to carcinoid syndrome, which occurs with metastasis and is characterized by chronic flushing and diarrhea. Only 8-10% of neuroendocrine tumors result in carcinoid syndrome. Many, as with this patient, are diagnosed incidentally and experience vague, nonspecific symptoms. Treatment includes the use of chemotherapy, radiation, somatostatin analogues, and surgery. Octreotide, a long-acting somatostatin analog, blocks hormone secretion by the tumor. This is helpful not only in reducing symptoms, but also in inhibiting tumor growth and slowing disease progression. Fortunately for patients, even when distant metastasis is present the overall five-year survival ranges from 40-85%, and ten-year survival from 40-60%.

*** This vignette was chosen for oral presentation on Research Day. Contact Luke Bailey at bailey00@uabmc.edu for a copy of the PowerPoint.**

Acquired Hemophilia A due to Alemtuzumab Treatment for Multiple Sclerosis

Jesse Faulk, MD, PGY2, Internal Medicine Resident; Dennis Sehgal, MD, PGY2, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Jorge Diaz, MD, Hematology/Oncology Specialist at Clearview Cancer Institute, Huntsville, AL; Pavan Panchavati, MD, Clinical Associate Professor, UAB Huntsville Regional Medical Campus

Learning objectives:

1. Diagnose acquired hemophilia A.
2. Treat acquired hemophilia A.

Case Presentation: A 66-year-old Caucasian female presented to the ED due to bruising. Her bruising began 4 days prior, beginning on her hands and feet then involving her torso. She also admitted to hematuria for 4 days but denied melanic stools, hematemesis, hematochezia, epistaxis, or joint swelling. She denied any recent trauma or history of easy bruising or bleeding. She has a medical history significant for multiple sclerosis and was treated nine months prior with alemtuzumab. She had a prior appendectomy and cholecystectomy without any significant bleeding. She has no family history of bleeding or clotting disorders. On examination, she was afebrile and normotensive. She had diffuse bruising. Her hemoglobin was 8.1 g/dL, platelet count was $381 \times 10^3/\mu\text{L}$, and WBC was $11.7 \times 10^3/\mu\text{L}$. Prothrombin time (PT) was 13.1 s and partial thromboplastin time (PTT) was 90.2 s. Factor 8 activity was 3% (normal: 50-150%). A PTT mixing study was performed with PTT failing to correct. Lupus anticoagulant was not detected. Factor VIII inhibitor was detected. Computed tomography of chest, abdomen, and pelvis showed no malignancy. Antinuclear antibody was negative. She was diagnosed with acquired hemophilia A secondary to alemtuzumab therapy and started on prednisone and rituximab, which was changed to cyclophosphamide due to allergic reaction. Her PTT began to decrease, and she was discharged on prednisone and cyclophosphamide.

Discussion:

Acquired hemophilia A is an uncommon autoimmune disorder that is caused by an inhibitory autoantibody to factor VIII. Acquired hemophilia may be associated with certain medications, autoimmune disorders, or malignancy. Almost half of patients may not have an identified etiology. Alemtuzumab, a humanized monoclonal antibody targeting the surface molecule of CD52 of B and T lymphocytes, has been reported to cause autoimmune conditions, and limited case reports exist of this monoclonal antibody causing acquired hemophilia A. The usual presenting symptom is bleeding that may occur after a surgical procedure or spontaneously. Patients may present with mucocutaneous bleeding, such as epistaxis, gastrointestinal bleeding, hematuria, ecchymosis, or large hematomas. Acquired hemophilia is characterized by prolonged PTT with normal PT. A mixing test that results in uncorrected PTT differentiates acquired hemophilia from inherited factor deficiency, and a Bethesda assay diagnoses the presence of a factor VIII inhibitor. Desmopressin, factor VIII concentrates, or activated prothrombin complex concentrate can be used to control bleeding. In order to eliminate the inhibitor, immunosuppressive modalities, such as glucocorticoids, cyclophosphamide, and rituximab, may be used.

Acquired Hemophilia A due to Alemtuzumab Therapy for Multiple Sclerosis

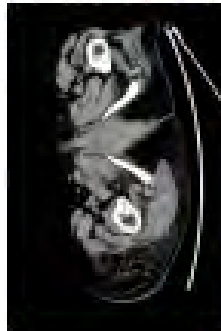
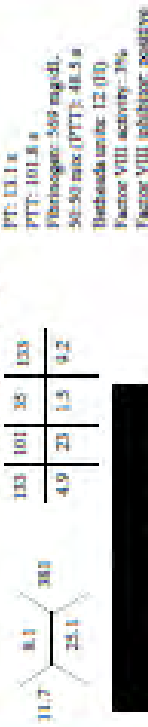
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Objectives

- To understand how to evaluate a prolonged PTT and diagnose acquired hemophilia A
- Understand the treatment for acquired hemophilia A
- Case Presentation**
 - A 66-year-old Caucasian female presents to the ED due to bruising and hematuria for past 4 days.
 - bruising began on her hands and feet and then progressed to her torso.
 - No recent trauma or previous history of easy bruising or bleeding disorders other than prior surgeries (appendectomy, cholecystectomy).
 - No medications, alcohol, or joint swelling.
 - hematochezia, epistaxis, or joint swelling.
 - PMH: Multiple sclerosis, migraines
 - Medications: Naproxen, Sumatriptan, Alemtuzumab (9 months prior)
 - Social History: No tobacco, alcohol, or illicit drug use.
 - Physical Exam: VS: T: 97.1° F, P: 107, BP: 114/50, RR: 18, O₂ sat: 100% on RA.
 - CV: HRR, no murmur, no S3/S4
 - PE: CTTAB
 - Musculoskeletal: no joint swelling
 - Neurologic: No focal deficits; alert and oriented x4.
 - Skin: Extensive ecchymosis to the bilateral upper and lower extremities



Diagnostic Work-Up



CT scan of the abdomen/pelvis without contrast revealed a 6 cm right gluteal muscle tear

Evaluation of prolonged PTT



Acquired Hemophilia A treatment

Hospital Course

- Patient was diagnosed with acquired hemophilia A
- Started on prednisone and danaparone
- CT scan of abdomen/pelvis to rule out malignancy and hematuria revealed a 6 cm right gluteal muscle mass, most likely a hematoma
- Started on cyclophosphamide and rituximab on day 5 in combination with prednisone due to no improvement in PTT
- Rituximab discontinued due to respiratory distress on initiation
- Developed atrial fibrillation and heart failure with reduced EF at day 7
- Given anti-inhibitor coagulant complex (FIIIIA) on day 7 due to encephalopathy; discontinued after CT head
- PTT improved to 40.9 seconds on day 14
- Discharged on prednisone and cyclophosphamide

Acquired Hemophilia A

- Suspect in someone with spontaneous bruising and elevated PTT
- Use both labs assay to determine inhibitor titer and treatment efficacy

Alemtuzumab

- Humanized monoclonal antibody targeting surface molecule of CD153 of T and B lymphocytes
- Five case reports exist of acquired hemophilia A from alemtuzumab
- Autoimmune condition development may be delayed by up to 5 years after therapy

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Acute Appendicitis Masking Mucinous Adenocarcinoma

Michael Tran, MS3, UAB Huntsville Regional Medical Campus; Roger D. Smalligan, MD, Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus

Learning Objective #1: Acknowledge the rare occurrence of mucinous adenocarcinoma in younger patients

Learning Objective #2: Symptoms of acute appendicitis and persistent constipation that continue after appendectomy can suggest possible malignancy in the colon.

Case: A 23yo man presented with RLQ pain and constipation for 2 months post appendectomy. PMH: Rt. Inguinal hernia repair age 5. FH: negative for hypothyroidism and cancer. Two months prior he had presented with RLQ pain without change of bowel habits or fever and was dx with appendicitis by clinical and CT findings. Pathology was reportedly c/w appendicitis. During the weeks post-op he had intermittent abd pain and continued severe constipation despite atbx, laxatives and an attempt with linaclotide; all without success. F/U CT abd showed bowel wall thickening of the distal small bowel and ascending colon with question of soft tissue density at the hepatic flexure. A trial of antibiotics failed and a week later he presented to the ER with nausea, vomiting and increased RLQ pain. On exam he was in pain, afebrile, VSS, lungs, heart nl, abd without rebound and bowel sounds diminished and no obvious mass. Labs: WBC 14.3, CRP 0.6, and CEA 2, chem 12 was wnl. Colonoscopy showed a partially obstructing cecal mass which was biopsied. Path showed mucinous adenocarcinoma with signet ring cells. He underwent hemicolectomy with a 5cm tumor at the hepatic flexure, ascites and enlarged lymph nodes. 9/45 nodes were positive for adenoCa resulting in a T4, N2, stage IV cancer.

Discussion: Mucinous adenocarcinomas (MACs) are malignant tumors that arise from epithelial tissue. They make up 10-15% of all colon cancers. They have a predilection for the right colon, with poor response to chemotherapy. These tumors are usually seen in older patients with the mean age of 66 and can occur in the appendix. One study reported 98.8% of MACs occur over the age of 30. About 1% of the time these tumors occur in the appendix although our patient's appendix was read as inflammation only. When a signet ring cell type of MAC is seen it usually originates from the stomach or ovaries and is seen in adenoCa of unknown origin cases. It carries a very poor prognosis (<30% 5y survival). Treatment is typically surgical debulking and right hemicolectomy as was done in our case followed by chemotherapy. Our case is rare due to the patient's very young age. Clinically it was difficult to diagnose him given the higher epidemiologic likelihood of infectious colitis or inflammatory bowel disease being the cause of the bowel thickening and the mass appearance of the distal colon. It was curious that he developed acute appendicitis and one wonders if tumor was overlooked there or if the distant tumor contributed to its occurrence. This case reminds physicians to keep cancer in the differential dx, including the rare mucinous adenocarcinoma, in young patients if response to usual treatment is atypical or unsuccessful.



Introduction

Mucinous adenocarcinoma is a rare and malignant form of cancer of the colon. It is more often seen in the older population, and rarely in the young. When young people are affected, it is more often found at an advanced stage.

Case Presentation

- A 23yo man presented with RLQ pain and constipation for 2 months after appendectomy
- PMH: right inguinal hernia repair at age 5
- PFH: negative for cancer, hypothyroidism
- Two months prior pt presented with RLQ pain without change of bowel habits or fever and was dx with appendicitis by clinical and CT
- Pathology at that time c/w appendicitis
- Weeks post-op he presented with intermittent abd pain and continued severe constipation despite after laxatives and laxocloids
- FU CT abd - bowel wall thickening of the distal small bowel and ascending colon with question of rectal time density at the hepatic flexure
- Antibiotics were given again
- One week later he presented to the ER with nausea, vomiting and increased RLQ pain
- On exam he was in pain, afebrile, VSS, lungs clear, heart nl, abd without rebound tenderness and bowel sounds diminished and no obvious mass
- Labs: WBC 14.3, CRP 0.6, and CEA 2, chem 12 was normal
- CT abd/pelvis with contrast - small bowel wall thickening in RLQ near terminal ileum suspicious for IBD

Hospital Course

Colonoscopy showed a partially obstructing cecal mass which was biopsied. Pathology showed mucinous adenocarcinoma with signet ring cells. Patient underwent right hemicolectomy with ileotransverse colonic anastomosis. A 3cm tumor at the hepatic flexure, active and enlarged lymph nodes were noted at surgery. 9 of 45 nodes were positive for adenocarcinoma resulting in a T4, N2, stage IV classification. Patient has been undergoing chemotherapy after resection and is doing well.

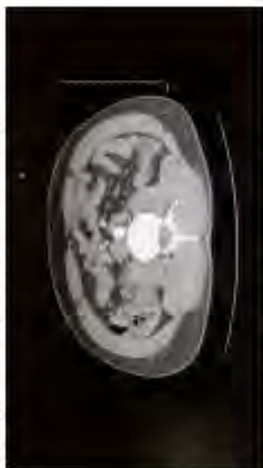


Fig 1 - Initial CT showing appendicitis only



Fig 2 - CT during admission showing findings of 'IBD'

Discussion

- Mucinous adenocarcinomas (MACs) are malignant tumors that arise from epithelial tissue. They make up 10-15% of all colon cancers and have a predilection for the right colon.
- These tumors are usually seen in older patients with the mean age of 66 and can occur in the appendix. One study reported 98.8% of MACs occur over the age of 30.
- About 1% of the time these tumors occur in the appendix although one patient's appendix was read as inflammation only. Signet ring cell type of MAC often originates from the stomach, ovary or in cases of adenocarcinoma of unknown origin.
- MAC carries a very poor prognosis (<30% 5y survival).
- Treatment is typically surgical debulking and hemicolectomy followed by chemotherapy.

Discussion (cont)

- Clinical diagnosis may have been delayed given the higher epidemiologic likelihood of infectious colitis or inflammatory bowel disease being the cause of the bowel thickening and the mass appearance of the distal colon.
- The initial presentation of acute appendicitis and the persistent symptoms after surgery also led the team to consider post-op complications over that of a new diagnosis.

Conclusion

This case reminds physicians to keep cancer in the differential diagnosis, including the rare mucinous adenocarcinoma in young patients if response to usual treatment is atypical or unsuccessful.

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Albuterol induced lactic acidosis, an under recognized phenomenon

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Learning objectives:

- To learn about types and causes of lactic acidosis and recognize beta-adrenergic agonist (albuterol) as a cause of lactic acidosis
- One can avoid extensive workup for sepsis in absence of tissue hypoxia for lactic acidosis

Case presentation:

73 year-old Caucasian male with chronic obstructive pulmonary disease (COPD) presented to the ER with progressively worsening dyspnea for 3 days. The symptoms had worsened despite the increasingly frequent use of albuterol/ipratropium combination inhaler at home. It was associated with a productive cough, wheezing and chest tightness. No fever, chills, weight loss or chest pain were reported. The patient was an active smoker with a 50 pack year smoking history. On evaluation, he was afebrile, tachycardic, tachypneic and saturating 94% on room air. Chest auscultation revealed diffuse expiratory wheezing. Initial laboratory data showed normal Complete Blood count (CBC), Comprehensive metabolic panel, C Reactive protein (CRP) and Pro BNP. Initial venous blood gas (VBG) analysis showed a lactate level of 4.21. Chest radiograph did not show any acute findings. The patient received an hour-long treatment of albuterol in the ER for COPD exacerbation. 3 hours later, repeat lactate level was 8.3. Arterial blood gas (ABG) analysis showed a normal value for pH, PCO₂, and PO₂. Workup for elevated lactate level including blood levels of salicylate and carboxyhemoglobin which were negative. Sepsis was ruled out with a normal CBC, CRP, negative blood/urine cultures and imaging. A presumptive diagnosis of albuterol induced lactic acidosis was made after excluding all causes of Type A and B lactic acidosis. Secondary to this, patient's albuterol treatments were spaced out from every four hours to every eight hours. In next 2 days, the lactate level was noted to have normalized to 2.1. The patient was discharged home after stabilization without any complication

Discussion:

The normal plasma lactate concentration is 0.5 to 1.5 mmol/L. Hyperlactatemia is if lactate concentration is 2-4 mmol/L without metabolic acidosis. Lactic acidosis is generally defined as a plasma lactate concentration greater than 4 mmol/L, even in the absence of overt acidemia. Beta adrenergic agonists are less recognized cause of lactic acidosis. Although the mechanism is not proven, the lactic acidosis in such patients may be due to adrenergic-induced glycolysis and lipolysis, which increase pyruvate and free fatty acid concentrations, respectively.

There are two types of lactic acidosis. Type A lactic acidosis is due to marked tissue hypoperfusion resulting from hypovolemia, cardiac failure, sepsis, or cardiopulmonary arrest. Type B lactic acidosis is due to either increased production of lactate or decreased clearance of lactate. Causes for Type B includes Drug induced, Diabetes mellitus, malignancy, B adrenergic agonist, chronic alcoholism,

thiamine deficiency, and HIV. In our case we have been ruled out type A lactic acidosis and we were directed to consider other causes of lactic acidosis. Lactate started trending down with spacing of albuterol nebulizer treatment. Lactate has been normalized on the day of discharge. Exact pathophysiology and why only proportion of people develop it remains to be elucidated.

Albuterol induced lactic acidosis, an under-recognized phenomenon

Sujatha Baddam¹, MD; Bhavyaa Bahl¹, MD; Lourdes Corman¹, MD;
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Case Description

- 73-year-old Caucasian male presented with progressively worsening dyspnea associated with a productive cough, wheezing and chest tightness for 3 days.
- On evaluation, vitals has been stable except for tachycardia, tachypnea and chest auscultation revealed diffuse expiratory wheezing
- Laboratory data showed normal CBC, CMP, CRP and CXR
- Initial venous blood gas (VBG) showed a lactate level of 4.21
- The patient received an hour-long treatment of albuterol in the ER for COPD exacerbation. 3 hours later, repeat lactate level was 8.3.
- Sepsis was ruled out with a normal CBC, CRP, negative blood/urine cultures and imaging.
- A presumptive diagnosis of albuterol induced lactic acidosis was made after excluding all causes of Type A and B lactic acidosis

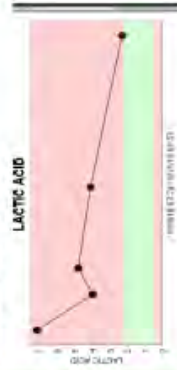
Discussion

- The normal plasma lactate concentration is 0.5 to 1.5 mmol/L.
- Hyperlactatemia occurs when the lactate concentration is 2-4 mmol/L without metabolic acidosis.
- Lactic acidosis is defined as a plasma lactate concentration greater than 4 mmol/L, even in the absence of overt acidemia
- There are two types of lactic acidosis, Type A and B.
- Type A lactic acidosis occurs due to marked tissue hypoperfusion resulting from hypovolemia, cardiac failure, sepsis, or cardiopulmonary arrest.
- Type B lactic acidosis occurs secondary to either increased production or decreased clearance of lactate. It is noted to be caused by drugs including beta-adrenergic agonists, diabetes mellitus, malignancy, chronic alcoholism, thiamine deficiency, mitochondrial dysfunction, and human immunodeficiency virus (HIV).
- Beta-adrenergic agonists are a less frequently recognized cause of lactic acidosis
- lactic acidosis in such patients may be due to adrenergic-induced glycolysis and lipolysis, which increase pyruvate and free fatty acid concentrations, respectively.
- Lactic acidosis is a frequently encountered condition among hospitalized patients requiring further evaluation as it is often associated with sepsis, timely management of which is crucial. In the setting where suspicion for sepsis is low, an extensive workup can be avoided by ruling out other causes particularly of type B lactic acidosis.

Learning objectives

- To learn about types and causes of lactic acidosis and recognize beta-adrenergic agonist (albuterol) as a cause of lactic acidosis
- One can avoid extensive workup for sepsis in absence of tissue hypoxia for lactic acidosis

Graph showing trend of Lactic acid during hospital course



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An Unusual Consequence of Alcoholism and Recurrent Pancreatitis

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LEARNING OBJECTIVES

Recognize an uncommon cause of ascites in patients with alcoholic liver disease and the treatment options available.

CASE

A 41 year old woman with history of alcoholism and recurrent pancreatitis presented with a three day history of abdominal pain and distension. She was seen in the emergency room a week ago for similar complaints, was diagnosed with cirrhosis and ascites, underwent paracentesis which was notable for a serum-ascites albumin gradient (SAAG) of 1.6 consistent with portal hypertension. During this presentation, physical exam was remarkable for ascites and diffuse tenderness to palpation. Laboratory studies were normal except for a low serum albumin of 2.1 g/dL, CT abdomen showed hepatic steatosis and cysts in the tail of the pancreas. Ascitic fluid analysis revealed total nucleated count of 414/microliter, total protein of 3.1 g/dL and a SAAG of 0.8 after which fluid amylase levels were checked and found to be elevated at 3671 U/L. Endoscopic ultrasound was done for further evaluation of pancreatic cysts and the aspirate was noted to be benign. The patient was diagnosed with pancreatic ascites, underwent an endoscopic retrograde cholangiopancreatography with biliary sphincterotomy for choledocholithiasis. She came back 2 days later with worsening ascites. Fluid analysis again showed elevated amylase of 2475 U/L. Repeat ERCP revealed pancreatic duct leakage and a transpapillary stent was placed. The patient improved clinically and symptomatically over the next few days. At a follow up visit 2 weeks later, there was no recurrence of ascites.

DISCUSSION:

Most common causes of ascites are cirrhosis, malignancy and heart failure. Pancreatic ascites is rare and is usually from leakage of the pancreatic duct or pseudocysts. It is characterized by SAAG <1.1, ascitic amylase level >1000 U/L and total protein >3 g/dl. Gold-standard for diagnosis is ERCP showing pancreatic duct disruption.

The most common cause of pancreatic ascites is chronic pancreatitis, other causes include acute pancreatitis and trauma. Estimated prevalence is 4% in patients with chronic pancreatitis and 6 to 14% in patients with pseudocysts.

There are only a few case reports and case series available to guide the management of pancreatic ascites. Conservative therapy includes serial paracentesis, parenteral nutrition and somatostatin analogues which decrease pancreatic secretions facilitating fistula closure. Interventions include either endoscopic therapy or surgery as the last resort. ERCP with transpapillary stent placement is a viable option in most cases. Surgical procedures include partial pancreatectomy and pancreaticojejunostomy.

CONCLUSION

Pancreatic ascites should be considered in the differential diagnosis of recurrent ascites in a patient with history of alcoholism and pancreatitis. Growing evidence supports interventional therapy as an initial treatment strategy especially endoscopic transpapillary stenting of the pancreatic duct.

LEARNING OBJECTIVES:

- Recognize an uncommon cause of ascites in patients with alcoholic liver disease and the treatment options available.

CASE:

- A 41 year old woman with history of alcoholism and recurrent pancreatitis presented with a three day history of abdominal pain and distension.
- She was seen in the emergency room a week ago for similar complaints, was diagnosed with carbon monoxide and ascites, underwent paracentesis which was notable for a serum-ascites albumin gradient (SAAG) of 1.6 consistent with portal hypertension.
- During this presentation, physical exam was remarkable for ascites and diffuse tenderness to palpation.
- Laboratory studies were normal except for a low serum albumin of 2.1 g/dL. CT abdomen showed hepatic steatosis and cysts in the tail of the pancreas.
- Ascitic fluid analysis revealed total nucleated count of 414/cubicmm, total protein of 3.1 g/dL, and a SAAG of 0.8 after which fluid analyses levels were checked and found to be elevated at 3671 U/L.
- Endoscopic ultrasound was done for further evaluation of pancreatic cysts and the aspirate was noted to be benign.
- The patient was diagnosed with pancreatic ascites¹, underwent an endoscopic retrograde cholangiopancreatography with biliary sphincterotomy for cholelithiasis and was discharged home on Octreotide.

- She came back 2 days later with worsening ascites. Fluid study no again showed elevated any less of 2415 U/L.
- Repeat ERCP revealed pancreatic duct leakage and a transpapillary stent² was placed.
- The patient improved clinically and symptomatically over the next few days. At a follow up visit 2 weeks later, there was no recurrence of a pain.

DISCUSSION:

- Most common cause of ascites are cirrhosis, malignancy and heart failure. Pancreatic ascites³ is rare and is usually from leakage of the pancreatic duct or pseudocysts.
- It is characterized by SAAG <1, acidic amylase level > 500 U/L, and total protein >3 g/dL.
- Gold-standard for diagnosis is ERCP showing pancreatic duct obstruction.
- The most common cause of pancreatic ascites is chronic pancreatitis, other causes include acute pancreatitis and trauma.

ERCP showing pancreatic duct leak with extravasation of contrast



ERCP following transpapillary stent placement

CONCLUSION:

- Pancreatic ascites should be considered in the differential diagnosis of recurrent ascites in a patient with history of alcoholism and pancreatitis. Growing evidence supports interventional therapy as an initial treatment strategy especially endoscopic transpapillary stenting of the pancreatic duct.

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Behcet's Disease: A case presentation

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Main Body:

A 16-year-old Caucasian male presented to the Huntsville Hospital pediatric emergency department due to oropharyngeal pain secondary to oral mucosal lesions. The patient has a 5-year history of recurrent oral, genital, and skin lesions occurring after viral illness or physical stress. This episode began after antigen proven influenza and the initial episode occurred after a clavicular fracture. On presentation differential diagnosis included Behcet's Syndrome, mouth and genital ulcers with inflamed cartilage (MAGIC) syndrome, periodic fever with aphthous stomatitis, pharyngitis, and adenitis (PFAPA) syndrome, recurrent aphthous stomatitis, herpes simplex virus, varicella viral infection, and autoimmune of unknown etiology. Physical exam showed multiple vesicular lesions with erythematous bases along the mucosa of the oropharynx, dry crusted vesicular lesions on an erythematous base along the arms and legs, and dry crusted vesicular lesions on an erythematous base along the genitalia. On initial evaluation other than a c-reactive protein 7.6 and sed rate of 54 the chest imaging was benign as was an extensive lab panel including comprehensive metabolic panel, complete blood count, urinalysis, HIV, blood culture, mycoplasma, myeloperoxidase antibody negative, total complement, complement C3 and C4, ANA, cANCA, pANCA, dsDNA, serum electrophoresis, proteinase 3 antibody, ENA antibody screen, and TB. There was a mild hypocomplementemia and neutropenia on chart review. A skin biopsy of the right leg showed lymphocytic perivascularitis with epidermal necrosis. A diagnosis of Behcet's was made based on the International Criteria for Behcet's Disease with skin lesions (1 point), genital aphthosis (2 points), and vascular lesions (1 point) for >3 points. The patient was started on steroids and will be followed as an outpatient with rheumatology.

Most common in Turkey and with a distribution pattern along the ancient silk road, Behcet's Disease affects almost 1 in 250 people of Turkish decent while only 1 in 500,000 North Americans. Generally presenting in years 20-40 of life this is a disease historically limited to immigrant and refugee populations in the United States, though as our patient represents must be considered whenever a patient presents with a disease pattern of recurrent oral aphthous ulcers with the potential for systemic involvement including skin and genital lesions, ocular involvement, arthritis, vascular disease (uniquely small, medium, and large vessels), gastrointestinal disease, and neurological disease. Many genetic and autoimmune theories exist about the underlying pathogenesis though none have consensus. Treatment involves broad, non-specific immunosuppression with prednisone and ciclosporin with new agents such as infliximab and antitumor necrosis factor alpha antibody showing promise. The prognosis is generally good with an overall estimated mortality of 5% at 7.7 years. The disease is most severe in young, male, middle-eastern or far-eastern patients. The presence of neurological disease is a poor prognosticator with the combination neurological disease and elevated protein or pleocytosis showing the majority of patients dead or with significant disability within 3 years.

Behcet's Syndrome: A case presentation

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2. Department of Pediatrics, UAB-Birmingham, Huntsville, AL, United States

Introduction

- 15-year-old Caucasian male presented to the Huntsville Hospital pediatric emergency department due to oropharyngeal pain secondary to oral mucosal lesions.
- The patient has a 5-year history of recurrent oral, genital, and skin lesions occurring after viral illness or physical stress.
- This episode began after antigen proven influenza and the initial episode occurred after a clavicular fracture.
- On presentation differential diagnoses included Behcet's Syndrome, mouth and genital ulcers with inflamed cartilage (MAGIC) syndrome, periodic fever with aphthous stomatitis, pharyngitis, and adenitis (PFAPA) syndrome, recurrent aphthous stomatitis, herpes simplex virus, varicella viral infection, and autoimmune of unknown etiology.
- Physical exam showed multiple vesicular lesions with erythematous bases along the mucosa of the oropharynx, dry crusted vesicular lesions on an erythematous base along the arms and legs, and dry crusted vesicular lesions on an erythematous base along the genitalia.
- On initial evaluation other than a t-reactive protein 7.6 and sed rate of 54, the chest imaging was benign as was an extensive lab panel including comprehensive metabolic panel, complete blood count, urinalysis, HIV, blood culture, mycoplasma, mycoplasma antibody negative, latex coagulation complement C3 and C4, ANA, cANCA, pANCA, cANCA, serum electrolytes, prothrombin 3 antibody, DNA antibody screen, and TB. There was a mild hyponatremia and neutrophilia on chest review.
- A skin biopsy of the right leg showed lymphocytic perivasculitis with epidermal necrosis.
- A diagnosis of Behcet's was made based on the International Criteria for Behcet's Disease with skin lesions (1 point), genital aphthosis (2 points), and vascular lesions (1 point) for >3 points.
- The patient was started on steroids and followed as an outpatient with rheumatology.



Figure 1. Intra-oral lesions along the soft palate and posterior pharynx of a patient with Behcet's Disease.



Figure 3. Cutaneous lesion in the setting of Behcet's Disease. Inset is same lesion with point of reference for ABCs.



Figure 2. Mucocutaneous lesions along the lips of a patient with Behcet's Disease.



Figure 4. Behcet's Disease is most prevalent along the ancient silk road which is highlighted along the red line.

Discussion

- Mixed common in Turkey and with a distribution pattern along the ancient silk road.
- Behcet's Disease affects almost 1 in 250 people of Turkish descent while only 1 in 500,000 North Americans.
- Generally presenting in years 20-40 of life this is a disease historically linked to immigrant and religious populations in the United States, though in our patient's case a patient presents with a disease pattern of recurrent oral aphthous ulcers with the potential for systemic involvement including skin and genital lesions, ocular involvement, arthritis, vascular disease (primarily small, medium, and large vessels), gastrointestinal disease, and neurological disease.
- Many genetic and autoimmune theories exist about the underlying pathogenesis though none have consensus.
- Treatment involves broad, non-specific immunosuppression with prednisone and colchicine with more targeted such as infliximab and anti-tumor necrosis factor alpha antibody blocking proteinase.
- Long treatment course of 15-24 months in severe disease.
- The prognosis is generally good with an overall estimated mortality of 5% at 7-7 years.
- The disease is most severe in young, male, middle-eastern or far-eastern patients.
- The prevalence of neurological disease is a poor prognosticator with the combination neurological disease and vascular protein or placental disease affecting the majority of patients dead or with significant disability within 3 years.

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Brain Abscess mimicking Brain neoplasm presentation

Sabrina Matosz, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical; Ali Hassoun, MD, Infectious Disease Specialist, Alabama Infectious Diseases Center, UAB Huntsville Regional Medical Campus

Learning Objective

Differentiating and confirming the diagnosis between a brain abscess versus a brain neoplasm.

Case presentation

A 32 year old African American male with 2 week history of severe central headache radiating to the left side and blurry vision. Past medical history significant for hypertension, recurrent sinusitis, and depression. He denies use of tobacco, alcohol, or illicit drug. Family history significant for diabetes mellitus, cervical and brain cancer. Physical exam was unremarkable including neurologic system. Magnetic resonance imaging of the brain showed 2.4 x 3.7 x 4.6 cm lobulated peripherally enhancing mass in the lateral left frontal lobe with vasogenic edema, localized mass effect, and slight rightward midline shift. A smaller lesion in the posterior inferior left temporal lobe suspicious for malignancy. CT of the chest, abdomen, and pelvis unremarkable. Patient underwent a stereotactically-guided craniotomy which revealed purulent drainage from mass. Cultures showed 2+ streptococcus intermedius and surgical pathology was consistent with abscess rather than a primary tumor or metastasis. Patients labs otherwise were unremarkable including immune deficiency work up. Patient initially started on dexamethasone and postoperatively ceftriaxone with clindamycin was added. His conditions improved within few days and discharged on ceftriaxone for 8 weeks.

Discussion

- Patient with brain lesion and vasogenic edema most commonly caused by malignancy or an infection including an abscess. Magnetic resonance imaging using diffusion weighted imaging can show restricted diffusion that helps to differentiate between a mass due to infection or neoplasm; however, it could still be very difficult. Any brain mass with vasogenic edema needs urgent treatment. Standard techniques including head elevation, intravenous mannitol, and hyperventilation should be started. Dexamethasone is used if the abscess is associated with edema and mass effect, impending cerebral herniation, or progressive neurological deterioration. Ventriculostomy or decompressive craniectomy can be attempted if all other treatments have been exhausted.
- Up to 2500 brain abscess cases per year reported in the US with contiguous source being the most common way of developing followed by hematogenous dissemination, head trauma, after any neurosurgical procedures, or cryptogenic. Routes of contiguous spread include direct extension through osteitis or via local lymphatics. Localization could be from otitis media, saphenous sinusitis, or dental infection.
- 30-50% of survivors are found to have neurological sequelae including seizures and hydrocephalus within 5 years. 5-10% of abscesses recur either due to inadequate or inappropriate antibiotics. Brain abscesses are still associated with high morbidity at about 20%. In a significant proportion of cases, source cannot be identified. The most frequent species causing brain abscesses includes *staphylococcus* and *streptococcus*. The most common location of abscess formation is the frontal lobe. Risk factors for

developing a brain abscess include: congenital heart disease, meningitis, chronic middle ear and sinus infections, dental infections, infections of the face, or head injury.

Conclusion

We present a case of a young immunocompetent male with brain abscess, initially thought to be malignancy due to magnetic resonance imaging finding. Brain biopsy might be the only way to confirm the diagnosis and differentiate between the two conditions.

Burger and AKI from uncommon cause

Alex Peña Garcia, MS3, UAB Huntsville Regional Medical Campus; Sabrina Matosz, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical; Parekha Yedla, MD, Associate Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus

LEARNING OBJECTIVES:

1. Identify unusual cause of bacteremia in the Madison County area.
2. Bring awareness of recent foodborne Salmonella outbreaks.

CASE PRESENTATION:

Patient is a 75-year-old male with nausea, vomiting, and diarrhea with a past medical history of coronary artery disease and prostate cancer. His symptoms began four days prior to admission. He ate a cheeseburger a day prior to the onset of symptoms and subsequently developed severe watery, non-bloody, non-mucoid, dark brown diarrhea; about twenty episodes per day. He later developed sudden onset nausea and multiple episodes of non-bloody vomiting. Vomiting resolved two days prior to admission, but the diarrhea persisted. He had a 17-pound weight loss during this period. He denied travel or sick contacts.

His HR was 57 and BP was 81/39. Mucus membranes were dry, and abdomen was non-tender, non-distended with hyperactive bowel sounds, but no guarding or rigidity. His CBC was unremarkable. Sodium 127, potassium 3.3, chloride 77, CO₂ 16, BUN 91, creatinine 9.6, anion gap 34. Chest x-ray and CT abdomen and pelvis showed no acute changes.

Patient was found to be in AKI secondary to severe volume contraction and was started on IV fluids and empiric antibiotics (Flagyl and Rocephin). Blood and stool cultures two days after admission came back positive for Salmonella B. Infectious Diseases was consulted and recommended 2 g Rocephin IV for Salmonella bacteremia. The patient's vitals normalized, and creatinine trended down to 3.5 by day three of admission. Repeat cultures showed no growth by day four of admission and the metabolic acidosis resolved. Patient was diagnosed with diabetes mellitus during admission, and elevated creatinine was likely due to chronic kidney disease and hypovolemia. Patient was discharged on ciprofloxacin 800 mg BID for 7 days and advised to follow up with Infectious Disease in two weeks.

DISCUSSION:

Salmonella genus are flagellated rod-shaped Gram-negative facultative anaerobes. Annually, there are an estimated 1.3 billion cases of Salmonella gastroenteritis, leading to approximately 3 million deaths worldwide. However, it is most prevalent in low-income countries. In North America, the incidence is less than 10 per 100,000 per year. Non-typhoidal Salmonella species are important food borne pathogens with acute gastroenteritis being the most common clinical manifestation frequently transmitted through poultry products. Invasion beyond the gastrointestinal tract (i.e. endovascularly, lungs, heart, urinary tract and central nervous system) occurs in approximately 1% of the patients with non-typhoidal Salmonella resulting in bacteremia. In various studies, Salmonella bacteremia occurs in the immunosuppressed, those aged 65 and older with underlying chronic disease, or children aged five or younger.

Despite the severe nature of invasive salmonellosis, there has been no recent published population-based determination of US incidences of invasive salmonella infection due to its low frequency in the US. Most published data are from sub-Saharan Africa and Asia. This case is important from a public health standpoint as there have been several cases of salmonellosis across the country necessitating recall (according to the CDC, 223 hospitalizations resulted from recent outbreaks associated with contaminated chicken, beef, and turkey products). As per our lab statistics, this was only the second reported case of Salmonella bacteremia in our area within the last year. It is necessary to consider Salmonella as an infectious cause of severe gastroenteritis in light of the recent outbreaks resulting in 741 total foodborne salmonella infections.

Learning Objectives

1. Identify unusual cause of bacteremia in the Madison County area.
2. Bring awareness of recent foodborne Salmonella outbreaks.

Clinical Vignette

Patient is a 75-year-old male with nausea, vomiting, and diarrhea with a past medical history of coronary artery disease and prostate cancer. His symptoms began four days prior to admission. He ate a cheeseburger a day prior to the onset of symptoms and subsequently developed severe watery, non-bloody, non-mucoid, dark brown diarrhea, about twenty episodes per day. He later developed sudden onset nausea and multiple episodes of non-bloody vomiting. Vomiting resolved two days prior to admission, but the diarrhea persisted. He had a 17-pound weight loss during this period. He denied travel or sick contacts.

His HR was 57 and BP was 81/39. Mucus membranes were dry, and abdomen was non-tender, non-distended with hyperactive bowel sounds, but no guarding or rigidity. His CBC was unremarkable. Sodium 127, potassium 3.3, chloride 77, CO₂ 16, BUN 91, creatinine 9.6, anion gap 34. Chest X-ray and CT abdomen and pelvis showed no acute changes.

Discussion

Salmonella genus are flagellated rod-shaped Gram-negative facultative anaerobes [Figure 1 (A) and (B)]. Annually, there are an estimated 1.3 billion cases of Salmonella gastroenteritis, leading to approximately 3 million deaths worldwide. However, it is most prevalent in low-income countries. In North America, the incidence is less than 10 per 100,000 per year. Non-typhoidal Salmonella species are important food borne pathogens with acute gastroenteritis being the most common clinical manifestation frequently transmitted through poultry products. Invasion beyond the gastrointestinal tract (i.e. endovascularly, lungs, heart, urinary tract and central nervous system) occurs in approximately 1% of the patients with non-typhoidal Salmonella resulting in bacteremia. In various studies, Salmonella bacteremia occurs in the immunosuppressed, those aged 65 and older with underlying chronic disease, or children aged five or younger.

Despite the severe nature of invasive salmonellosis, there has been no recent published population-based determination of US incidences of invasive salmonella infection due to its low frequency in the US. Most published data are from sub-Saharan Africa and Asia.

This case is important from a public health

standpoint as there have been several cases of salmonellosis across the country necessitating recall (according to the CDC, 223 hospitalizations resulted from recent outbreaks associated with contaminated chicken, beef, and turkey products) [Figure 2 (A) and (B)]. As per our lab statistics, this was only the second reported case of Salmonella bacteremia in our area within the last year. It is necessary to consider Salmonella as an infectious cause of severe gastroenteritis in light of the recent outbreaks resulting in 741 total foodborne salmonella infections.

People infected with the outbreak strain of Salmonella infants, by state of residence, as of February 19, 2019 (n=129).



Figure 2 (A)

People infected with the outbreak strain of Salmonella Newport, by state of residence, as of March 21, 2019 (n=403)



Figure 2 (B)

Figure 2: (A) Map of Salmonella infection linked to raw chicken products. (B) Map of Salmonella infections linked to ground beef



Figure 1 (A)



Figure 1 (B)

Figure 1: (A) Gram stain image of Salmonella enteritidis demonstrating Gram-negative properties. (B) Scanning electron micrograph image of Salmonella enteritidis demonstrating flagella

Catch your Breath with a Classic Crisis

Forrest Gamble, MS3, UAB Huntsville Regional Medical Campus; Dennis Sehgal, MD, PGY2, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Roger D. Smalligan, MD, Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus

Learning Objectives: (1) Recognize the signs and symptoms of primary adrenal insufficiency
(2) Understand the treatment of adrenal crisis

Case Presentation: A 28yo Mexican female presented to the ED with progressive shortness of breath and hyperpigmentation. She reported headaches, blurry vision and abdominal pain over the past week. She denied dysuria, fever, chills, diarrhea, or constipation. She was not taking any medications and denied current drug use. PMH: difficult to ascertain despite use of translator but history of Addison's disease, hypothyroidism, and methamphetamine abuse. PE: frail and in mild distress, BP 72/49, P 72, R 14, T 37C, O2 saturation 100% on RA. Marked hyperpigmentation of skin, gums, and tongue; guarding of LLQ, otherwise normal exam. Labs: WBC 4.5, with normal differential, hemoglobin 11.2, Na 123, K 4.7, Bicarb 21, BUN 18, Cr 0.5, glucose 87, Ca 9.5, TSH 18, FT4 0.8 (0.8-1.7); urine drug screen negative, HIV negative, UA with 187 leukocytes, bacteria and nitrites pos. ACTH 39 (normal). Imaging: CXR and CT abdomen/pelvis was normal. Hospital course: The patient was felt to be in adrenal crisis and was cultured, given ceftriaxone, 3L of NS IVF and hydrocortisone 100mg q8h. She improved rapidly, BP normalized, sodium rose to 135 over 2 days and she was discharged later with antibiotics, a steroid taper to 20 mg of hydrocortisone BID, fludrocortisone 0.1 mg and DHEA 20mg daily and close follow up.

Discussion: This case illustrates an adrenal crisis, which can be life threatening if not treated promptly. The chief complaint of shortness of breath is not included in the current literature as a symptom of adrenal insufficiency, but Dr. Thomas Addison, in his original description, observed patients as having "the breathing painfully hurried by the slightest exertion." She did report many of the other signs and symptoms of a patient with adrenal insufficiency including weakness, fatigue, abdominal pain, anorexia, and nausea. Her hyperpigmentation and hypoaldosteronism (evidenced by hyponatremia and high normal potassium) indicate she likely had primary adrenal insufficiency, which is most commonly caused by autoimmune destruction of the adrenals in the USA also called Addison's disease. Worldwide, including in Mexico, TB continues to be the most common cause of adrenal destruction, but our patient did not report or have evidence of TB. She was off treatment for Addison's and had the added stress of an acute UTI, which led to the classic adrenal crisis. Treatment of adrenal crisis is exactly as seen in our case: isotonic IV fluids and corticosteroids along with treatment of any inciting condition which in this case was the UTI. Long-term therapy includes short or long acting corticosteroid therapy with fludrocortisone for mineralocorticoid effect. In women, DHEA is recommended to help with psychological well being. Although adrenal insufficiency is rare, it is essential for physicians to recognize the sometimes subtle presentation of the disease.



Catch your Breath with this Classic Crisis

Forrest Gamble¹, Dennis Sehgal², Roger D. Smalligan⁴

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2. Family Medicine UAB-Huntsville, Huntsville, AL, United States

Learning Objectives

- (1) Recognize the signs and symptoms of primary adrenal insufficiency
- (2) Understand the treatment of adrenal crisis

Case Presentation

- HPI: A 28 yo Mexican female presented to the ED with progressive shortness of breath and hyperpigmentation
- She reported headaches, blurry vision and abdominal pain over the past week
- No diarrhea, fever, chills, diarrhea, or constipation. She was not taking any medications and denied current drug use.
- PMH: difficult to ascertain despite use of translator but history of Addison's disease, hypothyroidism, and methamphetamine abuse.
- PE: frail and in mild distress, BP 72/49, P 72, R 14, T 37C, O2 sat 100% on RA. Marked hyperpigmentation of skin, gums, and tongue; guarding of LLQ, otherwise normal exam.
- Labs: WBC 4.5, with normal differential, hemoglobin 11.2, Na 123, K 4.7, BICarb 21, BUN 18, Cr 0.5, glucose 87, Ca 9.5, TSH 18, FT4 0.8 (0.8-1.7); UDS negative, HIV negative, UA with 18% leukocytes, bacteria and nitrites pos. ACTH 39 (normal).
- Imaging: CXR and CT abdomen/pelvis was normal.
- Hospital course: The patient was felt to be in adrenal crisis and was cultured, given ceftriaxone, 3L of NS IVF and hydrocortisone 100mg q8h.
- She improved rapidly, BP normalized, sodium rose to 135 over 2 days and she was discharged later with antibiotics, a steroid taper to 20 mg of hydrocortisone BID, fluocortisone 0.1 mg and DHEA 20mg daily and close follow up



Case Presentation Continued

Discussion

- This case illustrates an adrenal crisis, which can be life threatening if not treated promptly.
- Dr. Thomas Addison, in his original description, observed patients as having "the breathing painfully hurried by the slightest exertion."
- She did report many of the other signs and symptoms of a patient with adrenal insufficiency including weakness, fatigue, abdominal pain, anorexia, and nausea.
- Her hyperpigmentation and hypoadosteronism (evidenced by hyponatremia and high normal potassium) indicate she likely had primary adrenal insufficiency, which is most commonly caused by autoimmune destruction of the adrenals in the USA also called Addison's disease. Worldwide, TB continues to be the most common cause of adrenal destruction¹
- She was off treatment for Addison's and had the added stress of an acute UTI, leading to the adrenal crisis.
- Treatment of adrenal crisis: isotonic IV fluids and corticosteroids along with treatment of any inciting condition¹
- Long-term therapy includes short or long acting corticosteroid therapy with fluocortisone for mineralocorticoid effect. In women, DHEA is recommended to help with psychological well being.
- Although adrenal insufficiency is rare, it is essential for physicians to recognize the sometimes subtle presentation of the disease

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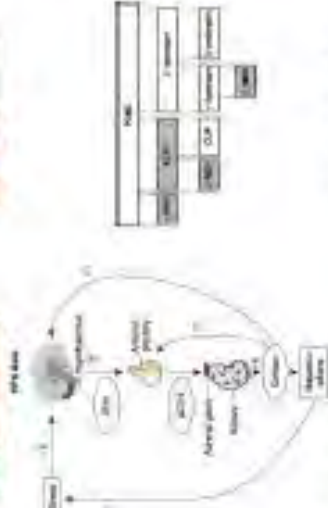


Figure 1: HPA Axis

Figure 2: Endocrine of Adrenal

Classic Findings May Not Be Typical: A Rare Case of Cutaneous Larva Migrans

Andrei Lojek OMS IV Alabama College of Osteopathic Medicine; Erin Britt, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical; Ali Hassoun, MD, Infectious Disease Specialist, Alabama Infectious Diseases Center, UAB Huntsville Regional Medical Campus

CASE:

History of Present Illness:

A Caucasian male in his late twenties, presented for evaluation of a foot rash that gradually worsened over the past 1.5 months.

- Described as painful, blistering, and pruritic rash on feet.
- No history of previous rashes or condition like this.
- Evaluated at a walk-in clinic. Received steroids, and antibiotics without resolution.
- A second trial of antibiotics and antifungal cream failed to alleviate symptoms.
- Next a dermatologist prescribed albendazole cream.
- Symptoms continued to gradually worsened, despite various treatment efforts.
- Referred to Infectious Disease.
- Pertinent history: the patient returned from Jamaica 2 weeks prior to initiation of symptoms; several friends who accompanied the patient on the trip reported a similar rash.

Past Medical History:

- No medical or surgical history. Denies any home medications or allergies.
- He owns two outside dogs. Social history otherwise non-contributory.

Review of Systems:

- Denies any fever, chills, weight changes, appetite changes, cough, dyspnea, abdominal pain, nausea, vomiting, diarrhea, hematochezia, melena, urinary changes, myalgias, arthralgias, edema, fatigue, or neurologic changes throughout time period that rash was present.

Physical Exam:

- Vitals: Temp: 97.7°F; BP 105/76
- Exam: Unremarkable, except for lesions (as pictured) on feet. Early lesions (left) arose two weeks after return from Jamaica, while later (right) serpiginous lesions developed approximately 1.5 months after his return.

Course:

- The patient was prescribed two 3mg tablets of Ivermectin to be taken two days in a row. Follow up two weeks later revealed the rash had drastically improved.

DISCUSSION:

Our patient was referred to infectious disease after seeing several physicians for a worsening, pruritic foot rash that erupted two weeks after travel to Jamaica and had been refractory to broad therapeutic management. Having an uncommon parasitic infection, a generic appearing rash, along with lack of continuity in his care likely contributed to a delay in diagnosis and appropriate treatment. At the point of diagnosis, the maculopapular rash had advanced to a more classically described serpiginous

rash, which finally aided targeted treatment. However, the patient's travel history, time line of rash eruption, along with travel partners exhibiting similar symptoms, would have been sufficient enough information to suspect parasitic infection.

Any new rash or cutaneous symptoms in the presence of travel to Africa, Asia, South America, or the Caribbean should prompt the clinician to include hookworms as a source of infection. Approximately one-sixth of the global population are infected by helminths transmitted via soil (eg *Ascaris lumbricoides*), thus, its inclusion in the differential is not unwarranted. With prevalence rates so high worldwide, it is important for the clinician to maintain a high degree of suspicion for atypical causes especially after travel to endemic areas and where bare feet or light footwear (eg sandals) are anticipated.

Appropriate initial management in suspected hookworm infection of any kind is a systemic anti-helminthic therapy. Ivermectin (200mcg/kg orally) has cure rates after one administration between 94 and 100%³. The condition could have been treated much earlier in the course had a helminth infection been considered

INTRODUCTION

Case:
Parasitic infections are not common in the United States. For this reason, parasitic infection often remains quite low on the differential of many general practitioners. Early hookworm infection manifests as a pruritic maculopapular rash often focused between the toes where the larvae penetrate the skin. The classic subcutaneous "serpiginous track" caused by larval migration is often not present during the course of the infection. Suspicion for helminth infection in anyone with cutaneous symptoms should be elevated in patients with recent travel history to the Caribbean or Latin America, but especially to sub-Saharan regions of Africa and some parts of Asia where prevalence is highest. Ancylostoma duodenale and Necator americanus are typical culprits, with the former being indigenous to Mediterranean countries and the Middle-East, and the latter, North America, South America, Africa, and the South Pacific.¹

Early recognition of hookworm infections by United States physicians can be challenging for several reasons including: low prevalence, rare diagnostic exposure, and ambiguous or subtle symptoms early in presentation. In countries with poor access to medicine, hookworm infection is a frequent cause of anemia. Symptoms progress concurrently with the parasitic life cycle. Cutaneous infection by the filarial parasite form occurs when the host comes into contact with infected feces. The parasite migrates through the bloodstream to the lungs and into the alveolar space. According to the pharynx, they are swallowed and move to the intestines to mature and feed on host blood. The adult form produces eggs that can be detected in the stool. Anemia is a common cause of morbidity in the chronically infected, but nausea, vomiting, diarrhea, and increased flatulence are often reported.



Image 1: Early lesions on medial aspect of right foot show blistered appearance with mild erythema. This appeared approximately 2 weeks after the patient returned from Jamaica.

CASE

History of Present Illness:
A Caucasian male in his late twenties, presented for evaluation of a foot rash that gradually worsened over the past 1.5 months.
-Described as painful, blistering, and pruritic rash on feet.
-No history of previous rashes or condition like this.
-Evaluated at a walk-in clinic. Received steroids, and antibiotics without resolution.
-A second trial of antibiotics and antifungal cream failed to alleviate symptoms.
-When a dermatologist prescribed albendazole cream. Symptoms continued to gradually worsened, despite various treatment efforts.
-Referred to Infectious Disease.
-Pertinent history: the patient returned from Jamaica 2 weeks prior to initiation of symptoms; several friends who accompanied the patient on the trip reported a similar rash.

Past Medical History:

- No medical or surgical history. Denies any home medications or allergies.
- He owns two outside dogs. Social history otherwise non-contributory.

Review of Systems:

- Denies any fever, chills, weight changes, appetite changes, cough, dyspnea, abdominal pain, nausea, vomiting, diarrhea, hematochezia, melena, urinary changes, myalgias, arthralgias, edema, fatigue, or neurologic changes throughout time period that rash was present.

Physical Exam

- Vitals: Temp: 97.7°F; BP: 105/76
- Exam: Unremarkable, except for lesions (as pictured) on feet. Early lesions (left) arose two weeks after return from Jamaica; while later (right) serpiginous lesions developed approximately 1.5 months after his return.

Course:

- The patient was prescribed two 3mg tablets of Ivermectin to be taken two days in a row. Follow up two weeks later revealed the rash had drastically improved.

DISCUSSION

Our patient was referred to infectious disease after seeing several physicians for a worsening, pruritic foot rash that erupted two weeks after travel to Jamaica and had been refractory to broad therapeutic management. Having an uncommon parasitic infection, a generic appearing rash, along with lack of continuity in his care likely contributed to a delay in diagnosis and appropriate treatment. At the point of diagnosis, the maculopapular rash had advanced to a more classically described serpiginous rash, which finally aided targeted treatment by the dermatologist. However, the patient's travel history, time line of rash eruption, along with travel partners exhibiting similar symptoms, would have been enough information to suspect parasitic infection.

Any new rash or cutaneous symptoms in the presence of travel to Africa, Asia, South America, or the Caribbean should prompt the clinician to include hookworms as a source of infection. Approximately one-sixth of the global population are infected by helminths transmitted via soil (eg. Ascaris lumbricoides), thus, its inclusion in the differential is not unwarranted. With prevalence rates so high worldwide, it is important for the clinician to maintain a high degree of suspicion for atypical causes especially after travel to endemic areas and where bare feet or light footwear (eg sandals) are anticipated.

The case depicts a situation in which an unexpected condition led to misdiagnosis, incorrect management, and worsening of the condition for almost two months before the correct diagnosis and proper therapy were established. With the travel history, several unsuccessful rounds of therapy, and a developing serpiginous rash, the patient was prescribed a topical albendazole to treat a suspected helminth infection. After a final evaluation with infectious disease, the classic rash pattern and, again, his history, prompted further treatment. Appropriate initial management in suspected hookworm infection of any kind is a systemic anti-helminthic therapy. Ivermectin (200mcg/kg orally) has cure rates after one administration between 94 and 100%.² The condition could have been treated much earlier in the course had a helminth infection been considered.

CONCLUSION

- Early stages of cutaneous hookworm infection are not obvious and are often confused with other diagnoses. A high degree of suspicion must be maintained when cutaneous symptoms arise after travel to regions endemic to hookworm species.
- Cutaneous larva migrans may be the textbook presentation, but is actually far less common in hookworm infection than medical education portrays to students in training.
- A pruritic maculopapular rash presenting within a month after traveling to hookworm endemic areas should greatly increase suspicion of hookworm as a cause.
- Reported treatment failure by several clinicians should increase suspicion of an atypical cause in the US, especially with history of travel outside the country.⁴
- One or two dose oral ivermectin or albendazole is the treatment of choice. Topical management is less effective and requires a longer course.

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- Travel medicine. *UpToDate*.
- Travel medicine. *UpToDate*.



Image 2: Later lesions on the dorsal and lateral aspect of the right foot. The classic serpiginous lesions of a hookworm infection are readily apparent under the skin.

Cryptococcal Pneumonia in immunocompetent host

Mohamed Hasan, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical Campus; Ali Hassoun, MD, Infectious Disease Specialist, Alabama Infectious Diseases Center, UAB Huntsville Regional Medical Campus

Clinical Vignette

Learning Objective 1: Cryptococcus pneumonia is a fungal infection caused by inhalation of cryptococcus typically in immunocompromised patients and rarely in immunocompetent. In immunocompromised host, it usually causes systemic manifestation including CNS involvement whereas immunocompetent host, it causes mild pulmonary infections.

Case: 44-year-old Caucasian male with a history of stroke with residual left-sided weakness and seizure disorder presented with chronic cough for 4 months associated with mild clear sputum. He also had upper left chest pain for 7 months duration. He has a significant history of smoking 26-year pack. There is no history travel or sick contact, no history of weight loss, and review of systems was negative. On physical examination he was afebrile and other findings were unremarkable. Chest Computed Tomography showed irregular peri bronchial mass with interstitial nodular consolidation measuring 3.1 x 2.3 cm in the left upper lobe with air bronchogram within the mass, left posterior apical pulmonary nodules, and small separate several adjacent nodules. Bronchoscopy was done, and histopathology was suggestive of fungal elements like Cryptococcus. Laboratory studies showed positive cryptococcal antigen with titer 1:320, he was HIV-seronegative, CD4 and CD8 were within normal limits, and immunoglobulin level was normal. Diagnosis of Cryptococcal Pneumonia was made, and the patient was treated with fluconazole and his symptoms improved.

Discussion: Infection with Cryptococcus occurs through inhalation of aerosolized basidiospores which can be found in soil and pigeon's excreta. Most cases caused by ubiquitous encapsulated yeast, Cryptococcus neoformans whereas cryptococcus gattii account for a fewer quantity of cases and mainly in immunocompetent hosts. The infection initially is localized in the lung then it disseminates depending on the immune status of the individual and causes systemic disease that involves the central nervous system (CNS), bone, skin, and viscera. The majority of pulmonary cryptococcal pneumonia are seen in immunocompromised patients including human immunodeficiency virus (HIV), post-transplant, hematological malignancy, and or other disorder associated with cell-mediated immune dysfunction. Pulmonary cryptococcal manifestations range from asymptomatic to severe respiratory distress which makes the diagnosis challenging. Immunocompetent individuals usually asymptomatic or develop mild respiratory symptoms. However, in immunocompromised, progression to acute respiratory distress syndrome (ARDS) can occur. The radiological finding of pulmonary cryptococcal has a variety of manifestations that include clustered nodules, solitary pulmonary nodules, scattered nodules, segmental or lobar consolidation.

Diagnosis of cryptococcal pneumonia is based on the radiographical finding, fungal culture, serum cryptococcal antigen, and histopathology. Fungal culture obtained from expectorated sputum and

bronchoalveolar lavage. Detecting serum and cerebrospinal fluid cryptococcal capsular polysaccharide by latex agglutination, enzyme-linked immunosorbent assay (ELISA), or lateral flow assay has high sensitivity and specificity values.

The Infectious Diseases of America Society (IDSA) guidelines recommended fluconazole for 6-12 months in patients with mild to moderate disease. Itraconazole, voriconazole, and posaconazole are acceptable alternative therapies. For severe pulmonary diseases, management includes amphotericin B plus flucytosine for two weeks followed by fluconazole for a minimum of 8 weeks.

Learning objective:

- Cryptococcal pneumonia is a fungal infection caused by inhalation of cryptococcus typically in immunocompromised patients and rarely in immunocompetent.
- In immunocompetent host, it usually causes systemic manifestations including CNS involvement whereas immunocompetent host, it causes mild pulmonary infections.

Case report

- 44-year-old Caucasian male with a history of stroke with residual left-sided weakness and seizure disorder presented with chronic cough for 4 months associated with mild chest tightness. He also had upper left chest pain for 7 months duration. He has a significant history of smoking 25-year pack. There is no history of travel or sick contact, no history of weight loss, and review of systems was negative.
- On physical examination he was afebrile and other findings were unremarkable.
- Chest Computed Tomography showed irregular peribronchovascular thickening with interstitial consolidation measuring 3.1 x 2.5 cm in the left upper lobe with air bronchograms within the mass, left posterior apical pulmonary nodules, and small separate several adjacent nodules.
- Bronchoscopy was done, and histopathology was suggestive of fungal elements like Cryptococcus.
- Laboratory studies showed positive cryptococcal antigen with titer 1:320, he was HIV-negative, CD4 and CD8 were within normal limits, and serum hemoglobin level was normal.
- Diagnosis of Cryptococcal Pneumonia was made, and the patient was treated with fluconazole and his symptoms improved.

Discussion

- Infection with Cryptococcus occurs through inhalation of aerosolized basidiospores which can be found in soil and pigeon's excreta.
- Cryptococcus gatii occurs rarely in tropical and sub-tropical regions. Most cases caused by ubiquitous encapsulated yeast, cryptococcus neoformans whereas cryptococcus gatii account for a lower quantity of cases and mainly in immunocompetent host.
- The infection initially is localized in the lung, then it disseminates depending on the immune status of the individual and causes systemic disease that involves the central nervous system (CNS), bone, skin, and viscera. The majority of pulmonary cryptococcal pneumonia are seen in immunocompromised patients including human immunodeficiency virus (HIV), post-transplant, hematological malignancy, and/or other disorder associated with cell-mediated immune dysfunction.
- Pulmonary cryptococcal manifestations range from asymptomatic to severe respiratory distress which make the diagnosis challenging. Immunocompetent individuals usually asymptomatic or develop mild respiratory symptoms include fever, productive cough, dyspnea. However, in immunocompromised, progression to acute respiratory distress syndrome (ARDS) can occur.

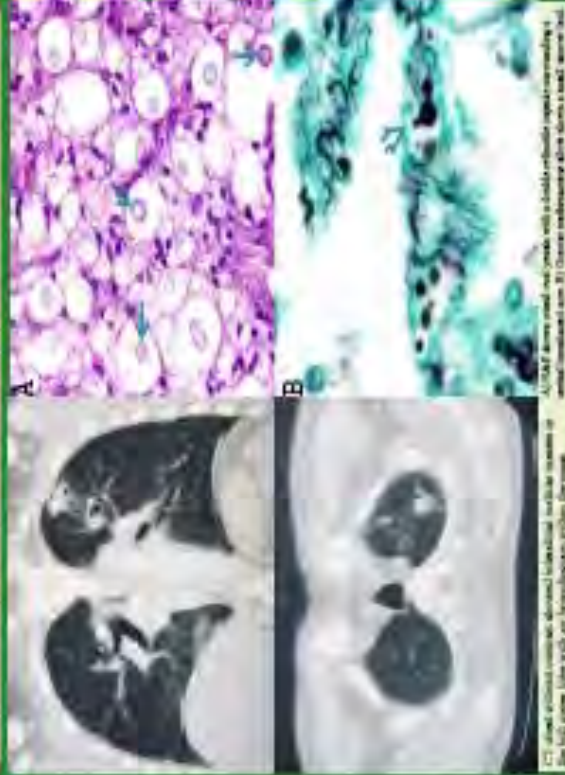


Figure 1: (A) Chest CT scan showing a large, irregular, peribronchovascular thickening with interstitial consolidation in the left upper lobe with air bronchograms within the mass. (B) Histopathology showing fungal elements (Cryptococcus) suggestive of Cryptococcus.

- Diagnosis of cryptococcal pneumonia is based on radiological finding, fungal culture, serum cryptococcal antigen, and histopathology.
- Fungal culture obtained from sputum, sputum and bronchoalveolar lavage.
- Dehiscence serum and cerebrospinal fluid cryptococcal capsular polysaccharide by latex agglutination, enzyme-linked immunosorbent assay (ELISA), or lateral flow assay has high sensitivity and specificity values.
- The radiological finding of pulmonary cryptococcal has a variety of manifestations that include clustered nodules, solitary pulmonary nodules, scattered nodules, segmental or lobar consolidations. Involvement of the pleura and lymphadenopathy are seen more commonly in immunocompromised patients.
- The Infectious Diseases of America Society (IDSA) guidelines recommended:
 - Fluconazole (400 mg daily orally) for 6-12 months in patient with mild to moderate disease.
 - Isotretinoin, voriconazole, and posaconazole are acceptable alternative therapies.
 - For severe pulmonary disease, management is similar to central nervous system disease which includes amphotericin B plus flucytosine for two weeks followed by fluconazole for minimum 8 weeks.
- Previously positive serum cryptococcal antigen titers are not criteria for cessation of therapy.

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Delayed Diagnosis of Annular Pancreas in 11 year-old girl with DiGeorge Syndrome – Case Report

Mary Margaret Barr, MS3, UAB Huntsville Regional Medical Campus; James Gilbert MD, Clinical Professor of Surgery, UAB / Huntsville Hospital; Zaria Murrell, MD, Clinical Associate Professor of Surgery, UAB / Huntsville Hospital

CASE

DiGeorge Syndrome is a collection of symptoms stemming from a heterozygous microdeletion on chromosome 22. Most patients have palate abnormalities and/or cardiac defects, but additional features vary in presentation. An eleven-year-old girl with DiGeorge syndrome presented to Pediatric Surgery with a history of episodes of vomiting since birth, and a recent hospitalization for aspiration pneumonia. Upper GI series demonstrated a large, distended stomach and duodenum with severely delayed passage of contrast into distal bowel.

At birth, the patient presented with a cleft palate, but no cardiac abnormalities. She was later confirmed to have DiGeorge Syndrome. However, she had recurrent non-bilious emesis, sometimes projectile, throughout infancy and childhood. Pyloric ultrasound was negative for pyloric stenosis, and her presumed GERD was treated medically. Over time, she adjusted her diet to primarily eat sauces and other soft foods, avoiding meat entirely. While being evaluated for recent aspiration pneumonia following emesis, a dilated duodenum and stomach was found on upper GI series, and she was referred to Pediatric Surgery. Originally suspecting a duodenal web obstructing the duodenum, the patient was scheduled for surgery to correct the obstruction. It was discovered during surgery that the obstruction was caused by an annular pancreas. The obstruction was treated by a duodenoduodenostomy using diamond anastomosis. By two week follow-up, the patient advanced to a mechanical soft diet and tolerated full liquids well.

CONCLUSIONS

Most annular pancreas patients present with complete obstruction in infancy. This patient presented with moderate symptoms in early adolescence. She was able to adjust her diet to allow for foods that could fit through the constricted lumen and grow appropriately.

CLINICAL SIGNIFICANCE

DiGeorge syndrome is loosely associated with various GI malformations, but annular pancreas has not been reported elsewhere in medical literature.

Delayed Diagnosis of Annular Pancreas in 11 year-old girl with DiGeorge Syndrome

Mary Margaret Barr¹, James Gilbert², Zaria Murrell²

¹University of Alabama at Birmingham School of Medicine, ²Huntsville Hospital Pediatric Surgery



Objectives:

- Evaluation of pediatric patients with treatment-resistant GERD should include investigation for mechanical causes.
- Lack of classic connection between a syndrome and pathophysiology should not rule out potential diagnosis.

Overview of DiGeorge Syndrome:

- Collection of symptoms stemming from a heterozygous microdeletion on chromosome 22
- Palate abnormalities and/or cardiac defects, but additional features vary in presentation
- Loose association with abdominal pathologies

Overview of Annular Pancreas:

- Most cases present with complete obstruction during infancy.
- Most adults are asymptomatic and are found incidentally.
- Should be on differential for any prolonged history of non-bilious vomiting associated with meals.

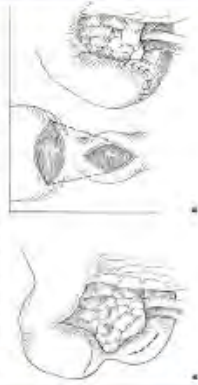
Patient Presentation:

At birth, the patient presented with a cleft palate, but no cardiac abnormalities. She was later confirmed to have DiGeorge Syndrome. However, she had recurrent non-bilious emesis, sometimes projectile, throughout infancy and childhood. Pyloric ultrasound was negative for pyloric stenosis, and her presumed GERD was treated medically. Over time, she adjusted her diet to primarily eat sauces and other soft foods, avoiding meat entirely. While being evaluated for recent recurrent aspiration pneumonia episodes, a dilated duodenum and stomach were found on upper GI series, and she was referred to Pediatric Surgery. Originally suspecting a windsock-type duodenal web, the patient was scheduled for surgery to correct the obstruction. During surgery, we found that the obstruction was caused by an annular pancreas. The obstruction was treated with a duodenoduodenostomy using diamond anastomosis. By her two week follow-up, the patient advanced to a mechanical soft diet and at 3 months after surgery is tolerating all foods.



Classic Double-Bubble sign of duodenal obstruction on CT (above) and GI series (right).

Duodenoduodenostomy:



Important Findings:

- Only known case of association between DiGeorge Syndrome and Annular Pancreas.
- Ability to adjust treatment plan due to discovery of annular pancreas in timely and efficient manner.

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Delayed diagnosis of pheochromocytoma: the importance of reevaluating the differential and the dangers of labeling patients

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Learning Objective 1: Illustrate importance of establishing broad differentials and reevaluating initial diagnoses despite pressures to accept a single exclusive etiology.

Learning Objective 2: Highlight the dangers of labeling patients as non-adherent to assigned medical regimen.

Case: A 53-year-old, wheelchair-confined, African American female nursing home resident with a past medical history of “brittle” type 2 diabetes, labile hypertension, prior stroke with residual right hemiparesis and dysphasia, breast cancer, seizure disorder, and diabetic autonomic neuropathy (DAN), presented to Huntsville Hospital for acute-onset confusion. She was found to be hypoglycemic [blood glucose: 24] and was admitted for further management. This was the most recent of multiple hospitalizations over the previous three years for hypertensive crisis and/or seizures assumed to be precipitated by hypoglycemia. Former admissions were attributed to non-adherence and acute dysautonomia. Despite medical oversight and appropriate dosing of various antihypertensive and insulin regimens, she continued to exhibit extreme fluctuations in blood pressure (BP range: 89/74 to 254/149) and blood glucose (range: 79 to 489) throughout admission. Other vital signs remained relatively stable with mild, persistent tachycardia (range: 105-125), oxygen saturations in mid-nineties, and no fever recorded throughout. She denied palpitations, diaphoresis, warm sensations, episodic headaches, or surges of anxiety. Review of records revealed an incidental LUQ mass seen on breast cancer staging CT in 2014. EUS with fine-needle aspiration was subsequently performed in 2014 and described a multi-cystic, hypoechoic lesion in the pancreatic body. The mass was labeled as a non-malignant, benign pancreatic cyst and was followed by serial abdominal CT scans biannually with annual follow-up with GI specialist. From 2014-2018 the mass remained stable, measuring approximately 9.5x7.7x7.2cm. On day 9 of hospitalization, urinary metanephrines were ordered and returned >58,500 (mcg/24hr). Subsequent I-123 MIBG scan confirmed increased uptake in the region of the LUQ mass consistent with pheochromocytoma (PCC). Surgery was scheduled after preoperative Prazosin management and a 427-gram left adrenal PCC was removed. The patient was discharged nine days post-op with no insulin requirement and no anti-hypertensive medications. Pt was scheduled for 2-week repeat urinary metanephrines and advised on annual testing.

Discussion: Our patient's presentation demonstrates the importance of establishing a broad differential that allows for consideration of diagnoses that may not assimilate perfectly into our textbook schemas of specific diseases. Our patient failed to exhibit the “classic symptoms” of episodic tachycardia, headaches, diaphoresis, and feelings of anxiety and instead manifested primarily with a glucose intolerance seen in only 35-50% of patients with PCC.¹ The delay in diagnosis was further instigated by long-history of reassurance from the radiology and gastroenterology services of the pancreatic origin of the cyst. Assumptions about the masses' origins led to a cognitive inhibition that blinded decision-making in a straightforward case of recalcitrant hypertension in the setting of a LUQ mass. Clinical critical-thinking was also initially impeded by a documented designation of the patient as “non-compliant,” which may or may not have been an accurate assessment. Regardless, these types of appraisals of culpability in a healthcare setting can serve as barriers to further diagnostic assessment, which may have dangerous consequences for patients.

Cryptococcal pneumonia in an immunocompetent host

Mehmet Hama, MD, Ali Hossain, MD, FACP, #1265A
Internal Medicine resident, OAB Pulmonary Medicine, AL 3, Alabama Infectious Disease Center, Birmingham, AL 3

Learning objectives

- Cryptococcal pneumonia is a fungal infection caused by infection of cryptococcus typically in immunocompromised patients and rarely in immunocompetent.
- In immunocompetent host, it usually causes systemic manifestations including CNS involvement whereas immunocompetent host, it causes mild pulmonary infections.

Case report

- 44-year-old Caucasian male with a history of stroke with residual left-sided weakness and seizure disorder presented with chronic cough for 4 months associated with mild chest tightness. He also had upper left chest pain for 7 months duration. He has a significant history of smoking 25-year pack. There is no history travel or sick contact, no history of weight loss, and review of systems was negative.
- On physical examination he was afebrile and other findings were unremarkable.
- Chest Computed Tomography showed irregular peribronchovascular thickening, nodular consolidation measuring 3.1 x 2.3 cm in the left upper lobe with air bronchogram within the mass, left posterior apical pulmonary nodules, and small separate several adjacent nodules.
- Bronchoscopy was done, and histopathology was suggestive of fungal elements like Cryptococcus.
- Laboratory studies showed positive cryptococcal antigen with titer 1:120, he was HIV-negative, CD4 and CD8 were within normal limits, and immunoglobulin level was normal.
- Diagnosis of Cryptococcal Pneumonia was made, and the patient was treated with fluconazole and his symptoms improved.

Discussion

- Infection with Cryptococcus occurs through inhalation of aerosolized basidiospores which can be found in soil and pigeon's excreta.
- Cryptococcus gatii occurs mainly in tropical and sub-tropical regions. Most cases caused by ubiquitous trophic yeast, cryptococcus neoformans whereas cryptococcus gatii account for a lower quantity of cases and mainly in immunocompetent host.
- The infection initially is localized in the lung then it disseminates depending on the immune status of the individual and causes systemic disease that involves the central nervous system (CNS), bone, skin, and viscera. The majority of pulmonary cryptococcal pneumonia are seen in immunocompromised patients including human immunodeficiency virus (HIV), post-transplant, hematological malignancy, and/or other disorder associated with cell-mediated immune dysfunction.
- Pulmonary cryptococcal manifestations range from asymptomatic to severe respiratory distress which make the diagnosis challenging. Immunocompetent individuals usually asymptomatic or develop mild respiratory symptoms include fever, productive cough, dyspnea. However, in immunocompromised, progression to acute respiratory distress syndrome (ARDS) can occur.

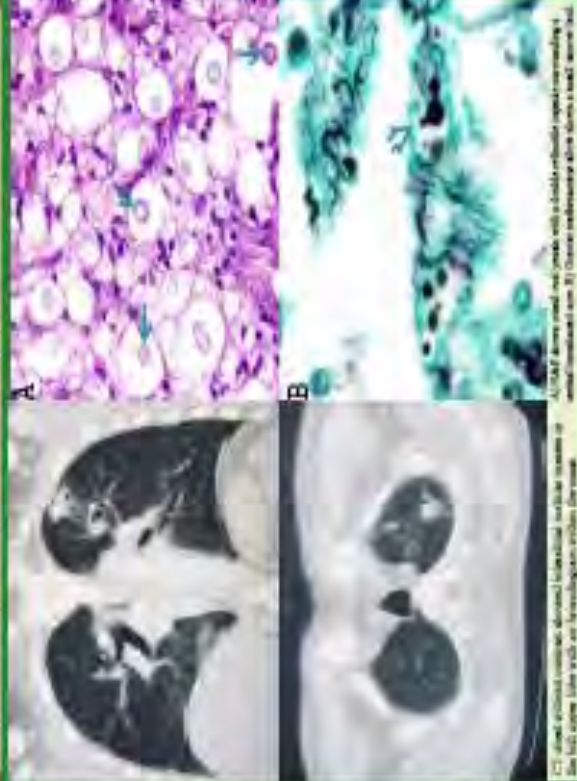


Figure 1: (A) Chest CT scan showing a large, irregular, enhancing mass in the left upper lobe with air bronchograms. (B) Histopathology showing numerous small, round, budding yeast cells with thick, refractile capsules, characteristic of Cryptococcus.

- Diagnosis of cryptococcal pneumonia is based on radiological finding, fungal culture, serum cryptococcal antigen, and histopathology.
- Fungal culture obtained from expectorated sputum and bronchoalveolar lavage.
- Identifying serum and cerebrospinal fluid cryptococcal antigen polysaccharide by latex agglutination, enzyme-linked immunosorbent assay (ELISA), or lateral flow assay has high sensitivity and specificity values.
- The radiological finding of pulmonary cryptococcal has a variety of manifestations that include clustered nodules, solitary pulmonary nodules, scattered nodules, segmental or lobar consolidation. Involvement of the pleura and lymphadenopathy are seen more commonly in immunocompromised patients.
- The Infectious Diseases of America Society (IDSA) guidelines recommended.
- Fluconazole (400 mg daily orally) for 6-12 months in patients with mild to moderate disease.
- Isoniazide, voriconazole, and posaconazole are acceptable alternative therapies.
- For severe pulmonary disease, management is similar to central nervous system disease which includes amphotericin B plus fluconazole for two weeks followed by fluconazole for minimum 8 weeks.
- Incidentally positive serum cryptococcal antigen tests are not criteria for commencement of therapy.

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Diagnostic challenges in a toddler struggling to walk after upper respiratory infection - case study of pediatric patient with Guillain-Barre syndrome (GBS)

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Introduction

Guillain-Barre syndrome (GBS) is a rare but important diagnosis to consider in children presenting with acute onset of neuropathic symptoms like paresthesia, progressive muscle weakness or difficulty walking. Systematic approach including combination of careful history and examination, and accurate interpretation of diagnostic testing is needed to recognize and differentiate GBS from other mimicking diagnosis.

It is important to recognize the variety and severity of the neurologic symptoms associated with GBS, especially with the diagnostic difficulties associated with the pediatric population

One proposed mechanism for GBS is that an antecedent infection evokes an immune response, which in turn cross-reacts with peripheral nerve components because of sharing of cross-reactive epitopes (molecular mimicry). The end result is an acute polyneuropathy.

Case Description

The case is one of a 2yo CM with history of asthma who presented during an upper respiratory tract infection with pain when he walks. Report URI symptoms were for 5 days prior to presentation with fever up to 103. Patient's mom later in hospital course gave a history of "food poisoning" after eating in a local Mexican restaurant 2 weeks prior to presentation. No history of recent vaccination. On presentation, patient work up was negative including normal WBC, normal bilateral lower x-ray and hip US. His ESR was mildly elevated at 22. On initial admission, patient was able to walk with support, had normal reflexes and normal tone. He also had tenderness with internal rotation of the left hip. Patient presentation was initial thought to be secondary to transient synovitis. On reevaluation within 24 hours of admission, patient had absent bilateral lower extremity reflexes and flaccid tone. MRI brain, cervical spine, thoracic spine and lumbar spine was done that was normal except for lumbar spine finding of avid enhancement of the cauda equine, Findings to be consistent with Guillain-Barre syndrome. Lumbar puncture was performed with CSF findings of total protein 90, glucose 56, lymphocytes 83, monocytes 16, RBC <1, TNC 2 and final CSF culture with no growth.

Outcome

Patient was treated with 5 doses of IVIG 400mg/kg/dose. Muscle tone gradually improved, however lower extremity reflex remain absent with continued inability to walk by the time of discharge. Pediatric neurology and physical therapy were both involved in patient's care throughout his hospital stay. He is planned to follow up with pediatric neurology within 2 weeks of discharge and continue physical therapy as outpatient.

Discussion

Guillain- Barre syndrome is considered a monophasic disease with several variants including acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor sensory axonal neuropathy, miller-fisher variant, Bickerstaff encephalitis, Polyneuritis cranialis and Pharyngeal-cervical-brachial weakness. Findings suggest that acute motor axonal neuropathy is an important subtype of guillain-barre syndrome seen in pediatric population [2]. GBS occurs world-wide with an annual incidence of 0.34 to 1.34 cases per 100,000 persons aged 18 years or less. While all age groups are affected, the incidence is lower in children than in adults. The incidence increases by approximately 20 percent with every 10-year increase in age beyond the first decade of life. GBS occurs rarely in children younger than two years of age, but can occur even in infants. Males are affected approximately 1.5 times more often than females in all age groups. The classic presentation of GBS begins with paresthesia in the toes and fingertips followed by lower extremity symmetric or modestly asymmetric weakness that may ascend over hours to days to involve the arms and, in severe cases, the muscles of respiration. In preschool-aged children, the most common symptoms are refusal to walk and pain in the legs [1]. Initial diagnosis is mostly done based on clinical presentation however lab, imaging and electrodiagnostic studies could also be useful in supporting the diagnosis. Findings consistent with GBS include: An elevated CSF protein (>45 mg/dL) with a normal CSF white blood cell count. Nerve conduction studies showing decreased amplitude of motor (and possibly sensory) responses, with normal conduction velocities. Contrast enhancement of the spinal nerve roots, cauda equina, or cranial nerve roots on MRI. The enhancement may be diffuse or predominantly involve the ventral (anterior) nerve roots.

Many disorders of the central nervous system, peripheral nervous system, neuromuscular junction and muscle can mimic GBS however a careful analysis of the clinical presentation, laboratory and imaging finding can help support the diagnosis of guillain-barre syndrome. The main modalities of therapy for Guillain-Barré syndrome include plasmapheresis and administration of intravenous immune globulin [1].



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Introduction

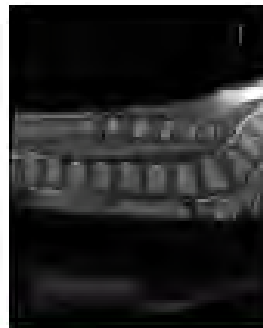
Guillain-Barre syndrome (GBS) is a rare but important diagnosis to consider in children presenting with acute onset of neuromuscular symptoms like periorbital, progressive muscle weakness or difficulty walking. Systematic approach including combination of case history and examination, and accurate interpretation of diagnostic testing is needed to recognize and differentiate GBS from other mimicking disorders. It is important to recognize the variety and severity of the neurologic symptoms associated with GBS, especially with the diagnostic difficulties associated with the pediatric population. One proposed mechanism for GBS is that an antecedent infection evokes an immune response, which in turn cross-reacts with peripheral nerve components because of sharing of cross-reactive epitopes (molecular mimicry). The end result is an acute polyneuropathy.

Case Description

This case is one of a 2yo GBS with history of asthma who presented during an upper respiratory tract infection with pain when he walks. Report URTI symptoms went for 5 days prior to presentation with fever up to 102. Patient's mom later in hospital course gave a history of "food poisoning" after eating in a local Mexican restaurant 2 weeks prior to presentation. No history of recent vaccination. On presentation, patient went up with negative including normal WBC, normal bilateral lower extremity and hip US. His GBS was initially elevated at 22. On initial admission, patient was able to walk with support, had normal reflexes and normal tone. He also had tenderness with internal rotation of the left hip. Patient presentation was initial thought to be secondary to transient syncope. On reevaluation within 24 hours of admission, patient had absent bilateral lower extremity reflexes and flaccid tone, left foot, cervical spine, thoracic spine and lumbar spine was done that felt normal except for lumbar spine finding of mild enhancement of the cauda equina. Findings to be consistent with Guillain-Barre syndrome. Lumbar puncture was performed with CSF findings of total protein 80, glucose 50, lymphocytes 23, monocytes 10, RBC -1, TNC 2 and low CSF culture with no growth.

Discussion

Guillain-Barre syndrome is considered a monophasic disease with several variants including acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor sensory axonal neuropathy, Miller-Fisher variant, Bickerstaff encephalitis, Polyneuropathy acralis and Pharyngeal-cervical-brachial weakness. Findings suggest that acute motor axonal neuropathy is an important subtype of Guillain-Barre syndrome seen in pediatric population [2]. GBS occurs worldwide with an annual incidence of 0.24 to 1.34 cases per 100,000 persons aged 15 years or less. While all age groups are affected, the incidence is lower in children than in adults. The incidence increases by approximately 20 percent with every 10-year increase in age beyond the first decade of life. GBS occurs rarely in children younger than two years of age, but can occur even in infants. Males are affected approximately 1.5 times more often than females in all age groups. The classic presentation of GBS begins with paresthesia in the feet and legs, followed by lower extremity symmetric or moderately asymmetric weakness that may ascend over hours to days to involve the arms and, if severe enough, the muscles of respiration.



Sagittal MRI of lumbar spine depicting enhancement of ventral nerve root



Cross section MRI of lumbar spine depicting enhancement of ventral nerve root

In preschool-aged children, the most common symptoms are refusal to walk and pain in the legs [1]. Initial diagnosis is mostly done based on clinical presentation however lab, imaging and electrodiagnostic studies could also be useful in supporting the diagnosis. Finding consistent with GBS include: An elevated CSF protein (and mg/dL) with a normal CSF white blood cell count. Nerve conduction studies showing decreased amplitude of motor (and possibly sensory) responses, with normal conduction velocities. Contrast enhancement of the spinal nerve roots, cauda equina, or cranial nerve roots on MRI. The enhancement may be diffuse or predominantly involve the ventral (anterior) nerve roots. Many disorders of the central nervous system, peripheral nervous system, neuromuscular junction and muscle can mimic GBS however a careful analysis of the clinical presentation, laboratory and imaging finding can help support the diagnosis of Guillain-Barre syndrome. The main modalities of therapy for Guillain-Barre syndrome include plasmapheresis and administration of intravenous immune globulin [1].

Outcome

Patient was treated with 5 doses of IVIG 400mg/kg/day. Muscle tone gradually improved, however lower extremity reflexes remain absent with continued inability to walk by the time of discharge. Pediatric neurology and physical therapy were both involved in patient's care throughout his hospital stay. He is planned to follow up with pediatric neurology while awaiting discharge and continue physical therapy as outpatient.

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Diagnostic Workup of Esophagitis in the Setting of Immunosuppression

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I. Case Presentation

76 year old male presented with generalized abdominal pain, constipation, reduced intake, and poor appetite for several weeks. He admits to dysphagia and odynophagia. He denies any fever or other symptoms. His past medical history was significant for recent diagnosis of lung cancer, currently undergoing radiation therapy to the chest. He continues to smoke but denies alcohol intake. On exam: patient malnourished, his BP was 80/45, afebrile, and O2 saturation 96% on room air. He was found to have fecal impaction. His white blood cell count was 12.0 , Hemoglobin 10 creatinine 1.8. His upper endoscopy showed severe inflammation of the esophagus with ulcerations. Pathology was consistent with CMV esophagitis. Patient was started on valganciclovir and symptoms improved with increased intake within one week.

II. Discussion

Esophagitis is inflammation of the esophagus that can be caused by a multitude of different conditions. If improperly treated, this condition can result in stricture, tearing, or even esophageal cancer. In the immunocompetent population, esophagitis is most commonly caused by gastroesophageal reflux disease. Medications such as aspirin and certain antibiotics may irritate the esophageal lining causing pill esophagitis or an allergic reaction causing a characteristic eosinophilic esophagitis.

However, in the immunocompromised patient, esophagitis may be the manifestation of an infectious etiology, most commonly Herpes Simplex Virus (HSV), candida sepsis, and cytomegalovirus (CMV). Thus, it is important to keep a wide differential in mind during the initial evaluation of any patient undergoing chemotherapy/radiation, infected with Human Immunodeficiency Virus (HIV), or with history of organ transplant on immunosuppressants. HSV is usually more common in organ transplant patients while candida species is seen in HIV infected patients.

Esophagitis of infectious origin usually presents with dysphagia and odynophagia. Candida infection usually presents with white plaque involving the oropharynx, palate, and tongue that can be easily scraped away while CMV and HSV infections manifest with oral ulcerations. However, when suspecting infectious esophagitis, the diagnostic test of choice is upper endoscopy. This allows visualization of esophageal inflammation and ulcerations, and pathological biopsies can be taken to confirm the diagnosis. Further testing with special pathology stains or serum CMV Polymerase Chain Reaction may also be used.

In suspected CMV esophagitis, there exists a diagnostic triad: clinical gastrointestinal symptoms (dysphagia), visualization of linear ulcerations on endoscopy, and intranuclear or cytoplasmic inclusion bodies seen on pathology. Treatment is with an antiviral such as ganciclovir or valganciclovir for 3 weeks induction therapy followed by a lower dose maintenance therapy.

III. Learning Objectives

1. Patients with esophagitis in the setting of immunosuppression (chemo/radiation, HIV, steroids, transplant medications) should be suspected to have infectious causes including HSV, CMV and candida species.
2. Diagnostic upper endoscopy should be done to identify the cause and extent of disease to establish an optimal treatment plan.

A Case Study in the Effective Diagnosis of Infectious Esophagitis

Dr. Bryan B. Grissett, DO, Dr. Ali Hassanali MD

Introduction

- **Esophagitis**-an inflammatory condition of the esophagus that can be caused by a multitude of infectious, physiological, or environmental conditions. If improperly treated or prevented, esophagitis can result in detrimental structure, tearing, or even esophageal cancer.
- **Risks (immunocompetent)**- In the immunocompetent population, esophagitis is most commonly caused by gastroesophageal reflux disease. In addition, medications such as aspirin and certain antibiotics may irritate the esophageal lining causing pill esophagitis or an allergic reaction causing a characteristic eosinophilic esophagitis.
- **Risks (immunocompromised)**- However, in the immunocompromised patient, esophagitis may be the manifestation of an infectious etiology, most commonly Herpes Simplex Virus (HSV), candida species, and cytomegalovirus (CMV). Thus, it is important to keep a wide differential in mind during the initial evaluation of any patient undergoing chemotherapy/radiation, infected with Human Immunodeficiency Virus (HIV), or with history of organ transplant on immunosuppressants.

Case Presentation

- 76 year old male presented with generalized abdominal pain, constipation, reduced intake, and poor appetite for several weeks. Admits to dysphagia and odynophagia, denies any fever or other symptoms.
- **Past medical history**
 - Recent diagnosis of lung cancer for which he was undergoing radiation therapy to the chest.
 - He continues to smoke but denies alcohol intake.
- **Physical Exam and Workup**
 - Malnourished, BP 80/45, afebrile, and O2 saturation 96% on room air. Has focal impaction. White blood cell count 12.0, hemoglobin 10, creatinine 1.8.
- **Upper endoscopy showed severe inflammation of the esophagus with ulcerations.**
- **Pathology** was consistent with **CMV esophagitis**.
- Patient was started on valganciclovir and symptoms improved with increased PO intake within one week.



Image Set 1. Linear ulcerations seen in CMV esophagitis

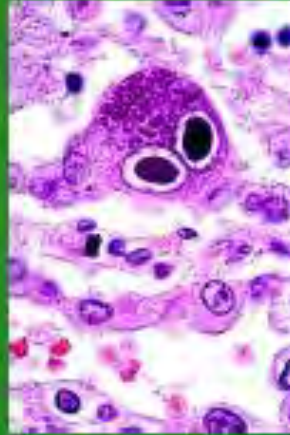


Image 2. Inclusion Bodies seen in CMV pathology

Discussion

- Esophagitis of infectious origin usually presents with **dysphagia** (difficultly swallowing) and **odynophagia** (painful swallowing).
- Candidal infections usually presents with white plaque involving the oropharynx, and tongue that can be easily scraped away while CMV and HSV infections manifest with **oral ulcerations**.
- When suspecting infectious esophagitis, the diagnostic test of choice is **upper endoscopy**. This allows visualization of esophageal inflammation and ulcerations, and **pathological biopsies** can be taken to **confirm the diagnosis**. Further testing with special pathology stains or serum CMV Polymerase Chain Reaction may also be used.
- In suspected CMV esophagitis, there exists a **diagnostic triad**- clinical gastrointestinal symptoms (dysphagia), visualization of **linear ulcerations** on endoscopy (Image 1), and **intracellular** or **cytoplasmic inclusion bodies** seen on pathology (Image 2).
- Treatment for CMV esophagitis is with an antiviral such as **ganciclovir** or **valganciclovir** for 3 weeks initiation therapy followed by a lower dose maintenance therapy.

Conclusion

The clinical evaluation of esophagitis in the immunocompromised patient requires the consideration of a broad differential diagnosis. The susceptibility to a wide variety of infectious disease in this particular population necessitates a high suspicion and swift, efficient workup to quickly diagnose the infectious agent and optimize management. The diagnostic test of choice for esophagitis is upper endoscopy for visualization with biopsies taken for pathological analysis followed by the appropriate treatment.

Contact Information

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Dry Beriberi in a Young Woman Following a Gastric Sleeve

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Learning Objective 1: Recognize the signs and symptoms of thiamine deficiency.

Learning Objective 2: Recognize thiamine deficiency as a possible complication of bariatric surgery that may result in adverse neurologic sequelae.

Case

A 23-year-old female with significant past medical history of laparoscopic sleeve gastrectomy four months prior to this presentation. A few days after gastric sleeve operation, she developed numbness in abdominal and pelvic areas, which gradually progressed to lower extremities. One month prior to this presentation, she had worsened paresthesias with burning pain in feet and lower extremity weakness leading to several falls and difficulty standing after squatting. Intermittent numbness and decreased grip strength in bilateral hands were also reported during the past month. The patient was a nondiabetic without history of alcohol consumption. Upon presentation, she had no gastrointestinal symptoms. Physical examination was significant for unsteady gait and difficulty walking with areflexia in knees and ankles, diminished proprioception and absent vibrational sensation in bilateral lower extremities. She was intact to light touch and had normal plantar reflexes. Strength was 4 out of 5 throughout. MRI of brain and spine showed no acute pathology. Electromyography and nerve conduction testing showed evidence of axonal peripheral neuropathy. Lumbar puncture revealed mildly elevated protein at 48mg/dL. CBC, CMP, B12 were normal. Thiamine level was significantly decreased at 35 nmol/L (normal range: 70-180). The patient's peripheral polyneuropathy with myopathy was determined to be dry beriberi, precipitated by malabsorption from her recent bariatric surgery. An initial dose of 200mg intravenous thiamine was immediately given. Within 24-48 hours, the patient reported a significant decrease in paresthesias, regained vibration sense in lower extremities, and was walking with less assistance. She received 200mg intravenous thiamine daily for a total of 5 days, followed by oral replacement of 100mg twice a day and multivitamin daily. She was educated about her predisposal to vitamin and mineral deficiencies, and advised on the importance of lifelong micronutrient supplementation and routine nutritional assessments with her primary care provider.

Discussion

Thiamine (vitamin B1) is an essential micronutrient involved in carbohydrate metabolism and ATP formation, and in pathways responsible for neurotransmitter production, amino acid modification, and lipid synthesis including myelin. Insufficient thiamine levels, often caused by alcoholism, can lead to beriberi and Wernicke-Korsakoff syndrome. Our patient developed dry beriberi due to axonal damage and impaired myelination of peripheral nerves. When recognized early and with rapid thiamine repletion, beriberi typically has a favorable prognosis. With no treatment, it can progress to Wernicke-Korsakoff, severe gait, ataxia, ophthalmoplegia, confusion, memory loss and death may occur. It is critical to provide bariatric patients with nutritional education and supplementation, and routinely assess vitamin and mineral levels. Unfortunately, our patient's medical record revealed she was discharged only on Vitamin D supplementation after her gastric sleeve. Among the various bariatric

surgeries, thiamine deficiency occurs more often with Roux-en-Y gastric bypass due to the duodenal bypass element, but may also present after gastric partitioning such as vertical banded gastroplasty and sleeve gastrectomy. The diminished holding capacity (about 75% of stomach removed) and consequently reduced functional time of the stomach following a sleeve gastrectomy predisposes to impaired nutritional absorption. Obesity itself can lead to malnutrition, with water and fat soluble vitamin deficiencies commonly observed. Deficiencies should be checked and treated prior to surgery, as they may worsen after surgery due to poor oral intake, nonadherence to bariatric nutrition protocol, or absence of dietary guidance. Measuring erythrocyte transketolase activity provides a reliable test of thiamine storage. Primary care providers, gastroenterologists, surgeons, and neurologists should be aware of the potential for underlying nutritional deficiency and/or malabsorption when working up a patient who presents with neurologic abnormalities; also recognize that patients may exhibit thiamine depletion symptoms acutely or even years after bariatric surgery.

Learning Objectives

1. Recognize the signs & symptoms of thiamine deficiency.
2. Recognize this rare deficiency as a possible complication of bariatric surgery that may result in adverse neurologic sequelae.

Introduction

- Thiamine (Vitamin B1; thiazolyl collector for carbohydrate & amino acid metabolism, ATP production, lipid/energy storage synthesis, succinate propagation & neuromodulation)
- Naturally occurring in whole grains, brown rice, yeast, legumes, pork
- Half-life: 10-20 days
- Active form: thiamine pyrophosphate (TPP)
- Measured via serum thiamine or erythrocyte transketolase activity



- Beriberi:** Deficiency of thiamine; Symptoms for "wet", "dry", "Wernicke", "Korsakoff"
- In Western world, primarily due to alcohol abuse
- Dry beriberi:** Symmetric peripheral neuropathy of the motor & sensory system (distal > proximal)
- Wet beriberi:** Heart failure, cardiomegaly, cardiomyopathy, peripheral edema, tachycardia
- Favorable prognosis when recognized early & with rapid thiamine repletion
- Wernicke-Korsakoff:** Chronic thiamine deficiency
- Hyptagmna, ophthalmoplegia, ataxia → confusion, memory loss; may be irreversible

Patient Presentation & Clinical Course

- 23yo white female presents after 1 month of progressive pain & paresthesia in bilateral lower extremities (BLL) with weakness resulting in falls and inability to walk; now also with intermittent numbness in hands
- Laboratory: *diagnosed 8 months prior*
 - Went discharged only on Vitamin D
 - Reports numbness started in abdomen 3-4 days w/o surgery → traveled to pelvic area followed by orthopedic symptoms in legs last 1 month before presentation
 - BMI: 31 → 35 (130 lb last)
 - Non-smoker, no alcohol or drug use
 - Non-diabetic, no family history of autoimmune disease
 - No recent travel, no recent illness, no significant past medical history
 - Neurologic exam: Alert & oriented with no memory/weakness/impairment; CNs I-VI intact; coordination intact; normal bulk in all muscles; strength 4/5 in BLL with decreased grip strength at 3.5-4/5; reflexes 2+ in BLL; intact to light touch but diminished proprioception & loss of vibratory sense in BLL; strength 3.5-4/5 in BLL; absent reflexes in BLL; ataxic/unstable gait

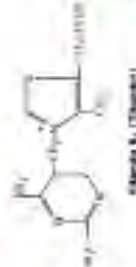
Labs/Imaging/Studies

- Vital signs within normal limits
CBC, CMP, Mg, Phos, TSH all normal
CRP: 3.1 → 0.7 mg/dL (normal <0.5)
ESR: 92 → 36 mm/hr (normal 0-36)
HIV/HBV negative
- Lumbar puncture LPx
Total protein: 48 mg/dL
Total nucleated cells: 0 per mm³
CSF Gram stain negative for any growth
CSF Green stain negative for any growth
- Visual evoked potentials (VEP): No evidence of sensory pathway conduction slowing
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Treatment

- 200 mg IV thiamine x 5 days
- Followed by 100 mg oral thiamine BID + multivitamin daily
- Gabapentin 600 mg TID for neuropathic pain
- Physical therapy & occupational therapy to assist with gait & grip strength



Discussion

- Our patient was found to have postprandial hyperglycemia without hyperlipidemia. She had an extensive workup due to concerns for autoimmune myelitis, multiple sclerosis (MS) or Guillain-Barré syndrome (GBS).
- While she did have a positive MRI, the total protein in the LP was only mildly elevated, making GBS unlikely. MS was ruled out as the patient had no lesions on MRI and had negative SEP & VEPs.
 - Our patient might have been spared such a workup (such as undergoing an LP as well as a 3 day hospital stay) if we had better suspicion and obtained a thiamine level earlier and began treatment immediately.
 - VEP deficiency can result from bariatric surgery; more often with a Roux-en-Y due to the duodenal bypass, but may also present after gastric partitioning such as vertical banded gastroplasty and sleeve gastrectomy.



Take Home Points

- Check for B1 treat vitamin deficiencies prior to bariatric surgery & routinely afterwards.
- Be aware of the potential for nutritional deficiencies and/or malabsorption following bariatric surgeries
- Provide nutritional education & ensure patients receive life-long vitamin supplementation
- Recognize thiamine depletion symptoms may occur acutely or even years later

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Gut instinct: an uncommon presentation of dilated cardiomyopathy

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Introduction

Dilated cardiomyopathies (DC) are a heterogeneous group of myocardial function disorders, most common in ages 40-59, characterized by mechanical and electrical abnormalities, with a wide variety of etiologies. These include both genetic and acquired causes. Undetected DC may progress to heart failure with reduced ejection fraction with concomitant arrhythmia and potential for thromboembolic events. Here, we report a less common presentation in an uncommon age cohort.

Case presentation

A 30-year-old male with history of autism, hyperlipidemia, and CKD presented in the outpatient setting with postprandial RUQ abdominal pain and diarrhea. On physical exam, his vitals were weight 196 lbs, BP 116/78, HR 71, RR 20, O2 98%, temp 98.5F and exam was notable for Murphy's sign and abdominal guarding. An abdominal US performed on day 10 of symptoms showed pericholecystic fluid without stones. At a followup visit at 3 weeks of symptoms, diarrhea had resolved but the patient was fatigued and had begun vomiting non-bloody fluid, initially non-bilious but now green-tinged. CMP showed sCr 1.55, BUN 23, AST 65, ALT 58; HIDA scan showed delayed gallbladder visualization, with questionable chronic cholecystitis vs. sphincter of Oddi dysfunction. Patient was admitted to the hospital for elective cholecystectomy evaluation.

Upon admission, vitals were weight 194 lbs, HR 62, RR 16, BP 112/62, O2 sat 100% RA, temp 98.4. CMP showed stable kidney function but worsening transaminitis AST 136, ALT 126 with normal T. bili. MRCP showed no biliary tract abnormalities and small bilateral pleural effusions. General surgery was consulted and plans were made for laparoscopic cholecystectomy. He was initially tachycardia and was given lorazepam 0.5 mg for anxiety. Upon anesthesia induction, he became bradycardic, cyanotic, and hypotensive; coded; was defibrillated; and had ROSC with a rhythm of atrial fibrillation with RVR. Surgical plans were aborted and the patient was given heparin and amiodarone. TTE showed mild enlargement of all 4 heart chambers with severe global hypokinesis. The patient remained hypotensive, eventually requiring 3 pressors for support as well as digoxin. He was found to have troponin 1218, proBNP 52900, sCr 2.4, and lactate 6.97. His AKI was thought secondary to ATN from cardiogenic shock, and his elevated lactate and original presenting postprandial abdominal pain caused by chronic mesenteric ischemia. The patient was transferred to UAB where he remained in a fib and was started on warfarin. He was treated with embolization for the bowel ischemia. He became aphasic with R hemiparesis, MRI showed embolic stroke. His ATN did not resolve and he was placed on dialysis.

Conclusion

Although 40-59 year olds represent DC's most common presenting adult age group, it is essential to retain cardiac causes on the differential when considering abdominal pain in younger patients. In this case, a careful retrospective cardiac history revealed this patient had exhibited dyspnea on exertion and fatigability for the past several months. The etiology of his cardiomyopathy remains unknown; however early recognition and exploration of cardiac symptoms with more timely interventions has been associated with improved prognosis in patients suffering from DC.

Introduction

Dilated cardiomyopathies (DC) are a heterogeneous group of myocardial function disorders, most common in ages 40-50, characterized by mechanical and electrical abnormalities, with a wide variety of etiologies. These include both genetic and acquired causes. Undetected DC may progress to heart failure with reduced ejection fraction with concomitant arrhythmia and potential for thromboembolic events. Here, we report a less common presentation in an uncommon age cohort.

Case presentation

A 30-year-old male with history of autism, hyperlipidemia, and CKD presented in the outpatient setting with postprandial RUQ abdominal pain and diarrhea. On physical exam, his vitals were weight 198 lbs, BP 116/78, HR 71, RR 20, O2 98%, temp 98.5°F and exam was notable for Murphy's sign and abdominal guarding. An abdominal US performed on day 10 of symptoms showed pericholecystic fluid without stones. At a followup visit at 3 weeks of symptoms, diarrhea had resolved but the patient was fatigued and had begun vomiting non-bloody fluid, initially non-bilious but now green-tinged. CMP showed sCr 1.55, BUN 23, AST 65, ALT 58; HIDA scan showed delayed gallbladder visualization, with questionable chronic cholecystitis vs. sphincter of Oddi dysfunction. Patient was admitted to the hospital for elective cholecystectomy evaluation. Upon admission, vitals were weight 194 lbs, HR 62, RR 16, BP 112/62, O2 sat 100% RA, temp 98.4. CMP showed stable kidney function but worsening transaminitis AST 136, ALT 126 with normal T. bili. MRCP showed no biliary tract abnormalities and bilateral pleural effusions.



General surgery was consulted and he was taken for laparoscopic cholecystectomy. He was initially tachycardia and was given lorazepam 0.5 mg for anxiety. Upon anesthesia induction, he became bradycardic, cyanotic, and hypotensive; coded; was defibrillated, and had ROSC with a rhythm of arial fibrillation with RVR.

Surgical plans were aborted and the patient was given heparin and amiodarone. TTE showed mild enlargement of all 4 heart chambers with severe global hypokinesia. The patient remained hypotensive, eventually requiring 3 pressors for support as well as digoxin. He was found to have troponin 12.18, proBNP 52000, sCr 2.4, and lactate 6.97. His AKI was thought secondary to ATN from cardiogenic shock, and his elevated lactate and original presenting postprandial abdominal pain caused by chronic mesenteric ischemia. The patient was transferred to UAB where he remained in a TB and was started on warfarin. His bowel ischemia was treated with embolization. He became aphasic with R hemiparesis. MRI showed embolic stroke. His ATN did not resolve and he was placed on dialysis.

Conclusion

Although 40-50 year olds represent DC's most common presenting adult age group, it is essential to retain cardiac causes on the differential when considering abdominal pain in younger patients. In this case, a careful retrospective cardiac history revealed this patient had exhibited dyspnea on exertion and fatigue for the past several months. The etiology of his CM remains unknown; however early recognition and exploration of cardiac symptoms with more timely interventions is associated with improved prognosis in patients suffering from DC.

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Headache: Not just a migraine or tension headache, a presenting symptom of bilateral vertebral artery dissection after a chiropractor neck manipulation

Sujatha Baddam, MD, PGY2 Internal Medicine Resident; Sanjay Muttineni, MD, PGY3, Internal Medicine Resident; Anjaneyulu Alapati, MD, Clerkship Director, Neurology Program, UAB Huntsville Regional Medical Campus

Learning Objectives

- To learn about secondary causes of headache
- To recognize vertebral artery dissection as a cause of headache after spinal manipulation to prevent fatal complications

Case presentation

30 year-old female with no past medical history presented to Emergency Department (ED) with complaints of headache. She reported that she hurt her neck approximately 2 weeks prior to arrival when holding her son and reached into the car to get her phone. Patient started having neck pain since then. She did go to chiropractor and neck manipulation was done. Patient developed headache shortly after neck manipulation and did have some continued neck pain. She presented to ED for further evaluation of occipital headache. No dizziness, nausea, vomiting, vision problems or any focal deficits were reported.

On evaluation vitals were stable. Neurological examination did not reveal any focal neurological deficits. Cranial nerves II-XII are intact. Initial laboratory data showed normal complete blood count, comprehensive metabolic panel and erythrocyte sedimentation rate. ANA screen was negative. CT Angiogram (CTA) head and neck with contrast showed bilateral vertebral artery stenosis suggestive of dissection. Patient was started on heparin drip for anticoagulation.

Neurology and Intervention

Radiology has been consulted, they recommended conservative management with anticoagulation. Patient was subsequently discharged on Coumadin with outpatient neurology follow up. Repeat CTA head and neck after 6 months showed interval healing of previously noted dissections.

Discussion

Vertebral artery (VA) aneurysm or dissections are the known complications of spinal manipulation procedure. Combined incidence of both VA dissection and carotid artery dissection is estimated to be 2.6 per 1, 00,000. Dissections of the vertebral vessels may occur spontaneously in patients with connective tissue diseases (Marfan syndrome, Ehlers- Danlos syndrome) or as a result of either trivial or serious trauma. Headache occurs in two thirds of patients with vertebral artery dissection but is rarely the only symptom. More than 90% of patients will have additional neck pain or associated neurologic features related to ischemic events within the territory of the affected artery. Vertebral artery dissection presents with ataxia, vertigo, nausea and vomiting, and brainstem findings. Pulsatile tinnitus or audible bruits can be present.

Vascular imaging is used to confirm an initial diagnosis of dissection and to guide serial treatment decisions. Diagnosis is usually established by MRI, MR Angiography, or CTA. No evidence supports the superiority of anticoagulation over antiplatelet therapy in prevention of stroke after vertebral artery dissections, and most patients are initially managed with heparin followed by anti-coagulant or anti

platelet therapy. There are no concrete data regarding optimal duration of antithrombotic therapy. The time course of healing of the vessel wall or resolution of vascular abnormalities may be used to guide duration of initial treatment. Most arterial abnormalities stabilize in appearance or resolve by three months, and vessels that fail to reconstitute a normal lumen by six months are highly unlikely to recover at later time points. We treated our patient with Coumadin/ for 6 months. There is complete resolution of symptoms/ imaging findings after 6 months of anti-coagulation.

*** This vignette was chosen for oral presentation on Research Day. Contact Sujatha Baddam at sbaddam@uabmc.edu for a copy of the PowerPoint.**

Herpes Zoster Ophthalmicus and related complications

Elizabeth Thottacherry, PGY2, Internal Medicine Resident, UAB Huntsville Regional Medical; Ashley Ford, MS3, UAB Huntsville Regional Medical Campus; Farrah Ibrahim, Program Director, Associate Professor of Medicine, UAB Huntsville Regional Medical Campus

Learning Objectives

1. Recognize the clinical features of Herpes Zoster Ophthalmicus
2. Anticipate, diagnose and treat complications following Herpes Zoster Ophthalmicus

Case Report

A 67-year-old female presented with right eye swelling and tenderness for five days. Symptoms began with tenderness over the right aspect of the forehead as well as vesicles appearing over right forehead, cheek and eyelid. Over the next two days, swelling increased and she could not open her right eye. The vesicles began to ooze and crust and at the time of presentation, she noticed swelling of the left eye. On examination, she was uncomfortable with periorbital swelling, right worse than the left with right side facial swelling, erythema and induration with open oozing and healing vesicles. Extraocular movements and vision was intact in the left eye, unable to assess vision in the right eye due to the periorbital edema. No other neurological deficits.

A complete blood count, kidney and liver function were unremarkable except for mild hyponatremia. A human Immunodeficiency virus test and blood cultures were drawn and were negative. A computer tomography maxillofacial view with contrast showed extensive soft tissue swelling with edema and skin thickening without abscess formation. Intravenous Valacyclovir started to treat Herpes Zoster involving V1 of the Trigeminal Nerve. Intravenous Vancomycin started for superimposed bacterial cellulitis and topical erythromycin ointment to treat possible conjunctivitis and keratitis. Due to a concern for acute retinal necrosis, the patient started on a methylprednisolone taper. She continued to improve during her hospital stay, and discharged home to finish a ten course of oral Valacyclovir, clindamycin, topical erythromycin as well as a methylprednisolone taper.

Discussion

Herpes Zoster Ophthalmicus considered an ophthalmic emergency requiring urgent treatment to prevent complications. These complications include superimposed bacterial infection of the periorbital skin and soft tissue as well as zoster conjunctivitis, episcleritis, uveitis, keratitis and acute retinal necrosis. Treatment involves early intravenous antiviral therapy to control the Herpes Zoster infection. Oral steroids may be initiated with a prolonged taper if there is concern for retinal involvement. Adjuvant use of antibiotics (intravenous and topical) can help treat and prevent concomitant bacterial infections. Delay in treatment, especially if the cornea or retina are involved, can lead to devastating permanent vision loss. What is more troubling is that while Herpes zoster infections are usually localized to unilateral dermatomes, untreated acute retinal necrosis can also lead to involvement of the contralateral eye with a potential for bilateral vision loss. Recognizing and treating these complications early is key in preventing permanent vision loss in these patients.

Herpes Zoster Ophthalmicus and related complications

Learning Objectives

- Recognize the clinical features of Herpes Zoster Ophthalmicus
- Anticipate, diagnose and treat complications following Herpes Zoster Ophthalmicus

Case Report:

- 67-year-old female without any relevant past medical history presented with progressive right eye swelling and tenderness for five days along with vesicles appearing over right forehead, cheek and eyelid.
- On examination, she was alert, sitting uncomfortably with stable vital signs.
- She was noted to have predominantly right sided periorbital swelling, facial swelling, erythema and induration with open oozing and healing vesicles.
- Extraocular movements and vision was intact in the left eye.
- Right eye movements and vision were unable to be assessed due to the periorbital edema. No other neurological deficits were noted.

Lab results and imaging:



HIV: negative

Blood Cultures: negative

CT Maxillofacial: extensive soft tissue swelling with edema and skin thickening without abscess formation

Hospital Course:

- Intravenous Valacyclovir initiated to treat Herpes Zoster involving V1 of the Trigeminal Nerve.
- Intravenous Vancomycin started for superimposed bacterial cellulitis and topical erythromycin ointment to treat possible conjunctivitis and keratitis.
- Due to a concern for acute retinal necrosis, a methylprednisolone taper was also started.
- She continued to improve during her hospital stay, and was discharged home to finish a ten course of oral Valacyclovir, clindamycin, topical erythromycin as well as a methylprednisolone taper.



Figure 1: Herpes Ophthalmicus with V1 distribution

Discussion:

- Herpes Zoster Ophthalmicus is an ophthalmic emergency
- Possible complications include:
 - Superimposed cellulitis
 - Zoster conjunctivitis, episcleritis, uveitis, keratitis
 - Acute retinal necrosis.
- Treatment involves early intravenous antiviral therapy
- Oral steroids and/or antibiotics may be initiated if there is concern for retinal involvement or concomitant bacterial infections
- Delay in treatment, especially with eye involvement can lead to permanent vision loss
- Untreated acute retinal necrosis can also lead to involvement of the contralateral eye

Conclusion:

Recognizing and treating complications early can prevent permanent vision loss in herpes ophthalmicus

“I feel like I’m in a fog”

Wade Edwards, MD, PGY1, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Kelly Diaz, MD, PGY3, Family Medicine Resident; Randy Turner, MD, PGY2, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Rajalakshmi Cheerla, MD, UAB Huntsville, Family Medicine Residency, Clerkship Director

Learning Objectives:

- Recognize signs of a thalamic and internal capsule stroke
- Correlate physical exam findings with underlying neuroanatomy

Case Presentation: 47 YOWF with past history of hypertension, hyperlipidemia, migraines with aura, hypothyroidism, and Vitamin D Deficiency presents with 3 day history of “brain fog”, and progressive 2 day slurring of speech and unsteady gait. She states that she feels like her brain isn’t working right, and she is walking around in a fog. Her boyfriend told her that some of her words didn’t make sense and were slurred. She had previously been on several medications, including anti-hypertensives, but hasn’t seen a physician in 2 years and is not currently on any medications. Social history is significant for 20 pack year smoking history, THC use, but no alcohol or other illicit drug use. Family history significant for CVA in an uncle at young age. ROS only positive for headache.

Physical Exam:

Vital Signs: Only abnormality was a blood pressure of 167/98.

Neuro Exam: Patient is awake and alert and oriented to self, place and time. She has mild expressive aphasia with full comprehension and can recall 3/3 objects. Cranial Nerves were tested and extraocular eye movements are intact with no gaze deviation. Pupils are reactive to light and accommodation. Visual fields full. Facial sensation intact and equal bilaterally. Patient does have dysarthria. No tongue deviation. Motor exam revealed 5/5 in LLE, 4/5 in RLE, 4/5 in Right deltoid, 5/5 in rest of right arm, 5/5 in LUE. No pronator drift. DTR’s were 2+ and symmetric. Cerebellar exam revealed noted dysdiadochokinesia on right, absent on left. Finger to nose and heel to shin test were normal bilaterally. Balance and gait were noted to have some unsteadiness, but otherwise normal. Sensation was decreased in RLE, most notable in S1/S2 dermatomes but otherwise normal

CV: Regular rate and NSR. All other systems examined and normal.

Lab work showed a normal CBC, CMP, negative troponins, negative pregnancy test, and EKG showed a normal sinus rhythm.

Where is the lesion??

Discussion: Both CT and MRI without contrast confirmed that this was an ischemic stroke affecting both the left internal capsule and left thalamus. The right sided weakness is explained by a lesion affecting the contralateral internal capsule, as seen in this patient. Figure 1 demonstrates this motor tract. The sensory findings in this case are also common in a thalamic stroke. What makes this case unusual is the finding of dysdiadochokinesia, usually seen in a cerebellar lesion. In some instances, ischemia affecting the thalamus can present with cerebellar findings (abnormal heel to shin, dysmetria,

dysdiadochokinesia). This is due to the connection between cerebellar cortex and the thalamus as outlined in Figure 2 below. It is also worth noting that it is common to see a stroke affecting both the internal capsule and thalamus concurrently. This is due to their close relationship anatomically.

Learning Objectives:

- ▶ Recognize signs of a thalamic and internal capsule stroke
- ▶ Correlate physical exam findings with underlying neuroanatomy

Case Presentation

47 YOWF with past history of hypertension, hyperlipidemia, migraines with aura, hypothyroidism, and Vitamin D deficiency presents with 3 day history of "brain fog", and progressive 2 day slurring of speech and unsteady gait. She states that she feels like her brain isn't working right, and she is walking around in a fog. Her boyfriend told her that some of her words didn't make sense and were slurred. She had previously been on several medications, including anti-hypertensives, but hasn't seen a physician in 2 years and is not currently on any medications. Social history is significant for 20 pack year smoking history, THC use, but no alcohol or other illicit drug use. Family history significant for CVA in an uncle at young age. ROS only positive for headache.

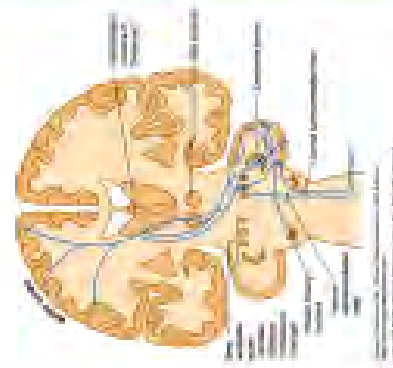
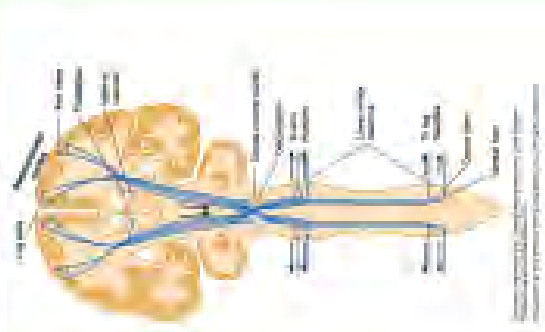
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Vital Signs: Only abnormality was a blood pressure of 167/98.
Neuro Exam: Patient is awake and alert and oriented to self, place and time. She has mild expressive aphasia with full comprehension and can recall 3/3 objects. Cranial Nerves were tested and extraocular eye movements are intact with no gaze deviation. Pupils are reactive to light and accommodation. Visual fields full. Facial sensation intact and equal bilaterally. Patient does have dysarthria. No tongue deviation. Motor exam revealed 5/5 in LUE, 4/5 in RLE, 4/5 in Right deficit, 5/5 in rest of right arm, 5/5 in LUE. No pronator drift. DTR's were 2+ and symmetric. Cerebellar exam revealed noted dysidiadochokinesia on right, absent on left. Finger to nose and heel to shin test were normal bilaterally. Balance and gait were noted to have some unsteadiness, but otherwise normal. Sensation was decreased in RLE, most notable in S1/S2 dermatomes but otherwise normal.
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Discussion

Both CT and MRI without contrast confirmed that this was an ischemic stroke affecting both the left internal capsule and left thalamus. The right sided weakness is explained by a lesion affecting the contralateral internal capsule, as seen in this patient. Figure 1 demonstrates this motor tract. The sensory findings in this case are also common in a thalamic stroke. What makes this case unusual is the finding of dysidiadochokinesia, usually seen in a cerebellar lesion. In some instances, ischemia affecting the thalamus can present with cerebellar findings (abnormal heel to shin, dysmetria, dysidiadochokinesia). This is due to the connection between cerebellar cortex and the thalamus as outlined in Figure 2 below. It is also worth noting that it is common to see a stroke affecting both the internal capsule and thalamus concurrently. This is due to their close relationship anatomically.



Lance-Adams syndrome: a post-hypoxic myoclonus after cardiopulmonary arrest

Sanjay Muttineni, MD, PGY3, Internal Medicine Resident; Sujatha Baddam, MD, PGY2 Internal Medicine Resident; Bhavyaa Bahl, MD, PGY2, Department of Internal Medicine; William Humphrey, MD, Huntsville Pulmonology / Critical Care Medicine

Learning objective

Predicting neurological outcome after a cardiopulmonary arrest is difficult. LAS is generalized myoclonus seen in survivors of cardiac arrest where the cause is respiratory arrest. It is rare complication of hypoxic ischemic brain injury, characterized by myoclonus with tactile, auditory, emotional stimuli. Early diagnosis and appropriate management improves functional outcomes.

Case presentation

A 18 year old woman with a history of asthma found unresponsive in respiratory arrest of unknown duration due to status asthmaticus. She was bagged en-route, intubated in emergency room and placed on sedation. She had generalized tonic clonic seizures in emergency room, hence neurology was consulted and she was started on keppra and dilantin. 48 hour video EEG showed generalized spike and slow wave discharges and MRI brain was normal.

On day 3 patient was awake, found to have myoclonic jerks on sedation vacation, which continued post-extubation. She had myoclonic jerks when she was awake and with movement and disappeared during rest and sleep. She was diagnosed as having Lance-Adams syndrome due to a history of hypoxic brain damage, the clinical features. She was started on depakote, klonopin, along with dilantin and increased doses of keppra. Physical therapy, occupational therapy, speech therapy were consulted for rehabilitation. Patient reported improvement in her myoclonus and functional capacity along with it. On discharge, patient demonstrated functional improvements in the areas of cognition, self-care, mobility, and locomotion.

Discussion

Lance adams syndrome is chronic post hypoxic myoclonus appearing after cardiopulmonary arrest. While our patient showed myoclonic jerks immediately after sedation vacation, they typically occur few days to few weeks after injury. Myoclonic jerks appear with attempting to move or position a limb and disappear with rest. Epilepsy, dysarthria, dysmetria and gait disturbance are frequently seen. Multiple medications are often used. Phenytoin, valproate, benzodiazepines, keppra, piracetam, zonisamide are used commonly. Early rehabilitation is important in regaining functional capacity. Time to recovery varies and early intervention results in favorable outcomes.

Learning Objectives:

- ▶ Recognize signs of a thalamic and internal capsule stroke
- ▶ Correlate physical exam findings with underlying neuroanatomy

Case Presentation

47 YOWF with past history of hypertension, hyperlipidemia, migraines with aura, hypothyroidism, and Vitamin D deficiency presents with 3 day history of "brain fog", and progressive 2 day slurring of speech and unsteady gait. She states that she feels like her brain isn't working right, and she is walking around in a fog. Her boyfriend told her that some of her words didn't make sense and were slurred. She had previously been on several medications, including anti-hypertensives, but hasn't seen a physician in 2 years and is not currently on any medications. Social history is significant for 20 pack year smoking history, THC use, but no alcohol or other illicit drug use. Family history significant for CVA in an uncle at young age. ROS only positive for headache.

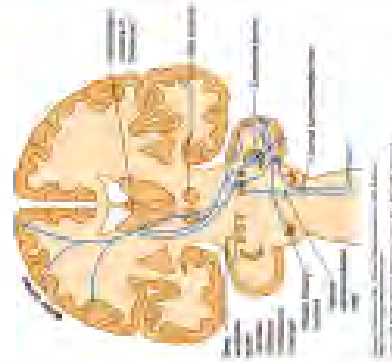
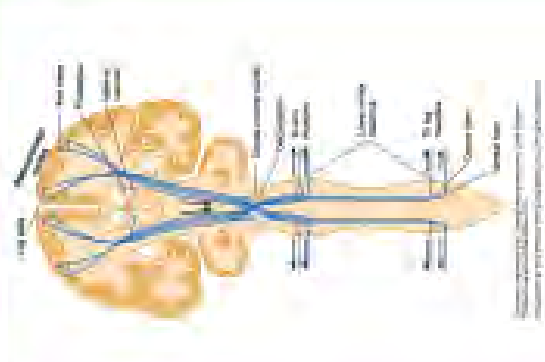
Physical Exam

Vital Signs: Only abnormality was a blood pressure of 167/98.
Neuro Exam: Patient is awake and alert and oriented to self, place and time. She has mild expressive aphasia with full comprehension and can recall 3/3 objects. Cranial Nerves were tested and extraocular eye movements are intact with no gaze deviation. Pupils are reactive to light and accommodation. Visual fields full. Facial sensation intact and equal bilaterally. Patient does have dysarthria. No tongue deviation. Motor exam revealed 5/5 in LUE, 4/5 in RLE, 4/5 in Right deltoid, 5/5 in rest of right arm, 5/5 in LUE. No pronator drift. DTR's were 2+ and symmetric. Cerebellar exam revealed noted dysidiadochokinesia on right, absent on left. Finger to nose and heel to shin test were normal bilaterally. Balance and gait were noted to have some unsteadiness, but otherwise normal. Sensation was decreased in RLE; most notable in S1/S2 dermatomes but otherwise normal.
CV: Regular rate and NRR. All other systems examined and normal.
Lab work showed a normal CBC, CMP, negative troponins, negative pregnancy test, and EKG showed a normal sinus rhythm.

Where is the lesion??

Discussion

Both CT and MRI without contrast confirmed that this was an ischemic stroke affecting both the left internal capsule and left thalamus. The right sided weakness is explained by a lesion affecting the contralateral internal capsule, as seen in this patient. Figure 1 demonstrates this motor tract. The sensory findings in this case are also common in a thalamic stroke. What makes this case unusual is the finding of dysidiadochokinesia, usually seen in a cerebellar lesion. In some instances, ischemia affecting the thalamus can present with cerebellar findings (abnormal heel to shin, dysmetria, dysidiadochokinesia). This is due to the connection between cerebellar cortex and the thalamus as outlined in Figure 2 below. It is also worth noting that it is common to see a stroke affecting both the internal capsule and thalamus concurrently. This is due to their close relationship anatomically.



Meningitis after Alemtuzumab Therapy for Multiple Sclerosis

Jesse Faulk, MD, PGY2, Internal Medicine Resident; Ali Hassoun, MD, Infectious Disease Specialist, Alabama Infectious Diseases Center, UAB Huntsville Regional Medical Campus

Learning objective 1: To identify patients at risk for *Listeria monocytogenes* infections, and know the treatment for listeriosis of the central nervous system.

Learning objective 2: To recognize potential complications in patients on monoclonal antibody therapies.

Case Presentation: A 51-year-old Caucasian female with medical history significant for multiple sclerosis presents to the emergency department due to fever and altered mental status for 4 days. Patient received a three-day course of alemtuzumab for her relapsing-remitting multiple sclerosis 8 days prior to admission. She then developed persistent fever four days prior to admission with associated decreased responsiveness. On day of admission, she began to have emesis and posterior head and neck pain. On physical examination, she was noted to be febrile to 40.5° C but was normotensive with normal pulse. She withdrew to painful stimuli and her pupils were equal and reactive. She was intubated for airway protection. Computed tomography of head showed no acute abnormality, and magnetic resonance imaging of brain revealed abnormal basilar meningeal enhancement pattern. Laboratory studies showed a serum WBC count of $12.8 \times 10^3/\mu\text{l}$ and cerebrospinal fluid glucose (CSF) of 4 mg/dL, protein of 312 mg/dL, nucleated cell count of $17/\mu\text{l}$ (90% neutrophils), and red blood cell count of $40/\mu\text{l}$. CSF gram stain showed gram-positive rods, and culture grew *Listeria monocytogenes*. She was initiated on ampicillin and trimethoprim-sulfamethoxazole. Patient remained poorly responsive off sedation and deteriorated further with loss of corneal, gag, and cough reflexes. She was ultimately pronounced brain dead and compassionately extubated on day 6 of admission.

Discussion: *Listeria monocytogenes* is a motile, aerobic and facultatively anaerobic gram-positive rod that is usually a food-borne organism. Most infections occur after oral ingestion. It most commonly causes diarrhea but can cause more severe disease of the central nervous system (CNS), such as meningitis, meningoencephalitis, or cerebritis. CSF analysis in meningitis due to *Listeria* shows a pleocytosis, reduced glucose, and moderately elevated protein. Though CSF gram stain has low sensitivity for listeriosis, CSF culture findings are positive in nearly 100% of patients. Ampicillin or penicillin G is first-line treatment of CNS listeriosis. Combination therapy with gentamicin can be considered in immunocompromised patients. Trimethoprim-sulfamethaxazole can be used for those with a penicillin allergy. Mortality is approximately 20%, thus it is very important for hospitalists to identify and treat *Listeria* early. Immunosuppression and increasing age are important risk factors for central nervous system (CNS) listeriosis. Limited case reports exist of *Listeria* CNS infection occurring after alemtuzumab therapy. Alemtuzumab, a humanized monoclonal antibody targeting the surface molecule of CD52 of B and T lymphocytes resulting in an immunocompromised state, is used in relapsing-remitting multiple sclerosis. With the growing use of immunotherapies, it is particularly important for those in hospital medicine to be familiar with complications of these therapies, particularly when those complications are associated with high morbidity and mortality.

Meningitis After Alemtuzumab Therapy for Multiple Sclerosis

uab.edu

Faulk, Paige MD, Hasegawa, Ali MD
Department of Medicine, University of Alabama School of Medicine, Huntsville Regional Medical Campus, Huntsville, AL

Objectives

- To identify patients at risk for *Listeria monocytogenes* infections and know the treatment for listeriosis of the central nervous system.
- To recognize potential complications in patients on monoclonal antibody therapies.

Case Presentation

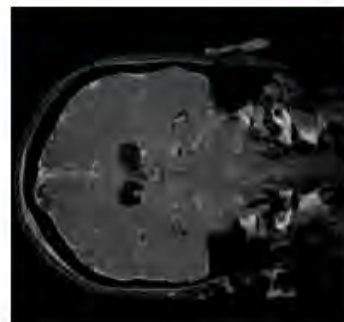
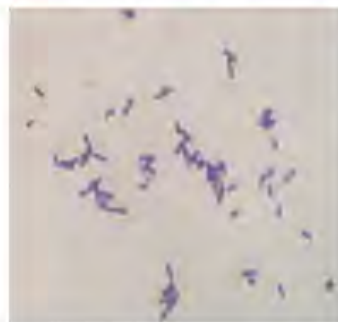
- A 51-year-old Caucasian female presents to the ED due to fever and altered mental status for 4 days.
- Patient received a 3 day course of alemtuzumab for multiple sclerosis 8 days prior to admission.
- She then developed persistent fever 4 days prior to admission with associated decreased responsiveness.
- On day of admission, she began to have emesis and posterior head and neck pain.
- In the ED, she became less alert and was intubated for airway protection.
- PMH: Multiple sclerosis, hypothyroidism
- Social History: No tobacco, alcohol, or illicit drug use.
- Physical Exam: VS: T: 40.5° C, P: 99, BP: 159/66, RR: 24, O₂ sat: 100% on 40 FIO₂
- CV: RRR, no m/r/g, no JVD
- Pulm: CTAB
- HEENT: PERLL
- Neuro: Localize to painful stimuli. Brachial reflex 2/4 and Patellar reflex 1/4 bilateral. Downgoing toes.
- Skin: No rashes, excoriations, or ulcerations.

Diagnostic Work-Up and Hospital Course

142	100	11	118
3.0	26	0.5	8.2



- Cerebrospinal Fluid:**
 - Glucose: 4 mg/dL
 - Total protein: 312 mg/dL
 - Cell count: 17 TNC/ μ L with 40 RBC/ μ L
 - 72% neutrophils
 - Gram stain: Gram-positive rod
 - Culture: *Listeria monocytogenes*
- CT head without contrast (day 1): no acute intracranial abnormality.
- Diagnosed with listeria meningitis secondary to alemtuzumab and started on ampicillin and trimethoprim-sulfamethoxazole.
- MRI brain with and without contrast (day 2): abnormal basilar meningeal enhancement pattern.
- Patient remains poorly responsive off sedation; brainstem reflexes intact.
- Video EEG (day 3-5): Generalized tonic-clonic seizure occurred during 1st 12 hours.
- CT head without contrast (day 4): interval effacement of the basilar cisterns and non-visualization of sylvian fissures.
- Loss of corneal, gag, and cough reflexes
- EEG (day 7): Near-isoelectric recording. Painful stimulation without change in background rhythm.
- Patient pronounced brain dead and compassionately extubated.



Discussion

- Listeria monocytogenes:**
 - Motile, aerobic and facultatively anaerobic gram-positive rod
 - Usually a food-borne organism
 - Most infections occur after oral ingestion
 - Can cause more severe disease of the central nervous system (CNS), such as meningitis, meningoencephalitis, or cerebritis.
- Cerebrospinal Fluid:**
 - Pleocytosis
 - Low glucose
 - Moderately elevated protein
 - Gram-stain with gram-positive rod (low sensitivity)
 - Culture almost 100% sensitive
- Treatment:**
 - Ampicillin or Penicillin G for CNS
 - Genitamicin addition for immunocompromised
 - Trimethoprim-sulfamethoxazole for penicillin-allergic
- Alemtuzumab**
 - Humanized monoclonal antibody targeting surface molecule of CD-52 of T and B lymphocytes
 - Results in immunocompromised state
 - Limited case reports exist of Listeria CNS infection occurring after therapy

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Metastatic neuroendocrine tumor to the brain: Unknown primary location

Swetha Srialluri, PGY1, Internal Medicine Resident, UAB Huntsville Regional Medical; Kapil Nath Veeravalli, MD; Farrah Ibrahim, Program Director, Associate Professor of Medicine, UAB Huntsville Regional Medical Campus

Learning Objective #1

Importance of considering metastatic neuroendocrine tumor when a clinician confronts with multiple cerebral lesions on brain imaging.

Learning Objective #2

The availability of image based stereotactic biopsy opened up new perspectives for the management of brain tumors.

Case

A 28-year-old Caucasian female presented with chief complaints of 2-week history of dizziness, ataxia, and worsening diplopia. Associated symptoms include nausea and one episode of vomiting per day for the last two weeks. She also had unexplained weight loss of twenty pounds in two months and fatigue. Neurological examination did not show any focal motor or sensory deficits. CBC and CMP were within normal limits. CT of the head showed multiple scattered lesions. MRI of the brain with and without contrast showed innumerable cerebral and cerebellar cystic and more solid lesions demonstrating peripheral enhancement and small amount of adjacent edema. HIV test was negative. Based on the imaging studies, neurocysticercosis was high on suspicion. Albendazole and praziquantel started. Neurocysticercosis antibodies were negative. Stereotactic brain biopsy done. Pathology showed high-grade poorly differentiated neuroendocrine tumor of unknown primary. Further primary tumor work up done. CT abdomen showed a low attenuating lesion within the right lobe of liver measuring 3.6 X 3cm in greatest cross section and several other low attenuating lesions within the liver are also present consistent with liver metastasis. Colonoscopy, capsule video endoscopy and upper GI endoscopy were normal. CT chest and pelvis with contrast is normal. CEA, CA-125, CA19-9, CA15-3, AFP are normal. Treatment started with Keppra for seizure prophylaxis and dexamethasone. Whole brain radiation started, after the first round of radiotherapy patient complained of worsening diplopia, dose of dexamethasone increased and patient improved during the hospital course. A chemo Port placed for outpatient chemotherapy.

Discussion

Cancer of unknown primary constitute 4-5% of all invasive cancers. Of these, neuroendocrine neoplasm account for less than 5%. The majority of metastasis of neuroendocrine tumors occur in liver, lungs and bone. Brain metastasis of neuroendocrine tumor is rare with incidence of 1.5-5%. Of all neuroendocrine neoplasm bronchopulmonary tumor appears to be the most frequent source of cerebral metastasis. In the largest series of brain metastasis, the primary tumor was located in bronchi or lungs in 45% and 71% of the patients respectively. In this case, CT chest, abdomen and pelvis, colonoscopy and upper GI endoscopy for primary tumor work up was negative. Even though the CT abdomen showed a low attenuating lesion in the right lobe of the liver and other attenuating lesions in the liver but smaller in size, these lesions were consistent with metastasis. The availability of image based stereotactic biopsy opened up new

perspectives for the management of brain tumors. Octreoscan not performed, as it is insensitive in poorly differentiated neuroendocrine tumor. Treatment for such tumor does not vary significantly based on primary site and the standard treatment includes initiation of platinum/etoposide combination.

Metastatic neuroendocrine tumor to the brain: Unknown primary location

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Learning objective

The availability of image based stereotactic biopsy opened up new perspectives for the management of brain tumors

Case Presentation

A 28 year-old Caucasian female presented with chief complaints of 2-week history of dizziness, ataxia and worsening diplopia. Associated symptoms include nausea and one episode of vomiting per day for 2 weeks.

Physical examination

Vital signs: Temp 98.5 F, RR 18/min, BP 140/90 mmHg, HR 68/min.

General: Well nourished Caucasian female lying on bed comfortably. Alert and oriented x3

Lungs: clear bilateral air entry.

Heart: S1, S2 present.

Neurological: No motor and sensory deficits.

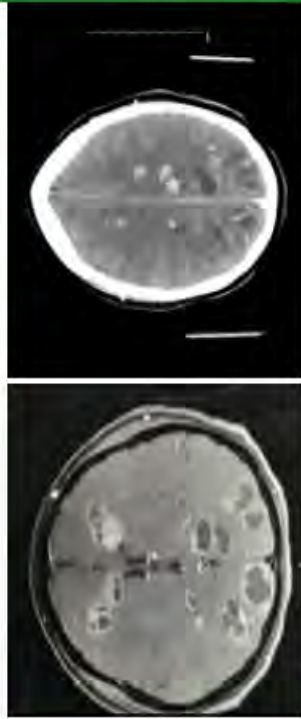
Labs & imaging:

CBC and BMP were within normal limits. HIV test was negative.

CT of the head showed multiple scattered lesions.

MRI of brain with and without contrast showed

innumerable cerebral and cerebellar cystic and more solid lesions.



Hospital course

Based on imaging studies, neurocysticercosis was high on suspicion.

Started albendazole and praziquantel. Neurocysticercosis antibodies were negative.

Stereotactic brain biopsy was done. Pathology showed high – grade poorly differentiated neuroendocrine tumor of unknown primary.

CT abdomen showed a low attenuating lesion within the right lobe of liver measuring 3.6 x 3 cm. and several other low attenuating lesions within the liver consistent with liver metastasis.

Colonoscopy, Capsule video endoscopy and upper GI endoscopy were normal. CT chest and pelvis with contrast normal. CEA, CA -125, CA 19-9, CA 15-3, AFP are normal. Started Keppra for seizure prophylaxis and dexamethasone.

Whole brain radiation was started and patient improved during the hospital course.

A chemo port placed for outpatient chemotherapy.

Discussion

Neuroendocrine neoplasm of unknown primary should be classified as well differentiated or poorly differentiated.

Most well – differentiated tumors of unknown primary site present with liver metastasis. In poorly differentiated tumors, in addition to the liver, common metastatic sites include bone, lung, and brain.

In the largest series of brain metastasis the primary tumor was located in bronchi or lungs in 45% and 71% of the patients

Initial evaluation of poorly differentiated should include CT/MRI of chest/ abdomen/ pelvis. Brain MRI should also be considered.

Carboplatin or cisplatin combined with etoposide is the recommended first line treatment for poorly differentiated tumors.

Conclusion:

Importance of considering metastatic neuroendocrine tumor when a clinician confronts with multiple cerebral lesions on brain imaging.

“Nerves vs Muscles” – An uncommon diagnosis for a young man who cannot swallow

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Learning Objectives:

1. Recognize the clinical approach to a patient with oropharyngeal dysphagia.
2. Appreciate the importance of collaborating with university hospitals in cases with diagnostic dilemmas.

Case Presentation:

A 19-year-old male with no past medical history presented with three months of worsening dysphagia, diffuse weakness, and a twenty-pound unintentional weight loss. Symptoms began with acute onset of left upper extremity swelling and weakness. The swelling resolved with Lasix from a local emergency department, but he had residual LUE weakness that eventually progressed to involve all extremities. He also developed odynophagia and dysphagia to both solids and liquids associated with nasal regurgitation. Physical examination revealed a thin, malnourished male with nasal speech, diffuse muscle atrophy, strength 4/5 in all extremities, DTRs 1+ in upper extremities and 2+ in lower extremities bilaterally. Skin exam was negative for any rashes or lesions. Remainder of physical exam was unremarkable. Initial laboratory workup revealed creatine phosphokinase 468 as the only abnormality. Further workup including HIV 1&2 Ag/Ab, hepatitis panel, ANA w/ reflex, MRI of brain and C-spine, and thyroid ultrasound were all unrevealing. Barium swallow demonstrated aspiration, and a subsequent modified barium swallow showed silent aspirations with complete absence of coordinated palatal movement or esophageal contractions, which confirmed severe oropharyngeal dysphagia. A trial of pyridostigmine was unsuccessful in improving symptoms. Paraneoplastic antibody panel, myasthenia gravis panel, and muscle biopsy were pending. Nerve conduction studies revealed no evidence of demyelination or neuromuscular junction dysfunction. Electromyography showed diffuse anterior horn cell dysfunction which made amyotrophic lateral sclerosis (ALS) as the most likely diagnosis. At this time, patient was transferred to UAB Birmingham with a presumptive diagnosis of ALS for further management. Several days after transfer, the muscle biopsy returned with a diagnosis of dermatomyositis. Further workup at UAB, including a repeat EMG, confirmed the diagnosis of dermatomyositis and glucocorticoid therapy was initiated.

Discussion:

Oropharyngeal dysphagia is characterized by difficulty transferring food from the mouth into the pharynx to initiate the swallowing process. It can be accompanied by nasal regurgitation, aspiration, and a sensation of an obstruction in the neck. It is an alarming symptom in any patient who presents with this complaint, and it is crucial to perform a thorough diagnostic workup in order to rule out life-threatening neuromuscular causes including ALS. In up to 80% of ALS patients, asymmetric limb weakness is the initial presenting symptom with eventual progression to the other extremities and

bulbar muscles, as seen in our patient. Based on this common presentation and neurodiagnostic studies that revealed diffuse anterior horn cell dysfunction, we made a presumptive diagnosis of ALS, despite the patient's young age. The decision was made to transfer the patient to a university hospital where the patient could receive more advanced care due to the severity of the diagnosis.

Dermatomyositis (DM) is a multisystem disorder that commonly presents with proximal skeletal muscle weakness accompanied by classic cutaneous manifestations, including Gottron's papules, heliotrope eruption, and photodistributed poikiloderma. Weakness may progress to involve the striated muscles of the oropharyngeal muscles, causing dysphagia, nasal regurgitation, and aspiration. The most common lab finding of DM is elevated muscle enzymes, including creatine kinase (CK), lactate dehydrogenase, aldolase, and aminotransferases. In most DM pts, the CK is elevated more than 10-fold the upper limit of normal (at least 2000-3000 U/L). In our patient, creatine kinase was only mildly elevated at 468, which is atypical of DM. Muscle biopsy is the gold standard for diagnosis of DM. The initial treatment of DM involves high dose systemic glucocorticoids. DM was lower on the differential diagnosis for our patient with progressive diffuse weakness and bulbar symptoms due to the absence of any skin findings and common lab findings not being distinct for DM.

Confirming the diagnosis of dysphagia may initially be difficult until the full clinical features are manifested. Although neurologists readily recognize ALS and its variants, about 1% of patients are misdiagnosed and delays in diagnosis are common. Many reasons may cause oropharyngeal dysphagia and many diseases mimic each other making it difficult to diagnose.

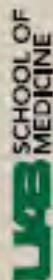


"Nerves vs Muscles" – An uncommon diagnosis for a young man who cannot swallow.

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Knowledge that will change your world

LEARNING OBJECTIVES

- Recognize the clinical approach to a patient with oropharyngeal dysphagia.
- Appreciate the importance of collaborating with university hospitals in cases of diagnostic dilemma.

CASE PRESENTATION

- A 19-year-old male presented with worsening dysphagia, diffuse weakness, and twenty-pound unintentional weight loss.
- He had no significant past medical, surgical, or family history.
- Symptoms began with an acute onset of left upper extremity weakness that eventually progressed to involve all extremities. He also developed dysphagia to both solids and liquids associated with nasal regurgitation.
- Physical exam revealed a thin, malnourished male with nasal speech, diffuse muscle atrophy, strength 4/5 in all extremities, DTRs 1+ in upper extremities and 2+ in lower extremities bilaterally. Skin exam was negative for any rashes or lesions.
- Initial laboratory workup revealed creatine phosphokinase 463 as the only abnormality. Further workup including HIV 1 & 2 Ag/Ab, hepatitis panel, ANA with reflex, MRI of brain and C-spine, and thyroid ultrasound were all unrevealing.
- Barium swallow demonstrated aspiration, and a subsequent modified barium swallow confirmed severe oropharyngeal dysphagia.
- A trial of pyridostigmine was unsuccessful in improving symptoms.
- Papanicolaou antibody panel, myositis panel, and muscle biopsy were pending at this time.
- Nerve conduction studies revealed no evidence of demyelination or neuromuscular junction dysfunction.
- Electrophysiology showed diffuse anterior horn cell dysfunction which made amyotrophic lateral sclerosis (ALS) the most likely diagnosis.
- At this time, patient was transferred to UAB Birmingham with a presumptive diagnosis of ALS for further management.
- Several days after transfer, the muscle biopsy returned with a diagnosis of dermatomyositis.
- Neurodiagnostic studies revealed that UAB confirmed the presence of myositis and ALS was ruled out at this time. Patient received IVIG and was discharged on glucocorticoids and methotrexate.
- At two-week follow up in our clinic, patient reported significant improvement in weakness and dysphagia as well as adequate weight gain.

DISCUSSION

- Oropharyngeal dysphagia is characterized by difficulty transferring food from the mouth into the pharynx to initiate the swallow-protective process. It can be accompanied by nasal regurgitation, aspiration, and a globus sensation.
- In up to 80% of ALS patients, asymmetric limb weakness is the initial presenting symptom with eventual progression to the other extremities and bulbar muscles, as seen in our patient.
- Dermatomyositis (DM) is a multisystem disorder that presents with proximal skeletal muscle weakness accompanied by classic cutaneous manifestations, including Gottron's papules, heliotrope erythema, and photosensitized poikiloderma.
- Weakness may progress to involve the striated muscles of the oropharyngeal muscles, causing dysphagia, nasal regurgitation, and aspiration.
- The most common lab finding of DM is elevated muscle enzymes, including creatine kinase (CK), lactate dehydrogenase, aldolase, and aspartate aminotransferase. In most DM pts, the CK is elevated more than 10-fold the upper limit of normal (at least 2000-3500 IU/L).
- Muscle biopsy is the gold standard for diagnosis of DM. Histopathology showing evidence of vacuolitis, perifascicular atrophy, and inflammatory infiltrate with CD4+ cells in the perimysial region is characteristic of DM.
- The initial treatment of DM involves high dose systemic glucocorticoids.
- Steroid-sparing immunosuppressants, either azathioprine or methotrexate, are generally added because of the potential for these agents to reduce the cumulative dose of glucocorticoids.

CONCLUSION

- Our patient presented with dysphagia, diffuse weakness, and weight loss concerning for ALS but was ultimately diagnosed with dermatomyositis.
- Oropharyngeal dysphagia is an alarming symptom that warrants a thorough diagnostic workup in order to rule out life-threatening neuromuscular causes including ALS.
- In situations of diagnostic dilemma, it may be beneficial to collaborate with university hospitals for further evaluation and management of atypical presentations.

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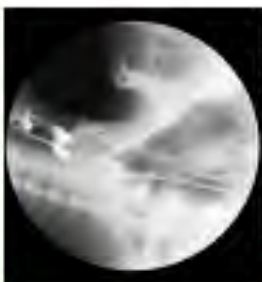


Figure 1. Barium swallow of our patient demonstrating small amounts of aspiration.



Figure 2. Modified barium swallow of our patient showing silent aspirations and laryngeal penetration. Also noted to have inadequate palatal closure, reduced tongue base retraction, inadequate laryngeal elevation & excursion, and absent epiglottic inversion.



Figure 3. Muscle biopsy showing perimysial and perinuclear inflammatory infiltrate as well as perifascicular necrosis.

Non-operative management of leaking thoracic aortic aneurysms

Jeremy Johnson, MD, PGY2, Department of Internal Medicine, UAB Huntsville Regional Medical Campus;
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Main Body: 93y F with PMH significant for HTN, cerebral aneurysm, and SH significant for non-smoker presents to HH ED with <24 hours of chest and epigastric pain. The pain was atypical in nature and her initial cardiac evaluation was negative for acute ischemic injury. Imaging revealed a 4.2cm ascending thoracic aortic aneurysm and a left apical lung mass with concern for a primary lung malignancy. Of note her systolic blood pressure was trending between 180mmHg and 220mmHg. Review of her home medications revealed amlodipine 10mg daily. Thoracocentesis was performed with plan for f/u biopsy. Thoracocentesis revealed frankly bloody fluid and concern was for leaking thoracic aortic aneurysm. Thoracic surgery and vascular surgery evaluated the patient and deemed her to be a non-surgical candidate. Aggressive hypertension control with goal systolic blood pressure of 105 to 120mmHg was initiated and achieved with the addition of labetalol 200mg BID, hydrochlorothiazide 25mg daily, along with her home amlodipine 10mg daily. Discussion with the patient revealed she did not wish for aggressive diagnosis or therapy of the lung mass. She was discharged home with home hospice. Thoracic aortic aneurysm (TAA) is defined as a permanent localized dilatation of the thoracic aorta having at least a fifty percent increase in diameter as compared to the expected normal diameter based on the specific level of the aorta. Many TAA's are asymptomatic. Symptoms if present are due to compression of adjacent structures, i.e. pulmonary symptoms, chest pain, back pain, hoarseness, or diaphragm paralysis. The most serious complications are dissection and rupture. Dissections and ruptures most always bleed into the left thoracic space. The imaging modality of choice is computerized tomography with many going unrecognized on plain film chest x-ray. When noted on plain film chest x-ray TAA's often appear as a widened mediastina, enlargement of the aortic knob, or with displacement of the trachea. In patients with asymptomatic disease recommendations include follow up imaging in six months to assess stability and evaluation for genetic syndromes known to be related to thoracic aortic aneurysm and dissection. The patient should be evaluated for other aneurysms as well. If symptomatic the evaluation can be performed post operatively. If the six-month follow up imaging shows a stable aneurysm imaging should be performed annually, however, if the aneurysm shows growth imaging every 3-6 months is recommended. Elective repair is only performed if the risk of rupture or other complications exceeds the risks associated with repairs. For asymptomatic patients the following are thresholds for repair: (1) Rapid expansion ($> \text{ or } = 10\text{mm per year}$) of aneurysms $< 5\text{cm}$. (2) For descending TAA a diameter $> 5.5\text{cm}$ for average risk patients, diameter of $> 7\text{cm}$ for high-risk patients, diameter 4-6cm for genetically mediated conditions, or a diameter $2\times$ the non-dilated aorta. (3) For ascending TAA an end-diastolic diameter $> 5.5\text{cm}$, an aortic size index $> \text{ or } = 2.75\text{cm/m}^2$, and non-Turner's patients undergoing valve repair with an end diastolic diameter $> 4.5\text{cm}$. Ascending aneurysms are managed with open repair utilizing cardiopulmonary bypass while descending aneurysms may be managed by open repair or endovascular repair depending on conditions such as aortic anatomy and the etiology being non-syndromic. For patients who are non-surgical candidates management consists of blood pressure control with systolic blood pressure goals of 105mmHg to 120mmHg. The preferred pharmacological agent is a beta-blocker followed by an angiotensin-converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB) for those intolerant of beta-blocker therapy. Management of cardiovascular risk factors such as smoking cessation, antiplatelet therapy, and statin therapy --- though unproved for TAA --- is indicated.

Overlap of Conus Medullaris and Cauda Equina syndromes in Compressive Myelopathy

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Learning Objectives:

1. Recognize the symptoms and clinical signs pertaining to Compressive Myelopathies, particularly in relation to Cauda Equina and Conus Medullaris.
2. Understand that overlap syndromes can occur in the setting of metastatic disease.

Case Description: A 57 year old African American male with history of metastatic Renal Cell Carcinoma presents with a two weeks history of paresthesia that initially started in his toes and gradually spreading to involve his forefoot and heels. During this time the patient also experienced bilateral lower limb weakness and on presentation was unable to walk. Both symptoms were associated with a constant, sharp, pulsating “tailbone” pain that was not relieved by changing position as well as headache. During this time, bowel incontinence, followed by urinary incontinence was also noted. Neurologic examination was significant for decreased sensations bilaterally involving the dermatomes at and below T9, as well as decreased power predominately at the knees and ankles. Decreased rectal tone and saddle anesthesia were noted as well as decreased reflexes at bilateral knee and ankle joints. Lab tests revealed a normal complete blood count, kidney and liver function except for the fact that the patient was mildly anemic. A MRI T spine revealed two masses. A large sacral mass approximately 8.9x5.7x8.9 cm involving S1 through S5 vertebrae and another mass involving the posterior arches of T7-T9 causing mass effect on spinal cord. A stat consult to Neurosurgery was placed and high dose of glucocorticoids were immediately administered. The patient underwent a decompression and stabilization surgery that consisted of a T7-T9 laminectomy and bilateral medial fasciectomy, requiring an impressive 14 units of PRBC due to the extremely vascular nature of the tumor. Unfortunately at the time of discharge, a CT scan performed to assess the patient’s headache revealed metastasis to the brain and skull. Palliative chemotherapy was initiated in hospital and patient was subsequently discharged home.

Discussion: Conus medullaris syndrome occurs due to an insult at the terminal end of the spinal cord proper and- presents clinically with a combination of upper and lower motor neuron deficits- owing to its close proximity to spinal nerve roots. Injury typically produces sudden onset bilateral symptoms including back pain, paresthesia, and weakness in lower extremities. Other hallmark features include bladder and bowel incontinence, impotence, and saddle anesthesia. Cauda equina syndrome typically manifests as pure lower motor neuron deficits that occur more progressively and asymmetric. Radicular pain is more severe and- clinical signs include decreased muscle tone, areflexia, and saddle anesthesia. Impotence is less frequent and urinary retention occurs later in disease. Our patient had features of both syndromes having both flaccid paralysis as well as saddle anesthesia. This was likely due to multiple impressive spinal metastasis he had involving both thoracic vertebrae and sacral nerve roots. Metastatic disease to the spinal cord can present with a confusing array of symptoms due to the extent of cord involvement, and must always be assessed in the setting of syndrome overlap.



Overlap of Conus Medullaris and Cauda Equina syndromes in Compressive Myelopathy

Aditya Puri, Bharat Thakur, Nitya Abraham Papp

Learning Objectives

1. Recognize the symptoms and clinical signs pertaining to Compressive Myelopathy, particularly in relation to Cauda Equina and Conus Medullaris.
2. Understand that overlap syndromes can occur in the setting of neoplastic disease.

Introduction

Compressive myelopathy has a vast array of etiologies—from congenital, degenerative, traumatic, or more ominous—tumor mass effect secondary to malignancy. A detailed history, thorough physical exam with special emphasis on the neurologic component, along with appropriate labs and imaging can help to delineate the underlying cause of the spinal cord compression.

Conus medullaris syndrome occurs due to an insult at the terminal end of the spinal cord proper and presents clinically with:

- Combination of upper and lower motor neuron deficits
- Sudden onset bilateral symptoms including back pain, paresthesia, and lower extremity weakness
- Bladder and bowel incontinence
- Saddle anesthesia

Cauda equina syndrome typically manifests as pure lower motor neuron deficits that occur more progressively and asymmetrically. Radicular pain is more severe. Clinical signs include:

- Decreased muscle tone
- Areflexia
- Saddle anesthesia
- Incontinence is less frequent and urinary retention occurs later in disease



Figure 1: MRI axial section of brain showing hyperdense lesions

Case Description
A 57-year-old African American male with history of metastatic Renal Cell Carcinoma presents with a two-week history of paresthesia that initially started in his toes and gradually spread to involve his forefoot and heels. During this time, the patient also experienced bilateral lower limb weakness and was unable to walk. These symptoms were associated with a constant, sharp, pulsating "balloon" pain that was not relieved by changing position. He also endorsed a headache of few days duration. During this time, bowel incontinence, followed by urinary incontinence, was also noted.

Neurologic examination was significant for decreased sensation bilaterally involving the dermatomes at and below T9, as well as decreased power predominantly at the knees and ankles. Decreased rectal tone and saddle anesthesia were noted as well as decreased reflexes at bilateral knees and ankle joints.

Labs: BMP and liver function panel unremarkable. Hemoglobin and hematocrits: 11.2 and 35, respectively.

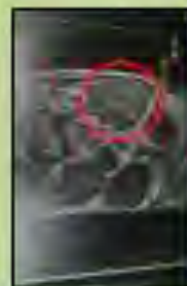


Figure 1: Sagittal MRI T1, T2, and contrast-enhanced T1 images of the spine showing a large enhancing mass at the T12-L1 level.



Figure 2: MRI axial section of sacral spine showing a large enhancing mass.

Hospital Course

- With the physical exam findings an MRI of the T spine with and without contrast was done. Imaging showed two large masses. A large sacral mass approximately 5.5x4.7x3.9 cm involving T1 through T12 vertebrae and another mass involving the posterior arches of T7-T9.
- Older Neurosurgery consult placed
- High dose of glucocorticoids administered

Decompression and stabilization surgery that consisted of a T7-T9 laminectomy and bilateral medial facetectomy, resulting in an impressive 14 units of NIHSS due to the extremely vascular nature of the tumor.

Discussion

Our patient had features of both syndromes having both sacral paresthesia as well as saddle anesthesia. This was likely due to multiple compressive spinal metastases he had involving both thoracic vertebrae and sacral nerve roots. Metastatic disease to the spinal cord can present with a confusing array of symptoms due to the extent of cord involvement, and must always be assessed in the setting of syndrome overlap. Emergent treatment is indicated because the most important indicator of long-term neurologic function is neurologic function at the time of decompression. Neurologic function is rarely regained as a result of therapy; however, further deterioration can be prevented.

Conclusion

On the morning of discharge to the Birmingham VA, after a 15-day stay, the patient experienced a headache that prompted a CT of the head. CT noted hyperdense hemorrhagic lesions consistent with metastatic disease, and a large destructive lytic lesion within right temporal bone, extending into right occipital condyle. Palliative chemotherapy was initiated in hospital and patient was subsequently discharged home.

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Panic Attacks, an Ominous Sign of a much Graver Disease

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Learning Objectives

Recognize the difference in symptoms between benign anxiety and a more worrisome disease process.
Eliminate gender bias in the workup of anxiety to avoid missing a critical diagnosis.

Case Presentation

34yo Caucasian female with past medical history of anxiety and “episodes that occur 3 times weekly of patient turning pale, shaking, increased heart rate, nausea, palpitations, and sweating” who complains of nausea, cold flu symptoms, decreased concentration, and fatigue of five day duration. Patient deteriorated upon admission and abdominal CT scan was performed after complaints of abdominal pain. A mass was discovered and idea of pheochromocytoma was entertained. Plasma and urine metanephrines confirmed the diagnosis. Treatment was delayed surgical excision with an excellent recovery.

Discussion

An ominous history of anxiety provided the only warning sign for pheochromocytoma, which was discovered after the patient was hospitalized with sepsis and severe cardiovascular compromise. It is important to exclude physical abnormalities before attributing panic attacks to anxiety. Elevated glucose and lack of anticipatory anxiety or agoraphobia can help distinguish pheochromocytoma from panic attacks. Severe sepsis and other illnesses can elevate catecholamines and their breakdown products, metanephrines, thus interfering with the diagnosis of pheochromocytoma during periods of physiologic stress. It is important to eliminate gender biases in the workup of panic attack symptoms.



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Pleural Effusion as An Initial Manifestation of Active Disease in Patient With CLL

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INTRODUCTION

Chronic Lymphocytic leukemia (CLL) is the most common of the adult leukemias and represents approximately 30% of all leukemias. Pneumonitis can occur in patients being treated for active disease with immunotherapy. Involvement of the pleural space is rare, especially in asymptomatic patients during the observation period. We present a case of 89-year-old male who presented with an exudative pleural effusion due to malignant pleural invasion from CLL.

CASE PRESENTATION

An 89-year-old male with past medical history of bladder cancer status post resection, and CLL presented with left sided chest pain, shortness of breath and dry cough for several days. He had been diagnosed with CLL four months prior and was being managed with close monitoring without active treatment. On presentation, his vital signs revealed a Temperature 98 F, blood pressure 114/56, pulse 97, respiratory rate 18, and O2 saturation of 93% on 2-liters nasal cannula. Physical exam was notable for decreased breath sounds at the left lung base. CBC showed WBC count of 37,000/uL, hemoglobin of 11.6 g/dL, and platelet of 381,000/uL. A CT of the chest showed a large left pleural effusion. He underwent thoracentesis where 1600 mL of straw-colored fluid was drained. Pleural fluid analysis was exudative in nature according to Light's Criteria, with a cell count of 17,060 with 67% lymphocytes. Gram stain and culture were negative. Flow cytometry of the pleural fluid showed monoclonal B-cells (positive for CD5, CD19, CD23) which were identical to the previous diagnosis of CLL. Chest X-ray obtained after thoracentesis revealed a persistent large left pleural effusion and a permanent pleural drain was placed for management of this malignant pleural effusion. He was discharged home in stable medical condition, and arrangement for outpatient therapy with Ibrutinib.

DISCUSSION

Chronic Lymphocytic leukemia (CLL) is the most common of the adult leukemias accounting for approximately 30% of all leukemias. Respiratory tract illnesses are common in patients with CLL, and result in significant morbidity and mortality. Pulmonary infiltrates are the most common complication and frequently caused by infection secondary to decreased immunoglobulins levels. Pleural effusion is a relatively rare complication of CLL. It can be the result of primary pleural involvement, central lymphatic blockage, infection or changes induced by previous irradiation or chemotherapy. Careful evaluation of the effusion to establish its etiology is required to direct therapy. When the effusion is caused by leukemic pleural infiltration (malignant pleural effusion), pleural fluid analysis will be exudative in nature and flow cytometry will show lymphocytes that are identical to those in the peripheral blood. After diagnostic thoracentesis, an asymptomatic pleural effusion does not require treatment. Patients who have symptoms secondary to effusion should undergo an initial therapeutic thoracentesis. The patient's symptomatic response and the rate of re-accumulation of the effusion should be determined along with

the patient's prognosis for duration of survival to direct other treatment options which include permanent pleural catheter placement. However, CLL directed treatment is indicated as soon as possible if not started yet for termed "active disease".

Introduction

- Chronic lymphocytic leukemia (CLL) is the most common of the adult leukemias and represents approximately 30% of all leukemias.[5]
- CLL is a progressive accumulation of functionally incompetent lymphocytes usually monoclonal in origin.[1]
- Pneumonitis can occur in patients being treated for active disease with immunotherapy.[3]
- Typical signs and symptoms of chronic lymphocytic leukemia include swollen lymph nodes and fatigue.
- Symptoms and staging is very important in determining a patient's survival.
- Asymptomatic early stage patients have a median survival greater than 10 years. Patients with symptoms of progressive disease have a median survival between 18 months and 3 years.[5]
- Involvement of the pleural space is rare as a presenting symptom of progressive disease, especially in asymptomatic patients during the observation period.
- "Active disease" is typically indicated for symptoms of weakness, night sweats, weight loss, fever, progressive increase in size of lymph nodes.[2]
- Other typical physical manifestations include lymphadenopathy, splenomegaly, hepatomegaly, skin, and any lymphoid tissue.[2]
- According to Rai and Bendit there is no clear staging for CLL. Diagnosis without symptoms is called the asymptomatic stage or Stage 0. Symptomatic CLL is called Advanced stage or symptomatic stage.[4]

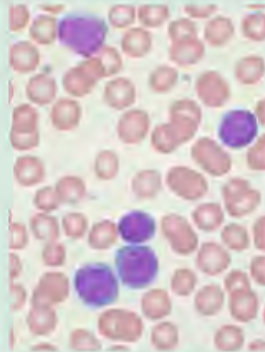


Figure 1: Peripheral blood smear of CLL of another patient. Reproduced here from O'Brien's Chronic Lymphocytic Leukemia.

Case Description

- An 89-year-old male with past medical history of bladder cancer status post resection, and asymptomatic Stage 0 CLL presented with left sided chest pain, shortness of breath, chills, and dry cough for several days.
- He had been diagnosed with CLL three weeks prior and was being managed with close monitoring without active treatment. Outpatient chest x-ray was done, and he was found to have a large left pleural effusion and referred to the hospital for further evaluation.
- Endorsed prior tobacco history many years in the past.

Physical Exam:

- T 97.8, HR 87, RR 17, BP 120/56, O2 96% on 2-Liters nasal cannula
- Patient is ill appearing, able to follow commands, not in distress.
- Cardiovascular: Regular rhythm, normal rate, with normal S1, S2.
- Pulmonary: Decreased breath sounds at the left lung base with no wheezing.
- Abdomen: Soft, nontender, normal bowel sounds.
- Neurological: Alert, awake, and oriented x3.
- Extremities: No edema. No clubbing. No cyanosis. Peripheral pulses intact.
- Remainder of physical exam was within normal limits.

Significant Labs:

- White blood cells 44,460/uL, Hemoglobin 13.0 g/dL, Hematocrit 38, platelets 381,000/uL
- Sodium 135, potassium 4.5, chloride 96, bicarbonate 25, BUN 14, creatinine 1.1, glucose 120, calcium 9.3
- INR 1.1
- Lactate 0.73
- Troponin 29
- Urine Analysis: RBC 245, large leukocytosis.

Hospital Course

Upon admission, patient was evaluated with CT chest/pelvis/abdomen for analysis of the pleural effusion. IR performed ultrasound guided thoracentesis with visualization of pleural fluid and drainage of 1600 mL of straw colored fluid which was sent to the lab for further analysis. Fluid gram stain and culture was negative for any growth. Fluid cell count was significant for 17,060/uL total nucleated cells, 44,000/uL RBCs. The differential 4% neutrophils, 67% lymphocytes, 22% monocytes, 7% eosinophils. Fluid LDH 3357/uL, Fluid Glucose 90 mg/dL, Fluid pH 7.26, IgG 533 mg/dL, IgA 81 mg/dL, IgM 18 mg/dL, Fluid total protein 4.6 g/dL.

After a 3 day hospital stay patient was sent home with a left sided chest drain for the malignant effusion. He wanted to function independently outside the hospital.

Pleural Fluid	PF/Serum Protein Ratio	PF/Serum LD Ratio	PF LD (U/L)
Transudate	< 0.5	< 0.6	< 2/3 URL
Exudate	> 0.5	> 0.6	> 2/3 URL

Table 1: This table shows the laboratory values for the Light's Criteria for determining transudate vs exudate pleural fluid. Effusions are identified as exudative if at least one of the conditions are met. LD – Lactate Dehydrogenase, PF – Pleural Fluid; URL – Upper Reference Limit of Serum LD.



Figure 2: CXR of large left pleural effusion. Reproduced here from Medical News Today.



Figure 3: CT image from the patient with visualization of the left-sided pleural effusion.

Discussion

- Respiratory tract illnesses are common in patients with CLL, and result in significant morbidity and mortality.
- Pulmonary infiltrates are the most common complication and frequently caused by infection secondary to decreased immunoglobulin levels.
- Pleural effusion is a relatively rare complication of CLL. It can be the result of primary pleural involvement, central lymphatic blockage, infection or changes induced by previous irradiation or chemotherapy.
- Careful evaluation of the effusion to establish its etiology is required to direct therapy.
- When the effusion is caused by leukemic pleural infiltration (malignant pleural effusion), pleural fluid analysis will be exudative in nature and flow cytometry will show lymphocytes that are identical to those in the peripheral blood.
- After diagnostic thoracentesis, an asymptomatic pleural effusion does not require treatment.
- Patients who have symptoms secondary to effusion should undergo an initial therapeutic thoracentesis.
- The patient's symptomatic response and the rate of re-accumulation of the effusion should be determined along with the patient's prognosis for duration of survival to direct other treatment options which include permanent pleural catheter placement.
- However, CLL directed treatment is indicated as soon as possible if not started yet for termed "active disease".

Conclusion

The patient remains stable, and followed up with his outpatient oncologist. After determining the new symptomatic staging of the patient's CLL, he was started on chemotherapy in the outpatient setting. No new symptoms or sequelae have occurred since the pleural effusion. He is still able to perform all activities of daily living and independent activities of daily living.

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Rapidly Progressive Acute Respiratory Failure as Initial Presentation of Idiopathic Pulmonary Fibrosis (IPF)

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LEARNING OBJECTIVE

The natural history of IPF is often described as insidious decline in lung function with progression to respiratory failure and death over approximately four years. Less than 5% of patients have no presenting symptoms when IPF diagnosed. This is a case of a 49-year-old male presenting with acute respiratory failure without chronic symptoms or radiological hallmark of IPF.

CASE

A 49-year-old male with past medical history of diabetes mellitus presented to the hospital with non-productive cough, shortness of breath, fever, and chest pain for one-week duration. Four weeks prior to current presentation, he completed four weeks of intravenous daptomycin for treatment of diabetic foot infection. He worked as a heavy machine operator in a coal mine. Upon arrival to the hospital, O₂ saturation was 85% on room air, which improved with 4 L of oxygen through nasal cannula. Physical examination of the lungs revealed bilateral coarse crackles. Chest X – ray showed nodular-appearing airspace disease bilaterally. CT chest without contrast showed ground glass opacities, areas of consolidation and interlobular septal thickening in both lungs. Chest X-ray obtained four weeks prior was normal. Vancomycin and cefepime started for possible pneumonia. Sputum culture and gram stain, respiratory diathrex panel, mycoplasma serology, urine *legionella* and *pneumococcal* antigen were normal. Echocardiogram showed reduced ejection fraction of 45% with mild global hypokinesis. There was no clinical improvement and oxygen requirement increased. Given the patient's recent exposure to daptomycin and peripheral eosinophilia, intravenous solumedrol started for possible Daptomycin Induced Eosinophilic Pneumonia. On day seven of admission, he became hypoxic and was placed on BIPAP. Patient underwent open lung biopsy. A few days later, he was intubated, and antibiotic was switched to zyvox and meropenem, Levaquin and voriconazole. Result of the lung biopsy was consistent with Idiopathic Pulmonary Fibrosis. The antibiotics were discontinued. There was no clinical improvement. His family decided on comfort care and passionate extubation, per patient's wishes.

DISCUSSION

IPF is a chronic, progressive fibrotic disorder of the lungs that typically affects adults over age 40. In the United States, IPF incidence estimates range from 7 to 16 cases per 100,000. The pathogenesis of IPF is complex and likely involves cycles of epithelial cell injury and dysregulated repair. Symptoms and signs of IPF are nonspecific. Most patients present with a gradual onset (often >6 months) of exertional dyspnea and/or a nonproductive cough. The diagnosis of IPF relies on the clinician to integrate and correlate the clinical, laboratory, radiologic, and pathologic data. The characteristics of high-resolution computed tomography include new bilateral reticular and ground glass opacification associated with architectural distortions including honeycomb changes and traction bronchiectasis on a background of findings consistent with usual interstitial pneumonia. Management of IPF generally includes a

combination of supportive care, use of selected medications (eg, pirfenidone, nintedanib), referral for lung transplant evaluation, and treatment of comorbidities. Systemic glucocorticoid is indicated only during acute exacerbation of IPF and is no longer part of the routine maintenance care for patients with IPF as there is no demonstrated efficacy and they may be potentially harmful.

*** This vignette was chosen for oral presentation on Research Day. Contact Swetha Srialluri at ssrialluri@uabmc.edu for a copy of the PowerPoint.**

Recurrent falls in a patient with suspected ovarian cancer: a rare case of lower extremity weakness

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Learning Objectives:

1. Identify paraneoplastic syndrome as the presenting manifestation of cancer
2. Significance of neuronal antibodies to voltage-gated potassium channel (VGKC) antibodies

Case presentation:

A 58-year-old Caucasian female with a PMH of type-2 diabetes mellitus and asthma presented to the ER with complaints of progressive generalized weakness for 5 days. She initially felt numbness and tingling in the tips of her fingers and toes. She then began to having recurrent falls with difficulty standing. She also endorsed progressive abdominal distention for 2 month. On exam, she was mildly tachycardic with a HR of 99. Her abdomen was moderately distended. Weakness was appreciated in her lower extremities, with strength of 2/5 in the lower extremities and 5/5 in the upper extremities. She had decreased sensation and absent reflexes in both upper and lower extremities. On laboratory studies, her CBC and BMP were unremarkable. Lipase and liver function tests were within normal limits. CPK and LDH was 292 U/ml and 289 U/ml, respectively.

CT of the abdomen and pelvis showed moderate ascites. MRI of the cervical, thoracic, and lumbar spine was unremarkable. CSF studies showed elevated protein of 150 mg/dl. Ascitic fluid showed malignant cells positive for MOC-31, BerEp4, and CK7. CEA and CA 19-9 were within normal limits. CA-125 was elevated at 1,089 U/ml. Of note, screening for neuronal antibodies was positive for the presence of anti-voltage-gated potassium channel (VGKC) antibodies. The patient completed 5 days of intravenous immunoglobulin (IVIG) therapy, after which her weakness improved and she was able to walk with support. She was started on neoadjuvant chemotherapy with carboplatin and docetaxel. Two weeks later, her weakness had completely resolved. Over the next two months, her CA-125 trended down over to 23 U/ml.

Discussion:

Paraneoplastic neurological syndromes (PNS) are rare disorders associated with malignancy. Presence of autoantibodies can assist in the diagnosis of PNS. A variety of syndromes have been associated with antibodies to VGKC. In 2010, it was discovered that patients do not truly have antibodies to potassium channels, but rather to associated proteins. This led to the distinction of three VGKC-positive subgroups: anti-LGI1 patients, anti-Caspr2 patients, and VGKC-positive patients lacking both antibodies. Patients with LGI1-antibodies may clinically manifest as limbic encephalitis. Caspr2-antibodies may cause a variety of peripheral or central nervous system symptoms, almost exclusively affecting older males. Half of the VGKC-positive patients lack antibodies to both LGI1 and Caspr2. A recent study did not show any clinical relevance of VGKC-positivity in the absence of antibodies to LGI1 and Caspr2.

However, we present a case with negative anti LGI1 and Caspr2 and positive neuronal anti VGKC antibodies that responded well to immunotherapy. Further research is needed to understand the significance of positive antibodies to VGKCs with negative anti LGI1 and anti-Caspr2.

Learning Objectives:

- Identify paraneoplastic syndrome as the presenting manifestation of cancer
- Significance of neuronal antibodies to voltage-gated potassium channel (VGKC) antibodies

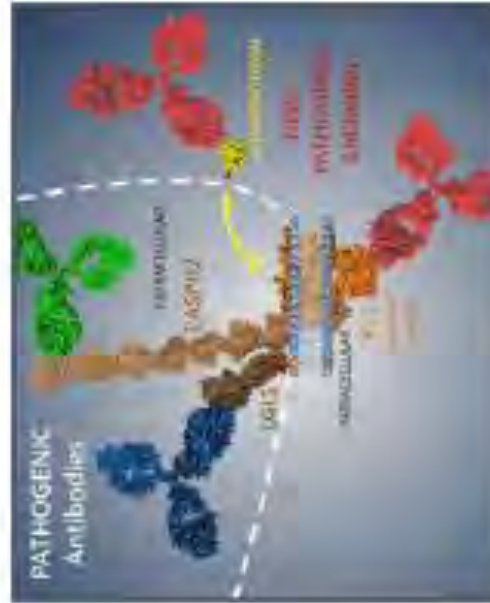
Case presentation:

- A 56-year-old Caucasian female with a PMH of type-2 diabetes mellitus and asthma presented to the ER with complaints of progressive generalized weakness for 5 days.
- She initially felt numbness and tingling in the tips of her fingers and toes. She then began to have recurrent falls with difficulty standing. She also endorsed progressive abdominal distention for 2 months.
- On exam, she was mildly tachycardic with a HR of 90. Her abdomen was moderately distended. Weakness was appreciated in her lower extremities, with strength of 2/5 in the lower extremities and 5/5 in the upper extremities. She had decreased sensation and absent reflexes in both upper and lower extremities.
- On laboratory studies, her CBC and BMP[®] were unremarkable. Lipase and liver function tests were within normal limits. CPE and LDH were 200 U/ml and 205 U/ml, respectively.
- CT of the abdomen and pelvis showed moderate ascites.
- MRI of the cervical, thoracic, and lumbar spine was unremarkable.
- CSP studies showed elevated protein of 150 mg/dL. Ascitic fluid showed malignant cells positive for MOC-31, BerEp4, and CK7.

Discussion:

- Paraneoplastic neurological syndromes (PNS) are rare disorders associated with malignancy.
- Presence of autoantibodies can assist in the diagnosis of PNS. A variety of syndromes have been associated with antibodies to VGKC.
- In 2010, it was discovered that patients do not truly have antibodies to potassium channels, but rather to associated proteins.
- This led to the distinction of three VGKC+ positive subgroups: anti-LGI1 patients, anti-Caspr2 patients, and VGKC-positive patients lacking both antibodies.
- Patients with LGI1-antibodies may clinically manifest as limbic encephalitis.
- Caspr2-antibodies may cause a variety of peripheral or central nervous system symptoms, almost exclusively affecting older males.
- Half of the VGKC-positive patients lack antibodies to both LGI1 and Caspr2.
- A recent study did not show any clinical relevance of VGKC-positivity in the absence of antibodies to LGI1 and Caspr2.
- However, we present a case with negative anti-LGI1 and Caspr2 and positive neuronal anti-VGKC antibodies that responded well to immunotherapy.
- Further research is needed to understand the significance of positive antibodies to VGKC with negative anti-LGI1 and anti-Caspr2.

- CSEA and CA-125 were within normal limits. CA-125 was elevated at 1,000 U/ml.
- Of note, screening for neuronal antibodies was positive for the presence of anti-voltage-gated potassium channel (VGKC) antibodies.
- The patient completed 5 days of intravenous immunoglobulin (IVIg) therapy, after which her weakness improved and she was able to walk with support. She was started on neurolytic chemotherapy with carboplatin and docetaxel.
- Two weeks later, her weakness had completely resolved. Over the next two months, her CA-125 trended down over to 23 U/ml.



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Septic Emboli and Aortic Rupture of Mycotic Aneurysm due *Salmonella enteritidis*

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Learning objectives:

1. Early recognition of mycotic aneurysm.
2. Treat mycotic aneurysms.

Case Presentation:

A 61-year-old alcoholic Caucasian male presented to the ED with acute encephalopathy. He was discharged from the ED with a discharge diagnosis of 'Alcohol withdrawal seizures' 5 days earlier after he developed a witnessed episode of "shaking" two days after cessation of alcohol use. One day later he developed 7/10 aching sensation in the chest. In the following days he developed nausea, vomiting, dysuria, episodes of disorientation, altered mentation, increasing weakness, gait instability, multiple falls and tremulousness at home. On day of admission he was found by family members outside of the house lying on the ground. CXR showed right upper lobe infiltrate concerning for aspiration pneumonia, but no change in appearance of mediastinum in comparison with CXR done 5 days prior and he was started on Rocephin. CT Head/C-spine showed an incompletely visualized soft tissue mass in the prevascular space 4.5 x. Patient had a 5-day 20lbs weight loss and laboratory findings consistent with starvation ketoacidosis. CT chest revealed ruptured thoracic aorta likely from ductal aneurysm with active extravasation, large mediastinal hematoma, hemopericardium, and bilateral pleural effusions. MR Brain showed multiple foci of diffusion restriction/small infarcts bilaterally. Patient was transferred to ICU and emergency consult was placed for vascular surgery, who performed immediate endovascular repair or graft performed aneurysm intravenously started. with placement of subclavian artery chimney stent. Patient became critically acidotic and was started on bicarbonate drip. He became hypotensive requiring two pressors. CXR revealed worsening pleural effusions. Left chest tube insertion revealed bloody discharge. He went for emergent mediastinal exploration, and upon opening there was a massive tamponade with a clot in the anterior mediastinum and left chest. He did not survive the surgery there was massive exsanguination and passed away. and the patient pronounced deceased shortly afterwards. Results from blood cultures sent to state lab revealed *Salmonella enteritidis*.

Impact/Discussion:

Bacteremia with subsequent endovascular infection is a known complication of gastroenteritis caused by nontyphoidal *Salmonella*. Infectious endarteritis, also known as mycotic aneurysm due to its appearance of "fresh fungus vegetations," are associated with high morbidity and mortality and therefore high clinical suspicion and early intervention are necessary. Infectious endarteritis occurs when degeneration of the arterial wall leads to an aneurysm, usually in the setting of bacteremia or septic embolization. Risk factors include: arterial injury as seen in IV drug users or in treatments with direct access to arteries; antecedent infections; immunocompromised states, including diabetes, HIV, cirrhosis, alcoholism and dialysis patients; atherosclerosis; and in preexisting aneurysms. The etiologies include bacteremic seeding of at-risk arteries (atherosclerosis or preexisting aneurysm), direct bacterial inoculation at the time of vascular injury, extension of an infection in the periarterial space, or infectious

metastases arising from septic emboli. The most common pathogens are *Staphylococcus* spp, including methicillin-resistant and vancomycin-intermediate (MRSA and VISA) *S. aureus*, and *Salmonella* spp. Bacteremia with subsequent endovascular infection is a known complication of gastroenteritis caused by nontyphoidal *Salmonella*. Case studies may suggest that *Salmonella* is associated with seeding of an atherosclerotic aorta and that aneurysm rupture is associated with infections by gram-negative pathogens. Other less common pathogens include *T. pallidum*, *Mycobacterium* spp, *Coxiella burnetii* as seen in chronic Q fever, and fungal pathogens. Other gram positives, gram negatives and anaerobes have also been reported. The classic clinical manifestation is an enlarging, pulsatile and painful mass with systemic symptoms such as fever, weight loss and elevated inflammatory markers. Other manifestations include GI bleeding, HF, expanding hematoma, mesenteric or peripheral ischemia, dysphagia, hoarseness, hemoptysis, osteomyelitis, psoas abscess and neuropathy. Definite diagnosis is made with imaging, of which CTA is the best modality. Treatment involves antibiotics, surgical debridement and may require revascularization. Antibiotic treatment is empiric with gram-positive coverage with vancomycin and gram-negative coverage with Rocephin, a fluoroquinolone or Zosyn, followed by targeted coverage based on cultures and susceptibilities. Surgical treatment typically involves debridement without revascularization (excision and ligation). If arterial reconstruction is needed, it may be done immediately if there is a high risk of distal ischemia, or it may be done following an interval of antibiotic treatment in cases with low to moderate risk of distal ischemia. Endovascular treatments are used when open surgery is not an option or as a temporizing measure in the setting of rupture and is becoming the preferred modality in thoracic aortic aneurysms.

Conclusion:

Infectious Endarteritis, though rare, is often caused by Nontyphoidal *Salmonella* spp. Infectious endarteritis is associated with significant morbidity and mortality, and is likely to have non-specific symptoms at presentation. Having a high clinical suspicion for early recognition and treatment can be lifesaving.

*** This vignette was chosen for oral presentation on Research Day. Contact José Cavo at jcavoacosta@uabmc.edu for a copy of the PowerPoint.**

Septic Knee Monoarthritis Due to *Histoplasma capsulatum* After Bone Marrow Transplant in a Patient with Aplastic Anemia

Bhavyaa Bahl, MD, PGY2, Department of Internal Medicine; Lourdes Corman, MD, Clinical Professor of Internal Medicine, UAB Huntsville Regional Medical Campus

LEARNING OBJECTIVES

1. Recognize monoarthritis as a rare presentation of Histoplasmosis.
2. Evaluate diagnostic testing for histoplasmosis in an immunocompromised host.

CASE DESCRIPTION

An 18-year-old male from Alabama presented with progressively worsening right knee swelling and pain for four days associated with fever and chills. He had undergone allogeneic bone marrow transplant a year ago as treatment of aplastic anemia for which he had been on an immunosuppressive regimen with prophylactic antimicrobials till two months ago. He was sexually active with his girlfriend and was recently treated for *Chlamydia trachomatis* urethritis. Magnetic resonance imaging (MRI) knee done five days ago did not reveal any abnormalities. On exam, he was febrile with a T_{max} of 102.9°F. His right knee was inflamed and tender with restricted range of motion. Arthrocentesis done for the knee effusion noted on radiograph revealed grossly purulent synovial aspirate with 19,000 total nucleated cells (90% neutrophils) along with 1+ yeast on gram stain. He was empirically started on vancomycin, piperacillin-tazobactam, and fluconazole for septic arthritis. Arthroscopic synovectomy revealed diffuse inflammation of the joint with synovial and vascular involvement noted on pathology. Blood, synovial fluid and biopsy specimens did not reveal bacterial infection on culture and molecular testing. (1,3)-Beta-D-Glucan assay (Fungitell®), T2Candida® and Histoplasma serology resulted as negative. He was switched to micafungin and discharged after being afebrile for 24 hours to complete 6 weeks of treatment for a fungal septic joint. Five days after discharge, Histoplasma polymerase chain reaction (PCR) on the synovial fluid was reported as positive following which he was changed to itraconazole. Two weeks later fungal cultures from blood and synovial fluid resulted as growing mold identified as *Histoplasma capsulatum*.

DISCUSSION

Histoplasma capsulatum is a dimorphic fungus endemic to the Ohio and Mississippi river valley with primarily asymptomatic pulmonary involvement in immunocompetent hosts. Immunocompromised individuals are particularly at risk for a severe and disseminated disease. Bone and joint infection is very rare and mostly manifests as monoarthritis of the knee due to local inoculation or hematogenous spread. Although tedious, fungal culture is the gold standard of diagnosis with 85% sensitivity in disseminated disease which was positive in both synovial fluid and blood specimen at four weeks in this case. Earlier diagnosis can be facilitated with PCR testing (100% specificity) in immunocompromised and disseminated states where Histoplasma antibody testing has a high false negative rate and antigen testing has low sensitivity as seen here.

CONCLUSION

Septic arthritis and tenosynovitis are rare complications of Histoplasmosis that must be suspected in immunocompromised hosts particularly in endemic areas. Molecular testing can aid in the earlier diagnosis of Histoplasmosis even in cases with negative serology.

LEARNING OBJECTIVES

1. Recognize monoarthritis as a rare presentation of histoplasmosis.
2. Evaluate diagnostic testing for histoplasmosis in an immunocompromised host.

CASE DESCRIPTION

18-year-old male from Alabama presented with progressively worsening right knee swelling and pain x 4 days.

Past Medical History

1. Aplastic anemia post allogeneic HSCT a year ago. He had been on prophylactic valacyclovir and voriconazole till 2 months ago.
2. Recently treated chlamydia urethritis.

Past Surgical History

Bone marrow biopsy

Review of Systems – positive for Fever, chills, decreased appetite, difficulty walking.

Physical Exam Findings

T_{max} – 102.8°F; HR – 113 bpm
BP – 114/76 mm Hg; RR – 20/min; SpO2 – 98% on RA

Local Exam: Right knee inflammation, swelling and restricted range of motion.

Imaging of the Rt. knee

- MRI done 5 days prior to admission - 187%.
- Abdominal X-ray – WNL.
- Knee US – joint effusion.

HOSPITAL COURSE

ARTHROCENTESIS

Laboratory Investigations

- Blood Cultures X 2 sent for gram staining, routine CBC, ABG, fungal cultures on SD4.
- Arthrocentesis fluid analysis – Grossly purulent synovial effusion
TNC – 15,000 with 90% PMN
14 yeast on gram stain.

DIAGNOSIS – SEPTIC MONOARTHRITIS

Therapy: broad spectrum antimicrobial vancomycin, piperacillin-tazobactam, fluconazole.
Investigations (blood)- (1,3)-Beta-D-Glucan assay (Fungitell®), T2-tandem® (Histoplasma serology)

Patient was taken for arthroscopic synovectomy which revealed diffuse inflammation of the joint with synovial and vascular involvement noted on pathology.
Synovial specimen sent for aerobic/anaerobic cultures. *

Day 4 :

No growth noted in blood and knee synovial cultures.

Negative (Fungitell®), T2-tandem®, (Histoplasma serology).
Antibiotics discontinued.

Patient afebrile since Day 3, now spleen liver T_{max} – 104°F,
fluconazole discontinued and patient switched to intrathecal.

Discharged home on oral micafungin 500mg and IV with ID specialist.

Day 10:

Histoplasma polymerase chain reaction (PCR) on the synovial fluid reported **POSITIVE**.
Antifungal switched to itraconazole.

Day 20:

Fungal cultures (blood, synovial fluid) with moldy growth identified as, **histoplasma capsulatum**.

DISCUSSION

1. *Histoplasma capsulatum* is a dimorphic fungus endemic to the Ohio and Mississippi river valley with primarily asymptomatic pulmonary involvement in immunocompetent hosts.
2. Transmissions presumed to be due to aerosolized spores, particularly at risk for a severe and disseminated disease.
3. Bone and joint infection is very rare and mostly manifests as monoarthritis of the knee due to local inoculation or hematogenous spread.
4. Although tedious, fungal culture is the gold standard of diagnosis with 85% sensitivity in disseminated disease which was positive in both synovial fluid and blood specimens at four weeks in this case.
5. Earlier diagnosis can be facilitated with PCR testing (100% specificity) in immunocompromised and disseminated disease, whereas histoplasma antibody testing has a high false negative rate and antigen testing has low sensitivity as seen here.

CONCLUSION

1. Septic arthritis and monoarthritis are rare complications of histoplasmosis that must be suspected in immunocompromised hosts particularly in endemic areas.
2. Molecular testing can aid in the earlier diagnosis of histoplasmosis even in cases with negative serology.

Sjogren's Syndrome: A case presentation

Jeremy Johnson, MD, PGY2, Department of Internal Medicine, UAB Huntsville Regional Medical Campus;
Leah Burch, MD, PGY1, Department of Family Medicine, UAB Huntsville Regional Medical Campus;
Parekha Yedla, MD, Associate Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus

Main Body: 64-year-old female presented to Huntsville Hospital emergency department with complaint of facial swelling. PMH significant for CAD, h/o 3-vessel CABG, DM II, HLD, and arrhythmia. Medications included Plavix, Tylenol, amaryl, norco, lantus, lisinopril, HCTZ, metformin, methylprednisolone, Toprol-XL, pravastatin, and triamcinolone. Social history includes a 50 pack year h/o tobacco smoking with a stop date 5 years prior. The swelling began 3 months prior and both her primary care physician and an ear, nose, and throat specialist evaluated her. She was given multiple courses of antibiotics and steroids. She endorsed improvement while on the steroids with return of symptoms once completed. She denied any change while on antibiotics alone. She endorsed associated symptoms of dry eyes and dry mouth to the point that her tongue would often stick to her cheeks. She denied any prior diagnosis and presented to the emergency department due to increased swelling and uncertainty of which provider to follow up with. She presented with outside labs inclusive of a weakly positive ANA titer and SSA/RO antibody greater than 8. Emergency department work up included imaging showing enlargement of the parotid and submandibular glands bilaterally with infiltrated nodules vs. multiple lymph nodes. A complete blood count and comprehensive metabolic panel were benign. She was diagnosed with Sjogren's syndrome started on prednisone 40mg daily and rheumatology was consulted for outpatient follow up. Rheumatology was able to see the patient prior to discharge and confirmed the diagnosis and agreed with the discharge plan and will follow the patient as an outpatient.

Introduction

- 60-year-old female presented to hospital's emergency department with complaint of facial swelling.
- PMH significant for CAD, type 2 diabetes, DM II, HTD, and emphysema.
- Medications included Plavix, Tylenol, emory's, norco, letairis, lisinopril, HCTZ, metformin, methylprednisolone, Toprol-XL, pravastatin, and lisinectrolone.
- Social history includes a 50 pack year history of tobacco smoking with a stop date 5 years prior.
- The swelling began 2 months prior and both her primary care physician and an ear, nose, and throat specialist evaluated her.
- She was given multiple courses of antibiotics and steroids. She endorsed improvement while on the steroids with return of symptoms once completed.
- She denied any improvement while on antibiotics alone.
- She endorsed associated symptoms of dry eyes and dry mouth to the point that her tongue would often stick to her cheeks.
- She denied any prior diagnosis and presented to the emergency department due to increased swelling and incapacity to follow up.
- She presented with visible facial induration of a weakly positive ANA, Rheumatoid Factor, and SS-A/Ro antibody greater than 8.
- Emergency department work up included imaging showing enlargement of the parotid and submandibular glands bilaterally with infiltrated nodules vs. multiple lymph nodes.
- A complete blood count and comprehensive metabolic panel were benign.
- She was diagnosed with Sjogren's syndrome started on prednisone 40mg daily and Rheumatology was consulted for outpatient follow up.
- Rheumatology was able to see the patient prior to discharge and confirmed the diagnosis and agreed with the discharge plan and will follow the patient as an outpatient.



Figure 1. Enlargement of the right parotid gland in a Sjögren's patient.



Figure 2. Enlargement of the submandibular



Figure 3.
Enlargement of the
parathyroid gland in a
Sjögren's patient.



Figure 4. Enlargement of the bilateral axillary glands in a 51-year-old patient.



Figure 5. Cutaneous involvement with vasculitis in a Sjögren's subject.



Figure 6. Elevated liver activity causes a shift in the distribution of activity in the body. In a 300-g patient.

Discussion

- [illegible]

References

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Stable, Low-Dose Quetiapine Causing Neuroleptic Malignant Syndrome in an Elderly Patient

Garrett Dunn, MS3, UAB Huntsville Regional Medical Campus; Tarak Vasavada, MD, Chair, Department of Psychiatry, UABSOM Huntsville Campus

Learning Objectives: Present an atypical case of NMS caused by quetiapine to raise awareness of NMS in patients taking newer, atypical antipsychotics, as early recognition and supportive treatment is key in reducing mortality.

Case Presentation: 72-year-old male nursing home resident with past medical history of vascular dementia, depression with previous suicide attempt, and anxiety who was previously easily redirected and reoriented, presented with worsening agitating and confusion, unable to follow commands for 2 days. Psychiatric medications included memantine 20mg twice daily, levomilnacipran ER 80mg daily, mirtazapine 15mg daily, quetiapine 25mg twice daily, and oxcarbazepine 150mg twice daily. Medications were started at a hospitalization 1 month prior for alerted mental status except for quetiapine, which he had been taking 25mg at bedtime for five months with the dose increased at last admission.

On physical exam, he appeared agitated and confused, unable to follow commands. His temperature was 106.8°F, blood pressure 117/102, pulse 150, and respirations 29. Neurologic exam included alert and oriented x0 with nonverbal speech, non-purposeful movement to all four extremities, no rigidity noted, down-going plantar response bilaterally without clonus, and 1+ deep tendon reflexes throughout. Oral mucosa was extremely dry. Remainder of physical exam was within normal limits. Significant labs included WBC of 14.54, hemoglobin 10.5, platelets 260, MCV 80, RDW 16.3, sodium 145, potassium 5.4, anion gap 23, bicarb 20, creatinine 1.8, glucose 70, CPK 2611, lactate 4.5, ferritin of 262, TIBC of 190, and iron saturation of 8%. UA was positive for nitrites, WBC's, and bacteria, with final urine cultures growing ESBL-Klebsiella pneumoniae.

Patient was admitted to the ICU for NMS with acute rhabdomyolysis, AKI, and complicated UTI. Psychiatric medications were held, antibiotic therapy initiated, and supportive care continued with mechanical ventilation. He was started on bromocriptine 2.5mg PO every 6 hours. Respiratory and mental status then gradually improved, and, after 5 days on the ventilator, he was extubated on day 8 with continued return to baseline mental status.

Discussion: The incidence of neuroleptic malignant syndrome (NMS) is 0.02 to 0.03 percent in patients taking dopamine antagonist, with the vast majority of cases involving high-potency, first-generation antipsychotics, and few in patients on atypical antipsychotics, such as quetiapine, with even fewer in patients on low doses of these medications.^{[1],[2]} The only dopamine antagonist medication implicated in causing NMS in our patient was quetiapine, which he had been taking for 5 months prior to presentation with an increase in dose by 25mg one week prior. Atypical antipsychotics, such as quetiapine, have fewer case reports of causing NMS as well as a less severe presentation of symptoms as opposed to the typical, first generation antipsychotics.^[3] It is important to recognize and be aware of the risk, although small, associated with these medications as early recognition and supportive treatment is key to reducing rates of associated mortality.



Stable, Low-Dose Quetiapine Causing Neuroleptic Malignant Syndrome in an Elderly Patient

Garrett Dunn, MS4, Tarak Vasavada, MD
UABSON Huntsville Campus; Department of Psychiatry
University of Alabama at Birmingham School of Medicine

Abstract

Quetiapine is a dibenzothiazepine, atypical antipsychotic used in the treatment of many psychiatric conditions. While the incidence of neuroleptic malignant syndrome ranges from 0.02 to 0.03 percent in patients taking neuroleptic agents, the vast majority of cases are reported with use of high-potency, first-generation neuroleptic agents, with few reports in patients on atypical antipsychotics, and even fewer with patients on low doses of these medications.^{1,2,3} This is a case of a 72yo man on a 50mg total daily dose of quetiapine, who presented with symptoms, history, physical exam, and clinical course suggestive of NMS.

Introduction

- Neuroleptic malignant syndrome (NMS) is a life-threatening neurologic emergency associated with the use of dopamine antagonists, which presents with mental status changes, neuromuscular abnormalities, and dysautonomia.⁴
- Patients generally have been exposed to a dopamine antagonist within 72 hours of symptom onset.
- No one symptom or test makes the diagnosis of NMS; however, certain symptoms are much more suggestive of this diagnosis over others:⁵
 - Hypertensive with extreme elevations in serum creatinine
 - Autonomic activation with tachycardia, diaphoresis, bradypnea, and elevated blood pressure
 - Generalized rigidity, usually described as "lead pipe"
 - Elevated creatine kinase, commonly four times the upper limit of normal or greater
 - Mental status changes, characterized by delirium and altered consciousness, usually preceding autonomic signs
- Agitation, exorbitant, dehydration, and iron deficiency heighten the risk of developing NMS when placed on a dopamine antagonist.⁶
- Treatment includes removing the offending agent, supportive therapy, and the use of bromocriptine, a dopamine agonist.⁷
- In most cases, NMS is self-limited after drug discontinuation, with a mean time of recovery of 7 to 10 days.⁸

Case Description

- 72yo Middle-Eastern male nursing home resident with extensive past medical history including vascular dementia, depression with previous suicide attempt, and anxiety, previously easily medicated and recruited, presented with worsening agitation and confusion, unable to follow commands for 2 days duration.
- He was discharged from the hospital one week prior for delirium secondary to constipated UTI.
- Psychiatric medications upon admission:
 - Mirtazapine 20mg twice daily
 - Levodopa/carbidopa ER 80mg daily
 - Mirtazapine 15mg daily
 - Olanzapine 150mg twice
 - Quetiapine 25mg twice daily
- Previously 25mg once daily at bedtime for the past 5 months. Increased to twice daily at admission one week prior.
- Reversal of psychiatric medications started at a hospitalization for altered mental status 1 month prior.

Physical Exam:

- T 104.8, HR 150, RR 20, BP 117/702, O2 94%, an RA.
- Patient appeared agitated and confused, unable to follow commands.
- Neurologic exam: No eye-opening response, oriented x 0, speech nonverbal with moans only, pupils equal-round-reactive to light, decrease response to visual threat bilaterally, gag reflex present, sensory intact to nociceptive stimuli, non-purposive movement to all four extremities, strength 3/5 throughout, no rigidity noted, down-going plantar response bilaterally, no clonus, and 1+ deep tendon reflexes throughout.
- Oral mucosa was extremely dry.

- Reversal of physical exam was within normal limits.

Significant labs:

200	770	79	10.3	10.3	100	100	100
5.4	28	1.8	13.3	12.5	100	100	100
				BDW: 15.3	Ferritin: 262		

- UA: Positive for nitrites, 8 WBCs, and large bacteria.
- Urine cultures: ESBL-Klebsiella pneumoniae.

Clinical Course

Patient was admitted to the ICU for NMS vs serotonin syndrome with acute rhabdomyolysis, AKI, and complicated UTI. Psychotropic medications were held, anticholinergic therapy initiated, and supportive care continued. CK trended upwards to 24,712 on hospital day 1, after which it trended down to 2,354 by day 5. Patient agitated further with continued respiratory and mental status decline ultimately requiring intubation. He was started on bromocriptine 2.5mg PO every 8 hours. Respiratory and mental status then gradually improved, and, after 5 days on the ventilator, he was extubated on day 8. He then continued to slowly return to baseline mental status.

Discussion

- NMS and Serotonin Syndrome were the top of the differential diagnosis, with NMS being ultimately diagnosed based upon key characteristics distinguishing the two syndromes.⁹

	NMS	Serotonin Syndrome
Onset	Days to Weeks	Within 24 hours
Neuromuscular Findings	Bradycardia, severe muscle rigidity	Hypertactivity (tremor, clonus, hyperreflexia)
Time to resolution	Days to Weeks	Within 24 hours
Treatment Agents	Bromocriptine	Serotonin Antagonists, Cyproheptadine
Quarantine Agents	Dopamine antagonist	Serotonin Antagonist

- Our patient was taking quetiapine at a stable dose of 25mg for the past 5 months with an increase to 50mg 1 week prior.
- Cases of NMS caused by atypical antipsychotics such as quetiapine are rare, and presentation is usually less severe as compared to the typical, first generation antipsychotics.¹⁰
- Always be aware of signs and symptoms of NMS in all patients, regardless of age, taking any dopamine antagonist, typical or atypical, as early recognition and supportive treatment is key.

References

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3. Dunn G, et al. Neuroleptic malignant syndrome: a review of the literature. *Psychopharmacol* 2002; 166: 1-10.

Suffocating sub-mental hematoma- A rare allergic reaction to a drug

Sanjay Muttineni, MD, PGY3, Internal Medicine Resident; Elizabeth Thottacherry, PGY2, Internal Medicine Resident, UAB Huntsville Regional Medical; Timothy Littman, MD, PGY1, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Nessy Abraham Philip, MD, Assistant Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus

Learning Objective : Certain drugs may trigger autoimmune response and production of auto antibodies. Drug induced hemophilia poses challenges in diagnosis and treatment due to its rare incidence and fatal consequences.

Case presentation : A 81-year-old Caucasian presented with gross painless hematuria, oropharyngeal bleeding and bilateral submental hematoma causing visible swelling. Initial labs showed H & H of 8.8 & 27.0, platelets 387, PTT 81.9 sec, PT17.9 sec, INR 1.4, fibrinogen . Past medical history is significant for PFO closure in 2013, BPH on flomax, migraine's.

Foley catheter was placed for persistent hematuria and patient was started on antibiotics for possible infectious etiology and Flomax. Cystoscopy did not show any concerning source. Submental hematoma progressed causing posterior displacement of tongue base, pushing epiglottis back and along with ecchymotic swollen epiglottis caused airway compromise needing intubation. No IV site or stoma bleeding was identified.

A PTT mixing study failed to correct PTT to normal level, indicating the presence of a coagulation factor inhibitor. A reflex Factor VIII assay inhibitor assay was positive and factor VIII activity was 1%. Bethesda assay resulted at 11.7 BU.

Patient was treated with blood transfusions, fresh frozen plasma and cryoprecipitate as needed. Once Factor VIII inhibitors were identified he was given recombinant factor VIIa to control bleeding. Steroids were started to eliminate factor inhibitors, but recurrent bleeding episodes and relative refractoriness in PTT levels was seen. His home medication were reviewed again and Flomax was stopped, as it has sulfa. 24 hours after Flomax was stopped, PTT levels down trended to 23.5 sec, factor VIII activity normalized and further bleeding episodes stopped.

Discussion: Acquired hemophilia is a rare autoimmune disorder due to acquired inhibitors against coagulant factors. Soft tissue bleeding is most common manifestation, followed by muscle, joint and hematuria. Most cases are idiopathic ~50%, other causes are pregnancy, malignancy, auto-immune disorders, drug reactions. Most commonly implicated drugs are penicillin, sulfamides, phenytoin, interferon and fludarabine.

Mixing study is the initial diagnostic test for isolated prolonged PTT. Bethesda assay is used to establish diagnosis and quantify antibody titer. Control bleeding with recombinant factor VIIa and activated PCC (FEIBA). Steroids and cyclophosphamide are used to eliminate factor inhibitors. Other therapies for inhibitor elimination are IVIG infusions, rituximab alone or in combination. Resistant forms of disease are treated with cyclosporine, cladribine and plasmapheresis for antibody adsorption. Monitor response with monthly PTT level for first six months.

This case illustrates that bleeding is often severe, constituting medical emergency and high index of suspicion required to quickly identify drug reactions, as cessation of the offending drug helps in disease resolution.

➤ Learning Objective :

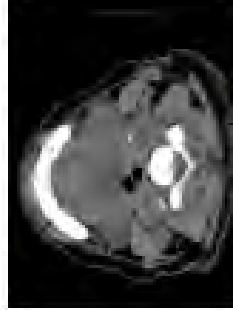
- Certain drugs may trigger autoimmune response and production of auto antibodies.
- Drug induced hemophilia poses challenges in diagnosis and treatment due to its rare incidence and fatal consequences.

➤ Case presentation :

- A 81-year-old Caucasian male presented with gross painless hematuria, oropharyngeal bleeding and bilateral submental hematomas causing visible swelling.
- Initial labs showed H & H of 8.8 & 27.0, platelets 387, PTT 81.9 sec, PT17.9 sec, INR 1.4, fibrinogen.
- Past medical history is significant for PFO closure in 2013, BPH on Flomax, migraines.

➤ Hospital course:

- Foley catheter was placed for persistent hematuria and patient was started on antibiotics for possible infectious etiology and on home dose of Flomax. Cystoscopy did not show any concerning source.
- Submental hematomas progressed causing posterior displacement of tongue base, pushing epiglottis back and along with echymotic swollen epiglottis caused airway compromise needing intubation. No IV site or stoma bleeding was identified.



- A PTT mixing study failed to correct PTT to normal level, indicating the presence of a coagulation factor inhibitor. A reflex Factor VIII assay inhibitor assay was positive and factor VIII activity was 1%. Bethesda assay resulted at 11.7 BU.
- Patient was treated with blood transfusions, fresh frozen plasma and cryoprecipitate as needed.
- Once Factor VIII inhibitors were identified he was given recombinant factor VIIa to control bleeding.
- Steroids were started to eliminate factor inhibitors, but recurrent bleeding episodes and relative refractoriness in PTT levels was seen.
- His home medication were reviewed again and Flomax was stopped, as it has alpha 1a beta after Flomax was stopped, PTT levels down trended to 23.5 sec, factor VIII activity normalized and further bleeding episodes stopped.

- **Discussion:** Acquired hemophilia is a rare autoimmune disorder due to acquired inhibitors against coagulant factors. Soft tissue bleeding is most common manifestation.
- Most cases are idiopathic ~50%, other causes are pregnancy, malignancy, auto-immune disorders, drug reactions. Most commonly implicated drugs are penicillin, valproic acid, phenytoin, interferon and fludarabine. Mixing study is the initial test.
- Control bleeding with recombinant factor VIIa and activated PCC (FEIBA). Steroids and cyclophosphamide are used to eliminate factor inhibitors. Other therapies for inhibitor elimination are IVIG infusions, rituximab, cyclosporine, cladribine and plasmapheresis for antibody adsorption.
- This case illustrates that bleeding is often severe, constituting medical emergency and high index of suspicion required to quickly identify drug reactions, as cessation of the offending drug helps in disease resolution.

Syncope as Presenting Feature of Superior Vena Cava Syndrome

Jesse Faulk, MD, PGY2, Internal Medicine Resident; Alan Baggett, MD, Assistant Professor, Department of Internal Medicine, UAB Huntsville Regional Medical Campus, Huntsville Hospital

Learning Objectives:

1. Recognize signs and symptoms of Superior Vena Cava syndrome.
2. Understand treatment for SVC syndrome.

Case Presentation: A 65 year-old Caucasian male with significant medical history of hypertension and tobacco abuse presented to the emergency department due to a syncopal episode. He described the episode as having lightheadedness while bending forward with subsequent loss of consciousness after standing erect without associated convulsions, incontinence, palpitations, or chest pain and with spontaneous recovery after 20 seconds. He had one prior syncopal event and multiple episodes of lightheadedness since 3 months ago, all occurring after standing from bending forward. He also complained of nonproductive cough for past 3 months as well as facial swelling after lying flat. Physical examination revealed blood pressure of 163/97 while lying and 135/78 while standing. He had slight facial plethora while sitting with significant worsening when leaning forward or straining. He had venous distention of chest wall. Computed tomography (CT) angiogram of the chest showed occlusion of the superior vena cava due to infiltrating adenopathy and mass effect of the left hilum with concern for infiltrating tumor measuring 4.1 x 5.4 cm. Bronchoscopy of the left upper lobe mass was performed with cytology most consistent with adenocarcinoma. A diagnosis of superior vena cava (SVC) syndrome secondary to adenocarcinoma was made, and a superior vena cava stent was placed with significant improvement in facial swelling and lightheadedness with no further syncopal events.

Discussion: SVC syndrome occurs when blood flow through the superior vena cava becomes obstructed, either through extrinsic compression, such as from a tumor, or intravascular obstruction. Mediastinal tumors are the cause of SVC syndrome in approximately 80% of cases, the majority of which are bronchogenic carcinomas. Nonmalignant causes of SVC syndrome include vascular disease, such as aortic aneurysm, and thrombosis related to central venous catheters. Partial obstruction of the SVC may cause few to no symptoms, whereas severe obstruction causes higher symptom burden. Early findings may include dyspnea, cough, hoarseness, ruddy face, and facial and arm edema, which typically worsen with bending forward. More advanced findings include facial and torso cyanosis, mental status changes, lethargy, and syncope. A contrast-enhanced chest CT is the most useful imaging modality for diagnosis of SVC syndrome as it can show the location and extent of venous blockage. Treatment of SVC syndrome due to underlying malignancy is focused on both treatment of cancer and relief of symptoms from obstruction. For many cases, initiation of chemotherapy with or without radiation causes rapid relief of symptoms. Cases with severe symptoms, such as syncope or respiratory distress, may require more urgent intervention, such as percutaneous placement of intravascular stent.

Conclusion: For a patient presenting with syncope, an expanded differential diagnosis must be maintained, and thorough history and physical examination are key in guiding work-up and management decisions.

Superior Vena Cava Syndrome Causing Syncope: When Cancer Catches Venous Congestion

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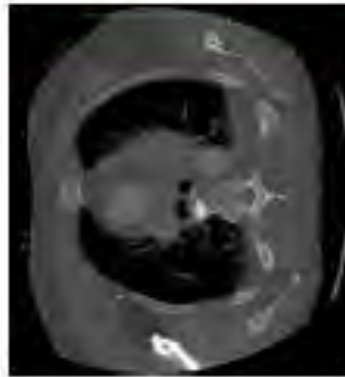
Objectives

- Recognize symptoms of superior vena cava syndrome
- Know common causes of SVC syndrome
- Case Presentation**
- 65-year-old Caucasian male presents due to syncope episode.
- Described the episode as having lightheadedness while bending forward with subsequent loss of consciousness after standing erect.
- Denies associated convulsions, palpitations, chest pain, or incontinence, and had spontaneous recovery within 20 seconds.
- Has had multiple episodes of lightheadedness upon standing, unproductive cough and facial swelling and numbness after lying flat for past 3 months.
- PMH: Hypertension
- Social History: 60 pack year smoking history
- Physical Exam: VS: T: 98.2 F, P: 77, BP: 164/88 (lying), 128/85 (standing), RR: 14, O₂ sat: 98% RA
- CV: RRR, no m/hg, no JVD
- Palp: CTAB
- Neck: No LAD
- HEENT: PEDEE.
- Neuro: 5/5 strength in all extremities, intact sensation
- Skin: Erythema of face and neck when arms raised. Facial swelling and erythema when lying flat

Diagnostic Work-Up and Hospital Course

$$6.8 \times \frac{13.6}{41.0} = \frac{140}{4.3} \times \frac{102}{22} = \frac{17}{1.0} \times \frac{90}{9.1}$$

- CT angiogram chest: Invasive mediastinal adenopathy, causing superior vena cava occlusion. Mass effect in left hilar region for infiltrating tumor measuring 4.1 x 5.4 cm
- Diagnosed with syncope due to SVC syndrome.
- Patient underwent angioplasty and stenting of the distal SVC and left brachiocephalic vein, with complete resolution of symptoms.
- Further attention was placed on the cause of patient's SVC obstruction.
- CT abdomen/pelvis and MRI brain: no evidence of metastasis
- Bronchoscopy: obstructing esophageal mass at left upper lobe apical segment. Transbronchial needle aspirations of mass and lymph nodes were performed
- Subcarinal lymph node bronchial FNA confirms non-small cell carcinoma, consistent with adenocarcinoma
- Nuclear medicine bone scan: increased activity within cervical, thoracic, and lumbar spine
- Patient was diagnosed with stage 4 lung cancer with metastases to vertebrae. He was discharged home on apixians after placement of SVC stent, with consideration of systemic chemotherapy versus palliative



Discussion

- Superior Vena Cava Syndrome: constellation of signs and symptoms resulting from obstruction of the superior vena cava
- Symptoms include:
 - Facial and arm edema
 - Cyanosis of head
 - Facial plethora
 - Distended subcutaneous veins
 - Cough
 - Hiccups
 - Headache, confusion
 - Hemodynamic compromise, syncope
- Causes include:
 - Malignancy (NSCLC, SCLC, lymphoma)
 - Thrombosis
 - Aortic aneurysm
 - Endocarditis
 - CT chest
 - Venography
 - Management:
 - Radiotherapy (if confirmed malignancy)
 - Systemic chemotherapy
 - Placement of intravascular stent
 - Surgery

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“SyphiLIPS”

Dallas Moran, MS3, UAB Huntsville Regional Medical Campus; Erin Baldwin, MS3, UAB Huntsville Regional Medical Campus; Haley Gates, MD, PGY1, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Rajalakshmi Cheerla, MD, Clinical Associate Professor of Family Medicine, UAB Huntsville Regional Medical Campus; Katherine Moody, Assistant Professor, Department of Family Medicine, UAB Huntsville Regional Medical Campus

Learning Objectives:

- 1.) Emphasize the importance of considering secondary syphilis in differential diagnosis of angioedema and oral ulcers.
- 2.) Remind clinicians of syphilis still being “the Great Imitator”.

Abstract Body:

Case Presentation: A 41 yo AAF with PMH of hypertension, hyperlipidemia, and alcohol abuse presented to the ER with lip swelling and increased heart rate. The patient reported several months of lip swelling and buccal mucosa masses, now significantly worse and preventing adequate PO intake. Patient’s PCP ordered ANA, rheumatoid factor, and HIV testing 7 months prior to this presentation, and referred to ENT for biopsy of the masses. Lab results and biopsy for malignancy were negative. On the day of presentation, the patient awoke with swollen lips, unable to eat, drink, or speak. In the ER the patient was tachycardic and hypotensive. Physical exam revealed significant upper and lower lip swelling resembling angioedema, removable white plaques and fissuring of the tongue somewhat resembling hairy leukoplakia, and cervical lymphadenopathy. There was no appreciable skin rash to her palms, soles, or the rest of her body. She was admitted for fluid resuscitation and further workup of mucositis and angioedema. Repeat HIV testing, hepatitis panel, and RPR were ordered. RPR was found to be reactive, and Infectious Disease was consulted for further workup. ID ordered VZV and HSV IgG and IgM, which were negative. Rheumatology was consulted for concerns of autoimmune disease, such as Behcet’s or Crohn’s. CRP, anticardiolipin, C3, C4, CH50, IBD panel, pANCA, and MPO were negative. During hospital course, the patient revealed she had worked in the sex trade industry for 20 years. The treponemal IgG and IgM was positive. She was treated with Penicillin G Benzathine 2.4 million units IM and followed up with the Health Department for 2 more doses, a week apart each. At 1 week follow up significant improvement was noted in her lip swelling and mucositis.

Discussion: Syphilis is a sexually transmitted infection caused by *Treponema pallidum*. In the US, there was a 76% increase in primary and secondary syphilis rates from 2013-2017. This increase was most prominent in men who have sex with men. However, risk factors include sex work, methamphetamine use, and acquiring a sexual partner via social media. Transmission occurs through direct contact with an infected lesion. Syphilis is classified into early or late. Early syphilis includes primary and secondary stages. Primary syphilis manifests as a painless chancre 3 to 90 days after initial infection. Chancres heal spontaneously within 3 to 6 weeks. Secondary syphilis manifests weeks to months later as constitutional symptoms, such as fever, malaise, headache, anorexia, myalgia, and weight loss with accompanying lymphadenopathy [epitrochlear highly specific]. The classic rash of secondary syphilis is diffuse, symmetric, macular or papular eruptions involving the trunk and extremities, including palms and soles. However, rash may present on mucosal surfaces such as the mouth or perineum as large, raised, white lesions known as condyloma lata. Often, mucosal rash occurs in the same location as the primary

chancre. Late syphilis occurs when patients remain untreated. This case report reminds clinicians to be aware of the change in epidemiology and risk factors for syphilis, as well as the varied manifestations of secondary syphilis, including isolated oral disease.

Centers for Disease Control and Prevention. *Syphilis Surveillance Supplement 2013–2017*. Atlanta: U.S. Department of Health and Human Services; 2019.



“SyphilisLips”

Dallas Moran MS3 and Erin Baldwin MS3
 Haley Gates M.D. PCYL, Katherine Moody M.D., and Rajalakshmi Cheerla M.D.
 UAB Department of Family Medicine and UAB School of Medicine

Introduction

A 41 yo AAF with PMH of hypertension, hyperlipidemia, and alcohol abuse presented to the ER with lip swelling and increased nasal pain. The patient reported several months of lip swelling and buccal mucosa masses, now significantly worse and preventing adequate PO intake. Patient's MCP ordered ANA, rheumatoid factor, and HIV testing 7 months prior



to this presentation, and referred to ENT for biopsy of the buccal masses. Lab results and biopsy for malignancy were negative. On the day of presentation, the patient arrives with swollen lips, unable to eat, drink, or speak. In the ER the patient was bedridden and hypotensive. Physical exam revealed significant upper and lower lip swelling, resembling angioedema, nonremovable white plaques and fissuring of the tongue somewhat resembling hairy leukoplakia, and cervical lymphadenopathy. There was no appreciable skin rash to her palms, soles, or the rest of her body. She was admitted for fluid resuscitation and further workup of mucositis and angioedema. During the hospital course, the patient revealed that she had worked in the sex trade industry for over 20 years. Patient denies any rash to her palms, soles, or any other part of her body in those 20 years working in the sex trade industry.



Methods

Rapid HIV testing, Hepatitis panel, and RPR were obtained. RPR was found to be reactive, and infectious Disease was consulted for further workup. ID ordered VZV and HSV IgG and IgM, which were negative. Rheumatology was consulted for concerns of an autoimmune disease, such as Behçet's or Crohn's disease. CRP, antinuclear Ab, C3, C4, CH50, IgD panel, pANCA, and MPO were negative. The treponemal IgG and IgM were positive. The patient was treated with 1 dose of Penicillin G Benzathine 2.4 million units IM and followed up with the Health Department for 2 doses subsequent a week apart each. At 1 week follow up significant improvement was noted in her lip swelling and mucositis.

Results

Rapid Plasma Reagin (RPR): reactive

Varicella Zoster Virus (VZV) IgG and IgM (titer): negative

Herpes Simplex Virus (HSV) IgG and IgM (titer): negative

Antinuclear (C-reactive protein, antinuclear Ab, C3, C4, CH50, IgD panel, pANCA, myeloperoxidase): negative

Discussion

Syphilis is a sexually transmitted infection caused by *Treponema pallidum*. In the US, there was a 76% increase in primary and secondary syphilis rates from 2013-2017. This increase was most prominent in men who have sex with men. However, risk factors include sex work, intravenous drug use, and acquiring a sexual partner via social media. Transmission occurs through direct contact with an infected lesion. Syphilis is classified into early or late. Early syphilis includes primary and secondary stages. Primary syphilis manifests as a painless chancre 3 to 90 days after initial infection.

Discussion (continued)

Chancres heal spontaneously within 3 to 6 weeks. Secondary syphilis manifests weeks to months later as constitutional symptoms, such as fever, malaise, headache, sore throat, myalgia, and weight loss with accompanying lymphadenopathy (epitrochlear highly specific). The classic rash of secondary syphilis is diffuse, symmetric, macular or papular eruptions involving the trunk and extremities, including palms and soles. However, rash may present on mucosal surfaces such as the mouth or perineum as large, raised, white lesions known as condylomata lata. Often, mucosal rash occurs in the same location as the primary chancre. Late syphilis occurs when patients are untreated during earlier stages of syphilis, and manifests as latent syphilis or tertiary syphilis.

Conclusion

This case report reminds clinicians to be aware of the change in epidemiology and risk factors for syphilis, as well as the varied manifestations of secondary syphilis, including isolated oral disease.

References

Centers for Disease Control and Prevention. Syphilis Surveillance Supplement 2013–2017. Atlanta: U.S. Department of Health and Human Services; 2019.

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“Thoughts of the Grave After Treating Graves”

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Case Report:

Case: An 18-year-old male with a hx of Graves disease presented 4 wks after radioactive iodine ablation. His methimazole was stopped per protocol 2 days before the ablation and resumed shortly thereafter. About 2 weeks later he developed what was thought to be a URI with rhinorrhea and cough that progressed to include fever, tachycardia, hypertension and clammy skin. Meds on admission included methimazole 10mg BID and atenolol 50mg QHS. PE: alert, BP 148/81, P 125, R 28, sats 99% on RA, HEENT – eyes nl with no lid lag or proptosis, neck with mild thyromegaly, no nodules, warmth, or erythema; lungs clear, heart RRR without murmurs, neuro exam nonfocal. While in the ED he admitted to recent suicidal ideation. Labs showed WBC 4k, 55S, 29L, Hgb 14, free T3 and free T4 higher than measurable levels and TSH below detectable limits. The patient was admitted on telemetry, his methimazole was increased to 10 mg TID and propranolol 30 mg TID was initiated. Over a two day hospital stay his vitals normalized and he had no further suicidal thoughts.

Discussion: Graves disease is the most common cause of hyperthyroidism in adolescents and young adults. Treatment options include pharmacotherapy, usually with methimazole, radioactive iodine ablation therapy or surgery. The advantage of radioactive iodine therapy is its ability to destroy the hyperactive thyroid tissue without the invasiveness of surgery; it is also less expensive. Most patients develop hypothyroidism after radioablation and are then able to be managed with simple, well-tolerated, inexpensive levothyroxine which has minimal side effects. A small subset of patients, however, can develop rebound hyperthyroidism. This can occur weeks to months after ablative therapy and effectively managed with increased methimazole as was done in our case. Thyrotoxicosis has been observed following trauma, non-thyroidal surgery, and infection. Although our patient’s hyperthyroid state was most likely one of the uncommon post-ablative reactions, his recent history of URI-like symptoms does suggest a possible alternative etiology: post-viral hyperthyroidism. Physicians must consider hyperthyroidism (even paradoxical as in this case) when new psychiatric symptoms, including suicidal ideation occur. Fortunately the suicidal ideation and other sx’s usually resolve once the condition is treated.



Thoughts of the Grave After Treating Graves

Luke Bailey; Roger D. Sinalligan, MD, MPH
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University of Alabama at Birmingham School of Medicine

Case History

- An 18-year-old male with PMH of Graves disease presented to the ED 4 weeks after radioactive iodine ablation.
- His methimazole was stopped, per protocol, 2 days before the ablation and resumed shortly thereafter.
- About 2 weeks later, he developed rhinorrhea, cough, fever, tachycardia, hypertension, fever, and clammy skin.
- While in the ED he admitted to recent suicidal ideation.
- Meds on admission: methimazole 10mg BID and atenolol 50mg QHS

Physical Exam

- Vitals: BP 148/81, P 125, R 28, 99% O2 Sat on room air
- HEENT: eyes normal with no lid lag or proptosis; neck with mild thyromegaly, no nodules, warmth, tenderness or erythema
- Lungs clear, heart tachycardic without murmurs, neuro exam non-focal

Labs

- CBC within normal limits, WBC 4,000
- Free T3 and free T4 higher above measurable levels
- TSH below detectable limits

Hospital Course

- The patient was admitted on telemetry, his methimazole was increased to 10 mg TID, and propranolol 30 mg TID was initiated.
- Over a two day hospital stay his vitals normalized and he had no further suicidal thoughts.

Discussion

- Graves disease is the most common cause of hyperthyroidism in adolescents and young adults.
- Treatment options include pharmacotherapy, radioactive iodine ablation therapy or surgery.
- The advantage of radioactive iodine therapy is its ability to destroy the hyperactive thyroid tissue without the invasiveness of surgery; it is also less expensive.



Diffuse radioactive uptake pattern seen in Graves disease thyroid

Discussion continued

- Hypothyroidism is typical after radioablation and it is managed with levothyroxine which is well tolerated.
- A small subset of patients develop rebound hyperthyroidism as did our patient. This can occur weeks to months after ablative therapy and requires increased methimazole.
- Although our patient's hyperthyroid state was most likely one of these uncommon post-ablative reactions, his recent history of URI-like symptoms does suggest the possibility of post-viral hyperthyroidism.
- This case provides an important reminder to physicians that hyperthyroidism can be the cause of new psychiatric symptoms.
- Fortunately suicidal ideation and other psychiatric symptoms typically resolve once the hyperthyroidism is treated.

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Toxic shock syndrome from protracted menstrual cup use

Jamie L. Kuck, MS3, UAB Huntsville Regional Medical Campus; Lourdes Corman, MD, Clinical Professor of Internal Medicine, UAB Huntsville Regional Medical Campus

Case

A 26-year-old African American female presented to the emergency department with a three-day history of progressive fever, headache, nausea, and diarrhea. She also reported vaginal itching and decreased urine output, but denied discharge and dysuria.

Five days prior to presentation, she appropriately inserted a DivaCup® menstrual cup, but forgot to remove it. She developed a low-grade fever and headache after 48 hours, both relieved by acetaminophen. At 72 and 96 hours, her temperature reached 38.9°C and 40.5°C, respectively, which prompted her visit.

She appeared lethargic with temperature 37.8°C, blood pressure 66/38 mmHg, heart rate 163 beats/min, and respiratory rate 22 breaths/min. Physical exam showed periorbital and facial edema, but no rash. Pelvic exam revealed a menstrual cup holding 150 ml of serosanguinous purulent fluid and microscopic analysis revealed Gram-positive cocci. Broad-spectrum antibiotics were initiated and she was admitted to the ICU.

Cultures from fluid within the retained menstrual cup grew methicillin-sensitive *Staphylococcus aureus*, extended-spectrum beta-lactamase-producing *Escherichia coli*, and *Bacteroides fragilis*. She developed acute kidney injury, shock liver, thrombocytopenia, and sterile pyuria. Blood cultures x2 and urine culture remained negative. On hospital day 12, diffuse desquamation was noted.

Conclusions and Clinical Significance

Our patient met the Centers for Disease Control and Prevention case definition for staphylococcal toxic shock syndrome (TSS): fever, hypotension, desquamation, multisystem involvement, and blood cultures negative for alternative pathogens. She failed to develop the rash characteristic of TSS, which is often subtle, and particularly difficult to distinguish in patients with dark skin.

Staphylococcal TSS is associated with tampon use. Reusable menstrual cups are marketed as a safe, environmentally-friendly alternative to tampons. Despite few reports of menstrual cup-associated TSS, *in vitro* data suggest that cups facilitate higher growth of *Staphylococcus aureus*. This case highlights the need for further investigation and informed provider-patient dialogue regarding the risks associated with menstrual cups.

¹UAB School of Medicine, ²Department of Internal Medicine, UAB Huntsville Regional Medical Campus

Abstract

The apparent isotherm with temperature 37 °C, blood pressure 66/38 mmHg, heart rate 163 beats/min, and respiratory rate 22 breaths/min. Physical exam showed petechiae and nasal edema, but no rales. Pulvic exam revealed a menstrual cup holding 150 ml of cervicovaginal fluid and microscopic analysis revealed Gram-positive cocci. Crossed-spectrum antibiotics were initiated and anal swab was submitted to the ICU.

Culture from fluid within the retained meningeal flap grew multidrug-resistant (*Mycobacterium avium*, susceptible-spectrum beta-lactamase-producing) bacteria and fungi (*Cryptococcus neoformans*). Blood culture (12) and urine culture remained negative. On hospital day 12, diffuse demyelination was noted.

Glyceryltrimethyl TBS is associated with benign use. However, intrauterine cups are marketed as a safe, environmentally-friendly alternative to tampons. Despite low reports of menstrual cup-associated TSS, *in vitro* data suggest that cups facilitate higher growth of *Clostridium* strains. This case highlights the need for further investigation and informed female patient dialogue regarding the risks associated with menstrual cups.

History of Present Illness

Patient inserted DivaCup® during menses and failed to retrieve it

48 hours after insertion, she developed mild fever and headache relieved by Tylenol

T_{max} at home at 96 hours was 40.9°C

- Patient inserted DivaCup® during menses and failed to retrieve it
- 48 hours after insertion, she developed mild fever and headache relieved by Tylenol
- T_{max} at home at 96 hours was 40.9°C

Presentation to ED	
Vital Signs	
Temperature (°C)	37.3
Blood pressure (mm Hg)	66/38
Heart rate (beats/min)	163
Respiratory rate (breaths/min)	22
Oxygen saturation (%)	98
Height (cm)	158
Weight (kg)	58.1
BMI	25

Physical Examination	
General	Drowsy, ill
HEENT	Periorbital and facial edema
Cardiovascular	Tachycardia
Pulmonary	CTAB
Gastrointestinal	HBS, NTND
Genitourinary	Suprapubic tenderness, 150 ml serosanguinous fluid within retained menstrual cup
Skin, Extremities	No rashes, desquamation, edema
Neurologic	Grossly intact

Results

- Hypotension not improved with 4L NS
- Staining of menstrual cup fluid → G+ cocci
- Vancomycin, piperacillin/tazobactam, clindamycin
- BP responded to Levophed

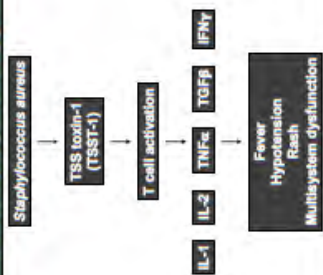
Lab Parameter (normal value)	Patient value
White blood cell count ($4.1\text{--}12.2 \times 10^3/\mu\text{l}$)	10.56
Hemoglobin ($11.5\text{--}14.7 \text{ g/dl}$)	12.3
Hematocrit ($37\text{--}52 \times 10^3/\mu\text{l}$)	74
Platelets ($137\text{--}352 \times 10^3/\mu\text{l}$)	4
Creatinine ($0.5\text{--}1 \text{ mg/dl}$)	31
Blood urea nitrogen ($6\text{--}20 \text{ mg/dl}$)	ND
Creatine Phosphokinase	40
Alanine aminotransferase ($<32 \text{ U/L}$)	48
Aspartate aminotransferase ($<33 \text{ U/L}$)	4.5
Total bilirubin ($<1 \text{ mg/dl}$)	6.76
Lactate ($0.5\text{--}2 \text{ mmol/L}$)	20 WBC/hpf
Urinalysis	Small bacteria
BHCG	Negative
Blood culture	Negative $\times 2$
Urine culture	Negative

Lab Parameter (normal value)	Patient value
White blood cell count (4.5-12.2 x10 ⁹ /l)	10.56
Hemoglobin (11.5-14.7 g/dl)	12.3
Platelets (137-352 x 10 ⁹ /pl)	74
Creatinine (0.5-1 mg/dl)	4
Blood urea nitrogen (6-20 mg/dl)	31
Creatine Phosphokinase	ND
Alanine aminotransferase (<32 U/L)	40
Aspartate aminotransferase (<33 U/L)	48
Total bilirubin (<1 mg/dl)	4.5
Lactate (0.5-2.2 mmol/L)	6.76
Urinalysis	20 WBC/hpf
	Small bacteria
BHCG	Negative
Blood culture	Negative x 2
Urine culture	Negative

```

graph TD
    A[Staphylococcus aureus] --> B[TSS toxin-1 (TSST-1)]
    B --> C[T cell activation]
    C --> D[IL-1, IL-2, TNF-α, TGFβ, IFN-γ]
    D --> E[Fever, Hypotension, Rash, Multisystem dysfunction]
  
```

The diagram illustrates the pathophysiology of Toxic Shock Syndrome (TSS) as a sequential process. It begins with **Staphylococcus aureus**, which produces **TSS toxin-1 (TSST-1)**. This toxin leads to **T cell activation**, which in turn triggers the release of various cytokines: **IL-1**, **IL-2**, **TNF-α**, **TGFβ**, and **IFN-γ**. These cytokines collectively cause the clinical manifestations of TSS, including **Fever**, **Hypotension**, **Rash**, and **Multisystem dysfunction**.



Clinical Criteria		Patient
Fever		$T_{max} \geq 38.5^{\circ}\text{C}$
Rash		Diffuse macular erythroderma
Desquamation		1-2 weeks after onset of rash
Hypotension		SBP < 90 mm Hg
Multisystem Involvement (≥ 3 systems)		
GI		Vomiting or diarrhea at onset
Muscular		Severe myalgias or CPK $> 2 \times$ ULN
Mucous membranes		Vaginal, oropharyngeal, conjunctival hyperemia
Renal		BUN or Cr $\geq 2 \times$ ULN or Sterile pyuria
Hepatic		Bilirubin _{max} ALT, or AST $\geq 2 \times$ ULN
Hematologic		Platelets < 100,000/mm ³
CNS		AMS without FND when fever and hypotension absent
Laboratory Criteria		
(-) Blood or CSF cultures		Blood cultures negative x 2
(+) Serology: RMSF, leptospirosis, or maulasia		

Clinical Criteria	Patient
Fever	Temperature $\geq 38.5^{\circ}\text{C}$ T _{max} 40.5°C
Rash	Diffuse macular erythroderma Undetected
Desquamation	Present
Hypotension	1-2 weeks after onset of rash SBP < 90 mm Hg
Multisystem involvement (≥ 3 systems)	
GI	
Muscular	Vomiting or diarrhea at onset
Mucous membranes	Severe oral dysplasia or CPK $\geq 2 \times$ ULN
Renal	Vaginal, conjunctival, conjunctival hyperemia BUN or Cr $\geq 2 \times$ ULN or Sterile pyuria
Hepatic	Bilirubin _{total} , ALT, or AST $\geq 2 \times$ ULN
Hematologic	Platelets < 100,000/mm ³
CNS	AMS without FND when fever and hypotension absent
Laboratory Criteria	
(+) Blood or CSF cultures	Blood cultures may be positive for Staphylococcus aureus
(+) Serology: RMSF, leptospirosis, or maulsies	Blood cultures negative x 2

Clinical Significance

- *Staphylococcal TSS* is associated with tampon use
- Reusable menstrual cups are marketed as a safe, environmentally-friendly alternative to tampons
- Another case involving TSS from menstrual cup use is reported in the medical literature²
- *In vitro* data suggest that cups facilitate higher growth of *S. aureus* compared to tampons³
- Menstrual cup manufacturers do not provide safety information to consumers
- Providers should acknowledge the increased use of menstrual cups and be prepared to discuss risks with patients

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- Reusable menstrual cups are marketed as a safe, environmentally-friendly alternative to tampons
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Unusual Case of Bilateral Foot Drop

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Learning objectives: a) Potential causes of bilateral foot drop
b) Diagnosis and management of peroneal neuropathy

Case Description: 31 yo Puerto Rican female presented to the ED with low back pain radiating to bilateral lower extremities (LE). Pain started after 3 weeks of falling onto her tailbone and buttocks. She mentioned unintentional weight loss of 40 lbs within 1 month. She mentioned weakness, paresthesia in LE, and difficulty ambulating after the incident. Denies any urinary or bowel incontinence, and fever. PMHx – Not significant. FMHx – Noncontributory. Patient takes no meds on a regular basis. PE: Weakness in bilateral LE, 0/5 strength with bilateral dorsiflexion of the ankle, ankle eversion, and the big toe. Compartments were soft and nondistended, inability to wiggle her toes bilaterally, +1 bilateral knee and Achilles reflex, abnormal slapped feet gait where she walks on her tips toes like walking on high heels. Initial labs: CMP and CBC within normal limits, vitamin B12 levels normal, HIV testing was negative. Neurology was consulted. MRI w/ and w/o contrast of the spine only demonstrated mild right foramina protrusion of the L5-S1 disc. EMG showed no definite electrophysiological evidence of any right lower lumbar radiculopathy. The nerve conduction study demonstrated bilateral peroneal neuropathy across the fibular segment. Extensive workup for weight loss did not reveal any cause. Lumbar puncture to evaluate Guillain-Barre syndrome was negative. Dx: Bilateral peroneal neuropathy due to acute onset of weight loss. Orthopedic surgery was consulted, recommended conservative management. They recommended no acute decompression surgery and believed it would improve with time. Ortho ordered ankle foot orthosis (AFO), Podus boots, and stretching to help. Advised to do physical therapy. Decompression surgery in the future if no improvement. Patient never followed up after discharge.

Discussion: Foot drop is the most common presentation of peroneal nerve neuropathy due to pressure or trauma. The mechanism of injury is by compression. Peroneal nerve injury is commonly unilateral and rarely bilateral. The incidence of unilateral vs bilateral is currently unknown due to the rare number of cases of bilateral peroneal neuropathy. The common location of compression was at the fibular head. Most common causes of bilateral peroneal neuropathy were after bariatric surgery (loss of 143 lbs over time in previous cases) or after prolonged lithotomy position during childbirth. Common presentation is usually difficulty walking and with the sensation of dragging the toes. Diagnosis of peroneal nerve neuropathy is a clinical diagnosis, and the gold standard is by EMG and nerve conduction studies. The treatment of peroneal neuropathy is watchful waiting, conservative management with AFOs, Podus boots help with ambulation, and physical therapy is recommended. Decompression surgery is only done if conservative management has failed. The percentage of recovery after the surgery is 70-75%.



Introduction

Bilateral peroneal neuropathy is a rare occurrence compared to unilateral. There are many causes for unilateral peroneal neuropathy, but extremely less for bilateral. This case explores the causes and treatments for bilateral peroneal neuropathy.

Case Presentation

- 31 yo Puerto Rican female presented to the ED w/ low back pain radiating to bilateral lower extremities (LE).
- Pain started after 3 wks of falling onto her tailbone and buttocks. She increased unintentional weight loss of 40 lbs w/ 1 month weakness.
- paresthesia in LE and difficulty ambulating after the incident.
- Dresses urinary, bowel incontinence, and fever.
- No significant PMHx
- Patient takes no meds.
- PE: Weakness in bilateral LE 0/5 strength w/ bilateral dereliction of the ankle, ankle eversion and big toe +1 bilateral knee and Achilles reflex, abnormal clapped feet gait when she walks.
- Initial labs: CMP, CBC, VB12 w/ normal limits, HIV testing negative.
- Neurology consulted
- MRI w/ and w/o contrast of the spine - mild right foraminal protrusion of the L5-S1 disc
- EMG - no definite electrophysiological evidence of any right lower lumbar radiculopathy
- NCS - bilateral peroneal neuropathy across the fibular segment
- Weight loss working negative
- Lumbar puncture - negative for Guillain-Barre syndrome

Management

Dr. Bilateral Peroneal Neuropathy most likely due to weight loss. Orthopedic surgery was consulted. Recommended conservative management, and believed it would improve with time. Ortho ordered ankle foot orthosis (AFO), Podiat boots, and stretching to help. Advised to do physical therapy. Decompression surgery in the future if no improvement. Patient never followed up after discharge.

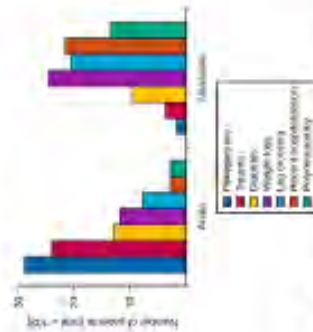


Fig 1 - Causes of unilateral peroneal neuropathy



Fig 2 - Foot Drop

Discussion

- Foot drop is the most common presentation of peroneal nerve neuropathy due to pressure or trauma. The mechanism of injury is by compression.
- Peroneal nerve injury is commonly unilateral and rarely bilateral. The incidence of unilateral vs bilateral is currently unknown due to the rare number of cases of bilateral peroneal neuropathy.
- Common location of compression was at the fibular head
- Most common cause of bilateral peroneal neuropathy were after bariatric surgery (loss of 143 lbs over time in previous cases) or after prolonged lithotomy position during childbirth.
- Common presentation is usually difficulty walking and with the sensation of dragging the toes.

Discussion (cont)

- Diagnosis of peroneal nerve neuropathy is a clinical diagnosis, and the gold standard is by EMG and nerve conduction studies.
- Treatment of peroneal neuropathy is watchful waiting and conservative management.
- Percentage of recovery after surgery is 70-75%.

Conclusion

This case shows that bilateral peroneal neuropathy can be caused by unintentional extreme weight loss. Our case was unique in that it was complicated by her fall, which was initially thought of as the cause for the foot drop.

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When the Cure Brings a Complication: Atypical Stevens-Johnson Syndrome After Gonorrhea Treatment

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Learning Objective #1: Diagnose Atypical Stevens-Johnson syndrome

Learning Objective #2: Understand cyclosporine can speed recovery in severe cases of Stevens-Johnson

Case: A 21yo male with recent gonorrhea presented with severe swelling of the lips, difficulty swallowing, and conjunctival injection. Two weeks prior he had dysuria, penile discharge and a swollen inguinal node. He had a swab done at an ER (later positive for gonorrhea, negative for Chlamydia) and was treated with ceftriaxone, azithromycin and sent home with doxycycline. One day later he noticed lip swelling and conjunctival injection. Despite concerns was allergic he continued taking doxy 2 more days, developed more swelling and severe sore throat and difficulty swallowing and returned to the ER. He denied continued penile discharge and oral sex and fever. On exam he was alert, anxious, drooling, afebrile, 132/80, P 80, R 14, O2 sats 95% on RA, extensive conjunctival injection, swollen lips and tongue with mucosal ulcers with whitish exudate, no cervical nodes, remainder of exam negative including no significant skin lesions. Labs: WBC 6.74 , Hgb 15.7 , platelets 240k, HIV neg. Although resistant gonorrhea was considered, the patient was felt to have atypical Stevens-Johnson syndrome (SJS) associated with one of his antibiotics, likely doxycycline. He was unable to handle his secretions and initially had supportive care. Ophthalmology was consulted for worsening conjunctival involvement and steroid eye drops were started. Worsening oral lesions/dysphagia prompted use of IV cyclosporine, at which point he began to improve rapidly and was discharged on the 4th day.

Impact/Discussion: Classic Stevens-Johnson syndrome includes rash, mucosal and conjunctival involvement, most commonly after some trigger such as a medication. HIV infection, malignancy and certain HLA types are also risk factors and the most commonly implicated drugs are antiepileptic antibiotics, sulfonamides, and allopurinol. This case is considered atypical in that there was no rash but the mucosal and conjunctival inflammation was otherwise classic and severe. This variation has been described some in children but rarely in adults. The pathogenesis is thought to be immune-mediated as in the classic form. Treatment is the same: although steroids are used by some, evidence leans towards supportive care as the treatment of choice. Careful monitoring of fluids and electrolytes is key. Some case series have shown cyclosporine to speed recovery and our patient responded better after it was started. Too few cases are reported to describe mortality of the atypical form but since mortality in SJS/toxic epidermal necrolysis is associated with >30% of skin sloughing, it can be presumed the rate is low in the atypical form.

Conclusion: Physicians need to be aware that an atypical form of Stevens-Johnson syndrome can occur with severe mucosal and conjunctival involvement but without the rash. Treatment and approach are similar to the classic SJS and outcomes should be favorable.

Introduction

Classic Stevens-Johnson syndrome (SJS) includes rash, mucosal and conjunctival involvement, most commonly after some trigger such as a medication. HIV infection, malignancy and certain HLA types are also risk factors and the most commonly implicated drugs are antiepileptics, antibiotics, sulfonamides and allopurinol. Although rare, an atypical case may present without rash.

Case Presentation

- A 21yo male with recent gonorrhea presented with severe swelling of the lips, difficulty swallowing and conjunctival injection.
- Two weeks prior he had dysuria, penile discharge and a swollen inguinal node. One week before presenting he had a urethral swab at an ER (later positive for gonorrhea, negative for Chlamydia) and was treated with ceftriaxone, azithromycin and sent home with doxycycline.
- He developed lip swelling, conjunctival injection and worried about an allergic reaction but took doxycycline several days. He ultimately presented with severe sore throat and difficulty swallowing and was admitted.
- On exam he was alert, anisocoric, drooling, afebrile, 132/90, P 80, R 14, O2 sat 95% on RA, extensive conjunctival injection, swollen lips and tongue with mucosal ulcers with whitish exudate, no cervical nodes, remainder of exam negative including no significant skin lesions.
- Rheumatology agreed the patient had atypical Stevens-Johnson syndrome associated with one of his antibiotics, possibly doxycycline.

Management

The patient was unable to handle his secretions and initially had supportive care. Ophthalmology was consulted for worsening conjunctival involvement and steroid eye drops were started. Worsening oral lesions/odynophagia prompted use of IV cyclosporine, at which point he began to improve rapidly and was discharged on the 4th day.



Figure 1 – Stevens-Johnson syndrome (similar to our patient)

Discussion

- Stevens-Johnson syndrome is a severe mucocutaneous reaction characterized by extreme necrosis and detachment of the epidermis.
- Classic Stevens-Johnson syndrome includes rash, mucosal and conjunctival involvement, most commonly after some trigger such as a medication.
- HIV infection, malignancy and certain HLA types are also risk factors and the most commonly implicated drugs are antiepileptics, antibiotics, sulfonamides, and allopurinol.
- This case is considered atypical in that there was no rash but the mucosal and conjunctival inflammation was otherwise classic and severe.
- This variation has been described occasionally in children; rarely in adults.
- The pathogenesis is thought to be immune-mediated as in the classic form.
- Too few cases are reported to describe mortality of the atypical form but since mortality in SJS/toxic epidermal necrolysis is associated with >30% of skin sloughing, it can be presumed the rate is low in the atypical form.

Discussion Cont.

- Treatment Options**
 - Although steroids are used by some, evidence leans towards supportive care as the treatment of choice.
 - Careful monitoring of fluids and electrolytes is key.
 - Some case series have shown cyclosporine to speed recovery and our patient responded rapidly once it was started.

Conclusion

Physicians need to be aware that an atypical form of Stevens-Johnson syndrome can occur with severe mucosal and conjunctival involvement but without the rash. Treatment and approach are similar to the classic SJS and outcomes should be favorable.

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Section II: Research Abstracts

Better Transitional Care & Reimbursement Outcomes with Introduction of TOC Template in HER

Kelly Diaz, MD, PGY3, Family Medicine Resident; MD, Assistant Professor, Department of Family Medicine, UAB Huntsville Regional Medical Campus

Introduction: In an era of value-based care, transitions of care are important. The Centers for Medicare and Medicaid Services (CMS) defines a “transition of care” as a transfer of patient care from one setting to another (e.g. hospital, skilled nursing facility, primary care office, etc.) and implemented CPT codes for transitional care management (TCM) in 2013. According to the National Transitions of Care Coalition, one in five U.S. patients discharged from the hospital experienced an avoidable adverse event within three weeks. Hospital readmissions account for \$15 billion dollars of Medicare spending and ~20% of Medicare beneficiaries are readmitted within 30 days.¹ Primary care providers and their staff face barriers in identifying recently hospitalized patients, obtaining discharge summaries, and scheduling follow-up visits. This translates to poor quality of care, patient dissatisfaction, increase in preventable readmissions, and lost income.

Hypothesis: As a family medicine residency program composed of 36 residents, we hypothesized that implementing a Transition of Care (TOC) note template in our EHR would allow for two major benefits: (1) improved handoffs between inpatient/outpatient providers and (2) improved capture of transitional care management charges.

Methods: On January 1, 2013, CMS established transitional care management CPT codes 99495 and 99496. These codes encompass a 30-day service period and may be billed the 29th day following hospital/facility discharge. The code may be billed once during that 30-day period. Three components comprise TCM: interactive contact, provision of certain non-face-to-face services, and a face-to-face visit within seven to fourteen days.² Our residency program created an EHR TOC note template based on Medicare guidelines including details of hospitalization, providers familiar with the patient, and medication reconciliation on the date of discharge from the hospital. Each note was sent to clinic ancillary staff who called patients to ensure that acute needs and non-face-to-face services were met and to schedule the patient for a follow-up visit in clinic. Then, the note was forwarded to the patient’s assigned primary care physician and attending physician for review and final signature. The TOC notes remained in the medical record as a visual trigger, source of information, and documentation of requirements needed for billing. Our TOC note template was released in late 2016 and residents, faculty, and clinic staff were educated throughout 2017 as the TOC note was piloted. A record of CPT codes 99495 and 99496 billed from 2016-2018 was kept.

Results: The number of transitional care management codes that were billed increased each year. In 2016, prior to the new note template being widely used, only 50 TCM codes were billed. In 2017 during the pilot year of the intervention, 149 TCM codes were billed. In 2018, 216 TCM codes were billed at the time of this investigation (excludes most of the month of December). This represents a 332% increase in

¹National Transitions of Care Coalition. “Improving Transitions of Care”. September 2010.
<<http://www.ntocc.org/portals/0/pdf/resources/ntoccissuebriefs.pdf>> Accessed 12/30/18.

²Department of Health and Human Services Centers for Medicare and Medicaid Services. “Transitional Care Management Services”. Medicare Learning Network. ICN 908628. December 2016.

TCM codes billed from 2016 to 2018. This correlates to a 348% increase in potential revenue generated from TCM codes.

Conclusions: Utilizing a specialized transitions of care note template in the electronic medical record can be beneficial in several ways: to serve as a visual cue of recent hospitalization; to prompt office staff to follow up by phone and schedule an appointment; to improve continuity of care and provide useful handoff information; and finally, to improve capture of CPT codes 99495 and 99496. These findings are likely not unique to our residency program and similar TOC/TCM note templates may be implemented widely in other primary care settings.

*** This vignette was chosen for oral presentation on Research Day. Contact Kelly Diaz at kdiaz@uabmc.edu for a copy of the PowerPoint.**

Case Series of Patients with Liver Abscess in a Large Tertiary Center

Aristotle Asis, MD, PGY3, Internal Medicine Resident; Ali Hassoun, MD, Infectious Disease Specialist, Alabama Infectious Diseases Center, UAB Huntsville Regional Medical Campus; Jesse Faulk, MD, PGY2, Department of Internal Medicine, UAB Huntsville Regional Medical Campus; Esmeralda Gutierrez-Asis, MD, Internal Medicine Hospitalist, UAB Huntsville Regional Medical Campus

Background

Pyogenic liver abscess is associated with significant morbidity, mortality and health care burden. Annual incidence has been estimated at 2.3 cases per 100,000 people.

Methods

Retrospective chart review of patients with liver abscess during 2012-2017 was done. Demographics, laboratory data, intensive care unit (ICU) and hospital stay, comorbidities, and mortality were collected.

Results

28 patients were included. 79% were males, Caucasians 78.57% and 17.86% African Americans. Most common presentation: abdominal pain (43%) and fever (17.86%). Mean age was 53 years (19-87 years) (73% >49 years). 78.57% of patients had percutaneous drainage. Most common organisms were gram negative bacilli (50%), gram positive cocci (17.86%) and no growth (32.14%). *Escherichia coli* (12.12%) and *Klebsiella pneumoniae* (12.12%) were among the most common isolates. 18% were polymicrobial with same predominant organisms. Fungal infection found in one patient with *Candida glabrata* was isolated. None of the gram-negative bacilli were extended spectrum beta-lactamase (ESBL) enzyme producer. Most common comorbidities were hypertension (39.29%), hyperlipidemia (21.43%) and type 2 diabetes (17.86%). Average hospital stay was 13 days (1-50 days). 14.29% of patients needed ICU admission. 17.86% of patients was re-admitted but those who stayed in the ICU had re-admission rate of 75% and longer hospital stay. 23% of patients treated empirically with piperacillin-tazobactam and 20% of patients were discharged on ceftriaxone.

Conclusion

Gram negative bacilli especially *Escherichia coli* and *Klebsiella pneumoniae* remain the most commonly isolated organisms in patients with pyogenic liver Abscess. Patients have prolonged hospital stay and high re-admission rate. Further studies are needed to guide appropriate effective management.

*** This vignette was chosen for oral presentation on Research Day. Contact Aristotle Asis at aristotleasis@uabmc.edu for a copy of the PowerPoint.**

Curriculum Development: Controlled Substance Prescribing and Diversion Prevention in a Family Medicine Residency Program

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Haley Phillippe, Assistant Clinical Professor of Pharmacy Practice, Auburn University Harrison School of Pharmacy

Main Body: With over 50 million patients in the United States receiving at least one prescription for opioid medications in 2017, combine with data showing Family Medicine physicians as prescribing the most opioid prescriptions of any specialty that year, it is more important than ever that Family Medicine residents are exposed to and trained in good opioid prescribing habits based on the current data and sociopolitical trends. Facing a legislative backlash and a culture of crisis the medical community now has no choice but to address opioid prescribing head on and in a way that is best for both physicians and patients. The UAB-Huntsville Family Medicine Residency Program has developed a controlled substances policy that is on the forefront of this issue and allows residents the opportunity to develop experience with opioids and other controlled substances. Along with a policy outlining requirements for starting chronic opioid and benzodiazepine therapy the program includes guidelines on risk assessment tools, prescription drug monitoring programs, urine drug screens, pill counts, documentation, escalation of therapy, de-escalation of therapy, and termination protocols. The program also has a controlled substance review committee to review charts and provide guidance to both residents and faculty alike on best practices and management options. This policy was approved and implemented January 1, 2019.

*** This vignette was chosen for oral presentation on Research Day. Contact Jeremy Johnson at jeremytjohnson@uabmc.edu for a copy of the PowerPoint.**

Effectiveness of a neonatal lactation nurse in preventing hypernatremic dehydration in a rural Kenyan hospital

Milza Oppen Howard, MS4, UAB Huntsville Regional Medical Campus; Arianna Shirk, MD, Clinical Instructor, Division of Pediatric Medicine, UAB; Annalise Sorrentino, MD, Professor, Division of Pediatric Medicine, UAB

Introduction: Hypernatremic dehydration in neonates is a potentially life-threatening condition commonly leading to a variety of adverse neurologic and vascular sequelae. It is a common reason for hospitalization in the neonatal period in East Africa, and is often caused by breastfeeding difficulties. In October 2015, a full-time lactation nurse was added to staff at Kijabe Hospital in Kenya in efforts to identify babies at risk of developing hypernatremia due to breast-feeding difficulties.

Objectives: Determine the impact of hiring a full time neonatal nurse to foster lactation and examine babies born in Kijabe Hospital. Specifically, to determine how this affected the rate of hypernatremic dehydration and related outcomes.

Methods: A database of all sick neonates admitted to Kijabe Hospital revealed 228 newborns (<28 days old) admitted to Kijabe Hospital with hypernatremia between October 1, 2014 – December 31, 2016. Chart review was then used to compare records of the neonates with hypernatremia between two groups: those born in Kijabe before the hiring of the full-time lactation nurse (October 1, 2015) and those born after. For each newborn, admission information, symptoms, laboratory findings, comorbidities, treatments, complications, and mortality, if documented in original chart, were recorded.

Results: The average serum sodium concentration was higher in the group of neonates born before the full-time nurse was hired than the group after ($p=0.0064$). Creatinine levels were significantly lower in the group after the nurse was hired compared to before ($p=0.0043$); significance remained when only maternal lactation issues were included ($p=0.0149$).

Conclusions: Hiring a full-time lactation nurse at Kijabe Hospital was associated with decreased sodium on admission and decreased creatinine. This indicates that hiring a lactation nurse is potentially beneficial and may decrease the severity of illness associated with hypernatremic dehydration.

*** This vignette was chosen for oral presentation on Research Day. Contact Milza Howard at milza@uabmc.edu for a copy of the PowerPoint.**

Epidemiology and Clinical Characteristics of Septic Bursitis

Sujatha Baddam, MD, PGY2, Internal Medicine Resident; Elizabeth Rosell Cespedes, MD, Internal Medicine Specialist; Ali Hassoun, MD, Infectious Disease Specialist, Alabama Infectious Diseases Center, UAB Huntsville Regional Medical Campus

Background

Septic bursitis is an inflammation of the bursa usually caused by bacterial inoculation either directly or spread from nearby soft tissues or hematogenous spread. Limited evidence based data available in evaluation and management of septic bursitis. We aimed to characterize the predisposing medical conditions, most common presentation, microbiology, therapy as well as complications.

Methods

We conducted a retrospective chart review of patients admitted to single center with the diagnosis of septic bursitis over the last 5 years from January 2012 to January 2017. Data were gathered from patients with culture-positive septic bursitis. Data collected include: demographics, predisposing medical conditions, clinical presentation, site of infection, type of pathogen, type and length of antibiotics used, as well as complications.

Results

220 charts with the admission diagnosis of Septic bursitis were reviewed. Only 54 patients had positive cultures of bursal fluid. Most patients were male (72%) and median age was 55 yrs (range 15-89). Olecranon bursa was the most common site of infection (57.4%), followed by pre-patellar bursa (35.1%), and less common was trochanteric bursitis (7.4%). Pre-patellar bursitis was more common in the younger age group. 61% of Patients were healthy adults without significant comorbidities, 12.96% of patients were diabetics, 3.7% had the combination of diabetes and Immunosuppression; 5.6% were alcoholics. The most common presentation was pain and erythema (n= 27). Almost all patients required an Incision and drainage. Most common organism isolated from bursa fluid was Methicillin resistant *Staphylococcus aureus* (MRSA) (40.7%), followed by Methicillin sensitive *Staphylococcus aureus* (MSSA) (31.5%) and *Proteus Mirabilis* in 5.6% of patients. 40 patients had blood culture done, of these only 10% (4/40) had concurrent bacteremia with most common organism MRSA (n=2), followed by MSSA (n=1) and *Proteus Mirabilis* with *Enterococcus faecalis* (n=1). Mean CRP was 8.4 and mean ESR was 33. Only 40% treated with Antibiotic monotherapy empirically. Most common monotherapy used was vancomycin (24%). Vancomycin and Zosyn was the most commonly used combination (12.9%), followed by Vancomycin and Clindamycin (7.4%) and Vancomycin and Rocephin (7.4%).

Conclusion

Septic Bursitis predominantly occur in Males. Majority were healthy Adults without any comorbidities and those with chronic diseases, diabetes and alcohol are common risk factors. *Staphylococcus aureus* was the most commonly isolated organism. Vancomycin was the most commonly used antibiotic. Further studies are required to elucidate appropriate antibiotic use.

*** This vignette was chosen for oral presentation on Research Day. Contact Sujatha Baddam at sbaddam@uabmc.edu for a copy of the PowerPoint.**

Epidemiology, Clinical Manifestations, and Outcomes of the 2017-2018 Influenza Season Among Hospitalized Patients at a Tertiary Care Center

Bhavyaa Bahl, MD, PGY3, Department of Internal Medicine; Rohini Ramamoorthy, MD, PGY3, Internal Medicine Resident; Soujanya Thummathati, MD, PGY3, Internal Medicine Resident

BACKGROUND

2017-2018 Influenza season has garnered much interest from the medical community due to it being classified as a “high severity” season by the CDC. There has been much curiosity in identifying the characteristics, morbidity and mortality, and its association with vaccination status, flu type and other factors for extrapolation and application of this data in preparation of the influenza seasons to come.

METHODS

Retrospective chart review of patients with Influenza admitted from September 1, 2017 to April 1, 2018. Diagnosis was confirmed by rapid flu test (RIDT) or Target Enriched Multiplex PCR (TEM PCR). Demographic, clinical, diagnostic, management, and outcomes data were obtained. Analysis included calculating prevalence and relative risk (RR).

RESULTS

220 patients were identified (47% males, 73% White). Median age was 70 years (range 18–99). Type A and B were noted in 65% and 27% respectively. 81% patient came from home and, 17% from a facility (nursing home, assisted living). 49% had been vaccinated for influenza. With a RR of 1.31 (95% CI 0.85–2.01, $P = 0.21$), no association was noted between the causative influenza strain and the vaccination status. Commonly seen comorbidities included lung disease (44%), obesity (41%), diabetes mellitus (36%), CAD (34%), congestive heart failure (31%). Common presentations were respiratory (79%) and constitutional (53%). 68% were hypoxic and 4% hypotensive on arrival. Sensitivity of RIDT was 38%. 91% were treated with oseltamivir (21% within 48 hours of flu detection). Median treatment duration was 5 days. Hospitalizations peaked in January. Median length of hospital stay was 6 days. 23% had severe flu (needed NPPV 13%, intubation 12%, pressor 5%, ICU stay 16%) which showed significant association with arrival from a facility RR 2.21 (95% CI 1.36–3.56, $P = 0.001$), lung disease RR 1.91 (95% CI 1.17–3.14, $P = 0.01$) and co-detection of respiratory pathogen (TEM PCR/sputum culture/serology) RR 2.65 (CI 1.60–4.38, $P = 0.0001$), but none with age >65 RR 1.46 (95% 0.83–2.56, $P = 0.18$), flu type RR 1.59 (95% CI 0.85–2.98, $P = 0.14$), active smoking RR 1.40 (95% CI 0.79–2.47, $P = 0.24$) or vaccination RR 1.21 (95% CI 0.70–2.12, $P = 0.48$). Fatality rate was 6% which showed significant association with arrival from a facility RR 4.56 (95% CI 1.55–13.40, $P = 0.006$).

CONCLUSION

2017–2018 Influenza season among hospitalized patients involved more elderly patients and peaked in January. There was no correlation between vaccination status and causative influenza type indicating similar efficacy of vaccination for all flu types involved.

Sensitivity of flu swab was 38% calling for better utilization of TEM PCR in hospitalized patients. Severe flu had significant association with arrival from facility, lung disease and co-detection of respiratory pathogen. Severe flu did not have association with age, type of influenza, vaccination status, smoking status or treatment with oseltamivir.

Fatality had significant association with arrival from facility. Confounders were not accounted for during analysis.

*** This vignette was chosen for oral presentation on Research Day. Contact Bhavyaa Bahl at bhavyaabahl@uabmc.edu for a copy of the PowerPoint.**

Epidemiology of Extended-Spectrum Beta-Lactamase (ESBL) Producing Organism Infections in a Large Tertiary Medical Center

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Background

Extended-Spectrum Beta-Lactamase producing organism-related infections is associated with significant morbidity and mortality.

Methods

Retrospective chart review of Extended-Spectrum Beta-Lactamase producing organism related infections in a 900-bed community hospital in North Alabama between 2013-2015. Data collected include demographics, hospital vs community-onset, mortality, length of stay, resistance to fosfomycin, readmissions and comorbidities.

Results

1068 total patients were identified, 975 patients with community-onset and 93 hospital-onset; median age 65 y (1 month -101 years old). 68 % were ≥ 60 years old. 74% were female. 77.9% Caucasians and 12.5% African Americans. Urine was the most common source of hospital-onset and community-onset, 84% vs 94% respectively while bacteremia was found only in 1.9% of total patients and more common in community-onset group. 8% of *Escherichia coli* produced Extended-Spectrum Beta-Lactamases.

Among hospital-onset and community-onset Extended-Spectrum Beta-Lactamase producing organism infections, *Escherichia coli* was isolated in 91% and was susceptible to fosfomycin in 99% community-onset vs 85% hospital-onset. Resistance to Doripenem (0.19%) and Ertapenem (0.28%). *Carbapenem-Resistant Enterobacteriaceae* reported in 2 patients each year. Only 13.6% received appropriate initial antimicrobial therapy in hospital-onset infections. Average length of hospital stay was 9.7-day (1-45 days), 32% readmission rate and 4% mortality. Most common co-morbidities: hypertension 72%, diabetes mellitus 41%, chronic kidney disease 27%, obesity 19%, cancer 19% followed by congestive heart failure and cirrhosis.

Conclusion

Extended-Spectrum Beta-Lactamase producing organism related infections are increasingly reported worldwide. The data presented supports this trend. Delay in appropriate therapy add to this burden and increase hospital stay, readmission and mortality.

*** This vignette was chosen for oral presentation on Research Day. Contact Aristotle Asis at aristotleasis@uabmc.edu for a copy of the PowerPoint.**



Epidemiology of Extended-Spectrum Beta-Lactamase Producing Organism Infections in a Large Tertiary Center

UAB

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(¹)UAB School of Medicine, Huntsville campus, Huntsville, AL. (²)Huntsville Hospital, Huntsville, AL.

Background

Extended-3pectrum Beta-Lactamase producing organism-related infections is associated with significant morbidity and mortality.

Methods

Retrospective chart review of Extended-3pectrum Beta-Lactamase producing organism related infections in a 800-bed community hospital in North Alabama between 2013-2015. Data collected include demographics, hospital vs community-onset, mortality, length of stay, resistance to cefotymoxin, readmissions and comorbidities.

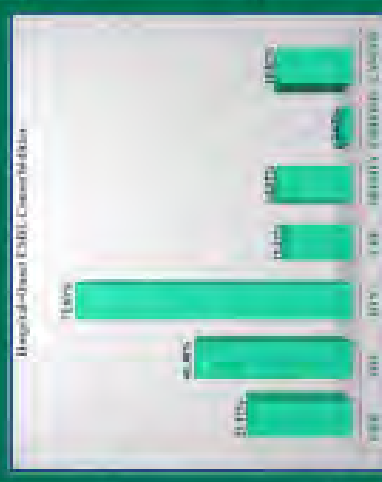
Results

1038 total patients were identified. 875 patients with community-onset and 83 hospital-onset; median age 65 y (1 month -101 years old). 88 % were ≥ 60 years old. 74% were female, 77.8% Caucasians and 12.6% African Americans. Urine was the most common source of hospital-onset and community-onset, 84% vs 84% respectively while bacteremia was found only in 1.8% of total patients and more common in community-onset group. 8% of *Escherichia coli* produced Extended-3pectrum Beta-Lactamase.

100 ESKAP Patient Age Distribution



100 ESKAP Patient Sex Distribution



Results...contd

Among hospital-onset and community-onset Extended-3pectrum Beta-Lactamase producing organism infections, *Escherichia coli* was isolated in 81% and was susceptible to cefotymoxin in 88% community-onset vs 86% hospital-onset. Resistance to Doripenem (0.18%) and Ertapenem (0.28%). Carbapenem-Resistant Enterobacteriaceae reported in 2 patients each year. Only 13.8% received appropriate initial antimicrobial therapy in hospital-onset infections. Average length of hospital stay was 8.7-day (1-46 days), 32% readmission rate and 4% mortality. Most common co-morbidities: hypertension 72%, diabetes mellitus 41%, chronic kidney disease 27%, obesity 18%, cancer 19% followed by congestive heart failure and anemia.

Conclusions

Extended-3pectrum Beta-Lactamase producing organism related infections are increasingly reported worldwide. The data presented supports this trend. Delay in appropriate therapy add to this burden and increase hospital stay, readmission and mortality.

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Epidemiology of Pneumococcal Bacteremia in a Large Tertiary Center

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Background

Streptococcus pneumoniae remains an important cause of bacteremia in the United States with high morbidity and mortality despite readily available treatment and vaccines.

Methods

Retrospective chart review of patients admitted with pneumococcal bacteremia over the last 2 winter seasons was done. Demographics, laboratory data, intensive care(ICU) stay, need for ventilation or pressor, comorbidities, and mortality were collected.

Results

53 patients enrolled. There were 66% white, 60% male, mean body mass index(BMI) of 27 (38% normal BMI). Mean age was 55 years (1-93) (57% > 61). Mean hospital stay was 7.8 days (1-30). >40% required ICU stay. Use of non-invasive positive pressure ventilation(NPPV), vasopressors and mechanical ventilation were 6%,15%, and 17% respectively. Most common presentation: dyspnea (30%) and fever (18%). 80% of patients with smoking history (55%) had pneumonia. Resistance to penicillin was 9% and intermediate susceptibility was 6%. Resistance to erythromycin (44%) and trimethoprim-sulfamethoxazole (12%) increased during winter 2017(52% and 12%) compared to winter 2016(30% and 10%). Only 2% of patients with pneumonia had positive sputum culture for pneumococcus and 62% had positive serum pneumococcal antigen with bacteremia. Positive co-detection of bacterial or viral targets in sputum using Multiplex polymerase chain reaction(PCR) did not correlate with mortality and hospital stay but they likely needed more ICU stay, use of vasopressors and mechanical ventilation. 43% of empiric therapy was as recommended by IDSA guidelines. Comparing 2016 vs 2017 seasons, mortality (15% vs 6%), hospital stay (9 days vs 7 days), use of NPPV (5% vs 6%) mechanical ventilation (15% vs 18%) and vasopressor (5% vs 21%). No correlation was found between influenza infection and bacteremia. Overall 6-month mortality and re-admission rate was 9% and 2% respectively. Mortality was higher in overweight patients (60% vs 20%), non-smokers (40% vs 20%), coronary artery disease (40%) and congestive heart failure (40%).

Conclusion

We observed less mortality and hospital stay but more use of NPPV, mechanical ventilation and vasopressor during winter 2017 which had widespread influenza-like activity and incidence of bacteremia.

*** This vignette was chosen for oral presentation on Research Day. Contact Aristotle Asis at aristotleasis@uabmc.edu for a copy of the PowerPoint.**

Factors Influencing Specialty Choice and Rural Family Medicine

Phillip Ingram, MS3, UAB Huntsville Regional Medical Campus; David Bramm, MD, Assistant Professor, Department of Family Medicine, Huntsville Regional Medical Campus; Paula Clawson, Rural Medicine Program Administrator, UAB Huntsville Regional Medical Campus; Kyle Siegrist, Systems Analyst, UAB Huntsville Regional Medical Campus

Submission category: Education innovation

Background

Entering first year medical students have a number of traits, which may serve as predictors of ultimate specialty choice. Several of these may also influence future practice location (urban or rural). This study investigated these influences in order to provide more insight on filling the need for rural family physicians.

Methods

Data was collected from a survey given to incoming first year medical students at the University of Alabama at Birmingham School of Medicine from 2003 to 2014. Responses from 970 students were then followed up using residency match data and locating their respective current practices. Analysis of various predictive factors self-reported by graduates were compared with choice of specialty and location of practice. Focused analysis was done on those practicing rural family medicine.

Results

Initial interest was found to be the strongest predictor in the fields of surgery, pediatrics, and family medicine, respectively. Commitment to service, and quality of life were significant traits found among students who selected family medicine. More rural physicians chose training in family medicine over other specialties. Among physicians working in rural communities, 80% either were born or grew up in rural areas. Initial interest in practicing rural medicine was expressed in 60% of current rural family physicians. Finances did not have a significant impact on the choice of any medical specialty. Prestige and exclusivity were also lacking a significant association with a decision of medical specialty.

Conclusions

Coming from a rural community was the strongest predictor for establishing future rural family physicians. Initial interest in living and working in a rural area was also a strong predictor of rural medicine. Traits of dedication to service and strong interest in quality of life are also significant predictors for the field of family medicine as a whole. Financial incentives or levels of prestige and exclusivity were not predictive for any choice of medical specialty.

*** This vignette was chosen for oral presentation on Research Day. Contact Phillip Ingram at pringram@uabmc.edu for a copy of the PowerPoint.**

Geographic Proximity to Healthy Food is Not Independently Associated with Ischemic Stroke Risk Factors

Alan Howard, MS3, UAB Huntsville Regional Medical Campus; Chigozirim Izeogu, MD, Neurologist; Adeniyi Idigo, MD; Michael Lyerly, UAB Department of Vascular Neurology

Category: Education Innovation

Objective: To examine the association of food desert residence (FDR), low income (LI), and low vehicle access areas (LVAA) to cardiovascular (CV) risk factors among ischemic stroke (IS) patients.

Background: Environmental/socioeconomic barriers may prevent people from obtaining diets rich in healthy foods that can reduce CV risk factors. The association between FDR and burden of CV risk factors has been previously attributed to low income rather than food access but has not been characterized well among IS patients.

Designs/Methods: We retrospectively collected data on IS patients between 10/1/2014 to 9/30/2015. Demographics, residence, and vascular risk factors were extracted. FDR, LI, and LVAA status were determined using The USDA Food Access Research Atlas. Spearman correlation and adjusted logistic regression were used to examine the relationships between CV risk factors and FDR, LI, and LVAA.

Results: We identified 353 patients (mean age 69, 45% black, 56% female, 67% Medicare, 56% FDR, 52% LI, and 40% LVAA). Black race correlated with FDR ($r = 0.16$; $p\text{-value} = 0.003$), LI ($r = 0.29$; $p\text{-value} < 0.0001$), and LVAA ($r = 0.51$ $p\text{-value} < 0.0001$). FDR was not associated with having hypertension ($OR = 0.65$, $CI = 0.32\text{--}1.3$, $p\text{-value} = 0.23$), dyslipidemia ($OR = 0.95$, $CI = 0.59\text{--}1.5$, $p\text{-value} = 0.82$), CAD ($OR = 1.1$, $CI = 0.64\text{--}1.7$, $p\text{-value} = 0.82$), diabetes ($OR = 0.76$, $CI = 0.45\text{--}1.2$, $p\text{-value} = 0.22$) or obesity ($OR = 1.1$, $CI = 0.7\text{--}1.8$, $p\text{-value} = 0.63$). LI area residency was found to be associated with hypertension ($OR = 3.2$, $CI = 1.4\text{--}7.4$, $p\text{-value} = 0.007$). FDR ($p\text{-value for interaction} = 0.05$) and LVAA ($p\text{-value for interaction} = 0.04$) were found to modify the association between LI and diabetes.

Conclusion: Among ischemic stroke patients, food access does not have an association with hypertension, diabetes, dyslipidemia, CAD, or obesity as an independent variable. However, FDR and LVAA modify the association between LI and diabetes. Further studies into FDR, LI, and LVAA should be conducted to examine this interaction in larger data sets.

*** This vignette was chosen for oral presentation on Research Day. Contact Alan Howard at adhoward@uabmc.edu for a copy of the PowerPoint.**

Geographic Proximity to Healthy Food Not Associated With Increased Prevalence of Cardiovascular Risk Factors in Stroke Patients

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Background

- Diets rich in fruits, vegetables, and low-fat dairy reduce the risk of cardiovascular (CV) risk factors.¹
- Environmental and socioeconomic barriers prevent people from obtaining these foods.²
- Previously, the association between food deserts (FD) and a higher burden of CV risk factors was attributed to low income rather than food access in the general population.³
- In this study, we examined this association in stroke patients who lived in food deserts, low income (LI) and low vehicle access (LV) areas.

Methods

Medical records of patients with discharge diagnosis codes for acute ischemic stroke (ICD 9) admitted at a tertiary center from 10/1/2014 to 9/30/2015 were reviewed.

Study Inclusion Criteria:

- Age 18+ years
- White/Black Race
- Diagnosis of acute ischemic stroke by a Neurologist or presence of restricted diffusion as evidenced by DWI and ADC correlate on MRI
- Valid Residential Street Address

Study Exclusion Criteria:

- Concurrent Intracranial Hemorrhage
- Venous Infarction
- Patients participating in clinical trials at the time of the hospitalization where they were diagnosed as stroke.

Demographics, history of cardiovascular risk factors, BMI, clinical diagnosis with stroke etiology, discharge mRS, and disposition were extracted.

The USDA Food Access Research Atlas⁴ was used to determine FD, LI, and LV status by residential street address.

Spearman correlation and adjusted logistic regression were used to examine the relationships between CV risk factors and FD, LI, and LV.

Results

Table 1. Baseline Characteristics (N= 353)

Age	Median (IQR (Q3-Q1))	Insurance	
		Medicare	Medicaid
67	23 (79-56)	67% (235)	8% (30)
Sex n(%)	Male	44% (144)	4% (16)
	Female	56% (197)	3% (9)
Race	Food Desert	Food Desert	
		Low Income	Low Vehicle
Black	45% (160)	52% (184)	40% (140)
White	55% (193)	52% (184)	40% (140)

Table 2. Odds Ratios for Cardiovascular Risk Factors by Food Desert, Low Income, and Low Vehicle (adjustments for age, race, sex, smoking status and exposure)

	Food Desert				Low Income				Low Vehicle			
	Unadjusted OR	95% Confidence Interval	P value	Adjusted OR	Unadjusted OR	95% Confidence Interval	P value	Adjusted OR	Unadjusted OR	95% Confidence Interval	P value	Adjusted OR
Hypertension	0.82	0.42-1.61	0.571	0.78	3.76	1.77-7.95	0.001	3.34	1.42	0.71-2.95	0.328	0.82
Diabetes	0.89	0.58-1.35	0.573	1.07	2.03	1.32-3.11	0.001	2.5	0.74	0.48-1.14	0.171	0.74
Hyperlipidemia	1.05	0.66-1.67	0.848	1.01	1.19	0.75-1.89	0.46	1.02	0.87	0.55-1.40	0.572	0.75
Coronary Artery Disease	1.06	0.85-1.72	0.826	1.02	0.79	0.49-1.28	0.341	0.7	0.79	0.47-1.29	0.327	0.93
Obesity	1.17	0.74-1.84	0.51	0.95	1.12	0.72-1.76	0.618	0.69	1.61	1.02-2.54	0.041	1.67

Interaction: Stratified Adjusted Logistic Regression was performed; the results demonstrated that Food desert (p-value for interaction = 0.05) and LV (p-value for interaction = 0.04) were found to be effect modifiers on the association between LI and diabetes.

Discussion

- Among ischemic stroke patients in our cohort, food access as demonstrated by geographical proximity to a healthy food source did not have an association with hypertension, diabetes, dyslipidemia, CAD, or obesity as an independent variable.
- Living in a low income area was the only independent variable that showed a statistically significant difference for hypertension and diabetes.
- Food desert and low vehicle access were found to modify the association between LI and diabetes.

Conclusion

Geographic proximity to food did not show an increased prevalence of CV risk factors in our sample. Further studies into food deserts, income, and vehicle access should be conducted to examine this interaction in a larger population of stroke patients.

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Interprofessional debate activity as a method of literature evaluation: a two-year analysis

Taylor Steuber, Assistant Clinical Professor of Pharmacy Practice, Auburn University Harrison School Of Pharmacy; Haley Phillippe, Assistant Clinical Professor of Pharmacy Practice, Auburn University Harrison School Of Pharmacy; Bradley Wright, Assistant Clinical Professor of Pharmacy Practice, Auburn University Harrison School Of Pharmacy; Miranda Andrus, Assistant Clinical Professor of Pharmacy Practice, Auburn University Harrison School Of Pharmacy

Description:

Interprofessional education and literature evaluation are emphasized in both medical and pharmacy education. Current literature studies support the idea of debates as an alternative to journal clubs in medical and pharmacy training.¹⁻⁴ However, there are no reports of interprofessional literature evaluation in the medical and pharmacy literature. The purpose of this study is to assess the impact of interprofessional debates as an alternative activity to journal club on perceived pharmacy-physician relationships and literature evaluation skills after two years of experience.

Methods:

Third year family medicine residents and fourth year pharmacy students participated together in an interprofessional debate activity. A pre-survey using the 10-item SPICE-R2 instrument (Likert scale; 1=strongly disagree through 5=strongly agree) was administered to the participants via Qualtrics at the beginning of the APPE rotation year to gauge interprofessional teamwork perceptions. Post-surveys were administered directly after the debate throughout the year and matched to assess changes in perceived pharmacist-physician relationships, and included questions to gauge perceptions of literature analysis and other skills via this method. Matched survey results were analyzed using descriptive statistics. Changes in perceptions of the SPICE-R2 instrument were analyzed using Wilcoxon Signed Rank test.

Results:

Fifty-five participants (18 medical residents and 37 pharmacy students) have participated in 9 unique interprofessional debates over a two year time period. Thirty-five participants (64%) completed both the pre- and post-surveys. Level of agreement increased on the post-survey for each item on the SPICE-R2 instrument, and was statistically significant for 7 out of 10 questions ($p < 0.05$). Overall, participants agreed or strongly agreed that the debate activity improved their literature evaluation, problem-solving, critical thinking, teamwork and communication skills.

Implications:

The debate was well received as a method of literature evaluation by both family medicine residents and pharmacy students. Participants reported positive and improved perceptions of working with an interprofessional team, as well as improved literature evaluation skills and other skills.

*****NOTE: this abstract is currently submitted to another meeting (American Association of Colleges of Pharmacy Annual Meeting) but is not yet accepted or published*****

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*** This vignette was chosen for oral presentation on Research Day. Contact Taylor Steuber at tds0038@auburn.edu for a copy of the PowerPoint.**

Interprofessional debate activity as a method of literature evaluation: a two-year analysis

Taylor Stauder^{1,2}, Haley Philpotts^{1,2}, Brad Wright^{1,2}, Miranda Andrus^{1,2}

1. Auburn University HSC; 2. Internal Medicine, UAB-Huntsville, 3. Family Medicine, UAB-Huntsville

Background

- Interprofessional education and literature evaluation are emphasized in health education
- Debates can serve as alternatives to journal clubs¹⁻⁴

Purpose

- Assess how an interprofessional debate activity has been perceived by medical residents and pharmacy students

Methods

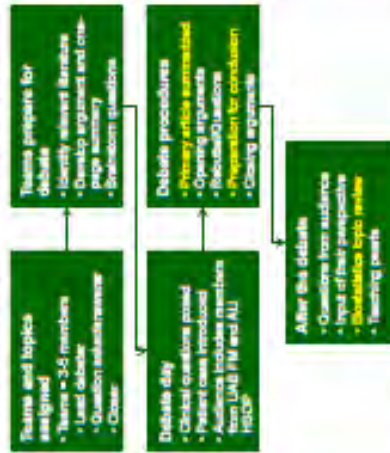
- Interprofessional debates started in August 2017
- PGY3 FM residents
- P4 pharmacy students
- Debate controversial topic in medical literature
- Pre-survey
- SPICE-R2 Instrument
- Post-survey
- SPICE-R2 Instrument
- Literature analysis
- Teamwork skills
- Matched survey results analyzed using descriptive statistics
- Changes in SPICE-R2 analyzed using Wilcoxon Signed rank test
- IRB-reviewed and exempt

Year 2 Topics

- Metoprolol XL vs Carvedilol
- Clopidogrel vs ASA/DO
- ARNI vs ACE-I
- Extended DAPT
- Evolocumab for CV event reduction

Debate Process

Year 2 topics assigned to teams for year 2 assigned in yellow



Results

- 55 participants (18 PGY3 residents, 37 P4 pharmacy students)
- 35 matched responses (64%), all areas improved with post-survey
- 9 PGY3 residents, 26 P4 pharmacy students
- Average age 27 ± 3.6 years, 19 females (54%)

	SPICE-R2 Survey Matched Data (n=38)	
	Pre-survey	Post-survey
1. Working with students from different disciplines enhances my education	5 (4.5)	5 (4.5)
2. My role within an interprofessional team is clearly defined	4 (4.5)	4 (4.5)
3. Interdisciplinary collaboration is improved when cases is delivered by an interprofessional team ¹	4 (4.5)	5 (4.5)
4. Participating in educational experiences with students from different disciplines enhances my ability to work on an interprofessional team of other health professionals ²	5 (4.5)	5 (4.5)
5. I have an understanding of the course taken by and training requirements of other health professionals ³	3 (3.0)	5 (3.4-5)
6. Healthcare costs are reduced when patients/care are treated by an interprofessional team ⁴	4 (4.5)	5 (4.5)
7. Health professional students from different disciplines should be educated to establish collaborative relationships with one another ⁵	5 (4.5)	5 (4.5)
8. I understand the value of other health professionals within an interprofessional team ⁶	4 (4.5)	4 (4.5)
9. Interdisciplinary education increases value care is delivered by an interprofessional team ⁷	4 (4.5)	5 (4.5)
10. During their education, health professional students should be involved in activities with students from different disciplines in order to understand their respective roles	4 (4.5)	5 (4.5)

SPICE-R2 survey questions and responses are available in the supplemental materials. ¹Working with students from different disciplines enhances my education. ²My role within an interprofessional team is clearly defined. ³I have an understanding of the course taken by and training requirements of other health professionals. ⁴Healthcare costs are reduced when patients/care are treated by an interprofessional team. ⁵Health professional students from different disciplines should be educated to establish collaborative relationships with one another. ⁶I understand the value of other health professionals within an interprofessional team. ⁷Interdisciplinary education increases value care is delivered by an interprofessional team.

Discussion

- Debate was well received by both parties
- Positive perceptions of working on interprofessional teams
- Also reported improved literature evaluation, problem-solving, critical thinking, and teamwork skills as a result of the process
- Some participants (n=8) disagree or strongly disagreed that they prefer the debate to a journal club
- Potentially due to more rigorous time commitment and preparation compared to a traditional journal club
- Debates provide an engaging platform and are effective alternatives to traditional journal clubs

Future Directions

- Quality improvement measure employed to enhance experience
- Involve more PGY2 residents and enhance audience involvement

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We will change your world

Using Vision Screenings to Educate Communities About Health

Meredith Lewis, LICSW, Behaviorist, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Amanda Stisher, MS4, UAB Huntsville Regional Medical Campus; Shivani Malhotra, MD, Assistant Professor, Department of Family Medicine, UAB Huntsville Regional Medical Campus; Bal Mainali, MS, Alabama A&M University, MSW Intern

Description

Stimulating collaboration with community organizations brings a partnership for scholarly activities in graduate medical education. One way to educate the community about health-related quality of life is through vision screenings. Many studies have shown that visual impairment can have a significant impact on keeping people healthy. Vision impairment has been associated with an increased mortality, increased risk of falls, a decline in performing activities of daily living (ADLs), decrease normal development in young children, and reasons why children struggle to develop socially.

Methods

To promote health initiatives, a total of three events in the community, two events for adults and one event for children, were performed. The Snellen acuity test, the gold standard for visual acuity, was used during vision screenings. The standard way to perform a vision screening is as follows: a) person stands 20 feet away from the Snellen eye chart, b) person covers their eyes one at a time and read each letter/object from the Snellen eye chart aloud, and c) person's visual acuity is determined based on their ability to read the chart. For example, 20/25 or 20/30.

Results

Data was recorded at the adult events. The mean age was 55.4. The average left eye score was 20/37 and the average right eye was 20/35. If vision impairment was detected, the adult population received resources from Eyesight Foundation of Alabama and New Eyes for follow-up.

In contrast, at the children's event, age range from 5 years to 13 years old. When performing the vision screenings for children, staff must identify which vision screening board is better for the children: pictures, shapes or letters.

Discussion

When utilizing the Snellen eye chart, patient should be positioned 20 feet from the eye chart. If a patient already wears glasses, it is important to leave them on unless they are only used for reading. The patient should cover one eye and read the smallest line on the eye chart possible. If more than half of the letters on that line are read correctly, then the visual acuity noted on the side of that line should be recorded and the opposite eye should be tested. If less than half of the letters are read correctly, then the patient should proceed to reading the line or lines above until half of the letters are read correctly.

Visual acuity is expressed as a fraction: the top number is the distance from the chart and the bottom number is the distance at which a normal eye can read the specified line of letters. For example, a vision of 20/30 means that, a person with normal vision can see the line of letters at 30 feet, but the patient being tested must be at 20 feet to read the same line of letters; therefore, a higher bottom numbers means worse vision. If a patient is wearing glasses during the exam and scores a 20/40, then results should be written as "20/40 corrected," meaning he or she could read the 20/40 line with glasses.

*** This vignette was chosen for oral presentation on Research Day. Contact Meredith Lewis at meredithlewis@uabmc.edu for a copy of the PowerPoint.**

Description

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Methods

To promote health initiatives, a total of three events in the community, two events for adults and one event for children, were performed. The Snellen acuity test, the gold standard for visual acuity, was used during vision screenings. The standard way to perform a vision screening is as follows: a) person stands 20 feet away from the Snellen eye chart, b) person covers their eyes one at a time and read each letter/object from the Snellen eye chart aloud, and c) person's visual acuity is determined based on their ability to read the chart. For example, 20/25 or 20/30.

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Watch Your Children Grow, Learning How to Address Obesity in Underserved Populations

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Submission Category: Research-Education Innovation

Obesity continues to be a health issue. In the US, four states including Alabama have rates over 35%. Obesity leads to diabetes, hypertension, and hyperlipidemia, which occur in younger children. A combination of poor eating habits, lack of nutritional education, social determinants, economic disparities, food insecurity, and lack of exercise are significant factors that precipitate this problem. These factors include *habits* that can be easily transmitted from parents to children.

Caloric Titration Method (CTM) developed by Cornell University researchers helps young/old adults to prevent age-related weight gain. CTM entails daily self-weighing as the principal method of controlling weight by establishing healthy habits. It relies on awareness of the effects of eating and exercise on changes in body weight to guide behavior.

A pilot study examined whether a modified version of the CTM for children could prevent weight gain in overweight and obese children, with the aim to grow into their weight. The study tested whether preadolescents would comply with *daily scale weighing*. The study had low attrition and after 6 months, the children with scales gained less weight than the controls and grew the same in height. The present project follows a similar protocol and includes reorganizing the home food environment.

Methods: A six-month in-home biofeedback program called Watch Your Children Grow was introduced to families, predominantly underserved, with at least one child with a BMI over 85th percentile (May 2016 - November 2017). The intervention consisted of bi-monthly home visits. At each visit fruits and vegetables were provided to the families and placed in a focal position in the eating areas. All family members weighed themselves daily on a Wi-Fi scale and received weekly mailings of their graph and maintenance goal. The study aimed to evaluate whether the home food environment can be assessed and custom-designed by Home Staging the eating areas, counters, refrigerator and cabinets based on specified criteria that are conducive to modifying behavior triggers. The changes in the home environment combined with the CTM Scale Biofeedback were provided to treatment participant families, and no information was provided to the control group.

Results & Conclusion: The treatment group included cohort 1 (28 participants, May 2016 - November 2016) and cohort 2 (40 participants, December 2016- May 2017). In the combined treatment groups the average net change in BMI percentile was -1.0 and average net BMI SDS change was -0.144. For the control group, 113 patients with similar qualification criteria are being retroactively reviewed.

Participants were more willing to engage in the research study due to the research team going into the home. Barriers to keeping participants engaged included Wi-Fi access for scale and ensuring community donations for food and other incentives for the intervention. Long-term impact needs to be studied

further in regards to individuals being able to sustain standing on the scale without external motivators such as the research team.

*** This vignette was chosen for oral presentation on Research Day. Contact Meredith Lewis at meredithlewis@uabmc.edu for a copy of the PowerPoint.**

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