Of Mice and Man
Hantavirus Pulmonary Syndrome in Central Indiana
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Introduction
Hantavirus pulmonary syndrome (HPS) in the US is rare, with 639 reported cases nationwide and only three in Indiana (since 1993). The primary reservoir for the virus is wild rodents with transmission via inhalation of urine, feces, or saliva. There is no proven antiviral therapy, and treatment is typically supportive. Survival increases markedly with support of cardiopulmonary function. Without treatment, mortality can reach up to 40%. We present a case of HPS treated at our hospital.

Our Case
A 19-year-old male college student with no significant medical history presented to an outside hospital ER with one week of worsening, nonproductive cough, shortness of breath, and fever of 102°F. The chest X-ray revealed diffuse bilateral infiltrates consistent with acute pulmonary edema (see right). He reported being a wildlife major. This involved exposure to field mice by catching wild rodents for analysis of blood and urine. Laboratory studies, including hantavirus antibody titers, were drawn at outside hospital. Due to exposure to field mice and non-specific viral prodrome, laboratory studies, including hantavirus antibody titers, were drawn at outside hospital. Due to concerns of hantavirus pulmonary syndrome, he was transferred to our facility for further care.

At our initial evaluation, the patient was in respiratory distress, complained of body aches and weakness. Initial vitals included a temperature of 98.6°F, heart rate of 88, respiratory rate of 43, and a 95% oxygen saturation on 5 L nasal cannula.

Physical Exam:
General: ill-appearing, in moderate distress
Chest: rales in bilateral lung fields
Cardiac: normal S1/S2, no M/R/G, RRR
Abdomen: soft, NT/ND, no organomegaly
Extremities: no edema
Neurologic: AAO x 3; no gross motor or sensory findings
Sympathetic: normal and he was comfortably breathing room air.

Clinical Course
The chest radiograph obtained at our facility showed bilateral pulmonary infiltrates (Figure 2). He was started on ceftriaxone and doxycycline for empiric pneumonia coverage. An Infectious Disease consultation investigated other infectious etiologies such as leptospirosis and viral infections and found them to be negative. The patient responded well to supportive care, with resolving leukocytosis and decreasing oxygen requirements. Follow-up serologies revealed positive IgM and IgG antibodies for hantavirus. At the time of discharge, his vital signs were normal and he was comfortably breathing room air. He was discharged home with Primary Care and Infectious Disease follow-up.

Laboratory Data and Imaging
Differential Showed 24% bands and reactive lymphocytes

<table>
<thead>
<tr>
<th>WBC</th>
<th>RBC</th>
<th>Platelets</th>
<th>Hemoglobin</th>
<th>Hematocrit</th>
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<td>10.9</td>
<td>59</td>
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<td>43.9</td>
<td>101</td>
<td>0.87</td>
<td>400</td>
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</table>

LDH: 400
AST/ALT: 54/21
Alkaline Phosphatase: 30
Total Bilirubin: 0.0

Take Home Points
- Clinical suspicion and appropriate history are key in diagnosing hantavirus pulmonary syndrome.
- Suspect hantavirus pulmonary syndrome in patients with a history of rodent exposure and non-specific viral prodrome, followed by acute worsening cardiorespiratory symptoms, with pulmonary edema on imaging.
- Proper diagnosis is critical considering the cardiopulmonary phase of the illness can lead to rapid deterioration and death.
- Treatment is focused on cardiopulmonary support.

References

Figure 1: Prevalence of Hantavirus cases in the United States

Figure 2: Chest radiograph showing bilateral pulmonary edema and small right apical iatrogenic pneumothorax.

Figure 3: Whitefoot mouse (left) and deer mouse (right) are the primary reservoirs for hantavirus in the United States.

Discussion
Hantavirus pulmonary syndrome in the US typically occurs in three stages. The first phase is the incubation phase typically lasting two to three weeks post-exposure. This is followed by the febrile phase which lasts two to eight days and consists of fever, chills, nausea, vomiting, weakness, and myalgias. The final phase is the cardiopulmonary phase. This phase consists of capillary leak into pulmonary beds and can rapidly progress to shock, coagulopathy, pulmonary edema, arrhythmias, and death. Referral to tertiary care center at the earliest stage is essential should critical care become necessary. Workup generally includes IgM and IgG antiviral serologies, basic laboratory studies, and chest imaging.

This case illustrates the importance of early recognition of rare zoonotic diseases based on history. Assessment of risks factors can lead to prompt laboratory testing for diagnosis and appropriate early transfer to a tertiary referral center. Although our patient did not decompensate, patients with Hantavirus cardiopulmonary syndrome can deteriorate over hours, requiring mechanical ventilation, ARDS protocols, and possible ECMO.
Leuconostoc Bacteremia: A Rare Cause of Gram Positive Bacteremia

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INTRODUCTION

Gram positive bacteremia is commonly seen in the inpatient setting. Vancomycin is often used as empiric therapy until organisms are identified. The following case describes a rare infection initially misdiagnosed as a viridans streptococcal bacteremia that was found to be identified as a vancomycin resistant Leuconostoc spp. bacteremia, an infection most commonly found in immunosuppressed patients, as well as those with central venous catheters and underlying pathology of the gastrointestinal (GI) tract.

CASE REPORT

A 35-year-old female with PMH of iatrogenic small bowel perforation complicated by peritonitis and multiple surgeries for reconstruction resulting in short bowel syndrome presented with sepsis from an outside hospital with complaints of fever, chills and general malaise. She had a low grade fever and tachycardia on admission with physical examination notable for a soft, non-distended, mildly tender abdomen with multiple surgical scars, ileostomy and J-tube.

She had a central venous catheter located in her left upper chest with the surrounding area appearing clean, dry and intact. The initial workup at OSH revealed blood cultures drawn from peripheral and central lines positive for viridans streptococci (sensitivities pending at transfer).

As was the case in this patient, the organism is often initially misidentified as a viridans streptococcus given its similar appearance. With vancomycin continuing to be commonly used for empiric treatment for gram positive bacteremia, it is important for physicians to be aware of vancomycin-resistant Leuconostoc spp particularly in this specific patient population.

Table 1. Microbiological characteristics of Leuconostoc spp

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram staining (Blood agar)</td>
<td>Gram positive coagulase-negative Staphylococcus aureus (CoNS)</td>
</tr>
<tr>
<td>Gram staining (Thioglycollate-broth)</td>
<td>Gram positive coagulase-negative Staphylococcus aureus (CoNS)</td>
</tr>
<tr>
<td>Catalase</td>
<td>Negative</td>
</tr>
<tr>
<td>Leucon arylamidase</td>
<td>Negative</td>
</tr>
<tr>
<td>Arginine dihydrolase</td>
<td>Negative</td>
</tr>
<tr>
<td>Pyridoxal phosphate</td>
<td>Negative</td>
</tr>
<tr>
<td>CO2 production from glucose in MRS broth</td>
<td>Positive</td>
</tr>
<tr>
<td>Bile esculin</td>
<td>Variable</td>
</tr>
</tbody>
</table>


REFERENCES


Atypical Hemolytic Uremic Syndrome: The Rarer and Deadlier Cousin of Shiga toxin-producing E. coli Hemolytic Uremic Syndrome

Sarah M. Jeong, MD, William B. Fisher, MD, Rodney Yuhico, MD
Indiana University Health Ball Memorial Hospital, Internal Medicine Residency, Muncie, IN

Introduction

- Atypical hemolytic uremic syndrome (aHUS) comprises about 10% of HUS cases, with an annual incidence of 2 per million. It has a more severe disease course than Shiga toxin-producing E. coli (STEC-HUS), with a mortality rate as high as 25% and with 50% - 80% of patients progressing to end-stage renal disease.

- aHUS, STEC-HUS, and thrombotic thrombocytopenic purpura (TPP) are the three main thrombotic microangiopathies (TMAs) and can be virtually impossible to clinically distinguish upon presentation.

- aHUS and STEC-HUS have differing pathophysiologies and therefore have differing disease courses and treatments. Rather than being induced by Shiga toxin-producing bacteria, aHUS is a genetic disorder that results from uncontrolled activation of the alternative complement system.

- For this reason, both HUS variants must not be erroneously grouped together on a TMA differential in order to facilitate swift recognition, diagnosis, and treatment of aHUS.

Differentiating among the TMAs

A 28-year-old male with no prior medical history presents with dyspnea and hemoptysis.

One week prior, he experienced 1-2 days of non-bloody diarrhea and vomiting with spontaneous resolution of symptoms after eating an undercooked hamburger. Surrounding this event, he developed progressive dyspnea on exertion as well as paroxysmal nocturnal dyspnea. The day prior to admission, he developed worsening cough productive of 500mL of frank hemoptysis. He denied associated fevers, chills, abdominal pain and dysuria, but did note increasing nocturia and peripheral edema.

Vitals on presentation: T 36.7°C   BP 207/133   HR 92   RR 18   O2 Sat 97%

Physical exam revealed jugular venous distension, bibasilar lung crackles, and 3+ pitting edema to the level of the thighs. No rash or skin discoloration was noted.

CT Chest without contrast revealed diffuse bilateral ground glass and nodular densities, as well as bilateral pleural effusions, a small pericardial effusion, and paratracheal and mediastinal lymphadenopathy. Blood and sputum cultures were negative, as well as fungal and tuberculosis workup.

Echocardiogram showed diastolic dysfunction grade III, mild concentric left ventricular hypertrophy, and moderate to severe mitral regurgitation, with ejection fraction greater than 55%

Evidence for non-immune microangiopathic anemia

- Hb 10.7 g/dL
- LDH 550 units/mL
- Hemoglobin < 6mg/dL
- Peripheral Smear Sischoctyes (Figure 1)
- Direct Coombs Neg
- Indirect Coombs Neg

Evidence for thrombotic microangiopathy

- Platelet 118 k/cumm
- Creatinine 5.60 mg/dL
- Kidney Biopsy Acute on chronic thrombotic microangiopathy with multiple fibrin thrombi (Figure 2)

Discussion

- Diarrhea is no longer a relevant means to separate typical and atypical HUS since both alike can present with the symptom. In fact, according to a European cohort study, diarrhea was the presenting symptom in 24% of aHUS cases, followed by respiratory infection at 18%. Other conditions known to precipitate aHUS include surgery, trauma, cocaine abuse, and pregnancy.

- Once labs indicate a TMA in a patient, plasmapheresis should be administered until TPP can be ruled out via ADAMTS13 level. If it is > 5%, plasmapheresis can be stopped, since it has no proven role in aHUS. If the patient is diagnosed with STEC-HUS, the treatment is largely supportive until the infection clears, while aHUS requires lifelong complement inhibition.

- Eculizumab (Soliris®) was approved by the FDA in 2011 for the management of aHUS. It is a humanized recombinant immunoglobulin against complement factor C5 and inhibits the generation of the cytotoxic membrane attack complex, C5b-9.

- Despite being a genetic disease, a mutation does not need to be confirmed for diagnosis of aHUS. Complement factor H mutations are the most common, occurring in 23% of cases, however, 35-40% of patients with a clinical scenario consistent with aHUS will have no demonstrable genetic mutation using current screening strategies. Complement levels are also unreliable diagnostic measures of aHUS, however low levels can contribute to the diagnosis.

- It is still unclear why our patient presented with hemoptysis, however literature review shows that the lungs are often involved in the late stages of aHUS, with some patients even requiring ventilator support.

- Patients with aHUS either present at end-stage renal disease (ESRD), or progress to ESRD due to inadequate response to therapy or delay in diagnosis. Those who require renal transplant should do so with concomitant treatment with eculizumab due to the high rate of recurrent disease without specific therapy. In theory, for complete cure of aHUS, a liver transplant is required because CFH, CFI, CFB, and C3 are all synthesized in the liver.

References

INTRODUCTION
60-70% of cases of hemoptysis are caused by infection, with as many as 23% of cases attributable to a primary lung cancer. Among all lung cancers, only 5% occur in patients less than 40 years old. Thus, when a young person presents with hemoptysis, it will rarely be secondary to malignancy.

CASE
A 30 year old lady presented to the Emergency Department for an initial episode of acute massive hemoptysis, producing approximately two cups of blood.

- **ROS**: Chronic, nonproductive cough , otherwise negative
- **PMH**: right sided pleurisy for one year
- **Surgeries**: appendectomy, C-section
- **Medications**: none
- **Family Hx**: negative for lung cancer
- **Social Hx**: nonsmoker, no alcohol, works in factory with exposure to fiberglass dust
- **Vitals**: 98 54 120/60 18 97% on RA
- **Exam**: Rhonchi at the right base

LABORATORY DATA

<table>
<thead>
<tr>
<th>Calcium 9.3</th>
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<th>INR 1.02</th>
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<tbody>
<tr>
<td>9 14 280</td>
<td>3.6 27 0.7</td>
<td></td>
</tr>
</tbody>
</table>

- Calcium 9.3
- CRP 19
- INR 1.02
- Bronchial washing culture: No yeast or fungal elements after 4 weeks

IMAGING

**Figure 1. CTA showed right lower lobe linear density and possible bronchiectatic airway**

**Figure 2. Bronchoscopy showed a shiny, white mass rich with vessels originating from the medial anterior wall of the right lower lobe airway, completely obstructing the basal right lower lobe**

Initial biopsy was withheld due to visible vascularity of the mass and significant bleeding upon touching the mass with the biopsy needle.

**Figure 3. Pulmonary function testing showed normal lung volumes, normal airway resistance, normal DLCO**

**Figure 4. PET-CT showed activity in the mass with nonspecific activity in the right hilum and subcarinal area.**

**Figure 5. Pathology report described a 1.7cm low grade endobronchial carcinoid tumor with 3 negative nodes**

DISCUSSION

- Massive hemoptysis in a young female nonsmoker is characteristic of carcinoid
- Global incidence of 2/100,000
- Most common primary lung malignancy in pediatric patients
- Delays lead to diagnosis in adolescence
- Comprise 1-2% of all pulmonary neoplasms
- Mean age of presentation is 45 years
- Gastrointestinal system: 80% carcinoids
- Lung is 2nd most common site
- Arise in lower lobe bifurcations, visible by bronchoscopy
- Biopsy despite tendency to bleed
- Typically low grade, favorable prognosis
- Hemothysis occurs in 50% patients
- More frequent presenting symptoms:
  - Wheezing
  - Atelectasis
  - Pneumonia
- Unique case:
  - Atypical presentation
  - Rare malignancy
  - Internists may encounter these cases
  - Confirm diagnosis with bronchoscopy and biopsy

REFERENCES


Introduction

Metformin is an effective and commonly used medication for the management of type 2 diabetes. While typically well-tolerated, it has one rare but potentially fatal adverse effect: metformin-associated lactic acidosis (MALA). This is distinct from metformin-induced lactic acidosis, which cannot be exclusively and specifically explained by metformin overdose.

• Normal lactate concentrations are <2 mmol/L. In patients taking metformin, levels usually fall within this reference range [1]. However, in MALA, lactate concentrations have exceeded 20 mmol/L.

• The incidence of MALA is quite low. One review found only 5.1 cases per 100,000 person-years of exposure [2]. Another systematic review of 347 cohort studies and randomized trials estimated the incidence of MALA at 1.1 cases per 100,000 person-years of exposure [3].

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Applications for Patient Care

• Although this is a very rare condition, mortality is approximately 45% [3] and patients should be made aware of this potential complication when starting metformin therapy.

• This case illustrates the importance of patient education regarding the side effects of diabetes medications, and specific instructions for when they are sick or unable to eat.

• Patients should be counseled to hold metformin for nausea and vomiting, decreased oral intake or severe acute illness.

• Patients should hold metformin prior to receiving iodinated contrast for CT scans, which can cause a temporary decrease in renal function.

Case

• A 65-year-old male with a history of type 2 diabetes mellitus and hypertension presented with five days of nausea and vomiting without hematemesis, along with abdominal and back pain, and minimal oral intake of food or fluids. He had no known history of kidney problems, but had not urinated for approximately 18 hours prior to presentation. He had no fever or chills, shortness of breath, cough, chest pain, extremity swelling or joint pain. Throughout the duration of his illness he had continued taking his prescribed lisinopril, metformin and levothyroxine.

• On arrival, his vital signs were as follows: temp 90.5°F, heart rate 67, blood pressure 114/56, respiratory rate 25 and oxygen saturation 98% on room air. Physical exam revealed only mild diffuse abdominal tenderness, with no rebound or guarding. Lab work on admission as listed in table below. Urinalysis revealed trace occult blood, 100 mg/dL protein, trace leukocyte esterase, 5-10 epithelial cells per HPF, slight mucus, 0-5 coarse granular casts per LPF, 0-5 hyaline casts per LPF, amorphous crystals and trace urine ketones. Chem radiographs and retroperitoneal ultrasounds were negative for acute processes.

• In the ER, the patient was given 100 mEq of sodium bicarbonate and started on a sodium bicarbonate drip at 150 mL/hr. The patient was admitted to the ICU, and his home metformin and lisinopril were held due to his acute renal failure. He underwent emergent hemodialysis later that day. While he did require pressors for hemodynamic instability, he never required intubation despite a pH of 6.8. After one session of hemodialysis, his renal function began to improve and eventually normalized.

• The patient was able to increase his oral intake and his IV fluids were stopped. He was transferred to the floor and then discharged home. He was instructed to stop taking metformin and to follow up with his primary care provider to establish a new regimen for management of his diabetes.

Mechanism of Action

The major effect of metformin is to inhibit gluconeogenesis and decrease hepatic output of glucose. Metformin inhibits glyceroiphosphatase, which plays a role in the gluconeogenic pathway. Without glyceroiphosphatase function, glycogen cannot be added to the pathway. It also leads to a decrease in lactate conversion to pyruvate. The excess glucose and lactate are released into the bloodstream. [4]

Discussion

• Metformin associated lactic acidosis is most commonly seen with comorbid conditions such as renal insufficiency, heart failure, hypoxic states or acute illness. In this patient, who had been taking metformin for many years without developing lactic acidosis, it is likely that his bout of gastroenteritis caused this acute problem. Multiple episodes of emesis along with decreased oral intake caused dehydration, which led to acute renal insufficiency that impaired his ability to metabolize metformin and likely triggered his MALA.

• Hemodialysis is only recommended for patients with severe metabolic acidosis, with a pH <7.1. This patient’s pH on admission was 6.8. He underwent one round of emergency dialysis, and his kidney function improved with IV fluids and improved oral intake.

Labs

<table>
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<tr>
<th>LABS</th>
<th>ADMISSION 07/9/15 15:02</th>
<th>POST IVF 07/10/15 8:10</th>
<th>POST EMERGENT DIALYSIS 07/10/15 16:15</th>
<th>DAY OF DISCHARGE 07/14/15 2:40</th>
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<td>SODIUM</td>
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<td>138</td>
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<td>POTASSIUM</td>
<td>8.5</td>
<td>4.2</td>
<td>4.3</td>
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<td>BICHLORIDE</td>
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<td>104</td>
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<td>13</td>
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<td>BUN</td>
<td>18</td>
<td>22</td>
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<td>22</td>
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<td>9.7</td>
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<td>17.7</td>
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<td>MAGNESIUM</td>
<td>2.8</td>
<td>LACTIC ACID POC (IN ED)</td>
<td>18.5</td>
<td>18.5</td>
</tr>
<tr>
<td>SODIUM</td>
<td>8.5</td>
<td></td>
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</tbody>
</table>
“I eat and then I pass out.”

Kathleen Morris, DO, Cynthia Misumi, MD, Areeba Kara, MD, MS, FACP
Indiana University School of Medicine, Indianapolis, Indiana

Learning Objectives
- List the clinical features of deglutition syncope.
- Describe the appropriate initial work up for deglutition syncope.
- Discuss the pathophysiology of deglutition syncope.

Patient Presentation
29-year old Burmese male was evaluated for syncope and weight loss.
- Daily episodes of loss of consciousness lasting 5-10 seconds
- Frequently associated with eating
  - Unable to relate to specific type of food
- Had monthly episodes between ages 6 and 15 which had abated until recent resurgence
- Developed significant anxiety related to food consumption and anorexia
  - Led to weight loss (20 lbs over past 3 months)
- No significant past medical history
- No family history of syncope
- Moved to US one month prior to presentation

Physical Exam
- VS: Tm 98.8. BP 110/70. RR 16.
- Thin appearing Burmese male in no apparent distress.
- Unremarkable exam including cardiac exam except for bradycardia.

Laboratory Evaluation
- Unremarkable labs including thyroid stimulating hormone that was within normal limits.
- EKG with sinus bradycardia (HR 47) but otherwise within normal limits.

Evaluation and Diagnosis
Outpatient Course
- Underwent echocardiogram that was within normal limits including normal septal wall thickness and normal left ventricular ejection fraction.
- Event monitoring showed transient high degree AV block with numerous pauses lasting up to 6 seconds over one week that were associated with deglutition.
- Rapidly evaluated by cardiology and assessed to have deglutition syncope due to patient history and event monitor episodes felt related to eating.
- Underwent placement of dual chamber pacemaker within 3 weeks with resolution of symptoms (3 months post-placement).

Introduction
- ‘Swallow’ or ‘deglutition’ syncope is a rare, but important syndrome as it can be lethal if not recognized.
- Although its pathophysiology is not well understood, it is thought to be vagally mediated.
  - It is hypothesized by Mitra et al that mechanoreceptors in the esophagus are activated when stretched, sending signals through the esophageal plexus to the vagus nerve.
  - The efferent impulses from the right vagus nerve reach the sinoatrial node while those from the left vagus nerve reach the AV node.
  - This may lead to bradyarrhythmias through either the SA or AV nodes that cause temporary reduction in cardiac output and result in cerebral hypoperfusion causing syncope.

Diagnosis
- The workup starts with careful history taking to establish the temporal relationship between swallowing and syncope.
- Provocative testing with various liquid and solid foods can be attempted.
- Esophageal and cardiac structural and functional abnormalities must be investigated and addressed if found.

Table 1. Case review of etiology.

<table>
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<tr>
<th>Underlying Disease</th>
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<th>Cardiac Disease</th>
<th>Other</th>
<th>None or Unknown</th>
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<tr>
<td>Number of cases</td>
<td>31</td>
<td>12</td>
<td>6</td>
<td>31</td>
</tr>
<tr>
<td>Percentage of total</td>
<td>38.75</td>
<td>15</td>
<td>7.50</td>
<td>38.75</td>
</tr>
</tbody>
</table>

Figure 1. Advanced Heart Block.

Clinal Features
- Deglutition syncope appears to be more common in males
- It has typically been described in those with underlying cardiac or esophageal disease.
- The severity of symptoms may be affected by the nature of ingested foods with some reports associating syncope to the consumption of carbonated, cold or hot beverages.

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<table>
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- The efferent impulses from the right vagus nerve reach the sinoatrial node while those from the left vagus nerve reach the AV node.
- This may lead to bradyarrhythmias through either the SA or AV nodes that cause temporary reduction in cardiac output and result in cerebral hypoperfusion causing syncope.

Treatment
- Lack of randomized control trials to guide treatment
- No comparison of efficacy of various treatments
- Avoidance of triggers and lifestyle modifications
- Discontinuation of medications with AV-blockade
- Anti-cholinergic medications may have a role in management
- Pacemaker placement has been successful in many of the recently reported cases.

TAKE HOME POINTS
- Swallow syncope is rare, but treatable and thus early recognition is necessary.
- Diagnosis requires the recognition of a temporal relationship between deglutition and syncope.
- Although there is a lack of RCTs, pacemaker placement has been successful in numerous case reports.
- The phenomenon of deglutition syncope needs to be further investigated particularly in those without structural heart disease.

References
Background

- The Adult Committee on Immunization Practices (ACIP) recommends that patients with chronic liver disease and cirrhosis (CLD) should be vaccinated against Hepatitis A and B, when applicable, to reduce additional liver-related morbidity and mortality.[1]
- Anecdotal observations suggested that the patients in the St Vincent Internal Medicine Residency outpatient continuity clinic were not receiving these vaccinations.
- It was hypothesized that vaccination rates in this clinic were low, with multiple possible causative factors.

Objectives

- To describe baseline hepatitis A and B immunization rates in clinic patients with a diagnosis of CLD and cirrhosis.
- To identify and prioritize root causes for low adherence to immunization recommendations.
- To implement counter-measures to address most important root causes to improve immunization rates.

Methods

- An interdisciplinary team comprised of physicians, nurses, and pharmacists was convened to address the quality improvement project.
- A3 quality improvement methodology was used to describe the current state and identify and prioritize root causes.
- Data collected includes the following:
  - Patient demographics
  - Primary service provider and qualifying diagnosis
  - Immunization status
  - Documentation of an offer to vaccination
  - Number of doses administered
  - Type of vaccine administered
- Adherence to immunization recommendations was defined as completing 3 doses of hepatitis B and 2 doses of hepatitis A vaccine or completing 3 doses of the combination vaccination in the appropriate time frame.
- This project was evaluated by the St Vincent Institutional Review Board and determined to be exempt.

Preliminary Results

- At baseline, 1 of 36 eligible patients were up-to-date with hepatitis A and B vaccinations.
- One month after notification letters were sent to patients, 9 of 36 patients had completed or initiated the vaccination series.
- Identified some confusion with use of hepatitis A/hepatitis B combination vaccination versus single vaccination products.

Limitations

- Short follow-up time frame does not demonstrate sustainability of results.
- Unclear if this process could be applied to other primary care settings
- All identified patients have not been evaluated at this time

Conclusions

A3 systemic problem solving was used to conduct process mapping and root cause analysis. This identified high-yield points for intervention and barriers to implementation of the ACIP recommendations as well as clinic insufficiencies. Counter-measures were devised to address these root causes. Initial results are encouraging. We will continue to approach these issues over the next several months through a multidisciplinary team involving nursing, residents, and attending physicians.

References

2) Prevention of Hepatitis A through active or passive immunization: Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 1999; 48:1
Doege-Potter Syndrome: When a Full Night’s Sleep is Deadly

Richard Nicolay, BS; Ryan Nolen, MD; Cassidy Menard, MD
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Introduction

Doege-Potter Syndrome (DPS) is a rare paraneoplastic syndrome characterized by a fibrous pleural tumor producing hypoglycemia through the ectopic production of high-molecular-weight IGF-II. We describe the medical management of a patient with DPS in the setting of a non-resectable tumor.

Case

Our patient is a 77-year-old female with a past medical history of a fibrotic pleural mass, who presented after 4 months of recurrent hypoglycemia.

During this time, she was consuming an 8-ounce Boost supplement every 3 hours through the night to maintain a morning blood glucose of 60-70 mg/dL.

On the morning of her presentation, she was found down, disoriented and hallucinating with a blood glucose of 24 mg/dL.

Physical Exam

Abdomen: Liver palpable 16 cm below the costal margin

Right Chest: Gross dorsal protrusion Dull to percussion Absent breath sounds

Laboratory Data

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Imaging

CT: Solitary, well-circumscribed 15 x 16 x 26 cm pleural mass (which approximately doubled in size since a PET CT performed 14 months prior)

Normal liver displaced inferiorly by pleural mass

Diagnosis

Previous biopsies indicated that the mass was a benign fibrotic tumor. Given her refractory hypoglycemia and pleural fibrotic lung mass, a clinical diagnosis of DPS was made.

Although an elevated IGF-II/IGF-I ratio suggests DPS, IGF-II was not measured because this expensive test would not have changed our approach to her treatment.

Treatment

The tumor was not resectable due to the patient’s age, tumor size, impaired lung function and extensive vascular involvement.

Symptomatic hyperglycemia persisted even after initiation of 150 mg of diazoxide daily and a 10% dextrose infusion. Her medications were optimized and dextrose weaned, and finally her blood glucose was controlled on a regimen of 40 mg of prednisone and 300 mg of diazoxide daily.

To prevent early morning hypoglycemia, a percutaneous endoscopic gastrostomy tube was placed for continuous nighttime feeds. On day 12, the patient was stable and discharged home.

References

Acute Onset Aphasia and a Ring Enhancing Lesion: a Unique Diagnostic Challenge

Jacob Reisner, DO; Laurel Fick, MD, FACP; Mark Janicki, MD
Department of Internal Medicine St. Vincent Hospital, Indianapolis, IN

INTRODUCTION

- Tumefactive multiple sclerosis (TMS) is a rare form of multiple sclerosis (MS) seen in 1-2,000 cases of MS or 3 cases per million per year in the general population.1,2,3,7,8,9
- TMS presents with lesions larger than traditional MS (>2cm) that may resemble intracranial neoplasm, infection, vasculitis or infarction.2,3,4
- Lesions are often ring enhancing and may open towards the cortex.2,3
- Lesions traditionally do not display mass effect on imaging.5,5
- Clinical presentation of TMS may mimic traditional MS; however, symptoms vary depending upon the location of the lesion.1
- Most commonly motor, cognitive, and sensory deficits have been described.1
- Most lesions respond to intravenous corticosteroids; however, refractory lesions may respond to plasma exchange (PLEX) therapy.2,3,7,8

CASE DESCRIPTION

- A 40 year old Caucasian male presented with new onset expressive aphasia and stated that he was “unable to get his words out.”
- Review of systems revealed a patch of burning across his upper back.
- A 40 year old Caucasian male presented with new onset expressive aphasia and stated that he was “unable to get his words out.”

TREATMENT COURSE

- The patient was treated with dexamethasone 40mg/day for 5 days without taper.
- He improved with steroids and was followed outpatient.
- Serology studies did not reveal vasculitis or infectious etiology.
- The patient was diagnosed with TMS based on MRI evidence of lesions disseminated in space and time.5
- Was placed on teriflunomide disease modifying therapy.
- At last follow up, his lesions had decreased in size, aphasia had significantly improved, and he had not experienced any more acute attacks.

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CONCLUSIONS

- The differential diagnosis of ring-enhancing brain lesions is wide, and the etiology is most commonly vasculitic, infectious, or neoplastic.
- Our patient presented with a large, open ring-enhancing lesion that decreased in size with glucocorticoid treatment consistent with a tumefactive demyelinating lesion.
- There is growing literature support for foregoing biopsy in such cases unless lesions are resistant to treatment or malignancy is highly suspected.2,3,4
- The patient also presented with several lesions with differing enhancement on MRI suggestive of dissemination of disease space and time—fitting the 2010 McDonald criteria for diagnosis of MS.5
- Cerebral spinal fluid demonstrating the presence of oligoclonal bands may suggest MS; however, this test lacks sensitivity and is not used in formal McDonald criteria.6,7
- No standard acute treatment for TMS has been established, but literature review of case reports and case series describes successfully treating patients with 4-5 days of glucocorticoid therapy.2,3,4,7,8
- Because TMS has been described to most commonly follow a relapsing remitting course, it is reasonable to start the patient on disease modifying therapy to decrease the number and frequency of future attacks.2,3
- When serologic testing is negative, as was seen in our patient, brain biopsy is often considered to establish a diagnosis. Therefore, it is critical that clinicians are aware of atypical presentations of more common diseases before subjecting patients to invasive and expensive testing.
- This case serves as a reminder to keep a wide differential diagnosis and to request early neurology consultation in patients presenting with ring-enhancing lesions and focal neurologic deficits.

REFERENCES

Olmesartan-induced enteropathy: an unusual cause of chronic diarrhea

F. Clay Smither, M.D., Kapil Mehta, M.D.,
St. Vincent Hospital, Department of Internal Medicine, Indianapolis, IN

Introduction

Chronic diarrhea can be challenging as the differential diagnosis is expansive. A thorough medication review should be performed as olmesartan, also known as Benicar, is a rare source of celiac-like enteropathy.

History of Present Illness

An 81-year-old female with past medical history only significant for hypertension presented to the emergency department with 4 to 5 months of diarrhea, weight loss of 40 pounds and profound weakness. Her weakness had progressed to the point that she was unable to perform her activities of daily living. She denied blood in her stools, fever or chills, and had no abdominal pain. Three years ago she had a normal colonoscopy. Her family history was significant for colon cancer in her mother. Her medications consisted of metoprolol, olmesartan, amlodipine and hydrochlorothiazide. She had been taking olmesartan for 13 months. On admission she was afebrile, normotensive and her abdominal exam was essentially normal. Her admission labs were significant for a potassium of 2.6, slightly elevated liver enzymes, and elevated BUN to creatinine ratio. Her WBC and hemoglobin were normal. Stool testing was negative for ova, parasites, and clostridium difficile.

Hospital Course

Gastroenterology was consulted and performed an EGD with biopsies and flexible sigmoidoscopy with biopsies. The duodenal biopsies showed marked villus atrophy and intraepithelial lymphocytosis, consistent with a sprue-like injury pattern. The left colon biopsies showed mild focal colitis, and the rectum biopsies showed acute proctitis with pseudomembrane-like inflammatory exudates. Tissue transglutaminase testing was negative with a normal level of IgA. The patient was hospitalized and placed on TPN. As her clinical course improved, she was transitioned to oral nutrition and discharged to a rehabilitation facility.

Case Resolution

Within six weeks of stopping olmesartan, the patient’s diarrhea resolved. She began to regain her weight and was able to return home.

Discussion

This patient had been on olmesartan for about 9 months before she had severe diarrhea. Her biopsies showed a sprue-like enteropathy, but her celiac tests were negative. This makes the cause of her diarrhea most likely olmesartan. This adverse effect of the medicine has only recently been published. The first recognition of this condition was in 2012 with a report on 22 patients who had been referred because of chronic diarrhea. These patients all had intestinal villous atrophy but negative IgA tissue transglutaminase antibody testing. The diarrhea resolved in all 22 patients once olmesartan was discontinued. In 17 out of 18 patients repeated biopsy showed resolution of the enteropathy within months. Since this report there have been other case reports of duodenal sprue-like enteropathy that resolved once patients were taken off of olmesartan. The mechanism of injury is not well-known, however it is hypothesized that it could be a cell-mediated hypersensitivity because it often takes months to years before the enteropathy occurs. This case underscores the importance of medication review when seeing a patient with chronic diarrhea.

References

### Introduction

Henoch-Schönlein purpura (HSP) or IgA vasculitis is the most common systemic vasculitis. HSP is a systemic small vessel vasculitis that is characterized by a leucocytoclastic vasculitis and deposition of immune complexes, IgA and C3 in the kidneys, skin, gastrointestinal tract and joints. Approximately 50% of patients with HSP will have kidney involvement. Therapy is typically steroids and typically runs a benign course. Cardiac involvement is considered an extremely rare and severe feature of HSP. Only 12 cases of cardiac involvement have been reported. None were steroid responsive but one was responsive to immunosuppressive therapy. We present a rare case of untreated HSP nephritis with cardiac involvement in an adult.

### Case Presentation

A 21-year-old white male presented with 3 weeks of worsening abdominal pain and constipation. He also endorsed progressive dyspnea on exertion, orthopnea, lower extremity swelling, and recent weight gain of 20 pounds. Past medical history revealed multiple bouts of gross hematuria with an associated petechial rash on his legs at age 8 which resolved spontaneously.

- Physical exam was significant for S3, bilobar rales, and 3+ lower extremity edema, but no rash.
- Initial laboratory evaluation demonstrated a troponin of 0.15 ng/mL, significantly elevated proBNP 20,000 pg/mL, renal insufficiency with a creatinine of 2.3 mg/dl and confirmed nephropathy with an elevated protein/creatinine ratio of 5.4.
- EKG demonstrated poor R-wave progression and nonspecific ST-T wave changes.
- Chest x-ray revealed cardiomegaly and vascular congestion.
- He was diagnosed with acute Heart Failure and nephrotic syndrome.
- A 2D-Echocardiogram demonstrated an ejection fraction less than 10% with diffuse, global hypokinesis.
- Further testing for nephropathy and heart failure included: ANA, Complement C3, C4, Hepatitis A antibody, Hepatitis B surface antigen, Hepatitis B Core Antibody IgM, Hepatitis C Antibody, Echo virus, Coxsackie type B1-B6, Adenovirus Antibody, CMV Antibody IgM, HIV, all negative.
- Cardiac MRI was done which showed an EF 9% with moderate to severe TR and global hypokinesis.
- The patient also underwent a cardiac catheterization that showed no evidence of coronary artery disease.
- Patient then underwent renal biopsy which showed focal segmental sclerosing glomerulonephritis consistent with Henoch-Schönlein purpura nephritis/ IgA nephropathy.

### Clinical Course

The patient was started on a regimen of Lasix drip and Milrinone. He symptomatically improved with diuresis.

### Conclusions

With as few as 12 reported cases of HSP with Cardiac involvement, only 3 of those cases had myocardial biopsy performed. All 3 cases demonstrated leukocytoclastic vasculitis. Cardiac involvement is rare, it is often a life threatening complication of HSP. Cardiac involvement can range from elevated troponin and reversible ECG changes, to myocardial necrosis and congestive heart failure. Patients often present with subtle symptoms such as abdominal pain due to intestinal deposition of IgA.

Cytotoxic immunosuppressive therapy may prevent infarction and deleterious outcome. Treatment with steroids and or cyclophosphamide in prior cases has deleterious outcome.

- Further investigation of the natural history of HSP complicated with cardiac involvement. Possibly delineating of the incidence of subclinical disease into adulthood.
- Cardiac involvement can range from elevated troponin and reversible ECG changes, to myocardial necrosis and congestive heart failure.
- Patients often present with subtle symptoms such as abdominal pain due to intestinal deposition of IgA.

### References

- Author's study. Oral presentation at the International AIDS Society's 2005 Conference.
Non-resolving pneumonia: TB masquerading as CAP
Jason Stegink MD and Lannie J. Cation MD, FACP

HPI: A 36 year old grocery store clerk with no past medical history was admitted to the hospital with persistent productive cough. She had been treated twice for community acquired pneumonia in the past 2 months - once as an outpatient with azithromycin, and then with a 10 day course of levofloxacin that included a 3 day hospital admission. However, her symptoms persisted and she presented to the ED for continued cough productive of yellow sputum and fever. Chest x-ray showed opacities in right lung that were slightly more pronounced than her previous admission (see CXR).

ROS: the patient endorsed right sided chest pain, worse with coughing, fevers and chills, mild nausea and occasional post-tussive emesis; she denied shortness of breath, hemoptysis, weight loss, night sweats, or dyspnea on exertion.

Past Medical History: None
Social History: Denies tobacco use, reports occasional alcohol use, and regular marijuana use prior to onset of cough.
Family History: Notes exposure to an uncle with history of active TB approximately 16 years ago.

Vital Signs: T 100.5    P 149   R 20   BP 118/70
Physical Exam: significant for decreased breath sounds in right upper chest with crackles in right lower lobe. Right anterior chest was tender to palpation; pain exacerbated also with cough. Remainder of exam unremarkable.

Laboratory Evaluation
Blastomyces antigen: negative
Legionella antigen: negative
Cryptococcus antigen: negative
Blood culture: no growth
Sputum culture: moderate acid fast bacilli
Quantiferon-GOLD: positive
HIV: negative
Urine HCG: negative
CT abdomen/pelvis: negative

Hospital Course:
On admission, patient was found to have multiple large cavitary lesions on CT chest and was placed in isolation. Infectious workup was largely negative until sputum culture was repeatedly positive for acid fast bacilli. Sputum culture later became positive for Mycobacterium tuberculosis. A workup for a cause for reactivation TB was not found including HIV or malignancy evaluations. Patient was begun on RIPE therapy and was discharged once AFB smears became negative. Follow-up with her PCP and the Health Department were arranged.

Discussion:
We present a case of active pulmonary TB in an otherwise healthy patient. She had presented to the healthcare system twice prior to her admission for respiratory symptoms not responding to standard CAP therapy. Unfortunately, TB (or other cause of non-resolving pneumonia) was not suspected or considered until multiple cavitary lesions were discovered incidentally on a chest CT done to rule out pulmonary embolism. Of note, the patient did not have any of the traditional symptoms associated with active tuberculosis like hemoptysis, night sweats, or weight loss, but she did have a history of TB exposure in the past which had not been previously elicited. Her TB exposure was presumed to have been her uncle years ago, but she had not received any workup or follow-up since that time. Unfortunately, the patient potentially exposed the community and many healthcare workers to active TB. This case illustrates the importance of broadening the differential diagnosis in a patient with non-resolving pneumonia to include TB even if the patient lacks classic symptoms. It also illustrates the importance of obtaining an accurate and thorough past medical history in patients with respiratory symptoms to ascertain if there a history of potential communicable infectious exposures.

CTA Chest Impression: Multifocal, cavitary, nodular pulmonary lesions with surrounding ground glass opacifications. Findings are suspicious for a multifocal infectious etiology. Subcarinal and right hilar lymphadenopathy. No PE.
**CASE PRESENTATION**

A 45-year-old male was admitted for epigastric pain, nausea, vomiting, and melena of two weeks duration.

- **Past Medical History:** coronary artery disease with coronary artery bypass graft (CABG) surgery in 2011 and diabetes mellitus type 2
- **Family History:** unknown because the patient was adopted
- **Social History:** denied tobacco, drug, or alcohol use.
- **Physical Examination:** stable patient with epigastric tenderness to palpation and minor extremity ecchymoses. Digital rectal exam revealed melena.

CT scan with contrast was significant for liver lesions. International normalized ratio (INR) was elevated despite adequate Vitamin K and fresh frozen plasma.

**LABS**

- **White Blood Cell count:** 8.1 k/cumm
- **Hemoglobin:** 7.8 g/dL from baseline of 11.0 g/dL
- **Platelet:** 118 k/cumm
- **Activated Partial Thromboplastin time (aPTT):** 47.5 seconds
- **Prothrombin time (PT):** 26.5 seconds
- **INR:** 3.0
- **Fibrinogen:** 656

**Mixing study:**
- Factor II level increased to 24%
- Factor IX level 82%
- Factor X level 94%
- Factor V level 82%
- Factor VII level 39%
- Factor II level 13%

**Inhibitor Present**

- **Hemoglobin:** 7.8 g/dL from baseline of 11.0 g/dL
- **White Blood Cell count:** 8.1 k/cumm
- **Liver function tests:** normal
- **Liver biopsy:**

**REFERENCES**

[11] [http://www.clinicai.org/content/1851517054-medium.png](http://www.clinicai.org/content/1851517054-medium.png)
Introduction

• Septic pelvic thrombophlebitis (SPT) is inflammation and infection of thrombosed pelvic vasculature
• Two types:
  • Ovarian vein thrombosis (OVT)
  • Deep septic pelvic thrombophlebitis (DSPT)
• Predisposition (Virchow’s triad):
  • Endothelial injury- acquired during vaginal birth, cesarean section, other gynecologic or obstetric procedures, and pelvic infection
  • Venous Stasis- dilation of pelvic vasculature after birth and inactivity postpartum or after surgery
  • Hypercoagulable state- pregnancy, malignancy

Case Description

• 22 year old female with history of severe preeclampsia, presented 6 weeks postpartum with intermittent fevers since delivery, and three days of dyspnea, dry cough and pleuritic chest pain
• Physical Exam:
  • Afebrile, mild respiratory distress
  • Coarse breath sounds in posterior right mid lung field without wheeze.
  • Tachycardia, without murmur
  • Right sided chest pain was reproducible on palpation
  • Abdomen was soft and non-tender
• Initial Imaging and Laboratory Data:
  • Elevated white count
  • Chest x-ray with patchy alveolar opacities in left lower lung field and cardiomegaly
• Hospital Course
  • Initially treated for sepsis secondary to suspected health care associated pneumonia with broad spectrum antibiotics
  • Two sets of blood cultures positive for enterococcus faecalis
  • Persistent intermittent fevers and tachycardia despite appropriate antibiotics
  • CT of chest showed large pulmonary embolus (Figure 1) and two thick-walled cavitary lesions (Figure 2); CT of the abdomen and pelvis did not demonstrate thrombosis of pelvic veins
  • Improved symptoms and defervescence with anticoagulation
  • Aortic and tricuspid valve vegetations noted on transeosophageal echocardiogram (Figures 3 and 4), consistent with endocarditis
  • She later developed severe aortic insufficiency requiring aortic valve replacement

Discussion

• OVT presents as fever and abdominal pain within a week of delivery or inciting event
  • Visualized by CT, MRI, or US in 20% of cases.
• DSPT is a diagnosis of exclusion
  • Presents with fever in the absence of abdominal pain or radiographic findings
  • SPT is notable for lack of response to antibiotic therapy alone and marked response to anticoagulation within 48 hours
  • Complications of SPT include:
    • Extension of the thrombosis into vessels outside the pelvis
    • Pulmonary embolus in 2% of cases
    • Overwhelming sepsis
    • Septic emboli
    • Death
  • Common pathogens
    • Streptococci
    • Enterobacteriaceae
    • Anaerobes
  • In uncomplicated cases, antibiotics and anticoagulation can be discontinued after resolution of fever and leukocytosis for 48 hours

Conclusions

• SPT should be considered in pre-disposed patients who present with persistent fever
• Absence of radiographic findings does not rule-out the diagnosis of SPT
• Treat with empiric antibiotics and anticoagulation
• Early identification and treatment is key as complications of SPT have the potential for significant morbidity and mortality

References


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