A 45 Year Old with Sudden Stroke: A Rare and Silent Culprit

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Introduction

Acute ischemic stroke in young individuals can be a sign of serious underlying pathology in which prompt recognition is essential for effective treatment and better physical outcomes. In most cases, initial presentation of these patients, cardiac embolism from either intramural thrombi or vegetations or even arterial dissection are most commonly considered where as much rare causes are often overlooked. This case demonstrates an unexpected disease entity that caused acute ischemic stroke in a young patient.

Case

- Pt is a 45 y/o F who presented with sudden syncope while taking a shower and was found to have left hemiparesis with left facial droop and dysarthria after she woke up.
- No chest pain, SOB, aura, convulsions, or post-ictal state.
- History of Hodgkin’s lymphoma treated with radiation 20+ years ago.
- History of severe menorrhagia with chronic anemia.
- LMP 2-3 weeks ago
- No significant surgical, family or medication history.
- No history of illicit drug use
- 20 pack year smoking history
- Except for leukocytosis of 16.8 k/μm, there are no major laboratory abnormalities on admission.
- Head CT w/o contrast on admission was unremarkable.
- The decision to use TPA was deferred in light of recent severe menstrual bleeding.

Differential Diagnoses

- Paroxysmal Atrial Fibrillation
- Acute Thrombotic Occlusion
- Infective Endocarditis
- Paradoxical Embolism via PFO
- Thrombophilia

2nd day

- MRI of brain on the second day confirmed bilateral cerebellar and right posterior frontal cortex consistent with cardio-embolic source.
- The patient also developed an acute inferior wall NSTEMI on the same day with a 3rd degree AV block requiring temporary transvenous pacing.
- Anticoagulation was not pursued given risk of hemorrhagic conversion.

- The list of differential diagnosis was rearranged with intramural thrombi vs. infective endocarditis being at the top.
- A transthoracic ECHO with bubble study was then performed and was negative for intramural thrombi or PFO.
- Subsequently the diagnosis of Mitral valve papillary fibroelastoma was confirmed on transesophageal ECHO as a 1 cm mobile mass on the posterior leaflet of the mitral valve as shown below.

Discussion

- Cardiac Papillary Fibroelastoma is a rare primary cardiac tumor with prevalence of less than 0.002%. It mostly originates on the mitral valve with the greatest predilection to aortic valves with sizes 0.2 cm <

- Additional work up was negative for infective endocarditis, clinically significant carotid artery stenosis, or thrombophilia.
- The patient underwent a left cardiac catheterization which found a chronic total occlusion of right coronary artery requiring no stent placement.
- The patient had a favorable outcome regaining most of her functional capacity with conservative management and physical therapy.
- She eventually underwent successful planned surgical resection of the fibroelastoma to reduce risk of future recurrence.

- Mitral Valve Papillary Fibroelastoma (MVPF) is comprised of a homogenous fibroelastic and collagenous core encased with endocardium.

- Transesophageal echocardiography is the best modality to establish the diagnosis. (76.8% sensitivity compared with transthoracic echocardiogram 61.9%). Sensitivity improves with sizes 0.2 cm <
- Characteristics on TEE:
  - Pedunculated and mobile
  - Homogenous
  - Peripheral speckled appearance
- Risk of stroke is directly proportional to size and mobility.

References


Acknowledgements

- Figure 1, Buttan A K et al. Circulation. 2012:125.
- Figure 2. Sun J P et al. Circulation. 2001:103:2687-2693
**Introduction**

Sarcoidosis is an inflammatory, granulomatous disease of unknown etiology. The disease most often presents with pulmonary involvement and symptoms such as cough and/or shortness of breath. Chest radiography demonstrates characteristic findings of hilar lymphadenopathy. Extrapulmonary sarcoidosis presents a diagnostic challenge.

**Case**

A 61 year-old Caucasian gentleman presented with malaise, anorexia, weight loss, mild cognitive changes and hypercalcemia.

Past medical history includes paroxysmal atrial fibrillation, coronary artery disease, congestive heart failure, obstructive sleep apnea, and diabetes mellitus. None of his medications were known to cause hypercalcemia.

No lymphadenopathy, pulmonary findings, hepatosplenomegaly or skin changes were found on exam.

**Laboratory Data**

- **Calcium**: 14.1 mg/dL (8.4 mg/dL-10.5 mg/dL)
- **Albumin**: 3.1 gm/dL (3.5-5.0 gm/dL)
- **PTH**: 5 pg/mL (10-65 pg/mL)
- **Angiotensin converting enzyme level**: 99 U/L (9-67 U/L)
- **1-25 hydroxy-vitamin D**: 99 pg/mL (18-72 pg/mL)
- **Bone marrow**
  - **Whole-body PET/CT**: heterogeneous uptake throughout the bone marrow.
  - **and mild splenomegaly**
  - **CT**: multiple <5mm calcified nodules throughout the lungs
  - **Skeletal survey**: negative for osteolytic lesions
  - **PA/lateral CXR**: cardiomegaly (Figure 1)
  - **Bone marrow biopsy**: normocellular bone marrow with non-caseating granulomas (Figure 3 and Figure 4)

**Imaging**

- **PA/lateral CXR**: cardiomegaly (Figure 1)
- **CT**: multiple <5mm calcified nodules throughout the lungs and mild splenomegaly
- **Whole-body PET/CT**: heterogeneous uptake throughout the bone marrow.
- **Bone marrow biopsy**: normocellular bone marrow with non-caseating granulomas (Figure 3 and Figure 4)

**Diagnosis**

The presence of non-caseating granulomas in the bone marrow along with the patient’s clinical presentation confirmed the diagnosis of isolated extrathoracic sarcoidosis as a rare etiology of refractory hypercalcemia.

**Discussion**

Extrapulmonary disease can be severe and life threatening and its presence affects the therapeutic approach. Diagnostic dilemmas arise when trying to search for affected organs, particularly when patients are asymptomatic. The use of PET/CT relies on glucose hypermetabolism by granuloma cells and can be used for mapping of inflammatory sites and identification of occult disease. In one study PET/CT revealed an occult site, not detected by exam or standard imaging (CXR/CT), in 15% of patients.

The sensitivity of PET/CT in detecting active sarcoidosis is 80-90%. Tissue biopsy is then required for diagnosis as specificity is low. The wide range of involvement of sarcoidosis does affect disease detection with PET/CT. In one study, PET/CT detected 100% of intrapulmonary lesions and 90% of extrapulmonary lesions in patients with biopsy-proven disease.

Our case illustrates the role PET/CT can play in the diagnosis of extrathoracic sarcoidosis. It is especially useful in those with unusual presentations and normal chest imaging.

**References**

A debilitated, 46-year-old, Caucasian woman presented from a nursing facility due to concern of superimposed infection on chronic non-healing ulcers. She developed wounds on her hips approximately 5 months earlier, after hospitalization for acute lower extremity deep venous thrombosis. At that time, anticoagulation with warfarin was started. Approximately 3 weeks following initiation of warfarin, she developed painful ulcerations that did not respond to standard outpatient wound treatment. She developed wounds on her hips approximately 5 months earlier, after hospitalization for acute lower extremity deep venous thrombosis. At that time, anticoagulation with warfarin was started. Approximately 3 weeks following initiation of warfarin, she developed painful ulcerations that did not respond to standard outpatient wound treatment.

**Past Medical History**
- Deep Venous Thrombosis
- Obesity
- Gastric Bypass
- Iron Deficiency Anemia

**Physical Exam**
- Obesity (BMI 36.9 kg/m²)
- Multiple ulcers with little surrounding erythema. The 4 cm x 5 cm lateral left thigh ulcer extended into necrotic subcutaneous tissue with no purulent discharge or erythema. Ulcers overlying the left medial thigh, right hip, left anterior tibia, and right heel had clean bases with granulation tissue.
- No abnormal black or blue skin discoloration.

**Clinical Presentation**

**Overview**
- Well-known to occur in end-stage renal disease, calciphylaxis can occur in other settings as well, such as treatment with warfarin.

**Warfarin as the Culprit of Calciphylaxis**

**Nicholas W. Creasap, MD St. Vincent Hospital, Indianapolis, Indiana**

**Pathophysiology**
- The exact mechanism is still unknown. It is postulated that vascular smooth muscle cells transdifferentiate into osteoblast-like cells through several cell-signaling pathways that converge on a common transcription factor, nuclear factor k-B, eventually expressing other factors such as bone morphogenetic protein and endothelin-1. Warfarin may inhibit matrix G1a protein, an inhibitor of bone morphogenetic protein.

**Labs/Imaging**
- WBC 5.5 k/cumm with normal differential
- Hgb 10 g/dL with MCV 78.1 fL
- BUN 6 mg/dL
- Creatinine 0.44 mg/dL
- Calcium 9.0 mg/dL (highest)
- Phosphorus 3.5 mg/dL
- Total protein 5.7 g/dL
- Albumin 1.9 g/dL with prealbumin 6 mg/dL
- ESR 105 mm/hour
- CRP 8.8 mg/dL
- ANA negative
- HIV non-reactive
- Hypercoagulation workup normal

**Nuclear Bone Scan:**

**Biopsy**
- Several biopsies had previously obtained, which demonstrated epidermal ulceration, fat necrosis, acute inflammation, granulation, and intramural calcification. No fibrin thrombi were present to suggest typical warfarin-induced necrosis.

**Outcome**
- Warfarin replaced by rivaroxaban. She was discharged back to the skilled nursing facility with wound therapy. The wounds completely healed 3 months later. Her severe pain resolved. She is now ambulating with a walker and is hopeful to walk independently soon.

**Final diagnosis**
- Patient was diagnosed with warfarin-induced calciphylaxis based on the timing of ulcers, biopsy & bone scan results, and improvement of ulcers with cessation of warfarin.

**Discussion**
- Risk factors include female sex; obesity; hyperphosphatemia; hypoalbuminemia; hypercoagulable states; white race; elevated ESR; and exposure to medications such as warfarin, calcium-based binders, vitamin D supplementation, and glucocorticoids. Skin biopsy with subcutaneous fat is the standard. Punch biopsy may not be sufficient, so more extensive excisional biopsy is required. Bone scan shows extensive calcification in 97% of patients.

**Pathophysiology**
- The exact mechanism is still unknown. It is postulated that vascular smooth muscle cells transdifferentiate into osteoblast-like cells through several cell-signaling pathways that converge on a common transcription factor, nuclear factor k-B, eventually expressing other factors such as bone morphogenetic protein and endothelin-1. Warfarin may inhibit matrix G1a protein, an inhibitor of bone morphogenetic protein.

**Therapy**
- The most imperative intervention is to address the underlying pathology promoting calciphylaxis. Examples include removing the offending agent; avoiding trauma; correcting calcium and phosphate abnormalities with non-calcium-phosphate binders; and cinacalcet for elevated PTH.

**Prognosis**
- In the presence of ESRD, calciphylaxis may have a 6-month mortality rate as high as 33% in non-ulcerating cases and 80% in those with ulcers. Rates in non-ESRD related cases are unknown.

**References**
Chronic diarrhea in pregnancy: Don’t blame it all on the baby
A peripartum presentation of ulcerative colitis
Kyle Glienke MD, Ishan Gohil MSIV, and Anthony Martin MD
St. Vincent Internal/Family Medicine Residency Program, Indianapolis, IN

Learning Objectives

- Identify inflammatory bowel disease (IBD) as a chronic disease of young patients in their reproductive years
- Review the general principles of management of IBD in pregnancy and the postpartum period
- Identify common complications of pregnancy associated with IBD

Background

- IBD is a common cause of subacute or chronic, often bloody, diarrhea in teenagers and younger adults
- IBD commonly affects women during reproductive years, therefore pregnancy may complicate the diagnosis and significantly alter treatment options
- Pregnant women with IBD are at a higher risk for complications, including preterm birth, small for gestational age birth, hypertensive disorders of pregnancy, and prolonged or premature rupture of membranes

Patient Presentation

- 22 year old G1P1 Caucasian female, postpartum day one
- Four month history of watery, non-formed stools, 3-5 episodes per day
- 22 year old G1P1 Caucasian female, postpartum day one
- Daily prenatal vitamin
- Review of systems positive for dyspnea on exertion and lightheadedness
- Denied recent antibiotic use, sick contacts, or travel
- Four month history of watery, non-formed stools, 3-5 episodes per day

Diagnosis and Treatment

- Pathology evaluation of biopsies: Diffuse moderate colitis with cryptitis and crypt abscesses, most consistent with ulcerative colitis
- Treatment started with prednisone orally and mesalamine after confirmation of safety with breastfeeding
- Stools found to be grossly bloody
- Hematologic consultation and evaluation negative for consumptive coagulopathy
- Obstetric consultation and evaluation consistent with normal post-partum changes
- Colonoscopy with random biopsies: Moderate to severely erythematous and ulcerated mucosa noted from the anus to the cecum

Further Evaluation

- Spontaneous improvement in platelets and renal function, persistence of anemia and metabolic acidosis
- Stools found to be grossly bloody
- Hematologic consultation and evaluation negative for consumptive coagulopathy
- Obstetric consultation and evaluation consistent with normal post-partum changes
- Colonoscopy with random biopsies: Moderate to severely erythematous and ulcerated mucosa noted from the anus to the cecum

Treatment Safety in Pregnancy and Breastfeeding

<table>
<thead>
<tr>
<th>Class</th>
<th>Pregnancy Category</th>
<th>Adverse Effects</th>
<th>Comments</th>
<th>Breastfeeding</th>
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<td>Aminosalicylate</td>
<td>Folate deficiency</td>
<td>Preparation-dependent risk</td>
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<tr>
<td>Prednisone</td>
<td>Corticosteroid</td>
<td>Orofacial clefts, premature ROM</td>
<td>F Fare treatment</td>
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<td>Folate antagonist</td>
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<td>Infliximab</td>
<td>Anti-TNF antibody</td>
<td>Limited human data</td>
<td>Not recommended</td>
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</table>

General Principles

- Counseling regarding heritability
- Fertility preserved or mildly reduced with adequate treatment
- Exceptions of surgical intervention
- Pregnancy not proven to increase likelihood of disease flare
- Risk of flare during pregnancy most likely to reflect disease activity prior to pregnancy, but may vary
- Management of flares in pregnancy similar to that in nonpregnant patients
- Cesarean section recommended with active perianal disease seen in Crohn’s disease
- IBD patients are at increased risk of complications
- Early and late preterm birth
- Small for gestational age birth
- Hypertensive disorders of pregnancy
- Prolonged or premature rupture of membranes
- Neonatal death
- Maternal—thromboembolism, malnutrition
- Likely higher risk with increased disease severity
- Achieving and maintaining disease remission key to reducing fetal and maternal complications
- Breastfeeding may reduce disease severity postpartum

Take Home Points

- Complete history and physical important for accurate diagnosis in setting of chronic diarrhea
- Poorly-controlled IBD in pregnancy is associated with pre-eclampsia, as well as other disorders
- Pregnancy has a variable effect on the severity of disease and frequency of flares
- Most medications used in the management of IBD are compatible with pregnancy, other than methotrexate

References

INTRODUCTION

Statin-associated necrotizing autoimmune myopathy is a rare condition with an incidence of approximately two in one million people per year. The etiology of this condition is thought to be due to an auto-antibody directed at 3-hydroxy-3-methylglutaryl-CoA reductase (HMGCR). Patients with this condition present with proximal muscle weakness, myalgia, and elevated creatine kinase (CK) levels. As opposed to other statin-induced myopathies, patients continue to be symptomatic even after discontinuation of the offending medication. Diagnosis is based on clinical presentation and muscle biopsy, which shows a necrotizing myopathy with little inflammatory component. The condition typically improves with systemic steroids.

PATIENT PRESENTATION

- 66 year old white male with a history of hypertension and hypercholesterolemia.
  - Medications are lisinopril and atorvastatin, both of which have been prescribed for years.
- Presented to his primary care physician with complaints of malaise and generalized weakness for approximately two weeks.
  - His symptoms began after working outside on his car during a hot day.
- One month prior to presentation the patient was noted to have an asymptomatic transaminitis.
  - AST 144, ALT 266 IU/L.
  - Atorvastatin was discontinued at this time.
- On physical exam the patient had 4/5 hip flexor strength bilaterally, but otherwise his vitals and exam were normal.
  - Initial laboratory data:
    - AST 570, ALT 553 IU/L.
    - CK 16,295 IU/L.
    - Serum aldolase 149 U/L.
    - Urine myoglobin was elevated beyond our laboratory’s upper limit.
    - ESR 85 mm/hr.

HOSPITAL COURSE

- The patient was treated for presumed rhabdomyolysis with aggressive intravenous hydration.
  - Despite this, the patient’s CK rose as high as 25,824.
  - The patient developed symmetric proximal weakness, most pronounced in the upper extremities.
  - Renal function remained normal.
- EMG was consistent with either a necrotizing or early inflammatory process.
- Muscle biopsy revealed significant myofiber necrosis with prominent infiltration of macrophages within necrotic fibers and the endomysium.
  - No evidence of polymyositis or dermatomyositis.
  - Necrotizing autoimmune myopathy, most likely related to statin use, is diagnosed.
- Upon initiation of prednisone at a dose of 1 mg/kg/day, the patient’s symptoms and CK levels improved rapidly.

DISCUSSION

Our patient presented with a subacute myopathic process consistent with an inflammatory myopathy. Within the category of inflammatory myopathies is a recently recognized subgroup entitled necrotizing autoimmune myopathy (NAM). This subgroup shares common histopathologic features which differentiate it from the broader category: myocyte necrosis without significant inflammation. Although sometimes idiopathic, recent case series have shown an association with this condition and statin use.

The pathophysiology of this condition is likely related to up-regulation of HMGCR and subsequent antibody formation to this enzyme. This antibody is 94% sensitive and 99% specific for statin-associated NAM. We did not test for this antibody in our patient as it not yet standard of care and it would not have changed our clinical management.

CONCLUSION

Knowledge of this diagnosis is important for clinicians as the number of prescriptions of statins is increasing in the United States. Statin-associated NAM may persist for weeks to months, and even progress, despite discontinuation of the medication. This is in contrast to the toxic myopathy associated with statins, which usually improves after the medicine is discontinued. Furthermore, treatment with steroids or other forms of immunosuppression is required to control this condition.

REFERENCES

Spinal Cord Ischemia After Methamphetamine Abuse: A Case Report

Maitri Kalra, MBBS ; Andrew Walker
Indiana University school of Medicine, Indianapolis, IN

Introduction

Methamphetamine is a stimulant type of recreational drug, very commonly abused in the United States. It enhances transmission at the catecholaminergic and dopaminergic synapses and produce elation and increased alertness, with increased motor activity. Stroke is the most common lasting adverse neurological event associated with methamphetamine use. Spinal cord effects are rarely reported. Here is a case of spinal cord ischemia after recreational use of methamphetamine.

Case Presentation

35 year old Caucasian male was brought to the emergency room after being found down by his roommate. On examination, his vital signs were stable except for oxygen saturation of 81% on room air. He was unresponsive; pupils were equal, round and reactive to light. Muscle tone was normal and deep tendon reflexes (DTR) were 2+ in all four extremities. Rest of his examination was unremarkable.

Laboratory investigations revealed serum creatinine 2.65, troponin 1.39 and lactate 5.1. Urine tested positive for amphetamines. CT scan of head showed no acute changes.

Clinical Course: He was intubated for airway protection in ER. He received aggressive fluid resuscitation and became responsive after eight hours.

He was subsequently extubated but, the following day, patient developed weakness in bilateral lower extremities acutely along with urinary retention. On examination, he had decreased muscle tone in bilateral lower extremities. Muscle strength was 3/5 for left hip flexion and knee extension, 1/5 for right hip flexion and knee extension, 4/5 for ankle plantar/dorsiflexion bilaterally. DTR were 2+ symmetrically in upper and lower extremities. Babinski’s sign was absent bilaterally. Sensations in lower extremities were decreased to light and sharp touch distal to the right knee, but normal on left leg. Rest of the neurological examination including cerebellar examination was normal.

MRI Images of Thoracic and Lumbar Spine

MRI of spine revealed abnormal T2 signal in the central cord at the level of the T11-T12 vertebral bodies. The lesion was in the watershed area with sparing of posterior column suggestive of infarct.

A lumbar puncture was planned but patient refused. After 24 hours, his lower extremity weakness started improving along with return of sensations gradually over the following days. Muscle strength was 5/5 in left lower extremity and 4/5 in right lower extremity muscles. At the time of discharge he was able to walk 2 feet with rolling walker.

Discussion

Patient had multi-organ injury with Type II NSTEMI, acute kidney injury and spinal cord ischemia. Though lumbar puncture could not be done, the transient, rapidly improving symptoms make spinal ischemia the likely diagnosis. The mechanism may be related to elevated catecholamine concentration, which causes vasospasm, platelet aggregation, and thrombus formation. Long-term use of amphetamines can cause repetitive episodes of vasospasm and paroxysms of hypertension, which may result in endothelial damage, and acceleration of atherosclerosis.

Conclusion

Methamphetamine use can cause spinal cord ischemia by vessel thrombosis or vasospasm.
Impact of Evidence-Based Guidelines for Management of Clostridium Difficile Infection

Emily Cochard, MD, PGY3; Carol Ruppert, MD PGY2; Stephen Knaus, MD; Lindsay Saum, PharmD

Introduction

Clostridium difficile infection (CDI) is a prevalent and potentially fatal cause of infectious diarrhea in hospitals, thus responsible for a significant cost to the healthcare system and adverse patient outcomes.

• 500,000 cases per year in the United States
• 15,000-20,000 deaths attributed to CDI

Primary CDI
• $2,870-$4,846 attributed to CDI per case
• $9,822-$13,854 total costs of treatment per case

Recurrent CDI
• $13,655-$18,067 attributed to CDI per case
• Total cost of treatment 3X higher than primary CDI

Background

In order to follow best evidence for treatment of CDI and improve quality care and patient outcomes, a group of internal medicine faculty, residents and pharmacists reviewed relevant literature to devise a set of evidence-based diagnosis and treatment guidelines for the teaching hospital service. These guidelines were publicized via a noon conference, an email, an internal website and a residents handbook. The guidelines emphasized severity-based treatment and provided institution-specific guidance for infection control and diagnosis of CDI.

Methods

We retrospectively analyzed data on all patients (79) treated for CDI at our hospital in a three-month period after publication of the updated guidelines in 2013. Outcome examined included length of stay after diagnosis (mLOS), mortality, cost and readmission rate in the group of patients treated with guideline-based (GB) therapy and those not treated with guideline-based therapy. IRB approval was obtained.

We also surveyed residents after the most recent conference on the updated CDI guidelines to assess our influence.

Results

• On a 5-point Likert scale, residents (11 respondents) reported that the conference was educationally beneficial (4.18) and that attending the conference would change their management (4.36).

Discussion

While none of our outcomes reached statistical significance, we did discover trends towards improvement in some of the outcomes measured.

• mLOS, though shorter in patients treated with guideline-based therapy, could have been confounded by other co-morbidities. We subtracted the days prior to diagnosis of CDI to attempts to obtain a better measurement of the LOS attributable to CDI.

• Mortality was lower in the group treated with guideline-based therapy but a larger sample size may be needed to reach significance.

• Readmissions were lower in the group treated with guideline-based therapy but a larger sample size may be needed to reach significance. All-cause readmissions were assessed, which may not reflect initial CDI therapy.

• Cost may have been higher due to multiple confounding variables.

• Interestingly, despite the educational resources provided, resident and hospitalist physicians followed guideline-based therapy only 45.2% of the time. Increasing awareness of the guidelines’ existence might improve utilization.

Conclusion

This study analyzed the effect of a best-practice guideline for treatment of CDI on patient outcomes and educational benefit within a hospitalist service in a teaching hospital. We discovered trends toward improved outcomes, but they did not reach statistical significance. Our study was underpowered and confounding factors (co-morbid conditions) may have affected results. We propose that development of best practice guidelines for quality care within an internal medicine residency program can improve education of residents and management of patients with CDI.

References

Intentional Isoniazid Overdose: An Uncommon Toxicity

Chris Kniese, MD • Grant Gilroy, MD • Praveen Mathur, MD
Indiana University School of Medicine, Indianapolis Indiana

Introduction

Newly diagnosed cases of tuberculosis in the United States have continued to decline over the past two decades due to increased diagnosis and management of latent tuberculosis infections1. Despite documented adverse events, isoniazid combined with pyridoxine remains first-line therapy. We present a case of status epilepticus and metabolic acidosis precipitated by intentional overdose of isoniazid and pyridoxine.

Case Presentation

Our patient is a 30 year old Hispanic female who was brought to the emergency department due to seizures. Empty bottles of pyridoxine and isoniazid were found next to her. She required multiple doses of benzodiazepines for recurrent seizures and pyridoxine and isoniazid were found next to her. She required the emergency department due to seizures. Empty bottles of pyridoxine has shown to be effective2. Although studies in intentional isoniazid overdose, and treatment with single-dose pyridoxine remains first-line therapy. We present combined with pyridoxine remains first-line therapy. We present...

Lab Results

| WBC | 19.3 | k/μL |
| Hemoglobin | 13.1 | g/dL |
| Hematocrit | 30% |
| Troponin | <0.03 | ng/mL |
| Sodium | 136 | mmol/L |
| Potassium | 3.3 | mmol/L |
| Chloride | 103 | mmol/L |
| Bicarbonate | 9 | mmol/L |
| Anion gap | 24 | mmol/L |
| Blood urea nitrogen | 36 | mg/dL |
| Creatinine | 0.62 | mg/dL |
| Glucose | 240 | mg/dL |
| Calcium | 6.8 | mg/dL |
| Albumin | 4 | g/dL |
| Bilirubin, total | 0.3 | mg/dL |
| Alkaline phosphatase | 101 | IU/L |
| AST | 47 | IU/L |
| ALT | 52 | IU/L |
| Protein, total | 8.5 | g/dL |
| Acetaminophen, serum | <2 | mg/dL |
| Ethanol, serum | <3 | mg/dL |
| Sarcosine | 2 | mg/dL |

Learning Objectives

1. Isoniazid (INH) toxicity can present with elevated anion gap metabolic acidosis with or without seizure activity.
2. Single-dose pyridoxine is safe and effective therapy for INH toxicity.
3. Transient abnormalities in liver function tests may be seen within 72 hours and can improve with supportive care.

Discussion

Status epilepticus with anion-gap metabolic acidosis has previously been described in patients who have taken an intentional isoniazid overdose, and treatment with single-dose pyridoxine has shown to be effective3. Although studies in animal models have demonstrated the potential neurotoxicity of significant doses of pyridoxine4, particularly sensory neuropathy, studies in humans suggest that this is likely to be seen with high doses given over days to weeks, or in lower doses given chronically5.

The mechanism of neurotoxicity due to isoniazid is poorly elucidated in the literature but it is thought to be related to interruption of pyridoxine-dependent metabolic pathways. While these commonly manifests as peripheral neuropathy, there are several documented cases of seizure activity as well. This is likely due to interruption of gamma-amino butyric acid (GABA) production in the central nervous system6, thus inducing a state of relative GABA deficiency.

Our patient initially experienced repeated seizures despite multiple rounds of treatment with benzodiazepines. However, after receiving a single-dose of intravenous pyridoxine, she experienced no further seizure activity. Isoniazid toxicity should be considered in patients presenting with seizures and metabolic acidosis of unknown etiology, particularly in the setting of possible ingestion or history of latent tuberculosis infection.

REFERENCES

It Takes a Village To Cure Recurrent Pneumothorax

Kimberly Ku, MD
Department of Medicine, Indiana University School of Medicine
Indianapolis, IN

Learning Objectives

‣ To recognize that catamenial pneumothorax (CP) represents a spectrum entity.
‣ To emphasize an early multi-disciplinary approach to diagnosis and management of CP.

Case Description

History
‣ 39 year old West African female with multiple prior right-sided pneumothoraces while on her period status post video-assisted thoracic surgery (VATS) pleurodesis presents to the ED with acute onset right-sided chest pain. She was on her menstrual cycle.
‣ Vitals and exam were normal.
‣ CXR showed interval development of right-sided septated pneumothorax (compared to a normalized CXR after pleurodesis three months ago). Chest CT confirms findings, also shows small pleural effusion.
‣ Instructions were to establish a primary care physician; and to follow up in pulmonary, thoracic surgery, and gynecology clinics.

Evaluation
‣ Symptoms began in 2009. Five total recurrent spontaneous pneumothoraces (recurred despite VATS talc pleurodesis done both 2011 and 2013).
‣ Vitals normal. Decreased breath sounds over right upper and lower lobes; decreased right-sided diaphragmatic excursion. Otherwise unremarkable exam.
‣ Pleural fluid cytology in 2011 noted “suspicious for endometrial cells”. Pleural biopsy in 2013 showed benign fibrous tissue; no malignancy in 2011.

Clinical Course
‣ Leuprolide acetate injection for three months; depot medroxyprogesterone acetate indefinitely.
‣ No further incidents of pneumothorax or chest pain.
‣ Pulmonary and thoracic surgery clinics following on symptom basis with no further planned surgeries.

Discussion

‣ Flexible definition: recurrent pneumothoraces, reproductive age, temporal relationship with menses, with or without histologic evidence of endometriosis.
‣ Etiology still not well understood.
‣ Incidence of CP ranges from 25-30% among all women with recurrent, spontaneous pneumothoraces referred for surgical treatment.
‣ Diagnosis based on clinical history is adequate in CP. Definitive diagnosis of thoracic endometriosis requires histological evidence.
‣ Combined surgical and hormonal treatment approach most optimally decreases recurrence rates.

CT chest with IV contrast - ED visit

Thoracic endometriosis

Highly suspicious of CP
Consider surgery + VATS
Bullec only → wedge resection + pleurodesis
Diaphragmatic fenestration + thoracotomy
consider resection + mesh repair + pleurodesis

Consider medical treatment post operatively
6-12 months

References
Evaluation of differences in statin recommendations between ATP3 and ACC/AHA guidelines in a primary care population

Stephanie N Martin, MD, Amanda J Place, PharmD, BCACP, Karie A Morrical-Kline, PharmD, BCACP, Victor Collier, MD, FACP
St. Vincent Hospital, Indianapolis, IN

Background

- In 2010, over 700,000 people died of heart attack or stroke
- Cholesterol reduction, specifically with statins, plays an important role in primary and secondary prevention of atherosclerotic cardiovascular disease (ASCVD)
- ATP III lipid guidelines published in 2001 have been widely implemented and were the standard of care until new AHA/ACC guidelines published in 2013
- The new guidelines have significantly change our approach to cholesterol treatment

Hypothesis

We theorized that more patients would qualify for statin therapy with implementation of the ACC/AHA guidelines

Objectives

**Primary:** To demonstrate the difference in recommendations for statin use, in statin naïve patients, between the 2001 and 2013 guidelines

**Secondary:** To demonstrate that patients already receiving statin therapy would require more potent formulations as a result of the new guidelines

Methods

- Retrospective chart review: of 3,194 patients in our residency clinic of which 299 were randomly selected for analysis
  - Active clinic patients aged 40-75
- Lipid panels performed between November 2011 and November 2013
- Demographic and cardiovascular information collected included the necessary variables to input into Framingham and ASCVD Risk Calculators.
- Need for statin determined using ATP III and ACC/AHA guidelines and calculators

Results

**Is statin therapy recommended?**

<table>
<thead>
<tr>
<th>ATP3</th>
<th>ACC/AHA</th>
</tr>
</thead>
<tbody>
<tr>
<td>No statin</td>
<td>Statin</td>
</tr>
</tbody>
</table>

**ACC/AHA recommends increased statin potency**

- 51% Yes
- 49% No

**% of patients who should receive statins**

<table>
<thead>
<tr>
<th>Group</th>
<th>ATP3</th>
<th>ACC/AHA</th>
</tr>
</thead>
<tbody>
<tr>
<td>African American patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male Patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients with CAD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients with HTN</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Discussion

- Many editorials published since the release of the ACC/AHA 2013 guidelines have hypothesized large increases in patients requiring statins; our data appears to support this hypothesis
- The limitations of this study include:
  - Relatively small sample size
  - Some patients already on statins at random time of the sampling

Conclusion

- Statin therapy was recommended in more patients overall with ACC/AHA than ATPII
- ACC/AHA guidelines recommended increased statin intensity in almost 50% of patients who were already receiving statin therapy
- Statin therapy was recommended for more patients in all special populations analyzed in this study

References


http://circ.ahajournals.org/content/130/25/e51.full

IRB # R2013-155

Authors have no conflicts of interest to disclose

A perinephric abscess is a collection of suppurative material in the perinephric space between the renal capsule and Gerota’s fascia, if the abscess is able to extend beyond the said fascia, it is termed a paranephric abscess. The perinephric space also contains some blood vessels and lymphatics, which facilitate the spread of infection. Most of perirenal results from either the rupture of an intrarenal abscess into the perinephric space, or chronic recurrent pyelonephritis. Approximately 30% of cases are attributed to hematogenous or lymphatic dissemination of organisms from focal sources of infection such as wound infection and furuncles. Occasionally, a perinephric abscess results from the spread of infection from extraperitoneal sites, such as in retroperitoneal appendicitis, diverticulitis, pancreatitis, pelvic inflammatory conditions and osteomyelitis of adjacent ribs or vertebrae.1

The case discussed here demonstrates the difficulty in diagnosing perinephric abscess due to the slow onset of presentation, radiographic limitations and the limited penetration of certain antibiotics. It also illustrates that unusual pathogens can complicate the clinical picture. The common etiologic agents of perinephric abscesses are E. coli, Proteus species, and S. aureus. The following is a very rare case in terms of the organism isolate Salmonella sp. from a perinephric abscess.

A 44-year-old man with past medical history significant for End stage renal disease (ESRD) from autosomal dominant polycystic kidney disease (ADPKD) presented to emergency department with a stage renal disease (ESRD) from autosomal dominant polycystic kidney disease. He was admitted for evaluation of fever and flank pain without any urinary symptoms. CT scan of the abdomen without contrast revealed bilateral renal enlargement and flank pain without any urinary symptoms. CT scan of the abdomen was not used due to his chronic kidney disease. He was discharged in stable condition to continue to yield negative growth. He was transferred to our clinic for further evaluation and treatment. The patient's blood cultures were switched to one due to improved cyst penetration and prior failure of cefotaxime. He was continued on ceftazidime for improved cyst penetration and prior failure of cefotaxime due to improved cyst penetration and prior failure of cefotaxime. Antibiotics were switched to cefotaxime due to improved cyst penetration and prior failure of cefotaxime. He was discharged with an abscess infecting left renal pelvis.

He was sent to interventional radiology for abscess drainage. Culture studies of this serous fluid subsequently grew Salmonella sp. Following drainage, he had resolution of fever and leukocytosis. He was discharged in stable condition to complete two weeks of outpatient antibiotic therapy. Further review of outside records revealed that he had grown a Salmonella species from stool culture collected on admission.

A repeat CT scan showed some left sided perinephric stranding complicating the clinical picture. The common etiologic agents of perinephric abscesses are E. coli, Proteus species, and S. aureus. The following is a very rare case in terms of the organism isolate Salmonella sp. from a perinephric abscess.

Case Description

A 44-year-old man with past medical history significant for End stage renal disease (ESRD) from autosomal dominant polycystic kidney disease (ADPKD) presented to emergency department with a fever of 102.5°F and left lower quadrant abdominal pain. Other pertinent medical history includes the patient being a hemodialysis recipient three times a week, and is also a type II diabetic. Two weeks prior to this particular visit, the patient had intermittent fevers, vomiting, and non-bloody diarrhea for 5 days which resolved with supportive treatment. The patient was a known positive for mild LLQ and flank pain without any urinary symptoms. CT scan of the abdomen without contrast revealed bilateral renal enlargement and innumerable cysts consistent with his condition without any evidence of an infectious process in the abdomen or pelvis. IV contrast was not used due to his chronic kidney disease. He was empirically treated with Vancomycin and Piperacillin-Tazobactam. However over the next few days, he continued to have intermittent high grade fevers with severe leukocytosis and was thus switched to intravenous cefotaxime concerning for possible cyst infection. A repeat CT scan showed some left sided perinephric stranding confirming the suspicion of a cyst super infection but no definitive abscess or fluid collection was evident. Patient's blood cultures also continued to yield negative growth. He was transferred to our institution for further evaluation and treatment, given his refractory fevers and leukocytosis. At this point, antibiotics were switched to cefotaxime due to improved cyst penetration and prior failure of cefotaxime. Patients with diabetes account for one third of all perinephric abscess cases. Patients with polycystic renal disease undergoing hemodialysis may be particularly susceptible to developing perinephric abscesses (62% of cases). The case in discussion fulfilled each of these latter criteria, and thus unfortunately our patient was at a high risk of developing the infection.

Pathophysiology

Perinephric abscess can be a life-threatening entity. Non-specific findings in patient’s history and physical exam make this a difficult diagnosis even for an astute physician. Perinephric abscesses carry morbidity rate of 8-22% and significant morbidity occurs in 35% of patients. This rate partly is due to long delays in diagnosis and the comorbid conditions. The mortality rate is higher in sepsis, urinary tract obstruction, the presence of more underlying diseases such as diabetic ketoacidosis, WBC count greater than 25,000 cells/μL, and positive blood cultures.2

Diagnosis

CT scan is considered the diagnostic technique of choice as it identifies the abscess and defines its extent beyond the renal capsule and the surrounding anatomy. Although US is non-invasive, CT has been documented to be superior to US for diagnosing renal or peri-renal abscesses, with an accuracy rate of 90~100%.5

Presentation

The symptoms of a perinephric abscess develop insidiously making early recognition at times difficult. Fever is the most common presenting symptom and occurs in virtually all patients. Our patient as described earlier, presented with fever and unilateral flank pain. Table 1 below provides a break down of the most common signs and symptoms depicting a clinical picture of perinephric abscess.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>(% patients affected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated temperature</td>
<td>89 Flank tenderness 73</td>
</tr>
<tr>
<td>Pain-Flank</td>
<td>80 Abdominal tenderness 63</td>
</tr>
<tr>
<td>-Abdominal</td>
<td>60</td>
</tr>
<tr>
<td>-Unspecified</td>
<td>72</td>
</tr>
<tr>
<td>Chills</td>
<td>42 Temp &gt;100°F 59</td>
</tr>
<tr>
<td></td>
<td>Temp &gt;102°F 11</td>
</tr>
<tr>
<td>Dysuria</td>
<td>39 Flank mass 47</td>
</tr>
<tr>
<td></td>
<td>Abdominal mass 35</td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>23</td>
</tr>
<tr>
<td>Weight loss</td>
<td>25</td>
</tr>
<tr>
<td>Weakness</td>
<td>14</td>
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</table>

Table 1. Clinical signs and symptoms of perinephric abscess1

Etiology

The common etiologic agents of perinephric abscesses are E. coli, Proteus species, and S. aureus. Perinephric abscesses are polymicrobial in 25% of cases. Figure 3 details a breakdown of perinephric abscess etiology.

Pathophysiology

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References

Quality Improvement: Safe Prescribing Habits of Citalopram in the Elderly

Staci Hollar MD, Amanda Place PharmD, Grace Greist MD
St. Vincent Joshua Max Simon Primary Care Center, Indianapolis, IN

Introduction

Citalopram is a commonly prescribed SSRI used in the treatment of depression and anxiety. In 2011 the FDA issued a drug safety communication regarding citalopram dosing.1 It was recommended that doses greater than 20mg daily be avoided in patients 60 years and older due to increased risk of QTc prolongation and potentially fatal arrhythmias.4,5,6 Additional risk factors for prolonged QTc include female sex, electrolyte abnormalities, renal or hepatic dysfunction, and use of other QTc prolonging medications.2,3

The St Vincent Joshua Max Simon Primary Care Center (PCC) is a medical residency training facility. Residents in the Internal Medicine (IM) and Family Medicine (FM) programs routinely see patients 60 years and older, and in many cases may be responsible for prescribing antidepressant therapy.

Hypothesis

It is hypothesized that PCC prescriber adherence to FDA recommendations for citalopram prescribing in patients 60 years and older will be low.

Objectives

Primary: Identify the percentage of patients over age 60 at the PCC prescribed higher than recommended doses of citalopram.

Secondary: Compare frequency of concurrent risk factors for QTc prolongation in patients taking recommended doses and those prescribed greater than recommended doses.

If adherence to prescribing recommendations is low, will perform an educational intervention to improve awareness and change prescribing patterns.

Methods

- Institutional Review Board exempt quality improvement project using retrospective chart reviews pre- and post-educational intervention.
- Inclusion criteria for baseline analysis: active PCC IM or FM patients 60 years and older who received a citalopram prescription between January 2013 and August 2014.
- Data collected included patient demographics, prescription data, and presence of concurrent risk factors for QTc prolongation.
- Educational intervention will be conducted in November 2014.
- Post-intervention data will be collected May 2015.

Results

- 69 patients over the age of 60 years identified as taking some dose of citalopram
- 98% of patients were female
- 42% of these patients on higher than recommended dose.
- QTc prolongation risk factor distribution:

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>GFR &lt;30 ml/min</th>
<th>Hepatic Disease</th>
<th>Hypokalemia or Hypomagnesemia</th>
<th>Pre-existing QTc Prolongation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients on any dose citalopram</td>
<td>4.35%</td>
<td>4.35%</td>
<td>5.80%</td>
<td>1.45%</td>
</tr>
<tr>
<td>Patients on 40mg dose</td>
<td>3.45%</td>
<td>0%</td>
<td>3.45%</td>
<td>3.45%</td>
</tr>
</tbody>
</table>

Concurrent use of interacting medications:

<table>
<thead>
<tr>
<th>Dose</th>
<th>QTc Prolonging Med</th>
<th>Other Interacting Medication*</th>
<th>Loop Diuretic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any Dose Citalopram</td>
<td>28.98%</td>
<td>84.06%</td>
<td>10.14%</td>
</tr>
<tr>
<td>40mg Dose Citalopram</td>
<td>31.03%</td>
<td>82.76%</td>
<td>10.34%</td>
</tr>
</tbody>
</table>

*Includes medications acting on 2CYP19 and CYP3A4. Most common interacting medications statins, PPIs, benzos

Conclusion

- Initial data analysis revealed a substantial number of prescriptions written for a higher than recommended dose of citalopram.
- Of those patients on higher than recommended dosage, very few had appropriate EKG monitoring.
- An educational intervention has been designed in the form of a presentation at resident conference.
- Data will be re-queried several months after educational intervention to determine whether prescribing habits have changed.

References

Hemoptysis: A red flag for pseudoaneurysm after pulmonary artery catheterization

Min Qi DO¹, Keriann VanNostrand MD², Mary Baker MD², William Carlos MD², Farzad Loghmani MD²

¹Indiana University Internal Medicine Department and
²Indiana University Pulmonary and Critical Care Department, Indianapolis, Indiana

INTRODUCTION

• Pulmonary artery catheters (PAC) are routinely used in the diagnosis and management of pulmonary hypertension with minimal risks.
• Pulmonary artery pseudoaneurysm rupture is a rare but potentially fatal complication.

CASE DESCRIPTION

• A 69 year old female with history of diabetes and hypertension was evaluated for 3 months of lower extremity edema, abdominal distension and orthopnea.
• Transthoracic echocardiogram showed diastolic dysfunction and RVSP of ~55mmHg.
• Left heart catheterization was notable for single vessel disease and LVEDP of 27mmHg.
• Right heart catheterization performed under fluoroscopic guidance: pulmonary artery pressure of 56/33 mmHg and wedge pressure of 21mmHg.
• Massive hemoptysis (~300 mL) occurred unexpectedly with removal of PAC.
• Chest CT revealed a right middle lobe pulmonary artery pseudoaneurysm (Fig 1-2).
• Pulmonary angiography confirmed this as the source of hemoptysis (Fig 3).
• Two coils were successfully deployed: one in the pseudoaneurysm and a second coil in the feeding branch (Fig 4).
• Patient subsequently required a transfusion for a 4 gram drop in her hemoglobin.
• She didn’t have any further hemoptysis during her hospitalization and was discharged home.

Table 1

<table>
<thead>
<tr>
<th>Risk factors for pulmonary artery pseudoaneurysm formation:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Chronic steroid use</td>
</tr>
<tr>
<td>• Systemic anticoagulation</td>
</tr>
<tr>
<td>• Age &gt; 60 years</td>
</tr>
<tr>
<td>• Female gender (69% preponderance)</td>
</tr>
<tr>
<td>• Cardiac decompression</td>
</tr>
<tr>
<td>• Cardiac manipulation during surgery</td>
</tr>
</tbody>
</table>

DISCUSSION

• Hemoptysis is the initial presenting symptom suggestive of PA injury in more than 80% of cases.
• For those who survive the initial hemoptysis from a PA rupture, pseudoaneurysm has been reported to occur between minutes to 7 months later.
• The right PA is involved in 93% of cases. A high index of suspicion is required when post-catheterization patients develop hemoptysis.
• Left untreated, risk of re-bleeding from the pseudoaneurysm is 30-40% with mortality rate of 40-70%.

CONCLUSION

• Hemoptysis in patients who have undergone PAC should raise suspicion for pseudoaneurysm formation. The R. PA is involved in 93% of cases.
• Prompt evaluation with imaging is crucial and can be accomplished through contrast-enhanced CT or, time permitting, a CT angiogram.
• Timely recognition and treatment with surgery or coil embolization are necessary to prevent morbidity and mortality.

REFERENCES

Ascaris Lumbricoides

- Most common parasitic infection in the world with 1/6 of population infected
- Uncommon in the developed world
- Spread by fecal oral route
- Complicated life cycle
- Adult worms can reach 35 cm in length
- Pulmonary stage: intense inflammatory response may occur producing Loffler’s Syndrome, an eosinophilic pneumonia secondary to a parasitic infection.

Objectives
- Discussion of typical characteristics of Ascaris Lumbricoides infection
- Review of a case presentation
- Discussion of Loffler Syndrome

Initial Presentation
- 75 y/o male Burmese refugee presented with acute respiratory failure and possible pneumonia
- Long history of heavy smoking and COPD
- Admitted to the MICU, intubated, and treated with empirically for COPD and possible Community Acquired Pneumonia
- Patient developed diffuse pulmonary infiltrates but all cultures remained negative including thoracentesis
- Patients condition improved and he was transferred to the floor after weaning off the vent.
- The medical staff received a call from the floor nurse that she had “removed several feet of iodoform packing from the patient’s rectum while cleaning him after a bowel movement”

Clinical Course
- In consultation with the Infectious Disease physicians, it was felt that this was an Ascaris Lumbricoides infection.
- Infection with possible exacerbation by the recent steroid therapy causing mobilization.
- The patient was placed on Albendazole and received a full course of therapy.
- Developed acute hyper-carbic respiratory failure when therapy was initiated necessitating a return to the MICU and ventilator.
- Bronchoscopy did not show any larval forms.
- Pulmonary and Infectious Disease services believe that this is the likely explanation of his initial presentation.
- The patient was able to return to his home after an intensive ventilator wean at an LTAC facility.

Loffler Syndrome
- Essentially is eosinophilic pulmonary disease secondary to the transit of helminth larval forms through the lung.
- Can be caused by Ascaris lumbricoides, hookworms, and Strongyloides stercoralis.
- Sputum may contain eosinophil-derived Charcot-Leyden crystals.
- Peripheral eosinophilia is usually present but not always (not seen in this case).
- No specific therapy required

Image 1: Ascaris life cycle (http://www.metapathogen.com/roundworm)

Image 2: “Packing foam” found by medical staff upon arrival to bedside.

Image 3: CT chest from this patient showing diffuse pulmonary infiltrates.

Image 4: Charcot-Leyden crystals

Conclusion
- Ascaris Lumbricoides is a common disease outside of the United States
- Loffler’s Syndrome is a well documented pulmonary complication of these parasitic worms
- Some foreign bodies are living!

Reference
Available upon request.

Disclosure
None.
Acquired Factor XIII deficiency due to anti-FXIII antibodies is a rare but life-threatening bleeding disorder. Factor XIII is a fibrin stabilizing enzyme which crosslinks fibrin monomers. Deficiency of Factor XIII results in destabilization of formed clots within 24-48 hours, resulting in delayed hemorrhage. Because the standard coagulation tests are normal, the diagnosis of this disease requires a high degree of suspicion and specialized testing. Here, we report a case of an 88-year-old female presenting with severe hemorrhage of unknown origin.

**CASE DESCRIPTION**

An 88-year-old female with a recent diagnosis of autoimmune hemolytic anemia on oral Prednisone developed left arm swelling, pain, and ecchymosis. CT scan of the arm showed a biceps muscle hematoma measuring 17cm in length. On the 4th day, she developed a rapidly evolving hematoma on the contralateral forearm, which prompted bilateral fasciotomies and evacuation. Patient continued to have bleeding from her right forearm hematoma, requiring further exploration and evacuation of multiple clots. Her Hg dropped from 9.2 g/dl to 6.6 g/dl and she needed packed RBC transfusions. Patient had a normal coagulation profile including PT, PTT, Fibrinogen and Thrombin Time. Peripheral blood smear revealed features of chronic hemolysis.

Patient’s Factor VIII, IX and X levels were normal. Platelet function checked by Platelet Function Analyzer, VWD antigen and assay were also unremarkable. Subsequently, patient’s Factor XIII levels came back low at 8%. We suspected this to be a case of acquired factor XIII deficiency as patient lacked any history of excessive bleeding. Testing for inhibitor with serial dilutions showed an antibody titer of 1:40. Patient was started on 100mg of Cyclophosphamide and continued on 20mg of Prednisone for inhibitor eradication. On day 66, patient had AIHA flare up (Hg: 6.6g/dL) and was started on Rituximab 375mg/m² for 4 infusions and her prednisone was increased to 40mg due to AIHA flare up. On day 98, IVIG was started due to increasing inhibitor levels and ecchymosis.

**PROPOSED MANAGEMENT**

Acquired FXIII deficiency due to anti-FXIII antibodies is a rare bleeding disorder that can cause moderate to severe bleeding and carries a significant mortality rate. This case illustrates the clinical paradigm that if a patient presents with bleeding symptoms in the setting of normal routine coagulation tests, the diagnosis of acquired FXIII deficiency should be considered. Prompt characterization of Factor XIII activity and inhibitor level is essential in order to provide the most appropriate therapy for inhibitor eradication and control of hemorrhagic complications.

**REFERENCES**

The Cost of A Cardiac Marker: It’s Enough to Give You Chest Pain

Stephen J. Schutzman, MD and Victor Collier, MD
Saint Vincent Hospital, Indianapolis, Indiana

The Question
The practice of ordering “serial cardiac markers” is ingrained in our practice culture. Inpatient and ER physicians frequently order cardiac markers for symptoms in and around the chest without considering the pre-test probability of acute coronary syndrome. This often leads to confusion in interpreting results, additional testing and cost. This leads us to question the cost and clinical value of the standard “cardiac marker” panel.

The Background
Cardiac markers have been conventionally defined as a Troponin along with CKMB or CK Panel (Total CK and CKMB). A cardiac marker series is held as three separate testing points usually at six to eight hour intervals. This is done as the laboratory values are known to peak hours after injury has occurred. Historically CK and CKMB were the first identified possible markers of cardiac tissue injury. These markers however were not specific enough to the cardiac tissue and have since been replaced by Troponin as the primary marker of cardiac tissue injury. This replacement with Troponin as the standard is evident in our clinical conversations. Discussion amongst physicians concerns “what was the troponin” and does not include the other laboratory values. Despite the shift in how clinical decisions are made CKMB Panels have remained as part of the “serial marker” set.

The Analysis
Saint Vincent Hospital, Indianapolis is a 700 bed inpatient facility located in Central Indiana. The facility is the tertiary hub for a 16 hospital system covering central Indiana. Laboratory and billing information were reviewed for a 6 month period. The laboratory processed 11,901 Troponins and 11,349 CKMBs during this period. Inpatient billing charges for both of these labs were: $323.00 Troponin and $194.00 CKMB. Thus for a 6 month period $6,045,729.00 in charges were created for cardiac markers. Of this total $2,201,706.00 of charges would have been from CKMB in only a 6 month time frame.

<table>
<thead>
<tr>
<th>Test</th>
<th>Patient Cost</th>
<th>Total Ordered</th>
<th>Total Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin</td>
<td>$323.00</td>
<td>11,901</td>
<td>$3,844,023</td>
</tr>
<tr>
<td>CPT Code: 84484</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CKMB</td>
<td>$194.00</td>
<td>11,349</td>
<td>$2,201,706</td>
</tr>
<tr>
<td>CPT Code: 82550, 82552</td>
<td></td>
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</table>

Table 1: 6 month totals for cardiac markers at St. Vincent Indianapolis and associated total charges

Conclusion
In the era of increasing cost conscience medicine and possible capitated care models providing high value care is going to be the gold standard of the future. One of the easiest places to improve care towards this goal is to examine practices that occur as part of a culture but do not actually influence clinical care or decision. Elimination of the CKMB from the standard cardiac marker series could eliminate as much as 4.5 million dollars of charges to patients in a year’s time at one facility alone. The examination of the use of this one lab, on a national scale, could represent significant savings.

CKMB’s may provide insight into the timing of the injury that can provide clinical guidance in certain circumstances. The lab should be available as an opt-in as opposed to the current opt-out.

Call To Action
- It is important for providers to be aware of testing costs.
- Pre-populated order sets while efficient can perpetuate charge heavy medicine.
- **ASK THE QUESTION:** Will this change my management?

Limitations
- We looked at total billable charges and not those that were reimbursed and we also did not look at the charge to the hospital.

References available upon request