

# Abstracts from the Louisiana American College of Physicians Associates Meeting

Each year medical students from the 3 medical schools in Louisiana and residents from the six Internal Medicine training programs in Louisiana are invited to submit abstracts for the annual Louisiana American College of Physicians (ACP) Associates meeting. The content of these abstracts includes clinical case vignettes or research activities. The abstracts have all identifying features removed (i.e., names, institutional affiliations, etc.) before being sent to three physician judges who are not directly affiliated with the medical schools or training programs. Each judge scores each abstract independently and then the scores from the three judges are averaged and ranked. This year we are excited to be able to publish the 23 most highly ranked abstracts in this year's competition. These abstracts (12 oral; 11 poster) were presented at the Ochsner Clinic Foundation in New Orleans on Thursday, January 19, 2006. We would like to thank the *Journal of the Louisiana State Medical Society* and appreciate its efforts to publicize the efforts of these young trainees.

Frank Incaprera, MD and Fred A. Lopez, MD  
Co-Chairs, Louisiana Associates Liaison Committee

Sandy Kemmerly, MD  
Governor, Louisiana ACP

### ORAL PRESENTATIONS

#### A Case of Systemic Inflammatory Response Syndrome Due to *Vibrio cholerae* O1 serotype Inaba in a Patient with a History of Hypertension and Diabetes Mellitus.

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Cases of cholera are rarely seen in Louisiana, but an increased number of all types of *Vibrio* infections have begun to appear along the gulf coast in the immediate period following Hurricanes Katrina and Rita in the autumn of 2005. We report a 43-year-old man who developed severe diarrhea for three days, followed by a one day period of nausea and vomiting. He and his wife reported the consumption of boiled shrimp approximately five days prior to the onset of symptoms. He also reported wading through waist-deep water that was associated with Hurricane Rita's storm surge one week prior. On admission he was weak and volume depleted. He was noted to have an anion gap metabolic acidosis that required mechanical ventilation. Intravenous volume replacement was instituted, along with broad-spectrum antibiotic coverage. Despite aggressive fluid administration, the patient subsequently suffered from hypovolemic shock and acute renal failure. Elevated creatine phosphokinase accompanied by myalgias indicated rhabdomyolysis. Although the patient underwent repeated boluses of intravenous fluids, his blood urea nitrogen and creatinine levels continued to rise. Hemodialysis was initiated due to metabolic acidosis and electrolyte

abnormalities unresponsive to medical management. The patient met requirements for, and was administered the indicated dose of drotrecogin. Isolation of *Vibrio cholerae* O1 serotype Inaba from his stool confirmed the diagnosis of cholera, and he was switched to ciprofloxacin. The patient's condition stabilized and following extubation, he was transferred from the intensive care unit to the medicine ward. His diarrhea and vomiting completely resolved. The patient was discharged, and he is currently undergoing hemodialysis three times weekly due to lingering effects from suspected hypovolemia-induced acute tubular necrosis. Cholera is a serious condition that can be lethal if untreated. Treatment with fluids to offset the large volume loss is essential to prevent multisystem organ failure and potential death.

#### An Unfamiliar Presentation of Mycobacterium Tuberculosis.

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A 33-year-old Vietnamese man with a history of HIV (CD4+ 181) presented with a six-month history of intermittent infraumbilical pain. He admitted to a thirty-pound weight loss, night sweats, fatigue and constipation. Vital signs were stable except for a mild temperature increase of 99.6° F. Abdominal exam was normal and no lymphadenopathy was appreciated. Upon initial workup, the PPD yielded a 17 mm reaction at forty-eight hours. The chest x-ray was normal and the patient denied pulmonary symptoms. Abdominal pain persisted and a small bowel follow through was performed which showed multiple small round lesions at the terminal

ileum. A colonoscopy then revealed 4-mm lesions throughout the colon as well as granulomatous type lesions concentrated in the small bowel and proximal colon. Biopsies of the terminal ileum and proximal transverse colon were determined to be *Mycobacterium tuberculosis*. The patient was then started on RIPE (rifampin, isoniazid, pyrazinamide, ethambutol) therapy.

One day after starting therapy, the patient developed acute mental status changes, spiked a temperature to 103° F, and became hypotensive and tachycardic. A lumbar puncture was performed which showed a lymphocytic pleocytosis with a normal glucose (WBC 44; L- 94, M-5, E-1; total protein 187; glucose 58). Although the findings were consistent with tuberculous meningitis, stains and cultures did not confirm this diagnosis. The patient was fluid resuscitated in the ICU and mental status returned to baseline prior to hospital discharge. After RIPE therapy for two months and rifampin and INH for an additional twenty-two months, his weight increased from 145 lbs to 200 lbs. Currently the patient is much improved clinically with no complaints.

The epidemiology of *Mycobacterium tuberculosis* has changed dramatically with the HIV epidemic, most likely on the basis of a reduced TH1 response. In addition to having HIV, this patient is also an immigrant of low socioeconomic status which adds to his TB risk factors. Of greater interest is the presence of TB within the GI tract and presumably CNS without evidence of pulmonary involvement. Although the classic mechanism for disseminated tuberculous infection is considered to be hematogenous spread from pulmonary or miliary TB, three other mechanisms are plausible: swallowing of infected sputum, ingestion of contaminated food, or contiguous spread from adjacent organs. It is recognized that a component of HIV gastropathy is hyposecretion of gastric acid. If one accepts the swallowing TB-infected sputum as the inciting pathogenetic event and then superimposes a low pH state on the basis of HIV gastropathy with secondary achlorhydria, one could postulate that multiple mechanisms of disease worked in at least an additive manner to lead to the tuberculous enteritis experienced by this patient.

#### M.R.E. (Meals Ready to Exacerbate).

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**Learning Objectives:** 1. To understand the importance of sodium restriction in the setting of CHF. 2. To consider the balance of neurohormonal interactions associated with CHF.

**Case:** A 77-year-old man with a history of congestive heart failure presented with one week of dyspnea and lower extremity edema. The dyspnea was exacerbated with activity and associated with orthopnea and a non-productive cough. He had evacuated temporarily from New Orleans to Lake Charles, Louisiana after Hurricane Katrina. Upon his return to New Orleans he has been receiving food supplies from the National Guard in the form of Meals Ready to Eat (MRE). His diet has consisted of primarily MREs up to three times a day. He had a BP130/57, P62, and R22. He had a JVP of 7 cm, bibasilar crackles and 2+ edema bilaterally in his lower ex-

trimities. A chest radiograph revealed cardiomegaly with bilateral basilar infiltrates. Laboratory studies included an initial BNP of 570, Troponin of 0.04, hemoglobin of 15 g/dl, and normal electrolytes. Diuresis led to resolution of his symptoms. Upon discharge he was instructed to adhere to a low-salt diet. The importance of avoiding regular consumption of MREs was specifically discussed.

**Discussion:** Acute exacerbations of congestive heart failure should be expected following major disasters. As our patient illustrated, evacuees are often relegated to either canned foods or MRE meals, both of which are sodium rich. One MRE contains 2.3 g of sodium, and the consumer may opt to add flavor with the included salt package to increase the sodium content by an additional 4g. MRE's are designed for active soldiers who have healthy cardiovascular systems and subject to sodium loss due to sweating during vigorous activity.

Distribution of bulk MRE's in post-disaster and emergency situations is an important component of immediate relief operations. However, the lack of effective labeling and consumer information can occasionally lead to negative consequences. Distribution of easy-to-understand content information and recommendations regarding certain health conditions along with the public disaster relief MRE packaging would be an excellent modification.

#### *Nocardia* Brain Abscesses in a Patient with Lupus Nephritis.

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*Nocardia* species are aerobic, gram positive, partially acid-fast bacilli found in plants and soil. They cause cutaneous, pulmonary, CNS or disseminated infections, though usually more invasive in immunocompromised hosts.

A 37-year-old man with a history of SLE and lupus nephritis on corticosteroid and cyclophosphamide therapy presented to our medical center with complaints of pain in his left shoulder. Radiographs showed an anteriorly dislocated humeral head that was reduced in the ER. However, on physical exam, the patient was febrile and had an indwelling dialysis catheter. He had been admitted a month prior with anasarca, at which time he developed sepsis and left leg cellulitis. Blood cultures at that time grew out *Pseudomonas* and *Streptococcus bovis* and he was successfully treated with IV clindamycin, piperacillin/tazobactam and levofloxacin. He had also been seen at another hospital two weeks prior for new onset seizure-like activity and slurred speech at which time he was started on phenytoin.

An MRI performed during this admission showed multiple ring-enhancing lesions in the right frontal, right occipital and left temporal lobes located at the gray-white junction suspected to be infectious based on diffusion-weighted imaging. Lumbar puncture was not performed second to mass effect and focal neurological findings and repeated blood cultures were negative. The patient did not respond well to empiric therapy with IV tobramycin, metronidazole and cefepime, and therefore underwent open craniotomy and excisional brain biopsy. A fungal culture from the biopsy grew

out *Nocardia asteroides complex* 12 weeks after biopsy. At this time, the patient was receiving home IV therapy with cefepime and metronidazole, with stabilization of the lesions, but continued problems with nausea and vomiting as well as some residual neurological deficits. The patient was readmitted for worsening nausea and vomiting six months after initial presentation, at which time a chart review revealed the positive *Nocardia* culture and he was started on IV TMP/SMX, with good clinical response. Follow-up MRI two months after starting TMP/SMX therapy showed stable encephalomalacia of the affected areas with no abnormal contrast enhancement.

This case illustrates the need to consider atypical organisms in severely immunocompromised individuals. In particular, the suppression of macrophage activity and activation by steroids causes increased susceptibility to *Aspergillus*, *Nocardia*, *Listeria* and *Salmonella*. In addition to influencing selection of empiric antibiotics, it is important to follow-up any pending cultures in these patients for much longer than routine cultures, particularly when they are sent out to other institutions.

#### **An Unusual Cause of Syncope and Cardiac Conduction Abnormalities.**

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**Case:** A 53-year-old male presented with a seven month history of syncopal episodes associated with chest pain and diaphoresis. After his last episode, the patient was taken to the emergency department where telemetry revealed ventricular tachycardia. At that time, he underwent defibrillation and developed complete heart block. An emergent echocardiogram revealed a golf ball sized mass in the interatrial septum extending into the posterior wall of the aortic root. The patient was subsequently transferred to a tertiary care center for further medical management. Upon arrival, the patient was in complete heart block with a heart rate in the 40's requiring a temporary pacemaker. A bedside echocardiogram showed global hypokinesis with an ejection fraction of 30%, moderate aortic insufficiency, and a large immobile non-homogenous echodensity partially involving the atrial septum, atrioventricular groove, and aortic annulus. A cardiac MRI was obtained to further delineate the echodensity. It revealed a high signal mass circling the right posterolateral aspect of the aortic valve root with an area of flow suggesting a Sinus of Valsalva aneurysm 2.99 cm in size with a perianeurysmal hematoma extending (6.2 cm) between the right and left atrium. Angiography demonstrated a Sinus of Valsalva aneurysm of the noncoronary cusp, severe aortic insufficiency, normal coronaries, and no chamber communication. Cardiothoracic surgery evacuated the Sinus of Valsalva aneurysm and repaired the septal dissection. Additionally, the patient required aortic valve replacement and reconstruction of both the left ventricular outflow tract and aortic root.

**Discussion:** Sinus of Valsalva aneurysms are rare cardiac anomalies whose incidence is higher in far eastern countries than western countries. They may be acquired or congenital and most often originate from the right Sinus of Valsalva.

Furthermore, only 10-30% originate from the noncoronary sinus and less than 5% from the left sinus. Unruptured Sinus of Valsalva aneurysms are less frequently reported because they are typically clinically silent. They can however result in pulmonary outflow obstruction, conduction disturbances, and sudden death. This case illustrates the need for prompt diagnosis of Sinus of Valsalva aneurysms as progressive aneurysmal dilatation or rupture can lead to death without prompt surgical intervention.

#### **Diabetic Ketoacidosis without Ketones: Quest for the Missing Anion.**

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**Introduction:** Diabetic ketoacidosis (DKA) has been described classically as a triad of ketosis, hyperglycemia, and high anion-gap metabolic acidosis (HAGMA). However, DKA may develop in the absence of measurable ketones in the serum and urine.

**Case:** A 46-year-old man with diabetes mellitus on sulfonylurea therapy, coronary artery disease, and severe peripheral vascular disease was admitted for worsening pain and erythema over an ulcer on the left foot. He also reported post-prandial abdominal pain. He denied consumption of toxic alcohols. Physical examination revealed no fever or abdominal tenderness. The left foot was cold and pulseless, with an ulcer over the fifth toe. The hemoglobin was 8 gm/dL; white blood cell count 13,000/mm<sup>3</sup>. Serum chemistry values included: glucose 461 mg/dL; bicarbonate 10 meq/dL; potassium 5.8 meq/dL; blood urea nitrogen 46 mg/dL; creatinine 2.4 mg/dL; amylase 242 u/L; lipase 1044 u/L; and lactic acid 1 mmol/L. The anion gap (AG) was 13. Serum ketones were not measured. Urine analysis revealed pH 5 without ketones. He was treated with intravenous fluids, bicarbonate, and regular insulin on a sliding scale. Arterial blood gas analysis the next day revealed: pH 7.24; PCO<sub>2</sub> 21 kPa; and HCO<sub>3</sub> 9 meq/L. Serum ketones were absent at that time. Over the next seven days, the serum creatinine decreased, but hyperglycemia and normal anion gap acidosis (NAGMA) persisted. The urine AG was +10 with pH 5 and osmolality 422mOsm/kg. Serum and urine ketones were then detectable. Subsequently, he was treated with high dose insulin for DKA and antibiotics for a diabetic foot infection, and all laboratory parameters normalized. He later underwent below the knee amputation.

**Discussion:** In retrospect, we think this patient had DKA at presentation. In the presence of infection and tissue hypoxia, the predominant ketoacid is beta hydroxy butyrate (BHB). The nitroprusside reaction commonly used in testing for ketones does not detect BHB. The positive urine AG in the presence of NAGMA in this patient was likely fallacious, since the urine pH suggests that there was no renal acidification defect. The presence of ketones in the urine invalidates the concept of the urinary AG, which assumes that chloride is the major anion. This case illustrates that the absence of serum and urine

ketones by the nitroprusside reaction does not rule out DKA and that acid-base disorders in DKA may include both HAGMA and NAGMA. It also shows the limitation of the urinary AG in determining the presence of renal tubular acidosis in association with DKA.

### Primary Mediastinal Liposarcoma.

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Primary mediastinal liposarcoma is an uncommon neoplasm of intrathoracic origin. It is usually encountered when the tumor has become large and symptomatic due to mass effect.

A 24-year-old male presented to our ER with severe dyspnea, particularly when laying supine. Chest radiograph revealed marked mediastinal widening and a nearly obliterated right lung field. Upon further questioning, patient was noted to have been successfully treated with surgical excision and chemotherapy for an embryonal rhabdomyosarcoma of the left lower extremity at age 10. He had continued follow-up with his pediatric oncologist annually until age 20. His family history was also significant for a father who died at age 36 of rhabdomyosarcoma originating in the neck. After hospitalization, CT of the thorax revealed a large low-density mass invading the mediastinum and right pleural space, occupying much of the right hemithorax. CT guided biopsy of the lesion revealed myxoid liposarcoma. As he was considered a poor surgical candidate due to the extent of his disease, the patient was initiated on chemotherapy. He underwent 3 months of therapy with modest improvement, but unfortunately succumbed to his cancer.

Mediastinal liposarcomas tend to vary histologically from myxoid and well-differentiated to dedifferentiated. Surgical excision appears to be the treatment of choice with smaller tumors, with chemotherapy and radiation therapy as adjuncts. Second primary soft tissue sarcomas, as this patient had, are also quite unusual, very rarely observed in patients without prior radiation therapy. Recent literature suggests that patients treated for a primary soft tissue sarcoma had a 0.2% chance of developing a second primary soft tissue sarcoma. However, there was a higher preponderance amongst those diagnosed with their first primary tumor at a younger age or with a strong family history such as that found in Li-Fraumeni syndrome. This patient highlights the possibility of the need for continual surveillance for second malignancy in those with childhood sarcomas.

### A Rare Case of Extra-nodal, Multicentric Castleman's Disease with Unusual Clinical Behavior.

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**Introduction:** Castleman's disease (CD), also known as angiofollicular hyperplasia, is an unusual benign lymphoproliferative disorder first described in 1956. The patho-

genesis is unknown. Typically, it occurs in the mediastinal lymph nodes, though cervical, retroperitoneal, and mesenteric nodes may be affected also. Systemic symptoms are usually present. Extra-nodal occurrence is rare. We report a patient with CD involving the subcutaneous tissue of the abdomen and right arm without systemic symptoms.

**Case:** A 73-year-old man presented with subcutaneous masses in his abdomen and on the posterior aspect of his right arm. These were slow growing and were associated with intermittent vague cramping abdominal pain. He denied fever, chills, night sweats, anorexia, or weight loss (typical B symptoms). On examination, two ill-defined masses, measuring 3x3 cm each, were noted. These were firm, non-fluctuant, non-tender, and mobile over underlying structures. His complete blood count and other basic laboratory studies were normal. A biopsy of the lesions was performed. Microscopic examination of the tissue demonstrated superficial and deep lymphoid infiltration of skin and blood vessel walls with diffuse spread of eosinophils. The findings were suggestive of CD of the hyaline vascular type. Computerized tomographic scans showed that the lesions were limited to the two subcutaneous foci. The masses were resected and have not recurred.

**Discussion:** Multicentric CD is a rare entity that typically results in "B" symptoms (driven by interleukin-6 overproduction), widespread lymphadenopathy, and hepatosplenomegaly. The condition may be associated with autoimmune hemolytic anemia, multiple myeloma, amyloidosis, pemphigus, human immunodeficiency virus infection, and herpesvirus 8 infection. There was no evidence of any of these in this patient. Multicentric CD may be classified histopathologically as hyaline vascular, plasmacytic, or mixed cellularity type. Though benign, it may have an aggressive course, and can progress to non-Hodgkin's lymphoma. In contrast, unicentric CD, which is usually the hyaline vascular variety, is slow growing and confined to the mediastinum or mesenteric lymph nodes. The only symptoms are from the mass effect of bulky lymphadenopathy, and there is no elevation of acute phase reactants. Resection is curative in over 90% of these.

**Conclusion:** This case had the typical clinical characteristics of unicentric CD, but was multicentric and extranodal in distribution. It was also unusual in that systemic manifestations were absent. Castleman's disease should be a consideration in undiagnosed lymphadenopathy, with or without systemic symptoms.

### Yet Another Reason Not to Smoke Crack.

D Victor, C Miller. Tulane Health Sciences Center.

**Learning Objectives:** 1. Interpret unusual EKG findings in a patient with heart disease. 2. Understand the clinical presentation of an ICD lead displacement.

**Case:** A 58-year-old man with ischemic heart disease presented with multiple firings of his recently implanted cardioverter-defibrillator (ICD). Fifteen minutes after smoking crack cocaine, he experienced six to seven shocks to his

chest. A reading of his chest x-ray demonstrated the coiling of the leads around the pulse generator with dislodgement of the ventricular lead. Interrogation of the device revealed no significant cardiac events. The defibrillator function was manually turned off. Two nights after admission, he began to complain of persistent hiccups and a feeling of his "heart beating in his stomach". On exam, he was noted to have repetitive abdominal heaving. An EKG showed aberrant pacer spikes that did not correspond with his cardiac rhythm. The spikes, however, corresponded to his rhythmic hiccups. The placement of a magnet over the ICD brought the hiccups to an immediate halt. The pacer function was disabled the following morning.

**Discussion:** Twiddler's Syndrome, first described in 1968 by Bayliss, is the permanent dysfunction of a cardiac pacemaker secondary to the spinning of a patient's pulse generator in the chest wall. The reeling of the pacemaker leads can result in stimulation of the phrenic nerve causing abdominal pacing as in our patient. Further coiling can lead to stimulation of the brachial plexus and pectoral muscles. Sudden cardiac death has been documented and is the most concerning feature of this syndrome. Not only is the patient at risk because the ICD may not be capturing, but there is also potential to precipitate a fatal arrhythmia through aberrant firing. Both features, cardioversion and pacing, need to be disabled when leads become dislodged. The incidence of Twiddler's Syndrome is estimated to be between one to seven percent in patients with ICDs and is more common in the elderly because of laxity of the subcutaneous tissues. The treatment is the surgical replacement of pacer leads and anchoring of the generator to the underlying fascia. A Dacron patch that promotes tissue in-growth has been advocated for all ICDs, especially cases of repeat lead placement and high-risk patients. With the increasing placement of ICD's, it is likely that Twiddler's syndrome will be increasingly encountered by the general internist.

**References:** 1. Bayliss CE, Beanlands DS, Baird RJ. The pacemaker-twiddler's syndrome: a new complication of implantable transvenous pacemakers. *Can Med Assoc J* 1968;99:371-3. 2. Nicholson, WJ.; Tuohy, KA.; Tilkemeier, P. Twiddler's Syndrome. *NEJM* Vol 348,2003;726-1727. 3. Mohsen S, Inbar S, Neckels B, et al. Twiddling to the Extreme. *J of Invasive Cardiology* 2005;17:195-196.

#### **Increased Phenotypic Expression in an LRP5 Gain of Function Mutation in an African-American Female.**

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**Introduction:** The LRP5 mutation has added new insight into the way endocrinologists and cell biologists look at bone. To date there has only been one African-American who has presented with the gain of function mutation. This gain of function mutation causes increased bone mass density, and in this case increased phenotypic expression.

**Case:** A 49-year-old African American female was referred to endocrinology clinic for elevated bone mineral density (BMD). The patient had been blind in the left eye since the age of 2 secondary to bone overgrowth affecting the optic nerve. At the age of 3 skull surgery was performed to relieve intracranial pressure. Acromegalic features were first noted in 1994. The patient had a history of osteopetrosis of the knee.

On physical exam the vital signs were normal. Examination was notable for obesity, and facial abnormalities; the chin was prominent and asymmetric with the right side larger than the left. Eye examination was significant for left sided blindness and strabismus. Oral examination revealed widely spaced teeth, a palatine tonsil, and maxillary gingival hyperplasia. There was also a 1.5 cm nodule in the occipital area, and an enlarged thyroid gland. Acanthosis nigricans was present. By inspection, the extremities appeared enlarged.

Significant laboratory values included the following: Alk Phos- 71 U/L, N- Telo peptide- 9.4 nmol/mmol creat (8.7-19.8), FSH- 10mU/mL. Radiographs showed bony sclerosis of the ribs, thick cortices of the long bones, and coarse trabeculae about the elbows, sclerosis about the knees and widening of the metatarsals. The cervical spine showed osteosclerosis and neural foramenal narrowing of C3-4 and C4-5. The skull radiograph showed a very sclerotic and thickened mandible and calvarium. The orbits were small and misshapen. There were no maxillary, mastoid, or ethmoid sinuses. Radionuclide bone scans showed a pattern of degenerative changes, and increased uptake of the mandible. BMD T-score and Z-score were 7 standard deviations above the reference. DNA studies identified LRP5 gain of function mutation.

**Discussion:** The LRP5 mutation was discovered in 2001 and 2002. The LRP5 gene is expressed in nearly all tissues and carries out various biologic processes. It is a cofactor in the Wnt pathway. This pathway regulates cellular activity including cell fate determination, proliferation, migration, and gene expression. Loss of function LRP5 mutations cause decreased bone formation and a condition known as osteoporosis-pseudoglioma syndrome, which affects bone and vision. The gain of function mutation leads to high BMD. Various other phenotypes of the mutation have been reported including sclerosis of the skull, macrocephaly, mandibular buccal exostoses and lingual exostoses. This is the first African-American to present with the LRP5 mutation. Her severe phenotypic findings, including osteopetrosis, separate her from other patients with LRP5 mutations. Other patients with this mutation have presented with relatively benign phenotypic manifestations. The LRP5 mutation discovery has added to the understanding of metabolic bone disease, and provides a new therapeutic target.

#### **Rapid Neuromyopathy Following Colchicine Administration in a Patient Receiving Chronic Simvastatin.**

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Both 3-hydroxy-3-methyl-glutaryl coenzyme A (HMG-CoA) reductase inhibitors and colchicine have been associated with

drug-induced myopathy. The time to development of myopathic symptoms with some HMG-CoA reductase inhibitors varies; however, reports of colchicine induced myopathy typically occur over several years following its initiation. We report a case of colchicine-associated myopathy fourteen days after its initiation.

A 61-year-old woman developed four days of progressive lower extremity weakness and inability to walk after taking colchicine for gout of the spine. Physical exam revealed absent deep tendon reflexes and two over five motor strength of her hip flexors. Her creatine phosphokinase (CPK) was measured at 4927 U/L, and her creatinine increased from 1.3 mg/dL to 2.2 mg/dL. Simvastatin was initially thought to be the cause; however her CPK continued to rise despite intravenous hydration. Following review of her medications, colchicine was discontinued. Her CPK then trended down and with rehabilitation she returned to her previous neuromuscular status. Electromyography suggested a drug-induced myopathy. The co-administration of colchicine and simvastatin therapies may have heightened her risk of neuromyotoxicity. There are limited reports of early myopathy in patients prescribed simvastatin therapy and subsequently given colchicine. The CPK elevation was ten to twenty times the upper limit of normal, consistent with previously reported colchicine myopathy. The metabolism of both colchicine and simvastatin by the CYP3A4 (part of cytochrome P450) pathway may overwhelm the cytochrome thus delaying the metabolism of drugs contributing to toxicity. This along with the subsequent renal elimination of colchicine in the presence of worsening renal function can further exacerbate the toxicity of these drugs. Patients who have been stable on simvastatin then started on colchicine should be carefully monitored and educated about the neuromyopathic consequences of this drug interaction.

### Chronic Mesenteric Ischemia Presenting as Postprandial Abdominal Pain.

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Patients suffering with chronic mesenteric ischemia usually present in their sixth or seventh decade and usually after symptoms have been present for 14-18 months. In the majority of cases, the patient has already undergone an extensive evaluation for long standing weight loss and abdominal pain.

A 64-year-old white man with a history of type II diabetes mellitus and tobacco use presented to the emergency room for the evaluation of abdominal pain that had been present for the previous six months. Patient described the pain as being non-radiating, dull, aching and located over the epigastrium that occurred approximately twenty minutes after meals. He denied any dysphagia, nausea, emesis, diarrhea, melena, hematochezia or history of peptic ulcer disease. Physical exam was pertinent for mild bilateral temporal wasting and bilateral carotid bruits. Rectal exam revealed heme positive brown stool. The patient was admitted to the medicine service and had an esophogastroduodenal endoscopy

which revealed antral gastritis, a small hiatal hernia as well as a 3mm pyloric ulceration without visible signs of bleeding. Colonoscopy revealed a 3cm white ulceration at the hepatic flexure. The patient was then referred to IR for an angiogram which revealed atherosclerotic changes in the aorta, stenosis of the celiac trunk, 95% stenosis of the celiac artery, 95% stenosis of the superior mesenteric artery and a 100% occlusion of the inferior mesenteric artery. A diagnosis of chronic mesenteric ischemia was established and stents were placed in the superior mesenteric artery and the celiac artery. Post procedure the patient reported great relief from his abdominal pain and was discharged from the hospital pain free.

This case illustrates how the diagnosis of chronic mesenteric ischemia should be made. According to the 2000 American Gastrointestinal Association technical review on intestinal ischemia, diagnosis "is based upon clinical symptoms, arteriographic demonstration of an occlusive process of the splanchnic vessels, and to a great measure, exclusion of other gastrointestinal disorders."

## POSTER PRESENTATIONS

### A Hole in my Heart.

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**Learning objectives:** 1. Understand the embryological physiology of the atria. 2. Recognize the clinical presentation and treatment of atrial septal defects in adults.

A 62-year-old woman with a history of stroke presented with one day of sub-sternal chest heaviness, dyspnea and palpitations. Upon admission, her heart rate was 160 bt/min. with an irregularly irregular pulse. She had a fixed split S2. The remaining examination was normal. Telemetry revealed atrial fibrillation. Owing to the split S2, an echo was obtained that revealed normal systolic function but an aneurysmal atrial septum, and an atrial septal defect with left-to-right shunt. A diagnostic cardiac catheterization revealed a pulmonary-to-systemic (P-to-S) flow ratio of 1.5/1 with mildly elevated pulmonary artery pressures.

A 12mm atrial septal defect was closed by a percutaneous ASD closure device. She declined radiofrequency ablation and was started on long-term warfarin regimen. She was placed on clopidogrel for six months, and aspirin 81 mg for life.

**Discussion:** Atrial septal defect (ASD) is a congenital heart defect caused by the improper closure of the ostium secundum during embryogenesis. Clinically significant ASD's are not rare, occurring in six percent of the population. Symptoms usually begin after the fourth decade and include fatigue, exercise intolerance, dyspnea, or overt heart failure. The incidence of atrial arrhythmias caused by atrial enlargement increases with age to 80% for those over the age of 60 years. Other symptoms include paradoxical emboli and cryptogenic stroke due to gradual right-to-left shunting and concurrent atrial septal aneurysms. The presence of a fixed split

S2 in the setting of these symptoms should prompt physicians to investigate the diagnosis of an ASD. Echocardiography with saline contrast bubbles is the test of choice, successfully diagnosing ASD with a 90-100% sensitivity. Closure of the defect is recommended for a P-to-S flow ratio greater than 1.5/1 to 2/1.

### Post-Ambulatory Oxygen Desaturation as a Predictor of Asthma and COPD Relapse in the ED.

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**Objectives:** Resting pulse oximetry has been used to predict the need for hospital admission in patients with acute asthma and chronic obstructive pulmonary disease (COPD) exacerbations, but the use of post-ambulatory oxygen desaturation as a predictor of Emergency Department (ED) relapse rates has not been studied. The objective of this pilot study was to determine if post-ambulatory oxygen desaturation could be used to predict which patients with asthma or COPD exacerbations discharged from the ED would return within two weeks or four weeks with a relapse.

**Methods:** This was a retrospective chart review of consecutive adult patients with an asthma or COPD exacerbation, and a documented post-ambulatory pulse oximetry prior to discharge from the ED.

**Results:** A total of 250 charts were reviewed, and of those 62 patients were identified that met inclusion criteria. The mean age was 43 years, 50 percent were male, 52 (83.9 percent) were diagnosed with asthma, and 10 (16.5 percent) with COPD. Post-ambulatory desaturation of any amount occurred in 37 (59.6 percent) patients, and desaturation of 3 or more occurred in 9 (14.5 percent). Overall 15 (24.1 percent) patients returned to the ED with a relapse, and of those, 7 (46.6 percent) experienced a post-ambulatory desaturation of any amount and 3 (20 percent) experienced a desaturation of 3 or more. There was no statistically significant relationship between post-ambulatory desaturation and relapse rates.

**Conclusion:** In this pilot study, post-ambulatory oxygen desaturation could not be used to predict ED relapse rates in patients with asthma or COPD.

### Cutaneous Manifestations of Histoplasmosis.

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Cutaneous manifestations of histoplasmosis are an unusual presentation of *Histoplasma capsulatum* infection seen most frequently in AIDS patients. It is a progressive extrapulmonary infection due to hematogenous dissemination occurring during the acute phase of the infection. Disseminated disease occurs in approximately 1 in 2000 acute infections; however, it has been observed more frequently in immunocompromised patients.

We present a case of a 48-year-old male with HIV and AIDS since 2001 who presented to our clinic with a 2 month history of a diffuse, pruritic papulo-nodular rash. He was also noted to have a 1 month history of fever, dyspnea, and cough productive of yellow sputum. Upon admission to our hospital, physical exam revealed 0.5 – 1 cm erythematous papulo-nodular lesions with numerous excoriations confluent over the face, neck, and distal extremities. His laboratory analysis showed a WBC  $13 \times 10^3$ /uL, HGB 10.4 gm/dL, HCT 31.8 %, PLT 220 103/uL, MCV 79.7 fl, Seg Neut 56 %, Bands 8 %, Lymph 25 %, Mono 8 %, Eosi 2 %, Baso 1 %; Na+ 132 mmol/L, K+ 4.0 mmol/L, Cl- 100 mmol/L, CO2 26 mmol/L, Ca2+ 9 mg/dL, Total Protein 7.9 gm/dL, Alb 3.0 gm/dL, Glob 4.9 gm/dL, Total Bili 0.8 mg/dL, AST 21 U/L, ALT 15 U/L, and Alk Phos 84 U/L. CD4 count was 7 cells/mm<sup>3</sup> and HIV viral load was 78,600 copies/mL. Plain chest radiograph revealed a small right upper lobe cavitory lesion. Serum antibody and urinary antigen for *Histoplasma capsulatum* were negative, as well as blood cultures for both bacteria and fungi. Sputum gram stain and culture, likewise, revealed no fungus. Skin biopsy, however, yielded numerous yeast cells of *Histoplasma capsulatum*.

Treatment of choice is typically itraconazole or amphotericin B, however studies have shown success with fluconazole as well. Our patient received itraconazole 200 mg orally twice a day for 6 weeks and was instructed to follow-up in 1 month. Treatment is critical due to the progressive and even fatal course of disseminated histoplasmosis. Studies have shown up to a 25 % reduction in mortality with antifungal therapy.

### Syphilitic Uveitis and HIV: The Great Masquerader Disguised as Meningitis.

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**Introduction:** Syphilitic uveitis, previously a classic manifestation of syphilitic infection, is now a rare complication. Because syphilitic uveitis is an extension of neurosyphilis, its symptoms may be mistaken for bacterial or fungal meningitis in HIV infected patients.

**Case Presentation:** A 38-year-old caucasian male with HIV presented to the University of Utah HIV Outpatient clinic for chronic headaches and acute worsening of his vision. Additionally, he had become anorexic, progressively confused, and ill appearing. He described some neck stiffness and eye pain, but denied fever, nausea, vomiting, abdominal pain, cough, or chest pain. On examination, he was afebrile. His blood pressure was 95/60. He was cachectic, toxic appearing, oriented to person and time but not place. The pupils measured 4mm on the right and 6 mm on the left and were poorly reactive. He could see light and movement, but not form. Conjunctivae and sclera were significantly injected bilaterally, but he had no eye tearing or watering. The patient had no Brudzinski's or Kernig's sign. The remainder of the neurologic exam demonstrated no focal abnormalities. Cardiopulmonary exam demonstrated tachycardia (rate 110 bpm)

and a capillary refill prolonged at 4 seconds. The skin was pale, and there was an erythematous rash of three centimeter, confluent, circular macules present over the patient's abdomen. His CD4 cell count was 472/mm<sup>3</sup>. with HIV viral load of >100,000/mm<sup>3</sup>. A lumbar puncture showed a glucose 23 mg/dL, protein 385 mg/dL, WBC 63/uL with 67% lymphocytes, 13% monocytes, and 20% PMNs. The CSF gram stain, cryptococcal antigen, bacterial culture, CMV PCR and HSV PCR were all negative. The serum RPR titer was 1:2048, and the CSF VDRL was 1:8. Ophthalmologic exam demonstrated anterior and posterior uveitis. During the hospital course, the rash advanced to the patient's thorax, back, palms, and soles. He was treated with IV aqueous penicillin G for two weeks, and he received systemic and topical steroids for the uveitis. After three weeks, he has had enough recovery of his vision to read large print.

**Conclusion:** Although syphilis is less common, it still can present with its very damaging symptoms, including vision loss. Physicians should be vigilant of this "great masquerader" and consider syphilis in HIV patients with symptoms of meningitis or ocular problems.

#### The EYES Have It.

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A 46-year-old female presents with a right temporal headache and visual loss. The headache has occurred on and off for 2 months. The visual loss has been gradual over the past 2 weeks and she now states that she is "blind". Past medical history includes NIDDM, hypertension and a recent history of viral meningitis. Review of systems includes a 17-pound weight loss over the past few months, the visual symptoms and headache mentioned above, and recent nausea and vomiting. She had two previous workups at outside facilities recently that were completely negative. Social history is negative for tobacco, alcohol and illicit drug use. Physical exam was significant for proptosis and small 1-2mm pupils that were minimally reactive. She variably reacts to visual confrontation. Vitals, all labs and head CT were normal. She was admitted to internal medicine and worked up by neurology and ophthalmology. The ophthalmology consult revealed no abnormality to explanation of her visual symptoms. Neurology ordered a MRI and MRA. There was leptomeningeal enhancement on the MRI, which could be explained by her recent viral meningitis and the MRA was normal. A lumbar puncture was performed and was negative for meningitis. Psychiatry was then consulted for possible conversion disorder. They placed her on Remeron and Seroquel. Her vision did not improve, however in light of her negative workup, she was discharged with a follow-up appointment with Neurology and Psychiatry. She was re-admitted 3 days later with the same symptoms as well as a catatonic-like state. She was again evaluated by Neurology and Ophthalmology and the work-up was negative. She was then transferred to Psychiatry for continued treatment of conversion disorder. The patient then began to experience dysarthria, combative behavior, confusion, mild right sided weakness and ataxic gait. She

was transferred to the ER for further evaluation. It was determined that she had a small lesion in the pons that was not on previous CT or MRI. A repeat MRI confirmed the new lesion in the pons. Angiography was normal. Shortly after the imaging, the patient became unresponsive. She was bradycardic and despite resuscitative measures, the patient died. An autopsy was performed and revealed metastatic poorly differentiated adenocarcinoma of the brain. No primary tumor was found, although immunohistochemical staining was suspicious for breast origin.

Leptomeningeal infiltration is recognized in only 4-7% of cancer patients although found in as many as 20% in autopsy. Pituitary metastasis is rare and accounts for only 1-2% of sellar masses. Both leptomeningeal and pituitary metastases are most commonly associated with breast and lung cancers. The prognosis is very poor in both subsets with average survival of 3-6 months.

#### Any Medication can be Dangerous.

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**Learning Objectives:** 1. Recognize torsades de points as a potential complication of low-dose haldol administration. 2. Understand the importance of cardiac monitoring in the setting of haldol administration for acute agitation.

**Case:** A 25-year-old woman with a history of schizophrenia and depression presented with one day of persecutory paranoia. On admission, she was noted to be hitting herself and thrashing about in bed. She was given lorazepam and haloperidol by intramuscular injection. One hour later, she was noted to be unresponsive. The initial telemetry reading revealed torsades de pointes. An immediate 200 J defibrillation resulted in a return to sinus rhythm. Intravenous magnesium was administered to prevent recurrence of the arrhythmia. She had no history of cardiac disease, and her physical examination and a echocardiogram following the event were normal. Her electrolytes and toxicology screen were also normal. She was monitored uneventfully overnight and discharged the following day.

**Discussion:** QT prolongation reflects delayed repolarization of the myocardium. In its extreme form, a PVC during this at-risk repolarization time results in torsades de pointes. The rate of repolarization is due to activity of each cell's delayed rectifier potassium channel. When there is both increased heterogeneity of ventricular repolarizations and early after repolarizations, slowly propagating extrasystoles can induce torsades de pointes.

Haldol is known to induce prolongation of the QT interval, especially when administered intramuscularly and in combination with other medications that retard its clearance. This is the first case of which we are aware that induced torsades de pointes after a single 5 mg dose of haldol. The co-administration of haldol and lorazepam may have induced torsades de pointes at doses not typical for this disorder. This case has implications for management of agitated patients in the emer-

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gency setting for whom haldol is considered for the acute management of agitation. Based upon this case, cardiac monitoring appears to be indicated even when low doses of haldol are administered, and especially if co-administered with other medications.

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### Non Small Cell Carcinoma in a Patient Presenting with Palpitations.

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**Case:** A 50-year-old man with past medical history of smoking, emphysema and bullous disease presented for routine physical exam. Chest x-ray demonstrated a large left pneumothorax requiring chest tube placement. Later, the patient disclosed that he had been having feelings of an irregular pulse for approximately one month. Physical exam was unremarkable except for an irregularly irregular heart rate. Electrocardiogram was consistent with atrial fibrillation. Repeat chest x-ray after chest tube placement revealed a left perihilar dense mass measuring 10.5 x 7.5cm. CT of the thorax confirmed a large left mid lung zone soft tissue mass with extensive left lung bullous disease. The bronchial biopsy demonstrated non-small cell carcinoma. The patient was referred to oncology. Cardiology initiated appropriate management for atrial fibrillation. A 2-D echocardiogram revealed biatrial enlargement with a normal ejection fraction. Atrial fibrillation persisted and four months later, a repeat CT of the thorax ordered because of increasing dyspnea showed a larger left lung mass measuring 11 x 10 cm and additional interval appearance of an abnormal soft tissue density in the pulmonary veins and the left atrium. A transesophageal echocardiogram demonstrated a large homogenous echodensity protruding from the pulmonary veins into the left atrium measuring 5.1 x 3.8 cm. A PET scan showed a left lung mass with extension into the left atrium consistent with malignant neoplasm. The patient was treated with radiation and chemotherapy. Over the next few months, the palpitations and tachycardia improved steadily until complete resolution of the atrial fibrillation. Follow up CT of thorax showed no identifiable tumor remaining. Follow up echocardiogram revealed no evidence of intracavity mass or thrombi.

**Discussion:** Metastatic disease generally occurs via three mechanisms: hematogenous or lymphangitic spread to the heart, direct invasion across the pleura and pericardium, or extension into the left atrium via the pulmonary veins. Pericardial effusion is the most frequent cardiac presentation of metastatic lung cancer, and rarely may be the presenting feature of lung cancer. Other complications related to pericardial and cardiac invasion, include tamponade, arrhythmia, and cardiac failure. Lung cancer may also invade the pulmonary veins and extend into the left atrium by direct extension within the lumen of the veins. Squamous cell carcinomas are the least likely of the non small cell lung carcinomas to metastasize with approximately less than 20% of cases at presentation. The unique feature of our case is the presentation of atrial fibrillation associated with non small cell lung carcinoma

extending into the pulmonary veins and left atrium and its resolution after chemotherapy and radiation.

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### Aspergillus terreus: The Bone Pathogen.

ML Juneau, MD.

**Introduction:** *Aspergillus terreus* is a saprophyte found ubiquitously in the environment which is rarely described as an invasive pathogen even in immunocompromised patients and even less commonly as a causative agent of osteomyelitis.

**Case:** We report a case of a 45-year-old man with no past medical history with *Aspergillus terreus* osteomyelitis. Approximately four months prior to presentation the patient fell on a friend's boat while fishing on an outboard motor bruising his chest without any evidence of skin breakage. The patient noticed a painful nodule developing on his chest approximately two to three days after the fall. He sought medical treatment at two other local hospitals and was told he had cartilage contusions and was advised to see his primary care physician for incision and drainage of suspected abscess. His primary care doctor incised the abscess and packed the wound with Iodoform. He then told the patient to follow up with a surgeon at which time he presented to our facility afebrile with greenish-yellow drainage from his chest wound. Chest CT at time of admission revealed inflammation of his right fifth anterior sterno-costal rib without extension to mediastinum. Shortly after admission a wound culture was sent and patient was started on Kefurox 1.5mg q8hrs IV. On hospital day 2, the patient was started on Vancomycin 1g IV q12hrs and Aztreonam 1.5g IV q12 hrs and taken to the operating room for incision and drainage of wound/abscess, at which time friable rib was palpated and resected and three specimens sent to pathology (rib cartilage, bone sequestration, and wound culture of right costal cartilage). The intraoperative wound culture grew *Aspergillus terreus* and the patient was started on Ketorolac tromethamine 15mg IV q6hrs for two days. The patient's hospital course was unremarkable. He remained afebrile without an elevated white blood cell count and no post-operative complications. The patient was discharged home with instructions to take Voriconazole 6mg/kg x1 day, then 4mg/kg x1week, then 200mg po bid for 4-6 weeks with weekly liver function tests, complete blood counts, and renal function tests. The patient also received an ophthalmologic evaluation prior to starting the voriconazole, which was normal.

**Discussion:** The case is unusual because the pathogen was *Aspergillus terreus*, the host was not immunocompromised, there was no definitive entry site, and the infection was only osteomyelitis.

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### A Rare Case of Malignant Tracheo-Esophageal Fistula (TEF) in Non-Small Cell Lung Cancer.

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**Introduction:** Malignant tracheoesophageal fistulae occur uncommonly. The most common primary tumor causing this condition is esophageal carcinoma. It is rare with lung cancers. We report a case of acquired TEF in a patient with non-small cell lung carcinoma (NSCLC).

**Case:** A 57-year-old man presented with progressive dysphagia, which was more pronounced for solids than liquids. Four months earlier, he was found to have unresectable non-small cell carcinoma of the upper lobe of the right lung. Radio- and chemotherapy were unsuccessful in inducing remission. He reported hoarseness and an occasional cough productive of clear sputum, but denied fever, chills, or hematemesis. Examination revealed emaciation and a fetid breath without significant oral pathology. On auscultation of the lungs, there were crackles and bronchial breath sounds in the right upper chest and axilla. A complete blood count showed hemoglobin of 11.3 mg/dL, with anisocytosis and microcytosis on peripheral smear, a mean corpuscular volume of 73.7 fL; and white blood cell count of 12,300/cm<sup>3</sup> with granulocytic predominance. Iron saturation was 7%. Blood cultures had no growth. A barium esophagram revealed aspiration of contrast material into a large air cyst in the right anterior lung. Following the study, the patient began coughing and a radiograph showed that the barium in the cyst had evacuated into the trachea and esophagus. Subsequently, computed tomography of the chest with contrast revealed a large cavity in the right upper lobe that communicated with the trachea and esophagus. It was surrounded by an infiltrating lesion. Esophagoscopy revealed a TEF with residual tumor surrounding the stoma. The proximal location of the lesion and friability of the tissue precluded placement of an esophageal or bronchial stent. Therefore, a gastrostomy tube was placed for palliation.

**Discussion:** Though rare, malignant TEF must be considered when patients with known lung cancers present with dysphagia. Because of the evidence of residual tumor at the fistula site, we do not believe that the TEF in this patient was secondary to radiation therapy. This complication of NSCLC can lead to rapid clinical deterioration and death from overwhelming pulmonary infection. Without prompt palliation, the mean survival is from one to six weeks. Effective management requires isolation of the fistula from the alimentary tract. The location of the fistula and the patient's general condition are major factors in the selection of palliative procedures. In this case, only a gastrostomy tube was feasible.

#### A New Look at an Old Draining Fistula.

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A 60-year-old white woman underwent a salpingo-oophorectomy, abdominal sacral colpopexy, pubovaginal sling, cystoscopy and fascia lata harvest in 1997 at age 52 with subsequent development of a wound abscess (15.5 cm x 4.5 cm) requiring surgical incision and drainage. Previously, the patient had undergone a total abdominal hysterectomy in 1978

at age 33. Following surgical drainage and extensive wound care, the open abdominal wound regressed to a small periumbilical sinus tract, which never closed and was attributed to the surgical procedure. Periodically, the sinus tract would become more edematous and drain exudative fluid, however, the tract never closed. Over the 8 year course, outpatient surgical consultation was made three times for possible closure of the sinus tract/fistula, each resulting in the decision that no surgical intervention would benefit the patient. Patient's laboratory studies (complete blood count and basic metabolic panel) were within normal limits prior to the second surgery. Gradually, the patient developed hypertension, diabetes mellitus, hypercholesterolemia, osteoporosis, mild obesity and a normocytic anemia with mild hypocellular marrow and mild erythroid hypoplasia (hemoglobin 8.9g/dl, normal 12-16 g/dl; hematocrit 26.9%, normal 35-46%). Patient denied any gastrointestinal complaints including diarrhea, abdominal pain, melena, hematochezia, arthritis, weight change, or other complaints. A flexible sigmoidoscopy in 1999 was found to be normal. Re-evaluation of patient's colon with colonoscopy in 2005 showed mild diverticulosis and normal colonic histology from a random sigmoid colon biopsy. A small bowel follow-through as part of anemia work-up revealed 30 minute transit time and diffuse, nodular, thickened small bowel mucosal fold pattern suggestive of an underlying process. Computed tomography scan of the abdomen was indeterminate of inflammatory process. Saccharomyces cerevisiae IgG was 34.3 units (normal 0.0 - 24.9 units) and IgA was negative indicating the diagnosis of Crohn's Disease in an essentially asymptomatic patient except for persistent draining abdominal sinus. Laboratory workup for sprue or other inflammatory processes was negative. Close to eighty percent of Crohn's disease patients are positive for either IgA or IgG and healthy controls are not reactive to this lab assay. The patient is currently undergoing surgical evaluation for resection of fistula with adjunctive pharmacological/immunotherapy with infliximab (5mg/kg) for chronic control of her autoimmune process. Lack of abdominal pain or diarrhea is rarely seen in Crohn's disease.

#### Shewanella putrefaciens Bacteremia and Pneumonia.

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Patient is a 73-year-old AAM with history of schizophrenia and NIDDM who normally gets his care at the local VA hospital, who presented to the hospital unresponsive, with severe volume depletion and hypernatremia secondary to Hurricane Katrina. During Hurricane Katrina, he refused to leave his home with the rest of his family. He was found unresponsive in his home on 9/7/2005 and covered with feces. He was rescued and transported to the MASH unit at the New Orleans International Airport where he responded to IV fluid hydration. He stated that he was trapped in floodwaters in his attic for a whole week. After initial resuscitation, he was transported to Ochsner Hospital for further resuscitation and workup.

Patient noted to have a left lower lobe pleural effusion and treated initially as a pneumonia with Zosyn and Ciprofloxacin, as the gram stain showed gram-negative rods. Patient was bacteremic with blood cultures positive for

*Shewanella putrefaciens*. Further studies delineated that there was a lung abscess in the left lung base with loculated pleural effusion and required Interventional Radiology to aspirate the fluid. Approximately 20cc of orange colored pleural fluid was removed. There was no growth from the pleural fluid. The patient's hospital course was prolonged secondary to multiple factors. He continued to show signs of infection with continued fever, elevated WBC, and shortness of breath requiring supplemental oxygen. He was also bacteremic with *Candida glabrata* later in the hospital course and also developed bilateral lower extremity deep venous thrombosis. Patient was afebrile with repeat blood cultures that were negative, and he was to follow-up with the VA hospital in the city his family moved to.

*Shewanella putrefaciens* is a gram-negative oxidative and nonoxidative bacilli that produces hydrogen sulfide gas. It is considered a *Pseudomonas* type organism. It is occasionally found as a human pathogen, but it is typically found in non-human sources. These include aquatic reservoirs (marine, freshwater, and sewage), natural energy reserves (oil and gas), soil, fish, poultry, dairy, and beef products. Most human isolates of *S. putrefaciens* occur as part of a mixed bacterial flora, clouding their clinical significance. However, a number of monomicrobial illnesses due to *S. putrefaciens* have been documented and include bacteremia, soft tissue infections, and otitis media. There have also been case reports of this bacteria causing peritonitis in patients on peritoneal dialysis and even causing multi-organ failure.

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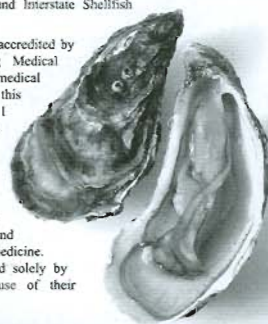
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