Each year medical students 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 31 most highly ranked abstracts in this year’s competition. These abstracts (18 oral; 13 poster) were presented at the Associates Meeting held at Tulane University Health Sciences Center in New Orleans in January 2008. We would like to thank the Journal of the Louisiana State Medical Society and appreciate its efforts to publicize the hard work of these young trainees.

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

Sandy Kemmerly, MD
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**ORAL PRESENTATIONS**

**Olanzapine-Induced Hypothermia and Polyuria.**

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Hypothermia and polyuria induced by atypical antipsychotic agents have been described separately and sparingly in the clinical psychiatric and psychopharmacological literature. We present a case report of simultaneous hypothermia and polyuria in a patient treated with olanzapine.

A 38-year-old woman was referred to our hospital for evaluation of altered mental status. Documentation accompanying the patient contained a recent psychiatric evaluation describing her as “grossly psychotic, agitated, and manic”. Carbamazepine 400 mg and olanzapine 20 mg twice a day by mouth had been initiated at the referring facility five days prior. Vital signs obtained during her initial evaluation in the Emergency Department revealed hypotension, bradycardia and hypothermia (core body temperature 28.8°C). Physical exam was significant for a depressed level of consciousness (GCS: E1 V1 M4) with hyperreflexia and a positive Babinski on the left. Electrocardiograph changes were consistent with hypothermia (sinus bradycardia, prolonged QTc and precordial lead early repolarization). The patient was immediately intubated for airway protection and mechanically ventilated. Volume resuscitation was initiated in conjunction with passive rewarming. All psychiatric medications were withheld. Toxicology screens were negative. Non-fasting blood glucose was 152 mg/dL. Serum lactic acid, luteinizing hormone (LH), follicle-stimulating hormone (FSH), and prolactin were within normal limits. Thyroid-stimulating hormone (TSH) was 0.91 IU/mL and a random cortisol was 24.2 mcg/dL. Blood cultures failed to grow identifiable pathogens and cerebrospinal fluid analysis, including bacterial cultures, herpes simplex viurs polymerase chain (HSV PCR), and cerebrospinal fluid venereal disease research laboratory test (CSF-VDRL), were also negative. Neurological consultation, electroencephalogram and intracranial imaging were also obtained without significant yield. General supportive care was provided in the intensive care setting with full recovery of the patient to her baseline without evidence of sustained neurological deficit. Of note, urine output during the first 48 hours of hospitalization totaled nine liters despite persistent hypotension, elevated plasma osmolality and rising serum sodium concentration. Olanzapine was implicated as a potential etiology of this patient’s hypothermia and polyuria only after the exhaustive inpatient investigation outlined above failed to identify an organic cause.

It has been speculated that Olanzapine’s potent 5HT-2a and D2 receptor antagonism is responsible for its ability to disrupt hypothalamic thermoregulation. Polyuria subsequent to Olanzapine administration is thought to represent early or partial central diabetes insipidus. To our knowledge, this is the first case report describing both of these adverse drug reactions occurring simultaneously in a patient treated with Olanzapine. Given the increase in use
of this drug over an ever-widening range of psychiatric diagnoses, we feel that heightened awareness of these potential adverse drug reactions is warranted.

Late Presentation of Congenital Diaphragmatic Hernia.

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A term infant presenting with respiratory distress on third day of life was found to have interval expression of a congenital diaphragmatic hernia (CDH). Patient is a 39 5/7 WGA male born to a 27-year-old woman. G1P0 with adequate prenatal care via primary C-section secondary to failure to progress. Within hours of delivery, the patient was noted to be tachypneic and evaluation revealed the presence of a right pneumothorax. After receiving oxygen therapy, the pneumothorax resolved within 24 hours. However, on the night prior to anticipated discharge, the infant developed respiratory distress. On exam, he was tachypneic with a respiratory rate of 115; however hemodynamically stable with adequate oxygenation. Chest X-ray then revealed the interval development of a right-sided CDH.

CDH is defined as a developmental defect in the diaphragm that allows abdominal viscera to herniate into the chest affecting 1 of 2200 live births. Typically, the diaphragm develops anteriorly as a septum between the heart and liver and extends posteriorly with final closure at the left Bochdalek foramen at 8-10 WGA. If bowel migrates into the abdominal cavity before the foramen closes, herniation of abdominal contents into the thorax may occur. The herniation may involve the liver, spleen, stomach, or intestines, and it is often associated with some degree of malrotation.

Late presentation of CDH may occur during the neonatal period or rarely in adulthood. Clinically, these cases may initially present with pneumothorax and later yield evidence of CDH. Late presentation of CDH has been associated with Group B streptococcal (GBS) infections in neonates. Though the causative relationship between GBS infection and delayed-onset CDH is unknown, the proposed mechanism is that increased intra-thoracic pressure coupled with abnormal lung compliance, due to inflammation, may result in expression of the CDH and subsequent migration of abdominal contents into the pleural cavity. Outside of the neonatal period, CDH most commonly presents with symptoms of respiratory distress or gastrointestinal obstruction.

The major morbidity due to CDH is persistent pulmonary hypertension (PPHN) due to pulmonary hypoplasia. The severity of PPHN is directly related to the degree of decreased bronchiolar as well as arterial branching. Therefore, if CDH is present either with pre-natal diagnosis or late presentation, ventilatory support with low peak pressures should be provided.

The current recommended approach to management is to maximize preoperative care avoiding factors such as acidosis, hypercarbia and hypoxia that worsen pulmonary hypertension followed by surgical repair to close the defect. Surgical repair is also recommended if CDH is found in adults, even if asymptomatic, to avoid herniation and possible strangulation of abdominal viscera.

I Have a Skin Rash.

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Introduction: Hepatitis C infections have multiple extra-hepatic manifestations including the skin involvement. Patients can present with purpuric rash which should raise the suspicion of leucocytoclastic vasculitis.

We report a case of a 48-year-old Caucasian patient with hepatitis C who presented due to complaints of bilateral lower extremities cellulitis superimposed on chronic vascular insufficiency skin changes. The skin changes included symmetrical brownish discoloration on both lower extremities up to an inch above the knee joint with a sharply demarcated area. Also seen were multiple purpuric lesions on her lower extremities and buttocks. Skin biopsy done from the buttock was consistent with leucocytoclastic vasculitis. Her labs included- abnormal liver synthetic function, high aspartate aminotransferase (AST) test/ alanine aminotransferase test (ALT), high rheumatoid factor (RF) blood test, negative antinuclear antibody (ANA) test, negative antineutrophil cytoplasmic antibodies (ANCA), and negative anti cardiolipin. Her C3 and C4 were low. Serum cryoglobulins were positive. She was started on antibiotics and skin cellulitis resolved. Skin discoloration and purpuric lesions persisted.

Discussion: Presence of skin change should prompt work up of cryoglobulin level, complement level and work up to rule out other vasculitides. Hepatitis C virus (HCV) is most commonly associated with mixed cryoglobulinemia which in turn can cause low complement levels. Skin biopsy is definitive for leucocytoclastic vasculitis. Treatment would involve treating hepatitis C in this case.

Spontaneous Internal Carotid Dissection: Uncommon but Significant Cause of Ischemic Stroke in Young Adults.

C Moll, MD, Louisiana State University Health Sciences Center, New Orleans, Louisiana.

Spontaneous dissections of the carotid artery (SCD) arise from intimal tears which allow blood to enter the wall of the artery forming an intramural hematoma. Typically seen in the fourth generation of life, most patients present with headache or symptoms of transient ischemic attacks.
but describe no precipitating traumatic events. The etiology of most cases remains unknown.

We report the case of a 28-year-old Caucasian woman with no known previous medical conditions who presented to the Emergency Department thirty minutes after the onset of right hemiplegia and aphasia. Review of systems was significant for brief episodes of unconsciousness followed by upper extremity shaking and headaches that were attributed to hypoglycemia. The patient had no history of recent trauma or risk factors for coronary artery disease or cerebral vascular accidents. Initial chemistries, including urine drug screen, were unremarkable. Computed tomography (CT) of the head without contrast was reported as negative for acute intracranial bleed but magnetic resonance imaging (MRI) of the brain showed a left middle cerebral ischemic stroke. A tranesophageal echocardiogram with contrast demonstrated good left ventricular contraction with no patent foramen ovale. Complete occlusion of the left internal carotid artery was visualized by carotid ultrasound. Findings on magnetic resonance angiogram (MRA) of the head and neck were suggestive of a left internal carotid dissection. Fibrinolytic therapy was not given and the patient was observed in the intensive care unit (ICU) on Lovenox and aspirin.

Worsening cerebral edema with midline shift was seen on repeat CT of the head and the patient underwent decompressive craniectomy. Hypercoagulability work-up was unremarkable. Repeat MRI showed recanalization of the left internal carotid artery and vascular surgery was consulted regarding possible stenting. They recommended physical rehabilitation and treatment with Lovenox and aspirin with re-evaluation for stenting in six weeks.

Given the patient’s negative medical history and no identifiable cause, she was given the diagnosis of SCD. Though SCD accounts for only 2% of all ischemic strokes, 10%-25% of ischemic strokes in young and middle-aged patients are attributed to this disease. Treatment with fibrinolytics is controversial and most SCDs are treated with anticoagulation or stenting. MRA is often the definitive diagnostic test. A high index of suspicion should be kept when evaluating a young patient presenting with stroke symptoms and an unremarkable history.

Severe Peripheral Neuropathy Related to Vitamin B6 (Pyridoxine) Deficiency After Gastric Bypass Surgery.

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Purpose: To discuss a not well documented cause of peripheral neuropathy in patients with gastric bypass surgery.

We present a 37-year-old Caucasian woman with history of morbid obesity status post gastric bypass surgery in 1997 who presented to the Emergency Department complaining of weakness, pain, and numbness in bilateral lower extremities over the last four months. Initial blood work showed marked anemia with very elevated mean corpuscular volume (MCV) but normal vitamin B12 levels. Further diagnostic tests including magnetic resonance imaging (MRI) of brain and lumbar spine as well as human immunodeficiency virus (HIV) and hepatitis panel were negative. Nerve conduction study of the lower extremity was consistent with severe peripheral neuropathy and axonal damage. Vitamin B6 levels were severely decreased (less than 2.5 nmol/L). Patient was started on B6 supplement and followed as an outpatient. Symptoms were improved by next time she was seen as outpatient.

Discussion: Vitamin B6 (pyridoxine) is a water-soluble vitamin used by the body as a catalyst in reactions that involve amino acids. Pyridoxine deficiency is a rare disease in the adult population because it is found very easily on our diet. People that are prone to B6 deficiency are alcoholics and patients treated with anti tuberculous medication. Its deficiency is not well documented in patients with gastric bypass surgery. Further studies should be done to see if there is a strong correlation between gastric bypass surgery and vitamin B6 induced peripheral neuropathy.

Alpha-Fetoprotein and Beta-Human Chorionic Gonadotropin (HCG) Producing Squamous Cell Lung Cancer in an Adolescent.

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Introduction: Among adolescents aged 15-19 years, malignancy occurs at a rate of 150-200 per million per year. The most frequent diagnostic groups are leukemias, sarcomas and germ cell tumors. It is unusual for primary lung cancer to present during adolescence, and even more unusual for lung cancer to be associated with elevated alpha-fetoprotein (AFP) and beta human chorionic gonadotropin (beta-HCG) levels.

Case Report: A 17-year-old man was referred for evaluation and treatment of newly-diagnosed lung cancer. He was a non-smoker who had injured his chest while playing basketball and noted a persistent bruise, dull pain, and worsening dyspnea over the next month. Evaluation by his primary care physician revealed localized tenderness with a 10 x 10 cm ecchymotic area and decreased breath sounds with dullness to percussion over the right lower chest. A radiograph showed a large pleural effusion, and computed tomography of the chest revealed a mass in the right middle lobe of the lung with post-obstructive pneumonia. Bronchial brushings and biopsy of the lesion suggested a moderately-differentiated primary cell carcinoma. Laboratory evaluation revealed hypercalcemia; AFP 2481 ng/mL (normal <15); beta-HCG 14 mIU/mL (normal <5), and Lactic acid dehydrogenase (LDH) 650 mg/dL (normal 100-190). A thoracostomy tube was placed for relief of dyspnea.
Cytology of the pleural fluid was non-diagnostic. Because of his age and clinical picture, a diagnosis of metastatic germ cell tumor was considered. However, ultrasonography of his testes revealed only bilateral microlithiasis, and chromosome analysis of the tumor specimen showed no duplication or trisomy of the short arm of chromosome 12. Magnetic resonance imaging of the brain did not reveal metastases. Chemotherapy was initiated with cisplatin and etoposide. He tolerated the treatment well, and some regression of his disease was noted with a decrease in AFP and beta HCG. However, repeat scanning three months later revealed extensive tumor involvement of the right hemithorax, with areas of osseous destruction. There was a dominant heterogenous, lobulated mass in the right middle lobe. Re-biopsy of the lesion again revealed squamous cell cancer. He died one month later.

Discussion: This case illustrates that primary lung cancer can occur in adolescence with no significant risk factors. The tumor in this patient was extremely rare in that it produced AFP and beta-HCG, and thus mimicked a germ cell tumor. There are occasional reports in the literature of such tumors. However, these have been poorly differentiated adenocarcinomas, unlike the moderately-differentiated squamous cell carcinoma in this case.

Lazarus the Kidney Transplantee.

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Leprosy has been eliminated from most of the developed world since the introduction of multidrug therapy. It is rarely reported in North America, but may take advantage of the immunosuppressed host.

A 49-year-old man received a cadaveric kidney transplant in February 1999 for kidney failure due to antineutrophil cytoplasmic antibodies (ANCA) associated vasculitis. His human leukocyte antigen (HLA) was comprised of A2, A3, B7 (BW6), B51 (BW4), CW4, CW7, DR8, DR15, DQB1*04, DQB1*06. He had an uneventful post transplant course on cyclosporine, mycophenylate mofetil, and prednisone, but was lost to follow-up after Hurricane Katrina. In April 2006, he returned reporting a one year history of progressive skin eruption of the chest, back, and arms without associated symptoms. On physical examination, he had a variety of different skin lesions including many pink to red oval shaped macules and papules on the back, chest, buttocks and arms, some of
which appeared inflamed. The face was spared. The lesions were pruritic, but not tender, and he had normal sensation over the lesions. A series of biopsies were performed. Fite’s stain showed numerous acid-fast organisms within granulomatous inflammation, and the patient was diagnosed with lepromatous leprosy (LL). The source of the infection could not be established. He had no close contacts with the disease, nor exposure to armadillos. The patient was treated with dapsone, clofazimine and rifampin. Repeat biopsy one year later showed evidence of persistent bacteria, and twenty-four months of therapy was recommended secondary to chronic immunosuppression.

Leprosy is caused by mycobacterium leprae, an acid-fast obligate intracellular bacillus, and is characterized by chronic granulomatous disease of the skin and peripheral nerves. It is probably transmitted through respiratory droplets from close contacts. Armadillos may act as a reservoir. Disease expression has been linked to HLA-DR3, and HLA-DQ1. Few cases of leprosy in renal transplant patients have been reported, including six in India, one in Taiwan, and one in the United States.

Skin lesions in transplant patients pose a formidable clinical challenge. The differential diagnoses include fungal, bacterial, or other atypical mycobacterial infections. Leprosy should be considered in this differential, and a biopsy is warranted in such cases.

The Skinny on a Growing Problem: Dry Beriberi Following Bariatric Surgery.

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Obesity has reached epidemic levels in the United States and incidence continues to rise. More than 60% of the adult population now meets criteria for being overweight or obese. Gastric bypass surgery has become a popular and effective way to combat this medical problem, with more than 100,000 procedures performed annually. However, surgery is not a benign treatment, and has multiple inherent risks and associated complications, including malnutrition and vitamin deficiency. It is vital for internists to be aware of these medical complications and their varied presentations.

A 37-year-old woman with morbid obesity underwent a Roux-en-Y gastric bypass three months prior to presentation. Her operation had been uncomplicated, but she received no follow-up care after the procedure. Two weeks prior to presentation the patient developed mild paresthesia of the anterior right leg. Two days prior to presentation her paresthesia worsened and began to involve the left leg. At this time, she also noted progressive weakness of the lower extremities bilaterally. One day prior to presentation she experienced acute problems walking, fell down after her knees buckled under her weight, and was then unable to stand due to weakness. Her review of systems was also positive for persistent nausea and vomiting and a 60 pound weight loss since her surgery.

She presented to the emergency room (ER). On admission to the medicine service, bilateral lower extremity proximal muscle weakness was noted from the thighs distally. In her lower extremities bilaterally, decreased sensations from the knees distally were noted and reflexes were diminished. Neurological exam further revealed no upper extremity abnormalities and intact cranial nerves. There were no signs or sequelae of cardiac disease. Computed tomography (CT) scan of the head was normal, and the magnetic resonance imaging (MRI) of the brain and spine were within normal limits. Complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) were within normal limits. Comprehensive metabolic panel (CMP) was unremarkable. Vitamin levels were drawn, revealing greatly reduced thiamine levels (<0.5). B12 and other vitamin levels were normal.

Aggressive thiamine replacement was begun immediately. After three days the patient showed little improvement and was still unable to walk. Physical therapy was initiated and the patient was transported to a rehabilitation facility, where she received daily therapy. After three weeks she had slowly, but progressively, improved and was able to ambulate about 150 feet with a rolling walker.

Although quite common in gastric bypass surgery patients, thiamine deficiency is rarely symptomatic. Symptomatic thiamine deficiency, known as dry beriberi (mostly neurological symptoms) and wet beriberi (cardiac symptoms), is seen in 0.0002% to 0.4% of gastric bypass patients. Internists and other primary care physicians should be diligent in thiamine and other vitamin replacement in post-gastric surgery patients, in order to prevent deficiencies. If beriberi develops, however, it should be aggressively treated with thiamine replacement. Symptoms of dry beriberi may persist for weeks to months following replacement of thiamine, but wet beriberi typically responds quickly to treatment.

References:
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Case Report: A 69-year-old woman was undergoing preoperative evaluation for rotator cuff surgery when she was found to have a prolonged activated partial thromboplastin time (aPTT) of >150, with normal prothrombin time (PT) and international normalized ratio (INR).

She denied a past history of bleeding. There were no signs of bleeding diathesis on physical examination. A thorough hematological work up was performed in which basic hematology, lupus anticoagulant, and von Willebrand factor levels were all within normal limits. However, mixing studies showed correction of the markedly prolonged aPTT, suggesting a factor level deficiency. Although factors levels were within normal limits, because of the prolonged aPTT, prekallikrein deficiency was considered and a contact factor study was obtained. Results showed a prekallikrein activity of less than 1%, as well as a normal HMW kinogen activity, thereby indicating a prekallikrein deficiency (also known as Fletcher factor) as the cause of her prolonged aPTT.

Discussion: Prekallikrein deficiency, also known as Fletcher factor deficiency, was first discovered in 1963 in Kentucky. Prekallikrein is a coagulation protein that is involved in early stages of pathway activation and fibrinolysis. The activation of factor XII is dependent upon Prekallikrein. When a deficiency is present, there is delayed activation of factor XII resulting in prolongation of the aPTT.

Conversely, in the fibrinolytic pathway, plasminogen is activated by several molecules including kallikrein and factor XII. Plasma kallikrein is also involved in the production of kinin from high molecular weight kininogen. These molecules are involved in various inflammatory processes, uterine contractility during labor and delivery, and various pathological states.

In addition to congenital deficiency, an acquired prekallikrein deficiency has been reported in cases of liver disease, septic shock, chronic renal failure, vitamin K deficiency, multiple trauma, DIC, typhoid fever, DVT, and phlebitis.

Severe prekallikrein deficiency in humans is not associated with any clinically significant impairment of hemostasis, fibrinolysis, or inflammatory response. Even though affected patients may have a markedly prolonged aPTT, there is no increased bleeding even when stressed by surgery. This observation suggests that contact mechanisms of clotting have only a minor role in normal in vivo haemostatic function. Several studies have demonstrated the role prekallikrein deficiency in arterial thrombosis. Nevertheless, most patients are asymptomatic with little to no post surgical complications. It is advisable to proceed with surgical management if necessary.
Tumorigenic Properties of Nanog-Expressing MCF-7 Breast Cancer Cells.

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Numerous studies within the past few years have provided growing evidence supporting the cancer stem cell (CSC) hypothesis, which states that a subset of phenotypically distinct tumor cells called cancer stem cells (CSCs), rather than all tumor cells, drive growth and metastasis of a solid tumor. If the CSC hypothesis is true, then CSC-specific therapy for cancer patients can provide potential improvements on current therapy standards by increasing efficiency and by allowing precise targeting without adverse effects on other cells. Before CSCs can be selectively targeted, it is necessary to identify markers that distinguish these cells from non-tumorigenic cells. In this study, our goal was to determine whether Nanog is a potential identifying marker of breast cancer stem cells, specifically in the MCF-7 breast cancer cell line.

Nanog is a transcription factor critical in maintaining the pluripotency of embryonic stem (ES) cells and is specifically expressed in the inner cell mass (ICM) and epiblast of the early embryo. Following implantation, expression is downregulated as ES cells differentiate into adult tissues; in other words, Nanog is expressed in pluripotent stem cells, but not in differentiated tissues. In fact, ablation of Nanog or its transcription factor (Oct4) expression in ES cells by genetic knock-out or by using RNAi-mediated knock-down procedures abolished self-renewal and pluripotency of such cells and resulted in their differentiation. Recent evidence indicates Nanog and Oct4 are expressed in human breast cancer tissue and MCF-7 cell lines.

For this study, a lentiviral vector bearing a 380-bp human Nanog promoter fragment upstream an EGFP coding region was constructed. The promoter fragment has been previously shown to give specific expression in ES and embryonic carcinoma (EC) cells. The vectors were transduced into MCF-7 cells and sorted using FACS. We then used reverse transcriptase–polymerase chain reaction (RT-PCR) to analyze Nanog mRNA expression by comparing Nanog mRNA levels in cells sorted for Nanog expression against those not sorted for Nanog expression with GAPDH as an internal standard. Preliminary results show detectable levels of Nanog mRNA in both samples. However, cells sorted for Nanog expression showed approximately 16 times more Nanog mRNA than unsorted cells. This preliminary study provides the basis for future studies involving injection of sorted and unsorted MCF-7 cells into the mammary fat pad of homozygous nude female mice and assessment of breast tumor onset and size, as well as metastatic burden in lungs. We also plan to assess Nanog-expression levels in MCF-7 cells sorted and unsorted for expression of various markers proposed by other groups as potential breast cancer stem cell markers.

Liver Disease is Skin Deep.

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Learning Objectives:
1. Recognize the presenting signs and symptoms of porphyria cutanea tarda.
2. Understand the pathophysiology of porphyria cutanea tarda.
3. Identify systemic diseases associated with porphyria cutanea tarda.

Case: A 62-year-old man presented with three months of “fragile skin.” He noted no additional symptoms, complaining only of recurrent vesicular lesions on sun-exposed areas of his body. He noted drinking three to five beers a day and he did not take any medications. He did not have a history of blood transfusion or intravenous drug use.

His vital signs were normal, as were his heart, lung, and abdominal examinations. Vesicles and bullae were present on the dorsa of both hands and on the face. He had facial hypertrichosis with underlying mottled brown-black pigmentation in the periorbital region.

His complete blood count and electrolytes were normal; human immunodeficiency virus (HIV) and antinuclear antibody (ANA) tests were negative. He had an elevated serum ferritin, elevated liver enzyme tests and he was hepatitis C positive. His urine was reddish, fluoresced under a Wood’s light, and revealed elevated levels of uroporphyrin.

After counseling, he stopped drinking and started weekly outpatient phlebotomy. His symptoms resolved slowly over two months.

Discussion: Porphyria cutanea tarda (PCT) is the most common disorder of porphyrin metabolism, and while it is frequently seen by the general internist, is frequently mis-ascribed to drug reactions. In normal heme biosynthesis, uroporphyrinogen decarboxylase converts uroporphyrinogen to coproporphyrinogen. PCT results from an accumulation of uroporphyrinogen in the skin due to either a decreased activity of the UPG-D enzyme, increased amino acid substrates entering the porphyrin metabolic pathway, or a combination of both. Photo-excited porphyrins in the skin serve as the catalyst by which ultraviolet light converts oxygen to two oxygen free radicals which are the cause of oxidative damage to the skin.

The cutaneous manifestations of PCT are found most frequently on light-exposed areas, most often on the dorsal hands and forearms. Patients complain of increased fragility, vesicles and bullae. Facial hypertrichosis and hyperpigmentation in the periorbital region are findings are highly suggestive of PCT.

PCT is autosomal dominant and, unlike the autosomal recessive acute intermittent porphyria, results in accumulation of water-soluble porphyrins that deposit in the skin.
and urine. For this reason, the diagnosis of PCT begins with examination of the urine, as porphyrins give the urine a reddish brown color and fluoresce under ultraviolet light. Confirmation by quantitative porphyrin analysis reveals elevated uroporphyrin and coproporphyrin.

Innate coding for the UPG-D enzyme varies from person to person, and those with genetically determined lower quantities of the enzyme can succumb to PCT if the amino acid substrates entering the porphyrin metabolic pathway are increased. An increase in amino acid entry is stimulated by starvation, alcohol use, and other inflammatory diseases such as systemic lupus erythematosus, hepatitis C, HIV, and hemochromatosis. For this reason, the internist must recognize the importance of evaluating for these conditions even after the diagnosis of PCT has been established. Our patient was subsequently diagnosed with hepatitis C and hemochromatosis.

Treatment of PCT begins with the elimination of alcohol, estrogen, or iron, decreasing the amount of amino acid substrates entering the porphyrin pathway. Phlebotomy is commonly used until either total urinary porphyrins diminish or the hemoglobin level decreases to 10 g/dL. In patients with hepatitis C, iron overload should be corrected with phlebotomy before consideration for interferon-ribavirin treatment.

Cholesterol to Stroke, Stroke to Coumadin, Coumadin to Cholesterol.

T Chang, Tulane University Health Sciences Center, New Orleans, Louisiana.

Leaning Objectives:
1. Identify cholesterol crystal embolism as a complication of anticoagulation.
2. Understand the presentation of renal failure due to cholesterol embolism.

Case Presentation: A 76-year-old man presented with altered mental status for two days. His family found him at home with disorientation, and generalized weakness. He had a history of stroke that occurred three months prior to admission, for which he had been taking coumadin.

His vital signs were normal. He was awake, oriented to self, and his speech was fluent but nonsensical. There were no carotid bruits, no jugular venous distention, and the cardiac exam was normal. There were diffuse myoclonic jerks of all extremities. Small, round purple skin lesions were found on the toes; they were tender and a few had small areas of central necrosis. His potassium was 6.2, a blood urea nitrogen (BUN) was 166, and creatinine, 10.8. His urinalysis was normal with the exception of moderate blood and crystals forming right angles. The urine sodium was 51.8, urine creatinine was 82, and fractional excretion of sodium was 5.1 percent. He was given intravenous fluids and when the BUN and creatinine continued to rise, was started on hemodialysis.

The records from his admission three months prior were reviewed. As part of his stroke evaluation, he had a transesophageal echocardiogram that revealed a grade IV atheroma of the ascending aorta and arch. He was started on warfarin for secondary stroke prevention. A biopsy of a skin lesion confirmed the presence of cholesterol emboli crystals.

Discussion: Cholesterol crystal embolism is an increasingly recognized cause of renal failure. It is common in elderly men and is associated with diabetes, hypertension, cardiovascular disease, and preexisting renal insufficiency. Although it may occur due to spontaneous rupture of an atherosclerotic plaque, the majority of cases have an identifiable triggering event. Embolization is a known complication of anticoagulation, angiography, and vascular surgery in patients with large vessel atherosclerotic disease. Cholesterol crystals dislodge from the plaque and may embolize to multiple locations including the kidneys, gastrointestinal tract, retinas, and extremities. The clear, biconvex crystals deposit in small vessels and induce a

Best performance in a supporting role. Arturo Guzman, school teacher, and Marcia Gay Harden, actress. Both have earned raves for their volunteer work for the American Red Cross. To learn more about volunteering, contact your local Red Cross chapter or visit www.redcross.org.
vasculitis in the endothelium. Inflammation, fibrosis, and vessel occlusion may occur over a time period of weeks to months.

The clinical presentation of cholesterol embolization includes fever, fatigue, and myalgias. Skin lesions are present in the majority of patients and may include purple toes, livedo reticularis, and necrosis or gangrene. Signs of uremia may be present and fundoscopy may reveal retinal crystal deposition. The erythrocyte sedimentation rate, C-reactive protein, and eosinophil count may be elevated. The diagnosis can be made definitively by renal biopsy. However, the diagnosis is best established by biopsy of other involved organs such as the skin, muscle, or gastrointestinal tract. Treatment involves discontinuation of anticoagulation, and there may be some benefit from statins or steroids.

Tuberculosis is Not in the History Books; It’s in the History.

SV Nguyen, MD, CS Miller, MD, Tulane University Health Sciences Center, New Orleans, Louisiana.

Learning Objectives:
1. Recognize an atypical presentation of tuberculosis.
2. Understand the diagnostic utility of peritoneal adenosine deaminase.

Case: An 80-year-old woman presented with one month of increasing abdominal girth. Other than mild dyspnea, she had no associated symptoms or further complaints. She had no significant past medical history and had been in good health. She had no risk factors for hepatitis or liver disease.

Her vital signs were normal. She had a diffusely distended, tense abdomen with shifting dullness. A hardened ridge of subcutaneous tissue could be palpated above the umbilicus, but there were no discrete masses. She had no evidence of cirrhosis or chronic liver disease.

Peritoneal fluid revealed a white blood cell count of 125 cells/mm³; 80% lymphocytes. The peritoneal protein was 3.9 g/dL, and the serum ascites albumin gradient (SAAG) was calculated at 0.7 g/dL. Based upon the peritoneal fluid studies, ovarian carcinoma was initially suspected, but cytology of the peritoneal fluid was negative and a computed tomographic (CT) scan of the abdomen showed no ovarian masses, but did show small 3-5 mm masses in the anterior peritoneum and omentum. Tuberculosis (TB) was also considered despite her adamant denial of risk factors. She refused a peritoneal biopsy.

The next day a family member explained that a nephew was treated for TB more than 25 years ago. Our patient and her nephew lived in the same house at that time. The results of her purified protein derivative (PPD) were 17 mm of induration. She was empirically treated for TB, and the peritoneal fluid sent for adenosine deaminase returned positive one week later.

Discussion: Tuberculous peritonitis should be considered in all patients with new onset ascites, lymphocytic ascites, serum-ascites albumin gradient of less than 1.1 g/dL and ascitic fluid protein content of greater than 3.0 g/dL. Adenosine deaminase activity of ascitic fluid has a sensitivity and specificity of 100 and 97 percent for the diagnosis of TB peritonitis when the level is greater than 45 U/L. The gold standard for the diagnosis of TB peritonitis is culture growth of mycobacteria or a peritoneal biopsy under direct visualization.

This is one of many unusual presentations of tuberculosis, but the general internist must keep this disease on his or her radar as it still plagues many of our patients today, especially in poor urban areas. A PPD is positive in 70 percent of TB patients, however a negative result does not exclude the disease. Adenosine deaminase is a useful diagnostic test when the clinical pre-test probability suggests peritoneal tuberculosis.

Pseu, Pseu, Psuedo-Heart Attack.

A Small, B Kunjummen, Tulane University Health Sciences Center, New Orleans, Louisiana.

Learning Objectives:
1. Recognize the complications of valve replacement.
2. Identify the treatment of a left ventricular pseudoaneurysm.

A 62-year-old man presented with a one day history of chest tightness and acute shortness of breath. Four months earlier, he had been diagnosed with methicillin-sensitive Staphylococcus aureus endocarditis and he had undergone...
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an aortic valve replacement surgery. The pre-operative evaluation at that time revealed normal coronary arteries, and normal systolic and diastolic function. One month prior to his current presentation he had a non-ST elevation myocardial infarction (MI) with a troponin I increase to 13.6 ng/mL, with T-wave inversions in V3 through V5.

He had a heart rate of 89 beats per minute; the blood pressure was 104/68 mmHg; and his SaO2 was 97% on room air. He had bibasilar crackles and a II/VI systolic murmur heard best at the apex. He had a moderately elevated jugular venous pressure (JVP) and bilateral pitting edema, but no ascites.

His chest X-ray showed bilateral pleural effusions without evidence of venous congestion. The electrocardiogram (EKG) showed reversal of the T-wave inversions, but no additional acute changes; troponin I levels were normal. A transthoracic echocardiogram revealed the left circumflex artery succumbed to a 90% collapse during systole. Subsequent imaging with computed tomography (CT) angiogram of the chest revealed a pseudoaneurysm that enveloped the left main coronary artery from its origin to the bifurcation of the circumflex and left anterior descending artery, contributing to the circumferential mass effect of these coronary vessels.

The cause of his myocardial ischemia was due to the left ventricular pseudoaneurysm occluding the left coronary artery after aortic valve replacement. An aortic valve replacement and resection of the pseudoaneurysm defect were performed. He tolerated the operation and was discharged on post operation day seven.

Discussion: Access to echocardiographic evaluation has increased, leading to earlier diagnosis of surgically repairable valvular disease. With increased valve surgeries being performed, the general internist must be cognizant of the potential complications of this surgery.

A left ventricular pseudoaneurysm is an abnormal outpouching of the left ventricular wall that arises after damage occurs to the layers of tissue. Typically, pseudoaneurysms are consequences of myocardial infarction, but as this case illustrates, can be a complication of aortic valve replacement. The anatomy of the coronary vessels places them juxtaposed to the aortic ring, and inadvertent compression of the vessels during aortic valve replacement surgery, either intraoperatively or due to scarring after the surgery, can lead to a pseudoaneurysm. Like the pseudoaneurysm seen following an anterior myocardial infarction, reversal of the T wave polarity is a clue to its presence. In our case, clinical suspicion, and the use of newer imaging procedures such as CT coronary angiogram led to the correct diagnosis.
To Whom Much Is Given, Much Can Be Received: The Hyperhemolysis Syndrome.

L Wasson, E Howe, Tulane University Health Sciences Center, New Orleans, Louisiana.

Learning Objectives:
1. To recognize the signs/symptoms of hyperhemolysis syndrome.
2. To understand theories concerning pathophysiology of hyperhemolysis syndrome.
3. To understand the clinical implications and treatment strategies concerning hyperhemolysis syndrome.

Case: A 24-year-old woman with sickle cell anemia presented with menorrhagia. One week earlier, she had stopped her Depo-Provera injections because of breakthrough bleeding, and she noted the menometrorrhagia shortly thereafter. She was evaluated in the clinic and received a blood transfusion to correct her hematocrit to 21%. At the time of admission, she presented with chest pain, left leg pain, and dark urine.

Her heart rate was 120 beats per minute, the blood pressure was 90/50 mmHg, and her temperature was 38°C. She appeared diaphoretic and somnolent, with a hyperdynamic precordium and diffusely tender abdomen. Her lungs were clear, and aside from appearing pale, her extremity examination was normal.

Laboratory tests revealed hyperkalemia, acute renal failure, and an elevated white blood cell count, liver enzymes, troponins, lactate dehydrogenase, D-dimer, and haptoglobin levels. Her hematocrit rapidly declined to a nadir of 12% from her baseline hematocrit of 28%.

Suspecting a transfusion reaction, her blood was analyzed for the presence of antigens and antibodies, both of which were present. She was treated with a transfusion of donor-matched red blood cells that had been washed, filtered, and premixed with intravenous immune globulin (IVIG). She also received fresh frozen plasma (FFP) and cryoprecipitate. Supportive care was initiated with hemodialysis and elective intubation. Her condition improved.

A review of her medical record revealed that she had been in another intensive care unit three years earlier where she had been treated for acute renal failure following a blood transfusion.

Discussion: Hyperhemolysis syndrome is a potentially life-threatening condition characterized by brisk hemolysis following a transfusion. Major histocompatibility reactions are rare outside of outright medical errors in failing to crossmatch the blood to A and B antigens. The general internist must be aware, however, that “minor” histocompatibility reactions due to the surface “i” antigens can induce a serious hemolytic reaction, as was the case in our patient. Severe complications may include acute chest syndrome, congestive heart failure, acute renal failure, and pancreatitis. Signs of this condition include dark urine, decreased hematocrit, hemolysis, and a positive Coomb’s test. The reappearance of similar symptoms with additional transfusions is suggestive of the diagnosis. Patients with sickle cell disease are especially prone to this complication, as repeated transfusions over time result in accumulation of antibodies to the “i” antigens after sensitizing transfusions. In addition to the lysis of the transfused cells, the host cells may also be lysed, and the mechanism by which this occurs is not fully understood.

The most important therapeutic step in the hyperhemolysis syndrome is to quickly recognize the signs and symptoms of this disease, and begin IVIG to sequester the antibodies responsible for the hemolysis. This case illustrates the important of careful vigilance following transfusions, as significant transfusion reactions can occur distant to the time of the transfusion. Those with multiple transfusions in the past are especially prone to this syndrome, and should be observed with extra vigilance.

Thyrotoxicosis induced Dilated Cardiomyopathy in a 25-Year-Old Man.

J Ardoin, MD, Louisiana State University Health Sciences Center, Lafayette, Louisiana.

Abstract: This is a case of a 25-year-old African American man who presented to the hospital with complaints of progressive...
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dyspnea on exertion and bilateral lower extremity swelling for two weeks duration. On further review of systems, the patient reported symptoms of heat intolerance, palpitations, intermittent diarrhea, and scalp alopecia all going back at least eight years. The patient had no past medical history and had never been to a physician regarding any of these symptoms prior to this hospital admission. Vital signs on admit were significant for tachycardia and low-normal blood pressure. On physical exam the patient had massive jugulovenous distention, S3 gallop, tachycardia, bibasilar crackles on lung exam, and 3+ pitting edema extending from the lower extremities bilaterally to the abdominal wall and scrotum. Chest X-ray showed bibasilar edema in lung fields and massive cardiomegaly. Electrocardiogram (EKG) revealed sinus tachycardia. Echocardiogram showed dilated cardiomyopathy with severe systolic dysfunction and a left ventricular ejection fraction of <10%. Labs revealed thyroid-stimulating hormone (TSH) of < 0.01; free T4 of 5.84; T3 of >20; and TSH 264(H). The patient was immediately started on propylthiouracil 200mg by mouth three times daily. Otherwise the patient was initially treated according to heart failure treatment guidelines with volume overload protocol with diuresis and Angiotensin converting enzyme (ACE) inhibitors. Once euvolemia was achieved and patient was symptomatically improved, beta blocker therapy with carvedilol was initiated and titrated up as tolerated. Repeat thyroid function tests showed improving thyrotoxicosis and the patient continued to improve throughout his hospitalization and had no lower extremity edema, clear lung fields, and much diminished JVD. The patient was discharged with cardiology follow up and repeat echocardiogram in three months.

**Conclusion:** This case is interesting because the mechanism of this patient’s heart failure is not the hyperdynamic, diastolic dysfunction that one would expect from hyperthyroid induced cardiac disease. Instead, it is believed that this patient was thyrotoxic for so long that he developed a hypodynamic systolic dysfunction dilated cardiomyopathy. This probably was the result of insult from long standing direct stimulation of the myocardium from thyroid excess compounded by a tachycardia induced cardiomyopathy. In cases of tachycardia induced cardiomyopathy as this, improvement in cardiac function is generally expected once the patient becomes euthyroid.

**Angiosarcoma: A Common Presentation of an Uncommon Disease.**

D Vaidyanathan,^2^ RM Muthuswamy,^2^ I Hasan,^2^ DM Johnson,^3^ Division of Hematology and Oncology,^1^ Department of Internal Medicine, University Medical Center, Louisiana State Health Sciences Center, Lafayette, Louisiana.

Angiosarcoma is a relatively rare sarcoma composed of anaplastic cells arising from vascular tissue. They may arise in almost any area, including the skin, liver, lung, breast, and bone. Due to low incidence and wide range of clinical presentation, angiosarcoma remains an elusive diagnosis, and consequently, many cases are diagnosed only at advanced stages.

We present a case of a 60-year-old man who presented with acute inability to open his right eye and progressively worsening back pain for several months. Other significant history included prostate cancer treated by brachytherapy nine years prior and a 100 pack per year history of smoking. Ophthalmic examination revealed total right eye ptosis with minimal movement of extraocular muscles. Due to his previous history, a Tc-99 bone scan was performed which revealed diffuse uptake suggestive of widely metastatic disease. Staging computed tomographic (CT) of chest showed numerous small bilateral pulmonary nodules, which had increased uptake on positron emission tomographic (PET) scan as well. CT of abdomen and head were normal. MRI of spine was performed, which showed lesions in C3, C7, T1, T4, and L5. A biopsy of a rib revealed a high grade angiosarcoma. The primary site was unclear, and may have either been the vertebrae or possibly the lungs. Multiple imaging including magnetic resonance imaging (MRI) and CT of his brain and orbits failed to reveal a specific cause for the extraocular findings. It was thought this may be due to microscopic invasion or perhaps some form of neoplastic syndrome.

Angiosarcoma is an uncommon neoplasm, accounting for less than 1% of soft tissue sarcomas. Due to its nonspe-
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cific symptoms, it is often recognized only at a later stage. Angiosarcoma tends to behave in an aggressive manner, both locally and with metastatic potential. However, in cases with a known primary and early stage, wide surgical excision with adjuvant radiation may offer good cure rates. Future treatment potentials include anti-vascular endothelial growth factor (VEGF) therapies such as bevacizumab.

**Hereditary Hemorrhagic Telangiectasia.**

RM Muthuswamy, I Hasan, D Vadyanathan, DM Johnson, Division of Hematology and Oncology, Department of Internal Medicine, Louisiana State University Health Sciences Center, Lafayette, Louisiana.

Hereditary hemorrhagic telangiectasia (HHT) is an increasingly recognized disease of vascular malformations that predisposes patients to repeated episodes of mucous membrane bleeding. It is characterized by mucocutaneous telangiectasias, visceral (typically gastrointestinal (GI), but including central nervous system (CNS) and pulmonary) arteriovenous malformations (AVM’s), repeated episodes of epistaxis, and a strong family history of HHT.

We present a case of a 37-year-old man who presented initially in 2003 with symptomatic anemia and repeated episodes of unprovoked epistaxis. He reports a history of unprovoked epistaxis for over 11 years which have increased in frequency to daily over the past 12 months as well as intermittent melena for several years. Fiberoptic examination of his nasal cavity revealed numerous large AVM’s. Careful examination of his oral mucosa revealed several small telangiectasias along his inner lower lip. Upper, lower, and capsule endoscopy performed showed scattered AVM’s in his stomach and jejunum. At this point the diagnosis of HHT was made, and indeed he had a strong history of epistaxis among first degree relatives. This patient has undergone treatment with estrogens, danazol, somatostatin, focused radiation with little effect on his epistaxis. Multiple embolizations of his nasal vessels has temporized his bleeding for short periods of time, however he continues to require intravenous (IV) iron and blood transfusions monthly. A regimen of aminocaproic acid and bevacizumab (an antiangiogenic monoclonal antibody) is currently being administered. This innovative approach has decreased his epistaxis and reduced his need for blood transfusions. This would need further study in refractory HHT patients.

HHT remains a clinically underdiagnosed disease, particularly due to the specificity of the criteria for diagnosis. Recent advances in genomic analysis have identified four-gene clusters responsible for nearly 80% of cases. However HHT is a clinically heterogeneous disease with the most serious complications including pulmonary and cerebral AVM’s. Screening and treatment guidelines still remain under debate, but the literature suggests that this is a disease internists should be more familiar with and consider in any patient with repeated mucous membrane bleeds.

**POSTER PRESENTATIONS**

**Thyroid Storm Presenting with Acute Hepatic Failure.**

DS Hsia, MD, DA Peacock, MD, Department of Internal Medicine and Pediatrics, Louisiana State University Health Sciences Center, New Orleans, Louisiana.

Thyroid storm is characterized by a constellation of signs and symptoms including tachycardia, hyperpyrexia, delirium, heart failure, and gastrointestinal (GI) dysfunction. Acute hepatic failure is a known but uncommon consequence of thyroid storm.

A 78-year-old woman presented to the emergency room with a three week history of decreased oral intake and a three month history of weight loss of approximately 25 kg. The patient denied any previous medical history, blood transfusions, medication use, smoking, and alcohol use. Initially the patient had a heart rate of 192 beats per minute, blood pressure of 190/103 mmHg, temperature of 98.4°F; and weight of 35 kg. On physical exam the patient was disoriented and cachectic. She had mild scleral icterus, dry mucous membranes, and temporal muscle wasting. Her thyroid gland was diffusely enlarged with a prominent mass over the left lobe. Cardiac rhythm was regular with tachycardia, and the chest was clear to auscultation. Her abdomen was diffusely tender with normal bowel sounds and no hepatosplenomegaly. There was no peripheral edema.

Initial lab data showed a thyroid-stimulating hormone (TSH) of 0.08 mIU/L (0.35-5.5 mIU/L), a total T4 of 27.1 μg/dL (4.5-12 μg/dL), and abnormal liver function tests: aspartate aminotransferase (AST) 300 IU/L (10-40 IU/L), ALT 191 IU/L (10-36 IU/L), total bilirubin 4.5 mg/dL (0.2-1 mg/dL), and abnormal liver function tests: aspartate aminotransferase (AST) 300 IU/L (10-40 IU/L), ALT 191 IU/L (10-36 IU/L), total bilirubin 4.5 mg/dL, and INR 1.6. A computed tomographic (CT) scan of her neck showed a markedly enlarged, irregular, nodular thyroid with the left lobe measuring 4.0 x 5.8 cm and the right lobe measuring 3.9 x 2.9 cm. Right upper quadrant ultrasound showed no abnormalities in the liver.

The patient remained tachycardic (131 beats per minute) after three liters of normal saline. She was admitted to the intensive care unit (ICU), and Endocrinology was consulted. Dexamethasone, propylthiouracil (PTU), and an esmolol drip were started. Transaminases peaked on day three (AST 1952 IU/L and ALT 1084 IU/L). After three days of therapy, total T4 decreased by nearly half to 13.9 μg/dL, and all of her liver function tests started to improve. A hepatitis panel and all blood and urine cultures were negative. The patient would require radioactive iodine or thyroidectomy once she became more stable.

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This case illustrates the rare complication of marked liver dysfunction in a patient with thyroid storm. Once thyroid function was controlled, signs of liver failure improved concurrently. It is important to recognize the symptoms, the multiple complications, and the appropriate treatment of a patient with thyroid storm.
An Unusual Case of Encephalitis.

J Johnson (R5), Louisiana State University Health Sciences Center, Internal Medicine and Emergency Medicine Program, New Orleans, Louisiana.

A 24-year-old African American man with no past medical history presented to the Emergency Department with complaint of severe headache, nausea and vomiting and fever. He was diagnosed with a urinary tract infection (UTI) and discharged on antibiotics. He returned to the hospital three days later with worsening headache, intractable vomiting, neck pain and altered mental status.

Physical exam revealed a temperature of 99.2 °F with lethargy and inability to follow simple commands. He also had meningeal signs and evidence of photophobia. Lumbar puncture was performed and was sent for bacterial, herpes simplex, and fungal cultures. Cerebrospinal fluid (CSF) was hazy in appearance and revealed 51 white blood cells, 140 red blood cells, with a differential of 1% segmented neutrophils, 89% lymphocytes, and 10% monocytes.

He was admitted with a diagnosis of encephalitis and started on vancomycin, ceftriaxone, and acyclovir. Although he remained afebrile, his mental status continued to deteriorate. He developed urinary/fecal incontinence, ataxia, and decreased muscle strength. Computed tomographic (CT) scan of head was unremarkable. Electroencephalogram (EEG) showed no abnormalities.

Neurology was consulted and suggested repeat lumbar puncture with viral cultures for arboviruses. While awaiting results of cultures, he developed acute renal failure possibly due to acyclovir. CSF serology demonstrated the presence of West Nile Virus IgG and IgM antibodies. He was diagnosed with West Nile encephalitis and slowly recovered, leaving the hospital with outpatient physical therapy.

West Nile encephalitis is an infection of the brain caused by a flavivirus and is commonly transmitted by Culex mosquitos. The number of cases of West Nile infections continue to increase each year in the United States, with cases being reported in 48 states. Older age remains the major risk factor for developing encephalitis. In 2002, the median age among neuroinvasive disease cases in the United States was 64 years.

Clinical manifestations include fatigue, fever, headache, neck stiffness, muscle weakness and difficulty concentrating. CSF usually shows pleocytosis with predominance of lymphocytes. Protein is universally elevated and the glucose is usually normal. Diagnosis is made through the detection of specific IgM antibody in cerebrospinal fluid. Although CT scan of brain usually doesn’t show disease, Magnetic resonance imaging (MRI) may demonstrate enhancement of leptomeninges or the periventricular area in one-third of patients. EEG typically shows generalized slowing which is more prominent in the frontal or temporal region. Treatment is usually supportive.

Although patients at highest risk are usually over the age of 50, it is important to remember that any age can be affected. This case demonstrates the presentation of West Nile encephalitis in an unusually young patient.

Pictorial Review of the Lung Segmental Anatomy: A Radiological and Bronchoscopic Correlation.

N Vergara, MD, J Ali, MD, FRCP(C), FCCP, Department of Medicine, Louisiana State University Health Sciences Center, New Orleans, Louisiana.

Introduction: Clinical diagnosis in pulmonary medicine requires a working knowledge of the segmental anatomy of the lung and the correlation between different proper diagnostic techniques is helpful. Correct interpretation of the chest X-ray, chest computed tomographic (CT) scan aided by bronchoscopic visualization whenever indicated assists in the specific anatomical localization of an abnormality. Although this topic has been addressed independently in the literature, understanding of this correlation, at times, poses a significant challenge to the clinician. This pictorial review derived from various sources is aimed at creating a comprehensive educational tool for clinicians in this field.

Methods: Selected graphics, diagrams and tables from referenced sources were reviewed and arranged in order to create a pictorial essay.
Discussion: The evaluation of the respiratory system entails a proper history, a comprehensive physical examination and aid of other diagnostic modalities. The expansive, yet tightly compact nature of the broncho-alveolar anatomy makes it difficult for the clinician to accurately and precisely localize lung pathology without correlating these diagnostic modalities. The examination of the chest and physical diagnosis is limited by the poor correlation of surface anatomy, auscultatory findings and radiographic imaging. Intrapulmonary processes may be underappreciated on routine physical examination and pathology in the airways and bronchopulmonary segments are not always identifiable on chest imaging. Differential diagnoses and therapeutic options related to the work-up and management of a pulmonary disorder or disease process depend upon an accurate correlation between these modalities.

Conclusion: This pictorial essay combines the imaging data available for ready reference in pulmonary clinical medicine and could also serve as a helpful educational tool for medical students and trainee physicians.

Rhabdomyolysis in an Immunocompromised Patient with Disseminated Histoplasmosis.

J Beedupalli, MD, R Penn, MD, Louisiana State University Health Sciences Center, Shreveport, Louisiana.

Introduction: Disseminated histoplasmosis in immunocompromised patients may present in a number of ways, including septicemia with multi-organ involvement. We report an unusual case in which rhabdomyolysis was a prominent part of the clinical picture. Previous literature shows varied presentation including septicemia with organ failure, and dermatologic lesions.1-3

Here we report a case of a 28-year-old man with human immunodeficiency virus (HIV)/acquired immunity deficiency syndrome (AIDS) with disseminated histoplasmosis presenting as active rhabdomyolysis that responded promptly to treatment of histoplasmosis.

Case Report: A 28-year-old man with recently diagnosed HIV infection and an absolute CD4+ T lymphocyte count of 3/μL, on no medications, was admitted with generalized myalgias, fatigue, subjective fever without rigors, and cough productive of yellow sputum. He reported modest alcohol consumption, and admitted to occasional crack cocaine use in the past. Examination revealed: Blood pressure 128/74 mm/Hg, temperature 102.7°F, pulse 120 beats per minute, respirations 18/min, and oxygen saturation 99% on room air. He was emaciated with temporal wasting. Small axillary and epitrochlear lymph nodes were palpable bilaterally. The chest was clear to auscultation, and the remainder of the examination, including the neuromuscular system, was unremarkable. Laboratory evaluation revealed: creatinine 1.5 mg/dl, BUN 25 mg/dl, total bilirubin 0.4 mg/dl, AST 374 U/L, ALT 80 U/L, alkaline phosphatase 315 U/L, LDH 2665 U/L, WBC 2960/mm3 with 77% neutrophils and 18% lymphocytes, and platelet count 127000/mm3. Urinalysis revealed large blood on dipstick with no red or white blood cells on microscopic examination. The serum creatine kinase (CK) was 2,013 U/L. Urine myoglobin was 3,321 ng/mL (normal 0-5 ng/mL). Serologic studies for hepatitis were negative. The urine drug screen revealed only marijuana. Cerebrospinal fluid analysis yielded no nucleated cells or elevation of the protein. Computed tomography of the chest showed right middle lobe infiltration. Imaging studies of the brain were normal. He was treated with antibiotics for community-acquired pneumonia and possible Pneumocystis jiroveci infection. Aggressive intravenous fluid therapy with bicarbonate was initiated for rhabdomyolysis. Because of persistent fever, liposomal amphotericin was begun on the third hospital day, and he promptly defervesced. Levels of creatine phosphokinase (CPK), which had remained elevated, decreased to normal over the next five days. Subsequently, he was reported to have histoplasma antigenemia and antigenuria, and blood, urine, and cerebrospinal fluid cultures submitted at admission grew Histoplasma capsulatum.

Discussion: This patient definitely had active rhabdomyolysis on presentation since he had myoglobinuria, which is highly specific and diagnostic of rhabdomyolysis though not very sensitive.3-5 This patient had multiple reasons for rhabdomyolysis, including HIV infection alone. However, he did not have the characteristic symmetrical proximal muscle weakness associated with HIV myopathy.6-7 We believe that his clinical course, with rapid resolution of the process after initiation of appropriate anti-fungal therapy for histoplasmosis,5 is indicative of rhabdomyolysis associated with disseminated histoplasmosis. This entity has been reported rarely in patients with acquired immunodeficiency syndrome.8 In patients with AIDS who present with findings suggestive of unexplained rhabdomyolysis, disseminated fungal infection especially disseminated histoplasmosis should be considered in the differential and prompt anti-fungal therapy along with aggressive hydration may prevent progression to acute renal failure and other life-threatening complications of rhabdomyolysis.

References:
Limb-Shaking Syndrome: A Hyperkinetic Presentation of Transient Ischemic Attack.

S Ittiara, (associate), Department of Medicine, Ochsner Hospital, New Orleans, Louisiana.

Introduction: Limb Shaking Syndrome (LSS) is a unique form of transient ischemic attack (TIA) characterized by seizure-like hyperkinesias resulting from cerebral hypoperfusion and autonomic dysregulation. The atypical presentation of TIA, with similarity to focal motor seizure, poses a diagnostic challenge which can delay appropriate and necessary treatment.

Case Presentation: A 98-year-old man with a history significant for moderate dementia, coronary artery disease (CAD), and recent CVA presents with multiple episodes of convulsions with unresponsiveness. Symptoms were first noticed by his caretaker several days prior to admission, and were described as head shaking, left arm shaking, and staring unresponsive to verbal or tactile stimuli. Each episode lasted approximately 10 to 15 seconds before resolving. There was no tongue biting or incontinence during episodes, nor was there any noticeable post-ictal period. Recent history only revealed one to two days of diarrhea occurring prior to the described symptoms. Review of systems was otherwise negative. Physical exam was benign, aside from left-sided carotid bruit and baseline dementia.

The patient was given a tentative diagnosis of new onset focal motor seizure and admitted for workup. An initial head CT showed no significant abnormalities. A subsequent head MRI also came back negative. The patient underwent extensive laboratory testing for seizure etiology, all of which were negative. An EEG attempting to confirm seizure showed only some nonspecific generalized slowing. The patient continued to have shaking episodes in the hospital and was started on a trial of carbamazepine. Symptoms persisted. Carotid dopplers exposed a state of critical/complete stenosis in the right internal carotid artery and 70-80% stenosis in the left. A head magnetic resonance angiogram (MRA) revealed diminished flow in the right middle cerebral artery and its branches. Additional history confirmed that symptoms occurred mostly with maneuvers inducing orthostasis, such as rising from bed or getting up from a chair. In light of the other negative tests, imaging results combined with the adjuvant history pointed towards a diagnosis of LSS.

This form of TIA was secondary to the patient’s prerenal hypertension with subcutaneous nodules and constitutional symptoms including fever. Though a rare disease, SPTL should be considered in the differential diagnosis of patients with fever of unknown origin and dermatological findings.

Subcutaneous Panniculitis-Like T Cell Lymphoma Presenting as Fever of Unknown Origin.

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Subcutaneous Panniculitis-like T cell lymphoma (SPTL) is a rare cutaneous form of T cell lymphoma. SPTL presents with subcutaneous nodules and constitutional symptoms including fever. Though a rare disease, SPTL should be considered in the differential diagnosis of patients with fever of unknown origin and dermatological findings.

A 39-year-old man with a history of hypertension, diabetes, hyperlipidemia, and chronic renal insufficiency presents with fever, chills, and a rash for three days. The rash is comprised of multiple, one to two centimeter, diffuse, erythematous, nonpainful, and nonpururitic nodules on his arms and legs. These nodules appeared at the same time that the patient began to develop fever and chills. He had temperature spikes to approximately 103 degrees at various times throughout the day. The patient was evaluated by his primary care provider who felt that the nodules were likely erythema nodosum since they appeared three weeks after he started him on Lisinopril and Simvastatin. These medications were discontinued one day prior to admission. Once admitted, the patient was empirically started on Vancomycin and infectious disease, dermatology, and rheumatology were consulted. He remained hospitalized for five days and received a thorough evaluation that included a biopsy of the skin nodules. He continued to have high fevers despite a negative work up. He was discharged home without antibiotics as it was believed that infection was not the etiology of his fevers. The skin biopsy was sent to Mayo Clinic and histological, immunohistochemical stains, and T cell receptor gene arrangement suggested SPTL. The pathologist did remark that additional biopsies were needed for definitive diagnosis. The nodules persisted and the patient continued to have fevers for an additional one to two weeks after discharge. The patient was referred to hospice.
hematology/oncology five weeks after discharge and was afebrile by then. The subcutaneous nodules had resolved at this time so his oncologist elected to observe the patient, not beginning treatment for SPTL. The patient will follow up with his oncologist for repeat biopsy if symptoms recur.

SPTL is rare malignancy that can have either an indolent, recurring course or an aggressive, fatal course. SPTL is a diagnosis that should be considered in patients with fever of unknown origin and skin findings. Multiple biopsies are often necessary to make a definitive diagnosis. These patients should be followed closely by an oncologist because SPTL can have a very poor prognosis and can rapidly become fatal. The most important negative prognostic indicator is the development of hemophagocytic syndrome, which results in hepatosplenomegaly, cytopenia, and organ infiltration caused by phagocytic blood cells.

Radiation Induced Peritoneal Mesothelioma: A Case Report and Review of the National Cancer Institute - Surveillance, Epidemiology, and End Results - SEER Database.

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As advances in treatment of cancer continue to improve survival in patients with cancer, it is increasingly important to understand the long term complications of therapy.

Case: A 54-year-old Caucasian woman presented in July 2006, with worsening indigestion and abdominal pain. She also noted a gradual weight loss and generalized fatigue. She was evaluated by her primary care physician and had a computerized tomography (CT) with a follow-up pelvic ultrasound, which showed some free abdominal fluid. Her past history is significant for left sided primary dysgerminoma of the ovary when she was 23 years old. She underwent left oopherectomy at that time which was followed by 68 fractions (27 to pelvis and 41 days of strip therapy to the abdomen) of radiation. She was initially treated with 2000 cGy to the tumor delivered to the pelvis through a 12 X 14 port. This was followed by strip therapy to the abdomen from the xiphoid process to the symphysis shielding the liver and kidney (2200 cGY was delivered by strip therapy to the tumor). The patient tolerated the radiation therapy well with only minimal diarrhea. Approximately 11 years ago, she had a hysterectomy for benign lesions in the uterus. At that time, there was no evidence of recurrence of her ovarian cancer. A CT scan done in August 2006 showed omental caking, peritoneal deposits, and ascites consistent with peritoneal mesothelioma. She has no known exposure to asbestos. It is likely that her peritoneal mesothelioma is from the pelvic radiation that she received 31 years ago for her ovarian carcinoma. She received six cycles of pemetrexed and cisplatin with stable disease and now continues on maintenance treatment with pemetrexed one year after her diagnosis with no evidence of disease progression.

Discussion: Malignant mesothelioma is a rare and often fatal disease arising from the mesothelial surfaces of the pleural and peritoneal cavities. A majority of patients with malignant mesothelioma have a history of asbestos exposure. The estimated latency between exposure to asbestos and development of mesothelioma is between 20 to 30 years. The increased risk of developing a second malignancy after radiation therapy has been well established. However, the causal role of radiation in mesothelioma has been widely debated. Several preclinical models have reported increased incidence of mesothelioma following exposure to therapeutic doses of radiation. Despite no known exposure to asbestos the possibility of asbestos-associated mesothelioma cannot be completely ruled out completely in our case. Pleural mesothelioma is four to five times more common.

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in men. These differences in incidence could potentially be attributed to both biological and occupational factors. We reviewed all the cases of malignant mesothelioma reported in the Surveillance, Epidemiology, and End results public-use database 1973-2004.

SEER analysis - Methodology: Surveillance, Epidemiology, and End Results (SEER) program is a National Cancer Institute-funded database that has been collecting clinicopathologic data on all incident cancer cases in selected geographic areas of the United States since 1973. From the SEER registry, we identified all cases of malignant mesothelioma ( International Classification of Diseases-Oncology-second revision, morphology code ICD-O-3: 9050/3, 9051/3, 9052/3 and 9053/3). Among these cases, we narrowed the focus to microscopically confirmed primary cancers diagnosed prior to autopsy. There were 9315 such cases. Variables analyzed included age, gender, ethnicity, race, disease stage, histology, and survival. SEER*Stat 6.3.6 software was used to estimate relative survival for all cases of malignant mesothelioma. Relative survival rates were defined as the observed probability of survival (calculated using Kaplan–Meier methods) adjusted for the expected survival rate of the United States population for that age, gender, and calendar year. One, three, and five year survival for pleural and peritoneal mesothelioma were calculated.

Results: There were significant differences in incidence of pleural and peritoneal mesotheliomas in men and women. Pleural mesothelioma is about four times more common in men compared to women. However, the incidence of peritoneal mesothelioma is nearly equal in both men and women. Epitheloid mesothelioma appears to be the most common histological subtype in both pleural and peritoneal mesothelioma. Peritoneal mesotheliomas have a higher proportion of epitheloid subtype than pleural mesotheliomas (79.5% vs. 63%). We found significant differences in the relative survival between pleural and peritoneal mesotheliomas. Men had worse prognosis compared to women and this difference was even more prominent in the patients with peritoneal mesothelioma.

Conclusions: There appear to be significant differences in presentation and outcomes of malignant mesothelioma arising from pleura and peritoneum. We report better prognosis of mesothelioma in women and that arising from the peritoneum. Our results are consistent with previous institutional studies reporting better prognosis of peritoneal mesothelioma, particularly in women. Some studies also report better prognosis in patients with non-asbestos related mesothelioma. Our findings suggest biological differences between pleural and peritoneal mesotheliomas. We hypothesize that these variations can at least partially be attributed to differences in the mechanism of the disease. Non-asbestos related mesothelioma may be more common in peritoneal mesothelioma and in mesothelioma in women. Our findings warrant further investigation of the biology of mesothelioma and the mechanism of onset of asbestos and non-asbestos related mesothelioma.

References:

Sphingomonas paucimobilis Chorioamnionitis.

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We report here the first case of pathologically demonstrated chorioamnionitis due to Sphingomonas paucimobilis. Chorioamnionitis is an infection that involves the amniotic cavity and chorioamnionitic membranes and is associated with 10% to 40% of febrile morbidity cases in the peripartum period. Chorioamnionitis can result in maternal bacteremia, serious post partum pelvic infections, and is associated with an increase in maternal death.

A 25-year-old G5P4 woman with twin intrauterine pregnancy at 31 and 3/7 weeks gestation was admitted to the hospital with vaginal discharge, urinary frequency, lower back pain and fever that had started five days prior. Temperature upon admission was recorded as 101°F. Uterine contractions were occurring every five minutes, causing physical discomfort. She had mild costovertebral tenderness but benign abdominal exam findings. Gynecological exam revealed a 3 cm dilated cervix. Wet prep was unremarkable. Pre-term labor was arrested with intravenous magnesium sulfate. Neisseria gonorrhea was isolated from cervical culture, for which the patient received ceftriaxone intramuscularly. The patient’s fever and abdominal pain continued and amnionecitosis was performed. Sphingomonas was identified from amniotic fluid cultures in the hospital microbiology laboratory and confirmed to be S. paucimobilis by DNA sequencing at the Mayo Medical Laboratory (Rochester MN). The patient’s fever persisted for two more days. A low transverse C-section was performed after failed induction of labor. Pathological evaluation demonstrated early chorioamnionitis of twin B placenta. The patient was treated successfully with cefazolin and gentamicin.

Risk factors for chorioamnionitis include cervical colonization with gonorrhea, preterm labor, ruptured membranes before labor, and young age. Typical causative bacteria associated with chorioamnionitis are Streptococcus agalactiae,
Steroids: Crazy Without, Crazy Within.

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Learning Objectives:
1. Recognize altered mental status as a presentation of systemic lupus erythematosus.
2. Identify the treatment for steroid-induced psychosis.
3. Recognize the complications of treatment of steroid-induced psychosis.

Case: A 42-year-old woman with no prior history of psychiatric disease presented with one day of aggressive and bizarre behavior. Her family discovered her uncharacteristically shouting vulgarities at her children. She was also exhibiting loose thought associations, hyper-religiosity, hyper-sexuality, and agitated behavior. Two weeks before presentation, she consulted an outside physician for “joint pains,” and was given a preliminary diagnosis of fibromyalgia and started on prednisone.

Her heart rate was 102 beats per minute. The remainder of her vital signs were normal. Her physical exam was normal, with no signs of trauma; her neurologic examination was normal. The joint swelling she had previously described was now absent. Her psychiatric examination was notable for non-linear thinking, irrational behavior, and several emotionally charged outbursts.

Her white blood cell count was 3500/ul, the hemoglobin was 7.4gm/dl, and the platelet count was 439,000/ul. A urinalysis revealed significant proteinuria and moderate hematuria. Electrolytes, liver function tests and urine toxicology were normal. A chest x-ray revealed a small left-sided pleural effusion. A head CT scan and the lumbar puncture were normal. An anti-nuclear antibody (ANA) test was positive (dilution 1:640) as well as an anti-double stranded DNA antibody test.

She was diagnosed with systemic lupus erythematosus (SLE), for which steroids were indicated. Because her altered mental status was antecedent to the steroid administration, the diagnosis of steroid-induced psychosis had a higher pre-test probability than acute lupus cerebritis. The prednisone was discontinued, and her altered mental status resolved over the next two days.

Discussion: Lupus is a common presenting complaint for the general internist. Recognizing lupus cerebritis as a cause of altered mental status is important, as over fifty percent of patients with lupus will have a neurologic complication during their lifetime. Recognizing steroid-induced psychosis is equally important, and the internist’s careful history, especially regarding the timing of the altered mental status as it relates to the administration of steroids is the key to distinguishing between these two diagnoses.

The internist must also recognize that relapses are common for patients who have had a previous episode of steroid-induced psychosis. In our patient, systemic complications of lupus such as glomerulonephritis remained, and prednisone alone was not an option for treatment. In this setting, immunosuppressive agents such as cyclophosphamide or mycophenolate mofetil are indicated. Attempts of using corticosteroid treatment with the addition of an antipsychotic has shown promise in a few, isolated studies. Our patient received cyclophosphamide which resulted in resolution of the acute glomerulonephritis.

A Big Belly Breaking the Rules: Ascites as the Presenting Symptom of a Case of Constrictive Pericarditis.

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Learning Objectives:
1. To recognize constellation of symptoms suggestive of constrictive pericarditis.
2. To understand the pathophysiology of constrictive pericarditis.
3. To appreciate the need for early diagnosis and intervention in constrictive pericarditis.

Case: A 48-year-old woman presented with one month of worsening ascites, orthopnea, and shortness of breath. She noted the presence of ascites for the one year; she did not drink or use drugs, and had no history of liver diseases. She noted no additional complaints.

Her vital signs were normal. She had significant JVD, abdominal distention, and bilateral lower extremity edema. Analysis of the ascitic fluid obtained during therapeutic taps directed toward a transudative, non-malignant process and hepatitis panel and liver function tests were normal. Anti-nuclear antibody (ANA) was negative. Chest X-ray showed cardiomegaly with signs of congestion. Liver ultrasound and echocardiogram were normal. CT and MRI of the chest showed thickened pericardium. Constrictive pericarditis was diagnosed as the etiology of the patient’s intractable ascites and the patient was taken for surgical intervention.

Discussion: Constrictive pericarditis symptoms are nonspecific and may be shared with diseases as diverse as myocardial infarction, aortic dissection, and connective
tissue disorders. Because the most impressive physical findings are often the insidious, development of ascites patients are often mistakenly thought to have hepatic cirrhosis or an intra-abdominal tumor, delaying early intervention and treatment.

The associated signs and symptoms of constrictive pericarditis result from a fibrotic, thickened, adherent pericardium restricting normal diastolic filling of the heart include: (1) fatigue, hypotension, and reflex tachycardia (reflecting reduced cardiac output) (2) jugular venous distension, hepatomegaly, peripheral edema (reflecting elevated systemic venous pressure) and (3) exertional dyspnea, cough and orthopnea (reflecting pulmonary venous congestion).

Constrictive pericarditis usually begins as an initial episode of acute pericarditis which may not be detected clinically. This then slowly progresses to a subacute stage of organization and resorption of effusion, followed by a chronic stage characterized by fibrous scarring and thickening of the pericardium with possible concurrent calcium deposition. Etiology of constrictive pericarditis includes idiopathic (nearly half of cases), tuberculosis, post-surgical, post-mediastinal radiation therapy, chronic renal failure, connective tissue disorders, neoplastic pericardial infiltration, inadequately drained purulent pericarditis, fungal and parasitic infections, and acute myocardial infarction. Diagnostic tests for constrictive pericarditis include chest radiograph, EKG, echocardiogram, CT chest, MRI chest, and cardiac catherization. Of note, though most patients with constrictive pericarditis will show thickened pericardium in one of these diagnostic modalities, there are cases when such studies indicate normal thickness of pericardium yet patients had surgically proven constrictive pericarditis. Thus, when clinical, echocardiographic, or invasive hemodynamic features indicate constriction in patients with heart failure, these patients should be considered to have constrictive pericarditis even in the absence of thickened pericardium.

Definitive treatment for constrictive pericarditis is complete resection of the pericardium – a surgery associated with a mortality rate of between 5%-20% depending upon the surgical center. A minority of patients may survive for many years with modest jugular venous distention and peripheral edema controlled by the judicious use of diet and diuretics. However, the majority of patients become progressively more disabled and subsequently suffer the complications of severe cardiac cachexia. Among the patients who survive pericardectomy, symptomatic improvement can be expected in about 90% of such patients with complete relief of symptoms in 50%. Five year survival ranges from 75%-85% with higher survival rates associated with earlier intervention and long-term survival.

Learning Objectives:
1. Understand the pathology behind acute renal failure.
2. Differentiate the causes of acute renal failure.
3. Examine the pathophysiology of abdominal compartment syndrome.

Case: A 69-year-old man was brought to the hospital by his landlord for altered mental status. He reported not having eaten anything or having had anything to drink for four days. He also reportedly had not urinated for several days and complained of difficulty breathing.

His vital signs were normal, as was his head and neck, cardiac, and pulmonary examinations. He had a global depression of mental status, but was able to recognize his name. His abdomen was distended and tight, but not tender, and his liver was not palpable. An ultrasound of his abdomen revealed distended loops of bowel, but no fluid. Flat and erect radiography of the abdomen showed no air-fluid levels and no ruptured viscus or free air in the peritoneum.

His blood urea nitrogen was 294 mg/dL and his creatinine was 12.2 mg/dL. His serum potassium was 7.8 mEq/L and the calculated fractional excretion of sodium was 0.3%. A foley catheter was placed and 200 cc of urine immediately returned. A nasogastric tube was inserted to aid in abdominal decompression and feculent material was immediately returned. He was aggressively volume resuscitated with intravenous fluids and taken to the operating room with a small bowel obstruction. Once his abdominal cavity was decompressed, his renal function returned to normal.

Discussion: Acute renal failure is commonly encountered by the general internist. Pre-renal acute kidney injury develops because of volume depletion, decreased cardiac output, systemic vasodilation, afferent arteriolar vasoconstriction, or efferent arteriolar vasodilation. Compression of the renal vasculature due to abdominal compartment syndrome is frequently omitted from the internist’s differential diagnosis of pre-renal failure, but is an important etiology to consider early, as life-saving interventions predicate upon early detection.

Abdominal compartment syndrome is a surgical emergency and its prompt recognition is essential to avoid life-threatening outcomes. The pathophysiology of abdominal compartment syndrome is marked by a rise in intraabdominal pressure beyond 20 mmHg. This can be a consequence of intraperitoneal changes such as bowel distention, hemorrhage, ascites, or mesenteric venous obstruction, or of retroperitoneal volume changes from aortic surgery and pancreatitis. Short-term treatment of abdominal compartment syndrome with concomitant renal failure involves aggressive fluid resuscitation, bowel decompression, and possibly hemodialysis, but ultimately urgent surgical intervention is required.

Under Pressure: Compartmentalizing Renal Failure.

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Length of Stay in Hospitalized Pneumonia Patients: A Cross Sectional Study.

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Background: Pneumonia was ranked the 8th leading cause of death in the United States in 2005. In 2004, 1.3 million patients nationwide were discharged with the diagnosis of pneumonia and had an average length of hospital stay (LOS) of 5.5 days. We evaluated the effectiveness of our pneumonia protocol and other factors associated with LOS.

Patients and Method: Retrospective chart review of 78 patients hospitalized with pneumonia was performed upon IRB approval. Information on patients’ demographic characteristics, smoking and drug use status, co-morbidity, initial signs of illness, laboratory analyses, initial radiograph findings and antibiotic regimens along with admission and discharge data was obtained. LOS was calculated and was categorized as high (LOS of >4 days) and low (LOS <4 days). LOS was compared in patients who were managed according to pneumonia protocol and patients who were managed otherwise. Statistical significance of trends and differences across categories was determined using least squares and maximum likelihood for continuous and categorical variables, respectively. In a linear regression model age, race, sex, obesity (BMI > 30 kg/m2), smoking status, co-morbidity, difference in time (in days) between initial signs of pneumonia and date of admission, prior outpatient management, bilateral lung involvement and application of pneumonia protocol were included to predict LOS. A multivariate logistic regression was also conducted to calculate prevalence ratio of higher LOS.

Result: Median LOS in overall study patients was 4 days with mean LOS of 5.7 days. However, mean LOS was 4.9 days in patient treated with pneumonia protocol versus 8.2 days who were treated without the protocol (p-trend <0.01). Multivariate adjusted linear regression model showed application of pneumonia protocol reduces LOS by 4 days (p <0.01). After adjustment for age, race, sex, BMI, smoking status and co-morbidity, patients not treated with protocol were 1.68 (95% CI: 0.88 - 3.21) times more likely to have longer LOS, than patients treated with the protocol.

Conclusion: Average daily cost of a hospital room in United States is $212. In our study using pneumonia protocol decreased LOS by approximately 4 days, which decreases the cost of hospital room on average $848 per admission.

Management of pneumonia patient according to the protocol is significantly associated with shorter duration of hospital stay and should be followed strictly to reduce morbidity, mortality and financial burden on patients and the health system in general.

The Hepatopulmonary Syndrome: An Overlooked Cause of Hypoxemia in Liver Disease.

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Hepatopulmonary syndrome (HPS) is an underrecognized cause of progressive oxygen decline in patients with liver disease. It is characterized by hypoxemia and intrapulmonary vascular dilations (IPVD’s) in the setting of severe liver dysfunction.

We present a case of a 48-year-old man who presented to our clinic complaining of fatigue, dyspnea on exertion, and edema of his legs for the previous few months. He had a history of chronic hepatitis C for about five years and alcohol abuse. Upon our examination, it was noted that the patient’s oxygen saturation was 93% while sitting, and 94% when supine. He was also noted to have a jaundiced appearance as well as 2+ pitting lower extremity edema. Lab studies showed a total bilirubin of 14.5, INR 1.3, albumin 2.3, and elevated AST and ALT. As the patient appeared to have hepatic impairment, we began to suspect the possibility of HPS in this person without any obvious pulmonary pathology. Pulmonary function tests revealed decreased diffusion capacity (DLCO) with preserved lung volumes and spirometry. Transesophageal echocardiogram with agitated saline was performed, confirming the presence of shunting (IPVD’s), and thus the diagnosis of HPS. After diuresis, his edema improved, however, the hypoxemia persisted. The patient was referred for liver biopsy and transplant evaluation.

Hepatopulmonary syndrome has been reported in up to 40% of patients being evaluated for orthotopic liver transplant. Progressive hypoxemia is due to shunting, ventilation-perfusion mismatch, and poor diffusion capacity, all secondary to the development of IPVD’s. Recognizing HPS is important in patients with liver disease as this may be a cause of significant morbidity and mortality, and the diagnosis can be made relatively simply in most healthcare facilities. Orthotopic liver transplant may offer a cure to such patients, and HPS is being investigated as an independent indication for transplant. In patients with liver dysfunction and chronic hypoxemia, hepatopulmonary syndrome should be considered as a diagnosis promptly.