American College of Physicians
Internal Medicine / Doctors for Adults

PUERTO RICO CHAPTER ABSTRACT PRESENTATIONS

Oral Presentations

OP-1 **Primitive Neuroectodermal Thoracic Tumor in an Adult: A Case Report**

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A 20-year-old Puerto Rican woman was evaluated at another institution due to progressive shortness of breath and left sided chest pain. Chest radiograph and computer tomography (CT) of the chest showed a large left sided mass. A 17 x 16 x 2 cm encapsulated, irregular mass was surgically resected and the patient recovered without complications. Evaluation by the Armed Forces Institute of Pathology (AFIP) reported an undifferentiated tumor comprised of small blue cells with cystic spaces and focal attempts at rosette formation. The tumor cells were strongly positive for vimentin and the Ewing's marker MIC-2 (CD99, O-3) supporting the diagnosis of primitive neuroectodermal tumor (PNET). No further therapy was given since she was lost to medical follow up.

Five years later, she came to our institution complaining of a two week history of progressive shortness of breath on exertion and left sided dull chest pain. Physical examination showed decreased breath sounds on the left lung field, but was otherwise unremarkable. Laboratory values were normal. CT of the chest revealed an amorphous large lesion extending throughtout the left upper lung field medially and downward throughout the left perihilar compartment into the left lower lung field with post obstructive pneumonitis at the left lung base.

Extensive surgical excision of the tumor was done. Pathological evaluation showed recurrent PNET. Now, five and a half years after initial diagnosis she is undergoing chemotherapy with Cytoxan, DTIC and Adriamycin.

PNET is usually seen in childhood but there are few reported cases in adults. It presents as a painful, rapidly growing mass that can be located in the mediastinum, chest wall or lung. The overall histologic appearance is that of a primitive round cell sarcoma in most cases, but can also present with pseudorosettes suggesting a neuroectodermal origin.

Primitive neuroectodermal tumors should be considered as part of the differential diagnosis of thoracic tumors in the young adult population. Management includes surgical resection, chemotherapy and radiotherapy. Without combination therapy, recurrence is a possibility.

OP-2 **Cold agglutinin disease: Clinical evolution into a Malignant process**

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A 55 years old female had mild anemia on a routine CBC done on 08/00 (WBC 6.0;48.5% lymphs,42.1% PMN). Her hemoglobin decreased to 10 two years later . Direct and indirect Coomb's test were negative. Mycoplasma pneumonia titers were elevated as well as cold agglutinin titers (1:256). On 09/01,she was found to have a polyclonal gammopathy. An autoimmune workup was negative. Hepatitis workup showed positive antibodies against hepatitis C; but HCV RNA was negative. She was diagnosed as autoimmune hemolytic anemia and cold agglutinin disease. Symptoms included purplish discoloration of ear lobes and hands upon exposure to cold temperatures. On 2/27/04 she had relative lymphocytosis with plasmacytoid features and hemoglobin of 11.2, WBC 8.4 (47.1% lymphs). For the first time she was found to have a "M spike"of 1.5 g/dL on SPEP(IgM lambda). Bone marrow aspirate had increase in lymphoplasmacyticelements (>30%)consistent with a lymphoproliferative disorder. Markers on peripheral lymphocytes were CD19 and CD20+ with a small populationCD5+; B cells had Kappa light chain restriction(74:1 ratio) Immunohistochemistry studies on bone marrow biopsy were compatiblewith small lymphocytic lymphoma B cell type. On 6/7/04 she was started on Fludarabine, Cytoxan and Rituxan,decreasing her M spike to 0.9 g/dL.

Cold agglutinins are produced either in response to infection or by neoplastic growth of a single B lymphocyte clone which manufactures the cold IgM antibody

responsible for the hemolysis. Conditions associated with cold agglutinins range from "benign" cold agglutinin disease to low grade lymphoma but high-grade lymphoma has also been described. The intriguing issue in this case is that the patient initially presented with a benign polyclonal gammopathy, perhaps triggered by Hepatitis C virus and aggravated by M. pneumonia, and with time evolved into a monoclonal gammopathy associated to a lymphoma that had a different light chain restriction to the serum M spike. A similar situation is known to occur with gastric MALT lymphoma in which H. pylori stimulation initially leads to a benign gastritis with lymphocytic infiltrate but with time can evolve into a low grade lymphoma and later on to an aggressive large cell lymphoma.

OP-3 **Sildenafil And Bosentan in Acute Secondary Pulmonary Hypertension: Case Report**

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A 67 year old man with history of arterial hypertension, COPD and 50 pack/year history came to our institution with a one week history of worsening shortness of breath and non productive cough. Upon initial physical examination, he was acutely ill, with use of accessory muscles, and with diminished breath sounds. His arterial blood gases showed a PaCO2 of 115 mm Hg, for which the patient was admitted to the ICU and placed on non-invasive ventilation (NIV), since he elected not to be intubated. After a few days of treatment, including antibiotics and bronchodilators, there was no improvement. An echocardiogram showed severe pulmonary hypertension with a systolic PA pressure of 77 mm Hg and right ventricular dilatation and D-dimers were found to be elevated. A ventilation-perfusion scan was of intermediate probability for pulmonary embolism, and a venous Duplex scan was negative. In lieu of the above findings, and with the working clinical diagnosis of acute pulmonary embolism, the patient was treated with thrombolytic therapy. A repeat echocardiogram showed mild improvement in pulmonary pressures, but the patient remained NIV-dependent.

While receiving heparin, he developed heparin induced thrombocytopenia for which he was started on coumadin; and an IVC filter was placed. Despite anticoagulation, the patient remained fully dependent on NIV. Following reports of treatment of secondary pulmonary hypertension with sildenafil and bosentan, the patient was started on these medications. After one week of treatment, the need for ventilatory assistance diminished, until NIV was finally discontinued . He was transferred to a medical ward on low flow O2. He was recently evaluated on the ambulatory setting, where a repeat echocardiogram showed significant improvement in PA pressures ,pulmonary artery systolic pressure of 57 mmHg, with decreased CO2 to 51 mm Hg. He is able to perform all activities of daily living independently.

Prospective randomized clinical trials of sildenafil, endothelin receptor blockers, alone and together**,** and as well as other therapies are needed to further define the role of these drugs in the therapy of acute secondary pulmonary hypertension such as the one this patient had.

OP-4 **Pulmonary Edema During A Triathlon**

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A 36-year-old male athlete, a secretary in a pharmaceutical company, developed shortness of breath (SOB) during the first section of a triathlon race. He requested a life-saver to complete the remaining 1,800 meters. He heard inspiratory stridor, coughed intensely around 16 times and spitted bloody mucus. A sudden episode of SOB and coughing had occurred three weeks earlier while training in the swimming pool. A stress test 2 years previously was normal.

On arrival the temperature was 36.1ºC , heart rate 93, respiratory rate 21 and blood pressure 156/86. While breathing with a 50% ventury mask, the pulse oxymeter showed a 90% saturation. There was no jugular distention. The lungs showed basilar crackles. The heart had a regular rhythm, no murmurs or gallops. There were no neurological deficits.

The arterial blood with an FIO2 of 50% showed a pH of 7.41, a pCO2 of 40.2 mmHG and a pO2 of 50. The chest films and the CT scan showed bilaterally heterogeneous ground glass pattern. Th Hgb was 14.6 g/dl, the white count was 10,800 cells/mm3, and platelets 236,000. The glucose was 116 g/dl, creatinine 1.1, sodium was 139mEq/L, potassium 4, chloride102, and HCO2 27. The urinalysis was normal. The sedimentation rate was 6 mm/hr. D-dimmer test was negative. The total CK was 134, CK-MB 5.8 with a relative index 4.3. The troponine was 1.33 ng/dl 6 hrs after admission. Electrocardiograms were within normal limits. The 2D-echo showed an ejection fraction of 65% and a mild inferior wall hypokinesis. He was started on Rocephin 1 gram IV BID, Zitrhomax 250 mg IV daily and Protonix 40 mg IV daily. He received 50% oxygen via ventury mask for the first 24 hours and Lasix 40 mg IV every 8 hours. The next day while breathing room air the

pO2 was 90 mm/Hg and the chest films showed clear lungs. A CT-scan with contrast of the lower extremities did not show thrombosis of the proximal veins and a myocardial scan showed no ischemia. The bronchial washing showed many red and white cells and some pigment laden macrophages. Exercise test with Sestamibi 2 weeks after discharge was negative for ischemia. Pulmonary function tests showed a slight restrictive defect.

Stremous exercise in athletes may cause ischemic injury, myocardial dysfunctions and pulmonary edema.

OP-5 **Postprandial Hypotension In A 77 Y/O Female With Hypertension.**

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This is a 77 year old female $G_9P_6A_3$, with hypertension, osteoporosis, osteoarthritis, and dyslipidemia. Past medical history is relevant to smoking 2-3 cigarettes daily for 20 yrs, having quit 35 yrs. ago. At the day of the first evaluation she was taking the following medications: raloxifene 60mg qd, atorvastatin 10mg qd, ramipril 5mg qd, and multivitamins qd. The patient comes to the Geriatrics Clinic complaining of a year of evolution of episodes of postprandial dizziness, drowsiness, giddiness, and blurred vision. She had taken her blood pressure during the episodes, and had noticed a drop in blood pressure. The symptoms lasted between minutes to an hour. They were relieved with bed rest and drinking coffee. She denied syncope, loss of consciousness, chest pain, palpitations, or shortness of breath. She had visited many doctors with this complaint and was even offered a Psychiatry consult. Physical exam was unremarkable, except for blood pressure (BP) = 190/100. No orthostatism was evoked. Her routine labs were unremarkable except for the following: total cholesterol 251 mg/dl, triglycerides 215 mg/dl, VLDL 43 mg/dl, LDL 153 mg/dl.

On follow/up appointments the patient brought a chart of pre-meal and post-meal blood pressures. The following is a sample of BP's taken before and after meals at different time intervals:

	BREAKFAST	LUNCH
PRE-MEAL	143/81	124/70
POST-MEAL	108/60	88/56

The process of aging is associated with changes in the cardiovascular system that may have different consequences. Our patient is presenting with what has been described as postprandial hypotension (PPH). PPH is a recognized cause of falls and syncope in the elderly, as well as angina or stroke. PPH is defined as a drop in systolic BP of at least 20 mmHG after a meal. Several etiologies have been proposed including excessive splanchnic blood pooling, sympathetic response impairment, and neurohumoral mechanisms. Treatment may consist of eating small low carbohydrate meals, avoid low salt diet, caffeine, and octreotide. All treatment options have been tried in this patient, except for the use of octreotide.

A literature review for PPH shows consistency in co-morbid conditions; 99% of these patients have autonomic dysfunction. Primary physicians taking care of elderly patients should increase their awareness about this condition and alternatives for treatment.

OP-6 **Hypokalemic Periodic Paralysis in a Young Female with RTA Type I**

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A 40 year old female was brought to ER after developed a sudden onset of generalized weakness while at home. She refers the weakness was principally involving her legs and arms. Her husband refer that the patient loss consciousness for approximately 2-3 minutes during the event but complains that the weakness continued for several hours. Denied headache, convulsions, relaxation of urinary or anal sphincter. Denied chest pain, palpitations, shortness of breath or coughing, also denied nauseas or vomiting during the event. She denied family history of neuromuscular disease or family history of electrolyte disturbance.

On physical exam: BP: 120/70, P: 76, R: 17, T: 37.2°C, WT: 170, HT: 5´6". Patient was alert, hypoactive and oriented in person, place and time. Neurological examination revealed the motor force in 3/5 at upper and lower extremities bilaterally. There were no sensory deficits and reflexes were 1 + on brachioradialis and 1+ on patellar.

Routine laboratory test displayed a metabolic acidosis, hypokalemia of 2.7 mmol/L, Hyperchloremia (117) with elevated BUN (21) and creatinine (2.4), sodium and glucose levels were normal. CBC was normal, CPK, PT, PTT, INR were within normal limits, urinalysis showed a SG: 1.00, PH: 7.00, Leu: ++, BLD: 10 UL, WBC: 10-15, Bacteria Few. Brain CT, carotid Doppler, chest x ray and EKG were normal.

The patient was treated with IV administration of potassium and Bicarbonate. At 48 hours of been admitted, patient was discharge home with normalized potassium levels correction of the metabolic disturbance and without symptoms.

This case is particularly interesting because it showed us a rare disorder in a patient that also is presenting with Renal Tubular Acidosis type I. Hypokalemic periodic paralysis occur as either inherited (primary) or acquired (secondary) illness, because there is no family history of hereditary periodic paralysis, this case probably is due to secondary causes of periodic paralysis as is urinary potassium wastage. The goals of treatment of Hypokalemic periodic paralysis are correction of potassium levels, relief of acute symptoms and prevention of further attacks. Potassium that is given during an attack may stop the attacks, a low carbohydrate diet and avoidance of alcohol are of help. Acetazolamide prevents attacks in many cases but triamterene or spironolactone may help to prevent attacks in people who do not respond to acetazolamide.

OP-7 **Hepatopulmonary Syndrome.**

Guibel Perera, MD; George P. Fahed, MD; Jose Luis Nieves, MD; William Rodriguez-Cintron, MD; Alfonso Torres Palacios, MD.

Introduction: In view of the elevated incidence of hepatic disease in western society, chest physicians should screen for hepatopulmonary syndrome (HPS). Estimates of HPS prevalence among patients with chronic liver disease vary‡. This syndrome is characterized by progressive hypoxemia which leads to deleterious physiologic effects which may worsen the prognosis of chronic liver disease patients. Case presentation: We report a 56 y/o man who had been well until day of admission was seen at the emergency room for complaints of sudden onset bilateral tonic-clonic movements, loss of sphincter tone, and unresponsiveness. These episodes occurred twice, lasting 10 minutes, and were followed by transient quadri-paralysis and respiratory distress; there was interval somnolence between the episodes. There was no history of previous pulmonary disease, trauma, fever, chills, or recent drug or alcohol use. The history was notable for schizophrenia, untreated hepatitis C, and previous alcohol, tobacco and cocaine abuse. His only current medication was olanzapine. In the ED, the patient was intubated, placed on ventilator support, and treated with phenytoin for new onset seizures. Physical exam was remarkable for lethargy, marked clubbing, isolated thoracic wall spider nevi, and rigid extremities. Chest x-ray showed mildly increased symmetrical vascular markings. While breathing 100% O2 on the ventilator, the SpO2 failed to rise above 96%. Chest radiography failed to show presence of infiltrates, consolidates, pneumothorax; pleura and mediastinal structures were unremarkable. The serum Na+ was 118 mmol/dL, and there was a high-anion-gap metabolic acidosis (AG =26); the white blood cell count was 19.5 x 10x3 cells/cu mm, and the total CPK was 13,000 U/L. Liver function tests, hemoglobin, platelet count, glucose, and coagulation times were normal. After correction of serum electrolytes the patient regained consciousness, remained free of seizures and was subsequently extubated. Hypoxemia with a wide A-a gradient (622 mmHg) and orthodeoxia (SpO2 88% while in the supine position, decreasing to 79% upon assuming an upright posture) were found. The diagnosis of psychogenic polydipsia with severe hyponatremia causing new onset seizures was made. HPS was considered to be the cause of hypoxemia in this patient. An agitated saline contrast enhanced 2D echocardiogram, chest CT scan, and a tagged-albumin V/Q scan were done, all showing intrapulmonary shunting with intrapulmonary vascular dilatations, which confirmed the diagnosis of HPS. Discussion: HPS is frequently related to any type of chronic liver disease. There has been no consistent relationship between biochemical indicators of hepatic dysfunction or Child-Pugh classification and either the severity of hypoxemia or shunt¨. Impaired arterial oxygenation is the hallmark of HPS even in the absence of parenchymal pulmonary disease. Hypoxemia in these patients can be life threatening and can progress without an associated decline in liver function. It is also an indication for liver transplant in severe cases¨. This syndrome should be suspected in any patient with platypnea and orthodeoxia in the absence of associated cardiopulmonary disease. Its diagnosis can be made early at bed-side, is simple, and requires only minimally invasive procedures including DLCO*. Conclusion: HPS is usually under diagnosed in the early stages and its presence prompts more aggressive treatment of the underling liver disease†.

OP-8 **Atypical Presentation of Disseminated Histoplasmosis in a Liver Transplant Recipient.**

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Infections in immunocompromised hosts can have atypical presentations or can disseminate more often. We report the case of a 53 year-old Puerto Rican male with diabetes, hypertension, chronic kidney disease, who had liver transplant in 2001 due to chronic liver disease secondary to hepatitis C virus infection. He complained of recurrent episodes of fever (101-102 degrees Fahrenheit). The episodes occurred weekly at the beginning, but then progressed to occur daily. In addition to fever and chills, he complained of general malaise, weakness, anorexia, and weight loss. CBC showed normochromic, normocytic

anemia (hemoglobin 8.8 g/dL) and monocytosis (24% monocytes).

The patient was using tacrolimus and sirolimus, and he was under evaluation for chronic transplant rejection and hepatitis C reactivation because of mild elevation in transaminases (AST 88 units/L, ALT 88 units/L) with increased bilirubin (1.9 mg/dL), GGT (>2000 units/L), and alkaline phosphatase levels (418 units/L). In view cholestatic/ granulomatous pattern of liver dysfunction, liver biopsy was done, but it failed to show rejection, cirrhosis, or granuloma formation. Special stains of liver biopsy were also negative.

Extensive work-up for opportunistic infections was done, of which serum histoplasmosis antigen was positive (2.46). The patient was admitted for treatment with intravenous amphotericin B for suspected disseminated histoplasmosis. Three days after starting therapy with antifungal agents, fever subsided. He was later changed to itraconazole and discharged home, where he continues with resolution of fever, chills, weight loss, and anorexia. Upon follow up, there has been diminution in GGT levels and transaminases.

Histoplasmosis is the most prevalent endemic mycosis in the United States and it is also endemic in Puerto Rico. *Histoplasma capsulatum* polysaccharide antigen may be detected in the blood (>75%) and in the urine (>90%) of patients with disseminated histoplasmosis. Antigen detection has a very high specificity (at least 98%) and in disseminated disease sensitivity approaches 92% in urine and 82% for serum samples.

Improvement in liver function tests after treatment with antifungal agents suggests that granulomatous liver dysfunction in this patient with a positive serum histoplasma antigen should have been secondary to histoplasmosis. Although cases of obstructive pattern of liver dysfunction secondary to histoplasmosis have been reported in literature, it is relatively uncommon and represents an atypical presentation of disseminated histoplasmosis.

OP-9 **A Case of Flaccid Paralysis in a Patient With HIV: Cmv Polyradiculomyelopathy.**

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Central Nervous System (CNS) infections in severely immunocompromised patients include a wide variety of etiologies and most can occur with unusual presentations. We report the case of a 40 year-old female patient with hypertension and AIDS (HIV diagnosed 20 years ago; last CD4 count of 0/mm^3 and VL >100,000 copies) on highly active antiretroviral therapy. She complained of headache (mostly in right parietal area), associated with nausea, vomits and photophobia for a month prior to admission.

Approximately 3 weeks before admission, she developed bilateral lower extremities paresthesias and right facial paralysis. Shortly afterwards, a Foley catheter was placed by Urology specialist because of urinary retention. Days before admission she noticed some weakness in lower extremities, which later progressed to inability to walk or move.

Upon evaluation, she was found with right peripheral cranial nerve VII palsy. Motor examination revealed flaccid areflexic paraplegia with an associated sensory level at T4. There was a neutral plantar response to Babinski bilaterally. Both CT and MRI with contrast enhancement of brain and thoracic spine were negative. Cerobrospinal fluid (CSF) analysis revealed: White Blood Cells 118 (83% polymorphonuclear cells), Red Blood Cells 3,496, Glucose 23 mg/dL, protein >500 mg. CSF India ink, gram stain, KOH, acid fast, culture, cryptococcal antigen, toxoplasma, rubella IgG, Herpes 1 and 2 IgG were all negative.

In view of Bell's palsy, meningeal symptoms, thoracic sensory level, and flaccid areflexic paralysis, pleocytosis with neutrophilic predominance, hypoglycorrhagia, and elevated protein in CSF, cytomegalovirus (CMV) infection was considered and the patient was started in ganciclovir intravenously. CMV IgG in CSF was later found to be positive in our patient. CMV DNA was detected in CSF by PCR technique, while PCR for Herpes Zoster and Herpes Simplex I and II was negative. This is highly specific of CMV neurologic disease, which was already suggested by clinical presentation and findings.

CMV neurologic disease is a serious complication of AIDS, more common in patients with CD4 cell count below 50/mm3. CMV can produce encephalitis, myelitis, peripheral neuropathy, and, like in this case, polyradiculomyelopathy with progressive Guillain-Barré-like ascending paralysis and urinary retention. CSF can show polymorphonuclear pleocytosis with hypoglycorrhagia. Treatment with ganciclovir or foscarnet, or both, has resulted in improvement in some patients, but prolonged or indefinite therapy is required.

OP-10 **Unsual Cause of a Prolonged Bleeding Time in an Elderly Female.**

Ismael Labrador, MD; Robert Hunter, MD, FACP

We present a patient that exemplifies the issues and controversies regarding preoperative cardiovascular risk assessment in the elderly patient. This is a case of a 77 year old female who underwent a dipyridamole pharmacologic stress test in relation to a planned surgery for a new parotid mass. As part of the preoperative blood

evaluation a bleeding time was found to be prolonged (11 minutes) with a second test performed 96 hours later also prolonged (14 minutes).Hematological consultation was requested in which congenital coagulopathy (Von Willebrands disease),cutaneous (skin atrophy) abnormalities and endocrine (hypothyroidism) dysfunction were exclude as possible etiologies for prolonged bleeding time. In addition the absence of a history recent medication ingestion which may alter platelet function was also excluded .A review of dipyridamole dosage used in the stress test was reviewed and found to be 0.14 mg/kg per minute for four minute (total dose of 40 mg) which may have been responsible for the observed prolongation of the bleeding time. Dipyridamole is a systemic vasodilator with important inhibition of platelet function. The medication inhibits platelet phosphodiesterase activity and cause platelet dysfunction and a prolonged bleeding time .In our patient a repeat test, 10 days later was normal .This clinical case illustrates the role of dipyridamole as a cause of a prolonged bleeding time. Several studies have suggested that dipyridamole-thallium scintigraphy is the ideal preoperative screening test for evaluating risk of cardiovascular events .For this reason and recognizing the potential effects of dipyridamole on platelet function we suggest a period of at least 7 days between the stress test and the evaluation of coagulation parameters. Ours patient underwent the planned intervention and continues to do well.

A Bluish Face

OP-11 Jorge E. Garayúa, MD (associate), José Ramírez Rivera, MD, FACP Universidad Central del Caribe, Hospital Universitario Ramón Ruiz Arnau, Bayamón, Puerto Rico.

Methemoglobin is formed when the iron in the hemoglobin is changed from the ferrous to the ferric state. The displacement of oxyhemoglobin may cause tissue hypoxia. While to see cyanosis requires 5g of desaturated hemoglobin, a concentration of 1.5 g per deciliter of methemoglobin is sufficient to cause a bluish face. A 46-year-old man with a mayor depression disorder was brought to emergency room (ER) unconscious. On the day of admission, he had drunk alcohol and ingested 30 pills of diphenhydramine, 30 pills of haloperidol, 20 pills of dolagesic, 20 pills of cyclobenzaprine, 20 pills of naproxen, 14 pills of cephalexin, and 48 pills of chlorzoxazone. A gastric lavage had been performed. He was on mechanical ventilation, and responded only to painful stimuli. Five hours later his face, hands and feet became bluish. The pulse oximetry revealed an SpO2 of 85%. The dark chocolate-color arterial blood showed a PaO2 of 290.8 mm Hg and an oxygen saturation (SaO2) of 99%. After the FiO2 was increased to 100%, the PaO2 was 554.2 mm Hg and the SaO2 99.9%. An unchanged SpO2 suggested the diagnosis of methemoglobinemia. One mg per Kg of intravenous methylene blue was administered in 5 minutes. Twenty minutes later, the cyanosis began to fade and the SpO2 was __. One hour later, the cyanosis disappeared and the SpO2 was 99%. Early treatment of methemoglobinemia is crucial in preventing tissue hypoxia. In the cyanotic patient, the prior medical history, the low saturation in oximetry and normal blood gases, are clues for making the diagnosis. Methylene blue is the treatment of choice for severe methemoglobinemia. This agent is an effective electron donor for NADH methemoglobin reductase, providing sufficient reducing power to eliminate most methemoglobin from the red blood cell. The initial dose of methylene blue is 1-2 mg/kg of a 1% solution administered over 5 minutes. Reduction of methemoglobin usually is complete in 1 hour. If methemoglobinemia persists, a second dose not to exceed a total dose of 5-7 mg/kg may be administered. Because headache, nausea, vomiting, diarrhea, angina, and discoloration of skin and urine may occur with therapeutic doses, methylene blue should only be administered to those patients with signs of hypoxia. An asymptomatic bluish face is not an indication for methylene blue.

Total Blindness In A Young Woman With Recurrent Deep Venous Thrombosis.

OP-12 Loyda R. Ayala-Rosado, (Associate), José Ramírez-Rivera, MD, FACP, Lynnette Ortiz-Toro, MD, Axel Velez-Santiago, MD, FACP, Francisco Jaume-Anselmi, MD, FACP, Hospital de La Concepción, San Germán, Puerto Rico.

A 32-years-old obese woman with 3 previous episodes of deep venous thrombosis (DVT) over the last year developed right leg DVT associated with cellulitis. She was treated with antibiotics and warfarin. Two days later she returned to the hospital with constant shoulder and left arm pain, a severe headache, progressive decrease in visual acuity and hematuria. There was no chest pain or shortness of breath. The temperature was 36.2ºC, heart rate 75/min, respiratory rate 22/min and blood pressure 110/80 mmHg. She weighed 255lb, with a height 64 inches and had a BMI of 43kg/m2. She was alert and oriented but pupils were dilated and did not react to light. There was bilateral papilledema but no hemorrhage or exudates. She had a regular cardiac rhythm and no murmurs. The lungs were clear. The liver and spleen were not palpable. There was no neurological deficit except she could only see light.

The Hgb was 9.7gm/ml, the white cell count 18,100 with 67% neutrophils and 12% bands. The platelets count 339,000. The sed rate was 60 mm/hr. The blood sugar was

121 mg/dl and the creat 0.6 mg/dl, Ca 9.5, PT 18.9/12.7, INR 2.35, PTT 32.8/26.3. The urinalysis showed 30mg/dl of protein, WBC 0-1/hpf, and RBC 28-30/ hpf. She was given Rocephin 1 g daily, Solumedrol 80mg IV x 3 then 1 g daily x 3. Two days after admission she was completely blind. An MRI showed complete opacification of the right and partial opacification of the left sphenoid sinus, and solid densities extending into the right temporal fossa . The cerebrospinal fluid showed no cells, a glucose 67 mg/dl with protein 38.8 mg/dl (blood glucose 101 mg/dl) . The CSF culture was negative after 5 days. HIV ELISA was non-reactive.

Five days after admission 2/3 of the right lower chest showed dullness and there were no breath sounds. The pleural fluid demonstrated WBC 69 cell/mm3, RBC 213 cell/mm3, pH 7.5, glucose 104 mg/dl, total protein 5.2, lactic dehydrogenase 289 and amylase 21. No yeast or fungal elements were seen. Chest CT scan showed a large right sided pleural effusion with a consolidation in the right middle lobe with air bronchogram and no hilar or mediastinal masses. Multiple lytic lesions involved the vertebral bodies, the thoracic spine, the left scapula and proximal left humerus consistent with a neoplastic process. A sphenoid sinus biopsy showed adenoid cystic carcinoma with acute suppurative inflammation containing septated fungal hyphal elements. The patient died 26 days after admission. Autopsy was not authorized. Adenoid cystic carcinoma is the most common adenocarcinoma of the upper respiratory passages. This carcinoma tends to spread along nerves sheaths, when it invades the optic nerves causes total blindness. In the presence of recurrent DVT suggesting a hypercoagulable state there should be an aggressive search for hidden malignancy.

Necrotizing Pneumonia Due to Macrolide Resistant Streptococcus Pneumonae.

OP-13 Luis Ortiz-Muñoz, MD; Marjery Lopez, MD; Miguel Boque, MD; J.J. Gutierrez-Nuñez, MD, FACP–Veterans Administration Medical Center, Puerto Rico

Background: In the last two decades strains of S. pneumoniae has shown resistance to the most commonly used antibiotics. Complications such as decrease in bacterial clearing from lung tissue with consequent necrosis and cavitations of lung parenchyma has been associated with antibiotic resistant isolates.

Case information: Case of a 21 y/o Puerto Rican female, resident of Kentucky, while visiting relatives in Puerto Rico developed common cold symptoms treated with over the counter medications. She progress with non-quantified fever, dry cough, diarrhea, anorexia and shortness of breath upon exertion and was treated with azithromycin. The day of admission patient was found to be febrile, in respiratory distress, hypoxemic, with bilateral alveolar consolidation requiring orotracheal intubation and mechanical ventilation. She was admitted to the ICU, where she was treated for ARDS, hemodynamics were compatible with a hyperdynamic state, consistent with sepsis. After appropriate cultures, including bronchoscopy with BAL patient was started on broad-spectrum antibiotics. Blood and sputum cultures were positive for S. pneumoniae resistant to macrolides. Despite aggressive therapy patient continued to deteriorate, with no improvement of oxygenation, presenting with bilateral spontaneous pneumothorax with further hemodynamic deterioration, and no improvement after bilateral chest tubes were placed. An overwhelming septic state ensued, with multiorgan failure including anuric renal failure, DIC and alveolar and gastrointestinal hemorrhage requiring large amounts of blood transfusions. That same day patients pulmonary hemorrhage worsened as well as oxygen saturation, she complicated with another episode of cardiac arrest, which progressed to death. An autopsy was done which showed multiple bilateral lung abscesses with cavitations and extensive alveolar hemorrhage.

Discussion: Macrolides should not be consider as an empiric therapy for pneumonia by S. pneumoniae when the patient is a resident from area where the prevalence of macrolide resistance is high.

Huge Atrial Myxoma: an Unusual Cause of Atrial Fibrillation

OP-14 Magda E. Sánchez, M.D. (Associate), José Kareh, M.D., Cid S. Quintana, M.D., Enrique Carrión, M.D. University Hospital, Puerto Rico Cardiovascular Center, San Juan, PR

Atrial fibrillation (AF) is the most common sustained arrhythmia in the general population. It is usually associated to advanced age, hypertension, coronary artery disease, heart failure and valvular heart disease. This case presents atrial fibrillation caused by a huge atrial myxoma.

A 53-year-old female developed palpitations as her sole symptom. She was diagnosed with atrial fibrillation by her primary physician and started on digitalis and beta-blockers. Despite treatment the patient's condition worsened over time now developing shortness of breath.

Patient sought further medical attention at the Puerto Rico Cardiovascular Center being admitted with the diagnosis of AF with a fast ventricular response. As part of her initial evaluation a transthoracic echocardiogram showed a huge mass occupying the entire left atrium and protruding into the mitral valve orifice. The patient was referred to cardiothoracic surgery undergoing a right

atriotomy plus partial left atriotomy for resection of the mass and of the interatrial septum. The atrial septal defect was closed with a pericardial patch and there were no residual defects detected by intraoperative transesophageal echocardiography. Postoperative recovery was uneventful and she was discharged home 5 days after the surgery. The pathologic report confirmed the mass to be a myxoma that measured 7cm x 5cm x 3.5 cm. The patient has remained free of symptoms and with sinus rhythm since the time of surgery.

Atrial myxomas are benign tumors of the heart that most commonly occur in the left atrium. Patients usually present with dyspnea, orthopnea, fatigue, syncope and may develop sudden cardiac death absent in this patient. This case report shows the importance of the echocardiogram as a simple, valuable and non-invasive tool in the workup of patients presenting with cardiac arrhythmias.

OP-15 **End Stage Renal Disease: a Dreaded Complication of the Use of a Chinese Herb**.

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A 59 year-old man with a history of PUD, esophageal hernia, hypertension, complained of nausea, bad taste, general malaise, decreased appetite, polyuria and polydypsia during the last month. He referred bilateral swelling of both legs and these symptoms appeared and worsened gradually.

The patient refers longstanding use of natural herbs and multiple natural supplements that, although he cannot recall their names, he used them to relieve the above symptoms. He also referred concomitant use of NSAIDS during this time. Physical exam was relevant for decreased sensation over both lower extremities, and bilateral leg edema. Cardiovascular, lung and abdominal exam were essentially negative. Kidney Sonogram failed to show obstruction and kidney size was normal. Labs on admission showed Crea=7.9mg/dl, BUN=66.5 mg/dl, CO2=26.5 meq/l, K=3.7 meq/l, Na=141 meq/l and Cl=104meq/l. U/A showed 4+ protein, mild pyuria but no RBC casts. 24 hr urine protein was 21 gms/day. The initial impression to explain the renal failure was that of acute glomerular disease. Therefore, a kidney biopsy was done and it showed focal segmental and global glomerulosclerosis with acute and chronic tubulointerstitial inflammation with fibrosis. Further serologic work-up for other systemic causes of acute renal failure was negative.

During his hospital stay, he required treatment with dialysis and he has not recovered renal function. The clinical picture and particularly the classic kidney biopsy findings suggest that the cause of the renal failure was the use of Chinese herbs contaminated with Aristolochic acid (AA)-induced nephropathy. The causal role of AA in the so-called Chinese herbs nephropathy (CHN) has been conclusively demonstrated only in the Belgian epidemic. We report a biopsy-proven hypocellular interstitial fibrosing nephropathy in a patient who had ingested a Chinese herbal preparation bought in Puerto Rico. Because the ingested preparation is very popular as an over-the-counter product, our observation raises the possibility that many such cases due to AA-induced nephropathy might be currently passed unrecognized. AA should be banned from herbal preparations worldwide.

OP-16 **Transverse Myelitis in an Immunocompetent Patient After an Episode of Trigeminal Herpes Zoster: Case Report and Literature Review.**

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Transverse myelitis as a consequence of herpes zoster has been occasionally reported in medical literature, most cases observed in immunocompromised patients and in the elderly. We report the neurologic complications of a young, immunocompetent patient after suffering an episode of trigeminal herpes zoster. A 29 year old male with history of recently diagnosed diabetes, well controlled with oral medication, was admitted in May 2004 due to abdominal pain, nausea, vomiting, and paresthesias of left side of face, left side of abdomen, and right upper and lower extremities. The patient had suffered an episode of trigeminal herpes zoster three weeks prior to admission which had been successfully treated with oral famciclovir. Her neurologic deficit progressed to quadriparesis, urinary retention, and constipation for which an initial assessment of transverse myelitis was made and confirmed with magnetic resonance imaging of the spine. Cerebrospinal fluid was negative for infectious etiologies and demyelinating disease. The patient was treated with IV acyclovir and corticosteroids with partial recuperation of motor and sensory deficit. The neurologic complications observed in this young, immunocompetent patient are unusual and unexpected, mostly because of lack of cervical or thoracic dermatomal involvement which usually precedes the type of lesion observed in her imaging studies. The development of transverse myelitis after trigeminal herpes zoster, up to our knowledge, has not been previously reported.

OP-17 **Patient With Complicated Aortic Deformities in Association With an Atrial Septal Defect and Infective Endocarditis.**

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Coarctation of Aorta (CoA) is strongly associated with bicuspid aortic valve (BAV). This is a risk factor for infective endocarditis. Ancurysm of a sinus of valsalva (SV) is a rare defect with a prevalence of 0.09% and are associated in 10% of cases with BAV and less frequently with COA and ATrial Septal Defect (ASD). The association of CoA with ASD is extremely rare. This is the first case reported in Puerto Rico of an adult patient with CoA in association with a ruptured aneurysm of SV and an ASD.

The patient is a 22 year old male with CoA diagnosed since childhood who was admitted at the Cardiovascular Center due to infective endocarditis secondary to teeth infection (caused by Streptococcus mitis) and signs of congestive heart failure. Upon evaluation with transthoracic and transesophageal echos, he was found to have CoA in aortic isthmus, aortic root dilatation, BAV with vegetation, severe aortic and tricuspid regurgitation, aneurysm of non coronary SV with perforation to right atrium, biatrial enlargement and dilated righ ventricle. Successful antibiotherapy of endocarditis was achieved. Afterwards, surgical replacement of aortic valve and ascending aorta and closure of non coronary SV aneurysm was done. A secundum ASD was found and was also closed. Color photographic registration of the findings confirmed at surgery was obtained. Surgical correction of CoA was postponed for a future time. The patient had a successful postsurgical recovery and was discharged home with anticoagulation treatment.

OP-18 **Pacemaker An Inusual Source Of Bacterial Endocarditis.**

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A 66-year-old man was admitted because myalgias, weakness and tenderness over pacemaker. The pacemaker was placed three years ago at left chest and got infected two weeks after placement with Staphylococcus aureous sensitive to nafcillin which was treated successfully as an inpatient. He started two weeks ago having 38.5 °C of fever and noticed redness over pacemaker area. At physical exam temperature was 38°C, pulse 86/bpm, respiratory rate 20/min and BP 130/70. Positive jugular venous distention , 2/3 systolic murmur at tricuspid valve and edema of lower limbs. CBC showed WBC 26,000, Hgb 13.0 g/dl, Htc 36.8%, platelets 310,000, bands 31 and neutrophils 56%. ECG was normal. Staphylococcus aureus sensitive to nafcillin was detected in blood culture.The incidence of pacemaker infections has not been carefully documented. In 21 studies of pacemaker recipients with variable follow-up, the rate of infections has ranged from 0.8 to 5.7. Failure to remove the infected intravascular electrode is associated with a high rate of relapsed infection and mortality, even if antibiotic therapy directed at the implicated organism is administered for the full duration of medical therapy for endocarditis. The mortality rate is significantly higher for patients treated with antibiotics alone compared to those treated with a combination of removal of the entire pacemaker plus antibiotics. Transesophageal echocardiogram showed vegetation of 1 x 1 cm at pacemaker tip, plus vegetations at tricuspid valve . Diagnosis of acute bacterial endocarditis was done related to pacemaker infection and treated with IV Vancomycin and Gentamycin .Cefepime was added later. Open-heart surgery was performed to remove infected pacemaker, tricuspid valve was repaired but unfortunately the patient died few days later. Pacemakers are a potential source of endocardial contamination and acute bacterial endocarditis that we should always take into account and where structural changes and high mortality is present besides rapid surgical removal of the devic.

Poster Presentations

PP-1 **Familial Idiopathic Thrombocytopenic Purpura (ITP) with Premature Ovarian Failure.**

Ariel Rosado-Rosa, MD Associate; Robert Hunter-Mellado, MD, FACP, Ramon Ruiz Arnau Hospital Universidad Central del Caribe.

The purpose of this report is to add a new family with Familial ITP to the existing six described in the medical literature. The published literature of Familial ITP presents families with one, two and three affected proposita, in clear contrast to our paper which contributes five patients. Of additional interest is the finding of Premature Ovarian Failure (POF) of apparent familial grouping in which the association with adrenal autoimmune disorders or Addison's disease is not present. We describe a family of twelve siblings in which an evaluation of platelet count was done in seven. These seven patients were characterized as all being female with an age range between 36-53 years. Three of these seven patients had a diagnosis of ITP formally made in their fourth decade of life (40-48), which required steroid therapy in all three and splenectomy in two. In the remaining four patients the platelet count was in the lower border of normal and in two an abnormally high mean platelet volume was present. The presence of an abnormal mean platelet volume is associated with a high platelet turnover in which older platelets are removed from the circulation. The father of the siblings had a history of low platelets and died secondary to a bleeding event related to minor trauma. During the review of the past medical history it was found that out of the seven proposita, five of them had developed premature ovarian failure with a clinical menopause between the ages of 32 and 39. There were no other endocrine abnormalities detected in this group of seven patients. An additional female with POF was detected in the siblings not evaluated for ITP. The presence of Familial ITP associated with POF has not been previously reported. We are uncertain of the nature of this association. Nevertheless it is known that both entities are of an autoimmune nature and as such genetic predisposition is a distinct possibility to explain these findings. We plan to expand the evaluation to include the other siblings.

PP-2 **Foregut Carcinoid Tumors.**

Eilyn De Jesus, MD, (Associate), José Ramírez Rivera, MD, FACP, Universidad Central del Caribe, Hospital Universitario Ramón Ruiz Arnau, Bayamón, Puerto Rico.

Carcinoid tumors have been reported in a wide range of organs but most commonly involve the lungs, bronchi, and gastrointestinal tract. The age of distribution of carcinoid tumors ranges form the second to the ninth decade with peak incidence between 50 and 70 years. The majority of patients with carcinoid tumors are asymptomatic. Initial symptoms may be nonspecific,when present correlate with the site of origin and extent of a tumor. 54-year-old woman with hypertension, coronary artery disease, and peptic ulcer disease came to the hospital with periumbilical pain. The pain presented two months prior to admission and became progressively worse up to intensity of 6/10 with radiation to right flank and lower back. At the time of admission it had intensified to a 9/10 and was accompanied by unquantified fever, diaphoresis, and coffee ground vomiting. She was found with a hemoglobin level of 8.3 mg/dl, Hctto of 25.3%, MCV of 75.3, MCH of 24.7, RDW of 15.6 and a platelet count of 425. At this time vitals signs were the following: temperature 37°C, respiratory rate 18, pulse (supine) 80, pulse (standing) 88, blood pressure (supine) 160/90, blood pressure (standing) 140/90. Patient was alert and oriented as to time and place and person with generalized pallor, pale conjunctivae. There was no adenopathy, lungs were clear with grade II/VI holosystolic murmur. The abdomen was soft, epigastric tenderness with radiation to the right side, no rebound or guarding, no edema or deformities were found. A gastric lavage showed traces of blood. Treatment was initiated with Ringer lactate, Protonix IV and transfusion of packed red blood cells. An upper gastrointestinal endoscopy revealed an ulcerated friable gastric mass in the cardiac area . A biopsy of the mass showed,on immunohistochemical analysis, chromogranins positive and synaptophysin positive consistent with neuroendocrine tumor. Patient developed many complications through the admission including right lobe pneumoniae, sepsis,ARDS, hypercalcemia and died

26th days after admission. Local excision is the usual treatment for carcinoid tumors that are smaller than one centimeter. Surgery for larger tumors taking more surrounding normal appearing tissue.

PP-3 **Lung Cavitating Process In An Inmuno-competent Elderly Patient.**

Gregorio A. Cortes-Maisonet, MD (Associate), Arnulfo Santana, MD, FACP; Jose H. Martinez, MD, FACP. San Juan Bautista Medical Center.

Among the different differential diagnosis of lung cavitating diseases, there are a few which are not well known or understood because of their rarity or relatively new appearance. Of those, only anecdotal reports exist among the medical literature.

Today, we bring to discussion a rare and almost unique case of a 71 years old retired salesman with past medical history of hypertension and a chronic smoker for 48 years. The patient is admitted with productive cough, brown sputum, fever, weight loss and fibrocavitating lesions on CXR of approximately 4 – 6 weeks of evolution. The patient has no risk factor for fungi, PPD negative, and no previous cancer history. At physical examination and laboratory reports the patient presented bilateral apical crackles, mild microscopic hematuria, mild hyponatremia, leukocytosis, and negative acid fast. CT Scan showed bilateral apical fibrocavitating lesions. After several negative bronchoscopic lavages, an open lung biopsy was performed and a pathologic report of Cavitating Bronchiolitis Obliterans Organizing Pneumonia was received. Treatment with steroids induced a rapid response with a complete remission of all the symptoms and a significant improvement of radiologic findings.

This case illustrates several findings. First, the need of ruling out cavitating BOOP in patients with a cavitary lung disease. Second, the need for further study of an idiopathic disease on which medical literature has been so sparse on reporting about. Finally, follow up of these patients should be closely monitored for any further relapses and their long term consequences as well as for new preventive, diagnostic and therapeutic advances.

PP-4 **Shortness of Breath in an Octagenarian: The Unexpected Diagnosis of an Atrial Myxoma.**

Felix Lugo-Adams, MD; Sonia Vicenty, MD; Jose Escabi, MD, FACC; Luis Rodriguez-Ospina, MD, FACC, FACP

Cardiac myxomas are the most frequent benign neoplasm of the heart in adults. The mean age of patient with sporadic myxomas is 56 years. The clinical presentation varies and constitutional non-specific symptoms are the only complaints in one third of cases. The diagnosis of an atrial myxoma in an octagenarian is extremely rare and its prompt management is important to relieve associated symptoms and avoid embolic complications.

We describe the case of an 85-year-old male patient who was incidentally found with a left atrial myxoma and was successfully taken to the operating room for its removal. He is the oldest reported patient with a myxoma to be diagnosed and operated in Puerto Rico.

PP-5 **Hermansky-pudlak Syndrome Presenting With Rectal Bleeding.**

Annabelle Lipsett M.D.(Associate); Teresa Montesinos, MD; Milton D. Carrero-Quiñonez, MD, FACP; Jose Ramirez-Rivera, MD, FACP – Dr. Ramon E. Betances University Hospital, Mayaguez, Puerto Rico.

A 25 year-old albino woman from Aguada was admitted with rectal bleeding. Three days earlier she had notices blood-tinged loose stools which progressed to bright red bleeding. She was taking Protonix 40 mg daily and Asacol 800 mg every 8 hours. She also referred mild colicky abdominal pain. Patient denied using aspirin or NSAIDS.

On admission temperature was 36.7 0C, pulse was 100/min., RR 22/min., and BP 109/81. The patient had a fair skin and looked extremely pale. She was blonde with light brown eyes and had horizontal mystagmus. Her abdomen was soft and the epigastrium was slightly tender. The white celll count was 12.5 x 10 3/ul with 83% neutrophils. The platelet count was 275 x 10 3/ul. The hemoglobin was 14.og/dL.

Prothrombin and partial thromboplastin times were normal. Bleeding time was 5 minutes and 30 seconds (normal:1 to 3 secs). Patient underwent Esophagogastroduodenoscopy and colonoscopy. Final diagnosis was: hiatal hernia, gastritis, non-specific colitis and external hemorrhoids; no active bleeding was found. Patient was treated with bowel rest, loperamide, pantoprazole and mesalamine. She was discharged three days later without further bleeding. Her lowest hemoglobin level was 12.9 g/dL.

Hermansky-Pudlak syndrome (HPS) describves a rare group of autosomal recessive diseases whose manifestations include: oculocutaneous albinism, bleeding & lysosomal cervid deposition. Bleeding is related to platelet storage tool defects, platelets lack dense bodies, thus rendering them dysfunctional. The HPS has been related to defects in 4 genes. The HPS I genes is located hand 10q 23. The carrier frequency in Puerto Rico is estimated at 1:21. HPS represents the most frequent type of albinism in the Puerto Rican population with a frequency of 1:1800 in Isabela, Puerto Rico. Our patient is from Aguada (about 30 miles southwest of Isabela). She

is the daughter of relationship between a niece and her uncle; most probably, both of them carriers.

Late complications of HPS include interstitial pulmonary fibrosis, inflammatory bowel disease, renal failure and cardiomyopathy due to cervid-like material deposition. Pulmonary fibrosis is the most common cause of death, but hemorrhage and granulomatous colitis can be fatal. This platelet defect conjoined by granulomatous colitis may leads to exsanguinating rectal bleeding.

PP-6 **An Unusual Case of Chest Pain – a Case Presentation.**

Jorge V. Alvarado, MD; Angie Rosado, MD (Associate); Roberto Leon, MD, 9Associate); Augusto Sepulveda, MD (Associate) – Department of Internal Medicine, Damas Hospital, Ponce School of Medicine, Ponce, Puerto Rico.

Introduction: Cardiac disease is common among patients with SLE (Systemic Lupus Erythematosus) as pericardial, myocardial, valvular, and coronary artery disease. The three major coronary abnormalities associated with myocardial injury in SLE include premature atherosclerosis, coronary arteritis and, less frequently, coronary aneurysms.

Case Description: A 26 year-old black male patient with a 5-year histoy of Anti-Phospholipid Syndrome sustained a lateral wall myocardial infarct and demonstrated angiographic evidence of multiple, diffuse, saccular coronary aneurysms without evidence of atherosclerotic occlusive disease in all three coronary arteries. Serologic studies where consistent with active SLE. Lupus-associated nephritis was also present. Radiographic studies showed no evidence of brain or thoracic aneurysms. 2D echocardiogram showed an estimated ejection fraction of 35% and moderate pericardial effusion. High dose IV steroid treatment was started along with systemic anticoagulation. Patient showed a benign course and was discharged by Day 14th on high dose oral steroids daily (60 mg), statins, full oral anticoagulation (warfarin 5mg), anti-hypertensives and aspirin. Patient was lost during follow up and developed steroid-induced hyperglycemia, 20 pound weight gain and warfarin intoxication. Three-months follow up coronary angiography showed complete resolution of aneurysms and serologic studies showed no active autoimmune disorder. Coronary artery aneurysms have been reported in 15 prior cases of patients with SLE.

Variation in format: An intensive Medline search of the literature revealed no previous reports of diffuse saccular coronary aneurysms involving all three coronary arteries associated with SLE presentation. This case highlights the unusual presentation of acute SLE in young patients complicated by multiple aneurysms and acute myocardial infarction.

Conclusion: In this case, we believe that a direct causal association exists between acute SLE-associated aneurysms and myocardial ischemic injury. Early recognition and prompt treatment may decrease mortality associated with this condition

PP-7 **Perirenal Abscess Secondary to Acute Pancreatitis.**

Guibel Perera, MD; Jose Nieves, MD; Sylvette Naxario, MD, San Juan V. A. Medical Center.

A 64 y/o male smoker with ethanolism, PUD sp vagotomy and 2 previous episode of pancreatitis was admitted to the hospital with abdominal pain, nausea and anorexia. He presented elevated amylase and lipase consistent with acute pancreatitis. Abdominal CT scan showed cholelithiasis and a stone at the pancreatic duct with dilation of the duct and extensive peripancreatic and perinephric inflammatory changes. He underwent an ERCP with papillotomy and stone extraction with gradual resolution of abdominal pain and improvement in amylase and lipase. Two days later he developed fever and repeated CT scan showed improvement in pancreatic inflammation but increased perinephric collection. The perinephric collection was aspirated and drainage left. Drained fluid had elevated amylase consistent with perinephric collection secondary to pancreatitis. Cultures were negative. Fever and leukocytes resolved. Drainage was discontinued and he underwent cholecystectomy with resolution of all symptoms. Conclusion: Perinephric abscess is an uncommon complication but should be kept in mind when persistent fever or leukocytes are present in subjects with pancreatitis.

PP-8 **Acquired Hemophilia Presenting as Intractable Bleeding in an Elderly Patient**.

Luis Ortiz, MD (associate), Nelson Matos, MD, Armando Garcia, MD, William Caceres, MD, FACP; Veterans Affairs Hospital, Internal Medicine Program ,San Juan ,Puerto Rico.

Case of a 72 y/o male puertorrican male, with Alzheimer's disease, seizure disorder and hypertension. Outpatient medication included Dilantin. Patient was brought to ER by family members because he developed hematuria of three days duration, associated with multiple body hematomas. Patient was found lethargic, hypotensive, and tachycardic with gross hematuria. Multiple hematomas were visible on right shoulder, upper back, left wrist and both feet. Family members denied any trauma or recent seizure episodes. Laboratory results showed PTT (78.7

sec), PT (18.8 sec), hemoglobin (5.7 g/dl) and hematocrit (17.2 %). Patient was admitted to Hematology Oncology ward were FFP was given with minimal improvement of bleeding without correction of PTT. Several blood transfusions were required for optimization of hemoglobin levels. Mixed 1:1 test done with no correction of PTT suggesting a factor inhibitor. During intrahospital stay at Hematology Oncology ward the patient received treatment with solumedrol, and human factor VIII (Humate-P) for the most common acquired factor inhibitor (antibodies against factor VIII) . Activity of factor VIII was found on 2% and levels of FVIII inhibitor on plasma were 287.3 H BU initially. One and a half month later the hemoglobin increased to 12.5 g/dl with a hematocrit of 37.2%, PTT 88.2 sec, activity of FVIII 0%, and FVIII inhibitor of 525.9 H BU. Hematomas resolved but patient still presenting with sporadic episodes of hematuria. Current treatment continues with FEIBA and Konyne as needed to control bleeding. The patient was started on weekly Rituximab.

Discussion: Acquired Hemophilia and its therapeutic options will be discussed.

PP-9 **Role Of Hyperinsulinemic-euglycemic Therapy In Calcium Channel Blocker Intoxication.**

Luis Ortiz, MD,Manuel Figueroa, MD, Ricardo Fernandez, MD, Miguel Boque, MD, George Fahet, MD, Michelle Lopez, MD, Alfonso Torres, MD; VA Hospital, San Juan, Puerto Rico.

A 77 y/o male with hypertension , hyperlipidemia, hypothyroidism, and a cerebrovascular accident in the past. Patient was found by family members after taking an overdose of atenol and nifedipine. At emergency room the patient was found aphasic with generalized weakness, not following verbal commands. No focal neurologic deficits were identified. Upon physical examination the patient was found to be hypotensive (Bp 57/41mm/Hg) and EKG showed junctional bradycardia(HR 35-40bpm) . Aggressive isotonic fluid therapy was started with two large peripheral IV bores. Gastric lavage and administration of activated charcoal was done . Calcium gluconate 2gm IV x 3 doses were given with only temporary improvement of blood pressure. Glucagon 6 mg IV was added to therapy after which pressure increase to 100 / 60mm/Hg and heart rate to 70bpm temporarily. Further efforts to improve hemodynamic compromised were made by starting insulin drip at .5u/kg/hr with concomitant use of Dextrose 10% solution . Remarkable neurological improvement was seen within minutes after insulin drip placement. Chest film showed pulmonary congestive changes and marked pulmonary edema . Patient was admitted to the Medical Intensive Care Unit were PA flotation catheter was placed. Further calcium gluconate and chloride infusion raised blood pressure only minimally for which dopamine was started titrated up to 14mcg/kg/min.Several episodes of hypoglycemia were documented. Dopamine and insulin drip were weaned off during the next few hours as resolution of sinus rhythm electrocardiographically was noted. The patient was transfer to the telemetry ward for a total length of stay at MICU of less than 18hrs. Further more, he was transferred to the psychiatry service were no medical complications were seen.

Disscusion: Hyperinsulinemic-Euglycemic therapy is of benefit in patients with hemodynamic compromised secondary to intoxication with calcium channel blocker not responding to conventional therapy .

PP-10 **Association of Parvovirus B19 Infection With Acute Leucocytoclastic Vasculitits in a Healthy Adult: Case Report and Review of the Literature.**

María del C. Rodriguez-Julbe, MD (Associate), José Fournier-Rebollo, MD, Aurea Rivera-Delgado, MD, José Gutierrez-Nunez, MD (Fellow), Glenda Gonzalez-Claudio, MD (Associate), VA Medical Center, San Juan, Puerto Rico.

Introduction: Parvovirus B19 was discovered in 1974 and is the only member of the family Parvoviridae known to be pathogenic in humans. Infection with this agent may cause a widespread benign and self-limiting disease in children and adults, known as erythema infectiousum or fifth disease. A variety of further manifestations are associated with the infection such as arthralgias, arthritis, leucopenia and thrombocytopenia, anemia and vasculitis, spontaneous abortion and hydrops fetalis. Both acute and persistent courses of B19 infections have been reported. We describe a patient who had pathological evidence of a leucocytoclastic vasculitis associated with parvovirus B19 infection documented by positive serology. All the known data and potential mechanisms involved in the pathogenesis will be discussed.

Abstract: 32 y/o male without systemic illness, no allergies, no medication use, no surgeries, social ethanol use, no other toxic habits who works as a physician was well until the development of headache not relieved with acetaminophen and 48 hours afterwards developed general malaise, bilateral knee pain with swelling and erythematous, non-pruritic skin macules on lower extremities. Skin biopsy confirmed the diagnosis of leucocytoclastic vasculitis. Serum complements were low, hepatitis B serology negative, normal sed rate and skin biopsy confirmed the diagnosis of leucocytoclastic vasculitis. Patient was treated with high dose steroid course with tapering down. Complete clinical recovery

was obtained in 3 days. Parvovirus B19 serology was positive and persisted positive after eight weeks of the clinical course.

Conclusion: Parvovirus B19 should be considered in the differential diagnosis of leucocytoclastic vasculitis.

Myopathy and Polyneuropathy of Critical Illness.

PP-11 Rafael Chiong, MD (Associate); Lizanabell Torres, MD; Frances Cruz, MD; Milton D. Carrero-Quiñonez, MD, FACP – Dr. Ramon E. Betances University Hospital, Mayagüez, Puerto Rico.

A 56 year old healthy man developed proximal weakness of four extremities during an admission in intensive care unit due to severe sepsis and acute renal failure. He suffered a partial dehiscence of the anastomosis producing intraabdominal infection three days later after a resection of a rectosigmoid adenocarcinoma. A left hemicolectomy and colostomy were performed. He was transferred to ICU on mechanical ventilation, diaphoretic with 39c of temperature. He had prodced less than 50 mililiters of urine in the last 24 hours. His blood pressure was stable around 145/71 mmHg.

At neurological examination: aware, alert, slightly obtunded, strength of right proximal muscles of upper and lower extremities was 0/5, on the left side 2/5. Strength of distal muscles in upper extremities 1/5 and in lower extremities 2/5. Reflexes 1+ in the four extremities. Sensation was unable to assessed. The white blood cells count was 24.9 x 10 3/ml, the hemoglobin was 7.6 g/dL, hematocrit 23.2 vol%, platelets 322 x 10 3/ml, bands 12, BUN 101 mg/dL, creatinine was 9,9 mg/dL, albumin 1.2 g/dL, potassium 5.9 meq/L, glucose 305 mg/dL, osmolality 349.3. The arterial blood sample (FiO2 35%) showed pH 7.30, pCO2 50.5, pO2 89. FENa 3%. The first blood cultures were negatives. Chest X rays showed small right pleural effusion.

Electrocardiogram with sinus tachycardia. Electromyogram and nerve conduction velocity studies concluded: 1) myopathy involving both distal and proximal muscles. 2) mild peripheral polyneuropathy. Intensive care support started immediately: intravenous fluids, daily hemodialysis, insulin pump, antibiotics (Garamycin, Vancomycin, Primaxin, Flagyl). Protonix, total parenteral nutrition therapy, blood transfusion, central venous pressure were measured. Final diagnosis: Severe sepsis; Acute renal failure; Myopathy and Polyneuropathy of critical illness.

Critical illness myopathy is an uncommon clinical picture which appears in ICU setting due to usual neuromuscular blocking agents, steroids, aminoglycosides, uremic status and sepsis. Critical illness polyneuropathy is also seen in ICU settings in patients with systemic inflammatory response syndrome. It is secondary to altered microcirculation, proinflammatory cytokines, axonal degeneration, damage from parenteral lipids, impaired transport of axonal protein and endoneural edema, and /or hypoxia.