ST. VINCENT CHARITY MEDICAL CENTER

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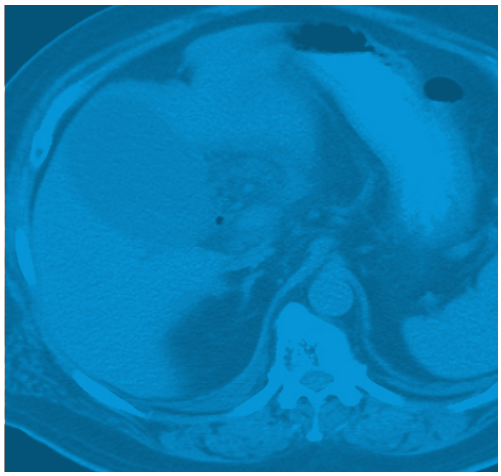

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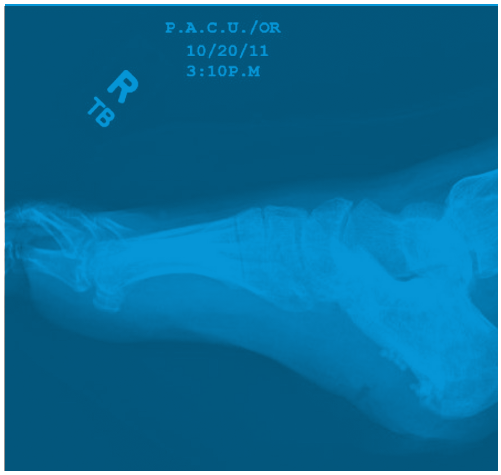

ST. VINCENT CHARITY MEDICAL CENTER JOURNAL
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ST. VINCENT CHARITY
MEDICAL CENTER

The St. Vincent Charity Medical Center family is committed to the healing mission of Jesus. We serve with: a deep respect for the dignity and value of all persons; our practice of quality care; our dedication to the poor; and, our commitment to education. St. Vincent Charity Medical Center, with the commitment of its Caregivers and physicians, will be a leading model for healthcare delivery in Northeast Ohio based on its faith-based mission, dedication to education, commitment to the communities it serves, excellence in the patient experience it provides, focus on surgical services, and partnerships with physicians and other constituencies.

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A MESSAGE FROM THE EDITOR

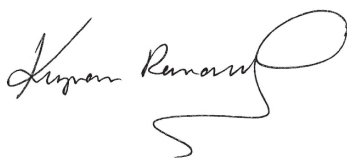
What are the characteristics of future doctors of America? This is a question I have been asking myself for a long time. Medicine is constantly evolving. I have witnessed changes—some I liked; others not as much. The primary concern remains whether or not we are moving in the right direction. I am not really sure.

When I think of the skills and qualities required of my ideal physician and compare it with today's general requirements for a physician, I see a mismatch. I do not blame our young doctors or students for this mismatch. We have problems in our medical schools, residency programs, financing of our health system, our legal system and many more.

Let me give an example. Who do you like more, Marcus Welby, MD, or Dr. House? I know these are fictional representations, but like most good fiction, there is a bit of truth in them. Marcus Welby, whom I prefer, was known for his caring and involvement in his patient's life. He had time for them. Maybe he wasn't a genius, but his main character was a loving, people person who took good care of his patients.

In contrast, Dr. House is a genius. He makes all the diagnoses that no one else can make but he is not my kind a doctor. Yet despite his poor communication skills, everybody loves him. I am not talking about his patients in the show, I am talking about us. My residents and students love him! And I fear that he has become an unhealthy role model for our future physicians.

In this latest issue of the *St. Vincent Charity Medical Center Journal*, we continue to balance the genius of clinical research with a focus on the human side of medicine.



Keyvan Ravakhah, MD, MBA, FACP
Editor in Chief

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Painless Gangrenous Cholecystitis

Timely Diagnosis and Treatment

By **Suresh Ponnada, MD**; **Amara Nidimusili, MD**; **Debbie Lim, MD**; **Khaldoon Shaheen, MD**; **Indukumar Sonpal, MD**; and **Donald Eghobamien, MD**

LEARNING OBJECTIVES

1. The importance of recognizing the unusual presentation of gangrenous cholecystitis
2. Predictors of Gangrenous Cholecystitis

INTRODUCTION

Gangrenous Cholecystitis (GC) is one of the most common complications of acute cholecystitis particularly in diabetics, older patients and those who delay definitive treatment. It results from sustained obstruction of the cystic duct, leading to vascular compromise and full-thickness gangrene of the gallbladder wall [1]. Patients with gangrenous cholecystitis are at increased risk of mortality and gallbladder-related complications, including perforation and abscess as well as an increased risk of conversion to open cholecystectomy. We report an atypical and unique presentation of a patient with painless gangrenous cholecystitis.

CASE PRESENTATION

History: A 66-year-old obese man with H/O DM and HTN was admitted to our hospital for atypical left-sided chest pain of several hours duration worsened by movement and relieved with nitroglycerin. Acute Coronary Syndrome was ruled out with a negative cardiac workup and the chest pain subsequently resolved. On presentation, he denied any abdominal pain, nausea, vomiting, fever /chills. Review of systems was negative.

Physical examination was notable for heart rate of 110, with no fever and unremarkable abdominal exam findings. The rest of systemic examination was within normal limits.

Assessment and hospital course: Initial work-up showed WBC of 17.4 with 13% bands; AST 6 u/L; ALT 23 u/L; Alkaline Phosphatase 80 u/L; Serum sodium 138; Leukocytosis persisted, with no possible source of infection identified. On Day 3, blood cultures grew MRSE and Enterobacter Sakazaki, and patient was started on IV Vancomycin and Meropenem. During this entire course, the patient was completely pain-free. An ultrasound showed normal-sized gallbladder and multiple gallstones with no wall thickening or pericholecystic fluid. HIDA scan was inconclusive; Indium WBC whole body scan showed questionable hyperemia/ inflammatory changes in the pericholecystic region. Finally, CT scan of the abdomen showed a distended gall bladder with posterior layering of calculi/sludge, suspicious for cholecystitis. Infiltration in the adjacent fat is seen. Air bubbles project over the region of the gallbladder neck suggestive of an infection with gas producing organism. He had a laparoscopic surgery, where the gallbladder was found to be gangrenous and the procedure was advanced into an open cholecystectomy. Post-surgical hospital course was uneventful and leukocytosis resolved.

DISCUSSION

GC is a fatal complication of cholecystitis with an incidence of 2%-29.6% [2]. The current case is unique, since our patient denied any prior episodes of abdominal pain and the only abnormality was leukocytosis. A high index of suspicion is essential for the early diagnosis and treatment of GC. Previous studies have shown that advanced age, male gender, leukocytosis and co-morbidities especially diabetes have been associated with the risk of developing gangrenous cholecystitis. Current studies have confirmed that if a patient has the following admission criteria (male gender, white blood cell count greater than 14,000/mm3, heart rate greater than 90/min and sodium 135 mg/dL or less) together with right upper quadrant pain, the likelihood of gangrenous cholecystitis is high [1]. GC has a mortality rate of up to 22% and a complication rate of 16-25% [2]. Complications associated with GC include perforation, which has been reported to occur in as many as 10% of cases of acute cholecystitis. Overall, high risk of suspicion and early surgical intervention for acute cholecystitis will prevent the development of gangrenous cholecystitis.

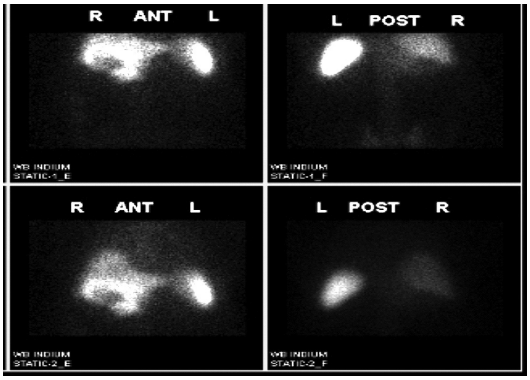
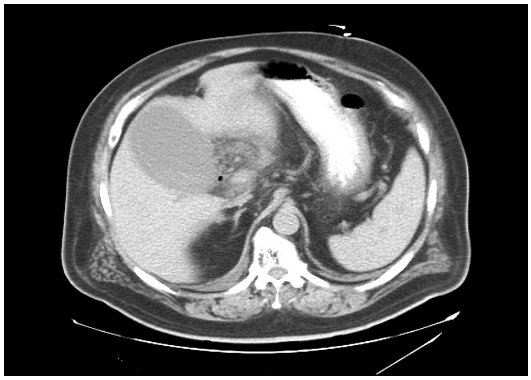


Fig 1. Indium WBC whole body scan shows and distended markedly gallbladder. Infiltration in the adjacent fat is seen, neck suggestive of an infection with gas producing organism.



CT scan abdomen with oral contrast shows air bubbles project over the region of the gallbladder area of hyperemia/ inflammatory changes in the Pericholecystic region.

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Not Just Another Case of Back Pain

Inferior vena cava filter penetration

By **Artan Kaso, MD**; **Buthayna Dinary, MD**; **Khaldoon Shaheen, MD**; **Ginius Pradhan, MD**; and **Keyvan Ravakhah, MD, MBA, FACP**

LEARNING OBJECTIVES

1. Identify indications for IVC filters
2. Recognize the late complications of IVC filters

CASE PRESENTATION

A 32-year-old woman admitted with one-day history of fever, chills and confusion. Her past medical history is remarkable for hypertension, diabetes mellitus, and end-stage renal disease on hemodialysis. Five years ago, she had pulmonary embolism, initially was treated with Coumadin, which was stopped due to subdural hematoma and an IVC filter was placed at that time. On presentation, temperature was 38.8°C, heart rate 120/min, respiratory rate 22/min and blood pressure 80/40 mmHg. Cardiopulmonary and other systemic examination was unremarkable. She was admitted to the ICU and started on vancomycin and piperacillin/tazobactam intravenously. Blood culture yielded methicillin-resistant S. aureus (MRSA). TEE was done with no evidence of infective endocarditis. A few days later, fever and confusion resolved. However, she started to have lower back pain. CT scan of lumbar spine was performed and showed a single strut of the IVC filter perforating inferior vena cava and anchor in L2 vertebral body with surrounding lytic changes *(Figure 1)*. As a result of these findings the patient is considered for IVC filter removal by vascular and neurosurgery teams.

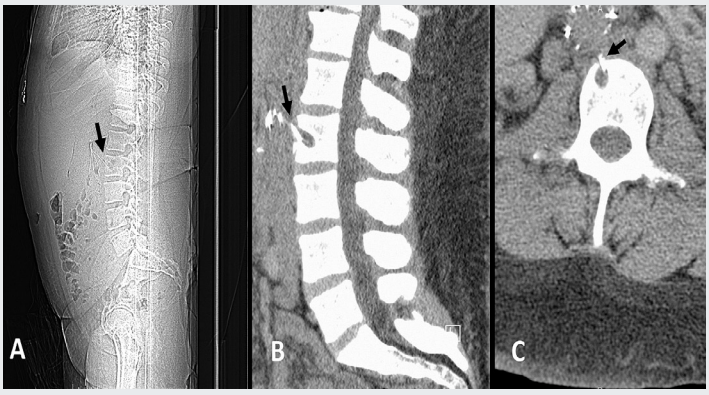

Figure 1: Computed tomography scan of lumbosacral spine, general scanogram (panel A), sagital view (panel B) and axial view (panel C): A single strut of the IVC filter (arrows) perforating the IVC and anchor in L2 vertebral body with surrounding lytic changes.

DISCUSSION

The use of inferior vena cava (IVC) filters has increased dramatically in the last two decades. Multiple factors have contributed to this phenomenon, including expanding indications for filter placement and continued development of new devices. Increased utilization has also coincided with the introduction of retrievable filters.

Retrievable filters are particularly attractive in patients who are considered to have a high risk for venous thromboembolic (VTE) events and a temporary contraindication to pharmacologic prophylaxis. Under these circumstances, patients can be bridged with a filter until their risk for VTE subsides and they no longer require protection, or until they no longer have absolute or relative contraindications to anticoagulation. Filters placed under these circumstances are considered prophylactic. Filters are considered therapeutic when placed in patients with documented VTE who have either a contraindication to or failure of anticoagulation.

When retrieval is attempted, it is usually successful, but the majority of retrievable filters are left in place indefinitely. There are multiple reasons why this occurs, including ongoing indications for the filter, large trapped embolus or thrombosis, inability to retrieve the filter due to filter tilt or ingrowth, and loss of patient follow-up.

It makes intuitive sense that a device placed in the vena cava for an extended period of time has a risk of complication. Even though IVC filters have been shown to be safe and effective, there have been a substantial number of filter-related complications reported in the medical literature.

Late complications of IVC filters include filter migration, tilting, penetration, strut fracture and IVC filter thrombosis [1]. Filter penetration is defined as an extension of the filter components of greater than 3 mm outside the caval wall [2]. Studies have | continued on p.8

Euglycemic Diabetic Ketoacidosis with Acute Pancreatitis in a Patient Not Known to Have Diabetes

By **Janna Prater, MD** and **Joumana Chaiban, MD**

LEARNING OBJECTIVES

1. The complex interplay between severe alcohol-related pancreatic injury, ketoacidosis and starvation physiology
2. In patient with severe pancreatitis; euglycemic diabetic ketoacidosis should be in the differential even in the absence of hyperglycemia, acid/base status and ketonemia

INTRODUCTION

Diabetic Ketoacidosis (DKA) is one of the two most serious acute metabolic complications of diabetes mellitus (DM), the other entity being hyperosmolar hyperglycemic state. DKA is usually easily recognizable, moreover if the patient has an underlying history of DM. It is characterized by a triad of uncontrolled hyperglycemia, metabolic acidosis and increased total body ketones [1]. Euglycemic DKA, a relatively uncommon presentation of DKA, is a lesser-known entity and can go unrecognized at initial presentation. True euglycemic DKA (blood glucose <200 mg/dl [11.1 mmol/l]) 2 in DM can be caused by starvation of any cause in conjunction with a concurrent illness. It has been described mainly in type 1 DM [3, 4] but has also been reported in type 2 DM and in gestational DM [3, 5]. We report herein a case of euglycemic DKA precipitated by starvation and severe pancreatitis in a patient with history of chronic alcoholism and no known underlying DM.

CASE PRESENTATION

Ms D.D. is a 36-year-old hairdresser who presented to the emergency room with severe epigastric pain (10/10) of three days duration, radiating to her back, aggravated by lying back, with no alleviating factors, and associated with nausea and vomiting for the past week. She denied fever but had chills on admission. The patient had not been eating for more than a week due to the above symptoms. She denied having similar episodes in the past. She admitted to drinking 1 liter of brandy daily for years, her last drink was three days prior to admission. She denied taking any medications. Patient also underwent rehabilitation in 2007 and 2008 for alcoholism and relapsed soon after. Her family history was significant for coronary artery disease, diabetes, hypertension and her mother had been struggling with alcoholism. Apart from alcoholism, her social history was significant for smoking (three Black & Mild cigars a day for years) and remote history of cocaine use.

On physical examination, her vitals revealed a temperature of 36.6°C, blood pressure of 123/71 mm Hg, respiratory rate of 24 per minute, heart rate of 80 per minute and oxygen saturation of 100% on room air. Her height was 5 feet, weight 50 kg with a BMI of 20 kg/m2. She was in mild distress due to abdominal pain, lethargic, easily arousable and followed commands. No fruity odor was reported. Abdominal examination was remarkable for hypoactive bowel sounds, moderate tenderness in the epigastric area with no rebound tenderness or palpable mass. Remainder of the physical examination was within normal limits. Laboratory investigation was pertinent for anemia with hemoglobin of 10.2 g/dL, hematocrit of 32.8% and normal MCV at 90.9 fL, normal WBC at 7.600 cells/m3 with normal differential and platelet count of 218,000 cells/mm3. Her chemistry profile revealed low bicarbonate at 10 mmol/L, BUN of 7 mg/dL, creatinine of 0.7 mg/dL, normal glucose level at 86 mg/dL. Her liver enzymes were elevated with a total bilirubin of 2.0 mg/dL, AST (SGOT) of 320 u/L, and ALT (SGPT) of 88 U/L. Amylase was elevated at 208 U/L, and lipase at 3378 u/l initially and exceeded more than 10,000 u/l on the next day. Arterial blood gases showed severe metabolic acidosis with a pH 7.21 and pCO2 of 19. Anion gap was 27 mmol/L. Urinalysis was positive for ketones at 80 mg/dL, serum acetone was positive (qualitative test showed large acetone, no quantitative test was performed) and lactic acid level was 0.9 mmol/l (normal range 0.4 – 2.0 mmol/l). Urine toxicology was positive for opioids. CT scan of the abdomen showed severe pancreatitis with peripancreatic phlegmon, edema and severe infiltration of regional fat *(Figure 1)*.

Patient was diagnosed with severe pancreatitis and she was admitted to the intensive care unit. Increased anion gap metabolic acidosis was thought to be secondary to ketoacidosis due to alcoholism/starvation. Patient was kept NPO, intravenous fluid resuscitation was initiated (normal saline followed by 5% dextrose in normal saline), and abdominal pain was managed with morphine. Elevated liver function | continued on p.8

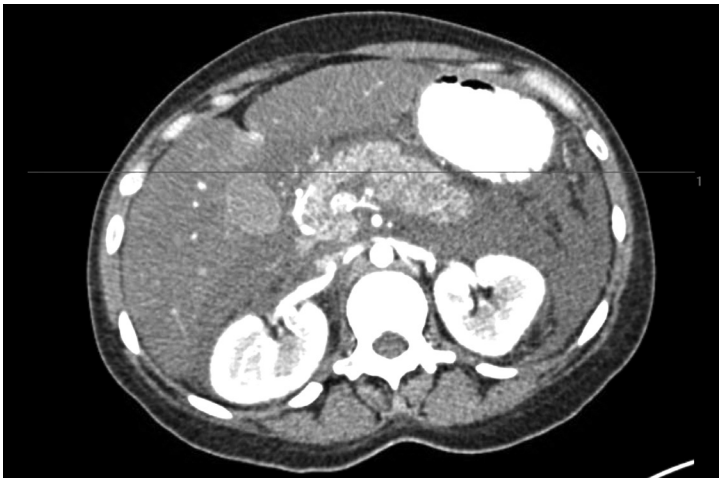

Figure 1: CT scan of the abdomen with IV and PO contrast showing severe pancreatitis with peripancreatic phlegmon, edema and infiltrative changes throughout the peripancreatic fat planes. Pancreatic necrosis and pseudopancreatic cyst is not seen.

Calcaneal Osteomyelitis Following Local Anesthetic Injection

By **Michael Canales, DPM, FAC FAS**; **Michael Bowen, DPM**; and **John Gerhard, DPM**

LEARNING OBJECTIVES

1. Heightening awareness of potentially limb-threatening complications following plantar fascial injection
2. Correct diagnosis and aggressive management of calcaneal osteomyelitis

INTRODUCTION

Injections of local anesthetic in combination with corticosteroids are an accepted choice in the treatment of recalcitrant heel pain diagnosed as plantar fasciitis [1]. When performed correctly, infections following injections are exceedingly rare [2]. There are two published reports documenting osteomyelitis following corticosteroid and local anesthetic injections [3, 4]. We present a case of calcaneal osteomyelitis following injection in a patient with a calcaneal stress fracture misdiagnosed as plantar fasciitis. Subtotal and partial calcanectomy have been posited as effective treatments for calcaneal osteomyelitis [5, 6]. We discuss the treatment options available when circumstances make this an unacceptable option to the surgeon or the patient.

CASE PRESENTATION

A 24-year-old female pharmacy technician reported to her primary care physician (PCP) with a complaint of unilateral heel pain. The patient reported a recent increase in activity following the birth of her first child. She states that her heel pain began after initiating running on a treadmill. Her PCP diagnosed the pain as plantar fasciitis and offered a treatment plan consisting of a series of three injections of local anesthetic and corticosteroid. When this treatment did not relieve her symptoms, the patient was referred to a podiatrist. The podiatrist added physiotherapy and cryotherapy to the treatment plan. These additions did not alleviate her pain and an MRI was obtained. The MRI revealed a linear decrease in T1 signal intensity suggesting that the patient had incurred a stress fracture of the calcaneus *(Figure 1)*. The patient was referred to our institution for further treatment.

Examination revealed an edematous and erythematous right foot and leg. No open lesions were appreciated. A two-layer Jones compression bandage with a fiberglass shell was applied to the patient's right leg. The patient was instructed to be non-weight bearing to the right foot with crutches.

Five days later, the patient contacted the office complaining of numbness to her digits, pain to the ankle and reported that blood was seeping through the cast. The patient was directed to report to the Emergency Department immediately.

Upon removal of the cast, hemorrhagic bullae were found encompassing the entirety of the heel *(Figure 2)*. Purulent drainage was expressed with compression of the lesions. No crepitance was noted within the soft tissue. The patient complained of chills and was tachycardic but was afebrile and normotensive. The patient was admitted with empiric IV antibiotic therapy initiated. The first of four soft tissue excisional debridements was performed as well as a biopsy of the calcaneus. After the first procedure, an additional MRI was obtained which revealed a mottled T1 signal intensity suggestive of osteomyelitis of the calcaneus *(Figure 3)*. Bone cultures revealed the calcaneus was infected by methicillin-resistant Staphylococcus aureus. | continued on p.10

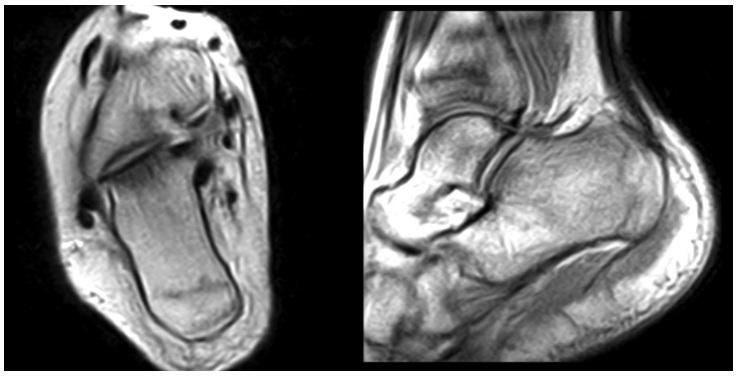

Figure 1

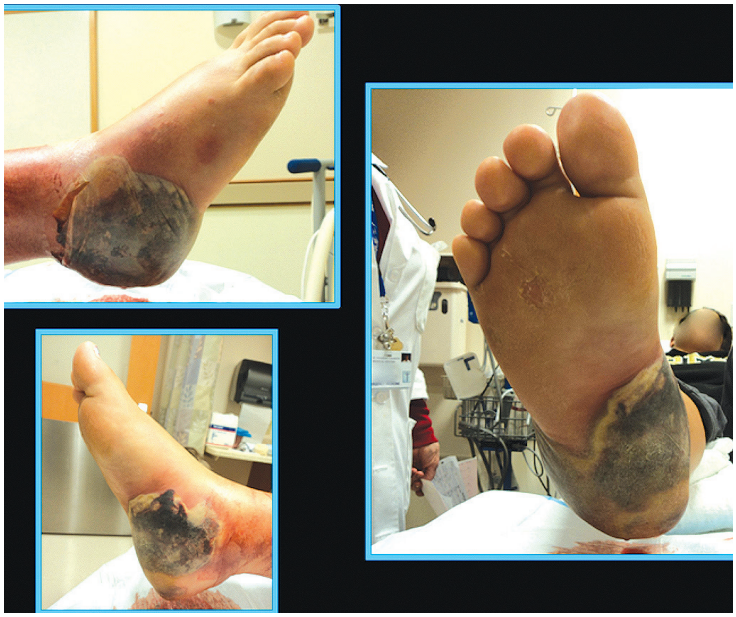

Figure 2

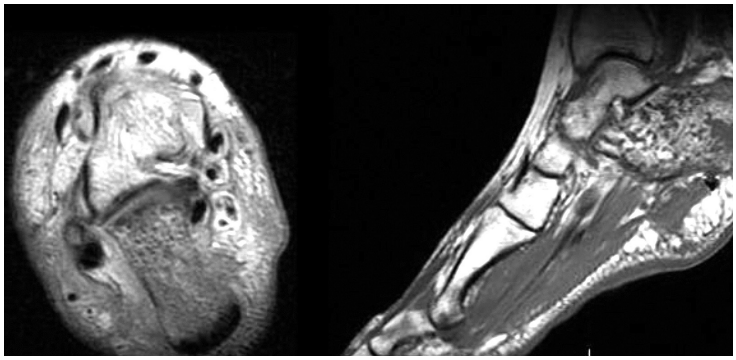

Figure 3

When Chest Pain is More Than a Heart Attack

By **Puja Karanth, MD**; **Khaldoon Shaheen, MD**; **Ismail Hader, MD**; and **Mona Reed, MD**

LEARNING OBJECTIVES

1. To identify non ischemic etiologies for cardiac chest pain
2. Coronary artery aneurysm—diagnosis and management

INTRODUCTION

Aneurysms of the left main coronary artery (LMCA) are exceedingly rare clinical entities, encountered incidentally in approximately 0.1% of patients who undergo routine angiography. The majority are atherosclerotic in origin. Other causes include trauma, connective tissue disorders, Kawasaki disease, vasculitis, congenital, mycotic, and idiopathic. The primary complication is myocardial ischemia or infarction, with rupture being rare. We describe here a 55-year-old female who presented with unstable angina and coronary angiogram revealed a saccular aneurysm of the LMCA.

CASE PRESENTATION

A 55-year-old female presented with acute onset of chest pain at rest that radiated to her jaw and left arm associated with a feeling of general malaise. She denied any history of dyspnea, orthopnea, dizziness or sweating. Her past medical history was notable for hypertension, hyperlipidemia, obstructive sleep apnea, and obesity status post gastric bypass surgery 10 years ago. She had a history of anemia and gastrointestinal bleeding of undetermined etiology a year ago. Medications included simvastatin, metoprolol, furosemide and folic acid. Family history is noncontributory and negative for coronary artery disease. Social history was significant for occasional alcohol use but no smoking or illicit drug abuse. On examination, she was obese and her temperature was 37.0°C, blood pressure was 145/80 mmHg, heart rate was 90 beats/minute, respiratory rate 20 breaths/minute, oxygen saturation 96% on room air and BMI of 55.

She appeared in moderate distress secondary to chest pain. The rest of her systemic examination was unremarkable, and the electrocardiogram showed T-wave inversion in lead II, without ST-segment changes. After the administration of sublingual nitroglycerin, her chest pain was substantially reduced and she remained hemodynamically stable. Troponin T levels were positive at 0.106. The rest of her laboratory results were significant for anemia with hemoglobin level of 7.1 g/dL and hematocrit level of 21.3%. Her complete metabolic profile including liver function test were normal.

On subsequent coronary angiography *(Figure 1)*, the patient was noted to have a 1.8-cm saccular aneurysm that involved the distal portion of the left main (LM) coronary artery and extended into the origin of left anterior descending (LAD) and left circumflex (LCx) coronary arteries, with no significant coronary stenoses. Left ventriculography revealed an ejection fraction of 60% with no regional wall motion abnormalities. Aspirin therapy was started, but warfarin was withheld due to the patient's reported history of melena and suspected recurrent upper gastrointestinal bleeding. She was given blood transfusion.

Emergent esophagogastroduodenoscopy (EGD) showed no identifiable etiology. This was followed by capsule enteroscopy, which revealed an isolated discrete arteriovenous malformation (AVM) in proximal small bowel without evidence of active bleeding. Later, noted no bleeding recurrence and she remained asymptomatic. Medical treatment for the LM coronary artery aneurysm was considered and was maintained on aspirin 81 mg daily without additional antiplatlet or anticoagulant therapy. However, on discharge, she was given appointment to follow-up in the cardiology clinic with the consideration to address either more intensive medical therapy or possibly a surgical procedure (ligation of the aneurysm and revascularization with coronary artery bypass grafting) if her symptoms recur or continuous enlargement of the aneurysm will be noticed through serial angiographic evaluations.

DISCUSSION

Coronary artery aneurism (CAA) is an uncommon disease which has been diagnosed with increasing frequency since the advent of coronary angiography. The incidence of coronary artery aneurisms lies somewhere within the range of 1% to 5% in large angiographic and autopsy series with male dominance and a predilection for the right coronary artery [1,2,3]. The majority of aneurisms occur as a consequence of atherosclerosis in adult population. Other causes may include trauma, congenital malformation, mycotic infections, Kawasaki disease, Takayasu's arteritis, polyarteritis nodosa, systemic lupus erythematosus, scleroderma, Ehlers-Danlos syndrome, Marfan syndrome, hereditary hemorrhagic telagectesia (HHT) and idiopathic [1.] It is also a recognized complication of drug-eluting and bare metal stents placement or angioplasty | continued on p.9

Not just another case of back pain

(cont. from p.4)

reported strut penetration rate of 3.5% to 40% [3, 4]. Approximately up to 10% of these perforations are symptomatic and may require intervention [3, 4]. Filter struts can injure any adjacent structures, including the duodenum, aorta, portal vein, small and large intestine, pancreas, kidney, renal vein, diaphragm, genitourinary system and the retroperitoneum [2]. IVC filter penetration and embedding in the vertebral column is extremely rare. Symptomatic IVC filter penetration is an indication for removal of the filter and repair of any injuries. If the patient undergoing filter removal is still at risk for thromboembolic phenomena, the decision to place a new filter or therapeutically anticoagulate must be made accordingly. Close follow-up of patients with IVC filters is required for reevaluation to minimize serious complications.

CONCLUSION

The dramatic increase in IVC filter utilization that we are currently witnessing may lead to significant increases in filter-related complications in the coming years. It is imperative that filter removal be attempted in a timely fashion whenever a retrievable filter is used and that physicians who place filters understand the implications of prolonged and unnecessary indwelling filters.

Close follow-up of patients with IVC filters is required for reevaluation to minimize serious complications.

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Euglycemic Diabetic Ketoacidosis

(cont. from p.5)

tests were attributed to alcoholic hepatitis and patient was placed on Valium alcohol withdrawal protocol.

Eighteen hours after intensive fluid resuscitation bicarbonate level remained low at 9 mmol/L, anion gap was still elevated at 22 mmol/L however glucose level increased to 315 mg/dL. Urinalysis showed glucosuria >500 mg/dL and ketonuria 80 mg/dL. Euglycemic DKA was suspected and decision was made to initiate low-dose continuous intravenous infusion of insulin together with continuation of intravenous infusion of 5% dextrose in normal saline. After seven hours of such therapy bicarbonate level increased to 16 mmol/L, anion gap normalized to 11 mmol/L, ketonuria and ketoacidosis resolved and the patient was still glucosuric at 50 mg/dL. Her HbA1C was 4%. Unfortunately, GAD or anti islet cells antibodies were not tested. Patient was placed on frequent glucose monitoring and sliding scale insulin for a week. Afterwards hyperglycemia resolved and she did not require further insulin therapy. Patient had a long hospital stay complicated by DIC, abdominal compartment syndrome, ARDS with intubation for four days and right-sided pleural effusion requiring thoracentesis. She was discharged home on no insulin with recommendation to follow up with outpatient rehabilitation program to remain sober. Afterwards, patient was lost to follow up.

DISCUSSION

DKA is defined by the American Diabetes Association's (ADA) diagnostic criteria of hyperglycemia [blood glucose >250 mg/dl (13.9 mmol/l)], acidosis (arterial pH <7.3 and serum bicarbonate <15 mEq/l), and ketosis (moderate ketonuria or ketonemia)[1]. In this situation, reduced effective insulin concentrations and increased concentrations of regulatory hormones lead to hyperglycemia and ketosis.

The fact that ketoacidosis in this patient did not resolve until intravenous infusion of insulin was initiated, supports possible underlying unrecognized diabetes and acute depletion of pancreatic beta cells reserve.

Epidemiologic studies suggest that two-thirds of DKA patients have type 1 DM and almost one third have type 2 DM[1]. It is usually associated with elevated blood glucose levels. However in 1973 Munro et al. reported a series of 211 episodes of DKA, around 17.5 % of those (37/211) episodes were described as euglycemic [defined at the time as blood glucose of 300 mg/dl (16.7 mmol/l) or less with plasma bicarbonate of 10 mEq/l or less][3]. Then later in 1993, in a larger analysis, Jenkins et al. reported 23 episodes (3%) of euglycemic DKA in a series of 722 episodes, based on the same diagnostic criteria [6].

It has since been argued that glucose readings above 200 mg/dl (11.1 mmol/l) cannot be considered to represent euglycemia, and therefore a blood glucose level of 200 mg/dl (11.1 mmol/l) or less should be used as the cut-off for defining true euglycemic DKA [2,7]. Based on this criterion, only 16 out of the 37 episodes in the study by Munro et al. [3] and 6 of the 23 episodes in the study by Jenkins et al [6] could be considered to have euglycemic DKA representing respectively 7% and 0.8% of patients presenting with DKA. As per the ADA consensus statement, approximately 10% of DKA population presents with euglycemic DKA which they define with a glucose value of less than 250 mg/dl though1. Euglycemic DKA is thought to be due to a combination of factors: exogenous insulin injection en route to the hospital, food restriction and starvation, and inhibition of gluconeogenesis.

One should always keep in mind that not all patients presenting with ketoacidosis have DKA, moreover if the patients have normal glucose levels. Starvation ketosis and alcoholic ketoacidosis (AKA) should always be in the differential. The acidosis can be relatively severe in AKA. The presence of ketoacidosis without hyperglycemia in an alcoholic patient is virtually diagnostic of AKA. However, modest elevations in serum glucose have been reported in AKA [8]. This may reflect underlying unrecognized diabetes or a catecholamine-mediated stress response. AKA usually occurs following heavy alcohol consumption with abrupt cessation in chronically malnourished alcoholics8. The undetectable alcohol level in our patient argues against AKA from acute intoxication.

In comparison, ketoacid levels in fasting ketoacidosis usually do not exceed 10 meq/L with prolonged fasting alone, which means that the serum bicarbonate concentration is typically above 14 meq/L [9]. The initial low bicarbonate level of 10 meq/l in this patient points towards another

additional mechanism for her ketoacidosis. With the low HbA1c level here, one would argue that diabetes here is of acute onset probably secondary to severe pancreatitis as evidenced by the marked pancreatic changes seen on CT scan *(FIG 1)*. Of course a low HbA1c is not evidence that patient did not have any underlying DM. Unfortunately we do not have an anti GAD level available and patient was lost to follow up to see if she ended up needing insulin therapy.

The fact that ketoacidosis in this patient did not resolve until intravenous infusion of insulin was initiated supports possible underlying unrecognized diabetes and acute depletion of pancreatic beta cells reserve. Severe acute on top of chronic destruction of more beta cells would have lead to acute decompensation and resultant insulin deficiency [10]. On the other hand, decreased glycogen reserve secondary to chronic alcoholism along with prolonged fasting would have lead to the euglycemic state. In DKA, hyperglycemia develops as a result of increased gluconeogenesis, accelerated glycogenolysis and impaired glucose utilization by peripheral tissues [1]. In cases of prolonged fasting, near total glycogen depletion contributes to the normoglycemia as metabolic acidosis continues to develop [11,12]. In practice there might be a considerable degree of overlap between starvation ketoacidosis and euglycemic DKA, as the relative normoglycemia in euglycemic DKA occurs as a result of prolonged fasting [3,6,7]. As evidenced in our patient, as soon as dextrose is infused, patients end up developing hyperglycemia.

Euglycemic DKA should always be considered in the differential diagnosis of ketoacidosis since intravenous insulin infusion and dextrose are the mainstay of therapy. The initial management should be, as in any case of ketoacidosis, to correct fluid/electrolyte abnormalities and re-establish carbohydrate metabolism [1,2,3,13]. Increased glucose administration using higher percentages of dextrose (10% or 20%) are sometimes required to facilitate the concomitant administration of the relatively large amounts of insulin that are needed to correct the severe acidosis in these patients [1,2,13]. Acidosis should improve with normalization of serum bicarbonate without the need for intravenous bicarbonate administration1.

This case is evidence that DKA can occur with normal glucose concentration. Although euglycemic DKA is a rare entity, its correct diagnosis is necessary to tailor therapy accordingly.

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Not Just Another Case of Cardiac Ischemia

(cont. from p.7)

without stenting [4,5,6]. A true arterial aneurysm can be fusiform, involving the full circumference of the blood vessel, or saccular, involving only a portion of the circumference. It can be single or multiple. Such lesions are generally defined as aneurysmal when their diameters exceed by at least 1.5 to 2 times the diameters of adjacent normal sigments or the diameter of the patient's largest coronary artery [1,2,3]. It rarely grows progressively large enough to be called a giant coronary aneurysm [7].

Aneurysm of the left main (LM) coronary artery is exceedingly rare and is the least frequent site of CAA involvement with an incidence of 0.1% [1,8]. To our knowledge, only a few cases have been reported in the world literature [8,9,10,11]. Clinically, most aneurysms are silent. The primary complication is myocardial ischemia or infarction as noted in our case, likely secondary to the abnormal flow within the aneurysm which predisposes patients to thrombus formation within the aneurysm and distal embolization, even in the absence of obstructive coronary disease [1,12].

Giant coronary aneurysm may also be associated with fistulas to a cardiac chamber, most commonly the left ventricle. Those with an associated fistula may have an audible murmur and sign and symptoms of congestive heart failure. Rupture of these aneurysms is rare [13]. We describe a case of LM coronary artery aneurysm present with striking features dissimilar to those observed in a majority of the cases reported in the literature is the relative patency of the coronary arteries in our patient compared to severe multi-vessels disease in up 75% of the reported cases [8]. Therefore, we expect also the possibility that the aneurysm was a rare congenital malformation. Our patient, however, had similar demographics to most of reported cases. As also noted in our patient, more than 50% of those reported cases had extension of the LM coronary artery aneurysm into adjacent branches [8].

Investigators have described a high risk of complete occlusion of aneurismal vessels and myocardial infarction after the early discover of nonstenosed and untreated CAAs [12]. Up to date, there are no controlled trials of therapy for left main coronary artery aneurysms but recommendations of therapy have been based on case study reports. These options which can effectively manage this condition include either medical or surgical treatments. Medical treatment consists of adequate anticoagulation and antiplatelet therapy with close monitoring of those patients. The surgical treatment is by resection or proximal and distal ligation of the aneurism as exclusion from the circulation and revascularization with bypass grafting which has minimal morbidity and mortality [9,14,15,16]. Surgical management is more preferred for symptomatic patients from myocardial ischemia who have evidence of thrombo-emboli from the aneurysm to the distal vessels. It is also indicated in cases of continuous enlargement of the aneurysm as documented by serial angiographic evaluation.

Overall, the prognosis of patients with coronary artery aneurysms associated with coronary atherosclerosis is not different than the prognosis of patients with coronary atherosclerosis without | continued on p.11

Calcaneal Osteomyelitis

(cont. from p.6)

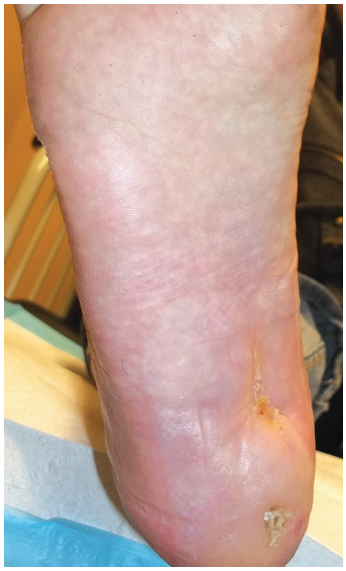

Figure 3

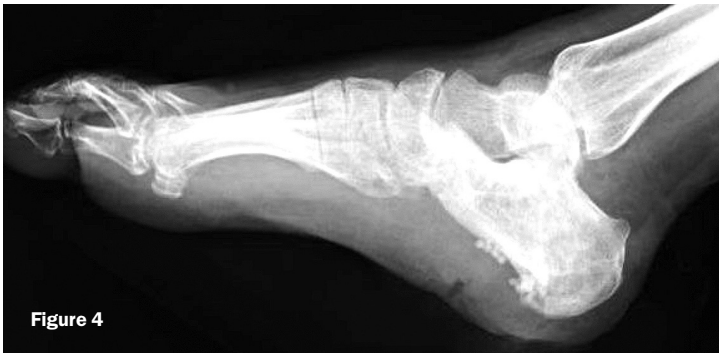

Figure 4

Due to the patient not being amenable to calcanectomy and the lead author's view that calcanectomies in young, sensate patients have poor outcomes; a course of long-term antibiotic therapy was formulated in conjunction with the Infectious Disease department. Weekly sharp debridements and other wound care modalities such as negative pressure and hyperbaric oxygen were employed. Three months after the first admission, the patient was hospitalized due to a Clostridium difficile infection secondary to chronic antibiosis. Ten months after her initial presentation, the patient was admitted for a third time with a diagnosis of cellulitis which emanated from the remaining open wound which was 1cm in circumference at this time *(fig. 4)*. One year from her initial presentation, the patient was admitted once again due to cellulitis and an abscess with a sinus tract. At this time the patient was amenable to a more invasive procedure.

The wound and sinus tract were excised and a calcaneal debridement was performed with multiple cores taken from the calcaneus via trephine. The deficits were packed with absorbable calcium sulfate antibiotic impregnated beads and the wound was left open to allow for drainage of the breakdown products of the beads and to allow for healing by secondary intention *(fig. 5)*. Upon discharge, the patient returned to outpatient visits at our wound care center and was placed on eight weeks of IV Cefazolin therapy.

At the nine-week post-operative visit, a small eschar was noted to the plantar foot *(fig. 6)* which was debrided. There were no local signs of infection noted. The patient complained of pain to the lateral calcaneus which was attributed to alteration of her gait due to the presence of the wound. The Infectious Disease department discontinued IV antibiotics and prescribed oral Keflex as suppressive therapy.

DISCUSSION

This case report presents what we found to be the only second documented case of iatrogenic calcaneal osteomyelitis following a heel injection. After being referred to our institution for management of an abscess, a diagnosis of methicillin-resistant Staphylococcus Aureus calcaneal osteomyelitis was confirmed by core bone biopsies. The initial procedures included extensive debridements, hyperbaric oxygen therapy, Intravenous antibiosis as well as oral antibiosis, negative pressure wound therapy, and aggressive local wound therapy.

At one year the patient presented with a draining sinus tract as well as a small abscess formation to plantar heel. She then underwent a second procedure which included soft tissue sinus tract resection, trephine calcaneal plugs packed with Calcium sulfate antibiotic beads impregnated with vancomycin and closure by secondary intention. The patient was again placed on intravenous antibiosis followed by five year suppressive oral Bactrim.

The literature has presented various techniques dealing with calcaneal osteomyelitis, including a technique first described by Gaenslen in 1931 in which a plantar incision was carried out followed by total resection of the cancellous bone leaving the cortical shell [1]. The defect was not packed with antibiotic beads. Another technique proposed by Oguachuba in 1983 used a closed instillation-suction technique for the treatment of chronic osteomyelitis. In that study only one of the subjects (n=28) had osteomyelitis of the calcaneus (the others being long bones of the lower extremity) and had no recurrence at over one year follow-up [8]. In fact, in this study only two out of the 28 subjects had recurrence of osteomyelitis utilizing this technique.

The use of antibiotic laden beads was first introduced in 1970 and later in 1976 with the advent of PMMA beads as an effective treatment option of acute and chronic osteomyelitis [9]. Studies have shown that minimum inhibitory concentration of antibiotics is released from PMMA beads from the first two days after implantation up to multiple weeks [10]. In our case, the choice was to use Vancomycin calcium sulfate beads to pack the trephine plugs. The bone cultures of the original debridement showed sensitivity to Vancomycin and calcium sulfate beads obviates the need for removal was the rationale behind using this product. A study performed by Chang and colleagues showed promising healing results when using Osteoset® pellets in conjunction with debridement versus debridement alone, even though the results were not statistically significant [11].

Other studies have had successful outcomes with either a calcanectomy or partial calcanectomy for the treatment of calcaneal osteomyelitis in the diabetic cohort [12,13], but due to the significant morbidities associated with partial or total calcanectomies, these procedures should be reserved as a last resort in the sensate, non-diabetic patient.

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(cont. from p.9)

aneurysms [17]. A higher mortality of patients with larger (giant) aneurysms has been shown in the literature suggests a possible threshold in size above which the prognosis is poorer [3.]

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HEALTH FACTS

THE DIRT ON MONEY

MRSA was found on 80 percent of dollar bills according to a recent study. It also showed that 50 percent of the credit cards tested also tested positive for MRSA.

Let them eat chocolate!

Chocolate is no longer just a guilty pleasure. According to a study published in the Journal of Nutrition, the daily consumption of dark chocolate rich in antioxidants can actually prevent future illnesses and provide protection against current environmental hazards. This doesn't mean anyone should start indulging in creamy milk chocolate on a daily basis. In order to reap in the benefits from this tasty treat, the chocolate must contain at least 65 percent cocoa, and if it's organically grown, it's even better for you.

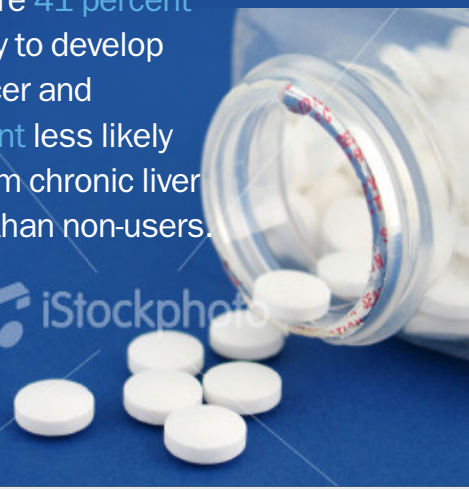
CAN YOU HEAR ME NOW?

There's "no scientific evidence" that cell phones are harmful

It's a common fear that cell phones are giving us cancer. Despite several studies assuring that there are no health risks associated with cell phone use, the fear persists. The latest study by the Norwegian Institute of Public Health gets right to the point: "There is no scientific evidence that low-level electromagnetic field exposure from mobile phones and other transmitting devices causes adverse health effects." The comprehensive 200-page study found that the electromagnetic fields generated by mobile phones are all below thresholds recommended by the International Commission on Non-Ionising radiation protection (ICNIRP). Those thresholds are set to be 50 times lower than the minimum value of electromagnetic radiation required to cause heating of human tissue or stimulation of nerve cells.

Aspirin may reduce risk of liver cancer and give hope to liver disease victims

According to a study of the National Cancer Institute in Rockville, Md., aspirin and other non-steroidal anti-inflammatory drugs or NSAIDS users were 41 percent less likely to develop liver cancer and 45 percent less likely to die from chronic liver disease than non-users.



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