Case Presentation: A 69-year-old male without any known medical problems presented to Albany Medical Center for chief complaints of confusion and “not acting like his usual self.” The patient reported anorexia, general malaise and lethargy. Complete review of systems otherwise was negative. The patient lived alone without family support, and was disconnected from the healthcare system. He did not take any home medications. He had not seen a physician for at least two years prior to admission. His social history was significant for an active 35 pack-year tobacco usage and alcohol consumption of six 12 oz. beers per day. Patient reported that for several days prior to admission, he could not tolerate alcohol consumption due to increasing malaise.

On examination, he was afebrile, cachectic and had normal vital signs other than his BMI of 17.5. There was a 14.7 kg weight loss since his last appointment two years prior to admission. Abdominal examination demonstrated a distended, palpable bladder. No costovertebral angle tenderness or organomegaly was appreciated. On the fingers, all ten digits had nail beds evenly divided by proximal whitish portion 50-60% with a distal pinkish appearance, demarcated at midpoint of the nails (Figure 1). Similar findings were not present on the toes. There was no asterixis on exam. Laboratory data were remarkable for a blood urea nitrogen level of 140 mg/dL and a serum creatinine of 11.5 mg/dL. A renal ultrasound demonstrated bilateral hydronephrosis and grossly enlarged prostate. A temporary hemodialysis catheter and bladder catheter were placed. He was treated for uremic encephalopathy secondary to obstructive uropathy. The patient’s weight loss was attributed to the patient’s longstanding uremia and alcoholism; the patient refused further evaluation.

Discussion: In the physical examination of a patient, the inspection of the hand, and especially the fingernails, can elicit a myriad of relevant data. Digital clubbing, A. Austin MD, PGY-2 Resident, Internal Medicine, Albany Medical College

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Half and Half Nails in Longstanding Obstructive Uropathy; A Clinical Clue of Chronic Kidney Disease (continued)

identified by either increased convexity with distortion of the ungophalangeal angle beyond 180 degrees or by the “floating nail”, is one of the most well known signs. However, there are numerous other cutaneous manifestations of systemic disease, particularly chronic kidney disease, which can have nail or nail bed findings.

Half and half nails, also referred to as Lindsay’s nails, are one such nail finding. The phenomenon was first described by an Iowan physician Philip G. Lindsay in 1967. The nails are identified by a proximal solid, white segment with a distal portion that is usually darker in color, such as brown or pink. The two halves are delineated by a regular and parallel border with the proximal white portion occupying 20-60% length of the nail (1). In comparison with Terry’s Nails, which is typically seen in cirrhotic patients, the white proximal aspect of the nail bed is more than 80%. This proportion of the white tissue is significantly higher with Terry’s nails. The link between Half and Half Nails to renal disease originates to Lindsay’s initial study of 25 patients whereby twenty-one patients had chronic kidney disease and two had some degree of renal insufficiency (1). The exact pathophysiology of Lindsay’s nails has not been clarified, however, since the whitish proximal portion does not change in length as the nail grows, there is a belief the hypopigmentation is related to a process involving the nail bed (2), which does not produce new nail. Although the mechanism is unclear, there has been a link between uremia and half and half nails whereby renal transplantation has been known to reverse the nail finding (3).


Case Report: Successful Removal of a Fully Covered Metal Biliary Stent in the Setting of Severe Sepsis

Jason Sloane, MD, PGY-3 Resident, Internal Medicine, Albany Medical College

Introduction: In 1992, Neuhaus et al became the first published data on the insertion of bare metal stents for palliation of esophageal malignancy (1,2). Since then, metal stents have been used for malignant hilar stricture, common bile duct (CBD) stricture, biliary leak, irretrievable biliary stones, and obstruction of the biliary tree. It was noted early on in their development that bare metal stents had their shortcomings. They had high rates of occlusion, perforation, infection and need for re-instrumentation (3, 10, 11). To address some of these complications, covered metal stents were created. While they did reduce rates of tumor ingrowth compared to uncovered stents, they had higher rates of migration and comparable rates of survival time and patency time (10). In general, patients are advised that plastic stents last 3 months, and metal stents can last up to 12 months. In a recent randomized control trial, average time to stent failure for covered metal stents was 185 days and time to failure for plastic was 185 day (7, 8). There is some thought that because covered stents have less tissue ingrowth that they are better used in patients with obstruction in the absence of a tissue diagnosis of cancer.

Case Description: We present a 60 year old male with history of stroke on Coumadin, chronic pancreatitis, history of ERCP for suspected malignant biliary stricture with placement of covered 10mm x 60mm metal stent and sphincterotomy in 2010, who presented to an outside hospital with two days of fever, nausea, vomiting, and left sided abdominal pain. Labs on admission were notable for significant leukocytosis and supratherapeutic INR. Imaging at the outside hospital revealed a 7.2cm ill-defined fluid collection adjacent to the pancreatic tail suspicious for an abscess. The patient was transferred to Albany Medical Center for further care.

On admission to our facility, he had severe sepsis with acute kidney injury and significant coagulopathy. His labs were significant for leukocytosis of 24,000 with 18% band neutrophils, platelets of 20,000, INR > 9, CO2 17, BUN 24, Creatinine 1.7, and lactic acid of 5.2. His liver function tests revealed a cholestatic pattern with a total bilirubin of 2.5, AST 104, ALT 52, alkaline phosphatase 267. Lipase was normal. His blood cultures grew Escherichia coli. Repeat CT was significant for a complex irregular fluid collection measuring 10 x 8 cm in the region of the pancreatic tail, suspected splenic infarction, enlarged gallbladder, cholelithiasis, and a biliary stent in the common bile duct (Figure 1). The initial treatment plan was to correct underlying coagulopathy and stabilize the patient with IV fluids and broad spectrum antibiotics. Subsequently, the patient underwent CT-guided aspiration of the large peri-pancreatic fluid collection with placement of a 10-French drainage catheter. The aspirate showed a polymicrobial infection including Proteus mirabilis, Escherichia coli, and Streptococcus anginosus. Once he was stable, patient underwent an ERCP, which revealed an occluded metal biliary stent. A 10 Fr by 10 cm plastic stent was successfully placed through the metal stent to provide adequate drainage.

There was initial improvement of liver function and sepsis, but the patient clinically worsened 8 days post procedure with abdominal pain, leukocytosis, and transaminitis. The patient underwent repeat ERCP as part of continuing treatment for sepsis. Two stents originating in the biliary tree were noted emerging from the major papilla. The metal stent was visibly occluded. Pus and sludge were seen emanating from the major papilla (Figure 2). The plastic stent was removed using a snare and the covered metal stent was removed from the biliary tree using a toothed forceps (Figure 3). Pus and sludge were drained from the common bile duct and two plastic stents were placed to maintain patency. The patient has done well since discharge.

Discussion: This case report illustrates complications from prolonged placement of a fully covered metal stent placed 4 years prior to presentation. Chronic pancreatitis can cause biliary obstruction from inflammatory edema, extrinsic compression, fibrotic stricture, and malignancy. In this case, a malignant biliary stricture was suspected and a Whipple procedure was proposed however patient declined and a palliative metal stent was placed. He was subsequently lost to follow up and presented to our institution with severe sepsis from complications of a chronically occluded biliary stent.

Covered metal stents for the treatment of biliary strictures have been in use for over 20 years and are becoming increasingly popular thanks to their durability and positive outcomes. Partially and fully covered metal stents are not labeled for “removability” in the United States but are often used for short-term applications such as benign strictures or intractable bile leaks (3, 4, 5). "Partially covered" stents are especially prone to tissue ingrowth and therefore difficult to remove so they are only suggested for use in a patient with a predicted maximum 24-month life span (10, 17, 18). Covered metal stents are less prone to tissue ingrowth, but as we have illustrated, also can become occluded over time. These long-term covered metal stents are removable but the procedure is technically complicated and has high risk associated (19, 20). We have shown that is series of steps. Several studies have concluded that the most cost effective...
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Jason Sloane, MD, PGY-3 Resident, Internal Medicine, Albany Medical College

Method for resolving an occluded stent was insertion of a plastic stent through the occluded metal stent. The most effective approach however was insertion of another metal stent through the existing one (21, 22). Rates of re-occlusion and longest time to re-intervention were improved. Our case involved insertion of plastic stents to drain infected bile and promote resolution of sepsis and then carefully removing the stent with forceps once inflammation was decreased. This is the first reported case of successful removal of covered metal stent after 5 years.

References:

Case Report: A Unique Etiology for Pleural Effusion

Paul Brasher, MD, PGY-3 Resident, Internal Medicine, Albany Medical College

Heart failure, infections, and malignancies cause the majority of pleural effusions. There are however, several uncommon causes associated with characteristic clinical contexts. This is a case of a patient presenting with urosepsis found to have an urinotherax. Urinotherax is a rare form of pleural effusion that should always be considered when evaluating a patient with obstructive uropathy. Urinotherax is defined as the presence of urine in the pleural space. It was first described in 1968 by studying ureteral obstructions in dogs. Since that time few reports of urinotherax have been reported in the literature.

A 54 year-old female presented from a nursing home with fever, tachycardia, and tachypnea. She had a history of mental retardation with advanced multiple sclerosis, complicated by spastic quadriplegia and a neurogenic bladder managed with a suprapubic catheter. She had had frequent UTI's and calculi requiring past placement of a left nephrostomy tube and a left percutaneous nephrolithotomy with stent placement. History was obtained from medical records as patient was nonverbal at baseline. She was diaphoretic and moaning, appearing to be in obvious pain. Breath sounds were absent in the left lung base. Abdomen was soft, but distended and was moderately tender on palpation. At admission patient had leukocytosis and a fluid collection with inflammatory changes within the left flank retroperitoneal space (Figure 2). A chest tube was placed to drain the pleural...
Case Report: A Unique Etiology for Pleural Effusion

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effusion. Analysis of the pleural fluid revealed a hazy yellow fluid with a creatinine of 1.0 mg/dl. Left flank fluid collection was also sent and found to have a creatinine of 9.4 mg/dl. Serum creatinine at that time was 0.9 mg/dl. Percutaneous nephrostomy tube placed in the left upper pole calyx and drainage tube placed in the left retroperitoneal space demonstrated a connection between them after injection of dye under fluoroscopy. Chest tube drainage progressively slowed once a nephrostomy tube was placed. Urine culture and left flank aspirate grew MRSA susceptible to vancomycin and doxycycline. Patient was treated with vancomycin and discharged on doxycycline until removal of the staghorn calculi could be done by urology at a later date.

Urinothorax can result from an obstructive process such as retroperitoneal fibrosis, malignancy, or nephrolithiasis; or from iatrogenic/traumatic causes such as percutaneous nephrostomy, renal biopsy, or blunt trauma. Urinothorax in this case was likely multifactorial from her previous nephrolithotomy and current obstructive uropathy. Several routes for movement of urine into the pleural space have been suggested but direct movement of leaked urine in the abdomen into the pleural space via defects in the diaphragm is the likely mechanism. Interestingly, unilateral obstruction at the ureteral or kidney level does not appear to cause urinothorax if the function of the contralateral kidney is preserved. Such was the case in our patient who was found to have bilateral staghorn calculi.

Establishing a diagnosis of urinothorax requires analysis of pleural fluid, which often has an appearance and odor consistent with urine. A hallmark of urinothorax is a pleural fluid to serum creatinine ratio of > 1.0. Though a very high ratio is certainly suggestive of urinothorax, there appears to be a degree of variability in pleural fluid creatinine level in urinothorax that may complicate interpretation. The pleural fluid protein level has consistently been reported to be low with a pleural fluid LDH in the exudative range. Other, less reliable indicators, are a low pH with an elevated glucose level in the pleural fluid.

In this case the diagnosis of urinothorax was made based on fluid analysis and high pre-test probability given the clinical context. If diagnosis is unclear a renal scan using technetium-99m labeled mercaptoacetyltriglycine (MAG-3) may aid in establishing the diagnosis by demonstrating leakage of the tracer from the urinary tract into the pleural space. This case demonstrates the importance of recognizing clinical contexts that warrant consideration of rare causes of pleural effusions.

The patient is a 34-year-old male who presented with migratory arthritis. He started feeling unwell 14 days ago with a dry cough, fatigue, night sweats, and nasal congestion. Four days after his syndrome started he developed ankle pain and swelling that began in his left ankle and spread to the right ankle a few days later. Initially gout was diagnosed and he failed to improve with NSAIDs. Five days into his illness he developed a rash on his lower left leg. The rash consisted of 3 tender violet colored sub centimeter lesions on his left lateral ankle and anterior shin. In addition to ankle involvement, also noticed bilateral knee pain without edema, and 1 day of left hip pain. No pain in elbows, shoulders, neck, or hands. Typically active, he was limited in his activities of daily life by the pain. He had decreased appetite but denied nausea, vomiting, diarrhea. Of note, he recently traveled to Beijing, China in early December and stayed for 3 days. During this time, he remained in the city, did not eat any food from street vendors nor did he travel to the countryside. He consumed raw oysters 1 week prior to the onset of his symptoms. Physical Examination was significant for fever of 100.5 F, and HR of 90. Bilateral adenoids were enlarged, and there was mild non tender anterior chain cervical lymphadenopathy. Lesions were located on left lower leg. Lesions appeared to have a cigarette burn appearance with black eschar with surrounding erythema (Figure 1). There was significant non pitting ankle edema on the left with decreased ROM secondary to pain. The right foot and ankle also had non pitting edema but was not tender on ROM. Rest of exam including lungs, heart, abdomen, nervous system was unremarkable. On labs AST and ALT were mildly elevated less than 1.5 x normal.

**Test Your Knowledge with our Picture quiz:**

Jason Sloan, MD, PGY-3 Resident, Internal Medicine, Albany Medical College

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**Answer:**

Tsutsugamushi disease

Also known as Scrub typhus, Tsutsugamushi disease is a rickettsial infection caused by Orientia tsutsugamushi. Rickettsiae are obligate intracellular, Gram-negative bacteria. Rickettsiae can cause a host of human diseases ranging from Rocky Mountain spotted fever to Typhus. A list of the typical presentation including rash appearance is seen in Table 1. The scrub typhus bacterium is introduced into the human host after a bite of a larval stage trombiculid mite. The mite and associated organism are endemic to areas all over Asia. Eschar is evident in 23-78% of cases. Scrub typhus symptoms typically include fever, headache, cough and gastrointestinal symptoms. Symptoms may also include diffuse lymphadenopathy, myalgias, arthralgias, and splenomegaly. Typically rash begins as a papule then enlarges, undergoes central necrosis, and eventually develops a blackened crust with erythema surrounding. This has been described to appear like a “cigarette burn”. Encephalitis and myocarditis have been reported in late phases of the illness. Diagnosis is made on clinical presentation alone in endemic areas. There have been multiple case reports of people returning to the United States with the illness from endemic areas but diagnosis took a long time because symptoms are similar to dengue, paratyphoid, and FUO. Choice of test for presence of the disease is controversial because no single test is all that sensitive or specific. Serologic testing is available and more sensitive at higher antibody titers. Skin biopsy of the eschar can show popular erythema with lymphocytic vasculitis with C3, C4, C5, IgA and IgM vascular immune deposits. PCR can be run from tissue biopsy to definitively diagnose this disease. This pattern can also show up in small vessel vasculitis so clinical correlation is needed. Treatment of choice is doxycycline. Azithromycin is a safer alternative in pregnant women, children, and when resistance to tetracycline is suspected. Mortality is less than 1% with adequate treatment and up to 60% in untreated cases.