Pylephlebitis and pyogenic liver abscesses: A complication of hemorrhoidal banding

Nicole G Chau BSc MD1, Sacha Bhatia MD MBA1, Maitreyi Raman MD FRCPC2

CASE PRESENTATION

A 49-year-old Vietnamese man presented to the emergency department of the Toronto Western Hospital, University Health Network (Toronto, Ontario) with a five-day history of fever, right upper quadrant pain, ascites, vomiting and hematochezia following hemorrhoidal banding treatment for symptomatic internal hemorrhoids with normal colonoscopy at an outpatient community clinic. Rectal varices were absent on colonoscopy. Past history included type II diabetes with retinopathy, hypertension, dyslipidemia, minor lacunar stroke and remotely treated pulmonary tuberculosis. His only medications were oral hypoglycemics (metformin and glyburide) and antihypertensives (amlodipine). He denied alcohol, illicit drug use, known liver disease or risk factors for thrombosis.

Liver function was abnormal with albumin at 17 g/L, an international normalized ratio of 1.1 and total bilirubin of 17 μmol/L. Creatinine was elevated at 170 μmol/L but normalized with hydration. Doppler abdominal ultrasound revealed cirrhosis with extensive portal vein thrombosis, ascites, two hypechoic liver lesions consistent with hepatic abscesses and an unremarkable biliary tree.

Empiric piperacillin/tazobactam (4.5 g given intravenously every 8 h) was quickly initiated for pylephlebitis (septic portal vein thrombosis) and pyogenic liver abscesses (PLA). There was immediate defervescence. A computed tomography (CT) scan of the abdomen confirmed right, left and main portal vein thrombosis, splenic and superior mesenteric vein thrombosis and five small, hypodense liver lesions in segments 1, 4A and 5 that were consistent with multifocal abscesses (Figure 1).

Blood cultures and ultrasound-guided aspirate from the largest abscess (2.5 cm) grew Klebsiella pneumoniae sensitive to ciprofloxacin. Therapeutic CT-guided aspiration was unsuccessful. Gastroscopy performed for melena revealed grade 2 to 3 esophageal varices that were banded. Colonoscopy confirmed no mass or rectal varices. Cirrhotic workup revealed only a positive hepatitis B core and surface antibody. Hypercoagulable workup was negative. Induced sputum for acid-fast bacilli was negative. Oral ciprofloxacin 500 mg twice daily was continued for six weeks after discharge. Follow-up imaging showed resolution of abscesses and improvement in portal vein thrombosis.
DISCUSSION

Septic complications of hemorrhoidal banding are rare but can be fatal. A review (1) of 39 studies including 8060 patients undergoing hemorrhoidal banding revealed a rate of infection of 0.05%, and a retrospective study (2) of 805 patients undergoing hemorrhoidal banding revealed only one case of bacteremia (0.09% sepsis). However, at least six deaths due to septic complications following hemorrhoidal banding have been published in nine papers (3). Septic complications posthemorrhoidal banding include abdominal pain, fever, urinary retention, local perirectal edema and cellulitis extending to the pelvis and thighs that occurred two to seven days following hemorrhoidal banding treatment (3). Responsible organisms confirmed at autopsy have included Escherichia coli and Clostridium species (4,5). Prevention of local septic complications include the use of enemas before banding, sterile instruments and povidone-iodine preparation (3). Early review of patients within a few days posthemorrhoidal banding is advised in patients with previous septic complications (6).

Risk factors for septic complications include HIV infection (6,7), immunosuppression, phenothiazines, intravenous drug abuse (4), diabetes and rheumatic diseases (6,7).

To our knowledge, this is the first reported case of pylephlebitis following hemorrhoidal banding and the second reported case of PLA following hemorrhoidal banding treatment. Interestingly, the only other case (8) of PLA following hemorrhoidal banding was also in a Vietnamese male with diabetes and a remote history of tuberculosis. Although K pneumoniae was isolated, the patient required right hepatectomy to control sepsis. These cases highlight the increased risk of PLAs due to K pneumoniae in patients with diabetes who experience gastrointestinal mucosal injury through hemorrhoidal banding.

It is possible that the present patient had pre-existing chronic liver disease secondary to hepatitis B or nonalcoholic fatty liver disease, and portal vein thrombosis that potentially resulted in rectal varices. Certainly, the presence of significant portal hypertension is supported by the presence of esophageal varices confirmed by endoscopy. However, colonoscopy at the community clinic and colonoscopy at the Toronto Western Hospital did not show rectal varices. If rectal varices indeed were missed in our patient, it would be intuitive to think that the presence of pre-existing liver disease and portal vein thrombosis would result in a higher complication rate following endoscopic rectal varical ligation compared with internal hemorrhoidal banding in a patient without pre-existing liver disease or portal vein thrombosis. A thorough MEDLINE literature search to date did not reveal clear data to ascertain the actual difference in complication rate.

Pylephlebitis (septic portal vein thrombosis) is a rare but serious condition that can complicate any intra-abdominal infection that is often secondary to diverticulitis, appendicitis, biliary tree infection or inflammatory bowel disease. Clinical presentation is often nonspecific; however, pylephlebitis should be suspected in patients with fever, right upper quadrant tenderness or jaundice. Diagnosis is best confirmed by CT scan or colour flow Doppler ultrasonography to demonstrate portal vein thrombosis in a patient with bacteremia. Mortality ranges from 11% to 32%, even with the use of antibiotics (9,10). The mainstay of treatment is early initiation of parenteral broad-spectrum antibiotics followed by specific antibiotics against the bacterial isolates that may lead to resolution of both portal vein thrombosis and hepatic abscess.

The median duration of therapy in surviving patients is 4.2 weeks (9). Bacteroides fragilis and Escherichia coli are the most common isolates. Portal vein thrombosis may progress to fatal mesenteric vein thrombosis (10) but the role of anticoagulation is controversial. In the absence of malignancy, hypercoagulable disorder and multiple thromboses, we elected not to anticoagulate this patient, who also had melena secondary to esophageal varices. Colonoscopy confirmed the absence of diverticulitis or malignancy in this patient.

PLA is rare, with an incidence rate of 2.3 per 100,000 people; incidence is higher in men, immunodeficient states and diabetes patients (11). Mortality ranges from 10% to 12% (12). Clinical manifestations are nonspecific and include fever, abdominal pain and enemia; diagnosis is confirmed by CT scan or ultrasound. The etiology of PLA is frequently from biliary pathology (often polymicrobial), hematogenous (portal system entry or hepatic artery from intestinal focus such as diverticulitis or inflammatory bowel disease), direct (percutaneous intervention or trauma) or by contiguity. PLA may be the first manifestation of colorectal cancer, even in the absence of hepatic metastases. Therefore, colorectal cancer screening is warranted in patients with PLA in the absence of biliary pathology (13). PLA has been reported in two patients following hemorrhoidectomy, and liver isolates grew Streptococcus viridans and K pneumoniae (14). PLA has also been reported following surveillance colonoscopy in a patient with inflammatory bowel disease (15).

K pneumoniae is the predominant cause of PLA in people of Asian origin (16,17), with rising incidence in Western countries (18,19). Minor mucosal injuries of the gastrointestinal tract are thought to allow entry of K pneumoniae into the bloodstream, with passage through the portal vein resulting in sequestration by Kupffer cells in the liver, leading to liver abscess formation. K pneumoniae liver abscesses (KLA) are usually monobacterial with bacteremia occurring in 95% of cases (20); however, 64% of KLA are cryptogenic (21). Standard ampicillin/gentamicin/metronidazole regimens can
be hazardous because K. pneumoniae is intrinsically resistant to ampicillin and metronidazole is ineffective against aerobes. The preferred empirical regimen for KLA is a combination of aminoglycoside and extended-spectrum beta-lactams (19). Drainage is essential for treatment in most cases; however, antibiotic therapy alone may be effective. One study (22) found percutaneous transhepatic drainage to be as effective as surgery to treat KLA.

Diabetes is the most common concomitant disease in patients with KLA (16,17,19-21). Of 160 patients with PLA, diabetes was present in 67.5% of patients with KLA compared with only 4.5% of patients with polymicrobial liver abscesses (RR of 11.1) (16). Diabetes is also a risk factor for extrahepatic metastases from KLA, including endophthalmitis (16,23) and septic pulmonary emboli (20). The increased risk of PLA in patients with diabetes may be related to the interference with neutrophil chemotaxis and phagocytosis (24-26).

**REFERENCES**