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CASE REPORT | INFLAMMATORY BOWEL DISEASE

Abdominal Pain in a Patient With Diverted Bowel and Inflammatory Bowel Disease

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ABSTRACT

Patients with medically refractory inflammatory bowel disease may undergo total proctocolectomy with ileal pouch-anal anastomosis. However, fecal diversion is necessary in patients with pouch failure. We present a rare case of pyogenic liver abscess (PLA) in a patient with ulcerative colitis with a history of ileal pouch-anal anastomosis complicated by chronic pouchitis requiring fecal diversion via loop ileostomy. The PLA was managed with computed tomography-guided aspiration drainage and antibiotics, followed by permanent ileostomy and pouch excision to prevent recurrence of PLA. This is the first case report of PLA in a patient with ulcerative colitis with a long-standing history of diversion pouchitis.

INTRODUCTION

Pyogenic liver abscess (PLA) is a rare disease with an incidence of 3.6 cases per 100,000 in the United States and a high mortality rate in untreated patients.[1] PLA is a recognized complication of Crohn's disease (incidence of 114 to 297 per 100,000); however, PLA is less frequent in ulcerative colitis (UC), with only 12 cases reported in the literature.[2–12] We present the first case of a patient with UC who had undergone long-term bowel diversion and developed a PLA.

CASE REPORT

A 46-year-old woman with UC after total proctocolectomy with ileal pouch-anal anastomosis at the age of 22 years complicated by chronic pouchitis requiring fecal diversion via diverting loop ileostomy at 43 years of age presented to an outside hospital with a 3-week history of intermittent right upper quadrant abdominal pain and increased nonbloody ileostomy output. Along with chronic pouchitis, she had a history of pouch fistulae and recurrent pouch strictures, raising the suspicion for Crohn's disease of the pouch, which precluded surgical reanastomosis and necessitated needle knife stricturotomy every 6 months. The last needle knife stricturotomy session was 4 months ago and revealed a 2-cm pouch outlet stricture with severe diversion pouchitis (DP). The patient denied the use of immunosuppressants, anti-tumor necrosis factor medications, short-chain fatty acids (SCFAs) enemas, topical mesalamine, or glucocorticoids for the past year. On presentation, her temperature was 100.7°F, blood pressure was 91/65 mm Hg, heart rate of 140 beats/min, and oxygen saturation was 98% on room air. On examination, the abdomen was soft and nondistended without hepatomegaly; right upper quadrant was tender to palpation, without rebound or guarding; and bowel sounds were present. The skin surrounding the stoma was intact without erythema, purulent drainage, or tenderness. Laboratory test results revealed a white blood cell count of 13,600/mm^3 and hemoglobin of 11.1 g/dL; liver function tests revealed a hepatocellular pattern of injury (aspartate aminotransferase of 157 IU/L and alanine aminotransferase of 170 IU/L, alkaline phosphatase of 95 IU, and total bilirubin 0.9 mg/dL). The urinalysis was negative for infection; urine drug screen and human immunodeficiency virus testing were also negative.

The patient was started on intravenous (IV) fluids, blood cultures were obtained, and empiric meropenem was administered for sepsis of unknown origin. Abdominal ultrasound revealed a liver mass, and abdominal computed tomography (CT) with IV contrast

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showed a 10.7 × 9.5 × 7.4 cm heterogeneous liver mass. An echocardiogram did not reveal valvular abnormalities. There was no recent travel history and no IV drug use.

The next day, a repeat abdominal CT scan showed a hypoattenuating 12.7 cm hepatic abscess with a mass effect on the main portal vein (Figure 1). CT-guided aspiration of the liver abscess yielded 35 mL of foul-smelling fluid, and a Jackson-Pratt drain was placed. Abscess culture was positive for *Streptococcus anginosus* and *Parvimonas micra*. Blood cultures from admission and stool testing for enteric pathogens were negative. The patient was discharged on hospital day 17 and continued on IV meropenem for an additional 3 weeks before being switched to amoxicillin-clavulanate for 2 more weeks. The Jackson-Pratt drains were removed after 5 weeks, at which point there was no further evidence of a residual PLA. With shared decision-making between the patient and the clinicians, 3 months after recovery, the patient underwent surgical excision of the diverted pouch and creation of end ileostomy to prevent future risk of PLA. At the 3-month follow-up, the patient was asymptomatic with good stoma function.

DISCUSSION

This is the first case report of PLA in a patient with UC and a long-standing history of DP and recurrent pouch strictures. DP results from a lack of SCFAs and other luminal nutrients because of the absence of the fecal stream. This leads to alteration in the pouch flora and inflammation, which predisposes to pouch stricture formation. Patients with DP present with bloody or mucoid discharge and cramping abdominal pain. Endoscopy reveals friability, erythema, and granularity. Biopsy of the pouch and stool testing is performed to exclude infectious etiology (such as *Clostridium difficile* and *Cytomegalovirus*).[13] Definitive treatment of chronic pouchitis and strictures is surgical reanastomosis. Medical therapy with SCFA enemas, topical mesalamine, or glucocorticoids is used if reanastomosis is contraindicated. However, pouch excision and permanent diversion is necessary if there is pouch failure because of pelvic sepsis, chronic pouchitis, or recurrent stricture/fistula formation in the pouch.[14]

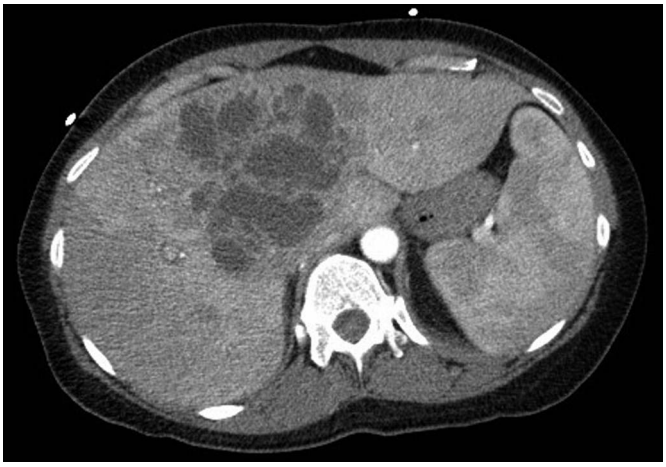

Figure 1. Abdominal computed tomography showing a multiloculated, cystic hypodensity centered in the central liver, extending from the hepatic dome to the inferior surface of the liver. There is heterogeneous enhancement throughout the liver on the arterial phase.

PLA is commonly due to biliary tract disease (such as cholangitis or cholecystitis), followed by intra-abdominal infections in which bacteria enter the liver through the portal venous system.[15] Patients present with fever and right upper quadrant abdominal pain. Laboratory studies reveal leukocytosis with elevation in aminotransferases and serum bilirubin. CT reveals a hypoattenuating lesion, with the right hepatic lobe being most commonly involved.[15] PLA is usually polymicrobial, with predominantly aerobic Gram-negative bacteria. Treatment requires 4 weeks of antibiotic therapy and drainage (either percutaneous or surgical).[16]

Previously, 12 cases of PLA associated with UC have been reported.[3–12] Most cases were associated with active inflammation, and none of the patients had a history of surgical intervention. The associated pathogens were usually normal gut flora, such as the *Streptococcus anginosus* family. It is believed that inflammation damages the colonic mucosal barrier of patients with inflammatory bowel disease, predisposing to microbial invasion of the portal tract.[3] One case of PLA occurred after surveillance colonoscopy with 26 biopsies in a patient with UC, highlighting the increased risk of bacterial translocation.[11] Animal models have shown that the mucosa in a diverted colon becomes a breeding ground for bacteria, increasing the probability of bacterial penetration through the mucosal barrier.[17,18] Likely, the combination of pouchitis and recurrent stricture formation in the pouch contributed to the development of PLA in our patient. Owing to the history of anal fistula, chronic pouchitis, and recurrent stricture, pouch excision was deemed as the next best step in improving the patient's quality of life and for prevention of PLA recurrence.[19] In summary, clinicians should have a low threshold for diagnosing PLA in patients with inflammatory bowel disease and long-term bowel diversion who present with abdominal pain and fever. Pouch excision may be necessary to prevent recurrence of the PLA.

DISCLOSURES

Author contributions: B. Hasan and R. Khalid wrote the manuscript. R. Charles and B. Chen revised the manuscript for intellectual content. B. Hasan, R. Khalid, R. Charles, and B. Chen approved the final manuscript. B. Hasan is the article guarantor.

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