Liver abscess of the caudate lobe due to Staphylococcus aureus in an ulcerative colitis patient: First case report

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Received 15 January 2011; received in revised form 15 February 2011; accepted 15 February 2011

KEYWORDS: Ulcerative colitis; Liver abscess; Staphylococcus aureus; Caudate lobe; Recurrence

Abstract

Liver abscesses are very rare complications of ulcerative colitis, with only nine cases described in the literature, to our knowledge. We report a case where a recurrence of liver abscess occurred in an ulcerative colitis patient, in two different hepatic lobes, which has not been previously described. The recurrence was in the caudate lobe having the cultures yielded Staphylococcus aureus. This is also the first case report of liver abscess in this location and caused by this microorganism in an ulcerative colitis patient. A review of the literature concerning liver abscesses involving ulcerative colitis patients is also provided.

1. Introduction

Diseases involving the hepatobiliary system are frequently encountered in patients with Inflammatory Bowel Disease (IBD) and comprise some of the most common extraintestinal manifestations. These complications include cholelithiasis, non-alcoholic fatty liver disease, primary sclerosing cholangitis, chronic active hepatitis, granulomatous hepatitis, amyloidosis, biliary tract carcinoma, and liver abscesses. Liver abscesses are however a rare complication of IBD. Most cases described to date were in patients with Crohn's disease.

2. Case report

A 67-year-old man with history of abdominal pain and bloody diarrhea was diagnosed with distal ulcerative colitis in 2005.
first total colonoscopy showed the sigmoid and rectal mucosa with edema, erythema, and friability. The remaining colonic mucosa had no lesions. Oral mesalazine 3 g/day and topical treatment with mesalazine enemas were started. He was a non-smoker, had no family history of ulcerative colitis, and had been submitted to a transvesical prostatectomy for benign prostatic hyperplasia in March 2006.

In May 2006, he was admitted to our hospital, with fever and abdominal pain. He did not have other complaints, such as diarrhea. The initial laboratory evaluation revealed leucocytosis (18.47×10⁹/L) with an elevated neutrophil count (14.22×10⁹/L), anemia (hemoglobin 10.4 g/dl), elevated C-reactive protein (283 mg/L), and elevated alkaline phosphatase (436 U/L). A computed tomography scan (CT) was performed and showed two liver abscesses in the right lobe with 8.2 cm (Fig. 1A) and 1.86 cm (Fig. 1B). They were drained percutaneously via ultrasound and antibiotherapy with meropenem was started. The abscess culture was negative. Abscess aspiration did not show neutrophilic features. Percutaneous drainage and antibiotherapy accomplished complete resolution of the liver abscesses.

A colonoscopy (progression until the descending colon) was performed in 2007 and showed edema, erythema, and erosions in the mucosa of the rectum, sigmoid and descending colon. A first course of corticosteroids (60 mg/day) was initiated. During a disease flare (bloody diarrhea) in May 2010, a total colonoscopy was performed and showed the sigmoid and rectal mucosa with edema, friability, and ulceration. Immunohistochemistry for the detection of cytomegalovirus (CMV) was negative. A full work up was done, including stool examination (including *Clostridium difficile* toxins), serum CMV antigen, and Polymerase Chain Reaction (PCR) for CMV DNA that was negative. A second course of corticosteroids was administered.

In August of 2010, he was readmitted to our institution with fever and abdominal pain, and a 5.5 cm pyogenic liver abscess was detected in the caudate lobe (Fig. 2) and it was drained percutaneously under sonographic guidance. He had completed a course of corticosteroids one week prior to admission. The culture of the pus was positive for *S. aureus* and blood cultures were negative. At this time, he did not have diarrhea, but maintained endoscopic activity.

No skin lesions were found during the clinical presentation of the liver abscesses.

In October 2010, he was asymptomatic, but colonoscopy showed the sigmoid and rectal mucosa with edema, erythema, superficial ulceration and an 8 cm sigmoid stenosis, overcome with the scope. The histology revealed chronic inflammation of the rectal and sigmoid mucosa with activity and ulceration, without dysplasia. No amoebae were detected.

Real-time PCR (protein chain reaction) assay of *E. histolytica* in the colonic mucosa and *E. histolytica* serology were negative. The parasitological stool exam was negative. The liver blood tests were normal.

Following the diagnosis of ulcerative colitis, he was medicated with oral mesalazine 3 g/day and topical treatment once a day with mesalazine enemas. In October 2010 immunosuppressive therapy with azathioprine was started.

### 3. Discussion

Liver abscesses are a possible hepatobiliary complication of IBD, but this entity is very uncommon, with only 60 cases reported to date, most of them in patients with Crohn’s disease. Clinical manifestations may include fever, chills, anorexia, weight loss, abdominal pain with right upper quadrant tenderness, and pulmonary findings, particularly right pleural effusion. Leucocytosis with an elevated neutrophil count, anemia, and elevated acute phase reactants are among the laboratory findings. An elevated serum alkaline phosphatase level is the single most reliable laboratory abnormality for liver abscess, but its specificity and sensitivity are limited.

![Figure 1](https://example.com/fig1.png)  
**Figure 1** A: Abdominal computed tomography (CT) scan in 2006, demonstrating hypodense lesion in the right hepatic lobe with 8.2 cm compatible with liver abscess. B: Abdominal CT scan in 2006 also showing a liver abscess in the right lobe with 1.86 cm.

![Figure 2](https://example.com/fig2.png)  
**Figure 2** Abdominal CT scan in 2010 revealing a 5.5 cm liver abscess in the caudate lobe.
Table 1  All reported cases of liver abscesses in ulcerative colitis patients.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>UC Location</th>
<th>Interval between onset UC and abscess</th>
<th>Activity/remission UC</th>
<th>Previous steroid therapy</th>
<th>Number of abscesses</th>
<th>Liver location</th>
<th>Bacteriology</th>
<th>References</th>
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<tr>
<td>Male</td>
<td>18</td>
<td>Information not available</td>
<td>2 years</td>
<td>Activity</td>
<td>None</td>
<td>Multiple</td>
<td>Information not available</td>
<td>Diplostreptococci</td>
<td>9</td>
</tr>
<tr>
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<td>4 years</td>
<td>Activity</td>
<td>None</td>
<td>Multiple</td>
<td>Information not available</td>
<td>Hemolytic streptococcus, E. coli</td>
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<tr>
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<td>45</td>
<td>Distal</td>
<td>4 years</td>
<td>Activity</td>
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<td>Solitary</td>
<td>Right lobe</td>
<td>Non hemolytic streptococci E. coli</td>
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<tr>
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<td>Unknown</td>
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<td>Solitary</td>
<td>Information not available</td>
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</tr>
<tr>
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<td>45</td>
<td>Information not available</td>
<td>0</td>
<td>Activity</td>
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<td>Multiple</td>
<td>Information not available</td>
<td>Negative</td>
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<tr>
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<td>84</td>
<td>Distal</td>
<td>0</td>
<td>Activity</td>
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<td>Right and left lobe</td>
<td>Streptococcus intermedius</td>
<td>3</td>
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<tr>
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<td>58</td>
<td>Pancolitis</td>
<td>10 years</td>
<td>Activity</td>
<td>Yes</td>
<td>Multiple</td>
<td>Right and left lobe</td>
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<td>Activity</td>
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<td>Multiple</td>
<td>Right lobe</td>
<td>Not done</td>
<td>8</td>
</tr>
<tr>
<td>Female</td>
<td>18</td>
<td>Pancolitis</td>
<td>0</td>
<td>Activity</td>
<td>None</td>
<td>Multiple</td>
<td>Right lobe</td>
<td>Negative</td>
<td>4</td>
</tr>
<tr>
<td>Male</td>
<td>67</td>
<td>Distal</td>
<td>1</td>
<td>Activity</td>
<td>None before 1st abscess/yes before 2nd abscess</td>
<td>Multiple</td>
<td>In 2006 multiple/ in 2010 solitary</td>
<td>Staphylococcus aureus</td>
<td>our case</td>
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Adapted and updated from Song et al3.
Liver abscess in ulcerative colitis

Hence, imaging techniques are required for an early diagnosis of liver abscess.

Streptococcus was the most common pathogen identified in liver abscesses in UC patients, followed by E. coli. S. aureus has been previously isolated from liver abscesses in patients with Crohn’s disease, but never reported in UC. In 1994, Vakil et al. reported six patients with liver abscesses as complications of Crohn’s disease, and two of these patients were S. aureus identified in abscess cultures. Both reported cutaneous infections in the two weeks preceding the diagnosis of the liver abscess. In IBD anaerobic cultures should be performed. Abscess cultures are positive in most cases (7 of 10 reported cases of UC). In contrast to liver abscesses detected in the general population, which are usually polymicrobial with several gram-negative strains, in most cases of liver abscesses in UC a single pathogen is isolated.

In 2006, the abscess cultures were negative and in this case aseptic abscess (AA) should be ruled out as a possible diagnosis. Aseptic abscesses (AA) are characterized by deep, sterile, round lesions consisting of neutrophils that do not respond to antibiotics but improve dramatically with corticosteroids. However, in our case, there was a complete response to percutaneous drainage and antibiotic treatment (meropenem), making AA an unlikely diagnosis. Furthermore, abscess aspiration did not show neutrophilic features. AA occurs in young adults, especially in those with IBD and the spleen is a privileged site of AA.

Early diagnosis with imaging studies, drainage therapy, and appropriate antibiotic treatment seem to improve outcome. Patients commonly receive antibiotics for 4–8 weeks, often intravenously for the entire duration. In the past, liver abscesses were considered a clear indication for surgical drainage. In recent years, a more conservative approach has been adopted and percutaneous drainage is now considered the procedure of choice with favorable outcomes.

Bacteremia involving the portal venous system is well documented in patients with IBD and studies of patients with ulcerative colitis suggested an increased incidence of portal bacteremia. Ulceration and loss of integrity of the normal mucosal barrier may predispose to microbial invasion of the portal venous system. Nine of the ten (90%) previously described cases of liver abscesses in UC were in active colitis (Table 1).

The predilection for the right lobe can be explained anatomically. The right hepatic lobe receives blood from both the superior mesenteric and portal veins, whereas the left lobe receives inferior mesenteric and splenic drainage. It also contains a denser network of biliary canaliculi and, overall, accounts for more hepatic mass.

The first case of liver abscess in a patient with ulcerative colitis was reported by Lansbury and Bargen in 1993, and since then only 9 cases have been published in the literature, to our knowledge. Six were described in men and four cases in women (taking into account this case), with ages ranging from 18 to 84 years. The interval between the onset of ulcerative colitis and liver abscess varied from 0 to 16 years, and three patients presented with an abscess as the initial manifestation of ulcerative colitis (Table 1). Intra-abdominal abscesses, fistulous disease, active colitis, and metronidazol therapy have been reported to be important predisposing factors to liver abscesses in IBD, mainly in Crohn’s disease. Corticosteroids have been recognized to dispose abscess development due to their immunosuppressive effects; however, only two patients with UC and liver abscesses had previously received corticosteroids (Table 1).

Herein, a patient with UC is reported and when a first liver abscess was diagnosed he had active colitis, but never taken corticosteroids. Mucosal barrier disruption probably predisposed to bacterial invasion of the portal venous system and consequently liver abscesses. In 2010 when the second abscess appeared he had recently (one week prior) suspended a two-month course of prednisolone following a disease flare. To our knowledge, this is the first case of a liver abscess recurrence in an UC patient, occurring in different hepatic locations: in 2006 in the right lobe and in 2010 in the caudate lobe.

In conclusion, liver abscess is a rare complication of UC patients and there is an increased tendency to develop multiple abscesses in this setting. A microorganism was isolated in most of the cases, and almost invariably involved the right liver lobe and occasionally the left lobe. Notably, this complication was consistently diagnosed during disease activity. To our knowledge, this is a rare report considering it is the first case with recurrence, which occurred in two different hepatic lobes, was caused by S. aureus, and located in caudate lobe of the liver in a patient with UC.

References