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Single Case

## **Pyogenic Liver Abscess Secondary to Sigmoid Diverticulitis: An Unusual Presentation**

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#### Keywords

Diverticulitis · Abscess · Infection · Streptococcus

#### Abstract

Pyogenic liver abscesses, despite being a rare complication of diverticulitis and inflammatory bowel disease, are potentially serious life-threatening pathologies. Diverticular diseases can lead to disruption of the colonic mucosal barrier and can serve as a route for bacterial infection via the portal venous system. This patient had such a delayed presentation due to his atypical symptoms; he developed large abscess formation, detected by computed tomography, eventually requiring an ultrasound-guided drain followed by a colonoscopy. The present report describes an elderly gentleman who developed a bacterial liver abscess due to seeding of a commensal organism caused by sigmoid diverticulitis.

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#### Introduction

The literature has described liver abscesses to have a characteristic presentation resembling that of cholangitis with symptoms such as fever, right upper abdominal pain, jaundice and weight loss. Usually, common bile duct infections are associated with up to 50% of liver abscesses [1]. The most common cause of a bacterial liver abscess is biliary disease. However, other causes exist, such as septicaemia, traumatic liver injury, inflammatory bowel disease, diverticulitis and a ruptured appendix [2]. A ruptured appendix with systemic spread was

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once deemed as the primary cause for a bacterial liver abscess; however, this is not the case with improved imaging and intervention. With respect to bowel pathology, sigmoid diverticulitis is an uncommon cause for this disease. The *Streptococcus milleri* group of bacteria are associated with abscess formation, but bacteria from colonic course are not often reported [3]. This case is related to *Streptococcus intermedius* as being the cause of a liver abscess secondary to diverticulitis in an elderly gentleman.

#### **Case Presentation**

A 75-year-old Caucasian builder with a known case of ulcerative colitis presented to the Southmead Hospital Emergency Department with a history of pyrexia associated with urinary symptoms. Three weeks prior to presentation, he had developed coryza, rigors and aching shoulders associated with pyrexia. He was seen by his GP who initially treated him for a suspected community-acquired pneumonia with amoxicillin. Seven days prior to admission, he returned to his GP with an ongoing history of intermittent pyrexia. He was treated for a urinary tract infection with a course of trimethoprim for 5 days. Despite this, he continued to deteriorate and developed progressively reduced mobility and muscle weakness. He was again treated with another course of antibiotics by his GP; however, he had deteriorated so much at this point that he was "unable to hold anything down," and it was then when he decided to go to hospital as he started vomiting. On admission, he was short of breath accompanied by wheezing. There was no cough or hemoptysis. There was no change in bowel habit, no history of foreign travel, weight loss, night sweats nor any recent history of unusual food substances. Interestingly, he mentioned that he had had occasional bouts of lower abdominal pain, which was "normal" for him. He had a past medical history of asthma, hypertension, non-ST elevation myocardial infarction, benign prostatic hypertrophy and ulcerative colitis. He lived with his wife in a house with stairs. He experienced one fall recently associated with a temperature spike. He was an ex-smoker (8–10 cigarettes per day for 55 years) and was an active builder. On physical examination, he was pyrexial with a temperature of 37.1°C and normotensive with a blood pressure of 107/62 mm Hg. Pulse was 62 bpm, and respiratory rate was 25 breaths per minute saturating at 99% SpO<sub>2</sub>. Cardiovascular examination revealed normal heart sounds accompanied by a soft systolic murmur. Upper airway wheeze was heard but not on auscultation as the lungs were otherwise clear. Abdomen was soft and non-tender with a small reducible umbilical hernia. Bowel sounds were present and normal. He was grossly moving his 4 limbs with ease. Investigations on admission revealed a white blood cell count of  $14.54 \times 10^{9}$ /L, haemoglobin of 80 g/L, mean corpuscular volume of 79.6 fL, mean corpuscular haemoglobin of 25.6 pg, neutrophils of 12.49 × 10<sup>9</sup>/L, C-reactive protein of 203 mg/L and estimated glomerular filtration rate of 52 mL/min. Interestingly, his liver function tests demonstrated a bilirubin of 11 µmol/L, alkaline phosphatase of 195 U/L, alanine transaminase of 36 U/L and albumin of 26 g/L. His chest X-ray revealed a suspicious 8-mm nodule within the left apex, no focal consolidation and clear pleural spaces with right apical pleural thickening (Fig. 1). Urinalysis was negative and electrocardiogram showed normal sinus rhythm. Venous blood gas was unremarkable. At first, the patient was presumed to have an infection from unknown origin accompanied with anaemia either secondary to iron deficiency anaemia or due to an underlying malignancy. The patient was commenced on empiric intravenous co-trimoxazole and gentamicin. Blood and urine cultures were sent to the laboratory prior to this. In view of the findings of abnormal urinary symptoms and the findings on the chest X-ray, an ultrasound of the urinary tract along with a computed tomography (CT) scan 166



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of the chest, abdomen and pelvis were obtained. An echocardiogram was performed to rule out infective endocarditis.

In the meantime, the patient was transferred to the Geriatric Ward. Despite all efforts, the patient continued to deteriorate with progressively worsening nausea and was no longer able to tolerate his oral medication. He began to develop new symptoms, including right-sided neck pain and intermittent rigors. CT scan of the chest, abdomen and pelvis was performed and showed 3 main findings: a right apical nodule, a small colonic lesion (being suspected malignancy or diverticulitis) and an irregular mass in the liver (Fig. 2a, b). Therefore, the differential diagnosis at the time was a liver abscess and colon carcinoma with metastatic deposits to the lung and liver. There was no evidence of intrahepatic or extrahepatic biliary dilatation. After discussion of these findings with the patient, he then openly admitted that he had a history of tuberculosis in his right lung as a child and was isolated at the age of 5 years for 9 months in Swansea. He recalled being told by medical personnel there that he had developed scarring ever since the treatment in his right lung. This would explain the appearance of the right apical nodule (Fig. 1). In view of the CT scan findings, an ultrasound of the liver was arranged, and if that would reveal a liver abscess, then an ultrasound-guided liver drain would be performed. Intravenous metronidazole was added to the antibiotic therapy regimen, more blood cultures were taken, and a colonoscopy was also arranged. The patient deteriorated so much that he began to lose the ability to mobilise without aid due to his progressive muscular weakness. He now complained of right upper quadrant pain accompanied by pain in his right shoulder with worsening nausea. After discussion with the microbiology team, S. intermedius was isolated as the main pathogen in his blood cultures, which was sensitive to teicoplanin. A liver ultrasound confirmed a large mixed echogenicity lesion at the superior aspect of the liver abutting the diaphragm measuring approximately 6.7 × 6 cm. Therefore, an ultrasound-guided drain was inserted in the cavity. Narrow-spectrum antibiotics, such as intravenous teicoplanin, metronidazole and gentamicin, were started. The patient expressed immediate relief following the drain insertion. Surprisingly, he was then able to tolerate food and was able to walk around the hospital freely for the first time. His colonoscopy revealed evidence of inflamed colon and areas of diverticulosis with no evidence of malignancy (Fig. 3). The diagnosis was most likely a liver abscess secondary to sigmoid diverticulitis caused by S. milleri group bacteria. The second differential diagnosis was an abscess as a complication of a resolving bout of ulcerative colitis in view of this patient's history. Otherwise, his neutrophilia began to improve significantly along with his liver function tests. A repeat CT scan of the abdomen was performed, which demonstrated significant improvement in the size of the liver abscess. The patient was discharged home to complete a 4-week course of oral antibiotics. A subsequent CT scan taken several months after discharge showed resolution of his abscess.

#### Discussion

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Diverticular disease of the colon is common in developed nations. High incidence rates of left-sided diverticulosis are common in the West and increase with age. The incidence increases to 50–66% in patients older than 80 years of age. Up to 25% of patients with acute diverticulitis develop complications. This includes abscess formation, fistulas, strictures/ob-struction and perforation. Sigmoid diverticulitis as the source of a liver abscess is uncommon. *S. intermedius* is the pathological organism which resulted in the development of the liver abscess. This bacterium forms part of the *S. milleri* group that consists of 3 distinct species: *S. anginosus, S. intermedius*, and *S. constellatus*. These are gram-positive, catalase-negative cocci.

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They are non-motile facultative anaerobes that demonstrate variable haemolytic patterns. This group of bacteria is known to be opportunistic in nature. Even though they constitute the normal flora of the oropharynx and genitourinary and gastrointestinal tracts, they have the potential to cause purulent infections, including meningitis, endocarditis and abscesses [4].

Pyogenic liver abscess is often a polymicrobial infection that has ascended from the gastrointestinal tract. *S. milleri* and gram-positive cocci are common causes of pyogenic liver abscesses. Interestingly, a *Klebsiella* liver abscess occurs without evidence of hepatobiliary disease. 90% of patients with the disease have positive cultures, and intestinal organisms, such as *Escherichia coli*, *Klebsiella* and *Proteus*, are isolated. *Candida* should also be considered in the immunocompromised [5]. Pyogenic liver abscess can be caused by an amoebic abscess, hydatid cystic cavities and metastatic and primary hepatic tumours. These are known to be a complication of liver transplantation, hepatic artery embolization when treating for hepatocellular carcinoma, and the ingestion of foreign bodies [5]. The main symptoms and signs of a pyogenic liver abscess include intermittent fever, rigors, enlarged liver with right upper quadrant pain, jaundice, nausea and anorexia. Other unusual symptoms include a cough secondary to diaphragmatic irritation and right shoulder pain due to the pain from the diaphragm. Though our patient did present with signs of an intra-abdominal pathology, it was not clear from the history alone that this was due to a pyogenic liver abscess.

In a study conducted by Mali and Muduganti [6], the most common laboratory abnormalities of a pyogenic liver abscess were hyperbilirubinemia, raised white blood cell count (68% of patients), low albumin level (70% of patients) and raised serum alkaline phosphatase level (67% of patients). Since hepatobiliary disease is the most common cause of liver abscesses, it is expected to have jaundice as a symptom due to the associated cholestasis [7]. However, this was not the case in this scenario, which made diagnosis difficult.

Ultrasound is the preferred modality for diagnosis; however, a CT scan can detect small abscesses (<5 cm) and has higher specificity for multiple small abscesses. When detected early, effective treatment of small abscesses involves the use of intravenous antibiotics alone. The antibiotic therapy should consist of a combination of an aminoglycoside with either metronidazole or clindamycin, or a  $\beta$ -lactam antibiotic with anaerobic coverage. Larger abscesses (>5 cm) prompt drainage for resolution of sepsis, and this can be done by CT-guided or ultrasound-guided percutaneous drainage and surgical drainage.

Percutaneous drainage is mainly indicated for lesions that have a single cavity, are easily accessible for proper localisation of drainage and are without any additional surgical pathology [8]. The indications for surgical drainage include multiple and/or loculated abscesses, abscesses not amenable to percutaneous drainage secondary to location, inclusion of intra-abdominal disease which necessitates surgery and failure of percutaneous drainage [6]. When deciding which drainage technique to use in the first instance, different institutes have their own preferences. Tan et al. [9] showed that surgical drainage mostly produced better resolution of fever and better success but with equally good morbidity and mortality rates as percutaneous drainage. They also demonstrated that surgical drainage has the advantage of allowing for breakdown of loculations and an overall more complete drainage of the collection.

Our patient had a history of ulcerative colitis, but this did not seem to be a true recurrence. Our initial differential diagnosis at the time was metastatic colon carcinoma. Reassuringly, the colonic lesion shown on the CT scan was investigated further with colonoscopy and was revealed to be inflammation of the sigmoid colonic mucosa (Fig. 2, 3). This was the only possible site of a primary infection focus found on investigation. There was no evidence of malignancy from the colonoscopy. Even though portal bacteraemia does occur in those affected with ulcerative colitis, the incidence of portal vein bacteraemia remains unknown. Manousos et al.

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[10] and Brooke et al. [11] sampled blood cultures via the portal vein during laparotomy and found that portal bacteraemia occurs in approximately 25% of patients with active ulcerative colitis [12]. The patient had bacteraemia with *S. intermedius*. The primary source of the infection was most likely a result of sigmoid diverticulitis. However, it is also possible that this may have been secondary to a resolving bout of ulcerative colitis. The patient recovered remarkably well after having a percutaneous liver drain and appropriate antibiotics and had a complete resolution of his infection on follow-up.

In conclusion, it is advocated that the presence of *S. milleri* group bacteraemia should alert the clinician to the possibility of an underlying abscess as a source of infection [13]. Although portal bacteraemia is a known complication of ulcerative colitis, the development of liver abscesses is rare. Elderly patients may develop a presentation uncharacteristic of that of the average adult with a pyogenic liver abscess.

#### Acknowledgement

We would like to thank the nursing staff on the Geriatric Ward for the great care that this gentleman had received.

#### **Statement of Ethics**

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

#### **Disclosure Statement**

The authors have no conflict of interest.

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#### **Author Contributions**

E.P.J. Muscat has written this case report and the discussion.

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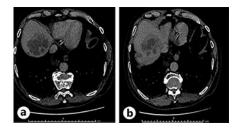
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Fig. 1. Chest X-ray showing left apical nodule and raised right hemidiaphragm.



**Fig. 2.** CT of the abdomen demonstrating the hepatic abscess. A characteristic "double-target sign" appearance of a hepatic abscess.



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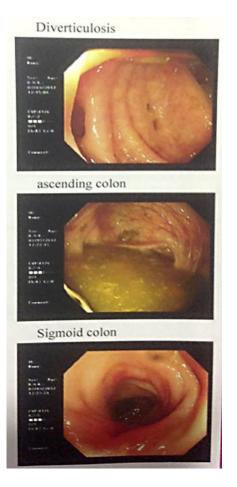


Fig. 3. Images taken from colonoscopy revealing inflammation of the sigmoid colon and diverticulosis.

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