CASE REPORT

Hepatic abscess caused by *Fusobacterium necrophorum* after a trip to the dentist

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SUMMARY

A 57-year-old man with no significant medical history was admitted to the hospital, with high-grade fever and right upper quadrant pain. He was found, on abdominal ultrasound, to have a right lobe hepatic cystic lesion. MRI of the abdomen confirmed a hepatic abscess. Cultures obtained under CT guidance showed the abscess to be caused by *Fusobacterium necrophorum*. This is a rare bacterium that can cause potentially fatal liver abscesses. Following drainage and intravenous antibiotic treatment, the patient improved and was discharged on a 4-week antibiotic course. An abdominal CT, performed 6 weeks after discharge, showed total resolution of the abscess. The patient had, 2 weeks prior to the development of the liver abscess, undergone routine dental cleaning. Neither upper respiratory symptoms nor sore throat had been identified prior to the presentation.

BACKGROUND

Liver abscesses are rare in the general population. They may be caused by different pathogens.

Amoebic liver abscesses are the most common form of liver abscess worldwide, whereas bacterial pyogenic liver abscesses are the most common form in North America,¹ and are usually the result of a polymicrobial infection.² Pyogenic liver abscesses are a rare condition in an immunocompetent host.

Pyogenic liver abscesses are potentially life-threatening, with an estimated incidence rate of 2.3 cases per 100 000 patients.^{3–4} Despite recent advances in the investigation and management of such abscesses, the mortality rate still ranges from 2% to 12%.⁵

Fusobacterium necrophorum is an anaerobic Gram-negative bacillus, part of the family Bacteroidaceae, and predominantly found as part of the normal oral flora.

As most pyogenic liver abscesses are polymicrobial, only a handful of cases of *F. necrophorum* hepatic abscesses have been described,⁶ and specific incidence of this pathogen in liver abscesses is unknown.

F. necrophorum is a recognised bacterial cause of liver abscesses as a complication of Lemierre's syndrome. This syndrome usually begins with pharyngitis that improves over the first 4–5 days,⁷ followed by a recurrence of symptoms with addition of bacteraemic manifestations including rigours, fever and night sweats. Patients also develop suppurative internal jugular thrombophlebitis and metastatic infections, especially in the liver, lungs and brain. Patients with Lemierre's

syndrome have an estimated mortality of 5%, with significant short-term and long-term morbidity.^{8–9}

The case presented here is an unusual case of a liver abscess caused by *F. necrophorum* not related to Lemierre's syndrome in an immunocompetent adult. The only possible aetiology of the infection is the oral cavity since the patient had undergone a routine dental cleaning prior to his presentation. Since the patient had good oral health, with neither gingival nor oral infections, and as no invasive dental work had been performed at that visit, this case represents an interest as it suggests that even routine dental cleaning may cause bacteraemia, which can seed in different areas of the body.

CASE PRESENTATION

A 57-year-old Caucasian man, with no significant medical history, presented to the hospital owing to fever and right upper quadrant (RUQ) discomfort that had been ongoing for 10 days. He had been seen in the emergency department (ED) a few days earlier for non-specific abdominal pain; at the time, no fever was noted, and routine blood work had shown no severe abnormality (no leucocytosis and no bandaemia). He was discharged without a definitive diagnosis and followed up with a primary care physician, by which time the pain had increased and the patient began 'feeling warm'; no temperature was measured. Abdominal ultrasound showed a hepatic lesion—a complex cyst versus abscess—on the right lobe (figure 1). He was then instructed to come to the hospital for further work up and treatment.

Aside from the presenting symptoms of fever and RUQ discomfort, the patient's review of systems was negative. The patient denied any recent upper respiratory infection or sore throat. He denied alcohol, tobacco and illicit drug use. Previous to this incident, he had not seen a physician for several years. He had never had a colonoscopy. He had been seen by a dentist 2 weeks earlier for routine dental hygiene. His oral health was found to be good—he was not aware of having any gingival or other oral infections; no invasive dental procedures had been performed at the visit, only a normal dental cleaning and examination.

On initial examination, the patient was tachycardic (112 bpm), normotensive and febrile (38.3°C). His abdominal examination revealed decreased bowel sounds in all four quadrants; he had minimal tenderness in the right upper quadrant, Murphy's sign was negative and no guarding or rebound was noted. He had no dental disease and no signs of oropharyngeal infection. The remainder of his physical examination was normal.

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Figure 1 Ultrasound of the liver.

Initial laboratory data showed significant leucocytosis of $22.8 \times 10^9/L$ with bandaemia of 14%, elevated erythrocyte sedimentation rate (ESR) of 79 mm/h and abnormal liver function tests with normal aspartate transaminase (AST) of 31 U/L, elevated alanine transaminase (ALT) of 61 U/L and elevated alkaline phosphatase (ALP) of 412 U/L. HIV test was negative. *Echinococcus* and *Entamoeba histolytica* titres were negative.

The patient was started on broad-spectrum intravenous antibiotics after blood cultures were drawn. An MRI of the abdomen with and without contrast combined with a MR cholangiopancreatography (MRCP) were performed. The MRI was chosen over the more frequently used CT, because close evaluation of the biliary tree with an MRCP was desired to investigate possible sources of infection, and it was decided to perform both tests in combination. The MRI showed a multiloculated collection with a thick rind of peripheral enhancement, compatible with hepatic abscess (figures 2–4). The MRCP was unremarkable.

Abscess drainage was performed percutaneously with CT guidance (figures 5 and 6) on the second day of hospitalisation. Thirty-five millilitres of thick purulent fluid was aspirated and Gram stain and cultures were obtained.

On the third day, the patient was feeling better but still had a low-grade fever. His white cell count was improving. The fluid culture obtained via percutaneous drainage grew *E. necrophorum*, β -lactamase positive, and resistant to amoxicillin/ampicillin.

A colonoscopy, performed as part of the work up for a liver abscess (as there might have been a relation with colonic neoplasm⁶), was normal.

Figure 2 MRI of the abdomen—T2 image.

Figure 3 MRI of the abdomen.

A repeat CT scan of the abdomen on the fifth day of hospitalisation showed almost complete resolution of the abscess (figure 7). The patient was subsequently discharged on the seventh day of hospitalisation after being afebrile for 24 h, to complete a 4-week course of intravenous antibiotic therapy as an outpatient.

INVESTIGATIONS

Laboratory studies

On initial presentation to the ED, the patient had undergone a complete blood count (CBC) with differential, which showed neither leucocytosis nor bandaemia (leucocytes of $8 \times 10^9/L$ with no bands); complete metabolic profile (CMP) was normal and no elevation of liver enzymes was present (AST of 27 U/L, ALT of 35 U/L). Presumably, the tests were not elevated because the patient had presented to the ED early, at the onset of his symptoms.

On admission to the hospital, however, the CBC was significant for leucocytosis of $22.8 \times 10^9/L$ with bandaemia of 14%. The CMP showed a normal renal function and electrolytes and an abnormal liver function with normal AST of 31 U/L, elevated ALT of 61 U/L and elevated ALP 412 U/L. Urine analysis was performed as part of the infectious work up in the patient, and was within normal limits. Blood culture remained negative. An ESR was obtained and was elevated at 79 mm/h. *Echinococcus* and *Entamoeba histolytica* titres were drawn to assess for

Figure 4 MRI of the abdomen—T1 image.

Figure 5 Pre-drainage CT of the abdomen.

possible aetiologies of the abscess—they were negative. The immune status of the patient was assessed with an HIV test, which was negative. The aspirated fluid was sent for a Gram stain, which revealed only a few polymorphonuclear leucocytes; and aerobic and anaerobic cultures showed a moderate amount of *E. necrophorum*, β -lactamase positive and amoxicillin/ampicillin resistant.

Radiological studies

Abdominal ultrasound as an initial evaluation of RUQ pain was obtained and showed a hypoechoic structure in the right lobe of the liver, likely a complex cyst, but an abscess, although less likely, could not be completely excluded. A CT of the abdomen is an imaging modality that is frequently used to evaluate liver lesions, but in our case, an abdominal MRI with contrast was performed for further detailing of the findings; this study revealed a 5.6 cm multiloculated abscess involving the hepatic segment VI. MRCP is another imaging modality that may be used for evaluation of a liver lesion and its possible aetiologies. The MRI was chosen over the CT because it was decided that a concomitant MRCP was going to be performed to look for possible sources of the suspected infectious process. An abdominal

Figure 6 CT-guided drainage.

Figure 7 Post-drainage CT of the abdomen.

CT was performed post drainage of the abscess for follow-up and showed a complete resolution of the collection.

Echocardiogram to rule out valvular structural abnormalities as a possible seeding source of the liver abscess was performed and was unremarkable.

Invasive diagnostic procedures

Percutaneous CT-guided abscess drainage was performed, and 35 mL of purulent fluid obtained.

A colonoscopy to rule out colon neoplasm, which can correlate with liver abscess,¹⁰ was performed and did not show any significant lesions.

TREATMENT

Intravenous antibiotic therapy was initiated on admission (after blood culture was obtained) with metronidazole and ceftriaxone, predominantly aimed at covering Gram-negative and anaerobic bacteria.

Later, the antibiotic therapy was changed to metronidazole and piperacillin/tazobactam. This decision was made after the initial results of the culture suggested anaerobic bacteria, to provide double coverage for this type of flora.

The final result of the fluid culture showed *E. necrophorum*, β -lactamase producing and resistant to amoxicillin/ampicillin.

CT-guided abscess drainage was performed with 35 mL of thick purulent liquid obtained and sent for culture.

Acetaminophen and probiotics were administered as well.

OUTCOME AND FOLLOW-UP

Metronidazole was discontinued on discharge after finishing a 7-day course of this antibiotic. The patient was discharged on piperacillin/tazobactam to complete 4 weeks of treatment.

The patient was followed in the outpatient setting with a CT scan of the abdomen 6 weeks after discharge from the hospital; it showed complete resolution of the liver abscess.

DISCUSSION

A liver abscess is the most common type of abscess in the visceral organs. Mortality rate is between 2% and 12%.¹¹ It is usually a complication of peritonitis via portal circulation or direct spread from a biliary infection. Liver abscesses may also happen in the setting of systemic infection via haematogenous spread.

Risk factors include underlying colorectal neoplasia, hepatobiliary, pancreatic disease, liver transplant, immunosuppression and diabetes mellitus. Geographic factors may also play a role; they are more common in East Asia, and typically caused by *Klebsiella pneumoniae* and in association with colorectal neoplasia; in India, Africa, Mexico and South America, they are caused by *Entamoeba histolytica*. Gender is another risk factor since liver abscesses are more common among males (3:1).⁴

Most pyogenic liver abscesses are polymicrobial; enteric facultative and anaerobic species are the most common pathogens. Potential pathogens include:

- ▶ *Streptococcus anginosus*
- ▶ *Staphylococcus aureus*
- ▶ *Candida* spp
- ▶ *K. pneumoniae*
- ▶ *E. histolytica*
- ▶ *Burkholderia pseudomallei*
- ▶ *F. necrophorum*

In the present case, the only risk factor the patient had was his sex. He did not have any immunodeficiencies, neoplasias or endocarditis, nor had he travelled. The only possible source for the infection was his oral cavity and the fact that he had had a dental cleaning performed.

The patient did not have problems with his oral health (no history of gingivitis and no infection with *F. necrophorum* in the past) and the dental visit was a routine cleaning. No special procedures were performed during the visit and no treatments preceded or succeeded the appointment. The cleaning might have involved some trauma to his oral mucosa that served as an entryway to the blood stream for his normal oral flora, in this case *F. necrophorum*.

Fusobacterium are Gram-negative anaerobes first recognised for their causative role in Lemierre's syndrome.¹² The pathogenesis of the liver infection remains unknown; however, the proposed mechanisms include haematogenous spread from dental caries, peritonsillar abscess¹³ or spread through the portal circulation in the setting of diverticular disease.¹⁴

The most common clinical presentation of a liver abscess is fever, abdominal pain, nausea, vomiting, anorexia and malaise.

Diagnosis is made by clinical history, physical examination and imaging studies followed by aspiration, drainage and culture of the fluid. It is crucial to rule out peritonitis, throat infection and dental cavities as possible sources of the liver abscess, through detailed history-taking and physical examination. Ultrasound, CT of the abdomen and MRI of the abdomen are the studies of choice for confirmation of the diagnosis. Aspiration and drainage are usually performed under CT guidance. A colonoscopy must be carried out to exclude colorectal neoplasia and diverticular disease. Echocardiography is also performed to check for possible structural heart-valve abnormalities and endocarditis as possible infection seeding sources for the liver abscess.

Blood tests should include: blood cultures, liver enzymes, CBC, ESR, HIV testing, and *E. histolytica* and *Echinococcus* titres.

Abscess drainage and antibiotic therapy are the treatment of choice. Drainage can be performed under percutaneous CT or ultrasound guidance, open surgical drainage or by endoscopic retrograde cholangiopancreatography.

For a single abscess with a diameter under 5 cm, either percutaneous catheter or needle aspiration are acceptable—the drainage catheter should remain in place for at least 7 days or until drainage is minimal. For a single abscess over 5 cm in diameter, catheter drainage is preferred over needle aspiration, and some

even prefer surgical intervention over percutaneous drainage.⁶ Surgical drainage is also appropriate for multiple abscesses, loculated abscesses, abscesses with viscous content that can block the drain tube and underlying diseases that require surgical management.⁶

Empiric intravenous antibiotic therapy with metronidazole plus ceftriaxone is first line; acceptable alternatives are ampicillin/sulbactam or piperacillin/tazobactam. Antibiotic therapy should be tailored when the aspirated/drained material culture and susceptibility are available to meet the specific needs of each patient.

Most *Fusobacterium* isolates are sensitive to ampicillin; however, 2% of isolates are resistant. Most isolates are sensitive to metronidazole.¹³ Antibiotic therapy should be continued for 4–6 weeks. Patients with good drainage of their abscess should receive 2–4 weeks of parenteral antibiotics, while incomplete drainage merits 4–6 weeks of parenteral therapy. The remainder of the course may be finished with oral medication selected based on sensitivity results,¹⁰ when appropriate. It may be prudent to repeat a CT scan after antibiotic therapy is completed to confirm complete resolution of the liver abscess.

Learning points

- ▶ Liver abscesses caused by *Fusobacterium necrophorum* are a rare condition.
- ▶ *F. necrophorum* is an anaerobic Gram-negative bacillus that belongs to the Bacteroidaceae family and is part of the oral bacterial flora.
- ▶ *F. necrophorum* is a well-known cause of Lemierre's syndrome. This syndrome is characterised by an acute primary infection of the oropharynx with secondary thrombophlebitis of the internal jugular vein, septicaemia and septic emboli frequently seeding to the liver, lungs and brain.
- ▶ This case suggests that liver abscesses caused by *F. necrophorum* are not always a consequence of Lemierre's syndrome and may occur in an immunocompetent host.
- ▶ When a liver abscess presents without an obvious source of infection, it is necessary to rule out:
 - Oropharyngeal infections (Lemierre's syndrome) as a seeding source;
 - Colonic neoplasm (via colonoscopy), as there is a relationship between colon cancer and liver abscesses;¹⁰
 - Infective endocarditis (via echocardiography) as a potential seeding source.
- ▶ A liver abscess caused by *F. necrophorum* may happen after a routine dental cleaning procedure, as suggested by this case.

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