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Case Report

Campylobacter jejuni, an uncommon cause of splenic abscess diagnosed by 16S rRNA gene sequencing

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SUMMARY

Splenic abscess is a rare disease that primarily occurs in patients with splenic trauma, endocarditis, sickle cell anemia, or other diseases that compromise the immune system. This report describes a culture-negative splenic abscess in an immunocompetent patient caused by *Campylobacter jejuni*, as determined by 16S rRNA gene sequencing.

1. Introduction

Splenic abscess is rare that may be misdiagnosed because of the existence of misleading or forms with antibiotics decapitated.[1,2] We reported here described the first case of a spontaneous splenic abscess caused by *Campylobacter jejuni* as determined by 16S rRNA gene sequencing in an immunocompetent patient.

2. Case report

In May 2013, a 20-year-old Caucasian man was admitted to the infectious disease department of the university hospital in Marseille for severe upper left quadrant pain that had appeared 4 days after febrile diarrhea. Prior to the diarrhea, he had been well, with nothing notable in his medical history. He had not had a splenic trauma or contact with animals, and he had not traveled to a tropical area. His body temperature was 39 °C, his pulse was 113 beats/min, and his blood pressure was 100/59 mmHg. Laboratory investigations revealed pathological values for C-reactive protein (266 mg/l; normal values ≤5 mg/l) and fibrinogen levels (7.7 g/l; normal values 1.8–4 g/l), an elevated leukocyte count (18×10^9/l, predominantly neutrophil granulocytes), a low hemoglobin concentration (116 g/l; normal 135–175 g/l), and a normal platelet count (339×10^9/l). Results of serum electrophoresis were normal. A computed tomography (CT) scan of the abdomen revealed an 11-cm splenic abscess with peripheral calcifications and a peri-splenic collection associated with a left pleural effusion (Figure 1a). ^{18}F-fluorodeoxyglucose positron emission tomography combined with computed tomography (PET/CT) showed a unilocular splenic collection with hypometabolic activity (Figure 1b). Repeat blood cultures were negative. Stool cultures to assay for *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia* species were negative. Indirect hemagglutination (IHA) and ELISA tests for *Entamoeba histolytica* were negative. Transthoracic echocardiography was negative for infective endocarditis and valve abnormalities.

Empirical antibiotic treatment with ceftriaxone and metronidazole was started. Nevertheless, a fever (up to 40 °C) persisted after 1 week of treatment, at which time the splenic abscess was percutaneously drained and 1200 ml of purulent fluid was aspirated. After drainage of the splenic abscess, the patient had definitive apyrexia. Bacterial cultures of the deep sample were

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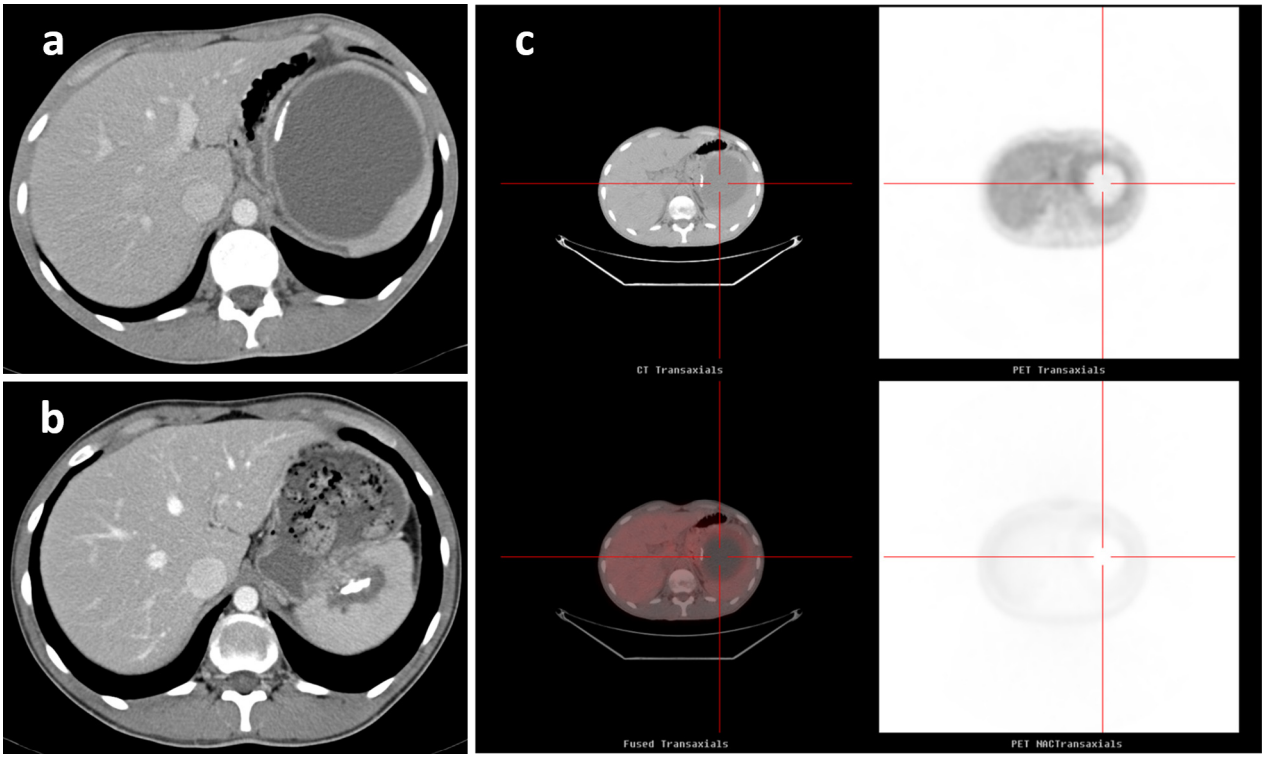

Figure 1. (a) CT scan of the abdomen showing an 11-cm unilocular splenic abscess. (b) FDG-PET scan showing a unilocular splenic collection with hypometabolic activity. (c) CT scan of the abdomen showing calcification sequelae in the spleen at 12 months after the end of treatment.

negative. Histology performed on the liquid confirmed the presence of a pyogenic splenic abscess, and 16S rRNA PCR was positive for *Campylobacter jejuni*. The patient was discharged on day 15 of hospitalization.

Post-discharge, the patient was given a 6-week treatment of ceftriaxone. During antibiotic treatment and 1 month after percutaneous drainage, the splenic collection relapsed without any clinical symptoms. This was drained under endo-ultrasonography in the stomach via two double-pigtail stents. A CT scan 9 months after intragastric drainage revealed a 5-cm splenic collection with calcification sequelae but no peri-splenic collection or pleural effusion (Figure 1c). The two double-pigtail catheters were removed at 10 months, and no relapses were observed at the 1-year post-antibiotic follow-up.

3. Discussion

In this report, we describe the first case of a spontaneous splenic abscess caused by *C. jejuni* in an immunocompetent patient. Pyogenic splenic abscess is a disease that is rarely reported.[1] The predisposing factors that are frequently recorded are splenic trauma, endocarditis, intravenous drug use, sickle cell disease, diabetes mellitus, and congenital or acquired immunodeficiency.[1] In this case, the splenic abscess was diagnosed by the presence of fever and pain in the upper left quadrant of the immunocompetent patient after 4 days of diarrhea and fever, without a splenic trauma or metastatic infection arising from endocarditis or a primary abscess.

A splenic abscess may be misdiagnosed because of non-specific clinical signs. The typical symptoms of fever, upper left quadrant pain, splenomegaly, and left pleural effusion are found in only 84–95% of cases, 39–50% of cases, 30–67% of cases, and 19–41% of cases, respectively.[1] Nonetheless, modern imaging techniques, such as abdominal CT scans[2] and PET/CT scans, have enhanced the diagnostic process. In our case, CT and PET/CT scans confirmed the diagnosis of splenic abscess and allowed us to exclude other localizations of *C. jejuni* infection.

Most splenic abscesses are due to a single organism,[2] and the main causal microorganisms identified in splenic abscesses are *Streptococcus spp*, *Staphylococcus spp*, *Salmonella spp*, *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas spp*, *Mycobacterium spp*, and some anaerobic bacteria.[1,2] In our case, blood and stool cultures prior to antibiotic treatment were negative for *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia* species. The diagnosis of *C. jejuni* infection was obtained by 16S rRNA gene PCR amplification and sequencing of culture-negative splenic abscess drainage fluid obtained 1 week after antibiotic treatment.

The most common human infections caused by *C. jejuni* are gastrointestinal infections, which are typically characterized by diarrhea, fever, and abdominal pain.[3] Extra-digestive infections are quite rare; cases of Guillain–Barré syndrome, acute cholecystitis, meningitis, pneumonia, urinary tract infection, thoracic wall abscess, arthritis, endocarditis, and transient bacteremia have occasionally been reported.[3,4] To our knowledge, the case reported here is the first of a splenic abscess caused by *C. jejuni* in an immunocompetent patient.

There is no gold standard for treating splenic abscesses. A splenectomy has long been considered to be the principal treatment for a splenic abscess.[1] However, for young patients, percutaneous drainage of a splenic abscess followed by antibiotic treatment is an alternative to splenectomy.[2] Antibiotic treatment alone without drainage of the pyogenic splenic abscess has been proposed by some authors.[1] Intragastric drainage of a splenic abscess with two double-pigtail catheters has been used in cases of splenic abscess complicated by pancreatitis[5] or gastrosplenic fistula.[2]

In this case, we performed percutaneous drainage and treated the patient with broad-spectrum antibiotics for 6 weeks. The splenic abscess was drained again 1 month later using an intragastric drainage procedure involving two double-pigtail stents.

In summary, our case highlights an uncommon splenic abscess caused by *C. jejuni* that appeared in a young patient who did not have an immune deficiency or splenic trauma. The infection was managed successfully with percutaneous and intragastric drainage combined with a prolonged course of antibiotic therapy.

Ethical approval: This study was approved by the institutional research ethics board and written informed consent was obtained from the patient.

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