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## Case Report

# Clostridioides Difficile beyond the Colon: A Case Report - @

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## ABSTRACT

Extra-colonic *Clostridioides difficile* infections are rarely reported. Presentations include bacteremia, enteritis, reactive arthritis, skin infections, or visceral abscesses. We present a case of monomicrobial *Clostridioides difficile* splenic abscess in a patient without colitis, preceding antibiotics, or bacteremia.

**Keywords:** Splenic abscess; *Clostridioides difficile*; Monomicrobial gut infection

## INTRODUCTION

*Clostridioides Difficile* (CD) is an anaerobic, spore-forming, gram-positive bacillus. It is a well-known colonizer of the human colon and is transmitted via the fecal-oral route. *Clostridioides Difficile Infection* (CDI) occurs following disruption of the host's gastrointestinal microbiota [1]. Risk factors are antibiotic therapy in previous 90 days, elderly, extended hospitalization, inflammatory bowel disease, cirrhosis, and chemotherapy [2]. Since CD is difficult to isolate in culture, the gold-standard diagnostic studies are limited to stool studies: Enzyme Immunoassays (EIA) for Glutamate Dehydrogenase (GDH) or toxin antigens and PCR for the toxin gene. Extra-colonic manifestations of CDI are sparsely documented in the literature. We present a case of an extra-colonic CDI presenting as a monomicrobial splenic abscess.

## CASE REPORT

A 54 year old male with history of cirrhosis due to chronic hepatitis B and C was seen in clinic for left upper quadrant pain, feeling febrile, and anorexia. Five weeks prior to presentation, a Transjugular Intrahepatic Portosystemic Shunt (TIPS) revision with splenic artery embolization was performed. He denied associated diarrhea, melena, or hematochezia. His temperature was 98.3 F with a pulse of 79 bpm, respiration rate of 20 breaths/min, and blood pressure of 125/63 mmHg. The physical exam revealed tenderness and guarding in the left upper quadrant along with pain exacerbated by inspiration. Comprehensive metabolic panel, lipase, and complete blood count were unremarkable. Ultrasound with doppler of liver revealed a patent TIPS. A CT of the abdomen was ordered which revealed a subcapsular splenic abscess which measured 17.6 x 15.7 x 11 cm (figure 1). He was admitted to the hospital for further management. Vancomycin and Piperacillin/Tazobactam were empirically administered. Laboratory studies were unremarkable. Blood cultures were negative. CT-guided aspiration with pigtail drain

placement yielded 900 mL of purulent drainage. Gram stain revealed white blood cells but no microorganisms. Six days later, *Clostridioides difficile* was isolated from the culture. He was discharged on oral metronidazole with planned repeat imaging and follow-up with infectious disease. The final CT scan six months after drainage and metronidazole therapy showed a residual collection measuring 4.4 cm x 1.8 cm. The patient stopped following-up, repeat imaging could not be done to document resolution of the abscess.

## DISCUSSION

CDI is a disease associated with the colon presenting as diarrhea, pseudomembranous colitis, ileus, or toxic megacolon. Extra-colonic CDI is a phenomenon rarely reported in the literature and usually occurring in the setting of colorectal surgery or colitis [3]. The few documented cases of extra-colonic CDI include: bacteremia [4], enteritis [3,5], reactive arthritis[5], polymicrobial visceral abscesses (splenic abscess, pancreatic abscess, empyema) [5], prosthetic device infections [5], and osteomyelitis [5]. This case involved a patient with cirrhosis, TIPS revision, and recent splenic artery embolization who developed a splenic abscess from which culture grew only CD. Upon chart review, the patient was not given any prophylactic antibiotics prior to TIPS revision. Notably, preceding colonic disease was absent. Over the last 40 years, only 6 cases of CD splenic abscesses have been reported. The first report of splenic abscess due to CD occurred in 1983. The patient had a history of liver cirrhosis and pancreatitis, in the absence of any gastrointestinal colitis or surgery [6]. The second case was seen in 1987 in a patient that had bacteremia with four *Clostridioides species* (*C. difficile*, *C. fallax*, *C. perfringens* and *C. ramosum*) 4 months prior to splenic abscess development. Cultures of the abscess contained *C. difficile* and *Pseudomonas paucimobilis j* [7]. A third case was seen in 1995 in a patient with atrial fibrillation and congestive heart failure that developed infarction of the colon and spleen. Subsequently, a splenic abscess infected with CD was formed [8]. In 1997, a patient that was given antibiotics for pneumonia following laminectomy surgery developed CD colitis. A few weeks later, a splenic abscess was found. The abscess culture grew CD and coagulase negative staphylococci [9]. Another patient with a history of adenocarcinoma of the cecum, right hemi-colectomy, small bowel infarction, recent *Pseudomonas aeruginosa* septicemia, treated with multiple antibiotics, presented with CD bacteremia. Upon further evaluation, a splenic abscess growing CD was found [10]. The last case reported occurred in 2003 in a patient with end-stage renal disease who had CD bacteremia and later developed a CD splenic and iliacus muscle abscesses [11]. These cases demonstrated shared preceding events: bacteremia, antibiotic exposure, pancreatitis, colitis, bowel and spleen infarction. Theoretically, these events may cause disruption of the bowel microbiota and mucosa. CD is then able to invade and translocate into the bloodstream then disseminate throughout the body. Splenic abscesses have been reported to follow splenic artery embolization procedures at an incidence of 6.8% [12]. With the absence of preceding colitis, pancreatitis, or antibiotic



**Figure 1:** CT scan of the splenic abscess upon diagnosis measuring 17.6 x 15.7 x 11 cm

exposure, the embolization procedure during which colonic translocation of bacteria must have occurred, remains the most likely cause of this patient's abscess. CD obtained its name by way of being difficult to isolate in culture. Traditionally, the diagnosis was made during endoscopy upon viewing pseudomembranes. The current gold-standard diagnostic studies involve EIA and PCR of stool samples. The lack of a reliable method to diagnose extra-colonic CDI implies that its incidence is underreported. Future areas of research should be dedicated to developing diagnostic tools to detect CD in blood or other body fluids.

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