



Yersinia enterocolitica Bacteremia and Multiple Liver Abscesses Disclose a Colonic Tubular Adenoma

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Abstract

We present a case of *Yersinia enterocolitica* bacteremia and sepsis with multiple liver abscesses mimicking metastatic liver disease. The patient was treated successfully with conservative therapies. Interestingly, colonic tubular adenoma was discovered during the diagnostic evaluation, and consequently we explore the possibility of an association between *Yersinia* bacteremia and colorectal neoplasia.

Introduction

Yersinia species are gram-negative coccobacilli, which are facultative anaerobes [1].

Three species of *Yersinia* produce human illness: *Yersinia pestis* (the causative agent of human plague), *Yersinia enterocolitica* (the causative agent of yersiniosis), and *Yersinia pseudotuberculosis*. The most common manifestation of yersiniosis is acute abdominal disease. Bacteremia and metastatic abscesses are rare [2]. These complications usually carry a poor prognosis.

Case Report

A 68-year-old male patient presented to the hospital with a three-day history of diarrhea. The stool was watery, three times per day, without any blood or mucus. The diarrhea was not accompanied by any abdominal pain, nausea, or vomiting. However, the patient had been complaining of lack of energy and generalized weakness. He denied any recent travel or exposure to animals (birds, sheep, cattle, goat, pigs, or livestock). He denied eating raw pork meat or using unpasteurized milk. He had no recent outdoor camping or hunting exposure. He does drink alcohol and does not use recreational drugs.

On physical examination, he had a temperature of 103.2°F, heart rate of 96/min, respiratory rate of 21/min, and blood pressure 130/73 mmHg. His quick Sequential Organ Failure Assessment score (qSOFA) was zero. Abdominal exam revealed a minimal tenderness in the right upper quadrant, but no distinct mass was felt. The rest of physical exam was normal.

Laboratory investigations revealed white blood count of $7.7 \times 10^3/\mu\text{L}$, hemoglobin of 11.3 g/dL, MCV of 85, platelets of $571 \times 10^3/\mu\text{L}$, creatinine 1.1 mg/dL, BUN 14 mg/dL, glucose 89 mg/dL. Liver function tests showed ALT 190 U/L, AST 147 U/L, total bilirubin 1.3 mg/dL, direct bilirubin 0.7 mg/dL, alkaline phosphatase 172 U/L, and albumin 2.4 g/dL.

An abdominal ultrasound demonstrated multiple hypoechoic lesions in the liver, likely representing metastatic disease (Figure 1). The portal vein was patent with appropriate directional flow. There was no biliary ductal dilatation and no splenomegaly. Computerized tomography (CT) scan of the chest and abdomen with contrast showed right infra-hilar airspace opacities and nodules measuring up to 1.6 cm. Along the right anterolateral mid lung field, there was an enhancing pleural mass measuring approximately 3.5 cm. Mediastinal and hilar lymphadenopathy is seen up to 2.6 cm in the subcarinal space. Furthermore, numerous low density lesions scattered throughout the hepatic parenchyma were seen (Figure 2). Scattered, subcentimeter lymph nodes were seen within the periaortic regions. Osseous structures were intact, with no lytic, or blastic-appearing lesions were appreciated. The patient underwent liver biopsy under CT-guidance using 17-gauge needle from the right lobe. The largest hepatic lesion was aspirated, and three millimeters of purulent fluid were obtained. Histological examination of the hepatic biopsy showed histomorphologic findings consistent with liver abscess. The adjacent liver tissue showed mild steatosis. There was no evidence of malignancy or hemochromatosis. Periodic acid-Schiff (PAS) stain did

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Figure 1: Abdominal ultrasound showed multiple hypoechoic lesions, likely representing metastatic disease. Patent portal vein with appropriate directional flow. No biliary ductal dilatation. No splenomegaly.



Figure 2: CT scan of the abdomen with IV contrast showed the presence of numerous low density lesions scattered throughout the hepatic parenchyma. The spleen, pancreas, kidneys, and adrenal glands are unremarkable.

not show diagnostic findings suggestive of Ameba organisms. Blood and aspiration specimen cultures yielded *Y. enterocolitica*. The patient was commenced on combination of piperacillin/tazobactam and tobramycin, to which the patient had a great response with normalization of abnormal laboratory results. The antibiogram showed that the strain was susceptible to all classes of antibiotics, including fluoroquinolone, aminoglycoside and penicillin. Subsequently, antimicrobial therapy was changed to ceftriaxone 2 g IV daily in combination with gentamicin 5 mg/kg IV daily. Repeat blood cultures showed no growth. The patient had bronchoscopy with bronchoalveolar lavage and ultrasound-guided needle biopsy of sub-carinal mediastinal lymph node. Pathology showed no evidence of malignancy and bacterial, acid-fast bacilli, and fungal cultures were negative. The patient was discharged home and further treated with ciprofloxacin 500 mg PO twice daily for additional 6 weeks as an outpatient. A repeat abdominal CT scan after finishing antibiotics showed complete resolution of the hepatic abscesses. Colonoscopy revealed a single pedunculated polyp in the descending colon and two small flat polyps which were removed by polypectomy. Pathology was consistent with tubular adenoma. Comprehensive workup excluded hemochromatosis, G6PD deficiency, thalassemia, sickle disease, and other chronic hemolytic conditions, as well as human immunodeficiency virus and other primary immunodeficiency, as these conditions carry higher index of suspicion for *Yersinia* infection.

Echinococcus, *Strongyloides*, and fungal serologies were negative. In addition, our patient had no chronic sequela of the infection such as reactive arthritis, myocarditis, glomerulonephritis, or liver failure.

Discussion

The number of cases with *Y. enterocolitica* has gradually risen over the years, and the bacterium is now recognized as a major enteric pathogen world-wide [3,4]. Conversely, mortality rates have also dramatically fallen, most likely due to the development of more effective antibiotics [3,4]. Clinical manifestations of yersiniosis include enteric disease, such as enterocolitis, mesenteric adenitis, appendicitis, and terminal ileitis [2]. Post-infectious manifestations are erythema nodosum, reactive polyarthritis, and Reiter's syndrome [4,5]. *Y. enterocolitica* invasive diseases such as bacteremia or hepato-splenic abscesses are extremely uncommon and mainly occur in patients with underlying conditions (diabetes mellitus, malignancy, immunosuppressive therapy, cirrhosis of the liver), especially those associated with iron overload [4,5]. This bacterium cannot thrive in low iron environments, as it cannot produce its own siderophores to bind iron [6]. Predisposing conditions are hemochromatosis, hemosiderosis, thalassemia major, acute iron poisoning, chronic hemodialysis, or long-term transfusion therapy [5]. Additionally, although deferoxamine is effective in reducing body iron load, it increases susceptibility to *Yersinia* infection in patients with hemosiderosis because the bacteria can utilize the siderophores from the deferoxamine [6].

Iron studies and liver biopsy results excluded the possibility of hemochromatosis in our patient. It is unknown if *Yersinia* species have any association with colorectal neoplasia. We believe that *Yersinia* bacteremia requires investigation for concomitant colorectal tumors. It is extremely important to know in the future if *Y. enterocolitica* could be a consequence of a gastrointestinal lesion or of triggering or promoting etiological nature. There are only approximately 50 cases of *Y. enterocolitica* liver abscesses being well documented to date, with the majority of cases happened outside the United States [3,5,7]. *Y. enterocolitica* is primarily transmitted to humans via contact with animal carriers, consumption of undercooked meat, especially pork, contaminated milk, and contaminated water [6,7]. The pathway for the spread of infection resulting in liver abscesses has been recognized as beginning in the alimentary tract and spreading through the portal vein [4]. *Y. enterocolitica* infection involves not only the small intestine but also the colon. Several studies have demonstrated that *Y. enterocolitica* can invade the epithelial barrier of the ileum via M cells, translocate into Peyer patches, and evade immune cells by translocation of Yop effector molecules [8,9]. This pathomechanism may be of relevance for bacterial spread to other organs, a prerequisite for post-infectious manifestations. The bacterial insult also leads to changes in tight junction (TJ) protein expression and architecture. Subsequently, *Y. enterocolitica* induced-diarrhea occurs via a leak flux mechanism [8].

Optimal treatment strategies for *Yersinia* spp. infections are unclear. Most cases of *Y. enterocolitis* do not merit treatment and no clinical benefit of treatment has been documented [10,11]. The treatment of yersiniosis, particularly in those patients who display predisposing conditions and systemic infection, should be pursued with prompt antibiotic treatment because of improved mortality [2,12,13]. The duration of therapy varied from two to six weeks, with a median of 22 days (intravenous followed by oral therapy) [13]. Trimethoprim-sulfamethazole, third-generation cephalosporins, or

aminoglycoside therapy is usually effective [6]. *Y. enterocolitica* has been shown to produce beta-lactamase and is resistant to ampicillin, erythromycin, and first-generation cephalosporins [5,6]. In adolescents and adults, tetracyclines or ciprofloxacin are alternative treatments [6].

Measures to prevent *Yersinia* infection include safe food processing and preparation and hand washing after exposure to pigs or raw pork products, as well as avoiding consumption of raw pork. Hand washing and the control of environmental cross-contamination are important measures to prevent zoonotic transmission of yersiniosis. Pigs are a natural reservoir for *Yersinia spp.* and thus, particular care after handling these animals is warranted.

In conclusion, *Y. enterocolitica* liver abscess is of rare occurrence. *Yersinia* bacteremia is not regularly associated with colorectal neoplasia, but further studies are needed to ascertain whether the bacterium can play a role in triggering or promoting colorectal neoplasia.

References

1. Bottone EJ. *Yersinia enterocolitica*: the charisma continues. *Clin Microbiol Rev.* 1997;10(2):257-76.
2. Cover TL, Aber RC. *Yersinia enterocolitica*. *N Engl J Med.* 1989;321(1):16-24.
3. Mert M, Kocaby G, Ozulker T, Temizel M, Yanar H, Uygun O, et al. Liver abscess due to *Yersinia* bacteremia in a well-controlled type I diabetic patient. *Pol J Endocrinol.* 2011;62(4):357-60.
4. Nemoto H, Murabayashi K, Kawamura Y, Sasaki K, Wakata N, Kinoshita M, et al. Multiple liver abscesses secondary to *Yersinia enterocolitica*. *Intern Med.* 1992;31:1125-7.
5. Hopfner M, Nitsche R, Rohr A, Harms D, Schubert S, Folsch UR. *Yersinia enterocolitica* infection with multiple liver abscesses uncovering a primary hemochromatosis. *Scand J Gastroenterol.* 2001;36(2):220-4.
6. Grigull L, Linderkamp C, Sander A, Schmid H, Mutschler U, Welte K, et al. Multiple spleen and liver abscesses due to *Yersinia enterocolitica* septicemia in a child with congenital sideroblastic anemia. *J Pediatr Hematol Oncol.* 2005;27(11):624-6.
7. Vadillo M, Corbella X, Pac V, Fernandez-Viladrich P, Pujol R. Multiple liver abscesses due to *Yersinia enterocolitica* discloses primary hemochromatosis: three case reports and review. *CID.* 1994;18(6):938-41.
8. Hering NA, Fromm A, Kikhney J, Lee IF, Moter A, Schulzke JD, et al. *Yersinia enterocolitica* affects intestinal barrier function in the colon. *J Infect Dis.* 2016;213(7):1157-62.
9. Heesemann J, Sing A, Trülsch K. *Yersinia*'s stratagem: targeting innate and adaptive immune defense. *Curr Opin Microbiol.* 2006;9(1):55-61.
10. Frydén A, Bengtsson A, Foberg U, Svenungsson B, Castor B, Kärnell A, et al. Early antibiotic treatment of reactive arthritis associated with enteric infections: clinical and serological study. *BMJ.* 1990;301:1299-302.
11. Press N, Fyfe M, Bowie W, Kelly M. Clinical and microbiological follow-up of an outbreak of *Yersinia pseudotuberculosis* serotype Ib. *Scand J Infect Dis.* 2001;33(7):523-6.
12. Jensen KT, Arpi M, Frederiksen W. *Yersinia enterocolitica* septicemia in Denmark 1972-1991: A report of 100 cases. *Contrib Microbiol Immunol.* 1995;13:11-5.
13. Gayraud M, Scavizzi MR, Mollaret HH, Guillevin L, Hornstein MJ. Antibiotic treatment of *Yersinia enterocolitica* septicemia: A retrospective review of 43 cases. *Clin Infect Dis.* 1993;17(3):405-10.