JOURNAL OF CLINICAL MEDICINE OF KAZAKHSTAN (E-ISSN 2313-1519)
Case Report DOI: https://doi.org/10.23950/jcmk/13256

Splenic infarction associated with Salmonella typhi infection: A rare case report

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Received: 2023-02-12.
Accepted: 2023-04-05

J Clin Med Kaz 2023; 20(3):109-111

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Abstract

We described a rare case of a splenic infarction associated with typhoid fever in a 26-year-old Bangladeshi man who returned from a vacation 10 days ago from his home country and presented to the emergency department with fever followed by left hypochondrial pain. Contrast-enhanced computed tomography of the abdomen showed multiple areas of splenic infarction. Salmonella typhi, which was sensitive to ceftriaxone, grew in the blood culture. The patient received intravenous ceftriaxone, analgesics, hydration, antiemetics, and other supportive care. He showed significant clinical improvement and was discharged. The patient was seen at the hospital two months after discharge, he was doing well and no splenic infarction could be detected by sonography.

Key words: typhoid fever, splenic infarction, antibiotics, analgesics

Introduction

Splenic infarction is an infrequent clinical entity that has various etiologies and presents with variable and nonspecific symptoms. Splenic infarction occurs when the splenic artery or any of its branches are occluded, either by distant emboli or by thrombosis in situ [1]. The most common causes of splenic infarction include infiltrative hematologic diseases that cause congestion of the splenic circulation by abnormal cells, or thromboembolic conditions that produce obstruction of larger vessels. Other causes include abdominal trauma, pancreatic disorders, hyper-coagulable states, autoimmune diseases, vascular disorders, and infections [2,3]. Additionally, iatrogenic splenic infarction following various procedures has been reported in the literature [4]. Infections complicated by splenic infarction have been reported in the literature, including the Epstein-Barr virus, cytomegalovirus, malaria, and brucellosis [5-7]. Typhoid fever cases accompanied by splenic infarction are rare; only a few cases have been reported in the literature [8-11]. In this report, we presented a rare case of splenic infarction associated with typhoid fever.

Case presentation

A 26-year-old Bangladeshi man, previously in good health, presented to the emergency department with a 6-day fever followed by rash and left hypochondrial pain. The pain was sharp 7/10 on a scale of 10, increasing with breathing and radiating to left shoulder. He came back from Bangladesh 10 days ago. On examination he appeared ill but conscious and oriented. The temperature was 39.4°C, the pulse rate was 120/minute and the blood pressure was recorded as 110/85 mm Hg. There was a tender enlarged spleen. The rest of the systemic examination was unremarkable.

Initial investigations revealed leukocytes of 13600/ul (mainly neutrophils); hemoglobin 10 g/dl; and platelets 450,000/ul. Blood sugar and kidney function tests were within the normal range. Liver enzymes were elevated (AST 220 u/l and ALT 122 u/l). A malaria parasite smear was negative and the urine dipstick and microscopy were normal. His C-reactive protein was 70 mg/L and blood samples were sent for Gram stain and culture.

Urgent abdominal ultrasound showed an enlarged spleen with irregular hypodense areas within the spleen suggestive of splenic infarctions. Contrast-enhanced computed tomography (CT) of the abdomen also showed findings consistent with splenic infarction (Figure 1). Further testing to determine the causes of the infarction revealed negative results for sickling, antinuclear factor, lupus antigen, and human immunodeficiency virus (HIV) serology, while no vegetations were visible on transthoracic echocardiography. Protein C and S activities were within normal limits and there were no abnormalities in splenic artery and vein Doppler.

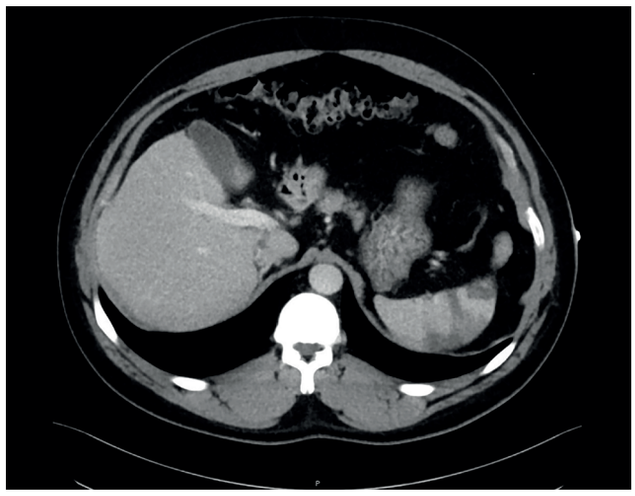

Figure 1 - Contrast-enhanced computed tomography of the abdomen shows hypodense splenic lesions consistent with splenic infarction.

He was admitted to the medical ward and given piperacillin-tazobactam intravenously while the sepsis workup was pending. On the following days, blood culture grew *Salmonella typhi* sensitive to piperacillin–tazobactam, ampicillin, ciprofloxacin, and ceftriaxone. The patient received intravenous ceftriaxone for 2 weeks. He showed significant clinical improvement and was discharged. Two months after discharge, the patient was seen in the clinic, he was asymptomatic and a repeat ultrasound of the abdomen was normal.

Discussion

Splenic infarction as a part of the extraintestinal complication of typhoid fever is rare, only a few cases have been reported worldwide. The exact incidence of splenic infarction in patients with typhoid fever is not well known because the diagnosis is often overlooked due to its nonspecific presentation, which offers no clues or indicators [1]. To the best of our knowledge, this is the first reported case of typhoid fever-associated splenic infarction in Qatar.

The clinical presentation of patients with splenic infarction may include nonspecific abdominal pain of variable intensity, nausea, vomiting, and fever [1-3, 12], probably resembling the symptoms of enteric fever. Therefore, in the setting of infectious diseases such as enteric fever, a splenic infarction may go unnoticed because clinicians are more concerned with determining the cause of the sepsis and frequently start broad-spectrum antibiotics, which can mask the condition. On the hand, presenting with severe abdominal pain and a tender abdomen may prompt physicians to request an abdominal CT scan resulting in the incidental detection of spleen infarction on a radiological test that was not intended to diagnose spleen infarction, as the case in our report.

Since the diagnosis of splenic infarction is based on clinical suspicion and imaging [12], a high index of suspicion is needed when a patient presents with abdominal pain and/or tender splenomegaly in the setting of febrile illness in endemic areas or after returning from an endemic area as in our case. However, between 17% and 20% of patients with splenic infarction reported no abdominal pain [1,2], making diagnosis in such cases challenging or impossible.

Although the precise pathogenesis of splenic infarction is still unknown, several mechanisms, including infection-induced injury to endothelium and nearby tissues, infected embolism, endophlebitis, triggering of the inflammatory cascade, infection-induced red blood cell rouleaux formation, and hypercoagulable state, have been proposed [5,12,13].

Laboratory workups are of no value in the diagnosis of splenic infarction, and contrast-enhanced CT of the abdomen is the imaging modality of choice to identify this clinical entity. Following the diagnosis of splenic infarction, all efforts should be directed towards finding the underlying etiology as these aids in patient management and prognosis [11-13]. We assumed that our patient had a typhoid-associated splenic infarction based on the isolation of Salmonella typhi from the patient's blood and the exclusion of common causes of splenic infarction by the absence of thrombi or vegetations on the echocardiogram, the absence of evidence of sickle erythrocytes, the negative results of antinuclear antibodies, lupus antigen, and human immunodeficiency virus. In addition to the normal splenic artery and vein doppler.

Treatment of splenic infarction in the setting of enteric fever consists primarily of administration of antibiotics, analgesics, hydration, antiemetics, and other supportive measures. There is uncertainty about the role of anticoagulation in the management of these patients. Our patient was treated conservatively with analgesics, analgesics, and antibiotics, with significant improvement.

Conclusion

Splenic infarction is a rare complication of typhoid fever that requires a high index of suspicion. Physicians should be aware that pain in the left hypochondrium that occurs during a febrile illness may be due to splenic infarction. Therefore, abdominal CT is the first option for diagnosis, and treatment consists mainly of the administration of antibiotics, analgesics, hydration, antiemetics, and other supportive measures.

Disclosures: There is no conflict of interest for all authors.

Acknowledgements: None.

Funding: None.

Patient informed consent: Obtained

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