CASE REPORT

Splenic infarct: A rare complication in extensively drug-resistant *Salmonella typhi*

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Abstract

Enteric fever is a common systemic infection characterised by high grade fever and variable gastrointestinal symptoms. Splenic abscesses, thrombosis, and infarcts are uncommon complications. We report a case of splenic infarction in a young female presenting with persistent fever and abdominal pain. Her blood culture showed drug-resistant Salmonella Typhi (S.Typhi). Due to persistent abdominal pain, abdominal imaging was performed, which revealed multiple splenic infarcts. Treatment with appropriate antibiotics led to complete resolution of symptoms. This case emphasises the importance of timely diagnosis with relevant investigations for the effective management of an unusual presentation of enteric fever.

Keywords: Enteric fever, splenic infarct, XDR Salmonella typhi.

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Introduction

Enteric, or typhoid, fever is a systemic bacterial infection caused by Salmonella enterica subtype Typhi and less commonly by Paratyphi A. It affects roughly 11 to 20 million people globally and according to the World Health Organization, it causes between 128,200 and 161,000 deaths annually. The clinical presentation typically includes high-grade fever, abdominal pain, diarrhoea, and vomiting. Around one-fourth of patients develop complications, with anaemia and delirium being the most prevalent.[1] Moreover, enteric fever has been associated with the involvement of nearly all body organ systems, including the spleen.[2] Although rare, splenic complications such as solitary or multiple micro abscesses of the spleen, splenic rupture, or infarct may occur during the course of illness.[3,4] Splenic abscesses are estimated to occur in about 0.2–0.7% of enteric fever cases.[5] However, data regarding the prevalence of splenic infarction as a complication of enteric fever remain scarce.

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Case Report

A 26-year-old woman with no known comorbidities presented to the Emergency Department at Aga Khan University Hospital, Karachi on 19th August 2022 with complaints of diarrhoea, left-sided abdominal pain, and daily high-grade fever unresponsive to antipyretics. These symptoms had been present for one week. She had non-bloody stools occurring three to four times per day. The abdominal pain was localized to the left upper quadrant on deep palpation. It was sudden in onset, non-radiating, and not relieved by over-the-counter analgesics. Due to persistent fever and abdominal pain, she sought medical evaluation at the hospital.

Her vital signs on admission showed a body temperature of 39.2°C, pulse rate 98 beats per minute, respiratory rate 23 breaths per minute, and blood pressure of 100/75 mmHg. Her physical examination was unremarkable, except for the tenderness in the left upper quadrant upon deep palpation, with no accompanying mass or organomegaly. Her haemoglobin level was 116 g/L (range: 123- 166g/L), and her white blood cell count was 4.5 x 10^9/L (range: 4.8- 11.3 x 10^9/L), with 68% neutrophils (range: 34.9-76.2%). The peripheral film did not how a leucoerythroblastic picture. Malarial parasites were not detected, and dengue antigen was also negative. Elevated C-reactive protein with value of 116.18 mg/L (range: 0- 10 mg/L) was observed.

ALT was 41 U/L (range: 0–45U/L), AST was 39 U/L (range: 0–35U/L), GGT was 34 U/L (range: 0-55U/L), total bilirubin was 5.13 umol/L (range: 1.71–3.42 umol/L), and direct bilirubin was 2.05 umol/L (0– 3.42 umol/L). Serum electrolytes and creatinine levels were within normal limits. Serum lipase was mildly elevated at 158 U/L (range: 6- 51 U/L).

An abdominal ultrasound was performed due to significant left sided abdominal soreness, which revealed no abnormal findings. Given the persistence of abdominal pain, a CT scan of the abdomen and pelvis was performed with contrast. This indicated the presence of splenic infarcts in multiple peripheral wedge-shaped hypodense regions of the spleen. (Figure).

A 12-lead ECG revealed no abnormalities. A Transthoracic echocardiogram revealed a normal ejection fraction with

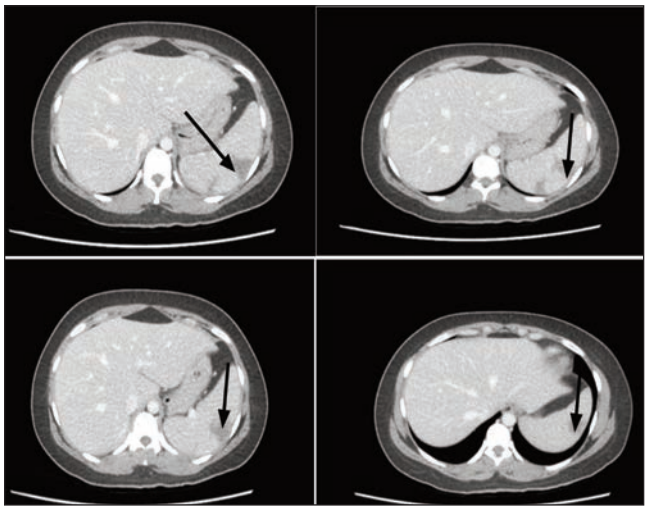

Figure: Splenic infarcts in the CT scan abdomen (axial view) with contrast (arrows are pointing to the infarcts).

no evidence of intracardiac thrombus or valvular vegetation. The group test for anti-nuclear antibodies was also negative. Blood cultures showed Extensively Drug-Resistant Salmonella Typhi (XDR), which was only carbepenem sensitive.

The patient was administered IV meropenem one gram every eight hours with adequate analgesia, resulting in the rapid resolution of her fever and abdominal pain within 48 hours. She was discharged on the same dose of IV meropenem, for a total of 14 days. On follow-up, a repeat blood culture yielded no organism, and the patient remained asymptomatic.

Discussion

Enteric fever is one of the most common causes of prolonged fever in low- and middle-income countries and carries a significant disease burden. It typically presents with high-grade fever and a range of systemic symptoms, including lethargy, nausea, and anorexia, as well as less frequent manifestations such as constipation, diarrhoea, abdominal pain, cough, and rash.[6]

Timely diagnosis and initiation of appropriate antibiotics are essential to prevent complications. In cases of delayed treatment, complications are commonly observed in 10-15% of patients during the second to fourth week of illness.[7,8] The Causes of complications are multifactorial and may include infection with an antimicrobial resistance strain, delayed treatment, and younger age.[7,8] The most commonly seen complications in the Asian population include delirium (32.1%), anaemia (19.4%), and disseminated intravascular coagulation (14.8%).[9]

The more severe and fatal complications include typhoid intestinal perforation (0.7%), gastrointestinal haemorrhage (3.1%), hepatitis (4.4%), shock (1.6%), encephalopathy (4%), and pneumonia (3%).[10]

Atypical complications of enteric fever are rarely reported but may involve multiple organ systems. Findings include abdominal lymphadenopathy, acalculous cholecystitis, Gullian-Barre syndrome, splenic and liver abscess, myocarditis, and pneumonitis. Splenic infarction is also infrequently reported. The exact mechanism of this complication is unclear; however, several theories have been proposed. These include direct tissue injury secondary to bacteraemia, septic embolization, endophlebitis, endothelial damage, inflammatory response, and a hypercoagulable state. However, the widely accepted theory suggests that Salmonella Typhi bacteraemia causes an increase in procoagulant factors and consumption of clotting factors with a subsequent decrease in anticoagulant factors which promotes the development of conditions such as splenic abscesses, DIC10 and arteritis. De Hong et al. in their case control study found increased production of in-vitro thrombin and high levels of plasma fibrinogen with prolonged prothrombin and partial prothrombin times further reinforcing this theory. Acute splenic infarcts (ASI) are typically characterized by left-sided abdominal pain; however, they may also be identified in individuals with vague abdominal complaints. These defects typically appear as a wedge-shaped area of hypodensity on the CT scan, which is the most sensitive modality for detecting them.

Splenic infarction has also been associated with tropical infections such as Cytomegalovirus, Epstein–Barr virus, and malaria.[10] This process has been linked to several processes, including a transient hypercoagulable condition brought on by viral complexes, an increase in circulating immune complexes that cause vascular blockage, and insufficient blood flow to an enlarged spleen. Leukaemias, lymphomas, sickle cell haemoglobinopathy, systemic embolisation, thrombosis following myocardial infarctions, infectious endocarditis, and traumas are among the more prevalent causes of splenic infarcts.[8] In our case, echocardiography showed no valvular vegetations, peripheral blood film revealed no leukoerythroblastic picture, and erythrocyte sedimentation rate (ESR) was within normal limits, thereby ruling out common etiologies. Additionally, a negative antinuclear antibody (ANA) test made autoimmune pathology a less likely cause. Based on these findings, it was concluded that enteric fever was the likely etiological factor contributing to the development of splenic infarction in our patient.

Conclusion

In conclusion, splenic infarction is an infrequently reported complication of enteric fever. Clinicians should maintain a high index of suspicion for this condition in patients presenting with persistent abdominal pain, to facilitate timely diagnosis and appropriate management.

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Conflict of Interest: None.

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AA: Supervision, review and final approval.