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An Unusual Cause Of Splenic Infarction In A Child With Enteric Fever

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Abstract
Splenic infarction is a rare but potentially serious complication of enteric fever, often presenting as left hypochondrial abdominal pain. We present a case of a 12 year-old female child from southern India who presented with fever and left hypochondrial pain. Ultrasound revealed hypoechoic lesions in the spleen, confirmed later by computed tomography (CT) scan showing multiple splenic infarcts. The diagnosis was supported by a high Widal titre and positive blood culture for Salmonella typhi. Treatment with intravenous Ceftriaxone led to clinical improvement. Splenic infarction in enteric fever, although rare, can occur early in the disease course. Early detection through imaging modalities like CT scan is crucial to prevent potential complications such as splenic rupture. Prompt antibiotic therapy is essential in managing this complication and averting morbidity. Awareness of this rare but reversible complication is crucial for timely intervention and management.

Keywords: Splenic infarct, enteric fever, rare complication, CT scan, infectious disease.

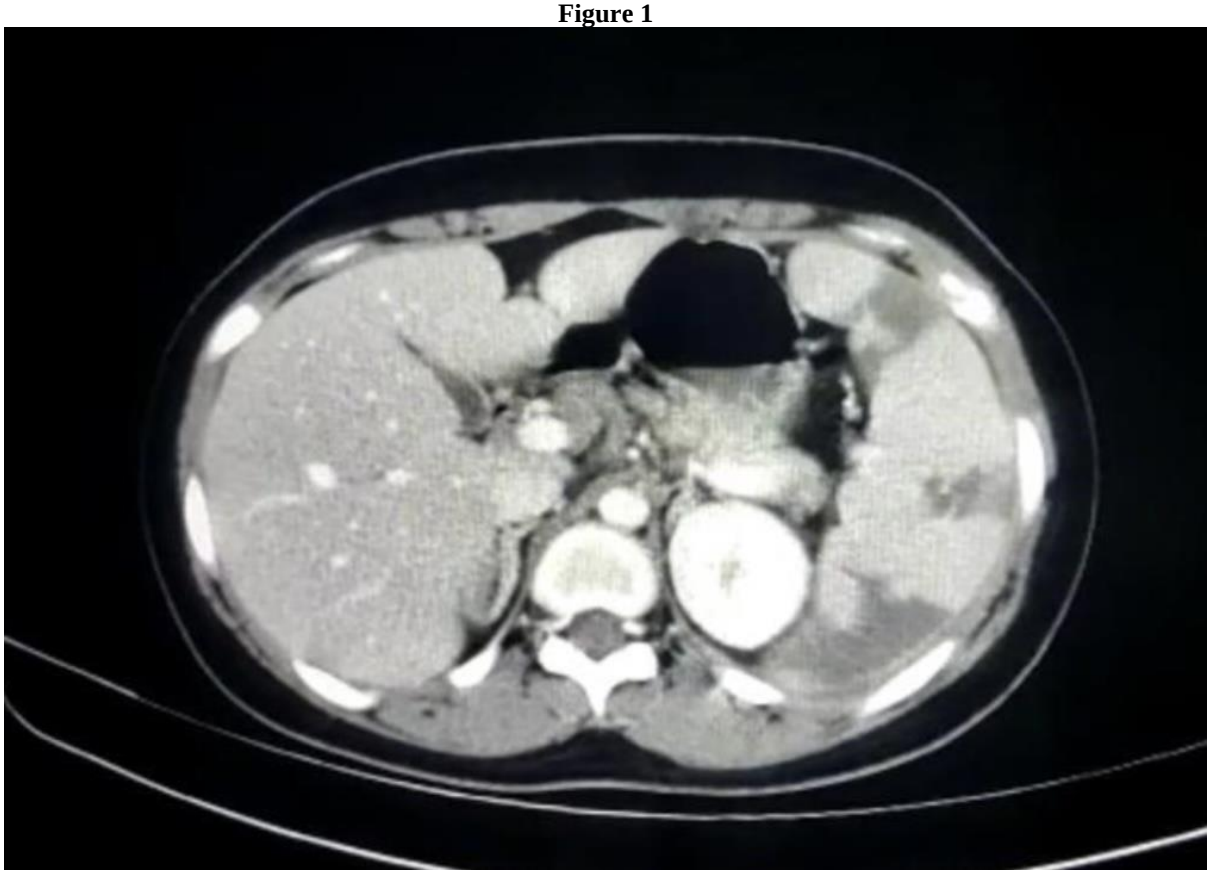
I. INTRODUCTION

Splenic infarction is an infrequent cause of acute abdomen in the emergency room. Splenic infarction occurs when blood flow to the spleen is compromised causing tissue ischemia and eventual necrosis. It may be the result of arterial or venous occlusion. Occlusion is usually caused by bland or septic emboli as well as venous congestion by abnormal cells (1). Infarction may involve a small segmental area of the spleen or may be global depending on which vessel is occluded. The two most common causes of splenic infarct are thromboembolic disease and infiltrative hematologic diseases (2). Sickle cell disease is a common cause of vaso occlusive disease in the paediatric population and can present as infarcts in the spleen, liver, brain, bone and lungs (3,4). In patients under 40 years of age, the most common cause is a hematologic disease (5). Splenic infarction can occur in enteric fever due to several mechanisms including septic emboli, vascular compromise, venous congestion and hypercoagulable state. Here we describe a rare case of splenic infarct caused by enteric fever.

II. CASE REPORT:

A 12 years old female child from the southern India presented with low grade continuous fever partly relieved by antipyretics, with no diurnal variation or chills and rigor. She also had on and off left hypochondrial abdominal pain lasting 4 days, without any aggravating or relieving factors. This was followed by nonprojectile nonbilious vomiting lasting 2 days, 2-3 episodes/day; without any blood tinge. After admission fever increased in intensity and became high grade. The examination was unremarkable with stable vitals, no lymphadenopathy, anemia or jaundice. There was no petechiae or purpura. Throat was not congested; but tongue was coated. Per abdominal examination was unremarkable except for the tenderness in left hypochondriac region with no rebound tenderness. Ultrasound abdomen showed moderate splenomegaly with hypoechoic lesions in it. Other Investigations done are depicted in Table 1. In view of persistent high grade fever and suspicion of Enteric fever child was started on Inj Ceftriaxone. High Widal titres of 1:320 and blood culture with growth of Salmonella typhi supported the antibiotic choice. Since USG showed hypoechoic lesion, CECT abdomen was done which detected multiple splenic infarcts (Fig. 1). Though child became afebrile after 48 hours of starting intravenous Ceftriaxone, the antibiotics were continued for 14 days after which child was discharged based on clinical improvement.

Figure 1



III. DISCUSSION:

Splenic infarction is a rare cause of abdominal pain in childhood enteric fever (6). It is commonly associated with hematological conditions such as leukemia and lymphoma, thromboembolic disorders such as infective endocarditis and rarely with intra-abdominal infections. In a study, cardioembolic events were the leading cause of splenic infarcts in 89 subjects (2). Tropical infections can rarely present or masquerade as splenic infarcts. Recently, splenic infarction has been described in infections such as malaria, Epstein barr virus and cytomegalovirus infections (7).Other less commonly known etiologies include autoimmune and vascular diseases such as polyarteritis nodosa, embolic events secondary to atrial fibrillation, sickle cell disease and trait, systemic infections and trauma. These disorders compromise the segmental vascular supply of the spleen, resulting in wedge-shaped infarcts (8). Enteric fever is an atypical cause of splenic infarction which may occur during the acute phase of enteric fever when the infection is actively replicating and spreading in the body due to the formation of septic emboli (9). However, splenic infarction can also occur later in the course of enteric fever, especially if the infection is not promptly diagnosed and treated. Chronic or recurrent bacteremia can contribute to ongoing vascular damage and thrombosis, increasing the risk of splenic infarction over time (10,11). However, in the present child, splenic infarct was noticed as early as 4th day of fever. The possible mechanism in this child could be the hyper inflammable state as suggested by high CRP, resulting in hypercoagulability. Discovering infarcts early in enteric fever might facilitate early appropriate antibiotic use, which may avert a potentially life-threatening complication like splenic rupture or hemorrhage which can occur later in enteric fever.

The exact mechanism for splenic infarct in infectious diseases is still obscure. It maybe due to septic emboli following bacteremia (9). The infection-related inflammation and vascular compromise associated with enteric fever can disrupt normal blood flow to the spleen. This disruption can result in reduced perfusion and subsequent ischemic injury, contributing to splenic infarction (11). Venous congestion in the spleen due to the accumulation of abnormal cells or inflammatory debris may also cause infarcts (12). Some theories propose that the hypercoagulable state occurring in the setting of hyperinflammation could be a cause (9). The present child had moderate splenomegaly with normal liver functions which ruled out hepatobiliary causes. Elevated liver function tests, bilirubin or lipase, may suggest a hepatobiliary or pancreatic source for pain. Though leukocytosis and elevated lactate dehydrogenase may be found in splenic infarction, the index child did not have any of these findings which is unusual (7).

Though USG can detect splenic infarct quite early as wedge-shaped hypoechoic lesions, which later mature to hyperechoic with retraction of splenic capsule, in the hyperacute stages, CT abdomen with I/V contrast is the imaging modality of choice. As in the present child, in the

hyperacute phase of infarction, splenic infarct appears as a wedge-shaped area of splenic tissue with the apex pointed toward the hilum and the base towards the splenic capsule. As the infarction matures, the affected tissue may normalize, liquefy or become contracted or scarred. Other cases reported were a case of splenic infarction a rare complication of XDR enteric fever in a 21 year old by Wania et al which was treated with 21 days of antibiotics. A case of Salmonella paratyphi-induced splenic vein thrombosis in a 25 year old by Ruchika et al in Chandigarh treated with 14 days of ceftriaxone. Shaukat ali et al described a case of splenic infarction in a 12-year-old male child with enteric fever who was treated with 14 days of ceftriaxone.

Treatment of splenic infarct is based primarily on the underlying causative disease state. Dangerous complications of splenic infarct include pseudocyst formation, abscess, hemorrhage, splenic rupture, and aneurysm (14).

Enteric fever encompasses typhoid fever, caused by infection with bacteria *Salmonella* typhi (*S* typhi), and paratyphoid fever, caused by *Salmonella* paratyphi A and B. *S* typhi is estimated to cause 76% of enteric fever globally. Patients present with a gradual onset of fever which typically rises to a plateau of 39-40°C (102-104°F) towards the end of a week. Abdominal symptoms such as diarrhoea, nausea, vomiting, and abdominal pain are common. Patients may also have headache, cough, and malaise. Physical findings are often non-specific. Soft tender hepatosplenomegaly, abdominal distension, mild ascites, and a diffuse or localised tenderness may be noticed on abdominal examination (15). Hepatitis and hepatomegaly are more common in children under 5 years old and are seen in 30-50% of children with enteric fever. A bradycardia relative to the height of the fever may be noted. Rose spots, blanching erythematous maculopapular lesions on the trunk, were considered characteristic of typhoid fever, but are now rarely reported. Blood culture is the optimum method to confirm the diagnosis by isolating the organism and testing antimicrobial sensitivity. A negative blood culture does not exclude enteric fever. Antibiotic pre-treatment, low sample volume, and low circulating bacterial load in the blood result in this low sensitivity. Bone marrow culture gives a higher yield, but it is rarely performed. Faeces, urine, or bile aspirate may be cultured, but a positive result may indicate chronic faecal carriage rather than acute infection. Serological tests, including the Widal test and newer rapid diagnostic tests, are not confirmatory in the acute phase of illness (13). Encephalopathy, gastrointestinal bleeding, nephritis, and hepatitis are common complications seen in 5-7% of hospitalised patients . But here we describe a rare case of enteric fever causing splenic infract in paediatric age group which required antibiotics and supportive management.

Table 1

	24/02/22	3/03/22
Hb	12.4	11.0
TLC	6010	7520
Platelet	2.07	3.73
PCV	37.7	32.9
CRP	40.2	11.8
Peripheral smear	Normal study	
Sodium	141	
Potassium	4.6	
chloride	105	
Bicarbonate	20.2	
PT	15.9	
APTT	22.2	
INR	1.38	
Urea	16	
Creatinine	0.7 (eGFR 120ml/hr)	
Serum Lipase	308	
Total Bilirubin	0.30	
Direct Bilirubin	0.22	
SGOT	32	
SGPT	16	
Alkaline Phosphatase	107	
Total Protein	6.7	
Albumin	3.3	
Serum Amylase	82	

IV. CONCLUSION:

Splenic infarct is a rare complication of enteric fever. In pediatric population, quite often we may not get the typical step ladder pattern as seen in adult. Relying on Laboratory findings are often unproductive as they are not very specific. Imaging modalities specifically CT scan with IV contrast, can help in clinching the diagnosis and avoid complication in suspected patients. Identifying possibility of infarcts early in possible enteric fever cases presenting with left hypochondriac pain by imaging, may avert potential complications like splenic hemorrhage /rupture in 2^{nd} or 3^{rd} week. At least in endemic countries, there should be Selection of appropriate antibiotic also will avert morbidity of prolonged illness. Being a reversible pathology, one must be aware of this complication for timely identification and management. One should always consider the possibility of splenic infarcts in countries where enteric fever is endemic and proceed to treat accordingly.

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FIGURE LEGENDS:

Figure 1 showing multiple splenic infarcts (hypoechoic regions)
Table 1 showing blood investigations of the child