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Splenic infarct secondary to salmonella typhi infection

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Abstract

Ultrasonography and Contrast enhanced CT done in a 12 year old female, which shows wedge shaped hypo echoic lesions extending from periphery to hilum with no evidence of vascularity within. On follow-up plain CT these lesion appear hypo dense wedge shaped areas with non-enhancement in post contrasting. Patient presented with complaints of fever and pain in left hypochondria. Antibiotic course of Ceftriaxone was given following which patient symptomatically improved and resulted in recovery. Rapid diagnosis and management of extra intestinal manifestations of salmonella typhoid and salmonella para typhoid group of organisms can be aided by imaging techniques.

Keywords: Salmonella typhoid; splenic infarct; typhoid fever

Introduction

Usual presentations of salmonella includes typhoid fever caused by salmonella typhoid and paratyphoid fever caused by salmonella paratyphoid A, B and C. Clinical course may vary from mild pyrexia to rapidly fatal disease. Typhoid fever also causes extra intestinal complications which may involve the central nervous system, cardiovascular system, pulmonary system, bones and joints system, hepatobiliary system, gastrointestinal system, and so forth¹. Typhoid fever is a systemic disease caused by Salmonella typhoid. Splenic abscess, sub phrenic abscess and pancreatitis are rare complications of typhoid fever. The extra intestinal manifestations⁸ of typhoid fever are caused by the seeding of organs during bacteremia. Sub phrenic abscess could be caused by intestinal perforation or by extension of splenic abscess^[2]. Splenic infarction is not common and it is usually associated with a hematological or rheumatologic disorder^[3]. We present a case of typhoid fever associated infarction of spleen that was identified during abdominal imaging. The diagnosis of typhoid fever was confirmed by the isolation of Salmonella typhoid from blood culture (WIDAL TEST). Spleen infarction was diagnosed earlier by imaging techniques such as USG and CECT. The unique contribution of the imaging towards diagnosis of Salmonella typhoid infarction of spleen has probed us to report this case.

Case Report

A 12-year-old female presented with fever for 1 week followed by pain in the left hypochondria. The pain was insidious in onset, gradually progressive shows no diurnal variation and was unrelieved by medication. On clinical examination, there was tenderness in the left hypochondria. However there is no evidence of organomegaly. The ultrasonography examination revealed hepatomegaly and multiple wedge-shaped hypo echoic lesions extending from the periphery to the hilum with no evidence of vascularity within (Figure 1) - Suggestive of splenic infarction and followed up with contrast-enhanced CT on the indicated, which showed wedge-shaped hypo dense areas in the spleen extending from the periphery to the hilum with no enhancement in post contrast (Figure 2b). Investigations to exclude hematological or rheumatologic factors responsible for splenic infarction were all negative. Echocardiography was normal.

Routine hematological and biochemical parameters (Table a) showed Reduced RBC levels (3.83 million / cu mm), Borderline increase in Lipase (308 U/L), increased C - reactive protein and the blood culture was positive for salmonella typhoid. WIDAL test showed titers suggestive of enteric fever (Table b). During the in vitro assay, the isolate was susceptible to ceftriaxone, chloramphenicol, tetracycline, ciprofloxacin, amikacin, cefaclor, ceftazidime, and ceftizoxime. The patient was prescribed Ceftriaxone, antipyretics, analgesics and IV

fluids. After 2 weeks of hospitalization and an antibiotic course of Ceftriaxone, the patient became afebrile and showed a gradual reduction in pain. The patient was discharged with oral antibiotics and was suggested repeat

ultrasound follow-up. Patient widely test was done 1 week later showed insignificant titers suggestive resolution of enteric fever.

Table (a): Routine hematological and biochemical parameters showing Reduced RBC, Borderline increase in Lipase, increased C - reactive protein

| Test | Result | Units | Reference range |
|--|----------|----------------|-------------------------|
| Hemoglobin (HB) | 11.0 | g/dl | Male 13-17 Female 12-15 |
| Total RBC Count | 3.83 | millions/cu.mm | 4.0 - 5.2 |
| Packed Cell Volume (PCV) | 32.9 | % | 35 - 45 |
| Mean corpuscular volume(MCV) | 85.9 | FL | 81-101 |
| Mean corpuscular hemoglobin (MCH) | 28.7 | PG | 27 - 32 |
| Mean corpuscular hemoglobin concentration (MCHC) | 33.4 | g/Dl | 31 - 37 |
| Red cell distribution width | 12.5 | % | 11.6 - 14 |
| Platelet Count | 3.73 | lakhs/cu mm | 1.7 - 4.5 |
| Total Leucocyte Count (TLC) | 7520 | cells/cumm | 5000 - 13000 |
| Neutrophils | 69.6 | % | 40 - 80 |
| Lymphocytes | 23.1 | % | 20 - 40 |
| Monocytes | 6.3 | % | 2 - 10 |
| Eosinophil's | 0.9 | % | 1 - 6 |
| Basophils | 0.1 | % | <1 - 2 |
| Absolute Neutrophil Count (ANC) | 5230 | cells/cumm | 2000-7000 |
| Smear for MP/MF | Negative | | Negative |
| Serum lipase | 308 | IU/L | 23-300 |
| C-reactive protein | 11.8 | mg/L | < 10 mg/L- Negative |

WIDAL Test

| | |
|----|----------------|
| TO | 1:320 Positive |
| TH | 1:320 Positive |
| AH | 1:20 Negative |
| BH | 1:20 Negative |

Titers suggestive of Enteric fever
 Table (b): WIDALS Test
 Blood culture and sensitivity:

Table (c): Blood culture and sensitivity

| | |
|-----------------|-----------|
| Ceftriaxone | Sensitive |
| Chloramphenicol | Sensitive |
| Tetracycline | Sensitive |
| Ciprofloxacin | Sensitive |
| Amikacin | Sensitive |
| Cefaclor | Sensitive |
| Ceftazidime | Sensitive |
| Ceftizoxime | Sensitive |
| Ampicillin | Resistant |
| Trimethoprim | Resistant |

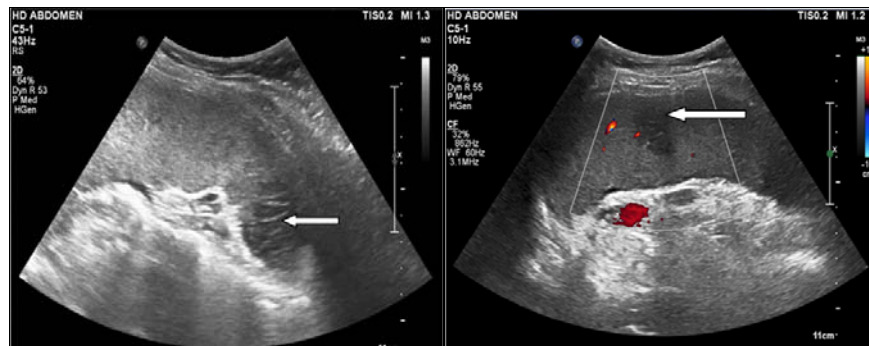


Fig 1 (a)

Fig 1 (b)

Fig 1: Sonographer of the abdomen showing few hypo echoic areas in spleen (white arrow in Fig. 1 (a) with no internal vascularity (white arrow in Fig. 1 (b))



Fig 2 (a)

Fig 2 (b)

Fig 2: Plain {Fig. 2 (a)} and post contrast {Fig. 2 (b)} computed tomography of the Abdomen showing splenic infarcts (white arrow)

Discussion

Splenic infarcts are credited to chronic myelogenous leukemia, hemoglobinopathy, systemic embolization; myelofibrosis, mural thrombosis following acute myocardial infarction, postpartum toxic syndrome, HIV-associated mycobacterium, splenic venous thrombosis and trauma⁴. Hematological and autoimmune pathology was ruled out following normal peripheral smear, negative rheumatoid factor, and negative antinuclear acid antibody. A negative HIV serology and absence of abnormalities in the splenic artery and vein Doppler studies were helpful in ruling out other well-known causes for splenic infarction like HIV, splenic artery thrombosis. A normal echocardiogram ruled out cardiac origin of the infarct.

In the aspect of any spleen involvement in enteric fevers, imaging techniques would prove to be constructive. In a previous study done on Turkey⁵, infarction of spleen was reported in an otherwise healthy patient caused by group B Salmonella. He was seropositive for the O antigen of Salmonella group B, and stool cultures were positive for group B Salmonellae. In another study done on France⁷, splenic abscess was reported in an otherwise healthy patient caused by gram negative salmonella typhoid. After treatment with susceptible antibiotics, the patient was normal four months later. As evident in our present case, aggressive therapeutic intervention and recovery can be made with an earlier ultrasonic diagnosis of splenic infarction. With multidrug resistant Salmonellae an emerging entity, contrast-enhanced sonographer would be of diagnostic value toward the elimination of any episodes of splenic infarct/splenic rupture in enteric fever. Sonographer examination would aid in early diagnosis and further antibiotic management in patients with salmonella infection who are liable for splenic infarction and splenic rupture⁶.

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Declarations

Ethics approval and consent to participate – Institutional ethics committee approval obtained and patient data anonymized and written informed consent of the patient for participation in the study wherever applicable obtained.

Consent for publication –All radiological images used in the study have been anonymized by removing patient identifiers.

Competing interests - The authors declare that they have no competing interests.

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All authors have read and approved the manuscript.

Conflict of Interest

Not available

Financial Support

Not available

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