Infarction of spleen in typhoid fever

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ABSTRACT

Ultrasonography and computed tomography scan with hypo echoic areas diagnosed a splenic infarction in a *Salmonella typhi* infected 30-year-old man with painful hypochondrium and epigastrium. An antibiotic recipe of ceftriaxone and amikacin resulted in recovery. Imaging techniques contribute remarkably to a rapid diagnosis and rational management of the extra intestinal lesions attributable to the *Salmonella typhi/paratyphi* group of organisms.

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Extra intestinal complications of typhoid fever involve the central nervous system, cardiovascular system, pulmonary system, bones and joints system, hematological hepatobiliary system, gastrointestinal system, and so forth.[1] There have been instances of multidrug resistant *Salmonella typhi* induced spontaneous rupture of spleen.[2] Splenic infarction is not common and it is usually associated with a hematological or rheumatological disorder.[3] We give an account of 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 typhi* from blood. Spleen infarction was diagnosed earlier by imaging techniques. The unique contribution of the imaging towards diagnosis of *Salmonella typhi* infarction of spleen has stimulated us to report this case.

Case Report. A 30-year-old male was reported with high fever, body ache, weakness, and anorexia for the past 3-4 days, and hospitalized at the Sant Parmanand Hospital, a tertiary care hospital in the Indian Capital, Delhi. Clinically, there was hepatosplenomegaly, with normal routine hematological and biochemical parameters. Abdominal ultrasonography showed hepatosplenomegaly. The blood culture was positive for *Salmonella typhi*. During in vitro assay, the isolate was susceptible to chloramphenicol, tetracycline, ciprofloxacin, amikacin, cefaclor, trimethoprim-sulphamethoxazole, ceftazidime, ceftriaxone, and ceftizoxime. He was prescribed ceftriaxone, antipyretics, and nutrients. After 2 days of hospitalization he developed sharp, severe pain in his left hypochondrium and epigastrium. The pain increased during inspiration/expiration and was radiating to the left shoulder. His pulse rate was 110/minute and the blood pressure was recorded as 150/90 mm Hg. There was tenderness in umbilical, epigastric, and left hypochondrium regions. Repeat biochemical and hematological parameters were normal. Radiological evaluations were repeated with no abnormality in chest and abdominal x-rays (**Figure 1**). The repeated ultrasonic examination revealed enlarged spleen with 2 hypo-echoic areas in the parenchyma. They measured 3.3 x 1.7 cm, rounded and 3.3 x 1.7 cm, triangular in shape suggestive of spleen infarction. Compute tomography scan of the abdomen was carried out showing a similar picture suggestive of splenic infarction (**Figure 2**). Investigations to exclude hematological or rheumatological factors responsible for splenic infarction were all negative. There was no erythrocyte sickling, and negative serology for antinuclear factor, lupus antigen, and human immunodeficiency virus (HIV). No abnormalities were evident during Doppler studies on abdominal viscera including splenic artery and vein. Echocardiography was normal. He was prescribed ceftriaxone and amikacin for 2 weeks. He responded well with a gradual reduction in pain and became afebrile, and was discharged on oral antibiotics. A repeat spleen ultrasonography was not feasible as the patient was lost for follow up.

Discussion. Splenic infarcts are attributable to hemoglobinopathy, chronic myelogenous leukemia, myelofibrosis, systemic embolization; mural thrombosis accompanying acute myocardial infarction, postpartum toxic syndrome, HIV associated mycobacterium,

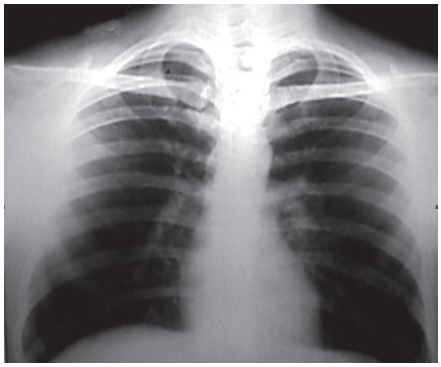

Figure 1 - Chest x-ray with normal postero-anterior view.

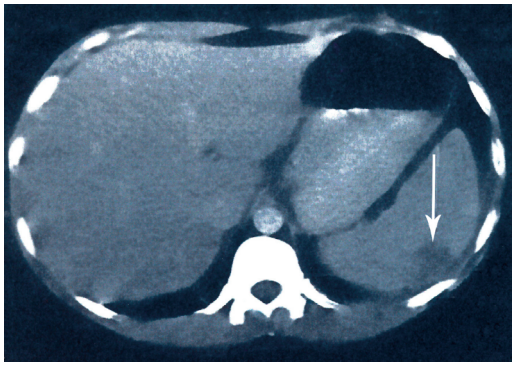

Figure 2 - Computed tomography of the abdomen with arrow pointing towards the splenic infarct.

splenic venous thrombosis, and trauma.[4] Negative erythrocyte sickling, rheumatoid factor, and antinuclear acid antibody ruled out hematological and autoimmune pathology. The well-known causes for splenic infarction were excluded by negative HIV serologic profile, absence of abnormalities in the splenic artery and vein Doppler studies, little myocardial involvement and no operative or traumatic events.

Imaging techniques would be a constructive aspect in the direction of any spleen involvement in enteric fevers. In Turkey, 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*. After appropriate antimicrobial therapy, her complaints disappeared and microbiological tests for *Salmonellae* became negative.[5] As evident in the present case, an earlier ultrasonic diagnosis of splenic infarction ensured aggressive therapeutic intervention and recovery. With increasing emergence of multidrug resistant *Salmonellae*, contrast-enhanced sonography would be of diagnostic value towards elimination of any episodes of splenic rupture in enteric fever.[2] Such procedures would be useful towards an early diagnosis and coherent antimicrobial guidelines in patients with emerging antibiotics resistant microbes liable for peri-splenic lesions, splenic infarction, splenic rupture, splenic hemangioma/splenoma, and accessory spleen.[6]

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