

A curious case of splenic infarction

Gauri Nilajkar¹, Aditya Phadte², Cyrus Pereira³

From ¹Lecturer, ²Junior Resident, Department of Medicine, ³Medical Student, Goa Medical College, Bambolim, Goa, India

Correspondence to: Dr. Gauri Malhar Nilajkar, Department of Medicine, Faculty Block, Goa Medical College and Hospital, Bambolim - 403 202, Goa, India. Email: gaurinilajkar@gmail.com

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ABSTRACT

Splenic infarction is an unusual diagnosis in infectious diseases. It is relatively more common in typhoid fever as opposed to nontyphoidal salmonellosis (NTS). A young male presenting with fever, abdominal pain, and tender hepatosplenomegaly, was investigated. Radiological tests showed the presence of areas of splenic infarction and his blood culture revealed the growth of NTS species. Only 5% of NTS infection present with extraintestinal manifestation such as splenic infarction, while the majority of the cases manifest as gastroenteritis.

Key words: *Computerized tomography, Fluoroquinolones, Nontyphoidal salmonella, Salmonellosis, Splenic infarction*

Splenic infarction is an uncommon cause of acute abdominal pain. Usually, the suspects are hematological or thromboembolic disorders. Splenic infarction occurs due to parenchymal ischemia, due to interrupted arterial supply which causes tissue necrosis. Infections causing splenic infarction are relatively rare. The spleen derives its arterial supply from the splenic artery and short gastric arteries, which form a good collateral system [1]. Splenic infarction is associated with various disorders which can compromise splenic arterial supply, which results in wedge-shaped infarction due to segmental perfusion. The major causes of splenic infarction are hematological disorders such as leukemia, lymphoma, sickle cell disease, and other conditions such as autoimmune diseases, vasculitis syndromes, embolic disorders such as atrial fibrillation and infective endocarditis, and trauma. Infective causes are relatively uncommon and include malaria, leptospirosis, kala azar, infectious mononucleosis, and meningococemia.

Infarction of the spleen may be an incidental finding or may present in the form of severe complication, such as peritoneal rupture of subcapsular splenic hematoma with resultant hemorrhagic shock [1]. However, the most common clinical presentation is of left upper abdominal pain with or without associated fever, nausea, vomiting, and left shoulder tip pain [2].

Imaging is confirmatory for the diagnosis of splenic infarct [3]. Imaging with ultrasonography is limited due to abdominal obesity and bowel gas. Contrast computed tomography is the diagnostic test of choice and reveals segmental wedge-shaped low attenuation defect. Rarely, the entire spleen is seen to be infarcted with contrast enhancement of capsule. Other tests such as magnetic resonance imaging with gadolinium contrast, scintigraphy, and radiolabeled autologous leukocyte scan are newer diagnostic modalities, available in few centers [4].

Medical management of splenic infarction includes analgesia and supportive care. There is no clear role for antibiotics or antiplatelets. If splenic abscess is suspected or confirmed, splenectomy is indicated. Surgical management is also needed in cases of hemorrhage or pseudocyst formation [5]. Here, we report a case of nontyphoidal salmonellosis (NTS) infection, with fever and abdominal pain due to splenic infarction, which is a rare presentation of NTS.

CASE REPORT

A 22-year-old male presented to the emergency department with high-grade fever, vomiting, and abdominal pain for 5 days. Abdominal pain was dull aching and localized to the left hypochondrium. He did not have diarrhea, jaundice, cough, shortness of breath, urinary disturbances, or altered mentation and his past and family history was insignificant.

On examination, he was febrile with a temperature of 102°F, pulse rate of 108/min, blood pressure of 130/80 mmHg, and respiratory rate of 24/min. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy, or edema. He had a tender, palpable, soft liver of 3 cm. Tenderness was elicitable in left hypochondrium with a soft, palpable spleen of 2 cm. There was no evidence of free fluid in the abdomen. Rest of the systemic examination was normal.

Laboratory reports revealed hemoglobin of 13.7 gram%, total white blood cell count of 4900/mm³ with differential counts of N56/L33/M10/E1/B0 and normal peripheral smear, and platelet count of 1.7 lakh/mm³. Urine routine microscopy was normal. His liver function tests showed total serum bilirubin of 0.9, aspartate aminotransferase of 118, alanine aminotransferase of 134, alkaline phosphatase of 167, total serum protein of 6.8, and albumin/globulin ratio of 3.8/3.

Other biochemical parameters were normal, fasting blood sugar 98, blood urea 26, serum creatinine 0.9, sodium 130, potassium 4, and chloride 101. Blood Widal test showed titers of T0 ≥ 1/320, TH ≥ 1/320, and AH < 1/80. Smear for malarial parasite and rapid malarial kit test were negative. Immunoglobulin M leptospirosis, dengue tests, hepatitis markers (A/B/C/D), human immunodeficiency virus (HIV) ELISA, and antinuclear antibody tests were also negative. His chest X-ray and echocardiography were normal.

Abdominal ultrasound, followed by contrast computerized tomography of the abdomen (Fig. 1), showed mild hepatomegaly and multiple wedge-shaped hypodense areas in spleen suggestive of splenic infarcts. Multiple mesenteric necrotic lymph nodes were also seen. There was no evidence of abscess.

Coagulation workup was planned but could not be done. Blood culture revealed the growth of NTS species sensitive to cephalosporins and cotrimoxazole. The patient was initially treated with ceftriaxone; however, due to poor clinical response, antibiotic therapy was upgraded to meropenem 1 g 8 h and continued for 14 days. The patient responded very well to treatment; his condition was afebrile and abdominal pain settled. Repeat blood culture was sterile and liver function tests normalized in 10 days (Table 1). The follow-up, 1 month later, revealed no abnormality on imaging of the abdomen, with complete resolution of splenic infarcts.

DISCUSSION

Splenic infarction due to typhoidal and NTS infection is not common. NTS-related splenic infarcts are a rarity with only a few reports in the medical literature [6]. Salmonellosis can result in splenic infarction as

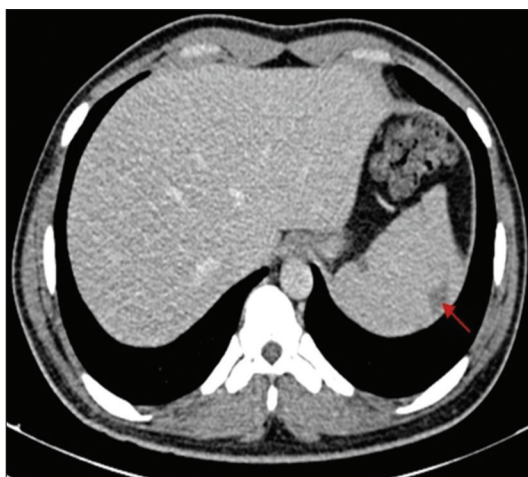


Figure 1: Computed tomography abdomen - areas of infarction in the spleen

Table 1: Clinical differences between enteric fever and NTS

Parameters	Enteric fever	NTS infection
Incubation period	14 days	6–12 h
Symptom duration	3 weeks	10 days
Clinical features	Fever, abdominal pain, rash, hepatosplenomegaly, nausea, vomiting, and cough	Mild gastroenteritis with watery stool passage, invasive disease in immunocompromised

NTS: Nontyphoidal salmonellosis

a part of extraintestinal complication. Various possible mechanisms are postulated such as rouleaux formation by red blood cells, infective embolization, low oxygen tension, and massive splenomegaly with relative insufficiency of blood supply [1].

NTS has a wide range of hosts and a strong association with agricultural products [7]. Less than 95% of NTS infections are foodborne. In contrast to typhoid fever, which is caused by *Salmonella typhi* or paratyphi infection, NTS transmission can also occur from person to person contact, contact with pets, and consumption of contaminated produce. Nosocomial infections and sporadic transmission due to undercooked eggs, infected transovarially from chicken, are also reported [7].

NTS infection in humans usually induces mild gastroenteritis, but in up to 5% of cases, invasive extraintestinal disease occurs. Some NTS serovars such as typhimurium, dublin, and choleraesuis have more potential for extraintestinal disease. Invasive disease occurs due to bacteremia and can cause complications such as meningitis, septic arthritis, osteomyelitis, cholangitis, and infectious endarteritis [8]. Malnutrition and coinfection with HIV or malaria are risk factors for invasive disease [9,10]. Diabetes mellitus and gastric hypoacidity can also increase the risk of NTS infection [10]. Long-term carriage of NTS is not described as with typhoidal species. NTS infection differs from typhoid in various ways [11].

Kupeli *et al.* reported a case of isolated splenic infarction caused by Group B salmonella in 2002 [6]. A similar case, caused by *Salmonella enteritidis*, was reported by Gupta and Kakar in 2004 [1]. NTS species are identified by direct plating of fecal samples and inoculation of standard enrichment broths [12]. Antibiotic therapy is indicated if the patient is severely ill or at risk for extraintestinal spread. Antibiotic treatment may prolong, rather than limit, the fecal shedding of organisms; hence, may be avoided in healthy patients with mild-to-moderate infection [13]. Fluoroquinolones, cotrimoxazole, ampicillin, or third-generation cephalosporins are used for the duration of 3–7 days. In case of invasive disease, single bactericidal drug therapy needs to be extended for 10–14 days. Splenic abscess may require surgery; treatment duration is 2 weeks if surgery is done and 4–6 weeks if there is bone or joint involvement [14]. Many strains of NTS species have the emergence of multidrug resistance and azithromycin may be effective in these cases [15]. There are no clinical data, suggesting that combination therapy is superior to single agent.

CONCLUSION

Splenic infarction is uncommon, but, in the setting of infectious disease process such as typhoid or paratyphoid fever, a high index

of suspicion is warranted when patient develops abdominal pain and/or tender splenomegaly. Splenic infarcts are easily confirmed radiologically and respond to conservative management with antibiotics, rarely requiring surgery.

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