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Isolated Ruptured Tuberculous Splenic Abscess in a Diabetic Patient

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ABSTRACT

Tuberculosis should be considered as a diagnostic possibility in patients presenting with pyrexia of unknown origin and splenomegaly; especially, in areas where the disease is prevalent. Splenic involvement is usually seen as a part of milliary form of tuberculosis. Isolated splenic abscess is an extremely rare form of solid organ tuberculosis and is mostly limited to immune compromised patients. This report describes the occurrence of isolated ruptured tuberculous splenic abscess in a man with poorly controlled diabetes mellitus who was successfully managed by splenectomy along with the anti tuberculous treatment.

Keywords: Tuberculosis, Diabetes Mellitus, Splenic Abscess, Splenectomy Anti tuberculous therapy.

Tuberculosis is primarily a disease of the respiratory system. Extra-pulmonary involvement accounts for 15% of all patients with tuberculosis [1]. Abdominal viscera involvement is observed in only 11% cases of extra-pulmonary tuberculosis [2]. Involvement of spleen in the form of tuberculous splenic abscess (TSA) is usually encountered as a part of milliary tuberculosis and it rarely manifests as an isolated entity [1,2]. Detailed online search of English literature revealed very few reported instances of TSA [3-6]. Here in, we report a case of isolated TSA with intra peritoneal rupture in a patient having longstanding history of chronic alcoholism and uncontrolled diabetes mellitus.

CASE REPORT

A 48-year-old immune-competent gentleman presented with dull aching left upper abdominal pain accompanied by intermittent low grade fever and malaise of 20 days duration. The patient was diagnosed with type II diabetes mellitus four years back; however, the glycemic control was inadequate with the prescribed oral hypoglycemic agents. The patient was addicted to alcohol for last 15 years. There was no history of chronic cough, chest pain, breathlessness, jaundice, and anorexia. The patient denied any history of trauma to the abdomen and having contact with patients suffering from tuberculosis. The patient was febrile to touch, pale, and anicteric. The pulse rate was 80 per minute; blood pressure was 110/70 mmHg. Abdominal palpation revealed tender, firm, splenomegaly (5 cm below the left costal margin). Overt signs of peritoneal irritation on the left upper abdomen were absent. Respiratory and cardiovascular system examinations were normal.

On investigation, his hemoglobin was 9.6 gm/dl; total leukocyte count was 15200/mm^3, and ESR was 35 mm in 1^{st} hour. Evaluation for human immunodeficiency virus, hepatitis B virus, amoebiasis, typhoid fever, malaria and sickle cell trait were non-contributory. Liver function and renal function tests were within normal range. Blood culture was reported to be sterile. A provisional clinical diagnosis of splenic abscess was made and the patient was administered intravenous antibiotics (combination of 3^{rd} generation cephalosporin and aminoglycoside) along with oral antipyretics and analgesics. Adequate glycemic control was achieved with initiation of intensive insulin

therapy. A single dose of polyvalent pneumococcal vaccine was also administered.

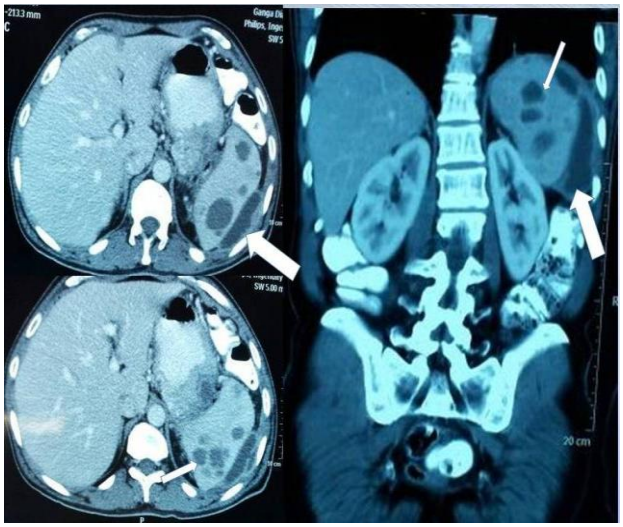

Figure - 1 A & B) Axial section of contrast enhanced computer tomography of abdomen showing multiple hypo-attenuating masses of varying size within the spleen (small arrow), with hypo-attenuated area in the perisplenic area (large arrow); 1C) Coronal section showing multiple hypo-attenuating mass of varying size within the spleen (small arrow), with hypo-attenuated area in the perisplenic area (large arrow).

The chest X-ray PA view was normal. Ultrasonography of abdomen revealed multiple hypoechoic lesions in the spleen with the largest one measuring 31x30 mm. There was evidence of perisplenic fluid collection of size 7.3x2.7 cm (volume approximately 82cc). Contrast enhanced computed tomography of abdomen demonstrated multiple hypodense, non-enhancing lesions, largest of size 30x35 mm at the lower pole of spleen with hypodense collection of size 7.5x2.8 cm between the spleen and diaphragm (**Fig. 1 A, B and C**). All other abdominal viscera were normal.

In view of persistent fever unresponsive to the prescribed antipyretics/antibiotics over a period of seven days, coupled with the findings of ruptured splenic abscess on imaging, decision for exploratory laparotomy was made. There was a large collection of thick, white, odorless pus in the left subdiaphragmatic space adjacent to the postero-superior surface of spleen. The pus collection was drained and splenectomy was carried out. The splenectomy specimen on histopathology revealed large areas of caseous necrosis with epitheloid cells at periphery indicative of tuberculous infection. No well-defined granulomas were identified. Ziehl-Neelson (ZN) stain for acid-fast bacilli (AFB) was negative.

The patient was advised daily combination antituberculous treatment (isoniazid 5 mg/kg, rifampicin 10 mg/kg, ethambutol20 mg/kg, and pyrazinamide 25 mg/kg) for two months followed by four months therapy of isoniazid and rifampicin in the postoperative period. Strict glycemic control was maintained with fixed dose insulin therapy and the patient was counseled for complete abstinence from alcohol. He was symptom free on follow up at six months following the surgical intervention.

DISCUSSION

The most common cause of splenic abscess is metastatic infection in the form of infective endocarditis [7]. Other potential causes are typhoid, malaria, infections in contiguous areas (pancreatitis, retroperitoneal/subphrenic abscess, and diverticulitis) and secondary infection of splenic infarction [7]. Tuberculous splenic abscess is usually encountered in patients having co-morbidities such as neoplasm, immunodeficiency, trauma, alcoholism and diabetes [2]. Our patient was immune-competent but the associated poorly controlled diabetes and chronic alcoholism were the predisposing factors for development of TSA. The more frequently encountered pyogenic splenic abscess presents with high grade fever and prominent localized manifestations like left upper abdominal tenderness, guarding, tender enlarged spleen, and pleuritic chest pain [7]. In contrast, the diagnosis of TSA is difficult because of vague local signs mimicking malignancy and fungal infection [1,2].

Roentgenogram can contribute to the diagnosis of pyogenic splenic abscess by revealing presence of left sided pleural effusion, left lower lobe atelectasis, elevated left hemidiaphragm, abnormal displacement of gastric shadow or extra-alimentary gas in left upper quadrant; however, these findings are considered nonspecific for a definitive diagnosis [8]. The chest roentgenogram was unremarkable in our case. Untreated, splenic abscess can rupture into peritoneal cavity, pleural space and bowel. Peritoneal rupture may presents as acute abdomen with free air confined to left upper quadrant on imaging [9]. The signs of peritonitis were not evident in our case, as pus was confined to the perisplenic area limited by left triangular, lenorenal and phrenecocolic ligaments.

In retrospect, ideal approach in our case would have been USG or CECT-guided aspiration of pus to confirm the etiological diagnosis. The tubercle bacilli are scarcely

identified on staining in patients with extra-pulmonary tuberculosis; thus, sole reliance on the staining methods to diagnose this form of tuberculosis is questionable [10]. Further, organic solvents used for making paraffin-embedded tissue samples, might reduce the affinity of mycobacteria for the acid fast stain (0.5% with the fluorescent method and 0.2% in ZN staining) [10]. This was reaffirmed in our case as the pus was negative for AFB and the diagnosis was confirmed only on histopathological examination. In solitary TSA, the bacilli are scanty; so, conventional and real time polymerase chain reaction test would have been a useful investigation for detection of tubercle bacilli but this test has a very limited penetration in the resource poor, developing countries. In Indian scenario, most of the cases have been diagnosed using staining and culture techniques, and clinical improvement following start of empirical anti tuberculous therapy (ATT) is often taken as an indicator of the tuberculous pathology.

The best treatment modality for splenic abscesses is still debatable. It ranges from antibiotics/ ATT, percutaneous drainage (67–100% success rate), and splenectomy [10,11]. Anti tuberculous treatment alone is considered effective, but few authors have showed suboptimal response to ATT without splenectomy [12,13]. Percutaneous catheter drainage is the treatment of choice for candidates unfit for surgical intervention. Splenectomy is a preferred option especially in patients with ruptured abscess to obtain source control along with control of gross peritoneal contamination. The current literature supports laparoscopic splenectomy as a safe and effective minimal invasive procedure in patients with splenic abscess avoiding unnecessary laparotomy [10,11]. The presence of a ruptured splenic abscess and lack of adequate expertise in laparoscopic splenectomy were the compelling reasons for contemplation of open splenectomy in this patient.

CONCLUSION

We believe that a detailed work-up is mandatory for all patients with splenic abscess, as effective treatment of the underlying cause is essential for complete recovery. Combination of ATT and splenectomy is of proven benefit in patients with ruptured TSA through rapid control of source of infection and arresting the progression of disease process.

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