

Splenic Abscess of an Unknown Etiology

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ABSTRACT

Splenic abscess is a rare condition, detected in individuals with certain predisposing factors that lead to abscess formation. This is a case report of a 20-year female presenting with high grade fever, weight loss, and left hypochondrial pain. She had an abdominal mass, that was reported as enlarged spleen with multiple abscesses on CT scan. Her laboratory analysis revealed neutrophil leukocytosis with a raised erythrocyte sedimentation rate (ESR) and normal peripheral smear, while her blood culture, brucella and viral serologies, transesophageal echo, antinuclear antibody (ANA) profile and hypercoagulability work up, were negative. Hence, no predisposing factor could be found as the cause of splenic abscess. She was successfully treated with intravenous antibiotics and recovered.

Key Words: Abscess, Splenectomy, Leukocytosis, Hypercoagulability.

INTRODUCTION

Splenic abscess is a rarely diagnosed disease, although the evolution of newer diagnostic techniques has increased its frequency. There are many predisposing factors leading to abscess formation, particularly an increase in the number of immunocompromised individuals, such as human immunodeficiency virus (HIV) infection, bacteremia secondary to trauma, or embolisation in infective endocarditis. It can occur from the spread of a local focus of infection. The triad of fever, left hypochondriac pain, and leukocytosis lead to the diagnosis. Here is a case of a young and immuno-competent female found to have splenic abscess clinically, which was confirmed by CT scan abdomen. No etiology found, despite extensive work up. She was successfully treated by intravenous antibiotics only. Splenectomy or any other intervention was not required, as she showed clinical and radiological improvement.

CASE REPORT

This is a case report of a 20-year female presented with 2 months history of high grade intermittent fever, associated with rigors, chills and severe left hypochondrial pain, nausea and decreased appetite. There was significant, but undocumented weight loss. On examination, she was a thin and lean built female, conscious, obviously pale and was hemodynamically stable. There was a tender epigastric mass extending up to left hypochondrium, giving the impression of tender splenomegaly. There was no peripheral lymphadenopathy and no murmur was audible on cardiac auscultation.

Keeping in mind the differential diagnosis of splenic abscess, infective endocarditis, hematological malignancies, lymphoproliferative disorder and autoimmune disorder, her laboratory workup was done that showed white blood cell count (WBC) of $11.5 \times 10^9/l$, hemoglobin (Hb) 7 g/dl, hematocrit 24, mean corpuscular volume (MCV) 77 fl, neutrophils 84% and platelet count $432 \times 10^9/l$, and international normalised ratio (INR) 1.1. Malarial parasites or sickle cells were not seen in the peripheral film. Serum creatinine was 0.55 mg/dl and electrolytes were within normal limits. Her transferrin saturation was 5.8%, erythrocyte sedimentation rate (ESR) was 92 mm, TSH was 2.07 iu/ml. LFTs showed total bilirubin of 1.24 mg/dl (direct=0.65, indirect=0.59), SGPT 15U/L, alkaline phosphatase 130, gamma GT 69U/L, and SGOT 14U/L. Her urine data of analysis and chest X-ray were normal. HbsAg and anti-HCV and HIV serology were non-reactive. Her CT scan abdomen showed enlarged spleen with multiple abscesses, the largest one measuring 3.1x2.9x6.8 cm. Two elongated areas of splenic infarcts were also seen. Splenic vein was dilated with partial thrombus in its lumen (Figures 1a and 1b).

Her antinuclear antibody (ANA) was sent, keeping in view an autoimmune cause, that came out to be negative. Her blood and urine cultures were negative and transthoracic echocardiogram did not show any pathology. Brucella serology was also negative. While she was being investigated for the causative factors, she was started on intravenous (IV) antibiotics (Inj. meropenem 1 gm iv three times a day (TDS) and Inj. metronidazole 500 mg iv TDS) and iron supplements, as she was hemodynamically stable, with no indication for urgent intervention at that time. Meanwhile, patient was kept on close follow-up. After 14 days of treatment on follow-up, there was significant improvement. Her fever and abdominal pain were settled and appetite had improved. CT scan showed spleen of normal size, with resolution of abscesses (the largest one measuring 2.2x1.0x4.4 cm, smaller abscesses showed complete

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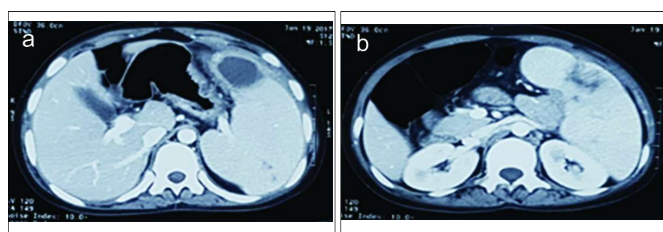


Figure 1 (a,b): CT scan showing multiple splenic abscesses (largest one measuring 3.1x2.9x6.8 cm) with dilated splenic vein.

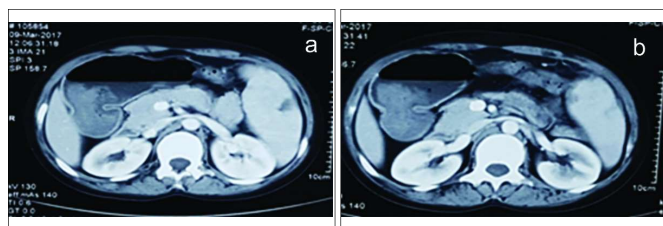


Figure 2 (a,b): CT scan showing resolution of abscesses, the largest one measuring 2.2x1.0x4.4 cm, and smaller abscesses showing complete resolution. Splenic vein is normal with resolution of thrombus.

resolution). Splenic vein was not dilated and thrombus in it showed resolution (Figures 2a and 2b). Repeat ESR was 15 mm per 1st hour. She is on regular follow-up with three-monthly ultrasound scan of abdomen, and she is clinically and symptomatically better.

DISCUSSION

Splenic abscess is seldom reported, with an estimated frequency of 0.05-0.7%, with a documented evidence of 600 cases in literature.^{1,2} It usually results from bacteremia secondary to trauma, embolisation, or hemoglobinopathy. Infective endocarditis causing systemic embolisation, can lead to splenic abscess formation in 10 - 20% of cases.³ Immunodeficiency is an important risk factor, as about 18 - 34% of patients with splenic abscess are immunocompromised (from disease, cancer chemotherapy, steroids use or HIV infection).⁴ It can also be a sequelae of a local focus of infection like abdominal abscess or diverticulitis. However, some systemic infections like enteric fever, malaria, pneumonia, urinary tract infection, and osteomyelitis can also lead to the formation of splenic abscess. The causative agents of splenic abscess are gram-positive cocci (*Staphylococcus aureus* and *Streptococcus pyogenes*) and gram-negative bacilli (*Klebsiella pneumoniae*, *Escherichia coli*, *Pseudomonas species*, *Salmonella species*, and *Proteus*). In 50% of cases, *Staphylococcus aureus* is the primary organism with splenic embolisation and abscess formation.⁵ Splenic infarction resulting from hemoglobinopathies or vasculitis can become infected and lead to abscess formation.⁶ The median age for initial diagnosis of splenic abscess is 40-50 years with no gender predominance.¹ The clinical triad of splenic abscess includes fever along with left upper abdominal pain and leukocytosis in more than 60% of cases.⁷ Ultrasound scan, with a sensitivity of 76%, can detect larger

abscesses.⁴ CT scan is the gold standard for diagnosis with a sensitivity and specificity of more than 90%.⁴ There is classical appearance of splenic abscess as a hypodense lesion on CT scan. The complications include rupture of abscess either in peritoneal or pleural cavity resulting in peritonitis with a guarded prognosis.⁴

The treatment of choice is total splenectomy along with appropriate antibiotic therapy, although treatment with antibiotics alone has been done successfully,⁸ as in this patient who was treated with antibiotics without any intervention. The administration of pneumococcal vaccine and antimicrobial prophylaxis before splenectomy can decline the mortality rate due to infectious causes.⁹ CT guided percutaneous drainage is considered as a safe and effective alternative to surgery, and should be considered as the first line of treatment, particularly in younger age group.¹⁰ Hemorrhage is the most common complication. However, pneumothorax, pleural effusion, and colonic injury have also been encountered. The indications for splenectomy include multilocular abscesses, fungal abscesses, infected hematomas, abscesses with thick contents and abscesses unresponsive to percutaneous drainage.¹⁰ The mortality rate can reach up to 100% without prompt treatment. However, with appropriate management, mortality can be decreased to <10%.⁸

In conclusion, an early and accurate diagnosis and prompt treatment can lead to dramatic response in splenic abscess, even of unknown etiology.

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