CASE REPORT

Successful CT Guided Needle Aspiration of Splenic Abscess: A Case Report

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Abstract

Splenic abcess is a rare clinical entity with an incidence of 0.2 to 0.7 % in autopsy based studies. Untreated, a splenic abscess is associated with nearly 100% mortality. An 44 year old male was admitted with fever and fatigue for three weeks and abdominal pain for two weeks was found to have large splenic abscesses on imaging studies. Extensive workup revealed no evidence of hematogenous spread from an endovascular source, no septic emboli in other organs and no proof of local spread from an infectious origin within the abdomen.Our case introduces an unusal presentation of splenic abscess without well described patho-physiologic mechanism to support their etiology.

Key Words

Splenic Abcess, Non immune compromised, Splenectomy

Introduction

Splenic abcess is a rare clinical entity with an incidence of 0.2 to 0.7 % in autopsy based studies. Untreated, a splenic abscess is associated with nearly 100% mortality. A splenic abscess warrants prompt institution of therapy. Splenectomy with antibiotics is considered to be the treatment of choice. Recent trends in the management of a splenic abscess have evolved successful techniques which avoid a splenectomy. Splenic abscesses are typically associated with bacterial endocarditis or a contiguous abdominal infection (1-6). In this case, our patient was diagnosed with splenic abscesses without having an identifiable source of infection. To our knowledge, this is one of the rare reported cases of spontaneous splenic abscesses in a non immune compromised host.

Case Report

An 44 year old male presented to emergency department with a three week history of fever upto 38.5°C and generalized weakness two weeks. There was history of pain abdomen in left hypochondrium radiating to back increasing on deep breath and movement on that side. Apart from a slight left pleuritic chest pain on deep inspiration, he reported no other additional symptoms. He denied cough, headache, dysuria or rash. Review of systems was otherwise negative. His past medical history included diabetes mellitus. He had no history of previous surgeries and no history of recent trauma. He did not smoke or drink alcohol and his medications included metformin, and glimpride. Examination revealed febrile patient with tenderness over left lower chest and left hypochondrium with spleen tip palpable.

Laboratory tests were significant for WBC of,6700/mm3 with 68% neutrophils, Hct 29.1% and haemoglobin 9.7mg/dl, blood glucose 155mg/dl, urea 96mmol/L, creatinine 2.26mg/dl, AST 192mg/dl ALT 171mg/dl, ALP 22mg/l, LDH 267mg/dl, HbA1c 9.1% and CRP 90mg/dl. Prothrombin time was 16 sec and the ultrasound findings showed spleen enlarged in size (12.4cm) and showed ill definied subcapsular area of mixed echogenicity near the superior pole 4.5x2.5x3cm in size , another small hypoechoic area is also seen within the spleen. The presence of splenic lesion was confirmed with computed (CT) which showed multiple splenic abcess with hepatosplenomegaly and left sided pleural effusion and basal atelectasis (*Fig-1-6*). A transthoracic echocardiogram was negative for vegetations or significant valvular disease and considering the pathophysiology of splenic abscesses. Multiple blood cultures taken before and after broad spectrum

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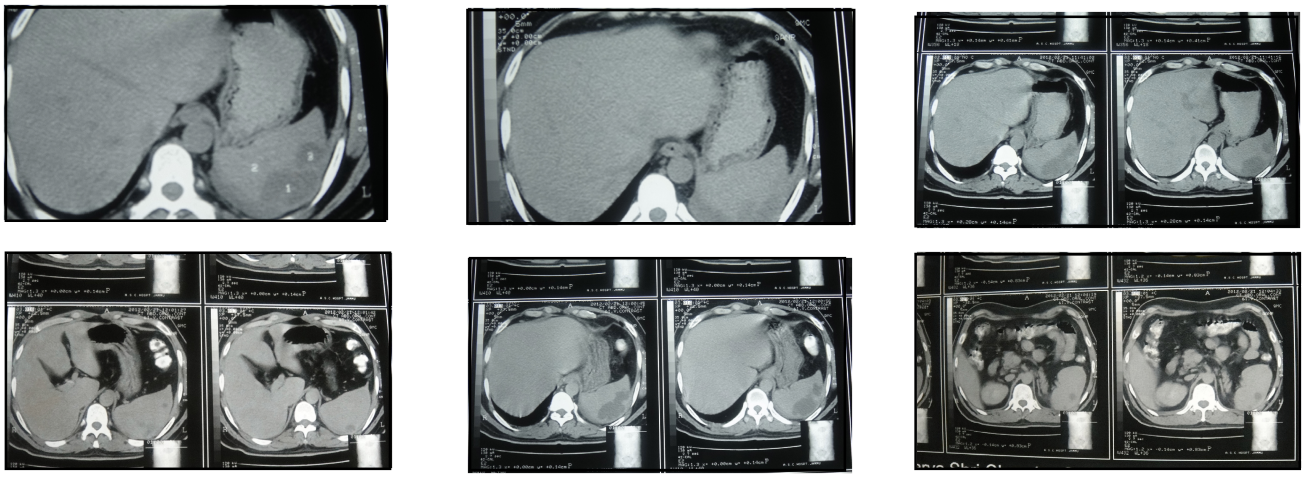

Fig 1-6. CT Showing Multiple Splenic Abcess with Hepatosplenomegaly & left sided Pleural Effusion & Basal Atelectasis

antimicrobial therapy was initiated, were negative. Serologic tests for,salmonella and HIV were negative. The patient remained hemodynamically stable but continued to have low grade fever despite improvement in his WBC count. Percutaneous computed tomography(CT) guided aspiration of about 50 cc of splenic abcess was done and was sent for culture and sensitivity which showed no growth after 48hours and AFB, gram staining was negative, total luecocyte count was 1000 with neutrophils 95%. A repeat aspiration was carried out on day 3 and about 25 cc pus was taken out. The patient receieved Inj. Meropenem and Metronidazole for ten days and was discharged subsequently. The splenic abscess has not recurred during 3 months of follow-up.

Discussion

The clinical signs and symptoms associated with splenic abscesses are non-specific and fever, chills, constitutional symptoms and abdominal pain (located in left upper quadrant or generalized) (7-14). Palpable splenomegaly or palpable abdominal mass was reported in only half of the patients in two case series (7,11). Leukocytosis is the most common laboratory finding (8).Therefore, in our case, the patient's clinical presentation was in accordance to the atypically described previously.

Moreover, although he was not typically immunosuppressed, he had diabetes mellitus which has been implicated as a major predisposing factor in two case series, occurring in half of the patients with splenic abscess(7,8). Although it is well known that diabetes mellitus has several compromising effects on the immune system ,it is not considered to cause profound immunosuppression as occurs in hematologic malignancies, high dose corticosteroid or immune suppressive therapy.

The leading micorganisms cultured from splenic abscesses are staphylococci, streptococci and gram negative aerobic bacteria (15). In a retrospective study of the microbiology of splenic abscesses involving56 isolates, aerobic bacteria were found in onethird of cases, an aerobic one third and one third of the cultures were mixed, while the rest were due to candida albicans. In the same study, the most common aerobic bacteria were Escherichia coli (E.coli), Staphylococus aureus, Streptococcus group D, Klebsiella pneumoniae and Proteus mirabilis. Peptostreptococcus species were the predominant anaerobes, followed by Bacteroides spp, Fusobacterium spp and Clostridiumspp (13). Polymicrobial etiology has been reported at approximately 50% of splenic abscess cultures (11,13). Some bacteria were related to certain predisposing conditions. Staphylococcus aureus, Streptococcus group D and Klebsiella pneumoniae were associated with endocarditis, E. coli with infections of the urinary or gastrointestinal tract, Bacteroides spp. and Clostridium spp. With abdominal infections, Fusobacterium spp with respiratory tract infections and Candida albicans with cancer and/or chemotherapy (13). Other microorganisms that have been implicated in primary splenic abscesses are Mycobacterium tuberculosis, Salmonella and Brucella (4,7,8). In two case series from Taiwan the most common

etiologic agent was Klebsiella pneumoniae (7,80 although in both these studies there was a high rate of either contiguous abdominal infection or concomitant hepatic abscess. In a European study of 22 patients, the most common etiologic agent was Mycobacterium tuberculosis and all tuberculosis cases had AIDS (4). Thus it is worth mentioning that the microbiology of splenic abscesses differs according to the geographic location and the patient population characteristics of each center. Negative cultures of the splenic aspirate have been reported in 30% of cases (1,7) possibly due to the effect of antimicrobial treatment or the challenge to culture anaerobic or fastidious micro organisms. Blood culture positivity ranged between30% and70% in multiple case series (7,11,13,15, 16).

It is unclear as to what the pathophysiologic mechanism of the splenic abscesses was in our patient. One potential explanation is that transient bacteremia, from a gastrointestinal or urinary source resulted in invasive disease and the development of splenic abscesses while effective antibiotic therapy lead to sterile cultures from the operative specimens. The fact that our patient had more than one abscesses supports the route of hematogenous spread. However, transthoracic echocardiogram failed to show evidence of large left atrial thrombus.

The obvious question in our case was whether he invasive candidemia that the patient had during his second admission to the hospital was related to the initial splenic abscesses. Nevertheless, both blood and the abscess fluid cultures were negative for time his primary admission during which he had not received any antifungals.

Conclusion

We present a rare case of spontaneous splenic abscess in a diabetic patient without other predisposing factors for profound immunosuppression. Although the causative agent was not identified, the patient was treated with empiric antibiotic therapy and aspiration of abcess. Splenic abscess is a life threatening condition hence prompt diagnosis and early initiation of appropriate treatment are key to the success full management to this disease. The availability of new techniques for early diagnosis and easier therapeutic options for the management of splenic abscesses have improved the overall outlook of patients. Our results suggest that CT-guided percutaneous drainage is a safe and effective alternative to surgery for the treatment of splenic abscess and allows preservation of the spleen.

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