

# Spontaneous abscesses of the spleen in an immunocompetent adult: Case report and review of the literature

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## ABSTRACT

**Introduction:** Splenic abscesses are typically associated with bacterial endocarditis or a contiguous abdominal infection. 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-immunocompromised host. **Case Report:** An 82-year-old male was admitted with fever and fatigue for two weeks and was found to have leukocytosis and two large splenic abscesses on imaging studies. Extensive work-up 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. **Conclusion:** Our case introduces an

unusual presentation of splenic abscesses without the well-described pathophysiologic mechanisms to support their etiology.

**Keywords:** Spleen, Splenic abscess, Abdominal abscess, Candida prostatitis

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## INTRODUCTION

A splenic abscess is a rather rare clinical entity, albeit with very high mortality, especially if left untreated. In an autopsy series, its incidence was 0.14-0.7% [1]. With the widespread use of advanced diagnostic imaging and with an increase in immunocompromised patients (chemotherapy, HIV/AIDS) the incidence of splenic abscesses seems to be on the rise [2-5].

There are three major pathophysiologic mechanisms for the development of splenic abscess [6-8]. The first one is via hematogenous spread and septic emboli to the spleen typically from bacterial endocarditis or another endovascular infection (infected pacemaker wire, mycotic aneurysm). The second mechanism is again related to bacteremia and hematogenous spread from a pyogenic infection but this time in the setting of disrupted splenic architecture, for example due to trauma or splenic infarcts (hemoglobinopathies, vasculitis). The third mechanism refers to contiguous

spread via fistula formation complicating an infection, trauma, perforation or malignancy of an adjacent organ (stomach, pancreas, colon) [9]. Splenic abscesses have also been reported after transcatheter arterial chemoembolization [8], and radiofrequency ablation [10].

Immunosuppression has been the leading predisposing factor in a number of case series [4, 11] and it was present in almost all cases of fungal splenic abscesses [1, 12]. To our knowledge, the presence of multiple spontaneous abscesses of the spleen in an otherwise immunocompetent adult is exceptionally uncommon [7].

## CASE REPORT

An 82-year-old male presented to our emergency department with a two week history of fever up to 38.5°C and generalized weakness. He was well until two weeks prior to admission when he started having fever for which he was treated empirically with oral antibiotics (names not known). The patient initially defervesced but the fever recurred when antibiotics were stopped. At that point he was prescribed clarithromycin empirically without resolution of fever and hence he was admitted to the internal medicine department. Apart from a slight left pleuritic chest pain on deep inspiration, he reported no other additional symptoms. He denied cough, abdominal pain, headache, dysuria or rash. Review of systems was otherwise negative. His past medical history included diabetes mellitus, hypertension, atrial fibrillation, dyslipidemia and hypothyroidism. He had no history of previous surgeries and no history of recent trauma. He was a retired grocery shop owner who lived almost all his life in Greece and had no history of recent travel outside the country. He did not smoke or drink alcohol and his medications included metformin, metoprolol, acenocoumarol, losartan, amlodipine, digitalis, spironolactone, glimepiride and omega-3 fatty acids.

On physical examination he was alert, oriented to self, time and place and not in distress. His blood pressure was 140/80 mmHg, heart rate 80 bpm and respiratory rate 20 breaths/min. There was no visible rash. Mucous membranes were mildly dry. His lungs were clear to auscultation, heart exam revealed irregularly irregular S1 and S2, without murmurs. Abdomen was soft, with mild tenderness at the epigastric area and without hepatosplenomegaly. He had no significant lower extremity edema and no lymphadenopathy. His neurological exam was grossly intact. Laboratory tests were significant for WBC of 33,600/mm<sup>3</sup> with 97% neutrophils, Hct-29.1% and Hb-9.7 mg/dl, blood glucose-155 mg/dl, urea-96 mmol/L, creatinine-2.26 mg/dl, AST-192 mg/dl, ALT-171 mg/dl, ALP-220 mg/dl, γGT-280 mg/dl, LDH-267 mg/dl, HbA1c-9.1% and CRP-90 mg/dl. Prothrombin time was 16 sec and INR 1.38 while on acenocoumarol. Chest X-

ray and urinalysis were within normal limits. Because of the paucity of signs and symptoms that could help localize a potential source of infection and the presence of abnormal liver enzymes, an ultrasound of the upper abdomen was performed. The ultrasound showed fatty infiltration of the liver and two large areas in the spleen, one target-like in appearance measuring 3.8 cm in diameter and another hypodense one measuring 9.5x4 cm.

The presence of splenic lesions was confirmed with computed tomography (CT) and magnetic resonance imaging (MRI) of the abdomen, both of which showed no other similar lesions in other organs. MRI of the abdomen showed two large cystic structures, one measuring 10 cm and the other five cm in diameter, which communicated with each other and did not sharply enhance after the administration of IV contrast (figure 1A-C). Their radiologic appearance matched the patient's clinical picture and the diagnosis of large splenic abscesses was made.

A transthoracic echocardiogram was negative for vegetations or significant valvular disease and it only revealed calcifications of the mitral and aortic valves. Considering the pathophysiology of splenic abscesses, our suspicion for bacterial endocarditis remained high and we proceeded to a transesophageal echocardiogram that also confirmed the absence of vegetations or abnormal valve function. Multiple blood cultures taken before and after broad-spectrum antimicrobial therapy was initiated, were negative. Serologic tests for brucella, salmonella and HIV were negative. The patient remained hemodynamically stable but continued to have low-grade fever despite improvement in his WBC count and CRP with antibiotic treatment. Although the possibility of percutaneous drainage was discussed in this patient, we elected to treat him with splenectomy weighing the risks and benefits of each procedure. After being vaccinated with the pneumococcal vaccine, he underwent splenectomy ten days after his admission while on antibiotics, namely piperacillin/tazobactam, which was later changed to ertapenem due to drug-induced cholestasis. The fluid taken peri-operatively from the splenic abscesses was grossly purulent, however both aerobic and anaerobic cultures were negative. The histopathologic findings of the spleen were consistent with two large abscess cavities without evidence of systemic disease or granulomas in the remaining parenchyma (figure 2).

Our patient tolerated the procedure well and he was discharged home ten days after surgery. However, ten days after his discharge, he was readmitted to our hospital with sepsis due to *Candida glabrata* fungemia. Upon his readmission the patient had urinary retention and significant prostate tenderness making the diagnosis of fungal prostatitis possible. *Candida glabrata* was isolated from the urine and the blood and imaging studies showed no new abscess formation in the upper or lower abdomen and pelvis.

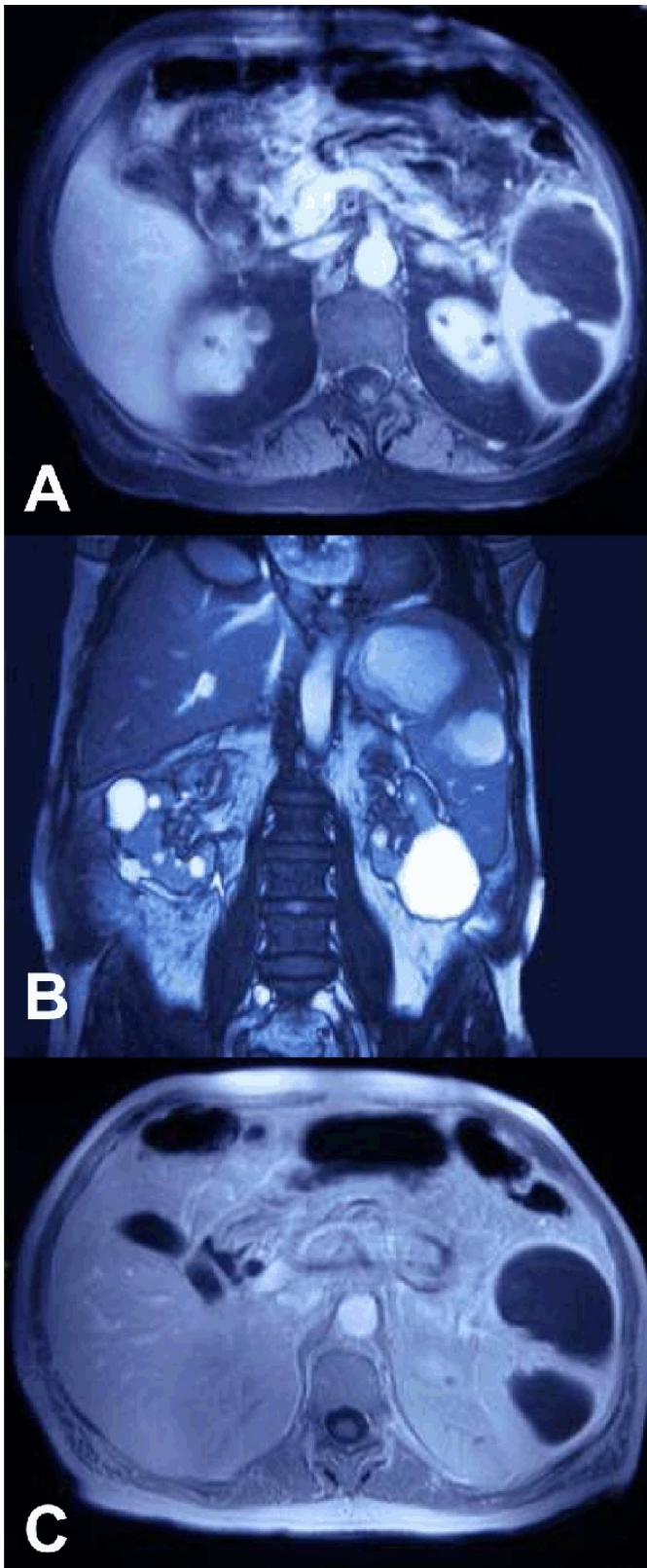


Figure 1: A-C) MRI of the abdomen depicting the two large communicating splenic abscesses.

He was treated with antifungals, his clinical condition improved significantly and he was discharged home two weeks later.



Figure 2: Photograph of the gross pathologic specimen of the spleen showing a large abscess cavity.

## DISCUSSION

The clinical signs and symptoms associated with splenic abscesses are non-specific and most frequently include fever, chills, constitutional symptoms and abdominal pain (located in left upper quadrant or generalized) [7, 8, 11]. 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 that typically 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 immunosuppressive therapy.

The leading microorganisms cultured from splenic abscesses are staphylococci, streptococci and gram-negative aerobic bacteria [15]. In a retrospective study of the microbiology of splenic abscesses involving 56 isolates, aerobic bacteria were found in one third of cases, anaerobic in 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*), *Staphylococcus aureus*, *Streptococcus* group D, *Klebsiella pneumoniae* and *Proteus mirabilis*. *Peptostreptococcus* species were the predominant anaerobes, followed by *Bacteroides* spp, *Fusobacterium* spp and *Clostridium* spp [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, 8] although in both those 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 14-30% of cases [1, 7] possibly due to the effect of antimicrobial treatment or the challenge to culture anaerobic or fastidious microorganisms. Blood culture positivity ranged between 30% and 70% in multiple case series [7, 11, 13, 15].

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. In addition, his INR being low on admission makes the presence of a left atrial thrombus possible, which in the setting of bacteremia, could have resulted in septic emboli to the spleen. However, both transthoracic and transesophageal echocardiogram failed to show evidence of large left atrial thrombus.

The obvious question in our case was whether the 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 yeast in his primary admission during which he had not received any antifungals. Given that our patient had many risk factors for fungemia (diabetes mellitus, abdominal surgery, splenectomy and the prolonged use of broad-spectrum antibiotics) it is most likely that this was a secondary complication, leaving the etiology of his splenic abscesses obscure.

## 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

splenectomy. Splenic abscess is a life threatening condition hence prompt diagnosis and early initiation of appropriate treatment are key to the successful management of this disease.

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## Author Contributions

Eirini Christaki – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Final approval of the version to be published

Apostolos Kabaroudis – Analysis and interpretation of data, Drafting the article, Final approval of the version to be published

Eleftherios Anagnostou – Acquisition of data, Drafting the article, Final approval of the version to be published

Vassilios G Athyros – Substantial contributions to conception and design, Drafting the article, Final approval of the version to be published

## Guarantor

The corresponding author is the guarantor of submission.

## Conflict of Interest

Authors declare no conflict of interest.

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