

The Rare Splenic Abscess

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Figure 1: Serosal surface of resected splenic abscess (formalin-fixed).



Figure 2: Multilocular cavities of a splenic abscess (formalin-fixed).

Clinical Image

Introduction

Abscesses of the spleen are relatively uncommon with an incidence of 0.2% [1]. Splenic abscesses have been reported periodically since the time of Hippocrates who postulated that the condition may take one of the following three courses; (i) the patient might die, (ii) the abscess might heal or (iii) the abscess might become chronic and the patient might live with the disease. He was essentially correct because the aetiology varies [1-4].

Case

We report a case of a 26-year old, farmer, with a 3-month history of a rapidly progressing left hypochondrial mass. This was associated with abdominal pain and intermittent fever. He had no history of trauma, infective endocarditis or tuberculosis and, was neither human immunodeficiency virus (HIV) positive nor diabetic. A full blood count revealed a low haemoglobin level (Hb 6.1 g/dl), a leucocytosis of $16.5 \times 10^9/l$, and, a normal platelet count of $307 \times 10^9/l$. Physical examination demonstrated a tender splenomegaly extending to the umbilicus (Hackett's 4). There was no associated lymphadenopathy and cardiovascular examination was normal. An abdominal ultrasound scan suggested a splenic abscess. Following the transfusion of 2 units of whole blood, he underwent a difficult splenectomy of a large necrotic fluid-filled spleen which was densely adherent to the tail

of the pancreas, inferior surface of the liver and greater curve of the stomach (Figure 1). Macroscopically, the resected spleen was large, multiloculated with total parenchymal destruction (Figure 2). He made good recovery and was discharged a week later after receiving vaccines against the encapsulated bacterial organisms, *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Neisseria meningitidis* that may cause an overwhelming post splenectomy infection (OPSI).

Discussion

Splenic abscesses are commonly caused by septic emboli from complications of infective endocarditis in about 5% of these patients, with the pathogens being *Streptococcus* or *Staphylococcus* [1]. It carries a very high mortality of greater than 70% if the diagnosis is missed, but with appropriate treatment the mortality can be reduced to less than 1%. With the availability of CT scan today the condition is rapidly diagnosed in addition to a potential treatment by aspiration [1-3]. The second common cause is a secondary infection of an infarcted spleen following trauma, interventional radiological embolization of splenic artery pseudoaneurysm complicating acute pancreatitis or a hemoglobinopathy such as sickle cell disease [1,3,5]. Other risk factors include immunocompromised states with 80% mortality, diabetes mellitus, illicit intravenous drug use in which splenic abscesses occur from a contiguous focus of infection. In these cases, the organisms commonly associated are polymicrobial (>50%), aerobes, anaerobes, fungi (usually candida) [1-4]. Splenic abscesses can also be associated with parasitic infection of the spleen and miscellaneous rare organisms such as *Burkholderia*, *Mycobacterium* and *Actinomyces* [3]. The common signs and symptoms include the triad of fever, left upper quadrant tenderness and leukocytosis [1,3,6]. The definitive treatment is splenectomy as most of the spleen is affected (Figure 2) plus pneumococcal, haemophilus influenzae type b and meningococcal conjugate vaccinations against the life-time risk (0.1-0.5%) but 50% mortality from a subsequent OPSI [7]. Percutaneous drainage is less likely to be successful in patients with multilocular abscesses, ill-defined cavities with necrotic debris and thick viscous fluid. Mortality rates of greater than 50% is reported in patients managed with antibiotics only [1,3].

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