Splenic Abscess: Etiology, clinical spectrum and Therapy

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ABSTRACT: Spleen abscess is a rare entity, only 600 cases reported in the literature, it has a high mortality in immunocompromised patients, and delay in diagnosis and treatment. Splenic abscess is more common in patients with endocarditis, immunocompromised and in patients with immunodeficiency virus infection. Common pathogens isolated include streptococcus spp, staphylococcus spp, mycobacteria, fungi, parasites, and Burkholderia pseudomallei isolates in melioidosis endemic regions. Computed tomography and ultrasonography guided percutaneous isolation in common in recent years. Medical treatment alone is insufficient. Splenectomy is considered to be a gold standard for splenic abscesses; different approaches based on abscess characteristics have shown success.

KEYWORDS: Splenic abscess, percutaneous drainage, splenectomy, and therapy

I. INTRODUCTION

The spleen is a highly vascular organ that is part of the reticuloendothelial arm of the immune system. If the spleen is surgically removed, its absence is marked by heightened susceptibility to overwhelming infection by capsulated bacteria and intraerythrocytic parasites. The abscesses of spleen usually result from bacteremia, particularly in the setting of abnormalities caused by trauma, embolization, or hemoglobinopathy. Immunodeficiency, such as that resulting from human immunodeficiency virus infection, is also risk factor [1]. Occasionally; splenic results from extension of a contiguous focus of infection [2]. Splenic abscesses are relatively uncommon. For example, in one series of 540 intra-abdominal abscesses, none were in the spleen [3]. Autopsy series have placed the incidence of splenic abscess at 0.2% to 0.07% [4,5]. Only approximately 600 have been reported in the literature [6]. There is a suggestion that incidence may be increasing because of improved detection, increase use of illicit intravenous drugs and the increased number of immunocompromised individuals [7,8]. Splenic abscess has a bimodal age distribution with peaks in the third and sixth decade of life [8]. Approximately two third of splenic abscesses in adults are solitary and one third are multiple. In children opposite holds true the majority are multiple and minority are solitary [9]. Mortality rates are high and vary with immune status and type of abscess; there is up to 80% mortality in immunocompromised patients with multilocular abscesses and 15% mortality in immunocompetent patients with unilocular abscesses [10,11]. Frequently isolated pathogens include strep to cococcus spp, staphylococcus spp, (due to endocarditis being the most common cause of splenic abscess), Mycobacteria, fungi, and parasites [2,12]. Researchers in Sabah, Malaysia reported Burkholderia pseudomallei being the common cause of splenic abscess in suitably predisposed individuals [13]. Early supportive care and parenteral broad spectrum antibiotics are of paramount importance while further diagnostic and therapeutic arrangements are made [14]. The gold standard for treatment of splenic abscess is splenectomy; however, recent studies have shown success using different approaches based on abscess characteristics [2,15]. With new medical advances in ultrasonography, computed tomography (CT), magnetic resonance imaging improved diagnosis and therapy has improved prognosis. The paper reviews the current literature, clinical presentation and therapy of splenic abscess.

II. ETIOLOGY AND INFECTIOUS AGENTS

Etiology. Splenic abscesses have diverse etiologies [16]. The most common is hematogenous spread originating from an infectious focus in the body. Infective endocarditis, a condition associated with systemic embolization in 22 to 50% of cases, has a 10 to 20% incidence of associated splenic abscess [17]. Other infective sources include typhoid paratyphoid, malaria, urinary tract infection, pneumonias, osteomyelitis, otitis media, mastoiditis, and pelvic infections. Pancreatic, other retroperitoneal and subphrenic abscesses, as well as diverticulitis, may contiguously involve the spleen. Splenic trauma is another well-recognized etiologic factor. Splenic infarction resulting from systemic disorders, such as hemoglobinopathies (especially sickle cell disease), leukemia, polycythemia, or vasculitis can become infected and evolve into splenic abscesses [14,18,19].

Infectious agents. Streptococci, staphylococci, salmonella, and Escherchia coli have been the major causative agents of splenic abscess for the past century. However, with the increased number of immunocompromised
patients, recent series have showed greater numbers of fungal isolates, including Candida albicans, Aspergillus spp, and agents of mucormycosis. Mycobacteria have also become more common. Anaerobic bacteria remain a relatively infrequent cause of splenic abscess compared with other intra-abdominal abscesses, despite improvements in culture techniques [2].

In HIV-infected patients, Salmonella spp, and Mycobacterium tuberculosis are common causes of splenic abscess, as are the opportunistic pathogens. Mycobacterium avium complex, Leishmania spp, Rhodococcus equi, and Pneumocystis jirovecii. Sickle cell anemia has classically been associated with Salmonella infections of the spleen, but more recent series noted a predominance of staphylococcus infection associated with this condition [8]. Many other organisms have been described as causative agents of splenic abscess at the case report level, including Bartonella henselae, Strep to bacillis monili form is and Nocardia spp [20,5]. In a series from Thailand, the agent of melioidosis, Burkholderia pseudomallei was the cause of splenic abscess in 24 of 41 cases from which a pathogen was isolated [21]. Overall blood cultures were positive in 24% to 60% of cases. Occasionally, aseptic abscess have been described in the spleen, particularly in patients with inflammatory bowel disease [22,23]. Patients with these apparently noninfectious inflammatory lesions of unknown etiology present with fever and respond to treatment with corticosteroids but not to antibiotics. They are often accompanied by aseptic abscesses, but the spleen is the most frequent site [2].

III. PATHOPHYSIOLOGY

Bacteremic infection from a variety of sites is the most common cause of splenic abscess. Classically, infective endocarditis has been most strongly associated with splenic abscess, and in most series, endocarditis is identified as the leading cause [23]. Other common sources of infection are the urinary tract, surgical wounds, and the gastrointestinal tract. Immunodeficiency has become a more important risk factor in the development of splenic abscess. In large reviews, 18% to 34% of patients were immunocompromised (from disease, cancer chemotherapy, or steroids use), including as many as 9% who were infected with human immunodeficiency virus [24]. Trauma to the spleen, either iatrogenic or accidental, accounts for 7% to 30% of cases, with lower numbers in more recent studies, and contiguous spread of infection (e.g. From an adjacent intra-abdominal process) continues to account for a small percentage of cases (2% to 7%) [24]. Other conditions associated with splenic abscess include splenic abnormalities such as Felty’s syndrome or amyloidosis, inflammatory bowel disease, hemoglobinopathy, and diabetes mellitus [2].

Complications of splenic abscess can be life threatening and include perforation into peritoneum which occurred in 19(6.6%) of 287 patients in recent series [24]. Rupture into adjacent organs can occur, with resulting fistulas into the gastrointestinal tract, the pleural space, or lung parenchyma. Overall mortality rates 0% to 14% have been reported with appropriate therapy, although higher rates occur among immunocompromised patients [2].

IV. CLINICAL SPECTRUM

Abscesses of the spleen have been reported periodically since the time of Hippocrates. He postulated that 1 of 3 courses was followed by a patient with splenic abscess: (1) the patient may die; (2) the abscess may heal; or (3) the abscess might become chronic and patient may live with the disease [25]. Splenic abscess is a rare entity, with a reported frequency of 0.05 to 0.7% its reported mortality rate is still high, up to 47% and can potentially reach 100% among patient who do not receive antibiotic treatment [18]. Appropriate management can decrease the mortality to less than 10% [26].

The history and physical examination are not sufficiently reliable to make the diagnosis of splenic abscess. However, information derived from the history and physical examination can suggest the diagnosis. Therefore, the clinicians must maintain a high index of suspicion, particularly higher risk clinical scenarios that include infective endocarditis, conditions associated with systemic embolization, pneumonia, osteomyelitis, mastoiditis and pelvic infections [12].

The signs and symptoms of splenic abscess have been well described but are not specific. Therefore, splenic abscess remains a subclinical diagnostic challenge. The classical triad of fever, upper quadrant pain, and splenomegaly is seen in only about one third of patients. The symptoms of splenic abscess can be variable and depend on the location, size, and progression of the process. They can also be acute, subacute, or chronic. Deep-seated, small abscesses and accompanied by symptoms [27]. Fever may be the only manifestation of splenic abscess, and fever is present in as many as 95% of cases. Another frequent finding is abdominal pain, which is either generalized or localized to the left upper quadrant and may radiate to left chest or shoulder. Nausea, vomiting, anorexia, and weakness are often present [2]. Abdominal tenderness is present in only half of cases, most often in left upper quadrant. Splenomegaly can be detected in a similar number of cases. Chest finding, including dullness, ales or both at the left base, have been detected in many patients. Other findings, less frequently present. Include splenic friction rubs, hepatomegaly, tenderness at costovertebral angle, and ascites.
Splenic Abscess: Etiology, clinical spectrum and Therapy

The only laboratory abnormality that is frequently present is leukocytosis, which is seen in 60% to 80% of cases [2].

Physical examination. (1) Abdominal tenderness (>50%) may or may not be accompanied by muscle guarding in the left upper quadrant. There may be edema of soft tissues overlying the spleen. Costovertebral tenderness may also be noted. (2) Splenomegaly (<50%) is less frequently observed, probably because of early diagnosis resulting from the widespread use of imaging methods. (3) Chest findings are nonspecific and reportedly include dullness at the left lung base (>30%), left basilar rales (>21%), or elevation of the left hemidiaphragm [28].

V. DIAGNOSIS

Diagnosis of splenic abscess remains a diagnostic challenge, because the symptoms and findings of splenic abscess are most frequently nonspecific, diagnosis depends on appropriate imaging studies. Plain radiographs are surprisingly sensitive, with abnormalities detected in 50% to 80% of chest films and 25% of abdominal films (e.g., basilar infiltrates, pleural effusion, elevated hemidiaphragm, shift of viscera, presence of gas), however the findings are most often nonspecific. The use of radionuclide scans are described in earlier literature, primarily technetium 99m sulfur-colloid liver spleen scans, has largely been supplanted by ultrasonography, computed tomography and magnetic resonance imaging [29].

Ultrasonography. The advantages of ultrasonography in the evaluation of splenic abscess include low cost, portability, and relatively high sensitivity, reportedly ranging from 75% to 93% [8,24,29,30]. It is therefore appropriate in the initial assessment of most suspected splenic abscesses. Ultrasonography typically demonstrates an area of decreased or absent echogenicity, sometimes with regular areas of echodensity (debris) or gas pattern within the lesion [31]. Splenomegaly can frequently be demonstrated. High resolution (7.5 MHz) ultrasonography can detect micro-abscesses in patients infected with human immunodeficiency virus that are missed with conventional ultrasonography [1].

Computed Tomography (CT scan). Computed tomography appears to be the single most sensitive modality for the detection of splenic abscess, particularly if enhancement by intravenous contrast is used [30]. A sensitivity of greater than 90% has been seen in most series [24]. The abscess is seen as an area of low-density fluid or necrotic tissue within the relatively homogenous spleen. Enhancement of the rim of the abscess cavity is seen in a minority of cases, so that splenic infarction are difficult to distinguish from splenic abscesses on contrast-enhanced computed tomography [24].

Magnetic Resonance Imaging (MRI). There is far less experience with magnetic imaging for the detection of splenic abscesses, and its role in the management is not yet established. Magnetic resonance imaging is sensitive for detection of other abdominal abscesses, and its sensitivity in the discovery of splenic abscesses is probably similar. Nonetheless, although magnetic resonance imaging has been successfully coupled with drainage procedures, it would appear to be more cumbersome and to offer fewer advantages than ultrasonography or computed tomography [32]. In the setting in which drainage may not be necessary, for example in immunocompromised patients in whom pathogens other than pyogenic bacteria are more common or if a noninfectious diagnosis such as malignancy or cyst is likely, fine-needle aspiration under the diagnostic imaging may be useful [33,34]. The sensitivity of this method is variable, and its role remains the subject of investigation [2].

VI. THERAPY

Untreated splenic abscess has a high mortality [8]. Splenectomy has been the traditional modality for treatment and remains the gold standard against which other therapies must be assessed. Antibiotics play an important role in the treatment of associated endocarditis and sepsis, in stabilization of the patient for splenectomy or a drainage procedure, and in treatment of selected pathogens (e.g., mycobacteria, fungi), but they are rarely curative alone for splenic abscess caused by pyogenic bacteria. Broad spectrum empirical therapy should be initiated as soon as splenic abscess is suspected, pending surgical or percutaneous drainage. Antibiotics should include agents against treptococci, staphylococci, and anaerobic gram negative bacilli, Vancomycin or oxacillin plus and aminoglycoside, a third or fourth generation cephalosporin, a fluoroquinolone, or a carbapenem would be reasonable empirical therapy. After blood or abscess culture results are obtained antibiotic coverage can be narrowed accordingly. If splenectomy is the possibility, it is advisable to administer vaccination for encapsulated bacterial pathogens as early as possible [2].

Experience with computed tomography and ultrasonography guided percutaneous aspiration of splenic abscesses has accumulated in recent decades. These procedures have advantage of lower initial morbidity and mortality than splenectomy and allow preservation of spleen. Success rates have ranged from 50% to 90% in several series [8,24,35,36]. In general, smaller (<3 to 4 cm), solitary, or uniloculate abscesses have larger rate of
successful percutaneous drainage in contrast, micro abscesses, as well as complex, phlegm nous or multicellular processes and those with thick fluid, tend to fare more poorly with this approach[2].

Percutaneous drainage may be useful at least initially, in those patients who are unstable or who present an unacceptably high surgical risk. Failure to achieve effective drainage or failure of the patient’s condition to improve is an indication for definitive surgery. Aspirated abscess fluid should be sent for Gram staining, bacterial, fungal and mycobacterial culture; and other studies as clinically indicated [2].

Some authorities advocate contraindications to percutaneous drainage include(a)multiloculated or debris-filled abscess(b)multiple small abscesses(c)uncontrollable coagulopathy(d)poorly defined abscess on CT scan or ultra-sonogram(e)diffuse ascites(f)not safe route for drainage[37,38,39].

The optimal duration of antibiotic therapy for splenic abscess has not been established in any clinical trial. For example patients, such as those with bacterial endocarditis, the duration is dictated by the underlying condition. If splenectomy is performed and the focus of infection is eradicated, briefer durations may be possible. With percutaneous drainage, duration of therapy needs to be tailored to the clinical course, including resolution of the abscess as assessed by the diagnostic imaging [2].

VII. CONCLUSION

The gold standard for treatment of splenic abscesses is a splenectomy; however, recent studies have shown success using different approaches based on abscess characteristics.

REFERENCES


