

CASE REPORT

Isolated splenic abscess in brucellosis

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Abstract

Brucellosis is a zoonotic infectious disease, which can attack any organ of the body but mainly involves lymphoreticular system. Our case report describes isolated splenic abscess diagnosed in a 50-year-old individual who is a milk vendor by occupation and has the habit of consuming raw milk. He was admitted with pain abdomen, high-grade fever with chills, generalized malaise, night sweats and weight loss. Ultrasound and computerized tomography of the abdomen showed splenomegaly and hypodense, nonenhancing lesion measuring 3.2 × 2.8 × 2.8 cm. Brucella slide and tube agglutination tests (Wright, at 1/640) were positive. The patient was successfully treated with percutaneous drainage along with oral doxycycline (200 mg/day) and rifampin (600 mg/day) for 6 weeks. A high index of suspicion is required for early detection, prompt treatment and prevention of complications of brucellosis, especially in endemic areas. Patients with fever of unknown origin should be evaluated for brucellosis.

INTRODUCTION

Brucellosis is a systemic zoonotic infectious disease caused by Gram-negative bacilli of the genus 'Brucellais'. It is also known as Bang's disease, Crimean fever, Gibraltar fever, Malta fever, Maltese fever, Mediterranean fever, rock fever or undulant fever. It is the most common zoonotic infection globally. A common cause of Brucellosis is due to consumption of unpasteurized milk and contact with infected animals. It can affect any organ in the body. Involvement of liver and spleen may be attributed to intracellular survival and replication of bacteria in the mononuclear-phagocytic system. Isolated splenic abscess due to Brucellosis is extremely rare and is fatal if not adequately treated [1]. The reported frequency of splenic abscess is 0.05–0.7%.

Main causes of splenic abscesses include (i) infection: anaerobes are the most common infectious agent [2]. It may be either metastatic infection or continuous infection such as the perinephric abscess or infected pancreatitis. (ii) Splenic infarction and superimposed infection. (iii) Trauma. (iv) Immunocompromised state (chemotherapy/transplant recipients, leukemia and AIDS). Clinical presentation of splenic abscess can be vague and non-specific with pyrexia being common. Other clinical features include left upper quadrant tenderness with rigors, chills and vomiting.

CASE REPORT

In June 2014, a 50-year-old man was brought to the emergency room with pain abdomen, high-grade fever with chills, generalized

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malaise, night sweats and significant weight loss (>10 kg in 45 days). The patient is a milk vendor by occupation in rural Bangalore and routinely consumes unpasteurized or raw milk due to a belief that raw milk is healthier. The patient is an ex-smoker and alcoholic with consuming about 20 g (whiskey) per day from 10 years. Patient's medical records and history from relatives revealed fever with night sweats and pain abdomen from 2 months for which he was evaluated as fever of unknown origin. The patient denied any recent travel or contact with persons having a similar illness.

His physical examination during admission was remarkable for fever (40°C), hypotension (BP: 86/60 mmHg), and increased respiratory (30 min⁻¹) and heart (120 min⁻¹) rates. He was severely dehydrated and pale, but no scleral icterus, clubbing, cyanosis, edema or lymphadenopathy was noted. Systemic examination of the abdomen showed tenderness in the left hypochondriac region. The liver was nontender with normal span. The spleen was not palpable but castell's sign was elicited (dullness noted in lower eighth intercostal space on inspiration). Cardiovascular and respiratory system examination was normal. Nervous system examination was normal.

Investigations revealed total white blood cell count of 32 100 mm⁻³ (neutrophils 64%, lymphocytes 30%, eosinophils 3% and monocytes 3%), ESR of 100 mm/h, C-reactive protein of 92 mg/l, hemoglobin of 10 mg/dl and platelet count was 199 000 μm⁻¹. The arterial blood gasses had pH of 7.36, a PO₂ of 68 mmHg, PCO₂ of 35.5 mmHg and 17.9 mmol/l of bicarbonate. Renal function test was abnormal with creatinine of 2.0 mg/dl and blood urea of 70 mg/dl. Liver function test was normal. Electrocardiogram showed tachycardia and non-specific ST-T changes in leads 2 and aVF. Echocardiography was normal with the ejection fraction of 60% and no features of endocarditis were found.

Brucella slide and tube agglutination tests (Wright, at 1/640) were positive. Ultrasound abdomen and computerized tomography of the abdomen showed splenomegaly and well-demarcated hypodense lesion, which is nonenhancing and measuring 3.2 × 2.8 × 2.8 cm in the upper pole of spleen with perisplenic fat stranding. Blood cultures confirmed infection by *Brucella melitensis* on Day 1 and Day 3. Chest radiograph showed bilateral pleural effusion with basal atelectasis. Upper gastrointestinal (GI) endoscopy was normal. The patient was treated with percutaneous drainage along with oral doxycycline (200 mg/day) and rifampin (600 mg/day) for 6 weeks.

The patient improved symptomatically over a period of 10 days with the absence of fever and reduction of pain abdomen. The patient was discharged on Day 10 with advice for review ultrasound abdomen examinations. The patient remained asymptomatic without recurrence at 6 months after completion

of treatment. Repeat ultrasound abdomen showed mild splenomegaly without any other abnormalities.

DISCUSSION

Brucellae are aerobic Gram-negative coccobacilli that can infect any organ in the body because of a unique ability to invade phagocytic and nonphagocytic cells and intracellular survival avoiding the immune system. Infection occurs through bacterial exposure to the skin, mucous membranes, conjunctivae, respiratory and GI tracts. Intracellular survival occurs due to lipopolysaccharide coat (smooth in *B melitensis*, *B abortus* and *B suis*; rough in *B canis*), which produces adenine and guanine monophosphate inhibiting oxidative damage and phagosomal fusion. Brucellae are less effective inducers of inflammatory cytokines (tumor necrosis factor and interferons) and alternative pathway of complement system due to low pyrogenicity, virulence and toxicity.

Recovery from infection is through cell-mediated immunity causing the development of granulomas in sites of infection indistinguishable from sarcoidosis. Highly virulent species (*B melitensis* and *B suis*) result in visceral microabscesses. Elevated IgG titer persistence occurs in chronic or relapsed infection. *Brucella* species identified till now are illustrated in Table 1. *Brucella melitensis* is the most virulent and prevalent worldwide. It is acquired by exposure to animals or animal products or by handling specimens in the laboratory. Males are commonly affected than females, and prevalence varies from country to country. In up to one-third of cases focal infection may be found [3]. An isolated splenic abscess is an extremely uncommon presentation [4, 5]. The incidence of the splenic abscess was reported to be <2% in many large series [6].

Several studies suggest that preexisting tissue injury of spleen and bacteremia is the basis for the formation of an abscess [7, 8]. The scenarios include: (i) hematogenous embolization to a normal spleen. Examples include septic endocarditis due to intravenous drug abuse, patients on chemotherapy who develop fungemia. These patients are usually immunocompromised or suffer from an overwhelming bacteremia. (ii) Hematogenous spread with an altered splenic architecture. This group includes single (from trauma) or multiple splenic infarcts (sickle cell disease and vasculitis) and bacteremia from a concurrent infection (central line sepsis, pneumonia, cholecystitis, etc.). (iii) Contiguous spread, which includes direct involvement from gastric or colonic perforations, pancreatic abscess and sub-phrenic abscess.

Organisms associated with splenic abscess include [9]: (i) aerobes—Gram-positive cocci (*Streptococcus*, *Staphylococcus* and *Enterococcus*); Gram-negative bacilli (*Klebsiella pneumoniae*,

Table 1: *Brucella* species and infections caused

Organism	Animal reservoir	Geographic distribution
<i>Brucella melitensis</i>	Goats, sheep, camels	Mediterranean, Asia, Latin America, parts of Africa and some southern European countries
<i>Brucella abortus</i>	Cows, buffalo, camels, yaks	Worldwide
<i>Brucella suis</i>	Pigs (biotype 1–3)	South America, Southeast Asia, United States
<i>Brucella canis</i>	Canines	Cosmopolitan
<i>Brucella ovis</i>	Sheep	No known human cases
<i>Brucella neotomae</i>	Rodents	Not known to cause human disease
<i>Brucella pinnipediae</i> and <i>Brucella cetaceae</i>	Marine animals, minke whales, dolphins, seals	Case reports describing some human cases (mainly neurobrucellosis)

Escherichia coli, *Pseudomonas*, *Proteus* and *Salmonella*). (ii) Anaerobes—*Peptostreptococcus*, *Bacteroides*, *Fusobacterium*, *Clostridium* and *Propionibacterium acnes*. Polymicrobial in up to 50% of cases. (iii) Fungi—*Candida*. (iv) Uncommon flora—*Burkholderia pseudomallei* (melioidosis), actinomycetes and mycobacteria (common in immunocompromised patients). Diabetic patients, alcoholics and immunosuppressed patients are susceptible to splenic abscesses.

According to a study published in 2009, the etiology of the splenic abscess was bacterial in 10 of 16 cases (62.5%), amebic in 2 (12.5%) and fungal in the remaining 4 (25%) [10]. A review of 22 cases of splenic abscess from single center showed that *Mycobacterium tuberculosis* was the frequent causative microorganism, found in 8 cases, and immunosuppression the main predisposing factor (in 63.6% of the patients) [11]. Rarely splenic abscess may be due to brucellosis [12].

In chronic brucellosis with hepatosplenic abscess treated with medical therapy alone, only 20–40% achieved a good response [13, 14]. Hence, such patients should be treated with both medical and surgical therapy to achieve the good response. Percutaneous drainage has reduced the need of splenectomy in the recent years. Prolonged medical treatment along with serial ultrasound examinations has been found to increase the chance of cure without the need for surgery. Duration of treatment should not be < 6 weeks.

In conclusion, splenic abscess caused due to brucellosis is very rare. High index of suspicion is required for early detection, prompt treatment and prevention of complications. In early stages, antibiotic treatment and percutaneous drainage is very effective and reduces need for surgery. Serial ultrasound examinations are needed to confirm complete cure. Patients presenting with fever of unknown origin should be evaluated for brucellosis and more specifically if patient resides in an endemic area. Patient education regarding the spread from raw milk, cheese and meat should be given importance.

CONFLICT OF INTEREST STATEMENT

None declared.

ETHICAL APPROVAL

Yes.

CONSENT

Yes.

GUARANTOR

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