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**Brucella: a rare causative agent of spontaneous bacterial peritonitis**

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We report a 54-year-old woman with hepatitis B-related chronic liver disease with ascites who developed spontaneous bacterial peritonitis. Blood and fluid cultures grew Brucella; the patient was working at an animal husbandry till one year ago. She responded to therapy with streptomycin and tetracycline. [Indian J Gastroenterol* 2003;22:190]

**Key words:** Ascites, Brucellosis, cirrhosis

Gram-negative enteric bacteria are the causative agents in more than 70% of cases of spontaneous bacterial peritonitis. We report a rare causative agent, Brucella, for this condition.

A 54-year-old woman was admitted with complaints of fever, chills, night sweats and abdominal distension. The patient had been well until one year earlier, when she noticed abdominal swelling. She had been working at an animal husbandry until one year ago. There was no history of tobacco or alcohol consumption. Physical examination revealed abdominal distension, hepatosplenomegaly and tense ascites.

**Investigations:** Hemoglobin 120 g/L, white cell count 4000 per mm³. ESR 70 mm in 1st hour. Urine was normal. Blood glucose level 4.8 mmol/L, urea 6.5 mmol/L, bilirubin 55 mmol/L, AST 101 IU/L, ALT 24 IU/L, LDH 414 IU, total proteins 71 g/L and albumin 31 g/L. Partial thromboplastin time 22 s, prothrombin time 18 s. Chest X-ray showed cardiomegaly.

Peritoneal fluid showed glucose 0.9 mmol/L, total proteins 23 g/L, LDH 112 IU/L, 1400 cells per mm³ with 94% mononuclear cells; no organisms were found on microscopy. Ultrasonography showed hepatomegaly with heterogeneous echogenicity, splenomegaly and intra-abdominal free fluid. Serological tests (enzyme immunoassay) showed HBsAg positive, anti-HBs antibody negative, anti-HBe antibody positive, anti-HIV antibody positive, IgG anti-HBe antibody positive, negative. Brucella serum agglutination test was positive (titer 1:1280). Blood and peritoneal fluid cultures for Brucella were positive. Liver histology showed chronic hepatitis (Knodell activity score 13/18 with severe fibrosis).

She was treated with intramuscular streptomycin (1 g/day), tetracycline (500 mg q5h) and oral spiramycin (100 mg/6h). Her symptoms disappeared in two weeks. No relapse was observed subsequently in a 3-month follow-up period.

Brucellosis is a zoonosis and generally is derived from infected animal and animal products. It is a systemic infection that can involve many organs and tissues. Brucellosis always affects the liver, but gastrointestinal complications are very rare. We found only two reports of peritonitis caused by Brucella in ascitic fluid due to chronic liver disease.

Oral tetracycline for 6 weeks in combination with intramuscular streptomycin for the first 3 weeks are the drugs of choice. We used this regimen successfully.

**References**


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**Abdominal wall abscess secondary to subcapsular tubercular liver abscess**

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We report a 22-year-old woman who presented with an abdominal wall lump in the right upper quadrant 15 days after starting antitubercular treatment for right pleural effusion. CT scan revealed a right liver lobe subcapsular abscess communicating with sub-
cutaneous tissue. Aspiration of pus revealed acid-fast bacilli. She responded to 9 months of antitubercular treatment. [Indian J Gastroenterol 2003;22:190-191]

Key words: Hepatic tuberculosis

Abdominal wall cold abscesses are commonly a result of tracking of a spinal or paraspinal tubercular lesion. We report a patient with abdominal wall cold abscess secondary to subcutaneous tracking of subcapsular tubercular liver abscess.

A 22-year-old woman presented with right lower chest pain, breathlessness, low-grade fever and anorexia of 3 months' duration. Chest X-ray showed right-sided pleural effusion. On pleural fluid analysis, she was diagnosed to have tuberculous pleural effusion and was started on 4-drug therapy.

After 15 days she started experiencing localized pain in the right upper quadrant of the abdomen, which was intermittent and throbbing. She continued to have fever and anorexia, but chest symptoms had decreased. General examination revealed mild pallor. Abdominal examination revealed a firm, nontender, globular mass, 3 cm x 3 cm, in the abdominal wall in the right hypochondrium. Liver was palpable 2 cm below the right costal margin. There was no splenomegaly or free fluid. Chest examination revealed right-sided pleural effusion.

Investigations: hemoglobin 10.9 g/dL, white cell count 9400 cells/mm^3 (P 70%, L 30%), ESR 75 mm in 1st hour, normal liver profile, serum electrolytes and blood sugar; HBsAg and HIV negative. Ultrasonography revealed an inhomogeneous hypodense lesion in the oblique muscles, suggestive of abdominal wall cold abscess. CT scan revealed mild hepatomegaly with a septated subcapsular abscess, 5.5 cm x 2 cm x 6 cm, anterior to the right lobe of the liver, and a similar 2 cm x 2 cm lesion anterior to the left lobe. The right lobe abscess was extending into subcutaneous tissues (Fig). There was no ascites or retroperitoneal lymphadenopathy. Ultrasound-guided aspiration of pus demonstrated acid-fast bacilli.

The patient was continued on the 4-drug regimen. After 3 months, since ultrasonography showed no decrease in the size of the abscess, percutaneous guided needle aspiration was done. On completion of nine months of treatment, the patient improved symptomatically and gained 3 Kg weight; the abdominal wall mass disappeared. Ultrasonography showed complete resolution of the abscess, with normal liver.

Focal liver involvement such as abscess secondary to tuberculosis is rare probably because of the relatively low tissue oxygen tension in the liver. Tubercular liver abscess is even more rare and to the best of our knowledge this is only the second report in indexed literature. Development of tuberculous abscess has been reported in patients on antitubercular drugs.

Complications described secondary to tubercular liver abscess include duodenal fistula and bronchobiliary and gastrobiliary fistulae. Our patient had abdominal wall abscess, which has been described secondary to paraspinal / spinal tuberculosis but not with tuberculous liver abscess. Antitubercular drugs remain the mainstay of therapy. Some authors recommend transcatheter infusion of these drugs into the abscess cavity. Percutaneous transcatheter or surgical drainage may be required in large abscesses.

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Micro pneumatosis coexistent with Helicobacter pylori and its improvement

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Micro pneumatosis intestinalis is the occurrence of gas-filled circular voids with diameter of 20-200 microns, not lined with epithelium. We report a 39-year-old man with superficial gastritis and Helicobacter pylori infection who also had gastric, duodenal and colonic micro pneumatosis. Endoscopic biopsy after treatment for H. pylori gastritis showed no micro pneumatosis in gastric, duodenal or colonic mucosal sections. We suggest that H. pylori may be one of the causative factors for micro pneumatosis. [Indian J Gastroenterol 2003;22:191-192]

Key words: Pseudolipomatosis

Fig: CT scan showing right lobe subcapsular liver abscess tracking into subcutaneous tissues.