There are few reports of skeletal infections in patients with cirrhosis. We present two such cases, both with alcoholic liver disease, seen over a period of one year. The first, a 46-year-old man, presented as pyrexia of unknown origin, and was found to have pyogenic discitis; he responded to antibiotic and surgery. The second, a 42-year-old man, presented with chest wall abscess and was diagnosed to have tubercular osteomyelitis; he expired despite treatment with non-hepatotoxic anti-tubercular drugs.

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Students with liver cirrhosis are well known to be prone to infections. These are a source of considerable morbidity and often mortality, and hence need prompt and aggressive treatment. Skeletal infections are an often-unrecognized group of infections in patients with liver cirrhosis. We present two such cases seen over a period of one year.

Case 1: A 46-year-old man with alcohol-related liver cirrhosis with portal hypertension and diabetes mellitus, presented with fever off and on for 4 months. There was no history of cough, dysuria, headache, or joint pains. Evaluation done outside was inconclusive. On admission he was pale, febrile, hemodynamically stable; physical examination was otherwise unremarkable.

Investigations: hemoglobin 7.6 g/dL, white cell count 3,800/cmm, platelets 91,000/cmm, ESR 70 mm in 1st hour, serum bilirubin 1.2 mg/dL (direct 0.6), AST 27 U/L, ALT 16 U/L, serum protein 6.4 g/dL (albumin 2.4), INR 1.7. Renal function tests and electrolytes were normal. Routine examination of urine was normal and culture was sterile. Chest X-ray was normal. Blood culture showed E. coli bacteremia. ELISA for HIV was negative. Echocardiography did not reveal any vegetations. He was treated with culture-sensitive antibiotics and discharged after the fever subsided.

He was readmitted with fever after one month. This time he gave history of severe low backache increased on prolonged sitting or walking. He had numbness of both lower limbs. He was found to have weakness of extensor hallucis longus and dorsal flexors of right lower limb. MRI spine showed disk protrusion at L4, with narrowing of neural foramen and lateral recess (Fig). He underwent disectomy. Intra-operatively the disk space was found to be infected. Culture of disk material showed E. coli. He was managed with culture-sensitive antibiotics and analgesics. However, as there was no relief in pain, re-explo- ration was done after 2 weeks and posterior lumbar intrabody fusion was done. He was discharged after one month and was doing well on follow up at 3 months.

Case 2: A 42-year-old man, previously diagnosed to have alcohol-related liver cirrhosis with portal hypertension, presented with upper GI bleed and right-sided chest wall abscess. On examination he was found to be pale and febrile, with a large parietal swelling on the right side of chest.

Investigations: hemoglobin 6 g/dL, white cell count 32,000/ cmm, serum bilirubin 8.6 mg/dL (direct 3.8), AST 84 U/L, ALT 49 U/L, alkaline phosphatase 64 U/L, serum protein 7 g/dL (albumin 1.6). ELISA for HIV was negative. Ultrasonography (USG) showed shrunken liver with coarse echotexture, splenomegaly and gross ascites. Diagnostic paracentesis of ascitic fluid did not suggest spontaneous bacterial peritonitis. USG chest showed a 19 cm x 8 cm heterogeneous cystic lesion with internal echoes abutting the right chest wall muscles and ribs, almost reaching up to the axilla, and right-sided pleural effusion. Pleural fluid aspirate showed 788 cells (64% polymorphonuclear), glucose 79 mg/dL, LDH 1430 U/L, and no AFB. Aspirate from the abscess was thick and hemorrhagic, and sterile on routine culture.

He was treated with parenteral broad-spectrum antibiotics, vitamin K, packed RBC, platelet, and plasma transfusions. As his coagulopathy worsened despite attempts at correction, incision and drainage of abscess was deferred on surgeon’s advice. However he continued to be febrile. CT chest showed...
a 15.6 cm x 13 cm x 6 cm multi-loculated chest wall abscess on the right side and right-sided pleural effusion; in addition there were osteomyelitis changes in the manubrium, axillary lymphadenopathy, and a small apical lung lesion. Considering the possibility of tubercular osteomyelitis and cold abscess he was started on non-hepatotoxic anti-tubercular drugs. However he developed progressive desaturation and hypotension needing mechanical ventilation and inotropic supports, and eventually expired.

Bacteremia in liver cirrhosis is well known, the risk appearing to increase with more advanced Child class. Escherichia coli is the most frequently isolated organism. Susceptibility to infection in cirrhosis is related to impaired defense mechanisms due to impaired reticuloendothelial function, portosystemic shunting, increased intestinal permeability. Recurrent bacteremia may also be due to bacterial factors that confer a survival advantage (e.g., adhesins that facilitate mucosal colonization).

Infections in atypical sites, often secondary to previous episodes of bacteremia, can often pose a diagnostic dilemma. Bacterial arthritis complicating cirrhosis has been described. Dental infections may also be a source of recurrent sepsis. However there are very few reports of skeletal infections in liver cirrhosis described in literature. They are often detected after repeated failed searches for source of infection. Co-morbidities like diabetes, alcohol abuse, and immunosuppression are factors likely to be correlated with mortality with such infections in cirrhotics.

A high index of suspicion can help in early recognition of skeletal infections as possible source of infection in cirrhotics that need appropriate and prolonged courses of antibiotics and other therapeutic interventions.

References

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Received November 3, 2004. Accepted December 22, 2004

Hepatitis-associated aplastic anemia: successful outcome following immunosuppressive therapy

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Hepatitis-associated aplastic anemia is an uncommon variant seen in young, previously healthy individuals. The pancytopenia follows hepatitis by a few weeks and is usually severe and prolonged. Bone marrow transplantation remains the cornerstone of therapy. However, immunosuppressive therapy has been found to be effective. We report an 8-year-old girl who had non-A, B, C and E hepatitis-associated severe aplastic anemia. She became transfusion-independent and had consistent, albeit incomplete recovery after immunosuppressive therapy with antithymocyte globulin and cyclosporine. [Indian J Gastroenterol 2005;24:175-176]

Aplastic anemia (AA) is a bone marrow failure syndrome, presumed to be of immune origin. In most cases, aberrant immune response is triggered by exposure to environmental agents such as drugs, toxins or chemicals. Aplasia is estimated to develop in less than 0.07% of all pediatric hepatitis cases. In most such cases, serological tests for hepatitis A, B and C viruses are negative. We describe our experience in one such patient.

An eight-year-old girl presented to us with history of spontaneous skin bleeds for one month. There was no history of pallor or fever. She had never received blood transfusion. She had history of jaundice three months back, which lasted for one month. Investigations done then revealed total serum bilirubin 24 mg/dL, (conjugated fraction 60%). Serum ALT and AST levels were 944 and 1056 U/L, respectively. Serum alkaline phosphatase level was 22 KAU/L (normal <15), respectively. Serum alkaline phosphatase level was 22 KAU/L (normal 3-13). Anti-HAV IgM, HBsAg, anti-HCV and anti-HEV IgM were negative. She was managed conservatively then, and recovered within one month.

On examination, she had moderate pallor and multiple skin bleeds. There was no icterus or adenopathy. Systemic examination was normal. Investigations: normocytic, normochromic anemia with hemoglobin 6 g/dL and corrected reticulocyte count 0.5%. Total leukocyte and absolute neutrophil counts were 1600 and 430 per mm3, respectively; platelet count was 10,000/mm3. Serum bilirubin was within normal limits; ALT and AST levels were 42 and 40 U/L, respectively, and alkaline phosphatase level was 22 KAU/L (normal 3-13). Anti-HAV IgM, HBsAg, anti-HCV and anti-HEV IgM were negative. She was managed conservatively then, and recovered within one month.

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