Myriad presentations of gall bladder tuberculosis

We would like to report our experience with myriad presentations of gall bladder tuberculosis.

Between 2001 and 2005 we managed five cases of gall bladder tuberculosis. All were women, aged 36-65 years (mean 53). Four patients presented with clinical features suggestive of chronic cholecystitis. One patient presented with a palpable gall bladder lump and systemic features such as weight loss and anorexia. Two patients had received antitubercular treatment for pulmonary tuberculosis 2 and 6 years prior to the current illness.

Hemogram and liver function tests were normal except for elevated ESR in all patients and hypoalbuminemia (serum albumin 2.8 g/dL; normal >3.5) in the patient presenting with gall bladder mass. Chest roentgenograms were normal in 3 patients and showed evidence of healed tuberculosis in 2 patients. Ultrasonography showed cholelithiasis and minimal wall thickening in 3 patients. One patient had diffuse thick-walled (8 mm) gall bladder with a small pericholecystic intrahepatic biloma. The fifth patient had a nodular mass in the gall bladder fundus, multiple gallstones and small pericoledochal lymph nodes. Dual-phase CT in this patient showed nodular mass arising from the fundus of gall bladder that was adherent to the duodenum, and small pericoledochal lymph nodes. There was no liver infiltration and the biliary tree and rest of the abdomen were normal. The preoperative diagnosis in four patients was chronic cholecystitis and carcinoma gall bladder in one.

Laparoscopic cholecystectomy was attempted in 4 patients. In three patients, it was successfully completed. In the fourth patient (with intrahepatic pericholecystic biloma) conversion to open procedure was required due to dense adhesions in the Callot’s triangle and cholecysto-duodenal fistula. Open cholecystectomy and repair of duodenal defect was done in this patient. Diagnostic laparoscopic evaluation of the general peritoneal cavity was normal in all four. The fifth patient underwent cholecystectomy.
with wedge resection of the gall bladder fossa. Intraoperative frozen section biopsy from the gall bladder mass, resected liver margin, pericholedochal lymph nodes and cystic duct stump were all negative for malignancy.

Diagnosis of tuberculosis was made in all 5 patients after histological examination. Typical granulomas were found in the gall bladder wall in all five patients. Stain for acid-fast bacilli was positive only in the patient with gall bladder mass. Caseating granulomas were also seen in the cystic lymph node in this patient.

All patients had uneventful recovery and were given multi-drug antitubercular treatment for 1 year. After a mean follow up of 22 months (range, 7-39) all patients are asymptomatic and disease-free.

Gall bladder tuberculosis is uncommon, with only 50 cases reported in literature till 2003. The intact gall bladder mucosa is resistant to Mycobacterium tuberculosis due to the presence of concentrated bile acids in the gall bladder lumen.1,2,3 The infection usually spreads via the hematogenous route, or from adjacent caseating lymph nodes or peritoneal tubercles.1,2,3 Cholelithiasis is associated in more than 70% of cases.3,4 In our series all patients had associated cholelithiasis. All patients in this series were women, which is similar to the experience of other authors.1,3 Clinical presentation is variable and includes features of chronic cholecystitis, carcinoma gall bladder and nonspecific systemic symptoms.1,2,3 Essop et al reported that liver enzymes were elevated in more than 60% of their patients with tuberculous involvement of liver.5 However, none of our patients had deranged liver enzymes. Sonographic and CT findings of gall bladder tuberculosis, such as an enlarged gall bladder, thickened gall bladder wall, soft-tissue masses, intrahepatic bilomas are nonspecific, and diagnosis is usually made on histological examination.1,4

Sorabh Kapoor, Ajit Sewkani, Saleem Naik, Sandesh Sharma, Aruna Jain,* Subodh Varshney
Departments of Surgical Gastroenterology and
*Pathology, Bhopal Memorial Hospital and Research Center, Raisen Bypass Road, Karond, Bhopal 462 038

References

Correspondence to: Dr. Kapoor, Assistant Professor. Fax: (755) 274 8309. E-mail: sorabhkapoor@gmail.com

G-6-phosphate dehydrogenase deficiency in patients with acute viral hepatitis presenting with severe hyperbilirubinemia

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is common in northern India, the incidence being variously reported as 2.2% to 14%.1,2 Although hyperbilirubinemia may occur in patients with severe hemolysis, it is usually mild (<4 mg/dL) and self-limited when liver function is normal.3,4 Similarly, in uncomplicated viral hepatitis, serum bilirubin levels of over 20 mg/dL are uncommon.2,4,5,6 Association of these two conditions may cause severe hyperbilirubinemia and occasionally protracted clinical course.6,7 Hemolytic anemia with hemoglobinuria as a complication of acute viral hepatitis may occur in normal patients, but is more common when the patient has G-6-PD deficiency.3,4,5,7,8 We studied deficiency of G-6-PD in patients with viral hepatitis with bilirubin levels >20 mg/dL in northern India.

Forty patients (mean [SD] age 35 [10] years, M:F 1.7:1) with acute viral hepatitis with marked hyperbilirubinemia (>20 mg/dL) were recruited from the medical outpatient department, excluding those who had history of blood loss, intake of oxidant drugs, any systemic immunological disease or chronic hemolytic state. Hemogram, liver biochemistry and serological markers for hepatotropic viruses (A, B, C and E) were tested. G-6-PD quantitative assay was performed by the modified spectrophotometric methods. Patients who had evidence of hemolysis were followed up for G-6-PD quantitative assay weekly for a period of 6 weeks, based on previous studies that showed that test for G-6-PD deficiency may be negative during and immediately after a hemolytic episode.3,7,8

G-6-PD values of all patients were the within normal range (146-376 U/1012 RBCs or 4.6-13.5 U/g hemoglobin) during the acute phase of the illness. Three cases (7.5%) developed severe hemolysis, characterized by marked anemia, reticulocytosis and findings on peripheral smear. These 3 patients had the highest bilirubin levels (>30 mg/dL).