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LETTER TO THE EDITOR

AN UNUSUAL CAUSE OF ACALCULOUS CHOLECYSTITIS DURING PREGNANCY: HEPATITIS A VIRUS

To the Editor:

Acalculous cholecystitis accounts for 5–10% of all acute cholecystitis cases and is usually a complication of various other medical or surgical conditions. Acute cholecystitis due to hepatitis A infection is quite a rare entity and hydropic and wall-thickened gallbladder and sludge formation have been described (1, 2). We present a 19-year-old pregnant woman with acalculous cholecystitis induced by hepatitis A infection.

The patient was initially admitted to the obstetrics clinic with complaints of nausea and malaise of 2 weeks duration. She was in the twelfth week of pregnancy and was discharged as the complaints pertained to pregnancy. One week after discharge she developed jaundice, fever, and severe right upper abdominal pain. On physical examination the patient was normal except for slight icterus and right upper quadrant tenderness (a positive Murphy's sign). Laboratory studies showed the following: white blood cell count, 4100; hematocrit, 35%; platelets, 215,000; alanine aminotransferase (ALT), 1213 U/L (normal range, 0–40); aspartate transaminase (AST), 984 U/L (0–40); alkaline phosphatase (ALP), 296 U/L (38–155); γ-glutamyltranspeptidase (GGT), 124 U/L (15–60); total bilirubin, 11.6 mg/dL (0.1–2.0); direct bilirubin, 5.7 mg/dL (0.1–0.8); albumin, 3.4 g/dL; globulin, 2.7 g/dL; C-reactive protein, 17.5 mg/L (0–5); erythrocyte sedimentation rate, 34 mm/hr; anti-HAV IgM, positive; HbsAg, negative; anti-HBc IgM, negative; and anti-HCV, negative. Abdominal ultrasonography revealed hepatomegaly, hydropic gallbladder with a thickened wall (8 mm), and 12-week intrauterine pregnancy. There was no calculus, pericholecystic, or intraabdominal fluid, however, a positive Murphy's sign induced by ultrasound probe was present. After hospitalization and strict bedrest, the patient's complaints regressed in the first week and her laboratory findings improved in the second week (ALT, 562 U/L; AST, 213 U/L; ALP, 194 U/L; GGT, 81 U/L). A month later, the patient was free of symptoms, biochemistry studies returned to normal, and the gallbladder was normal in sonographic examinations.

Our case had hepatitis A infection proved by serological and biochemical tests and her clinical acalculous cholecystitis resolved without any medical or surgical intervention. To the best of our knowledge, only four cases of acalculous cholecystitis induced by hepatitis A infection have been reported in the literature to date. Two of them demonstrated a similar clinical course with conservative management (3); whereas one underwent surgery (4) and the other died of acute fulminant hepatitis (5). Pregnancy may have been a predisposing factor for cholecystitis in the present case.

We recommend keeping hepatitis A infection in mind in the clinical approach to patients with acalculous cholecystitis.

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