Case Report

Emphysematous cholecystitis: A rare complication of hepatitis A virus infection

Ahmet Karakas, Cumhur Artuk, Ömer Coskun, Hanefi Cem Gür, Gürkan Mert, Can Polat Eyigun

Abstract

Hepatitis A virus (HAV) causes an acute hepatitis associated with significant elevation in liver enzymes. The liver is the main target of HAV, and extrahepatic manifestations are scarce. The diagnosis of acute HAV infection is made by the detection of anti-HAV IgM antibody in patients with the typical clinical presentation. Herein, we present a 22-year-old male presented with malaise, fever, nausea, and intensive stomach ache. In physical examination, the patient had fever, jaundice, and hepatomegaly. Laboratory tests resulted in elevated liver enzymes. Abdominal ultrasonography demonstrated thickening of the gallbladder wall, and intramural air. As the patient’s symptoms failed to regress in follow-up, laparoscopic cholecystectomy was performed. To sum up, HAV infection can be seen in all age groups, in developing countries, like our country. It must be kept in mind that although very rarely, HAV infections may exhibit extrahepatic complications.

Introduction

Acute hepatitis A virus (HAV) infection, a common viral infection, is seen especially in childhood in developing countries (1). Epidemiologic studies which recently conducted in Turkey have shown that the age of first encounter with HAV has shifted from childhood to the adult age group (2). HAV may cause a variety of clinical presentations from asymptomatic course to fulminant hepatitis. HAV infection is usually asymptomatic in children, however, symptomatic in adults, and commonly presents with complications. The major complications are cholestasis, relapse, prolonged hepatitis, and fulminant course. Extraphepatic findings are rarely seen in the course of HAV. Urticaria, hemolysis, acute renal failure, Guillain-Barre syndrome, pleural and pericardial effusion, acute reactive arthritis, pancreatitis, arthralgia, thrombocytopenic purpura, cryoglobulinemia, hemophagocytic syndrome, and acalculous cholecystitis comprise the previously described clinical presentations (1,3-5). These presentations usually resolve after the hepatitis subsides. During HAV infection, thickening of the gallbladder (GB) wall, and mud in the GB may be present (6). Acute acalculous cholecystitis, is a very rare presentation of HAV infection. There are very few reported cases in the literature (7-10). Emphysematous cholecystitis is a type of acute cholecystitis which involves the accumulation of gas independent of the gastrointestinal and biliary systems, in the GB lumen, wall, and pericholecystic tissues. This form is usually gangrenous, and unlike other cholecystitis types, is seen more frequently in men. The initial appearance of this entity usually includes an acute aseptic inflammation. Afterward, there is a secondary infection of gas forming bacilli. Herein, we present a previously unreported case of emphysematous cholecystitis which occurred due to HAV infection.

Case Report

A 22-year-old male who serving in army as a private deployed in Afghanistan presented to a primary
care center with fever, nausea, and vomiting and received a diagnosis of an upper respiratory tract infection and he was treated with antibiotics. 4 days after the onset of complaints, the patient’s urine color darkened (tea color), and jaundice in the sclera was noticed. The patient was referred to our clinic. At presentation, the patient had constipation and upper right quadrant pain of the abdomen. In physical examination, the patient looked exhausted. His fever was 38.5°C, pulse rate was 92 beats per minute and rhythmic. His arterial blood pressure and respiratory rate were 110/60 mmHg and 14 per min, respectively. His skin and sclera were jaundiced. In his abdominal examination, his liver crossed over the midclavicular line about 3 cm. There were significant pain and tenderness in the upper right quadrant. Because the patient’s oral and fluid intake was unwell, intravenous fluids were initiated. Furthermore, a lactulose suspension for the constipation was started.

In laboratory evaluation, the following findings were present; white blood cell count 11000/mm³ (40% neutrophil, 41% lymphocyte, 16% monocyte), hemoglobin 16.4 mg/dL, trombocyte 174.000/mm³, erythrocyte sedimentation rate 10 mm/h, aspartate aminotransferase (AST) 559 U/L (N:10-40 U/L), alanine aminotransferase (ALT) 1830 U/L (N:10-40 U/L), alkalen phosphatase (ALP) 357 U/L (N:38-155 U/L), gamma glutamiltransferase (GGT) 327 U/L (10-55 U/L), direct bilirubin 8,71 mg/dL (N:<0,2 mg/dL), total bilirubin 16,09 mg/dL (N:<0,2 mg/dL), total protein 6.39 g/dL, albumin 3.85 mg/dL, prothrombin time (PT) 17.4 s, INR (international normalized rate) 1.26, C-reactive protein <5 mg/L, bilirubin and urobilinogen in the urine (+++). Other laboratory tests and the patient’s chest X-ray were normal.

An abdominal ultrasonography (USG) was performed which revealed that the liver was 174 mm, with a Grade-1 diffusely increased in parenchyma echo. Furthermore, free fluid was present in the pelvic and perihedral space. The wall of the GB was measured as 14.6 mm (normal:<3 mm) (Figure 1). There was intramural air present in the wall of the GB. Also in the pericholecystic area, a reticular heterogenous hypechoic structure, 15 mm in diameter was observed. The spleen was normal in size. In the light of these findings, a diagnosis of emphysematous cholecystitis was established, and ampicillin-sulbactam was initiated empirically by the intravenous route.

In serological evaluations that were conducted HBsAg, anti-HBc IgM, anti-HBc IgG, anti-HCV, anti-HIV, anti-CMV IgM, anti-HSV IgM, anti-toxoplasma IgM, salmonella, and brucella agglutination tests were negative. However, anti-HAV IgM and anti-HAV IgG were positive. There were no ova and parasites in stool.

After 48 h of admission, since there was no improvement in the patient’s clinical status in addition to an increase in the abdominal pain a laparoscopic cholecystectomy was performed. Intra-abdominal fluid was drained surgically, and samples were sent culture. Cultures remained negative after incubation for 2 days. The empirically initiated ampicillin-sulbactam treatment was halted after its completion in 8 days. Twenty days after the operation the patients laboratory findings were; AST 109 U/L, ALT 150 U/L, direct bilirubin 3.0 mg/dL, total bilirubin 6.7 mg/dL, albumin 4.2 mg/dL, PT 11.4 seconds and INR 0.9. The patient was hospitalized for 22 days and was called for a follow-up examination 4 weeks after being discharged. In the follow-up examination, the patient’s abdominal USG and liver tests were normal. However, anti-HAV IgM remained positive.

Discussion

HAV infection is seen in the pediatric age group in developing countries and adult age group in developed countries. The disease is asymptomatic in children; however, it can be symptomatic and presents with complications in adults (3,5). Cholecystitis caused by HAV infection is rarely reported in the literature (8-10). The case presented in this report had emphysematous cholecystitis as a complication of the HAV infection.
Özaras et al. (7) has reported two HAV related acute viral cholecystitis cases. One of these patients was a 28-year-old male, with jaundice and a portal mass suggestive of cholangiocarcinoma, and the other was a 20-year-old woman with jaundice, stomach ache, nausea, vomiting, pruritus, and lack of appetite. Both patients were considered for operations however as their fever, leukocyte, and acute phase reactant findings were unresponsive, and because there were no gallstones present, bacterial cholecystitis ruled out; hence, the surgeries were canceled. Both patients’ clinical findings subsided spontaneously. Their report underlined that the patients were approached conservatively and that surgical intervention was prevented. There are a few studies reporting similar complications in the pediatric age group (8,11). The incidence of cholecystitis in children has been reported to be %1-4 (9,10). In these cases, the diagnoses were established by USG findings, and surgical intervention was deemed unnecessary.

Emphysematous cholecystitis is divided into three stages according to the presence of gas in the lumen of the GB (Stage I), wall (Stage II), pericholecystic area (Stage III) (11). There are differing views regarding how gas is formed in the GB. Usually, gas-forming bacteria such as Clostridium welchii, Escherichia coli, Klebsiella spp. and anaerobic streptococci are pointed out. Decrease in the volume of the GB, increase in wall thickness, and the presence of gall mud have been reported (6). Most publications point out that GB wall returns to normal thickness within a few days, and that surgery is generally unnecessary (9). In our cases, due to the fact that GB wall was thickened in USG, and intramural air was present in the wall, a diagnosis of emphysematous cholecystitis was established. Because the patient’s complaints persisted for 48 h after initial presentation, surgery was performed. Bacteria were not present in the isolated cultures. We did not encounter a previously reported HAV related emphysematous cholecystitis case in our literature review. Therefore, we believe that this presented case is significant.

**Conclusions**

To sum up, HAV infection can be seen in all age groups, in developing countries, like as our country. It must be kept in mind that, although very rarely, HAV infections may exhibit extrahepatic complications. In patients presenting with upper right quadrant pain, severe abdominal pain, prolonged fever, nausea and vomiting, an accompanying cholecystitis complication must be kept in mind as a possibility.

**References**