

Acute Acalculous Cholecystitis Due to
Viral Hepatitis B: A Case ReportOmer A Mahmoud¹, Mustafa Z Mahmoud^{2*} and Maram A Fagiri²¹Medical Ultrasound Imaging Department, Dr. Mohamed Abdel Mageed Ali Medical Complex, Alnohood, Sudan²Radiology and Medical Imaging Department, College of Applied Medical Sciences, Prince Sattam bin Abdulaziz University, Al-Kharj, Saudi Arabia

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Abstract

Hepatitis B is a potentially life threatening liver infection caused by the Hepatitis B Virus (HBV). It is a major global health problem. It is not possible, on clinical grounds, to differentiate HBV from hepatitis caused by other viral agents and, hence, laboratory confirmation of the diagnosis is essential. Sonographically, gallbladder findings of increased wall thickness and pericholecystic edema are very common. It can be used as auxiliary findings in case of acute viral hepatitis; especially when serological testing facility is not available it can back up in the diagnosis. A twelve year old Sudanese male, developed classic symptoms of acute HBV. In the emergency department, he presents with nausea for 9 days accompanied by vomiting, anorexia, fatigue, mild upper abdominal pain, loss of appetite, jaundice with pale stool, and dark urine. Laboratory investigation revealed an increased level of White Blood Cells (WBC), Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), and total serum bilirubin. Serological testing was positive for the Hepatitis B (HB) Surface Antigen (HBsAg), HB e Antigen (HBeAg), and anti-HBc (both IgM and IgG). Abdominal ultrasound revealed marked edematous gallbladder wall thickening. Also, gallbladder hyperemia was detected by Doppler ultrasonography. The patient received his initial HB vaccinations and was discharged with considerable improvement in biochemical tests. Gall bladder findings on ultrasound can be used to diagnose acute HBV when serological tests are not available.

Introduction

Hepatitis B Virus (HBV) is a small (diameter of 42 nm), incompletely double-stranded DNA hepadnavirus. Substantial genetic variations occur within distinct regions, globally facilitating classification of eight distinguishable genotypes (A through H), which have treatment implications [1,2]. All genotypes are present in the United States, with genotypes A and C comprising 35 and 31 percent of viruses, respectively [3]. The HBV genome produces a nucleocapsid that contains the Hepatitis B Core Antigen (HBcAg). This nucleocapsid is encompassed with an outer envelope referred to as the Hepatitis B Surface Antigen (HBsAg). One segment of HBcAg results in the production of the Hepatitis B e Antigen (HBeAg), which is associated with viral replication and high infectivity. The DNA polymerase reverse transcriptase is a target for antiviral therapy [4]. HBV is transmitted in blood and secretions (e.g., semen, saliva) and is infectious outside the body for seven or more days [5]. Globally, an estimated 350 million persons are chronically infected with HBV [6]. Even with the advent of an effective hepatitis B vaccine, more than 50 million new cases of HBV infection occurs annually [7]. High-risk populations should be screened for HBV infection [3]. The Centers for Disease Control and Prevention recommends routine HBV screening in populations in which HBsAg prevalence is at least 2 percent, including immigrants from these regions [6].

Case presentation

A twelve year old Sudanese male, developed classic symptoms of acute hepatitis. At the time when his general practitioner referred him to the emergency department, he had nausea for 9 days accompanied by vomiting, anorexia, fatigue, mild upper abdominal pain and loss of appetite. He had developed jaundice with pale stool and dark urine 4 days before referral. He had no pruritis. On physical examination, body temperature was 39°C, heart rate of 80 beats per minute, breath rate of 23 breaths per minute and blood pressure of 95/60 mmHg. He was jaundiced and had a tender liver and palpable 6 cm below right costal margin.

At admission, laboratory studies revealed the following: White Blood Cells (WBC), 6000/mm³; hemoglobin, 13 mg/dl; platelets, 175000/mm³; Alanine Aminotransferase (ALT), 3700 U/L (normal 7-40 U/L); 2330 U/L for Aspartate Aminotransferase (AST) (normal 7-40 U/L); total serum bilirubin of 137 µmol/L (normal 3-25 µmol/L). No signs of liver failure developed during the course of the disease. Serological testing was positive for the Hepatitis B (HB) Surface Antigen (HBsAg), HB e Antigen (HBeAg) and anti-HBc (both IgM and IgG), while other serological tests were negative. Acute infection with acute Hepatitis B Virus (HBV) was subsequently diagnosed.

Abdominal ultrasound revealed hepatomegaly (Figure 1) with the cranio-caudal diameter of the right lobe of 15.7 cm (normal up to 15.5 cm), splenomegaly (Figure 2) with a splenic length of 14 cm

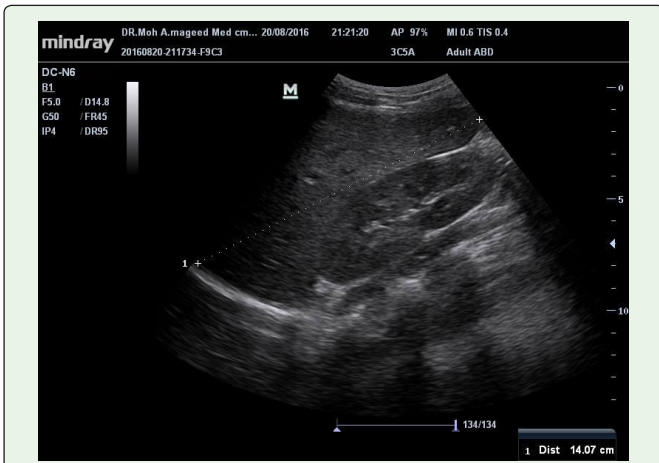


Figure 1: Gray scale ultrasound image of the liver revealed features of hepatomegaly (right liver lobe cranio-caudal diameter >15.5 cm).

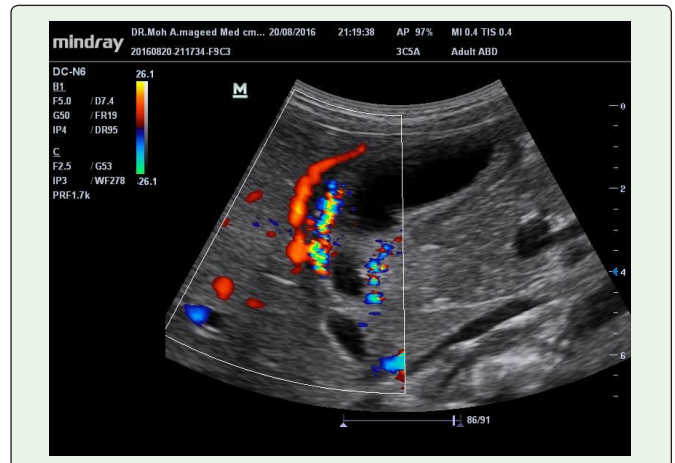


Figure 4: Color Doppler ultrasonography presents an excess blood flow (hyperemic gallbladder) due to acute HB infection.



Figure 2: Gray scale ultrasound image of the spleen revealed features of splenomegaly (spleen length >13 cm).

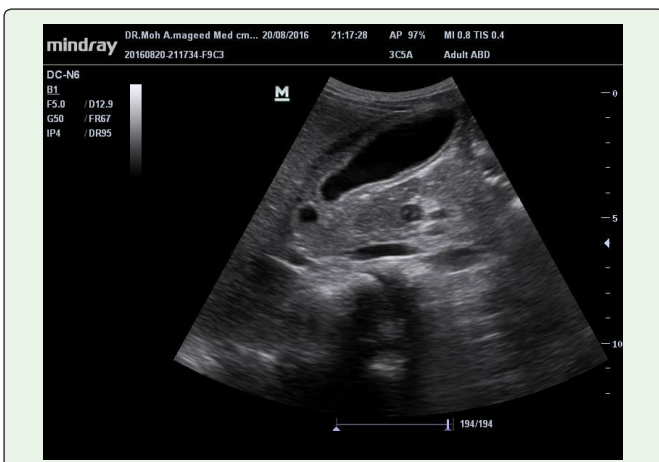


Figure 3: Gallbladder with features of acute acalculous cholecystitis; diagnosed on the basis of marked gallbladder wall thickening (10 mm), no gallbladder calculus, and the evidence of pericholecystic fluid collection.

(normal up to 13 cm), marked gallbladder wall thickening (10 mm), no gallbladder calculus, evidence of pericholecystic fluid along with hyperemia, as revealed by Doppler ultrasonography (Figures 3 and 4).

The patient received his initial HB vaccinations; RECOMBIVAX HB (the vaccine contains one of the viral envelope proteins). A series of 3 doses (0.5 mL each) given on a 0-, 1-, and 6-month schedule. During follow-up, his tenderness regressed, total serum bilirubin levels decreased, and respectively; there was also a marked decrease in serum AST and ALT levels. He was discharged in good clinical condition and with considerable improvement in biochemical tests.

Discussion

Symptoms of acute HBV infection are nonspecific and include fatigue, poor appetite, nausea, vomiting, abdominal pain, low grade fever, jaundice, and dark urine. Clinical signs include liver tenderness, hepatomegaly, and splenomegaly. Acute HBV infection typically lasts two to four months [8].

It is not possible, on clinical grounds, to differentiate hepatitis B from hepatitis caused by other viral agents and, hence, laboratory confirmation of the diagnosis is essential. A number of blood tests are available to diagnose and monitor people with hepatitis B. They can be used to distinguish acute and chronic infections. Laboratory diagnosis of hepatitis B infection focuses on the detection of the hepatitis B surface antigen HBsAg. Acute HBV infection is characterized by the presence of HBsAg and IgM antibody to the core antigen, HBcAg. During the initial phase of infection, patients are also seropositive for HBeAg. HBeAg is usually a marker of high levels of replication of the virus. The presence of HBeAg indicates that the blood and body fluids of the infected individual are highly contagious [9].

In acute course of HBV, ultrasound revealed a collapsed gall bladder with increased wall thickness and pericholecystic edema as a most consistent finding [10]. Several hypotheses have been proposed to explain the mechanism of gallbladder wall thickening in patients with acute hepatitis. One hypothesis is that gallbladder wall thickening, together with a decrease in gallbladder volume, occurs when hepatocytes injury at the time of onset of acute hepatitis causes a temporary decrease in bile production and excretion [11].

A second hypothesis is that gallbladder wall thickening is due to a direct injury and inflammation of the mucosal and muscular layers of the gallbladder by hepatitis virus contained in the bile [12]. A third hypothesis is that hepatocytes necrosis, which is extensive in patients with acute hepatitis, causes an inflammatory reaction in the tissues surrounding the liver, including the gallbladder wall [13].

In conclusion, ultrasound findings are present in most of the acute HBV. Gallbladder findings of increased wall thickness and pericholecystic edema are very common. It can be used as auxiliary findings in case of acute HBV. Especially when serological testing facility is not available, it can back up in the diagnosis when clinically acute HBV is suspected.

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