

ACUTE ACALCULOUS CHOLECYSTITIS IN A CHILD WITH HEPATITIS A INFECTION

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ABSTRACT

Acute acalculous cholecystitis (AAC) is an unusual and atypical manifestation of acute viral hepatitis A which is endemic in many developing countries. It may cause gangrene and perforation of the gallbladder wall if not picked early but mortality from acute acalculous cholecystitis in these patients is far less than AAC of other origins that need surgical intervention. Usually conservative management helps improving the condition of these patients with hepatitis A virus (HAV) infection. Here we are going to present a case of a 7-year-old girl who was diagnosed with atypical cholecystitis clinically and on ultrasound examination.

Key Words: Hepatitis A virus, Acute acalculous cholecystitis, Gall bladder wall thickness

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INTRODUCTION

Hepatitis A is a self-limited benign viral infection commonly found in children in developing countries. Most of the cases are asymptomatic, however, the cases may present with nausea, vomiting, diarrhea, fever and jaundice¹⁻⁴. Acute acalculous cholecystitis (AAC) is the inflammatory disease of the gallbladder in the absence of gallstones. It is estimated to represent at least 50-70% of all cases of acute cholecystitis during childhood⁵. Acute acalculous cholecystitis (AAC) is a rare disease in children, and its spectrum has not been well established⁴. Clinical manifestations are typical bile duct pain, jaundice and diffuse right hypochondrium mass or fullness commonly referred to as acute abdominal pain. The diagnosis is clinically suspected and then confirmed by ultrasound⁶. The occurrence of AAC during HAV infection is uncommon and has been specifically described in the literature⁷.

CASE REPORT

A 7 years old, previously healthy girl, presented to the emergency department of Dar-ul-Sehat Hospital in Karachi, Pakistan, in May 2016 with 5 days history of abdominal pain, anorexia, nausea, vomiting and malaise.

Initially the pain was diffuse but later it became intense only at the periumbilical region and epigastrium. There was no history of dark urine, fever or diarrhea. Patient was not being vaccinated for Hepatitis A. Her medical history was unremarkable.

On physical examination, the patient was afebrile and restless with a heart rate of 93 beats/min. Scleral icterus was present. Abdominal examination showed tender epigastric area and periumbilical region and a negative Murphy's sign. Her liver was palpable 3cm below the right costal margin without associated splenomegaly. Serology for viral hepatitis suggested acute hepatitis A infection (anti-HAV IgM positive.) Laboratory data at the time of admission were total bilirubin 3.9 mg/dl, bilirubin direct 2.65 mg/dl, bilirubin indirect 1.25 mg/dl, ALT/SGPT 1690 u/l, ALP 1310 u/l, gGT 196 u/l, Hb 12g/dl, total leucocyte count 5,400/ul, neutrophils 52%, lymphocytes 44% and platelets 242,000/ul. Ultrasound scan showed thick and edematous walls of gall bladder. Gall bladder wall thickness (GBWT) measured 0.5 cm (Figure 1). There was no evidence of mass or calculous in the gall bladder. Multiple enlarged hypoechoic lymph nodes were seen at portahepatis and peripancreatic region, one of them measured 1.6x1.0 cm. Liver

Figure 1: Ultrasound scan of the abdomen showing thick and edematous walls of gall bladder, measuring 0.5 cm

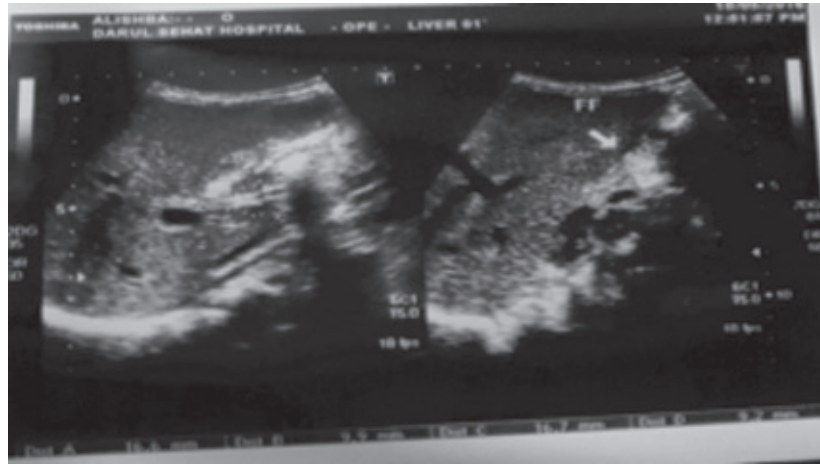
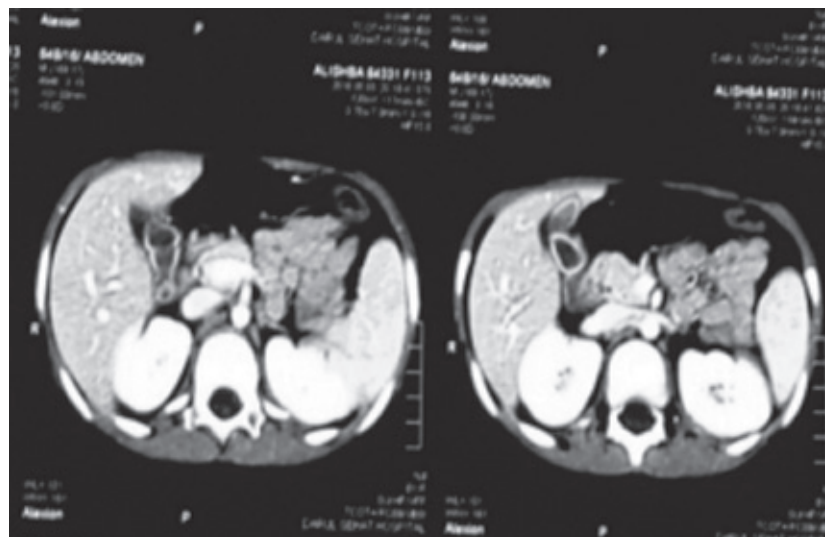


Figure 2: CT abdomen with contrast showing diffuse gall bladder wall thickening and enhancement with free fluid and surrounding lymph nodes



showed hypoechoic parenchyma with prominent periportal radicals suggestive of inflammation. Streak of free fluid was seen around the liver. CT abdomen with contrast was ordered which validated the findings of ultrasound showing diffuse gall bladder wall thickening and enhancement with free fluid and surrounding lymph nodes were most likely representing cholecystitis (Figure 2). There was no evidence of radiopaque calculi. Mild inflammation of the gastric pylorus was also noted. Patient was treated with supportive therapy; including IV fluids, IV paracetamol (for severe abdominal pain) with IV nalbuphine when needed and intravenous antibiotic (ceftriaxone) was administered. Patient improved with the conservative management hence no surgical intervention was needed. Her symptoms regressed and biochemical markers including ALT, AST and total bilirubin were normalized. She was discharged on the 7th day of admission and was advised follow up to outpatient department for monitoring.

DISCUSSION

Infection with hepatitis A is usually benign and a self limiting clinical pathology. In the literature, however, gangrenous cholecystitis, pleural effusions and ascites are rarely due to HAV infections. Although these complications of HAV infection are rare, gall bladder abnormalities usually occur during acute phase of viral hepatitis infection. These abnormalities include gall bladder wall thickening, gall bladder deposition and double-wall appearance⁸. The pathophysiology of gall bladder wall thickness in acute viral hepatitis is unclear; hypoalbuminemia, prolonged local liver inflammation and portal hypertension can be reflected as gallbladder wall edema⁹.

Most part of viral AAC has been described in last few years, which could be due to an increase of the diagnostic sensitivity, as ultrasonography has been used more often during acute liver diseases⁸. Hakala et al¹⁰ reported that a marked decrease in vascular filling of the gallbladder in the acalculous cholecystitis patients was observed when compared with the specimens of calculous cholecystitis patients. Ozaras et al² described two adult patients (28 and 20 years of age) with acute cholecystitis due to HAV infection. Neither administration of antibiotics nor surgical intervention was necessary for any of these patients. With a close follow up, both of the patients fully recovered. Hermier et al⁶ reported 3 cases of children with acute cholecystitis caused by HAV infection. Initial clinical presentation in children with acute hepatitis suggest acute cholecystitis (pain and guarding associated with right hypochondrium pain, fever and delayed jaundice) and associated with significant ultrasound findings including: a wall thickness greater than 10mm in the gallbladder, 2 or 3 layers of different echogenicities, ultrasonographic Murphy's sign and

gallbladder echogenic contents. The ultrasonographic gallbladder findings in children suspected of acute acalculous inflammatory gallbladder disease might result in better outcomes. In a study, 39 children hospitalized for Hepatitis A virus infection were evaluated ultrasonographically and operated; gallbladder walls with 10 mm or more striation were found in 10 patients¹¹. All of their anomalies returned to normal within 4 weeks.

CONCLUSION

Acute viral hepatitis A frequently occurs in childhood. The rare manifestations of acute viral hepatitis such as acute acalculous cholecystitis should be kept in mind by pediatricians and gastroenterologists to avoid unnecessary invasive procedures.

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