Indian Journal of Clinical Biochemistry, 2009 / 24 (3) 316-318

CASE REPORT

ACALCULOUS GALLBLADDER DISTENSION IN A YOUNG CHILD DUE TO HAV INFECTION : DIAGNOSTIC DILEMMA

D R Suresh, R Srikrishna, S K Nanda, V Annam, K Sunil* and B Arjun*

*Departments of Biochemistry and *Radiology, Sri Siddhartha Medical College, Tumkur-07, India*

ABSTRACT

Gall bladder distension with acute viral acalculous cholecystitis is a rare event in pediatric cases with a high incidence of perforation, gallbladder necrosis and mortality. We report a two and a half year old female child presenting with fever, vomiting, pain abdomen, mild hepatosplenomegaly and tenderness in right hypochondrium. Laboratory investigations revealed hyperbilirubinemia and elevated alkaline phosphatase, but there was no evidence of bacterial or parasitic infection. Serology for viral hepatitis suggested acute Hepatitis A infection. Ultrasonographically, distended inflamed gallbladder without calculous was observed. Finally acute acalculous cholecystitis due to Hepatitis A virus was diagnosed and the child responded to the conservative management.

KEY WORDS

Acalculous cholecystitis, Hepatitis A, Pericholecystic fluid, Gallbladder thickening.

INTRODUCTION

Acute acalculous cholecystitis is an inflammation of the gallbladder in the absence of gallstones. It usually occurs in critically ill patients but is rare in the pediatric age group (1). Though, acute hepatitis A virus (HAV) infection is frequently encountered in developing countries in children, acute viral acalculous cholecystitis due to viral hepatitis A, has rarely been described in children. The diagnosis of AAC is sometimes ambiguous and confusing especially in children (2). Here we report two and a half - year old female child with acute viral acalculous cholecystitis due to hepatitis A virus infection which is rarely reported at such an early age.

CASE HISTORY

Two and a half - year old female child was presented with four days of relapsing-remitting moderate degree fever, nausea and vomiting associated with abdominal pain, loss of appetite, dark urine and pale stool. Nothing was significant from past history and family history. On physical examination, child was moderately built and nourished (body weight-12.5 kg and height-92.5 cms), she was irritable, temperature - 38.5°C, heart rate - 86/minute, breath rate - 22/minute and blood pressure - 100/70 mm Hg. She appeared pale, fatigued with mild icterus, but cyanosis or lymphadenopathy was absent. The right side of the abdomen was tender with painful fullness in the right hypochondrium (a positive Murphy's sign). Mild hepatosplenomegaly was present. Other systemic examinations were normal. Laboratory studies revealed the following: normal hemoglobin - 13.6 g/dL; normal WBC - 6100 / mm^3, elevated C reactive protein - 12.5 mg/dL; normal platelet count - 186000/mm^3; normal ALT - 23.4 U/L; normal AST - 20.6 U/L; increased total bilirubinemia - 2.8 mg/dL with a direct fraction of 0.9 mg/dL; markedly elevated alkaline phosphatase - 551 U/L ; elevated GGT - 102 U/L, normal amylase – 38 U/L, normal albumin - 2.9 g/dl and normal prothrombin time. Serology for viral hepatitis suggested acute hepatitis A infection (anti HAV IgM +), and was negative for other causes [HBsAg (-), anti HBcIgM (-), anti HCV (-)]. Serology for salmonellosis and brucellosis were negative. Widal test was nonreactive. Bile salts and bile pigments were present in urine. Blood and

Address for Correspondence :

Dr. D. R. Suresh
3/1, Seethappa layout, 5th Block,
Doddabommasandra, Vidyaranyapura.P.O,
Bangalore, Karnataka- 560097
M: +91-9900954628
E-mail: drsuri77@yahoo.com; drsuri77@gmail.com

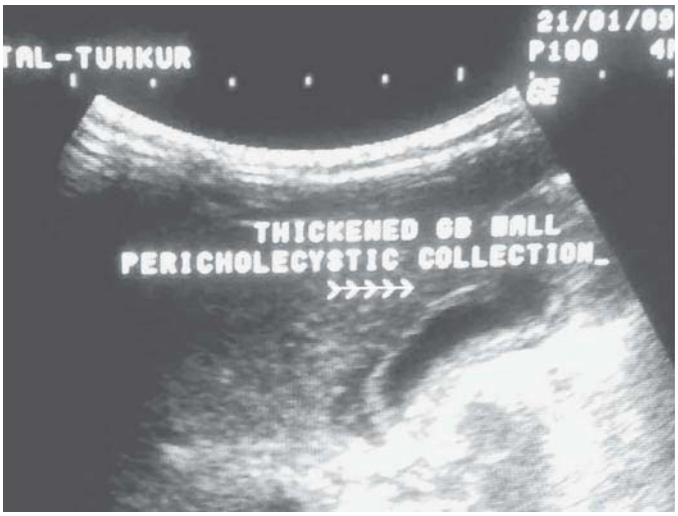

Fig 1: Trans-abdominal Ultrasonography showing thickened gallbladder and pericholecystic fluid collection

urine culture were sterile. Stool culture and parasitological examination were negative. Abdominal ultrasound revealed hepatosplenomegaly, hydropic gallbladder without calculus, thickened gallbladder wall and pericholecystic fluid. Sonographic Murphy sign was elicited when the probe was pushed directly on the gallbladder (Fig 1). Thus by clinical, laboratory and ultrasonographic findings, acute viral acalculous cholecystitis was diagnosed.

With conservative management, after four days child showed improvement in clinical and biochemical tests. She became afebrile, her tenderness regressed, serum total bilirubin and its direct fraction levels decreased to 1.7 mg/dL and 0.6 mg/dL, respectively; there was also a marked decrease in serum ALP levels. Surgical intervention was not required in our patient.

DISCUSSION

AAC, an uncommon condition in children, is an inflammation of the gallbladder in the absence of gallstones. HAV is a self-limiting, usually asymptomatic infection that occurs predominantly among children. HAV induced AAC has been very rarely reported in children. Although the origin is obscure, demonstrated invasion of the gallbladder and bile duct epithelium by HAV and cell-mediated immunologic response have been proposed in the pathogenesis of HAV infection-induced cholecystitis (3, 4).

Low grade fever, colicky abdominal pain and vomiting are common, as is right subcostal tenderness. Within a few hours, Murphy's sign (deep inspiration exacerbates the pain during palpation of the right upper quadrant and halts inspiration) develops along with involuntary guarding of right-sided, upper abdominal muscles. Untreated, the disease can rapidly progress to gallbladder gangrene and perforation, leading to sepsis, shock, and peritonitis (5). In uncomplicated acute cholecystitis, liver function tests are normal or only slightly elevated. Mild cholestatic abnormalities (bilirubin up to 4 mg/dL and elevated alkaline phosphatase) are common, probably indicating mechanical obstruction of the cystic duct resulting from intrinsic inflammation of the biliary tract (6). Ultrasonographic criteria for the diagnosis of acalculous cholecystitis include gallbladder thickening (more than 6 mm), pericholecystic fluid, pericholecystic stranding, high attenuation of bile and subserosal edema (halo). It may also elicit local abdominal tenderness over the gallbladder (ultrasonographic Murphy's sign) (7).

In the present case, child had acute HAV infection documented by clinical features, biochemical and serologic tests. Viral acalculous cholecystitis developed during the course of the disease. Ultrasonographic examination revealed the diagnosis of acalculous cholecystitis. The treatment of AAC varies, depending on the clinical presentation. Most cases are self-limited and the gallbladder may spontaneously decompress with treatment of the underlying systemic disease within approximately two weeks. Associated complications such as gallbladder perforation and deterioration of abdominal signs have been suggested as indications for surgery (8).

Acute viral acalculous cholecystitis is a rare complication of HAV infection which should be suspected when a child is admitted with right upper abdominal tenderness and fever, as it can lead to surgical emergency.

REFERENCES

1. Venuta A, Bertolani P. Acute cholecystitis in a child with HAV infection. Int J Pedia Neonatology 2007; [cited 2009 Jan 25]: 6(2): [about 2p]. Available from: http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijpn/vol6n2/hav.xml
2. Mourani S, Dobbs SM, Genta RM, Tandon AK, Yoffe B. Hepatitis A virus-associated cholecystitis. Ann Intern Med 1994; 120(5): 398-400.
3. Casha P, Rifflet H, Renou C, Bulgare JC, Fieschi JB. Acalculous acute cholecystitis and viral hepatitis A. Gastroenterol Clin Biol 2000; 24(5): 591-2.
4. Portincasa P, Moschetta A, Di Ciaula A, Palmieri VO, Milella M, Pastore G, et al. Changes of gallbladder and gastric dynamics in patients with acute hepatitis A. Eur J Clin Invest 2001; 31: 617-22.

5. Klar A, Branski D, Nadjari M, Akerman MY, Shoseyov D, Hurvitz H. Gallbladder and pancreatic involvement in hepatitis A. J Clin Gastroenterol 1998; 27: 143-5.
6. Gulnar U, Reha C, Akif GM, Bayram O. Hydrops of gallbladder associated with infection: A report of two cases. Turk J Gastroenterol 2000; 11(1): 84-7.
7. Dalgic N, Ince E, Cifti E, Oncel S, Gunes M, Fitoz S, Dogru U. Acute viral acalculous cholecystitis due to viral hepatitis A. J Ankara University Faculty of Medicine 2005; 58(2): 78-80.
8. Ozaras R, Mert A, Yilmaz MH, Celik AD, Tabak F, Bilir M, et al. Acute viral cholecystitis due to hepatitis A virus infection. J Clin Gastroenterol 2003; 37(1): 79-81.