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Case Report

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A Rare Case of Childhood Hepatitis A Infection with Bilateral Pleural Effusion Acalculous Cholecystitis and Massive Ascites

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Abstract

Hepatitis A virus infections are mostly asymptomatic or mildly symptomatic in pediatric population. Generally this disease has a benign course and resolves spontaneously. However, intrahepatic and extrahepatic manifestations can complicate typical course of acute hepatitis A in children. Pleural effusion, acalculous cholecystitis and ascites are extremely rare extrahepatic complications. Pleural effusion or ascites does not change the prognosis or require any invasive treatment usually.

We report a case of 8 years old boy presented with both pleural effusion and gross ascites accompanying hepatitis A infection. Diagnosis was done using serological testing and imaging studies. He was treated with supportive management only with full recovery after 4 weeks.

Keywords: Hepatitis A, Pleural effusion, Ascites, Unusual

Objective

Acute hepatitis A virus (HAV) infection is a self limiting viral disease in childhood. The seroprevalence of hepatitis A is reported in India is as high as 33% [1] and is responsible for sporadic outbreaks. The common cause of transmission is contamination of water with feces.

The patients often presents with nausea, vomiting, and fever with or without an icterus with a case fatality rate varying from 0.3% among children to 1.8% in elderly.[2]

Although hepatitis A usually presents with mild symptoms in children, intrahepatic and extrahepatic manifestations are reported rarely in 6.4-8% of cases. [3,4] These manifestations include arthralgia, cutaneous vasculitis, cryoglobulinemia, hemophagocytic syndrome, acalculous cholecystitis, pancreatitis, aplastic anemia, Guillane-Barre syndrome, transverse myelitis, acute tubular necrosis, nephrotic syndrome, vasculitis, reactive arthritis and pleural effusion. Among these, pleural effusion and acalculous cholecystitis are rare complications of acute viral hepatitis

A. Pleural effusion accompanying ascites in the course of hepatitis A is reported rarely in literature. [5-7] However, we found only three cases in the literature with all these three complications being presented simultaneously. So we present a case of hepatitis A complicated by pleural effusion, massive ascites and acalculous cholecystitis.

Case Presentation

A 8 years-old boy presented to the pediatric outpatient clinic with mild

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grade fever for 2 days one week back along with recurrent vomiting. He took OPD based treatment and symptoms got relieved. Now for last 3 days he had complaint of abdomen distension which was progressive in nature and associated with dull aching pain in right subcostal region. He had no previous history of traveling, blood transfusion, bleeding, or previous medical, drug, or surgical treatment.

Upon presentation, during physical examination, he had normal body temperature, gross abdominal distention, hepatomegaly with normal spleen size, unilateral basal right-sided decreased breathing sound. He was tachypnoic on admission due to abdomen distension. The rest of the examination was normal, including normal mental status.

The patient was admitted, and laboratory investigations were carried out (Tables 1, 2). Ultrasound examination revealed bilateral mild pleural effusion, gall bladder wall thickening of 5 mm suggestive of acalculous cholecystitis, severe free fluid in abdomen and hepatomegaly [Figure 1]. Etiological work up revealed a positive immunoglobulin M anti-hepatitis A serology. Serologies for hepatitis B, hepatitis C, Dengue fever and enteric fever were negative. Color Doppler examination of portal system was done which also came positive. Echocardiography was free of any abnormality. Our patient was diagnosed with HAV acute hepatitis associated with bilateral pleural effusion, acalculous cholecystitis and massive ascites. Treatment consisted of Inj Vitamin K, low protein low fat carbohydrate-enriched diet, while no antibiotics were used. Oral spironolactone was given as diuretic in view of massive ascites. She was managed conservatively and no invasive interventions were done. The patient was discharged on day 7 after significant improvement and significant reduction in abdominal girth. He achieved full clinical and biochemical recovery in 3 weeks post discharge.

Table 1: Laboratory analysis

Laboratory analysis	Result
Hb	11.6 g/dl
WBC	6.79 × 10^3cells/mm3
PLT	433 × 10^3/mm3
Serology HAV IgM	+ve HAV IgM
PT	16.1 (13.5 control)
PTT	22.6 (26 control)
INR	1.19
Albumin	2.7 g/dl
Procalcitonin	0.04 ng/ml
ALP	348 IU/l
Serum ammonia	84 UG/dl

Hb: Hemoglobin, WBC: White blood cells, PLT: platelets, PT: Prothrombin Time, PTT: Partial Thromboplastin Time, INR: International normalized ratio, ALP: alkaline phosphatase.

Table 2: Liver biochemical markers tracing during hospital stay

Liver biochemical markers	1st day	3rd day	5th day
AST (U/l)	236	159	142
ALT (U/l)	436	355	294
Total bilirubin (mg/dl)	0.7	0.8	0.6
Direct bilirubin (mg/dl)	0.3	0.2	0.2

AST: Aspartate aminotransferase, ALT: alanine aminotransferase.

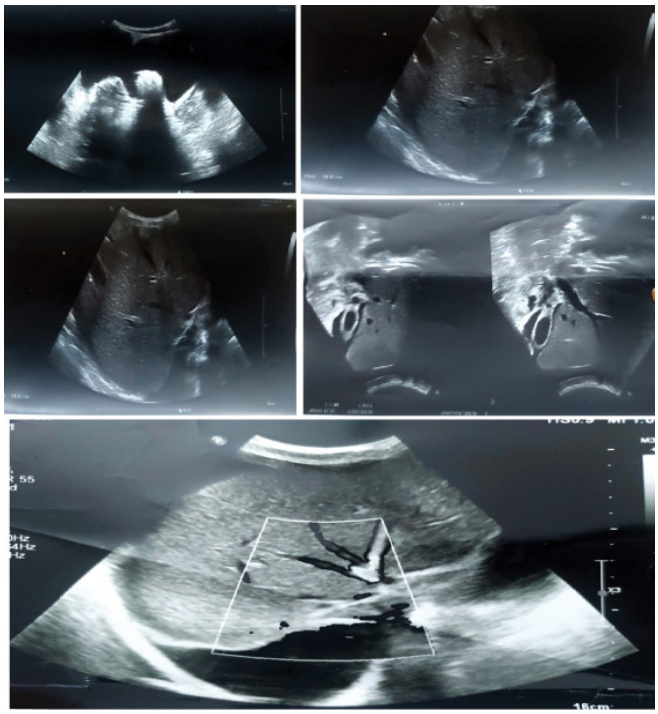

Figure 1: Ultrasound images.

Discussion

Hepatitis A virus (HAV) causes acute hepatitis associated with significant morbidity and occasional mortality. Although it can infect other tissues, clinical manifestations are usually associated with liver inflammation. The severity of disease increases with age. In children, it manifests usually with mild symptoms or asymptomatic. Jaundice is infrequent finding in pediatric HAV infection. In this self limiting infection, 85% of patients recover completely in three months.

HAV acute hepatitis may be associated with many complications, including:

1. Intrahepatic: such as cholestatic hepatitis, relapsing hepatitis, and autoimmune hepatitis. Rarely, hepatitis A can progress to acute liver failure.
2. Extrahepatic manifestations are infrequently reported in HAV acute hepatitis (6.4-8%) and may include: urticarial and maculopapular rash, acute kidney injury, autoimmune hemolytic anemia, aplastic anemia, acute

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pancreatitis, mononeuritis, reactive arthritis, Guillain-Barre syndrome and pleural or pericardial effusion, ascites, glomerulonephritis, polyarteritis nodosa, cryoglobulinemia, and thrombocytopenia. [3,4,8]

Pleural effusion is known to be an early and benign complication of the disease. Pleural effusion is an extremely rare co-occurring condition. The exact mechanism of pleural effusion is unknown and could be multifactorial. Theories regarding pathogenesis include:

1. Direct viral invasion of pleura, immune complex deposition or inflammatory response to HAV
2. Co presenting ascites could contribute to the pleural effusion via small diaphragmatic defects or diaphragmatic lymphatics
3. Decrease in the plasma oncotic pressure as well as a transient rise of the pressure in the portal vein or lymphatics due to compression by the hepatic sinusoids

Ascites has been reported in later stages of disease especially in older children and adults, is thought to occur from venous or lymphatic obstruction due to liver involvement or reduction of oncotic pressure due to hypoalbuminemia during the course of infection. [9,10]

Acalculous cholecystitis is rare in children. It has an uneventful course and usually recovers in two to three weeks. Exact pathogenesis of this manifestation is unknown. Mourani et al detected HAV antigen in bile duct epithelium and the gall bladder wall suggesting a direct effect of viral antigen rather than a secondary phenomenon. [11] Gallbladder changes may be variable during the course of HAV infection. Gallbladder thickening is the most common finding. [12]

All these three complications are usually transient and self-resolving. These usually do not require any surgical intervention. Our case shows hepatitis A infection complicated with extrahepatic manifestations and presents a success story of its conservative management. Pleural effusions and ascites do not change the prognosis or require any invasive treatment.

Conclusion

Pleural effusion, ascites and acalculous cholecystitis are benign, rare, extrahepatic complication of HAV acute hepatitis. These resolves spontaneously with supportive management. Thus, further invasive procedures would only complicate this self-resolving benign condition and should be minimized.

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