Case Report

Pleural Effusion: An Uncommon Manifestation of Hepatitis

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ABSTRACT

Hepatitis A virus infection is the commonest form of hepatitis in pediatric age group and common health problem in developing countries due to poor sanitation. The clinical spectrum of hepatitis A virus infection ranges from asymptomatic infection to fulminant hepatitis and the symptoms are primarily hepatobiliary or constitutional. Here, we present a case of 16 years old male who presented with complains of fever, vomiting, anorexia, right sided abdominal pain, shortness of breath and cough. On clinical examination, patient has hepatomegaly and decreased breath sounds on right sided mammary, infra- mammary and infra scapular region. Blood investigations showed aminotransferases while pleural effusion and ascites with thickened gall bladder were found in chest x-ray and abdominal ultrasonography respectively. In absence of others causes, pleural effusion can be a rare complication of hepatitis A. Thus, this complication should be kept in mind in endemic countries in clinically matching scenarios.

Keywords: Acalculous cholecystitis**;** ascites; hepatitis A; pleural effusion

INTRODUCTION

Hepatitis A is the most common form of acute viral hepatitis in the world and its endemicity are closely related to hygienic and sanitary conditions and other indicators of the level of socioeconomic development and is common in children of developing nations.[1] Younger children usually have a mild form of the disease, whereas older children may present with more serious and rarer manifestations. [2] The patients often present with nausea, vomiting and fever with or without an icterus with a case fatality rate varying from 0.3% in children to 1.8% in elderly. [3]

In this case report, we describe a patient with hepatitis A infection with pleural effusion, ascites and acalculous cholecystitis which improved with supportive treatment.

CASE REPORT

A 16 years old male presented in emergency department with complains of fever, right sided upper abdominal pain, vomiting, fatigue and anorexia for seven days. Patient complained of shortness of breath and nonproductive cough for two days. On examination, he was afebrile and vitals were stable. He had no history of exposure to patient with hepatitis A and previous history of jaundice or blood transfusion. Patient had no history of medical illness in past. On physical examination, patient was anicteric, liver was palpable 3 cm below the right costal margin and spleen was not palpable. On chest auscultation, there was decreased air entry on right infra-scapular and infra-axillary region.

On laboratory investigations, hemoglobin was 10.3 g/dl, hematocrit 31.3%, total leukocyte counts 17300 cells/mm^3 with 75% neutrophils and 25% lymphocytes, alanine aminotransferase (ALT) 1277 U/L, aspartate aminotransferase (AST) 1250 U/L, alkaline phosphatase (ALP) 81 U/L. Bilirubin, albumin, total protein and lactate dehydrogenase (LDH) were within normal limits. Prothrombin time was 30 seconds with international normalized ratio (INR) 2.5 and renal function tests (RFT) were normal.

Chest radiograph (Figure 1) showed moderate pleural effusion on right side with minimal pleural effusion on left side with no parenchymal involvement. Ultrasound examination of lung (Figure 2) revealed right-sided moderate pleural effusion. Abdominal ultrasound

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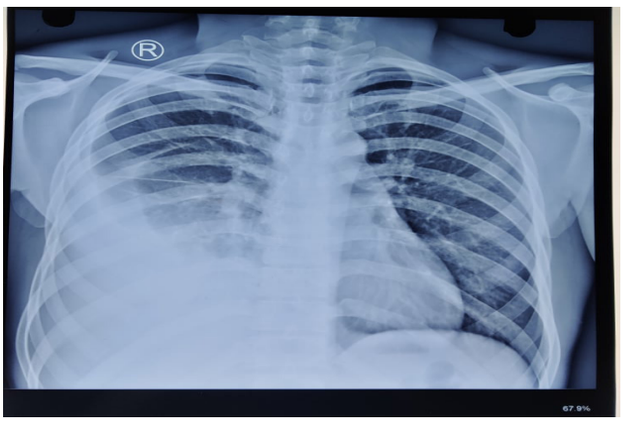

Figure 1. Chest Xray showing right sided moderate pleural effusion.

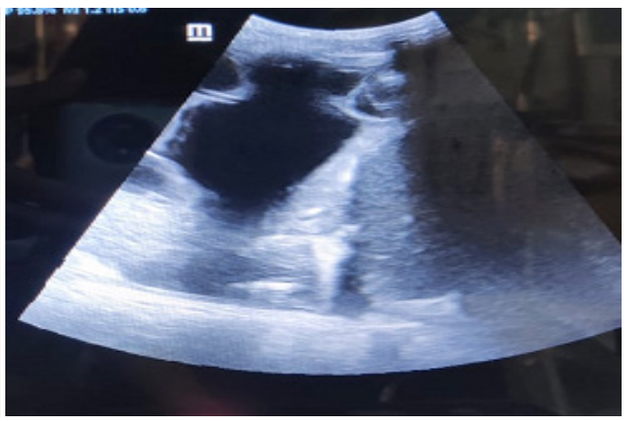

Figure 2. Chest ultrasound showing anechoic effusion in right pleural cavity with few internal septations.

revealed mild hepatomegaly with normal echotexture, thickened and edematous gall bladder with sludge, moderate pleural effusion on right side with minimal pleural effusion on left side. and mild ascites. Antibodies, immunoglobulin M (IgM) and immunoglobulin G (IgG) to hepatitis A virus were detected by enzyme linked immunosorbent assay (ELISA) whereas other viral markers like hepatitis B, hepatitis E, hepatitis C were negative. To determine the etiology of pleural effusion, ultrasound-guided thoracentesis was performed Pleural fluid analysis showed total count of 30 cells/cumm with 75% polymorphs and 25% lymphocytes, glucose 145 mg/dL, total protein 3.2 g/dL, lactic acid dehydrogenase LDH 341 U/L. As pleural fluid was exudative, to rule out parapneumonic effusion and tubercular pleural effusion, pleural fluid culture and pleural fluid adenosine deaminase (ADA), sputum sample for Gram stain, culture and sensitivity and for acid fast bacilli (AFB) were sent. Sputum sample was negative for gram stain and for acid fast bacilli and pleural fluid culture was negative whereas ADA was 18 U/L. Contrast enhanced Computed Tomography (CECT) of the chest showed right sided moderate pleural effusion with few internal septations with normal parenchyma and did not show any features suggestive of pulmonary embolism. Antinuclear antibody (ANA) was negative. Thus, the pleural effusion was attributed to concomitant Hepatitis A. The patient was managed with intravenous fluids and antibiotics. His fever resolved on 5th day of admission, Xray showed that the effusion was resolving and the patient was discharged after two weeks. On follow up at 20 days after discharge, chest x-ray showed complete resolution of pleural effusion and liver function tests were normal.

DISCUSSION

Hepatitis A virus is transmitted primarily through fecal-orally after close contact with an infected person and it is the most common cause of viral hepatitis worldwide, typically causing acute and self-limited symptoms, although rarely liver failure and death can occur.[4] Pleural effusion and acalculous cholecystitis are very uncommon manifestations of childhood hepatitis A infection. [5] A few cases of hepatitis A with pleural effusion and ascites reported worldwide.[2,5]

The commonest causes of the exudative pleural effusion are tuberculosis and parapneumonic effusion which may be associated with other medical illnesses. Parapneumonic effusion was considered less likely in this patient as there was no evidence of radiographic parenchymal involvement and the pleural effusion was bilateral. In presence of lymphocytosis in an exudative pleural effusion, parapneumonic pleural effusion is unlikely. [6] With short duration of illness, negative contact history, low level of ADA in pleural fluid analysis, normal lung parenchyma, negative sputum profile for gram stain and AFB tuberculosis was ruled out. Autoimmune causes of pleural effusion were also ruled out through antinuclear antibody (ANA) test, Rheumatoid Arthritis (RA) factor and Anti-cyclic citrullinated peptides (anti-CCP) which all came out to be negative. Hence, the diagnosis of acute viral hepatitis A infection with associated pleural effusion was made. Consistent with the diagnosis, the resolution of pleural effusion mirrored clinical and biochemical resolution of Hepatitis A in the patient. Tesovic *et al*. have mentioned a possibility of pleural effusion being an early benign complication of acute hepatitis A infection which does not require treatment that resolves spontaneously.[7]

The mechanism of pleural effusion in hepatitis A infection could be multifactorial but the exact pathogenesis is still unknown. One possible mechanism is virus induced inflammation of liver, through which unknown mechanisms leads to effusion 4. Probable mechanism might be direct effect of virus invasion on pleural membrane Kurt et al. demonstrated HAV

directly from pleural fluid by polymerase chain reaction (PCR) procedure.[8] Transport of fluid from diaphragmatic lymphatics or leakage from a diaphragmatic defect to the pleural cavity from co-existent ascites has also been postulated as a cause of pleural effusion.[9] Transport of fluid from diaphragmatic lymphatics or leakage from a diaphragmatic defect to the pleural cavity from co-existent ascites has also been postulated as a cause of pleural effusion.[10] The mechanism of ascites may occur as a result of venous and lymphatic obstruction or decrease in osmotic pressure of plasma colloid 6.

In this patient, pleural effusion, ascites and acalculous cholecystitis were detected in HAV infection. Pleural effusion, ascites and acalculous cholecystitis were transient and gradually disappears in our case similar to other previously reported cases in literature

CONCLUSIONS

A rare case of pediatric hepatitis A with extrahepatic manifestation was identified and managed conservatively. Although HAV infection is known to be self-limiting disease, it may reveal rare atypical extrahepatic complications in course of disease. This case report has important implication in primary care practice making clinicians aware about extrahepatic manifestation in hepatitis A in pediatric age group.

CONFLICT OF INTEREST

None

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