

Case Report

Anasarca-an atypical presentation of hepatitis A

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Abstract. Hepatitis A (HA) is a common illness, with prevalence rates highest in areas with limited hygiene and sanitation practices. In developing countries, where infection is endemic, most people are infected during the first decade of life. The illness is self limited, and severity is age dependent. One of the rare extrahepatic complications of hepatitis A is anasarca. The anasarca presenting as pleural effusion with ascites is a rare and possible benign complication of hepatitis A, and its appearance doesn't seem to correlate with seriousness of illness in children. We present a case of anasarca as an atypical presentation of hepatitis A.

Key words: Anasarca, atypical, hepatitis A

1. Introduction

Acute hepatitis A virus infection is usually a self-limited disease conferring lifelong immunity. Fulminant hepatitis is uncommon but has been described in some settings such as in patients with preexisting chronic hepatitis C. Chronic liver disease does not occur except in rare individuals in whom hepatitis A virus infection serves as a trigger for the development of autoimmune hepatitis. Three atypical clinical manifestations of acute infection are recognized: prolonged cholestasis, relapsing hepatitis, and extrahepatic disease associated with acute infection. The anasarca is a rare extrahepatic complication. We present a case of HA complicated pleural effusion with ascites.

2. Case report

A 3 year-old-male child born of consanguineous parents presented with generalized swelling of the whole body for 10 days prior to admission. The mother also complained of yellowish discoloration of urine. The whole body swelling started from legs followed by abdomen and face. There was no history of bleeding or past history of jaundice.

Examination revealed scleral icterus with bilateral pitting edema. His vital signs were as pulse 118/min with good volume, respiratory rate 30/min; and blood pressure of 70/56 mm of mercury. On systemic examination, there was abdominal distension with engorged veins in the upper aspect. The liver was enlarged, slightly tender, span 9 cm, surface smooth, no nodularity, palpable even in epigastrium. The spleen was not palpable.

After admission, we commenced with medication of intravenous fluids and vitamin K together with syrup lactulose along with protein restricted diet. Laboratory results were as follows: hemoglobin 10.2 g/dL, total leukocyte count 29500 cells/cm³, (neutrophile 53%, lymphocyte 44%, monocyte 1%, eosinophile 2%) platelets 450 x 10⁹/L, erythrocyte sedimentation rate 15mm/1st hr. Red blood cells were anisochromic and normocytic. Liver function test revealed bilirubin 9.67mmol/L, conjugated 8.56 mmol/L, AST 570U/L, ALT 1180U/L, alkaline phosphatase 428 U/L, total protein 6.1g/dL, albumin 2.9 g/dL globulin 3.2 g/dL, A:G = 0.9:1, HbsAg negative, prothrombin time test 17sec, control 12.5 sec. Ultrasonography of abdomen revealed mildly enlarged liver with otherwise normal ultrasound alongside mild ascites. Bilateral pleural effusion was also noticed. Spleen was not enlarged. Chest X-ray also revealed left sided pleural effusion with left lower zone haziness.

We confirmed the diagnosis of viral hepatitis A with unusual associated clinical features like ascites, and pleural effusion. A crucial diagnostic dilemma was now set up - whether we were dealing only with infective hepatitis or hepatitis

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alongside with another associated illness. The most likely associated diseases could be enteric fever or leptospirosis. These possibilities were ruled out by negative widal, blood culture and leptospira IgM tests. After 4 days of therapy, the swelling subsided and appetite were improving. On 5th day after admission, 3 more viral tests were performed in which hepatitis A IgM was positive, hepatitis E IgM and antibody to the hepatitis C was negative. Routine blood check after 9th day revealed hemoglobin 11.2 g/dL, total leucocyte count 14500 cells/cm³, (neutrophile 57%, lymphocyte 40%, monocyte 1%, eosinophile 2%), platelets 450 x 10⁹/L. Liver function test performed on 12th day revealed bilirubin 2.7mg/dL, conjugated 2.1mg/dL, AST 92U/L, ALT 101U/L, alkaline phosphatase 212U/L.

3. Discussion

Children almost universally recover from HA infections. Pleural effusion is a rare complication of acute viral hepatitis. The first case was reported in 1971, and thereafter 14 additional cases were reported. Among those, 5 were associated with hepatitis B and 2 had hepatitis A infection. The exact mechanism of this condition is unknown, though immune complexes have been cited as possible etiological factor. Pleural effusion is a possible benign and early complication of acute hepatitis A infection that resolves spontaneously regardless of illness

outcome (1,2). Ascites is also a known complication of hepatitis A (3). Available literature revealed a single case with three complications (acalculus cholecystitis, pleural effusion and ascites) simultaneously (4). However none of these features are markers of serious illness, and tend to resolve spontaneously. Samanta et al has assessed the clinical course and biochemical profile of children who had symptomatic viral hepatitis A with atypical manifestations. Of 229 children with hepatitis A, atypical manifestations were found in 32 (14%) subjects. Prolonged cholestasis (n=14), acute liver failure (n=9), relapse (n=9), ascites (n=8), and hematological problems (n=8) were the common presentations (5). So, we report a case of anasarca - an atypical presentation of hepatitis A.

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