A Case of Pleural Effusion and Ascites Associated with Hepatitis A

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Abstract

Acute hepatitis A is a frequent public health problem that is mostly seen in childhood, particularly in developing countries. It is the most frequent viral hepatitis. Our country is classified as a moderate endemic region for hepatitis A virus infection. Hepatitis A infection is usually asymptomatic in childhood; however, the course of infection can be more severe at older ages and the infection may end with fulminant hepatic failure, although this is rare. Different extrahepatic findings have been reported to be associated with hepatitis A virus infection. Pleural effusion is a rare complication of hepatitis A. In this report, we present a pediatric case of hepatitis A together with pleural effusion and ascites in which the patient fully recovered by supportive care after presenting to the hospital. *(J Pediatr Inf 2016; 10: 33-5)*

Keywords: Hepatitis A, pleural effusion, ascites

Introduction

Hepatitis A virus-associated (HAV) hepatitis is a frequent infection in developing countries. Clinical symptoms depend on the age of the host. Less than 30% of the infected infants and nearly 80% of the adults suffer from the symptomatic disease. Although hepatitis A is benign disease that heals without any sequelae, it may rarely end with fulminant hepatic failure. Following the two-six week incubation period, clinical symptoms such as fatigue, loss of appetite and nausea begin. Subsequently, hepatitis and darkness in the urine color is observed. Firstly clinical and then biochemical and histopathologic improvement is completed in a total of 6-12 months. Rarely extrahepatic complications such as arthralgia, cutaneous vasculitis, kriyoglobuline, hemophagocytic syndrome, acalculous cholecystitis, pacreatitis, aplastic anemia, Guillain-Barré syndrome, transverse myelitis, acute tabular necrosis, nephrotic syndrome, vasculitis, reactive arthritis and pleural effusion may develop (1). A pediatric case with pleural effusion and ascite during the course of symptomatic hepatitis A infection was presented; and this extrahepatic complication was discussed by reviewing the relevant Turkish and English literaure, this extrahepatic complication was discussed.

Case Report

A four and half-year of female patient was admitted with the complaints of fever and exhaustion that had started a week earlier. It was learnt form the history of the patient that the patient had an abdominal ache for three days and jaudince was detected. On the physical examination of the patient who was exhausted and had pale appearance, it was found that the patient had mild icterus, decrease in the breath sounds in the right hemithorax basal, and minimal sensitivity in the abdomen. The liver could not be palpated. In the laboratory tests, it was found that hemoglobin was 10.9 gr/dL, peripheral blood leukocyte count 10700/mm^3, C-reactive protein (CRP) 3.1 mg/L (n:0-8 mg/L), total bilirubin 6.6 mg/dL (n:0-2 mg/dL), direct bilirubin 4.8 mg/dL (n:0-0.2 mg/dL),

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aspartate aminotransferase (AST) 1180 U/L (n:<48 U/L), alanine aminotransferase (ALT) 2137 U/L (n:0-39 IU/L), gamma glutamyl transferase (GGT) 169 U/L (n:<23 U/L), lactate dehydrogenase (LDH) 326 IU/L (n:100-190 IU/L), alkaline phosphatase (ALP) 426 IU/L (n:110-302 IU/L), total protein 5.1 gr/dL (n:6-8 gr/dL), albumin 2.5 gr/dL (n:3.1-4.8 gr/dL), prothrombin time (PT) 21.5 seconds (n:10.6-14 seconds). Urine microscopy revelead 25 leukocytes and urine glucose was negative while urine bilırubin, protein and urobilinojen were positive. Urine culture was negative. There was no growth in the urine culture. It was found that serum enzyme-linked immunosorbent assay (ELISA) HAV immunoglobulin (Ig) M and anti-hepatitis B Ig G werepositive while HBsAg, anti-HBc IgM and IgG anti-HIV were negative. On the chest X-ray, there was a view that compatible with the pleural fluid in the right lung basal (Figure 1). In the ultrasonographic examination of the abdomen and thorax, it was observed that there were increase in liver parenchyma echo and heterogenic appearance; free abdominal fluid measuring 42 mm in the perihepatic, perisplenic zone; and in its deepest part in the pelvic region, and pleural effusion measuring 20 mm in the deepest part in the right hemithorax in the basal. On the microscopic examination of the pleural fluid obtained via thoracentesis for diagnostic purposes; it was found that there were 100 leukocytes/ mm^3 zone and 25 erythrocytes and 80% mononuclear cells in the methylene blue staining; in the biochemical examination, it was found that the pH was 7.37, glucose 90 mg/dL, and LDH 248 IU/L. Pleural fluid culture was negative. In the pleural fluid, HAV IgM was positive. The patient was hydrated with intravenous fluids and 5 mg of intramuscular vitamin K was given. AST 193 U/L (n:<48 U/L) regressed to ALT 754 U/L on the fourth day of hospitalization with the supportive care, and the control PT was normal. The patient was discharged from the hospital on the seventh day of the treatment when the pelvic fluid was regressed to 11 mm in the abdomen ultrasonography and no pleural fluid was detected on the thorax ultrasonography (Figure 2). On the outpatient clinical controls two months later, it was found that the liver enzymes and bilirubin values went totally back to normal.

Discussion

Severity of acute hepatitis A infection is related with the age of the patient. A infection is usually asymptomatic and anicteric in childhood. This is self-limiting disease and 85% of the patients recover completely in three months. Risk of mortality increases by age (2). Extrahepatic findings are seen in in 6.4-8% of children with acute viral hepatitis (1).

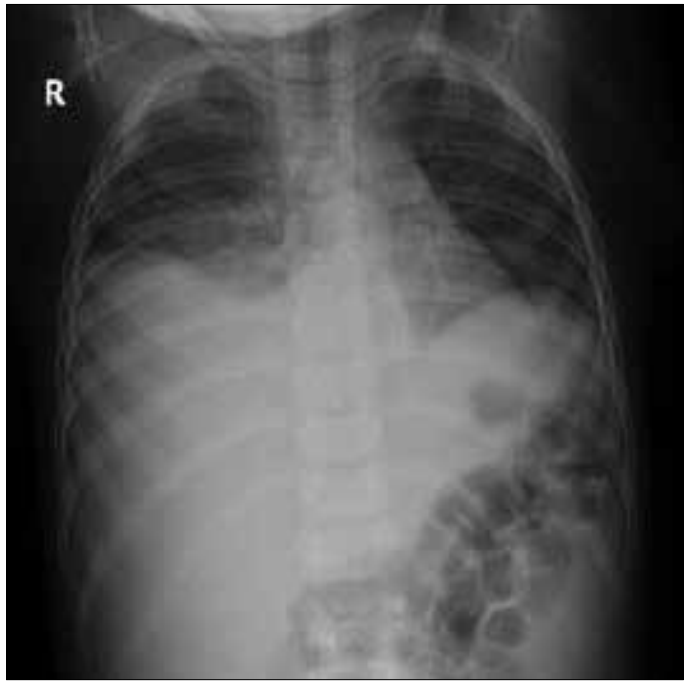

Figure 1. View of pleural effusion on the right

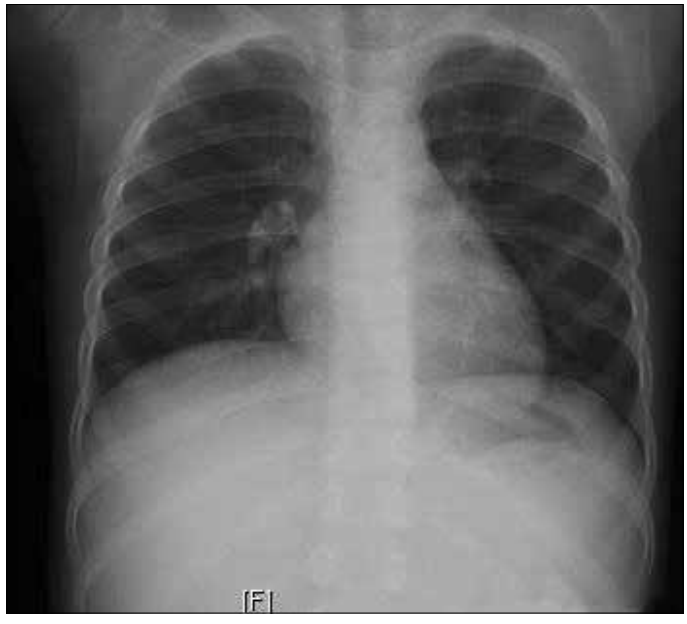

Figure 2. Normal chest x-ray in patient follow-up

In a study in which seventy eight children with acute HAV infection, it was found that 35 (44.9%) cases had ascite and 11 cases (14.1%) pleural effusion (3). Cases of hepatitis-associated had isolated ascite, cholecystitis together with pleural effusion and isolated pleural effusion have been reported (4). In the liver diseases, ascite may develop based on venous and lymphatic obstruction, or the situations that reduces plasma osmotic pressure such in as hypoalbuminemia (5). It was thought that hepatitis A-associated ascite was related to a temporary increase

in the portal venous or lymphatic pressure based on the compression of the hepatic sinusoids; related to the ascite in the pleural effusion, it was related to the direct passage of the fluid through diaphragmatic lymphatics or diaphragmatic defect (5). In our symptomatic and icteric case, the ascite accompanying the pleural effusion at the onset of the disease continued with a decreasing manner despite the loss of pleural effusion in the first week. Pleural effusion is a rare complication seen in the early period of acute viral hepatitis (5). Hepatitis A-associated pleural effusion was initially reported in an adult patient in 1971 and later in a five year old child in 1989 (6). Second and third hepatitis A-associated pleural effusion cases are the two patients aged 12 and 5 reported in 1999. In both cases, pleural effusion was detected in the right hemithorax and HAV IgM was positive in the pleural fluid. The authors concluded that although the pathogenesis of the effusion is uncertain, it is associated with the infectious inflammation of the liver more than immune complexes (7). Similarly in our case, HAV IgM was positive in the pleural fluid obtained from the right hemithorax. In a different case report of two patients aged 7, acalculous cholecystitis, bilateral pleural effusion and ascite that appeared as hepatitis A complication and healed spontaneously were reported; and it was thought that three serosal involvements occurred simultaneously depended on the roles played by the immune complexes in the pathogenesis (8). During the course of hepatitis A in an eight-year old patient, right hemithorax-located pleural effusion that healed spontaneously was reported. It was thought that the fact that the cases reported in this study were mostly in the right hemithorax might depend on a local inflammation more than a systemic incident; however, it was discussed that the existence of cases of bilateral pleural effusion without hypoproteinaemia supported this assessment (9). In our study, our case had a mild hypoproteinemia and hypoalbuminemia. In a case report of a six- year old patient, it was thought that autoimmunity was responsible in the pathogenesis of pleural effusion during the course of hepatitis A infection, because of the presence of thrombocytopenia, leukopenia, hypoproteinemia, coagulopathy and rashes with the right pleural effusion (10). Pleural effusion that appeared in our case at the beginning of icteric phase disappeared spontaneously at the end of the first week and at the end of the second month, the level of hepatic transaminases improved completely at the end of the second month. In Turkey, children and adult patients diagnosed with hepatitis A-associated pleural effusion and/or ascites were reported. Distinctive bilateral pleural effusion and ascite was reported in a six-year-old child (11). In this patient, hepatitis A RNA in the pleural fluid was screened by PCR, unlike the existing theories, it was though that pleural effusion might depend on the direct effect of pleural membrane of HAV RNA (11).

Conclusion

Although the exact mechanism of hepatitis A-associated pleural effusion is not completely known, inflammation-related there are some views suggesting that liver inflammation may be directly related to secondary ascite or the direct effect of HAV RNA on the immune complexes. Although our case had hypoproteinemia and hypoalbuminemia, it was thought that it was not at a sufficient level to explain ascite or pleural effusion; therefore, rather than a systematic reason, it might depend on local inflammation neighborhood and/or ascite; thus, the presence of HAV proof in the pleural fluid might be considered that pleural effusion may be due to viruses rather than immune complexes.

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Peer-review: Externally peer-reviewed.

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