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# Pleural Effusion, Ascites, and Thrombocytopenia Associated with Acute Hepatitis A Virus Infection

## Akut Hepatit A İlişkili Plevral Efüzyon, Asit ve Trombositopeni

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Dear Editor,

Although acute hepatitis A virus (HAV) infection is usually self-limiting, it may be rarely accompanied by extrahepatic manifestations such as evanescent rash, leukocytoclastic vasculitis, arthritis, glomerulonephritis, pleural effusion, ascites, cryoglobulinemia, autoimmune hemolytic anemia, and thrombocytopenia (1-3). The development of ascites may be associated with the transient elevation of pressure in portal and hepatic veins. It is caused by increase in hepatic sinusoidal pressure and decrease in oncotic pressure because of hypoalbuminemia. Pleural effusion is likely related to inflammation of the liver, immune complex mediated or transport of ascites to pleura through the lymphatics (4). Thrombocytopenia is associated with the accumulation of immune complex on the platelet surface (5). To our knowledge, this is the first case in which all these three extrahepatic manifestations were observed in a single adult patient.

A 35-year-old man admitted with a five-day history of nausea, vomiting, and jaundice. He had no comorbidities and no medical history. On physical examination the patient was icteric with normal vital signs, and liver was palpable 3 cm below the costal margin. Laboratory examination yielded the following; hemoglobin: 16.4 g/dL, white blood cell count: $10.3\times10^3$/μL, platelet count: $255\times10^3$/μL, ALT: 1970 U/L, AST: 1104 U/L, ALP: 102 U/L, GGT: 218 U/L, total bilirubin: 9.4 mg/dL, direct bilirubin: 6.9 mg/dl, total protein: 6 g/dL, albumin: 3 g/dL, prothrombin time: 17.5 s, and INR: 1.29. HAV immunoglobulin (Ig) M and HAV IgG were positive, whereas all viral markers for hepatitis B, C, D, and E were negative. Serological analyses for herpes simplex virus, Epstein-Barr Virus, cytomegalovirus, and parvovirus were negative. The right costophrenic angle was observed blunted in the chest X-ray. Abdominal ultrasonography and contrast-enhanced abdominal computerized tomography (CT) revealed ascites in perihepatic, perisplenic, and subdiaphragmatic areas, and acute hepatitis without biliary tract pathologies. In contrast-enhanced thorax CT, a right-sided pleural effusion with no parenchymal infiltration was observed. Supportive treatment was initiated. Until the 3rd day of admission, the platelet count decreased progressively to $60\times10^3$/μL, and it was verified by peripheral blood smear examination. Laboratory analyses, including direct and indirect Coombs tests, antinuclear antibodies, liver-kidney microsomal antibody, mitochondrial antibody, smooth muscle antibody, anti-soluble liver antigen, anti-cardiolipin IgM-IgG, anti-phospholipid IgM-IgG, protein electrophoresis, serum and 24-hour-urine copper, serum ceruloplasmin, and fecal alpha-1 antitrypsin did not reveal any abnormalities. Thrombocyte count gradually returned to normal values by the 11$^{th}$ day without treatment. Radiological examinations on the 11$^{th}$ day of admission showed regression of pleural effusion and ascites. Level of liver function tests decreased to completely normal values by the 18$^{th}$ day of admission.

Although the complications related to acute HAV infection is usually self-limiting and resolves spontaneously, as in the present case, it should be borne in mind that it may progress to a life-threatening condition in some cases.

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