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Case Report

Pancytopenia developed in the course of hepatitis A

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Hepatitis A has been reported with anemia, thrombocytopenia, leucopenia and rarely pancytopenia. A 5-year-old girl patient presented with complaints of yellowing of the eyes, abdominal bloating and abdominal pain 10 days ago. Skin and sclera were found to be icteric. Anti-HAV IgG and anti-HAV IgM tests were positive. In this case, we presented a 5-year-old girl who had pancytopenia with acute Hepatitis A infection as this case was rarely seen.

Key words: Hepatitis, child, infection, anemia, thrombocytopenia.

INTRODUCTION

Hepatitis A infection (HAV) is an infectious disease that is frequently encountered in childhood and rarely causes death, without a chronic course (Altindis et al., 2006). HAV can sometimes lead to significant complications. A large majority of these complications are hematologic complications such as leukopenia, leukocytosis, neutropenia, anemia, thrombocytopenia, thrombocytosis, and pancytopenia (Ulug et al., 2010). While anemia, thrombocytopenia, leukopenia have been reported hematologic disorders, pancytopenia has been reported very rarely (Venkataravanamma and Rau., 2004). In this case, we presented a 5-year-old girl who had pancytopenia with acute HAV infection as this case was rarely seen.

CASE REPORT

A 5-year-old girl patient presented with complaints of yellowing of the eyes, abdominal bloating and abdominal pain 10 days ago. It was stated that jaundice was increasing gradually and abdominal bloating was developed subsequently. The patient's history and family history were normal. There aren't a exposure to toxins or chemicals, blood transfusion or parenteral treatment in her history. Physical examination revealed moderate level of general condition, and that she was conscious and appeared to be exhausted. Skin and sclera were found to be icteric, conjunctivas were pale and turgor tonus of the skin was decreased. Abdominal examination revealed that the liver was palpable 3-4 cm below the subcostal margin. The examination of other systems was normal.

Laboratory studies were as follows; white blood cell count 1700 mm^3, hematocrit 25%, hemoglobin 5.7 g/dL, red blood cell count 3,080,000 mm^3, mean corpuscular volume 82 fL, and platelet count 26,000 mm^3. The rate of neutrophils was 48% and the rate of lymphocytes was 52% in peripheral blood smear. Red blood cells were found to be normochromic and normocytic, platelets were rarely seen and single and atypical cells were not seen. Liver function tests were as follows; aspartate aminotransferase: 131 U/L (N: 15-45 U/L), alanine aminotransferase: 164 U/L (N: 7-35 U/L), alkaline phosphatase: 369 U/L (N: 100-320 U / L), gamma-glutamyl transferase: 34 mg/dL, total serum bilirubin: 6,08 mg/dL and direct bilirubin: 0,8 mg/dL. The serum level of albumin, glucose, urea, creatinine and electrolytes were normal. Prothrombin time was 11 seconds. Sedimentation rate was 20 mm/h, and the level of C-reactive protein and fibrinogen were 3 mg/dL and 135 g/L, respectively. Anti-HAV IgG and anti-HAV IgM tests were positive. PCR could not technical inefficiency. There was negative result with the serologic tests for hepatitis B, hepatitis C, parvovirus, cytomegalovirus, and human immunodeficiency virus. Salmonella and brucella agglutination tests were negative. Bone marrow was heterogeneous and normocellular in structure. Bone marrow results not confirm aplastic anemia Abdominal ultrasound showed that parenchymal echo of the liver was decreased and gallbladder was slightly thickened.

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The case was considered to be pancytopenia induced by HAV and HAV infections. The patient was given frosh frozen plasma and red blood cell suspension. Follow-up values of the first week were as follows; white blood cell count 5500 mm^3, hemoglobin 10.5 g/dl, platelet count 134,000 mm^3 and anti-HAV IgM test negative. The patient is in the second month of follow-up without a symptom and hematologic parameters were normal.

DISCUSSION

Transient hematological disorders, such as bone marrow suppression can be seen during HAV infection (Cohen et al., 1993). However, pancytopenia after the HAV infection has rarely been reported in children. (Walia et al., 2006) presented a 12-year-old male patient who was diagnosed with pancytopenia through complete blood count and bone marrow smear. It was reported that pancytopenia was not seen in the study including 241 patients and investigating the complications of HAV infections. However, the rates of thrombocytopenia, thrombocytosis, anemia, leukopenia, leukocytosis and neutropenia were reported to be 5.8%, 7.8%, 11.4%, 2%, 10% and 5.8%, respectively (Ulug et al., 2012). In a study that was conducted by Taskesen et al. (Taskesen et al., 2008) and including 42 patients, thrombocytopenia (26.1%) and anemia (7.1%) were reported to be the other complications without pancytopenia. In a study that was conducted by Yasa et al. (Yasa et al., 2005) thrombocytopenia (11,3%), anemia (9,5%) and leukopenia (9,5%) were reported to be the other complications without pancytopenia. In a study that was conducted by Akarsu et al. (Akarsu et al., 2008) thrombocytopenia (4.3%), thrombocytosis (11.5%), anemia (15.5%), leukopenia (0,9%), leukocytosis (20.8%) and neutropenia (6.2%) were reported to be the other complications without pancytopenia. In a study conducted by Kanra et al. (Kanra et al., 2002) thrombocytopenia (11,3%), anemia (9,5%) and leukopenia (9,5%) were reported to be the other complications without pancytopenia. While pancytopenia was not reported in these studies, Venkataravanamma et al. (Venkataravanamma and Rau., 2004) reported pancytopenia with the rate of 0.9% in their study. In addition, thrombocytopenia (19.3%), anemia (12.5%), leukopenia (7.4%) and leukocytosis 10.8% were also reported to be hematologic complications (Venkataravanamma and Rau., 2004) .

In addition to the findings of the pancytopenia, detection of acute HAV infection and another infection that can cause pancytopenia, absence of a history of drug intake, normal being of the bone marrow structure, and detection of a full recovery of clinic and laboratory findings of pancytopenia during the recovery period of HAV infection suggested that pancytopenia was caused by HAV infection. Furthermore, the recovery of pancytopenia as well as the improvement of HAV infection has confirmed our diagnosis.

The hematological abnormalities induced by HAV infection were reported to be healed spontaneously without the need for aggressive treatment (Akarsu et al., 2008). In literature, it was reported that the patient with pancytopenia associated with HAV infection has recovered spontaneously without treatment (Walia et al., 2006). As in literature, our case was seen to be recovered spontaneously with symptomatic treatment.

In conclusion, we would like to remind that pancytopenia could develop due to HAV infection as in this case.

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