

Immune Thrombocytopenic Purpura Associated with Hepatitis A Infection in a Five-year Old Boy: A Case Report

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ABSTRACT

Acute hepatitis A infection is usually a self-limiting disease and mostly asymptomatic in children younger than six years old. Extrahepatic autoimmune manifestations such as immune thrombocytopenic purpura (ITP) have been reported rarely in children with acute hepatitis A infection. We report herein a paediatric case with ITP which is due to hepatitis A virus infection.

Keywords: Children, hepatitis A, infection, thrombocytopaenia, thrombocytopenic purpura

Púrpura Trombocitopénica Inmune Asociada con la Infección de Hepatitis A en un Niño de Cinco Años: Reporte de un Caso

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RESUMEN

La infección aguda por el virus de la hepatitis A es generalmente una enfermedad autolimitante y mayormente asintomática en niños menores de seis años de edad. Las manifestaciones autoinmunes extra-hepáticas, tales como la púrpura trombocitopénica inmune (PTI), raramente han sido reportadas en niños con infección aguda de hepatitis A. Reportamos aquí un caso pediátrico de PTI a causa de la infección del virus de la hepatitis A.

Palabras claves: Os niños, infección, hepatitis A, púrpura trombocitopénica, trombocitopenia

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INTRODUCTION

Acute hepatitis A infection is usually a self-limiting disease and is mostly asymptomatic in children younger than six years old. Extrahepatic autoimmune manifestations have rarely been reported in children with acute hepatitis A infection. Immune thrombocytopenic purpura (ITP) is also a benign and self-limiting disorder which may follow a recent viral infection or immunization in childhood. Immune thrombocytopenic purpura associated with hepatitis A virus (HAV) infection has been reported only in a small number of children in the literature (1–12). Immune thrombocytopenic purpura may be the sole manifestation of acute hepatitis A infection without other associated symptoms. We report herein a paediatric case with hepatitis A infection who was admitted to the Paediatric Emergency Department with severe thrombocytopaenia.

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CASE REPORT

A five-year old boy was referred to the Pediatric Emergency Department of Sisli Etfal Training and Research Hospital (Istanbul, Turkey) with thrombocytopaenia. He had oral lesions during the last week and bruising on both legs, purpuric, petechial lesions on his face, trunk and extremities had appeared over the last three days before admission. His two siblings had hepatitis A infection two months previously. He had no remarkable family history of haematologic disorder. He received no drugs and had no prior hospitalizations. He had no previous immunization record against hepatitis A.

On physical examination, he was alert, awake and anicteric. He had ecchymosis of the anterior aspect of both lower legs and petechiae all over his body. His weight was 15.1 kg (3–10th centile) and height 105 cm (10–25th centile). Body temperature (axillary) was 37.1 °C. Pulse rate was 88/minute and regular. Blood pressure was 80/40 mmHg. His oropharynx was hyperaemic and had non-ulcerated lesions. He had normal heart sounds and no murmur. Peripheral perfusion was normal. He had normal breath sounds bilaterally and respiratory rate was 16/minute. His abdomen was soft

and nondistended with no hepatosplenomegaly. He had no palpable lymphadenopathy. The remainder of the physical examination was normal.

The laboratory investigators revealed normal serum glucose, blood urea nitrogen, creatinine, total bilirubin, conjugated bilirubin, electrolyte levels, C-reactive protein and coagulation profile. Aspartate aminotransferase (AST) was 1940 U/L (normal: 15–55 U/L), alanine aminotransferase (ALT) 1503 U/L (normal: 5–45 U/L) and alkaline phosphatase (ALP) 360 U/L (normal: 100–300 U/L). The peripheral blood smear revealed severe thrombocytopenia. Complete blood cell count showed haemoglobin 12.5 g/dL, white blood cell count 5600/mm³ with a differential of 36% neutrophil, 60% lymphocyte and 4% monocyte and platelet count 4000/mm³. The laboratory findings are shown in the Table. Viral serologic studies were positive for anti-HAV IgM antibody and negative for anti-HAV IgG, hepatitis B and C, Epstein-Barr virus, cytomegalovirus, toxoplasma, rubella, and parvovirus B19.

Intravenous immunoglobulin (IVIG) with a 1 g/kg dose was given to the patient as a single dose for two days, and his platelet count increased to 43 000/mm³ on the third day and 265 000/mm³ on the second week. The patient remained haemodynamically stable and after a period of ward follow-up was discharged to the outpatient clinic. His clinical and biochemical profiles normalized in two weeks (Table). He is currently being followed up over the last five months, and has had no further history of thrombocytopenia.

is thought to be the result of bone marrow depression, immune-mediated peripheral destruction of platelets due to anti-platelet antibodies or circulating immune complexes or increased platelet consumption associated with disseminated intravascular coagulopathy (1–4, 13, 15).

Immune thrombocytopenic purpura was the sole manifestation of hepatitis A infection in some of the reported cases (4–7, 9, 11, 12). In others, thrombocytopenia was noted after or during the course of infection (2, 3, 10, 11, 13). Our patient was admitted with severe thrombocytopenia defined as a platelet count below 20 000/mm³ without any symptoms associated with hepatitis A infection.

Although life-threatening bleeding, such as intracranial haemorrhage, is rare in children with acute ITP, guidelines suggest children with platelet counts < 10 000/mm³ and minor purpura should be treated with specific regimens of IVIG or glucocorticosteroids. All of the cases reported except one (13) have been treated with IVIG or glucocorticosteroids. The cases that received treatment had a shorter duration of thrombocytopenia compared with the case which was only closely observed (14). Our patient received IVIG to increase the platelet count above 20 000/mm³ and shorten the duration of thrombocytopenia. His thrombocytopenia and biochemical profiles normalized gradually in two weeks.

In conclusion, ITP may actually be the only manifestation of viral hepatitis A. Therefore, it should be considered in the differential diagnosis of ITP in children.

Table: The laboratory findings

	Initial	3 rd day	2 nd week	2 nd month	
White blood cell count (mm ³)	5600	3300			(n: 6000–17 500)
Platelet count (mm ³)	4000	43 000	265 000	137 000	(n: 150–400 x 10 ³)
AST (U/L)	1940	1470	59	32	(n: 15–55)
ALT (U/L)	1503	1569	130	16	(n: 5–45)
GGT (U/L)	–	119	25	5	(n: 0–23)
LDH (U/L)	–	1159	–	–	(n: 100–290)
ALP (U/L)	–	373	235	195	(n: 100–300)
PT (s)	–	15.7	12	13.4	(n: 10–12.8)
aPTT (s)	–	30.6	35	30	(n: 28–40)
INR	–	1.3	1.0	1.14	(n: 0.8–1.2)

AST – aspartate aminotransferase; ALT – alanine aminotransferase; GGT – gamma glutamyl transferase; LDH – lactate dehydrogenase; ALP – alkaline phosphatase; PT – prothrombin time; aPTT – activated partial thromboplastin time; INR – international normalized ratio

DISCUSSION

Immune thrombocytopenic purpura is a self-limiting disorder which may follow a recent viral infection or immunization in childhood. Epstein-Barr virus, parvovirus, human immunodeficiency virus (HIV), and hepatitis C and B viruses (HCV, HBV) are among the viruses causing ITP. The occurrence of ITP due to hepatitis A virus infection is very rare. In the literature, a limited number of cases has been reported (1–14).

Although the exact mechanism is still unknown, immune-mediated thrombocytopenia in hepatitis A infection

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