Case Report

Rare manifestation of a common infection: Immune thrombocytopenic purpura due to hepatitis-A

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Abstract

Hepatitis A virus (HAV) is a benign self-limiting gastro intestinal infection of the children belonging to developing countries. Haematological complications like immune thrombocytopenic purpura (ITP) is rarely reported as a manifestation of acute hepatis A infection. We report a 6 year old female child with bleeding manifestations diagnosed to have ITP associated with acute hepatitis A infection.

Key words: Hepatitis A virus, Immune thrombocytopenic purpura, Bleeding

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Introduction

Acute hepatitis due to hepatitis A virus is usually a benign self-limiting disease during childhood. Many viral infections such as hepatitis B virus, Parvovirus, and Epstein-Barr virus are associated with extra hepatic autoimmune phenomena such as immune thrombocytopenic purpura, a plastic anemia, vasculitis, hemophagocytic syndrome. Immune thrombocytopenic purpura rarely manifests in patients with acute hepatitis A infection. So acute hepatitis A should be included in the differential diagnosis of thrombocytopenic purpura [1-3].

Case Report

A 6- year old previously well female child presented with history of fever, headache and vomiting for one week, jaundice for 3 days with passage of red coloured urine, black coloured stools and reddish spots all over body for one day prior to hospitalisation. There was no history of prodrome of typical infectious hepatitis, myalgia, arthralgia, sore throat, drug intake, recent travel or history of jaundice in close contacts. On examination child was conscious afebrile and icteric with no pallor. There was no lymphadenopathy and edema. She had petechiae all over the body with ecchymotic patch over left upper eyelid and forearm and haematuria was observed. On systemic examination moderate hepatomegaly and mild splenomegaly were present. In view of fever, jaundice and bleeding manifestations differentials considered were enteric fever, malaria, leptospira and viral hepatitis. Investigations at admission revealed normal hemoglobin and total leucocytecounts, platelet count 11000/cumm, reticulocyte count 1%. Total bilirubin was 6.5mg/dl (normal range 0.3-1.2) with a direct bilirubin of 6.3mg/dl (0.1-0.3) indirect bilirubin of 0.2mg/dl. Alanine (ALT) and aspartate aminotransferases (AST) were 381U/land 1338U/l (15–40 U/l), and alkaline phosphatase (ALP) was 385U/l (100–320 U/l).

Prothrombin time (PT), activated partial thromboplastin time (APTT), electrolytes, urea, and creatinine were normal. Serumferritin 625 ng/ml, serum triglycerides 191 mg/dl, LDH 666 U/L. Peripheral smear for malaria, serological tests for leptospirosis, typhoid, dengue and HIV were negative. Blood culture was sterile. Anti HAV IgM was positive (14.4OD units) but viral markers for hepatitis B and C were negative. Abdominal sonogram revealed pericholecystic edema and mild ascites. As the platelet count was 11000/cumm, single donor platelet transfusion (SDP) were given twice. As there was no improvement in platelet count even after 2 SDPs, immune mediated destruction of platelets was suspected and bone marrow aspiration was performed on day 3 of hospital admission which revealed normal trilineage haematopoiesis

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with megakaryocytic hyperplasia consistent with ITP. She was started on intravenous immunoglobulin's (IVIg) in view of fatal complications like intra cerebral haemorrhage. Platelet counts improved to 1.1lakh/cumm within 48 hrs and bleeding manifestations resolved gradually. She was discharged from the hospital with a platelet count of 2.3 lakh/cumm with improved liver function tests after 1 week of hospital stay. Child was brought for follow up after 2 weeks and at one month, her platelet count and liver function tests showed normal values at both visits.

Discussion

Immune thrombocytopenia is a benign self-limiting disease in children responding well to treatment and generally associated with viral infection [2]. Immune mediated extra hepatic manifestations and haemato-logical complications are mainly reported in adults with acute and chronic hepatitis B & C. Manifestations such as ITP, aplastic anemia, vasculitis, hemophagocytic syndrome are rare with hepatitis A infection. Till now very few number of cases of ITP associated with hepatitis Awere reported in the literature [1-3]. Tanir et al [1] reported a 5year old child with purpura and ecchymosis secondary to HAV infection that was recognised only on serological studies.

Decreased platelet count can be due to various causes like bone marrow depression, secondary to haemo-phagocytosis and immunologic destruction of platelets in the peripheral circulation secondary to circulating immune complex depositsor anti cardiolipin and anti-phospholipid antibodies or disseminated intravascular coagulopathy [3-6]. Thrombocytopenia may be the initial presentation [1,3] or may develop during the course of HAV infection in some of the reported cases [4]. In our case child was admitted with icterus, bleeding manifestations and with severe thrombocytopenia with platelet count 11000/cumm. Although life threatening bleeding such as intracranial haemorrhage is rare in children with acute ITP, guidelines suggest children with platelet count <10000/cumm should be treated with specific regimens like IVIg and steroids [7]. Our patient received IVIg as there is severe thrombo-cytopenia and for the prevention of fatal complications like intra cerebral haemorrhage. Her thrombocytopenia and biochemical profile normalised within 2 weeks. No evidence of hemophagocytic syndrome or bone marrow suppressionandincreased megakaryocytes in the one marrow examination and rapid response of platelet counts to immuno globulins suggests immune mediated destruction of platelets.

Conclusion

This case highlights that viral hepatitis A could be a cause for ITP and the importance of IVIg as a treatment option for ITP which can prevent fatal complications like intra cerebral haemorrhage.

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