Immune Thrombocytopenic Purpura secondary to Hepatitis A infection in a 15 years old girl

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Abstract: Acute hepatitis A infection is the most prevalent form and is responsible for most cases of acute and benign hepatitis. Fulminant hepatic failure is although rare. There had been few case reports of immune thrombocytopenic purpura (ITP) in children with acute hepatitis A infection. We therefore report a paediatric case of 15 years old female with Acute hepatitis A infection and ITP as an extrahepatic manifestations of the disease.

Keywords: Children, hepatitis A, infection, thrombocytopaenia

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I. Introduction

Acute hepatitis A infection is caused by RNA virus (picornavirus) which usually follows a self-limiting course. Hepatitis A virus (HAV) causes acute infections only and often it has a clinical course (anicteric illness) which is indistinguishable from other viral illness. There has been recent reports of extrahepatic autoimmune manifestations in children with acute hepatitis A infection in form of ITP. Immune thrombocytopenic purpura (ITP) is also a benign which may follow a recent viral infection or immunization in childhood. ITP associated with HAV infection has been reported only in a small number of children in the literature (1–12).

We report a case of 15 years old female who was admitted in paediatric ward of AIIMS Rishikesh with concerns of fever and yellowish discoloration of eyes and urine. On examination, she was found to be hemodynamically stable with icterus and her per abdomen examination revealed non tender hepatomegaly with no splenomegaly. There were no palpable lymph nodes and remainder of systemic examination were within normal limits. Her laboratory examination are as follows:

<table>
<thead>
<tr>
<th>Date</th>
<th>Hb (g/dl)</th>
<th>TLC (cells/mm³)</th>
<th>DLC N/L</th>
<th>Platelets (cells/mm³)</th>
<th>T. bil/ D. Bil (mg/dl)</th>
<th>SGOT/SGPT (U/L)</th>
<th>ALP/GGT</th>
<th>Total protein (g/dl)</th>
<th>Albumin (g/dl)</th>
<th>PT (sec)</th>
<th>INR</th>
<th>Urea/creatinine</th>
<th>Na/K/Ca</th>
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<td>3319</td>
<td>8049</td>
<td>10000</td>
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<td>1948/2158</td>
<td>692/27.5</td>
<td>5900</td>
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<td>17.8</td>
<td>1.32</td>
<td>18.3/0.62</td>
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<td>12.1</td>
<td>3000</td>
<td>N33/L39</td>
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<td>9.08/5.2</td>
<td>539/1170</td>
<td>246/53</td>
<td>5000</td>
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<td>13.8</td>
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<td>12000</td>
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<td>53/260</td>
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<td>N52/L30</td>
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<td>N46.4/L35.9</td>
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Table 1: Depicting laboratory parameters (complete blood count, Liver function test, kidney function test and PT and INR)

Her peripheral smear revealed normocytic normochromic red blood cells with severe thrombocytopenia and normal white blood cell count. Her HAV IgM was positive. Her HEV IgM and HBsAg were negative. She was given supportive management in form of proper nutrition and adequate rest. She was started on oral steroids (Prednisolone) at the dose of 2mg/kg/day and serial monitoring of platelet count was done. But despite oral prednisolone her platelet count remain dangerously low (5000 cells/mm³). She was then given intravenous immunoglobulin at the rate of 1g/kg/dose as a single dose over 2 days and platelet count was repeated after 48 hours and 96 hours which showed an improving trend (80000 and 1.08 lacs cells/mm³ respectively). Her other laboratory parameters (Liver function test and PT/INR) also showed an improving trend as depicted in table 1 above. She was then discharged in stable condition after 2 weeks of admission.

She is currently being followed up over the month, and has had no further history of thrombocytopenia.

It is considered to be the result of either bone marrow depression or 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). In some of the cases of acute hepatitis infection, very rarely ITP is the sole manifestation(4–7, 9, 11, 12). Although there are cases in which thrombocytopenia is noted during the course of illness (2, 3, 10, 11, 13). Our patient was admitted with features of acute hepatitis and on laboratory examination was found to have severe thrombocytopenia defined as a platelet count below 20000/mm³. Although life-threatening bleeding, such as intracranial haemorrhage, is not very common in children with acute ITP, but treatment should be vigourously prompted in children with platelet counts < 10000/mm³ and any evidence of bleeding. The mainstay of treatment of ITP are IVIG or glucocorticosteroids. All of the cases reported except one (13) have been treated with IVIG or glucocorticosteroids. It was noticed that the cases who are receiving treatment had a shorter duration of thrombocytopenia compared to the case which were only closely monitored (14). Our patient received both oral prednisolone in the starting and later on IVIG in view of inadequacy of expected response. Her thrombocytopenia and biochemical profiles normalized gradually in two weeks.

In conclusion, Hepatitis A virus can be the cause of ITP or ITP may actually be the only manifestation of viral hepatitis A.

II. 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 immuno-deficiency virus (HIV), and hepatitis C and B viruses (HCV, HBV) are among the viruses causing ITP. Although Hepatitis A virus is still not the common cause of ITP but possibility should always be considered. Very limited number of cases has been reported in the literature so far (1–14). Although the exact mechanism is still unknown, immune-mediated thrombocytopenia in hepatitis A infection can be considered as the mechanism.

References