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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. Wethereforereport 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 abenign which may follow a recentviral infection or immunization in childhood. ITP associated with HAVinfection has been reported only in a small number ofchildren in the literature (1–12).

We reporta 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:

Date	24/2/20	26/2	27/2	28/2	29/2	2/3	5/3	7/3
Hb (g/dl)	12.2	12.1	12.6	11.7	11.2	10.8	11.3	11.3
TLC (cells/mm^3)	5319	5000	5800	4175	8400	8300	9100	10300
DLC	N39.4/L33.7	N36.5/L39	N35/L59	N40/L17	N52/L30	N68/L22	N47/L36	N46.4/L35.9
Platelets (cells/mm^3)	8049	10000	5000	12000	12000	5000	80000	1.08 lacs
T. bil/ D. Bil (mg/dl)	7.29/4.22	9.08/5.2				3.2/1.5		
SGOT/SGPT (U/L)	1948/2158	539/1170				53/260		
ALP/GGT	692/27.5	246/53				255/397		
Total protein (g/dl)	5.9	6.7				7.5		
Albumin (g/dl)	3.2	3.1				3.4		
PT (sec)	17.8					8.8		
INR	1.32					0.75		
Urea/creatinine	18.3/0.62							
Na/K/Ca	128/3.95/9.3	135/3.9/-						

Table 1: Depicting laboratory parameters (complete blood count, Liver function test, kidney function test and PT and INR)

{Hb: Hemoglbin, TLC: total leukocyte count, DLC: Differential leukocyte count (N: neutrophils and L: lymphocytes), T.bil/D.bil: tota and direct Bilirubin, SGOT/SGPT: serum glutamic oxaloacetic transaminase/ serum glutamic pyruvic transaminase, ALP/GGT: Alkaline phosphatase/ gamma glutamyl transferase, PT: prothrombin time, INR: International normalised ratio, Na/K/Ca: sodium/ Potassium/ Calcium.}

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/mm^3). 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/mm^3 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 iscurrently being followed up over the month, andhas had no further history of thrombocytopaenia.

It is considered to be the result of either bone marrow depression or immune-mediatedperipheral destruction of platelets due toanti-platelet antibodies or circulating immune complexes orincreased platelet consumption associated with disseminatedintravascularcoagulapathy (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 thrombocytopaenia

defined as a platelet count below 20000/mm^3. Although life-threatening bleeding, such as intracranialhaemorrhage, 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 reportedexcept one (13) have been treated with IVIG orglucocorticosteroids. It was noticed that the cases who are receiving treatment had ashorter duration of thrombocytopaenia compared to thecase 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. Herthrombocytopaenia and biochemical profiles normalizedgradually in two weeks.

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

II. Discussion

Immune thrombocytopenic purpura is a self-limiting disorderwhich may follow a recent viral infection or immunization inchildhood. 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 thrombocytopaenia in hepatitis A infection can be considered as the mechanism.

References

[1]. Ertem D, Acar Y, Arat C, Pehlivanoglu E. Thrombotic and thrombocytopenic complications secondary to hepatitis A infection in children. Am J Gastroenterol 1999; 94: 3653–5.

[2]. Ertem D, Acar Y, Pehlivanoglu E. Autoimmune complications associated with hepatitis A virus infection in children. Pediatr Infect Dis J2001; 20: 809–11.

[3]. Avci Z, Turul T, Catal F, Olgar S, Baykan A, Tekfam O et al.Thrombocytopenia and emperipolesis in a patient with hepatitis Ainfection. PediatrHematol Oncol 2002; 19: 67–70.

[4]. Tanir G, Aydemir C, Tuygun N, Kaya O, Yarali N. Immunethrombocytopenic purpura as sole manifestation in a case of acutehepatitis A. Turk J Gastroenterol 2005; 16: 217–19.

[5]. Leblebısatan G, Tumgor G, Sasmaz I, Ozgur O, Antmen B. Hepatitis A-associated immune thrombocytopenia. Turk J Gastroenterol 2012; 23:195–7.

[6]. Venkataravanamma P, Rau AT. Severe thrombocytopenia in associationwith hepatitis A. Indian Pediatr 2004; 41: 1178–9.

[7]. Scott JX, Gnananayagam EJ, Gupta S, Simon A, MukhopadhyaA.Thrombocytopenic purpura as initial presentation of acute hepatitis A.Indian J Gastroenterol 2003; 22: 192–3.

[8]. Choulot JJ, Coquard JL, Pariente A, Saint-Martin J, Mensire A.[Hepatitis A and immune thrombocytopenic purpura]. Arch Pediatr1994; 1: 213–4.

[9]. Shenoy R, Nair S, Kamath N. Thrombocytopenia in hepatitis A – anatypical presentation. J Trop Pediatr 2004; 50: 241–2.

[10]. Hein N, Luizetto CM, Sato ME. Immune thrombocytopenic purpuraassociated with hepatitis – a case report. Pediatria 2004; 26: 59–62.

[11]. Gultekingil A, Yarc E, Unal S, Kara A. Immune thrombocytopenicpurpura during the course of acute hepatitis A virus infection. Infect DisClinPrac 2009; 17: 331–2.

[12]. Yildirim ZK, Buyukavcı M. Thrombocytopenia with findings of acute hepatitis A infection: case report. J PediatrInf 2011; 5: 80–2.

[13]. Ibarra H, Zapata C, Inostroza J, Mezzano S, Riedemann S. Immunethrombocytopenic purpura associated with hepatitis A. Blut 1986; 52:371–5.

[14]. Jose W, Kumar K, Unnikrishnan A, Palaniappan M, Nair A, PavithranK. Autoimmune thrombocytopenic purpura in acute hepatitis A – areport and review of literature. Asia Pac J Oncol Hematol 2010; 2: 137.

[15]. Stasi R, Chia LW, Kalkur P, Lowe R, Shannon MS. Pathobiology andtreatment of hepatitis virus-related thrombocytopenia. MediterrJHematol Infect Dis 2009; 25: e2009023. doi: 10.4084/MJHID.2009.023.

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