LETTER TO EDITOR (VIEWERS CHOICE)

METHEMOGLOBINEMIA DUE TO HEPATITIS A VIRUS IN A PATIENT WITH G6PD DEFICIENCY

Naresh Tayade, Manjusha Deotale***

Keywords: Methemoglobinemia, Glucose-6-phosphate deficiency, hemoglobinuria, hepatitis A infection

A 5 years old male child presented with fever for five days and red color urine along with vomiting since morning. No history of rash, impetigo, throat pain, joint pain was present. He had been treated with oral antibiotics and chloroquine. On presentation, patient was afebrile, had tachycardia (heart rate – 160/min), icterus, pallor with duskiness around mouth. Respiratory rate was 30/min. There was no clubbing and blood pressure was normal. Oxygen saturation (SpO2) on pulse oxymetry was 76% at room air which did not increase with oxygen. Other systems were normal. Urine was brown red in colour. Investigations at time of admission showed haemoglobin of 9 gm% with total leukocyte count 18,800/cumm and normal platelets. Liver function tests were not done. The colour of blood was chocolate brown. Peripheral smear showed fragmented red blood cells (RBCs) and features of hemolysis like spherocytes, fragmented cells & blister cell. Urine examination showed red colour urine with no red blood cell suggesting possibility of hemoglobinuria. Glucose 6 phosphate dehydrogenase (G6PD) level was 1.6 U/gHb (Normal: 6.4-18.7 U/gHb). Arterial blood gas (ABG) showed normal Pao2 (104) and Sao2 99% indicating paradoxical elevation of Po2 despite clinical cyanosis and a normal calculated oxygen saturation. Methemoglobin level could not be calculated due to technical issues. Bedside test on white filter paper by placing 2 drops of blood: one form patient and other form normal person showed that the patient's blood colour remained as it is while that from normal person brightened with exposure to oxygen. Patient was started on hydration along with antibiotics. However, abdominal pain, vomiting, hematuria, low oxygen saturation persisted and progressive anemia was noted. Repeat haemoglobin was 6 gm%. AST and ALT were 337 IU/L and 465 IU/L. Hepatitis A IgM was positive while HbsAg was negative. Patient was given blood transfusion and supportive treatment. Subsequently SpO2 reached 100% on room air on Day 8 with no tachycardia, abdominal pain or vomiting.

Patients with G6PD deficiency present in many ways. Our patient presented with hemolysis complicated by hepatitis due to hepatic A virus (HAV) infection. Similar features have been described in the far East. (1,2) In G6PD deficiency oxidative drugs can cause hemolysis or methemoglobinemia rarely at same time. (3,4) In our case methemoglobinemia, was noted without any use of oxidant agent. In literature search we got only adult case with this type of presentation with hepatitis E virus infection, (5) though there are many cases developing methemoglobinemia due to oxidant drugs. Methemoglobinemia patient presents with cyanosis that fails to respond to supplemental oxygen, a lack of significant pulmonary findings and clear chest radiograph. A distinguishing feature of methemoglobinemia on an arterial blood gas analysis is the paradoxical elevation of PO2 despite clinical cyanosis and normal calculated oxygen saturation. (6) The treatment of choice for severe acute methemoglobinemia is methylene blue. (6) However, in G6PD deficiency subjects, methylene blue should be used with caution as it can cause hemolysis, and paradoxically methemoglobinemia. (6) The most effective treatment in severely G6PD deficient patients with life threatening methemoglobinemia may be exchange transfusion. (7) However many patients have only partial enzyme deficiency in them and methylene blue is still the first line treatment and exchange transfusion is reserved only for selected patients. We managed this patient only with supportive care and blood transfusion.

Contributor Statement

NT diagnosed, worked up and managed the case and wrote the manuscript. MD reviewed the literature, prepared the final manuscript and followed up the case.

Funding : None

Conflict of Interest : None

References :

1. Chan TK. Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency: A Review Available form:www.cchi.com.hk/specialtopic/case1/case1.htm. Accessed May 23,2013.
2. Chan TK, Todd D. Hemolysis complicating viral hepatitis in patients with glucose-6-phosphate dehydrogenase deficiency. Br Med J. 1975; 1:131-133.
3. Abid S, Khan AH. Severe hemolysis and renal failure in glucose-6-phosphate dehydrogenase deficient patients with hepatitis E. Am J Gastoenterol. 2002;97:1544-1547.
4. Schurman M, Waardenburg DV, Costa JD, Niemarkt H, Leroy P. Severe hemolysis and methemoglobinemia following fava beans ingestion in glucose-6-phosphatase dehydrogenase deficiency- case report and literature review. Eur J Pediatr. 2009;168:779-782.
5. Wing YA, Chun WN, Chan WM, Leung RYY, Chan SC. Hemolysis and methemoglobinemia due to hepatitis E virus infection in patient with G6PD deficiency. Ann Hematol. 2011; 90:1237-1238.
6. Wright RO, Lewander WJ, Woolf AD. Methemoglobinemia: Etiology, Pharmacology, and Clinical Management. Ann Emerg Med. 1999;34:646-652.
7. Harrison MR. Toxic methemoglobinemia. A case of acute nitrobenzene and aniline poisoning treated by exchange transfusion. Anaesthesia. 1977; 32:270-272

From: *Department of Pediatrics, Life Care Neonatal and Pediatric Critical Care Centre Amravati, Maharashtra, India and **Department of Community Medicine, Dr Dr Panjabrao Deshmukh Memorial Medical College, Amravati, Maharashtra, India.

Address for Correspondence: Dr. Naresh Tayade, Meghdootnagar, Near Amar Colony, Amravati (Maharashtra), 440606, India.

Email: nt_2005@rediffmail.com

DOI No. 10.7199/ped.oncall.2015.38