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Case Report Open Access

Hematite Ingestion in G6PD Deficiency: Case Report

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Case Report

We report a case of severe hemolysis due to G6PD deficiency manifesting as methemoglobinemia in a 3.5-year-old Punjabi male, not known to have any earlier hemolytic episodes or diagnosis of G6PD deficiency.

A 3.5-year-old boy presented to the Emergency Department with history of fever, irritability, yellowish discoloration of eyes and red color urine for 2 days. There was significant history of intake called 'Geru', a local remedy for urticaria. Patient was referred to the us with increasing dark color urine, in view of ongoing hemolysis and persistent desaturation. At the time of admission, examination revealed pulse rate-135 beats per minute, respiratory rate-25 per minute, blood pressure -138/60 mmHg, SpO_2-66% on non-rebreathing face mask @ 15 L/min of O_2. Child was pale, icteric, centrally pink, and irritable, not in distress, conscious, oriented and responding to commands. Abdominal examination revealed hepatomegaly. Cardiovascular and respiratory system examination were essentially normal.

Hematology	
Hemoglobin	9.1 gm/dl
Platelet count	3.6 lac/mm^3
Total leucocyte count	21360 (P-80%, L- 10%, M-1%, E-9%)
Peripheral smear	Suggestive of hemolysis
G-6-PD	Quantitative deficiency
Biochemistry	
Na	131 mEq/L
K	4.8 mEq/L
Blood urea nitrogen	38 mg/dl
Creatinine	0.6 mg/dl
Lactates	1.54
Coagulation parameters	
PT-INR	0.9
APTT	35 sec

Table 1: Depicting hematological and biochemical parameters at admission.

Venous blood gas (VBG) analysis at admission revealed pH-7.41, PaO_2-22 mmHg, PCO_2-29 mmHg, Bicarbonate-18 mEq/L, Lactate-1.54 mmol/L, and methemoglobin-21.3%. His hemoglobin revealed a drop of 1.6 gm/dl since afternoon.

Further investigations revealed hemoglobinuria, features of hemolysis in peripheral smear, increased reticulocyte count (6.8). Direct Coomb's test was negative, Coagulation profile was normal, Liver function tests were deranged, while renal function tests were normal (Table 1). There was quantitative deficiency of G6PD enzyme (1.36 against a normal value of 7.0 to 20.5). Iron studies revealed serum iron of 497 mcg/dl (normal: 45-182), transferrin saturation was 93.2% (normal: 17.0-37.0). The overall picture was consistent with an active hemolysis due to G6PD deficiency.

He was started on double maintenance intravenous fluid, furosemide and soda bicarbonate (target pH 7.5) to prevent progression of acute kidney injury secondary to hemolysis.

As patient had a very high WBC and fever, he was covered with broad-spectrum antibiotics but later stopped, as all cultures were sterile.

Methylene blue was not given as it is contraindicated in G6PD deficiency. Serial VBG (Table 2) revealed decline in methemoglobin levels following fluid therapy.

On third day of admission, hemolysis subsided. He was shifted to ward on fourth day.

Time (hours)	**At admission**	**5 hours**	**9 hours**	**24 hours**	**36 hours**	**48 hours**
Methemoglobinemia On co-oximetry	21.3%	18%	8.2%	7%	1.6%	0.8%

Table 2: Depicting the methemoglobinemia levels of the patient by co-oximetry during hospital stay.

Discussion

Geru is a red coloured product containing ferric oxide, which is used to color pots, jewelry and ground nuts. It is pounded into fine powder before use. It is commonly taken as a decoction. There are many problems that may result from iron toxicity, these include: anorexia, oliguria, diarrhea, hypothermia, diphasic shock, metabolic acidosis, and death. In addition to these, the patient may experience vascular congestion of the gastrointestinal tract, liver, kidneys, heart, brain, spleen, adrenals, and thymus [1].

Severe hemolysis due to G6PD deficiency may manifest as methemoglobinemia, [2] in which the heme iron is in the oxidized ferric state rather than the ferrous state [3]. This resultant hemoglobin,

known as methemoglobin (met-Hb), cannot carry oxygen and the remaining oxyhemoglobin develops increased oxygen affinity resulting in impaired oxygen delivery. This results in a left shift of the oxygen–hemoglobin dissociation curve and secondary tissue hypoxia. Normal people generate met-Hb but in very low levels in the range of 0.5% to 3% [4].

Plasma exchange therapy, hyperbaric oxygen therapy and ascorbic acid therapies should be considered as second-line treatments for patients of methemoglobinemia unresponsive to methylene blue [5].

Even though, evidence for plasma exchange in literature is limited to case reports [6], this therapy was not given to our patient, as levels started decreasing without any major intervention.

There is a specific antidote, desferroxamine for hematite toxicity, but we didn't use in our patient as child improved clinically after admission on symptomatic management only. Gastric lavage was not done as the patient presented after 24 hours of ingestion.

As per our knowledge, no case has been reported till date to have hemolysis and methemoglobinemia secondary to G6PD deficiency after hematite ingestion.

Conclusion

Even though ferric oxide poisoning is common in children, complications such as intravascular hemolysis and methemoglobinemia may coexist. Special care should be taken while handling such cases and all the possibilities should be considered before intervention. Timely referral to a higher center that has a facility of secondary management for suspected methemoglobinemia is required.

References

1. (2012) Iron Toxicity, What You Don't Know, Plants Poisonous to Livestock. Cornell University Department of Animal Science.
2. Clark BB, Morrissey RW (1951) Relation of methemoglobin to hemolysis. Blood 6: 532-543.
3. Skold A, Cosco DL, Klein R (2011) Methemoglobinemia: pathogenesis, diagnosis, and management. South Med J 104: 757-761.
4. Taber KH, Migliore PJ, Pagani JJ, Ford JJ, McLauren T, et al. (1990) Temporal changes in the oxidation state in in vitro blood. Invest Radiol 25: 240-244.
5. Rees SM, Nelson LS (2004) Dyshemoglobinemias In: Emergency Medicinea comprehensive study guide (6thedn.) McGraw-Hill, New York, pp. 1169-1171.
6. Sachdev A, Gulla KM, Anand K, Raheja K, Gupta N, et al. (2008) Transfluthrin Poisoning Resulting in Intra Vascular Haemolysis and Methemoglobinemia in G6PD Deficiency-Treatment Challenge. J Clin Toxicol 5: 76-77.