

Petrol Ingestion Causing Methaemoglobinaemia in Glucose 6-Phosphate Dehydrogenase (G6PD) Deficiency Patient

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Abstract

A 31-year-old male found unconscious in his parked car on road side was admitted for evaluation and management in the hospital. The patient presented with central and peripheral cyanosis and arterial blood gas (ABG) report revealed 54.4% oxyhaemoglobin, 44.5% methaemoglobin and 1.1% carboxyhaemoglobin. The patient was put on 100% oxygen. Keeping in view the ABG report, the patient was treated as methaemoglobinaemia and started on 1% methylene blue solution intravenously. The patient, on regaining consciousness, told that he had accidentally ingested petrol a few hours back. Later on, further investigations revealed that the patient had bite cells in peripheral blood smear suggestive of hemolytic anaemia. The patient informed that he is a known case of Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency. The patient was managed as a case of petrol ingestion methaemoglobinaemia with G6PD deficiency and discharged from hospital.

Keywords: G6PD Deficiency; Methaemoglobinaemia; Methylene Blue; Petrol Ingestion.

Case Report

A 31-year-old male was found unconscious in his parked car on road side. He was brought to A & E department and admitted in the hospital for evaluation and management. The patient presented with central and peripheral cyanosis, bilateral crackles at the bases of lungs with spleen 3 cm below costal margin. Arterial blood gas (ABG) report revealed 54.4% oxyhaemoglobin, 44.5% methaemoglobin and 1.1 % carboxyhaemoglobin. While the blood samples were being drawn, the sister-in-charge noticed the chocolate-brown colour of blood and informed the treating physician. The G6PD level estimation and High Performance Liquid Chromatography (HPLC) could not be done as these facilities were not available in the hospital. The laboratory parameters on admission were: Haemoglobin 115 g/L, Haematocrit 33%, C-reactive protein (CRP) 72 mg/L (Normal 0-5 mg/L), direct Coomb's test (DCT) and sickling tests were negative.

The patient developed jaundice and his serum lactic dehydrogenase (LDH) levels increased which returned to normal after a few days. The reports of laboratory tests/ABG are tabulated (Table 1). The urine of the patient was dark in colour. Renal function tests were normal. Chest X-ray showed bilateral haziness at bases and CT chest and abdomen showed bilateral lower lobe consolidation (probably due to aspiration) and spleen 15 cm long in long axis. Electrocardiography (ECG) was within normal limits. Keeping in view the ABG report and his oxygen saturation levels and non-availability of G6PD level estimation and HPLC chromatography, the patient was treated as a case of methaemoglobinaemia and was started on IV 1% methylene blue solution and put on 100% oxygen. Methylene blue (1 mg/kg body weight) was given in the dose of 50 mg IV slowly over 5 minutes and after 30 minutes each two more IV doses of methylene blue 50 mg IV were given (Total 3 doses of methylene blue 50 mg each were administered). The patient was also given Tazobactam/piperacillin 4.5 g thrice a day for five days and initially Inj.

Omeprazole 40 mg twice a day IV and the shifted on oral omeprazole besides IV dextrose saline. The patient, on regaining consciousness, told that he had accidentally ingested petrol a few hours back and he is a case of G6PD deficiency. Peripheral blood smear revealed mild anisocytosis, normocytic normochromic polychromasia with occasional nucleated RBC and presence of bite cells suggestive of hemolytic anaemia due to G6PD deficiency (Figure1).

Levels of benzene were not measured in blood and urine due to non-availability of facilities. The patient received five units of blood and fresh frozen plasma during hospitalization. The patient was managed as a case of methaemoglobinaemia with underlying haemolytic anaemia due to G6PD deficiency and discharged from hospital.

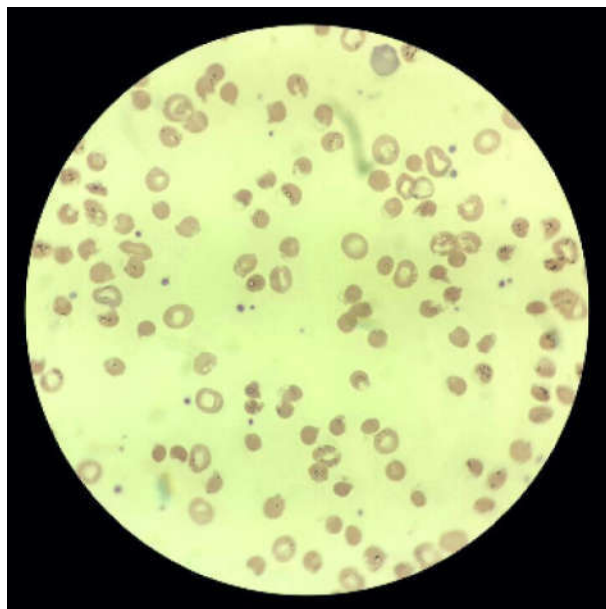


Fig. 1: Peripheral blood smear showing mild anisocytosis, normocytic normochromic polychromasia, occasional nucleated RBCs and bite cells (Wright Stain, 10x100 Magnification)

Discussion

Petrol is a life-line for any developed or developing country. Countries like USA and Europe have their petrol (gasoline) marketed with 1-5% benzene [1,2]. Sultanate of Oman, one of the middle East countries is also marketing petrol (gasoline) with less than 5% benzene content [3].

Benzene is acutely toxic by inhalation, causing mucous membrane irritation, neurological and other symptoms due to respiratory failure. Chronic exposure has been reported to result in bone marrow depression, aplasia and leukaemia, cardiac abnormalities, heart attack and other cancers of lung, brain and stomach. Following inhalation, benzene vapour is rapidly absorbed into the blood and distributed throughout the body. One of the effects of benzene in the body is the production of methaemoglobin (MetHb) which contains iron in ferric state (Fe^{3+}) [4].

Methaemoglobinaemia is a rare condition characterised by increased quantities of haemoglobin in which the iron of haem is oxidised to the ferric (Fe^{3+}) form. Clinically the condition presents with cyanosis and low oxygen saturations on pulse oximetry but normal oxygen saturation on arterial blood gas analysis. Most cases are acquired and are frequently drug-related.

Udonwa NE et al [5] studied the exposure of petrol station attendants and auto-mechanics to premium motor spirit fumes in Nigeria and suggested increased exposure to petrol fumes among automobile mechanics, petrol station attendants and MetHb as a useful biomarker in determining the level of exposure to benzene in petrol vapour.

Our patient had ingested petrol by accident and as petrol is volatile, some of the petrol may have gone into the respiratory tract causing bilateral consolidation and chemical pneumonitis.

Table 1: Relevant ABG/ blood parameters of the patient on Day 1-3 in the hospital

Relevant Blood/ ABG Parameters	Normal Range in our Hospital	Day 1 1346 hrs	Day 1 1949 hrs	Day1 2134 hrs	Day 1 2344 hrs	Day 2 0705 hrs	Day 2 1847 hrs	Day 3 1448 hrs
Met Hb	(0.0-1.5%)	44.5%	30.0%	26.7%	25.6%	15.0%	9.8%	9.7%
O ₂ Hb	(94.0-98.0%)	54.4%	68.0%	71.7%	72.4%	83.0%	87.3%	82.5%
CO Hb	(0.0-3.0%)	1.1%	1.3%	1.5%	1.8%	2.0%	2.9%	5.1%
SO ₂	(94.0-98.0%)	100%	99.1%	99.8%	99.7%	100%	100%	96.9%
Hb	(11.5-17.8g/ dL	12.7g/ dL	11.6g/ dL	11.1g/ dL	10.3g/ dL	8.1g/ dL	8.1g/ dL	7.3g/ dL
Hct	(36.0-53.0%)	41.8%	38.5%	37.5%	34.5%	29.6%	29.2%	27.4%

Table 2: Signs and symptoms associated with different levels of methaemoglobin in blood [6]

Methaemoglobin levels	3-15%	15-25%	25-50%	50-70%	Above 70%
Signs and symptoms	Pale, gray or blue discoloration of the skin may be present	Mild cyanosis otherwise relatively asymptomatic	Headache, dyspnoea, lightheadedness, syncope, weakness, confusion, palpitations, chest pain	Cardiovascular- Abnormal cardiac rhythms CNS- Altered mental status; delirium, seizures, coma Metabolic- Profound acidosis	Death

Acute methemoglobinemia can be life-threatening and usually is acquired as a consequence of exposure to toxins or drugs. Therefore, obtaining a detailed history of exposure to methemoglobinemia-inducing substances is important. Such history may not always be forthcoming, but it should always be sought actively since long-term or repeated exposure may occur. Consultation with a toxicologist may be necessary, especially with exposure to a new medication, because the list of medications known to cause methemoglobinemia changes constantly. Symptoms are proportional to the fraction of methemoglobin. A normal methemoglobin fraction is about 1% (Range 0-3%). Symptoms associated with various levels of methaemoglobin are shown (Table 2)[6].

G6PD deficiency, the most common human enzymopathy, affects 10% of the world's population, causing haemolysis due to intake of various drugs and other conditions [7]. G6PD deficiency is common in Oman with the G6PD Mediterranean mutation accounting for most cases [8].

Clarification regarding known family history of methemoglobinemia or glucose-6-phosphate dehydrogenase (G6PD) deficiency is important. Even patients who are heterozygous for methemoglobin reductase enzyme deficiencies are susceptible to low doses of oxidant drugs with resultant methemoglobinemia. In our case report, we were unable to ask the history at the time of admission to rule out G6PD deficiency as he was found in an unresponsive state. Since the facilities were not available at the hospital to assess the level of percentage of G6PD deficiency and HPLC chromatograph, it was decided to treat the patient with IV 1% methylene blue solution.

Conclusion

The case report is unique because of accidental ingestion of petrol by the patient and lying unconscious at roadside in his car. The patient's

health status was further complicated by his being a G6PD deficiency patient which was unknown till he regained consciousness. Patients of G6PD deficiency should be encouraged to carry an identity card or bracelet which may be life-saving and help them getting the best treatment in emergency situations.

Conflict of Interest and Funding

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