

A Case of Nitrate Induced Methemoglobinemia in 17 Years Old Boy

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ABSTRACT

Methemoglobinemia is life threatening emergency. It is bizarre presentation of oxygen delivery form of hemoglobin and presents with non-specific clinical features. Oxygen at lung site bind with heme iron in Fe+2 form while transport in Fe+3 form. At cellular level need further reduction to Fe+2 form for release of oxygen. Increased methemoglobin leads to functional anemia. The oxygen-dissociation curve shift to the left. We present a case of methemoglobinemia in 17 years old boy

Keywords: Nitrate; Methemoglobinemia; Boy

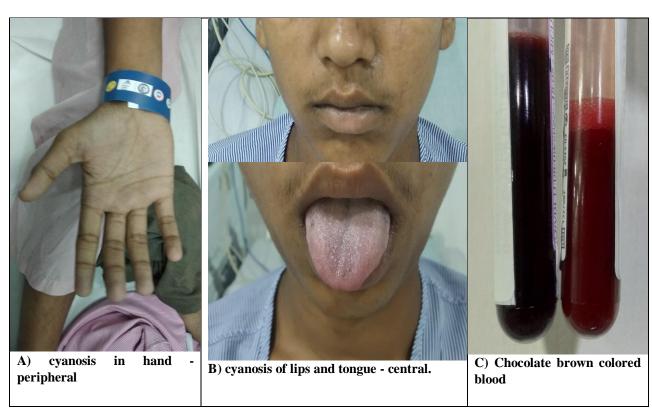
INTRODUCTION

Normally methemoglobin level in blood is 0% to 2%. This level is maintained by cytochrome B5 reductase (CYB5R), NADPH-MetHb and glutathione. Cytochrome-B5 reductase utilizes NADH formed during glycolysis to reduce methemoglobin back to functional hemoglobin. An alternate pathway for the reduction of methemoglobin is through the function of nicotinamide adenine dinucleotide phosphate hydrogen methemoglobin (NADPH-MetHb) reductase. NADPH-MetHb reductase utilizes NADPH that forms through the action of glucose-6-phosphate

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dehydrogenase (G6PD) in the hexose monophosphate shunt, for its reducing power.^[1,2] NADPH-MetHb reductase reduces methylene blue to leukomethylene blue using NADPH from the G6PD-dependent hexose monophosphate shunt. Here Leuco methylene blue act as electron donor which converts methemoglobin to Functional Hb. Methemoglobinemia may be acquired or congenital. Such as drugs used in Anaesthesia and other environmental factors are responsible for acquired causes. Clinical Presentation depends on the level of Methemoglobin^[3]. Cyanosis Starts to appear at 10%. Chocolate brown colored blood at 15% ^[4]. Upto 20% Patient may feel anxiety and light headaches. Tachypnea, confusion and loss of Consciousness occurs upto 50%. Around 50% it can cause seizures, dysrhythmias, metabolic acidosis, and coma. Above 70% it is fatal. ^[4,5]



CASE PRESENTATION

A 17 years old boy presented to emergency department with complain of shortness of breath, palpitation with bluish discoloration of peripheral extremities, tongue and lips. On physical examination patient was tachypneic, diaphoretic, cold and and clammy skin with presence of mixed cyanosis. Vitals were blood pressure 150/78 mm Hg, 150 pulse per minute, Spo2 84% on room air. Arterial blood gas analysis showed metabolic acidosis with methemoglobinemia (MetHb-39.9%). Patient was put on O2 mask (6L/min), but no improvement shown in Spo2. In the emergency room cardiorespiratory causes were ruled out by history, 2D echo and chest x ray. In the emergency room methylene blue 50mg in 100ml isotonic normal saline was given. Patient was shifted to critical care unit. In critical care unit a detailed drug history was taken which revealed ingestion of tablet paracetamol 500 mg and ondansetron 4 mg for fever and vomiting. 2 days before hospitalization patient had a

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history of eating aloo fries with Tomato sauce. He is habitual to eating this as routinely. Family history was insignificant in view of inherited methhemoglobinemia. On the night of admission patient developed sleepiness, anxiety, restlessness, disorientation which was improved later on the night. On investigation CBC, RFT, LFT, G-6-PD Level and USG Abdomen was done which was within normal limit. Patient was managed with serial ABG monitoring and methylene blue total dose of 350 mg and ascorbic acid 1.5 gm IV 6 hourly. On improvement patient was discharged after 6 days of admission.

CONCLUSION

Methhemoglobinemia is rare. More prevalent causes of methemoglobinemia are drug induced. [5,6] Hereditary causes are rare and include deficiency of methemoglobin reductase or the presence of hemoglobin M. Acquired causes are due to oxidative stress, most commonly from local anesthetic agents, such as benzocaine. [3] In our case it was tomato sauce was used as routine food habit and leading to development of methemoglobinemia. Many studies have shown that nitrate and nitrite compounds can threaten the environment and living health. [6] These compounds may accumulate in plants tissues and are very dangerous substances for human health, leading to health disturbances like methemoglobinemia. The results of mean content of nitrate level is in the following order: ketchup > pizza sauce > tomato paste > tomato fruit > tomato juice is remarkable. [6,7,8] The usual starting dose of methylene blue is 1–2 mg/kg (0.2 mL/kg of a 1% solution) infused intravenously over 3 to 5 minute. The dose may be repeated at 1 mg/kg if methemoglobinemia does not significantly decrease within 30–60 min. Methylene blue should reduce MetHb levels significantly in less than an hour. Ascorbic acid is the treatment of choice when MB is not available and in cases of methemoglobinemia and G6PD deficiency. [1,9] The role of N-acetylcysteine in the treatment of methemoglobinemia is unclear. [3]

Table 1: Levels of MetHb and the Methylene Blue dose administered

Day	Time	Met Hb level	Methylene blue dose
1 st Day	5:40 pm	39.9 %	50 mg
	6:50 pm	14.9 %	
	11:30 pm	21 %	50 mg
	-		
2nd Day	4: 00 am	15 %	
-	7: 46 am	23.4 %	100 mg
	1:00 pm	15 %	
	5: 00 pm	21.5 %	
	7: 00 pm	24.8 %	50 mg
	11: 35 pm	16.4 %	
3rd Day	3: 22 pm	20.5 %	
	8: 00 am	23.8 %	50 mg

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	4: 42 pm	19 %
	10: 16 pm	22.1 %
4th Day	2:00 am	18.3 %
	5:04 pm	20.7 %
	11:58 pm	16.8 %
5th Day	7:53 am	16.1 %
	2:11 am	13.4 %
6th Day	6:29 am	8.9 %

Table 2: Initial ABG on room air – results

Ph-7.3	PCO ₂ -31.1 mmHg	PO ₂ - 72 mmHg	SaO ₂ - 98%	BE _{ecf} 7.3 mmol/L
Na ⁺ -135.5 mmol/L	K ⁺ - 4.69 mmol/L	Lac- 2.71 mmol/L	HCO ₃ -17.9mmol/L	MetHb- 39.9 %

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