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Methemoglobinemia: A Case Report

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Abstract

Methemoglobinemia refers to the oxidation of ferrous iron (Fe++) to ferric iron (Fe+++) within the hemoglobin molecule.[1] This reaction impairs the ability of hemoglobin to transport oxygen and carbon dioxide, leading to tissue hypoxemia and in severe cases, death. Methemoglobinemia most commonly results from exposure to an oxidizing chemical, but may also arise from genetic, dietary, or even idiopathic etiologies.[2,3]

This case report describes the development of acquired methemoglobinemia in a 2 years old boy after accidental ingestion of Blue Detergent Dye , we are reporting this case because of these disparate causes, confusion may arise in the recognition and management of methemoglobinemia. Physicians may be aware of one etiology, such as dietary nitrites in well water, but may be unfamiliar with others, such as the ingestion Blue dye- ultramarine blue, chemical composition sodium aluminosulfosilcate, commonly used as fabric whitener in India.

Key words: Methemoglobinemia; Blue dye;Ultramarine blue; Methylene blue.

Introduction

Methemoglobinemia is a condition in which ferrous iron within hemoglobin is oxidized to ferric iron resulting in impaired oxygen and carbon dioxide transport leading to cyanosis. This ferric state leads to the formation of methemoglobin that causes brownish discoloration of the blood. In healthy normal children, the ferric iron is reduced to ferrous state by the aid of cytochrome b5 oxidase (methemoglobin reductase) along with other systems such as NADH reductase, glutathione reductase and Glucose-6-phosphate dehydrogenase (G-6-PD). Normal concentration of methemoglobin is maintained below 1% in healthy individuals by these enzyme systems.[4]

Methemoglobinemia occurs as an acquired or congenital variant. Acquired methemoglobinemia is more common and occurs from exposure to oxidizing agents such as nitrates, nitrites, aniline dyes and medications such as lidocaine, prilocaine antimalarials. Pyridium etc.[5] Congenital methemoglobinemia is due to either presence of altered hemoglobin (Hemoglobin M) or NADH reductase deficiency. Patients with congenital methemoglobinemia are generally asymptomatic other than cyanosis from birth.

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Acquired methemoglobinemia is usually mild but can be severe depending upon the cause and can present with cyanosis, dyspnea, lethargy-headache, dizziness and deterioration of mental functioning. A history of exposure to a known toxin or drug may be elicited.

Case Report

A 2 years old male child was brought with complaints of vomiting 5-6 episodes/day since 5days, fever since 2 days , bluish discoloration of skin since 2 days, 3 episodes of convulsion since 1 day and altered sensorium since the episodes of convulsion. There was history of ingestion of blue dye 4 days back, the blue dye is commonly used as detergent for washing white clothes in india. There was no history of cyanosis in the past. On general examination cyanosis was present, severe pallor was also present, patient was afebrile, tachycardia, tachypnoea, blood pressure was within percentile for that age and sex, SPO2 was 78% on room air on admission, even after the administration of oxygen there was no significant change in SPO2. On systemic examination lungs were clear , liver was enlarged 3cms below right coastal margin span was 8.5cms,spleen was just palpable, GCS was 11/15, pupils were normal size bilaterally equal reacting to light. Investigation showed, arterial blood- dark brown, Hemoglobin was 4.1g/dl, ABG showed PaO2 of 97 mmhg on room air, oxygen saturation-88%, Methemoglobin level, measured on admission was elevated at 48.5 %, IV methylene blue (1-2 mg/kg) was given and was repeated after 6 hours with the same dose. Methemoglobin level was reduced to 1.25 % after 3 days of treatment.

Discussion

Methemoglobinemia can be easily diagnosed by pulse oximetry. The severity of cyanosis does not correspond to the pulse oximetry reading. The patient may appear extremely cyanotic but have a pulse oximetry reading in the high 80s. The arterial blood sample will show a normal PaO2 though the blood is chocolate brown. This blood when exposed to 100% oxygen continues to remain brown while deoxygenated blood changes to bright red. The diagnosis and severity of methemoglobinemia can be determined by methemoglobin levels. Methemoglobin levels greater than 1% are normal and symptomatic individuals usually have levels greater than 40-50%.[6]
Treatment consists of IV methylene blue (1-2 mg/kg) as a 1% solution over 5 minutes, repeated in our hour if necessary if methemoglobin levels are greater than 30%. Methylene blue is contraindicated in patients with G-6-PD deficiency as it can lead to severe hemolysis. Methylene blue is an oxidant at levels greater than 1 mg/kg and therefore can cause methemoglobinemia at higher doses. Ascorbic acid (200-500 mg) is found useful in congenital methemoglobinemia when methemoglobin level is above 30%. Rarely exchange transfusion may be required. Patients with acquired methemoglobinemia should avoid future exposure to precipitating agent.[7]

Ultramarine is a blue pigment consisting primarily of a zeolite-based mineral containing small amounts of polysulfides. It occurs in nature as a proximate component of lapis lazuli. Ultramarine is the most complex of the mineral pigments, a complex sulfur-containing sodio-silicate ($Na_{8-10}Al_6Si_6O_{24}S_{2-4}$) containing a blue cubic mineral called lazurite (the major component in lapis lazuli).[8] The available information indicates that ultramarine blue is insoluble in water and is not readily absorbed by any route of exposure. The only effects noted in any of the studies were effects consistent to those of other siliceous earth materials. The available toxicity information indicates that ultramarine blue is of low or no toxicological concern. Additionally, given its use as a colorant, the amount of ultramarine blue that would be incorporated into any pesticide product is limited by the need for a certain shade or hue of blue. Therefore, EPA concludes that use of ultramarine blue in pesticide products as a colorant is not likely to pose a dietary risk under reasonably foreseeable circumstances.

There is a reasonable certainty of no harm to the general population, including infants and children, from aggregate exposure to residues of ultramarine blue.[9]

We are reporting this first case of toxicity of ultramarine blue in infants or toddlers, no other similar case reports were found in the medical literature.

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