Case Report

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Severe Methemoglobinemia Secondary to Gun-Powder Sniff in an Infant to Treat Infantile Colic: A Case Report

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Abstract

Methemoglobinemia is an increase in the methemoglobin levels in the blood due to inherited enzyme deficiencies or exposure of oxidizing agent. We report a case of life-threatening methemoglobinemia in an infant secondary to ingestion of Potassium Nitrate in the form of Gun-powder sniff as a remedy to treat infantile colic. The patient was successfully treated with methylene blue. Based on this case, the authors suggest that the use of Gun-powder sniff as a remedy should be avoided in infants, and pediatricians as well as emergency care providers should be aware of suck toxicity and educate parents appropriately.

Keywords: Potassium nitrate; Gun-powder; Saoot; Infant; Methemoglobinemia

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Introduction

Methemoglobinemia can be inherited or acquired. It can cause significant tissue hypoxia, leading to severe and life-threatening clinical features and death. We present a case of severe methemoglobinemia in an infant secondary to ingestion of Potassium nitrate as a remedy for infantile colic.

Case Report

A 4-months old Saudi girl, who was in a good health before, presented to the emergency department with vomiting and shortness of breath. Her acute symptoms had worsened throughout the day. Subsequently she refused feeding and became lethargic. There was no history of aspiration, fever or diarrhoea. She was born at full term without any complications and she was exclusively breastfed. Systematic review was unremarkable apart from infantile colic. No past medical history. She was not on any medication and has no known allergies. She lives with both parents and has two older healthy siblings.

The initial physical examination showed a deeply cyanosed and obtunded infant. Her respiratory rate was 80 breaths/min, heart rate-210 beats/min, BP-55/30 mmHg, rectal temperature was 37.1°C. Her oxygen saturation was 78% on 15 L/min O_2 through a non-rebreather mask. Chest examination revealed good air entry in both lung fields with normal vesicular breathing and no added sounds. Heart examination revealed normal first and second heart sounds without murmurs. Both pupils were mid-size and reactive to light. Other system examination was unremarkable.

Because of the hypotensive shock, immediate fluid resuscitation with normal saline was instigated. Dopamine and epinephrine infusions were titrated at maximum dose until the blood pressure improved. The decision to intubate the patient was made due to her persistent hypoxia and tachypnea and the patient was connected to mechanical ventilation (synchronized intermittent mandatory ventilation mode). Venous blood gas showed pH=6.9, PCO_2=14, HCO_3=2. Complete blood count revealed a WBC of 12,000, Haemoglobin=8.0, Platelets=160,000. Electrolytes panel showed a Na-142, K-4.2, Cl-112 and an anion gap of 25. Liver function test revealed three-fold elevated transaminases. The dark brown blood was sent for methemoglobin, which turned out to be 55%. In addition, her chest X-ray was normal.

It is worth to mention that during endotracheal intubation black silver granules were noticed in the patient nostrils. The parents disclosed that they had been giving the patient gun-powder sniff as a remedy to treat infantile colic. Benzodiazepine was pushed to control a generalized tonic-clonic convulsion she had developed in the ED. Consequently, the patient received 2 doses methylene blue (1 ml/kg each) to reduce her methemoglobin percentage to 15%. Ascorbic acid was not available in the hospital. Blood transfusion (10 ml/kg) was given to treat her anemia and improve tissue oxygenation. The patient improved dramatically after initiation of treatment and her clinical status and biochemical markers recovered. After 2 weeks stay in hospital, including intensive care unit admission; the patient was discharged home with no complications.

Discussion

Haemoglobin molecule consists of four iron atoms that can each bind a molecule of oxygen. Methemoglobin (MetHB) is a modified form of normal haemoglobin that occurs when haemoglobin is oxidized from the ferrous to ferric state, rendering the iron in heme unable to bind to oxygen and resulting in tissue hypoxia [1].

Methemoglobinemia occurs when the levels exceed 2%, and can cause tissue hypoxia and death depending on the concentration and rapidity of diagnosis and management [2]. Normally, red blood cells produce low levels of methemoglobin, as a result of frequently encountered oxidizing chemicals and oxygen free radicals, which the body converts back to haemoglobin through the NAD cytochrome b5 reductase enzyme. The process of conversion back to haemoglobin is much faster in adults than in infants, owing to the fact that the level of enzymes present in infants under the age of 6 months is much lower than in adults, hence resulting in slower rate of conversion among this age group [3].

Methemoglobinemia can be inherited or acquired. The inherited form of methemoglobinemia is characterized by multiple enzymes deficiencies and genetic components. More commonly, the acquired form occurs when a patient is exposed to certain oxidizing agents. These agents include pesticides, fertilizers, nitrates, nitrites, topical anaesthetics (benzocaine, lidocaine, and prilocaine), dapsone, primaquine, and aniline dyes (found in shoe polish, varnish, paint, and ink) [4]. Topical anaesthetics are widely used to facilitate multiple procedures and are implicated in the most serious of toxic methemoglobin cases [5]. Popular forms in which infants are exposed to pharmaceuticals that can induce methemoglobinemia include the use of local anesthesias during procedures or the use of anaesthetic gel to alleviate teething pain. A retrospective review of 138 cases of acquired methemoglobinemia showed that Dapsone accounted for (42%) of all the cases, followed by Benzocaine (4%) and primaquine (4%) [6]. Another single study demonstrated that Benzocaine was responsible for 66% of the topical anaesthesia-related cases of methemoglobinemia among 41 children and 120 adults [7].

The severity of clinical symptoms is directly related to the percentage of methemoglobin in the blood. At levels less than 15%, patients usually are asymptomatic and may have a greyish pigmentation of the skin. When levels exceed 15%, the patient will develop central cyanosis and a brown coloration of the blood. At levels of 20% to 50%, the patient may present with headache, dizziness, tachycardia, nausea, vomiting, syncope, dyspnea, and weakness. Levels at or greater than 50% can result in seizures, coma, and respiratory or cardiovascular failure. Methemoglobin levels greater than 70% are generally fatal [8].

Potassium nitrate (also known as Gunpowder, saltpetre) is an ionic salt of potassium ions and nitrate ions. Potassium nitrate is odourless, water soluble, white to grey crystalline solid. It is used in clear liquid fertilizers, toothpaste component, food preservation, cheese processing, explosives, and most importantly in the production of gunpowder (black powder) [9].

Historically, potassium nitrate was used in asthma and arthritis to relieve respiratory symptoms and pain respectively. To this date, the use of potassium nitrate as a laxative, analgesic and diuretic agent is still practiced by several herbalists and ancient traditional medicine practitioners in different regions worldwide [10].

The first case of methemoglobinemia in a 5-weeks-old infant secondary to *F. asafeotida* ingestion was reported by Kelly et al. [11]. The patient in the mentioned case presented with severe cyanosis and tachypnea a few hours after the presumed ingestion, it was then discovered that *F. asafeotida* exerts its major oxidative effect on the purified fetal haemoglobin HbF.

In Saudi Arabia, it is culturally believed that potassium nitrate (commonly known as Sa'oot) along with other non-medicinal remedies such as Heltit "*Ferula asafoetida*" relieve infantile colic and promote digestion. In Saudi Arabia, a recent case of severe methemoglobinemia has been reported in an infant following the use of *Ferula asafoetida* to treat infantile colic [12]. However, to the best of our knowledge, this is the first reported case of methemoglobinemia secondary to Gunpowder use as a remedy for treating infantile colic.

It has been estimated that the lethal oral dose of potassium nitrate for an adult is be-tween 4 and 30 g (about 40-300 mg NO^{3-}/kg). Adults tolerate large doses of nitrate as sodium and ammonium salt (>100 mg NO^{3-}/kg) with only minor effects in some individuals (light methaemoglobinaemia, diarrhoea and vomiting) [9].

Nitrate itself appears to be relatively non-toxic, but approximately 5% of all ingested nitrates are rapidly reduced to the more toxic component, nitrite, by bacteria in the saliva and the gastrointestinal tract [13]. Nitrite has a higher oxidative potential and therefore a higher ability to cause symptoms, mainly through the formation of met-haemoglobin. Moreover, nitrite is a potent vasodilator and oral ingestion of inorganic nitrate contribute to the development of large quantities of nitric oxide (NO) in the acidic environment of the gastric lumen, which can also induce vasodilation and hypotension [14].

In addition to intravenous fluids and high oxygen treatment, methylene blue, which is a thiazine dye with antiseptic and dose-dependent redox properties, is commonly used as an effective antidote for methemogolbinemia with levels over 20-30%. Alternatively, when methylene blue is unavailable or in cases of G6PD-deficiency, exchange transfusion, hyperbaric

oxygen therapy and high dose vitamin C have been reported to be beneficial in severe cases of methemoglobinemia [15]. More importantly, Prompt recognition and management usually leads to full recovery.

Conclusion

We present this case to emphasize the need to keep a high index of suspicion and the need to control and label potentially harmful material in order to prevent serious outcomes.

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