

## A Successful Case of an Acquired Methemoglobinemia Treated with Methylene Blue

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### ABSTRACT

*Methylene blue has been advocated for multiple indications, but data reporting its use on intoxication is rare. Acquired methemoglobinemia can be caused by a wide variety of chemicals and toxins and on severe cases, with high methemoglobin levels (>20%), methylene blue is the first line treatment. We describe a case report of a 45-year-old man with acute methemoglobinemia due to involuntary exposure to n-propyl nitrate. The patient came into the emergency department conscious, with profound peripheral and central cyanosis and oxygen saturation of 88% on air, with no response to oxygen supplementation. Arterial blood gas showed methemoglobin of 22.9%. Methylene blue was administered intravenously and symptoms improved dramatically, with control arterial blood gas with 2.6% of methemoglobin. Despite the rarity of occurrence of methemoglobinemia in occupational set, and since methylene blue is highly effective, a high degree of suspicious and good training aiming for prompt diagnosis is crucial in treating this emergency situation.*

### Keywords

Emergency medicine, Intoxication, Propyl nitrate.

### Introduction

N-propyl nitrate is a hazardous substance, a colorless liquid with an ether-like odor, used as rocket fuel and sometimes found on cleaning products [1]. It is a highly flammable and reactive chemical that requires local exhaust ventilation and use of personal protective equipment when manipulated. Exposure to this toxic can be made through inhaling and skin or eye contact. Toxic exposure has never been reported, however can be presented as a life-threatening hypoxia. Methylene blue has been advocated for multiple indications, but data reporting its use on intoxication is rare. We reported a 45-year-old man with an acute methemoglobinemia due to n-propyl nitrate intoxication solved with methylene blue.

### Case Report

Caucasian, 44-year-old man, with a history of dizziness, blurred

vision and mild respiratory distress of 2-hour duration, after involuntary exposure to n-propyl nitrate through manipulation of cleaning product without personal protective equipment. Patient was present to emergency department conscious, symptomatic, with peripheral and central cyanosis (mostly lips, ears and hands), respiratory rate of 24 cycles per minute and oxygen saturation of 88% on air, without response to oxygen supplementation with a non-re-breathing face mask. His pulse rate was 80 beats per minute, at sinus rhythm, and blood pressure was 100/60 mmHg. Upper and lower limbs were well perfused, warm, but profoundly cyanotic. Although patient referred dizziness and blurred vision, neurological examination was normal. Arterial blood gas, with a non-re-breathing facemask showed pH 7.55, partial pressure of oxygen 275 mmHg, oxygen saturation 97%, partial pressure of carbon dioxide 26 mmHg, methemoglobin 22.9%, bicarbonate 26.9 meq/L and base excess +8.9. Full blood count showed hemoglobin of 16.1 g/dl, platelet of  $292 \times 10^6/L$ , and white cell count of  $13.56 \times 10^6/L$ . Serum biochemical profile was normal.

Methylene blue (90mg, 1mg/kg) was administered intravenously and symptoms improved dramatically. Control arterial blood gas showed pH 7.41, partial pressure of oxygen 215mmHg, oxygen saturation 98% and methemoglobin 2.6%. Twelve hours later, patient remained asymptomatic, with blood methemoglobin of 1.3% and partial pressure of oxygen on air 93.1mmHg, with later discharged in stable condition.

## Discussion

Methemoglobinemia is a rare cyanosis cause, resulting from oxidation of ferrous iron ( $Fe^{2+}$ ) to ferric iron ( $Fe^{3+}$ ), yielding hemoglobin unavailability for oxygen transport, with potential life-threatening hypoxia [1]. In healthy individuals, normal methemoglobin level ranges from 0.5% to 3.0% of available hemoglobin [2]. Clinically, methemoglobinemia is present in two categories: acquired and hereditary. Congenital methemoglobinemia is due to structural abnormalities in the hemoglobin molecule or due to erythrocyte metabolism defects. Acquired methemoglobinemia can be caused by a wide variety of drugs, chemicals, and toxins [3,4]. Methemoglobinemia symptoms are unclear and nonspecific but are typically related to the degrees of hypoxia and methemoglobin levels, ranging from respiratory distress, dizziness, headache, fatigue, lethargy to seizures, arrhythmias, cardiovascular failure and coma. Methemoglobinemia should be considered in all cyanotic patients with a normal arterial PO<sub>2</sub> and low peripheral oxyhemoglobin saturation refractory to oxygen supply. Definitive diagnosis is based on ABG analysis with measure of methemoglobin levels. Pulse oximetry is inaccurate and unreliable in patients with high methemoglobin fractions [4,5]. Initial treatment of suspected methemoglobinemia is supportive and includes removing inciting agents from clothes, skin and gastrointestinal tracts, as well as administering oxygen. With

severe symptoms or high methemoglobin levels (>20%) methylene blue is the first line treatment [4,5]. It accelerates the enzymatic reduction of methemoglobin by NADPH-methemoglobin reductase. Note that methylene blue is contraindicated in patients with G6PD deficiency. First dose of is 1-2 mg/kg over 5–10 min is highly effective [6]. A second dose can be administered if cyanosis does not disappear or methemoglobin levels remain high within 1h [3,4]. Blood transfusion or exchange transfusion may be helpful in patients who are who are in shock. Hyperbaric oxygen has been used with anecdotal success in severe cases.

Despite the rarity of occurrence of methemoglobinemia in occupational set, and since methylene blue is highly effective, a high degree of suspicious and good training aiming for prompt diagnosis is crucial in treating this emergency situation.

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