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## Life threatening rebound methemoglobinemia following paint ingestion

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## CASE REPORT

## Life threatening rebound methemoglobinemia following paint ingestion

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### Abstract:

A 4-year-girl presented with cyanosis and altered sensorium after ingestion of paint. She was diagnosed to be having methemoglobinemia and treated appropriately with methylene blue. She responded to treatment but again deteriorated the next day for which a repeat dose of methylene blue was needed. One should be aware about rebound methemoglobinemia and the need for further monitoring and treatment.

**Keywords:** Methemoglobin, Paint ingestion, Methylene blue.

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### Introduction

Methemoglobinemia is a condition in which the iron within hemoglobin is oxidized from the ferrous ( $\text{Fe}^{2+}$ ) state to the ferric ( $\text{Fe}^{3+}$ ) state, resulting in the inability to transport oxygen and carbon dioxide. Methemoglobinemia occurs when methemoglobin levels is more than 2%. Presentation can vary from cyanosis to frank seizures, coma and death reflecting the level of methemoglobinemia. The failure of 100% oxygen to correct cyanosis is suggestive of methemoglobinemia.

Acquired methemoglobinemia is much more common than congenital form. Although exposure to drugs is the most common cause of acquired methemoglobinemia but accidental intake of substances containing oxidizing agents, as happened in our case, should not be overlooked. Methylene blue is treatment of

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choice for acquired methemoglobinemia [1]. Rebound methemoglobinemia though rare, but is known to occur and should always be looked after to avoid unexpected fatalities [2].

We present a case of 4-year-old girl who developed cyanosis and altered sensorium after ingestion of unknown and unquantified liquid which was later on suspected to be paint due to its typical odour coming from her clothes and mouth; she was diagnosed as methemoglobinemia and treated appropriately with intravenous methylene blue. The patient's methemoglobin levels decreased, but this was followed by a critical rebound phenomenon on day 2. A repeat dose of methylene blue was given following which she recovered and was discharged on day 6 after remaining asymptomatic for 3 days.

### Case Report

A 4-year-old girl presented to casualty with complaints of vomiting for 3-4 hours followed by altered sensorium and bluish discoloration of body for last 2-3 hours. It was preceded by intake of an unknown and unquantified liquid 4 hours prior to presentation. On examination the child was in altered sensorium with Glasgow Coma Scale of E<sub>3</sub>M<sub>4</sub>V<sub>2</sub>. There was marked central and peripheral cyanosis. Oral cavity examination had a strong odour suggestive of paint. Rest of the systemic examination was normal.

Investigations showed hemoglobin of 11.2 g% with normal white cell and platelet count. Arterial blood gas done while patient was breathing 100% oxygen from an oxygen mask showed a pO<sub>2</sub> of 137 mmHg, pCO<sub>2</sub> 34 mmHg and pH of 7.38 mmHg and an oxygen saturation of 85% with pulse oximetry. There was no improvement in cyanosis despite giving high flow oxygen. Filter paper test for methemoglobinemia was done and found to be positive.

On basis of history suggestive of paint ingestion, child having cyanosis and positive filter paper test, the diagnosis of methemoglobinemia was kept and a blood sample for estimation of methemoglobin levels was sent. Methylene blue at the dose of 1 mg/kg was given intravenously

following which patient showed marked improvement. Glasgow Coma Scale reverted to E<sub>4</sub>M<sub>5</sub>V<sub>6</sub>, colour improved and she became more responsive. Repeat arterial blood gas analysis showed pO<sub>2</sub> of 138 mm Hg, pCO<sub>2</sub> 33 mm Hg, pH 7.39 and oxygen saturation of 94%. Methemoglobin levels came out to be 54% making it evident that we were dealing with a case of acquired methemoglobinemia. On day 2 of admission patient again developed cyanosis which was soon followed by an episode of seizure. Glasgow Coma Scale dipped to E<sub>2</sub>M<sub>4</sub>V<sub>2</sub>. A repeat dose of methylene blue was administered; dramatic improvement was found. Patient regained consciousness and soon became oriented in time, place and person. She remained asymptomatic for next 3 days and was discharged on day 6.

### Discussion

Methemoglobin is a non-oxygen binding form of hemoglobin where iron is in the ferric form. Normally methemoglobin forms less than 1% of the total hemoglobin. The physiologic reduction of MethHb Fe<sup>3+</sup> to Hb Fe<sup>2+</sup> is mainly accomplished by red cell NADH-cytochrome b5 reductase [3]. When methemoglobin levels are greater than 15%, cyanosis is visible [4]. Acquired methemoglobinemia is more common than congenital form and occurs from exposure to oxidizing agents such as nitrates, nitrites, aniline dyes and medications such as lidocaine, prilocaine, antimalarials, pyridium etc. Paint contains aniline dyes which acts as an oxidizing agent. When the oxidizing capacity of dyes exceeds the reducing capacity of body, methemoglobin level rises. In many cases, there are no clinical features other than cyanosis, but when the concentration of methemoglobin reaches 30-45%, anoxic symptoms commonly develop. These include headache, dizziness, tachycardia, dyspnea on exertion, muscle cramps and weakness. In cases of acute poisoning, the concentration may exceed 60-70%, when vomiting, lethargy, loss of consciousness, circulatory failure and death may occur [1].

Diagnosis is mainly based on the clinical picture, filter paper test and methemoglobin levels. ABG and pulse oximetry are unreliable and only helpful to rule out other differentials. Oxygen saturation is generally maintained around 85%. Co-oximetry, if available, can help to reach the diagnosis [5].

In addition to the supportive management, methylene blue is the drug of choice for the specific management. Methylene blue is an oxidizing agent. Methylene blue is converted to leucomethylene blue which is the reducing agent. Leucomethylene blue increases the action of diaphorase-2 by more than five times though normally it has a very low activity. The drug is available as an ampoule of 1% solution containing 10 mg/ml. It is recommended at a dose of 1-2 mg/kg followed by bolus of 25-30 ml of normal saline. The dose can be repeated after an interval of an hour till a maximum dose of 7 mg/kg over 24 hours [3].

Second dose may be given if cyanosis has not cleared within 1 hour [1]. Any dose beyond 7 mg/kg/day will be harmful as the oxidizing action of methylene blue will become more than the reducing action of leucomethylene blue. Even after resolution of symptoms one should be cautious to look for recurrences which though rare but have been reported [2], as happened in our case.

Methylene blue belongs to a group of drugs considered to potentially cause hemolysis when given to persons with G6PD deficiency [6]. G6PD is required for production of NADPH which is utilized by NADPH MetHb reductase to convert methylene blue to leucomethylene blue, the main reducing agent. Hence in absence of G6PD methylene blue becomes ineffective. Hyperbaric oxygen is another modality recommended for the treatment of acquired methemoglobinemia [7].

Ascorbic acid has been recommended in chronic hereditary methemoglobinemia but its use in acquired methemoglobinemia is controversial [8].

### Conclusion

We concluded that it is important to diagnose methemoglobinemia at an early stage as it is potentially treatable, if delayed can be life threatening. One should be cautious in administering methylene blue in right doses, recognizing rebound phenomenon and avoiding it in G6PD deficiency cases.

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