

## Case Report

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# Indoxacarb poisoning induced methemoglobinemia-a rare presentation in paediatric age group

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## ABSTRACT

Indoxacarb, an oxadiazine insecticide, has high insecticidal and low mammalian toxicity. The insecticidal activity of indoxacarb is attributed to its ability to block sodium channels in the insect nervous system. Several cases of indoxacarb-induced methemoglobinemia have been reported in adults, but presentation in paediatric age group is rare. Methaemoglobinemia occurs after indoxacarb ingestion because its aromatic metabolites are biotransformed to active intermediates that produce methaemoglobin. The case involved is an adolescent girl who presented to the PICU 3 hours after suicidal ingestion of 10-15 ml of indoxacarb. After 15 min of consumption of poison she developed pain abdomen and vomiting. At PICU, she complained of nausea, vomiting and dizziness. Her vital signs were heart rate 96/min, BP 110/80 mmHg, oxygen saturation 78% on room air and GCS 15/15. Despite receiving 10L/ min of oxygen via NRB mask. Her ECG showed normal sinus rhythm without ischaemic change, and CXR showed no specific abnormality. Initial arterial blood gas analysis revealed pH-7.413,  $\text{pO}_2$ -123.6 mmHg,  $\text{pCO}_2$ -37.7 mmHg,  $\text{HCO}_3$  23.5 mmol/L. Other laboratory results were normal. Methaemoglobinemia is a rare clinical presentation of indoxacarb poisoning. The treatment plan includes timely administration of injection methylene blue.

**Keywords:** Indoxacarb poisoning, Methaemoglobinemia, Methylene blue

## INTRODUCTION

Indoxacarb is an oxadiazine insecticide that was designed to kill insects resistant to organophosphate, carbamate and pyrethroid insecticides.<sup>1</sup> Its activity is attributed to its ability to block sodium channels in the insect nervous system. Although it is known that indoxacarb has low mammalian toxicity, limited data are available on human poisoning. Although there are many causes of methaemoglobinemia, recently several authors have suggested that methaemoglobinemia occurs after indoxacarb ingestion because its aromatic metabolites are biotransformed to active intermediates that produce methaemoglobin. Methaemoglobin is an altered state of haemoglobin in which the ferrous ( $\text{Fe}^{2+}$ ) ions of haem

are oxidised to the ferric ( $\text{Fe}^{3+}$ ) state.<sup>6</sup> Moderate to severe symptoms of methaemoglobinemia might be treated with methylene blue. The most widely accepted treatment is administration of 1 or 2 mg/kg bodyweight of methylene blue infused i.v. over 5 min, followed by IV flush with normal saline.<sup>5</sup> Methylene blue is generally indicated for symptomatic patients (signs of hypoxaemia) and considered in asymptomatic patients with methaemoglobin level >20%. In some cases, repeat methylene blue doses might be necessary. In severe life threatening cases and/or when treatment with methylene blue is ineffective or contraindicated, exchange transfusion should be considered. If this is not available consider immediate administration of O negative red cells to increase the oxygen carrying capacity of blood.

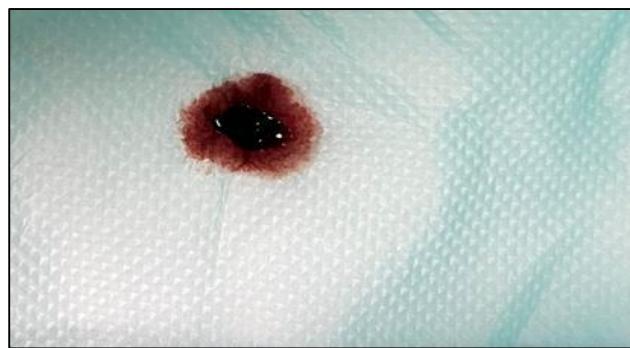
## CASE REPORT

The case involved 16 year old adolescent girl who was presented to the PICU 3 hours after suicidal ingestion of 10-15ml of indoxacarb. The patient had no previous medical history, with previous attempts of suicide in the last 1 day. After 15 minutes of consumption of the poison she developed pain abdomen and vomiting. At PICU, she complained of nausea, vomiting and dizziness. Her vital signs were heart rate 96/min, BP 110/80 mmHg, oxygen saturation 78% on room air and GCS 15/15. Despite receiving 10L/ min of oxygen via NRBM mask (Figure 1). Her ECG showed normal sinus rhythm without ischaemic change, and CXR showed no specific abnormality. Initial arterial blood gas analysis revealed pH-7.413, pO<sub>2</sub>-123.6 mmHg, pCO<sub>2</sub>-37.7 mmHg, HCO<sub>3</sub>-23.5 mmol/L. Other laboratory results were normal.



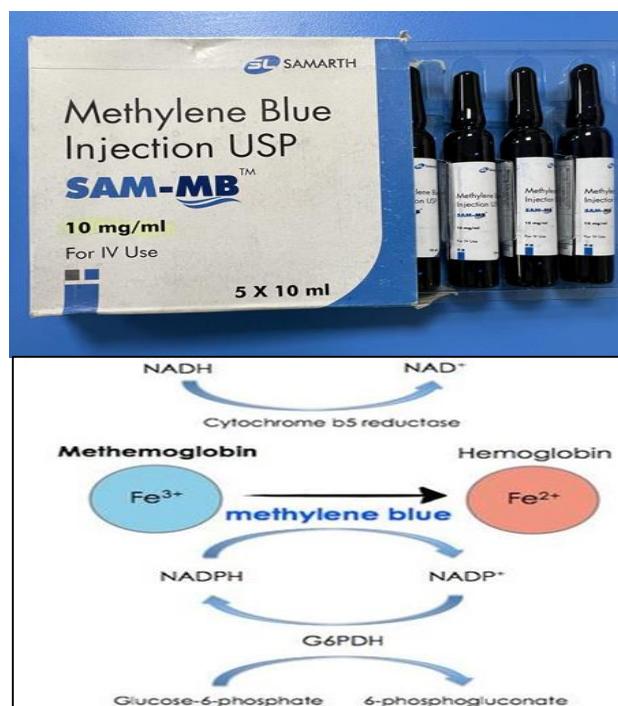
**Figure 1: Saturation at the time of admission.**

In our case, the patient reported nausea, vomiting and dizziness, and was noted to have hypoxia inspite of administering 100% oxygen.



**Figure 2: Filter paper test, blood remains dark on exposure to air.**

Filter paper test (Figure 2) was positive for methaemoglobinemia IV methylene blue (2 mg/kg) (10 mg/ml) was administered with 100 ml 5% dextrose. She improved immediately on administering methylene blue (Figure 5).

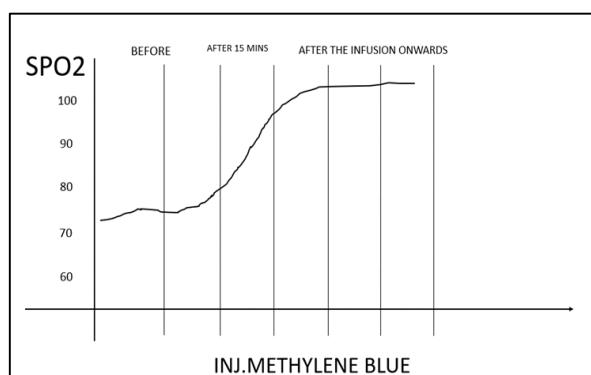


**Figure 3: Mechanism of action of inj. methylene blue.**

Her saturation improved to 100% with first dose of methylene blue (Figure 6), followed by 1 mg/kg BD maintainence dose was given for the next 24 hours.



**Figure 4: Urine colour change following methylene blue administration.**



**Figure 5: Effect of inj. methylene blue on saturation.**



**Figure 6: Improvement in saturation after administration of inj. methylene blue.**

## DISCUSSION

A case report by Wu et al of a patient who ingested indoxacarb only required a single dose of methylene blue and the patient recovered without sequelae.<sup>4</sup> This is one of the few case reports of indoxacarb poisoning that reports the duration of methemoglobinemia and administration of multiple doses of methylene blue. Another case report by Prasanna et al shows that indoxacarb poisoning can result in the treatment of methemoglobinemia that may require multiple doses of methylene blue, which can be life saving.<sup>3</sup> In patients with G6PD deficiency or at higher doses, methylene blue can paradoxically induce methemoglobinemia. Other minor side effects like emesis, retrosternal chest pain, tachycardia, hypertension, anxiety, green-blue urine (Figure 4), oxidative haemolysis, headache, dizziness, factitious cyanosis have been reported.<sup>5</sup> Patients not responsive to methylene blue, G6PD deficiency, severe symptoms will require exchange transfusions or hyperbaric oxygenation. Ascorbic acid is useful for congenital methemoglobinemia, its use is limited in cases of acquired methemoglobinemia.

## CONCLUSION

Methaemoglobinemia is a rare clinical presentation of Indoxacarb poisoning. MetHB concentration was not available in our setting. Clinically the child was symptomatic and the treatment plan was timely administration of injection methylene blue. Supplementation with ascorbic acid was given. We suggest that careful initial observation and management are needed in cases of indoxacarb poisoning because multiple doses of methylene blue maybe required to treat the rare presentation of methemoglobinemia.

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