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CASE REPORT

# A Rare Case of Methaemoglobinaemia Secondary to Indoxacarb Poisoning

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## Abstract

***Introduction:*** Indoxacarb containing insecticides are commonly used for pest control in the agricultural industry in Sri Lanka and deliberate self-poisoning with insecticides is not uncommon in the farming communities in Sri Lanka. Methaemoglobinaemia is a rare but fatal condition known to occur as a consequence of indoxacarb toxicity in humans.
***Case report:*** We report a case of a 35-year-old Sri Lankan male who presented with severe methaemoglobinaemia following ingestion of an indoxacarb containing insecticide to Weera Denzil Kobbekaduwa Base Hospital, Dambulla, Sri Lanka, in January 2024. Methaemoglobinaemia was suspected due to his presentation with central cyanosis with low oxygen saturation which did not improve with high flow oxygen therapy. Estimation and monitoring of blood methaemoglobin level was carried out using a bed-side qualitative assessment with colour charts. The patient required administration of repeated doses of methylene blue for complete recovery from methaemoglobinaemia.
***Discussion:*** The management of methaemoglobinaemia can be challenging in a resource poor setting due to unavailability of standard diagnostic facilities. Early identification and prompt treatment of methaemoglobinaemia are crucial in the management of indoxacarb poisoning to prevent fatal outcomes.
***Conclusion:*** This case highlights the importance of being aware of the fatal complication of methaemoglobinaemia with certain insecticide poisonings, clues to identify methaemoglobinaemia early in a resource poor setting and the treatment strategies of methaemoglobinaemia.

**Keywords:** Indoxacarb, Methaemoglobinaemia, Insecticide, Methylene blue

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## INTRODUCTION

Indoxacarb is an oxadiazine-class insecticide commonly used for pest control in the agricultural sector in Sri Lanka. Human exposure to indoxacarb is rare but can result in severe toxicity, particularly after deliberate ingestion. The most significant toxicological effect of indoxacarb poisoning is methaemoglobinaemia which is fatal if left untreated. In methaemoglobinaemia, haemoglobin is oxidized to methemoglobin, reducing its ability to transport oxygen. Cases of methaemoglobinaemia occurring following indoxacarb ingestion are particularly uncommon [1]. Prompt diagnosis and rapid initiation of treatment, particularly with intravenous methylene blue, are critical in managing this condition [2].

## CASE PRESENTATION

A 33-year-old Sri Lankan male, a farmer, presented to the emergency department two hours after deliberate ingestion of approximately 50 ml of Avaunt, an indoxacarb-containing insecticide. His main complaints were shortness of breath, abdominal pain, nausea, and headache. He did not give any prior history of psychiatric illness or substance abuse. On initial examination, he was conscious with a Glasgow Coma Scale score of 15/15. Notably, he exhibited both central and peripheral cyanosis, though his peripheries were warm. He was dyspnoeic, with a respiratory rate of 24 breaths per minute and on pulse oximetry the oxygen saturation was 86% on room air. Auscultation of the lungs was unremarkable with clear lung fields. His pulse rate and blood pressure were 96 beats per minute and 130/80 mmHg, respectively, and no cardiac murmurs were detected. The abdomen was soft and non-tender on palpation. Pupillary examination revealed equal, round, and reactive pupils, and no focal neurological deficits were identified.

Despite receiving 15 litres of oxygen per minute via a non-rebreather mask, his oxygen saturation remained low at 88-90%, which raised the suspicion of methaemoglobinaemia [3]. The patient's venous blood was noted to have a muddy-brown appearance, a classic visual sign of methaemoglobinaemia [4]. Capillary blood glucose was 124 mg/dL, and the electrocardiogram showed normal sinus rhythm without ischaemic changes. Based on clinical presentation, a diagnosis of indoxacarb-induced

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methaemoglobinaemia was suspected. Arterial blood gas (ABG) testing revealed the following results: PH: 7.39, PaO2: 220mmHg, PaCO2: 25mmHg, Bicarbonate: 20 mmol/L and oxygen saturation: 98%. In the absence of facilities to detect and quantify the methaemoglobin level in the blood, bedside methaemoglobin assessment was carried out using an absorbent paper stained with patient's blood and comparing it with a standard colour chart. This method has been validated to be used as a reliable tool to estimate the percentage of methaemoglobin in the resource limited setting [5]. Estimated methaemoglobin level was in the range of 50-60%.

The patient was treated with gastric lavage followed by activated charcoal to reduce the systemic absorption of Indoxacarb. As the methaemoglobin level was greater than the treatment threshold of 20%, intravenous methylene blue (MB) at 1 mg/kg (60mg) was administered over five minutes, after excluding contraindications [6]. The patient's vital parameters were closely monitored, and supplemental oxygen therapy was continued. One hour after the first dose of methylene blue, the patient's symptoms and oxygen saturation improved to 92-94%, although he remained visibly cyanotic. A repeat methaemoglobin assessment showed levels had decreased to 30-40%, and a second dose of intravenous methylene blue (60 mg) was administered. One hour after administration of the second dose the patient's oxygen saturation improved to 98%, and he experienced relief from dyspnoea. The methaemoglobin level was noted to be reduced to 10% at this point on reassessment. The patient was gradually weaned off supplemental oxygen over the next six hours as his clinical status improved.

Chest X-ray showed clear lung fields and no evidence of aspiration. Basic blood test results, including full blood count, renal function tests, serum electrolytes and the liver enzymes were within normal limits. Values of the blood and serum investigations are summarized in table 1. The patient remained haemodynamically stable throughout hospitalisation, with no alterations in sensorium, seizures or other signs of organ dysfunction. Two days later he was discharged following a mental health evaluation, during which he was diagnosed with moderate depression. Upon discharge, mental health follow-up was arranged to address the underlying psychiatric issues contributing to the deliberate self-poisoning.

**Table 1. Summary of blood investigations**

| Investigation | Value | Normal range |
|---|---|---|
| White cell count | 4.6 x $10^9$/L | 4 -11 × $10^9$/L |
| Haemoglobin | 14 g/dL | 13.5 – 17.5 g/dL |
| Platelet count | 304 x $10^9$/L | 150 - 450 × $10^9$/L |
| Serum creatinine | 0.84 mg/dL | 0.74 - 1.35 mg/dL |
| Serum sodium | 140 mmol/L | 135 - 145 mmol/L |
| Serum potassium | 4.2 mmol/L | 3.5 - 5.0 mmol/L |
| Serum calcium (ionized) | 1.18 mmol/L | 1.12 - 1.30 mmol/L |
| Serum magnesium | 0.8 mmol/L | 0.75 - 1.05 mmol/L |
| ALT | 24 U/L | 10 - 45 U/L |
| AST | 28 U/L | 10 - 40 U/L |
| Serum Bilirubin (Total) | 0.7 mg/dL | 0.3 - 1.2 mg/dL |

## DISCUSSION

Indoxacarb is an insecticide that blocks voltage-gated sodium channels in insects, causing neuronal dysfunction and death. In humans, its mechanism of toxicity involves the induction of oxidative stress, which leads to the formation of methaemoglobin [1]. This case highlights the serious complication of methaemoglobinaemia following Indoxacarb ingestion and the critical importance of timely intervention. Indoxacarb poisoning has also reported to cause acute kidney injury, although it was not seen in our patient.

Clues for the diagnosis of methaemoglobinaemia are refractory cyanosis and low oxygen saturation despite high flows of supplemental oxygen, muddy-brown colour of blood and saturation gap between the oxygen saturation by pulse oximetry and the saturation in arterial blood gas. Saturation gap is not diagnostic of methaemoglobinaemia as a saturation gap of greater than 5% can also be seen in carboxyhaemoglobinaemia and sulphaemoglobinaemia as well [7].

Methaemoglobinaemia takes place when the iron within haemoglobin is oxidized from the ferrous ($Fe^{2+}$) to the ferric ($Fe^{3+}$) state, impairing its ability to bind and transport oxygen. Clinical manifestations of methaemoglobinaemia vary depending on the severity of methaemoglobinaemia and the spectrum of clinical features includes cyanosis, fatigue, weakness, headache, central nervous system depression, metabolic acidosis, seizures, arrhythmias, coma, and death [8]. Normal blood methaemoglobin level is less than 1%, and levels above 20% cause significant cyanosis and hypoxia [9]. Levels above 30% can cause significant hypoxia and carry a high risk of mortality. Left untreated, severe methaemoglobinaemia can result in organ failure and death [4].

The first-line treatment for symptomatic methaemoglobinaemia is intravenous methylene blue, which reduces methaemoglobin back to haemoglobin through the NADPH-methaemoglobin reductase system. Methylene blue acts by reducing methaemoglobin back to haemoglobin via the NADPH-dependent methaemoglobin reductase pathway, thereby restoring the oxygen-carrying capacity of haemoglobin [10]. It should be administered as a bolus of 1-2mg/kg. Whilst most patients respond well to a single dose, repeat doses may be necessary in cases of persistent symptoms or having methaemoglobin levels above the treatment threshold of 20%, as was seen in this case [10].

It is important to note that methylene blue has contraindications, particularly in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency, where it can cause severe haemolysis. In such cases, or when treatment with methylene blue is not successful, high dose intravenous ascorbic acid (10 grams per dose) can be used as an alternative treatment, although the response to ascorbic acid may not be as effective and rapid as with treatment with methylene blue [6]. Exchange transfusion or hyperbaric

oxygen therapy may be required in severe cases [11]. In our patient, there was no history of G6PD deficiency, and methylene blue was administered without complications.

## CONCLUSION

Indoxacarb-induced methaemoglobinaemia is a rare but life-threatening condition that requires prompt recognition and treatment. This case underscores the importance of early administration of intravenous methylene blue, which leads to rapid improvement in oxygenation and resolution of symptoms. Healthcare providers should be aware of this fatal complication of indoxacarb ingestion and competent in promptly recognizing and estimating methaemoglobinaemia in relevant clinical contexts, especially where facilities for biochemical assessment of methaemoglobin levels are not available.

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