Isolataed Severe Methaemoglobinaemia: A Rare Presentation Of Indoxacarb Poisoning

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ABSTRACT Indoxacarb pesticide ingestion in humans is associated with multiple complications such as methaemoglobinaemia, acute kidney injury, rhabdomyolysis, acute respiratory distress syndrome and seizures. Among those, methaemoglobinaemia is common. Here we present a case of deliberate ingestion of indoxacarb pesticide causing methaemoglobinaemia. Early recognition and prompt treatment with methylene blue have made a complete recovery without any significant morbidity.

KEYWORDS Methaemoglobinaemia, indoxacarb, methylene blue

Introduction

Indoxacarb is an oxadiazine insecticide. It is initially developed for the killing of insects' strains that are resistance to organophosphates, carbamates, and pyrethroids. The binding of active metabolite will blocks the voltage-dependent sodium channels within the insect nervous system and leads to paralysis and death of the insect. The information regarding the toxic effect of indoxacarb in humans is scarce. Here, we present a case of methaemoglobinaemia following deliberate ingestion of indoxacarb. Early recognition and prompt administration with methylene blue have led to complete clinical recovery.

Case report

A 77-year-old previously healthy farmer, presented to the emergency department(ED) 4 hours after deliberate ingestion of 50 ml of a pesticide containing indoxacarb in an attempt to commit suicide. He had vomited 2-3 times while at home and on arrival to the emergency department he was conscious with a GCS 15/15. He was mildly dyspnoic with oxygen saturation of 85% on ambient air. He was not cyanosed; pulse rate was 88 bpm, blood pressure was 140/80 mmHg and no lung crepitations suggestive of aspiration of gastric contents. In the ED

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patient was treated with high flow oxygen via mask, gastric lavage performed and activated charcoal given.

A few hours later patient becomes deeply cyanosed centrally, mildly tired and confused and pulse oximetry reading dropped to 80% even with high flow oxygen indicating severe hypoxia. Arterial blood gas revealed a PH of 7.37, pO2 187 mmHg, pCO2 39 mmHg, HCO3 - 22.5 mmol/L. Chest radiography does not reveal any abnormalities. A muddy brown colour blood sample was noted on blood for investigations. A blood sample was sent for analysis to determine the methaemoglobin level, confirmed a level of 48%. Methylene blue was administered at a dose of 1 mg/Kg intravenously. Due to the unavailability of intravenous ascorbic acid, an enteral preparation was given.

Within an hour patient showed signs of dramatic clinical recovery. The cyanosis has disappeared, and the pulse oximetry reading was 90%-92%. Repeat arterial blood gas showed a PH of 7.41, pO2 277 mmHg, pCO2 36 mmHg, HCO - 22.8. Other necessary investigations such as full blood count, renal and liver profile were within normal range. ECG was unremarkable. Methaemoglobin level was repeated after 12 hours of administration of methylene blue showed a marked reduction to 8.5%. The patient was managed in ED for two days and showed complete recovery, and oxygen saturation improved to 96%. On day 5, the patient was discharged after counselling with follow up plans.

Discussion

Indoxacarb is a pyrazoline-type insecticide. It was discovered in 1991 and registered in 2001. It was developed for use against insect strains that are resistance to organophosphates, carbamates, and pyrethroids. The active metabolite will bind and

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block the voltage-gated sodium channel within the insect's nervous system and leads to paralysis and death. There are few reports recorded in the medical literature to date regarding indoxacarb poisoning in humans causing complications such as methaemoglobinaemia, acute kidney injury, rhabdomyolysis and ARDS around the globe and from Sri Lanka as well [1,2,3,4]. But surprisingly our patient did not develop any complications other than methaemoglobinaemia.

Methaemoglobinaemia is a commonly observed complication of indoxacarb poisoning. Methaemoglobin is formed by the oxidation of heme iron moieties from ferrous to ferric state, causing a characteristic bluish-brown muddy colour resembling cyanosis as observed in this patient. Methaemoglobin has a high affinity for oxygen, and it shifts the oxygen dissociation curve to the left causing impairment in oxygen delivery to tissues. The presence of methaemoglobin can be suspected when the oxygen saturation as measured by pulse oximetry is significantly different from the oxygen saturation calculated from arterial blood gas analysis ('saturation gap') [5, 6], as demonstrated in our patient with an SPO2 of 80%, however, the pO2 in ABG was 187 mmHg. In humans, normally the methaemoglobin levels are less than 1% and depending on the methaemoglobin levels the symptoms vary. Levels unto 15% cause colour changes in skin and blood. As levels rise above 15%, neurologic and cardiac symptoms arise as a consequence of hypoxia. Levels higher than 70% are usually fatal. In our patient methaemoglobin level was 48%, and he had muddy-brown discolouration of blood, mild drowsiness and confusion without any cardiac complications.

Methylene blue is the first line antidote for methaemoglobinaemia, and it acts as a cofactor in the NADPH-dependent methaemoglobin reductase system. It is available as a 1% solution (10mg/ml). Methylene blue is given in a dose of 1-2 mg/kg (up to a total of 50 mg in adults, adolescents, and older children) in IV saline over 3-5 minutes. Administration may be repeated at 1 mg/kg every 30 minutes as necessary to control symptoms, but most patients require only one dose, as our patient also improved with one dose.

Conclusion

Methaemoglobinaemia is commonly observed in indoxacarb poisoning and could be the sole manifestation. Early recognition and prompt treatment with methylene blue will prevent significant morbidity and mortality.

1. Abbreviations*

GCS – Glasgow Coma Scale. ECG – Electrocardiogram. ABG – Arterial Blood Gas. ARDS – Acute Respiratory Distress Syndrome. NADPH – Nicotinamide Adenine Dinucleotide Phosphate.

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