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Indoxacarb Poisoning - Induced Methemoglobinemia Leading To Cardiac Arrest: A Case Report

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ABSTRACT:
Indoxacarb is an insecticide used to control pests in agriculture and residential settings. It belongs to a class of insecticides known as sodium channel blockers, which disrupt the normal functioning of sodium channels in insects, leading to paralysis and death. Indoxacarb is generally considered to have low toxicity to humans and animals when used according to label instructions. However, like many chemicals, exposure to indoxacarb can have adverse effects, including the potential to induce methemoglobinemia. Methemoglobinemia may occur following the ingestion of indoxacarb, as its aromatic metabolites are transformed into active intermediates that generate methemoglobin. We report a case involving a 20 year old male with Indoxacarb poisoning presented with cyanosis due to methemoglobinemia, which was improved after giving methylene blue and supportive management.

Keywords: Indoxacarb, Methemoglobinemia, Methylene blue

I. INTRODUCTION:

Indoxacarb is a versatile oxadiazine insecticide used in both industrial and agricultural settings to control lepidopteran pests. It is effective against insects such as the beet armyworm, cotton bollworm, and native budworm, particularly in crops like cotton and soybeans.

Indoxacarb inhibits the movement of insect sodium channels in certain nerve cells, resulting in tremors, feeding cessation, and death within a few hours. In trials including dogs, rats, and mice, neurotoxicity and hemopoietic effects were observed. Intentional intake of indoxacarb suggests that this chemical causes methemoglobinemia in humans.

Despite the 'carb' ending, this is not a carbamate pesticide. It is a low-risk insecticide and an organophosphate substitute. Overdoes are quite rare. Inadvertent exposures are rarely toxic; nevertheless, massive or deliberate ingestions have resulted in considerable morbidity. Abrupt indoxacarb intake has resulted in severe methemoglobinemia, hypotension, mental status depression, abrupt renal failure, rhabdomyolysis, and metabolic acidosis.

II. CASE REPORT:

A 20 year old male arrived atthe emergency room early in the morning in a semi-conscious state presented with symptoms of headache, blurring of vision, giddiness followed by sudden onset of Shortness of breath and suddenly patient went into cardiac arrest. On examination, his saturation levels were very low i.e. SpO2 - 40% and the Arterial Blood Gas analysis indicated severe Respiratory acidosis. Immediately, CPR was done according to the ACLS protocol; the he was intubated and supported to a Mechanical ventilator. Despite being revived, the patient’s oxygen saturation remained at 70% even with 100% FiO2.After intubation, the patient's condition was still not improving and the patient's Chest X-ray revealed no Pulmonary edema, no pleural effusion, normal air bronchogram pattern with normal costo-phrenic and costo-cardiac angle; The ECG displayed a normal sinus rhythm without any ST-T

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changes and 2D ECHO showed no Left Ventricle Regional Wall Motion Abnormality, Good LV Function, No Atrial Regurgitation, No Aortic valve Stenosis, IVC – Normal >50% collapsing.

His Arterial Blood Gas analysis had shown the levels of pH 7.120, pCO2 64.61 mmHg, pO2 65.08 mmHg and HCO3- 24.43 mmol/L. After investigating the patient's attenders, they said that he consumed some unknown poison and they had searched for the poison container at their home. They had found the poison bottle and it was confirmed that he had consumed Indoxacarb poison (KENTO) which is a non-organophosphorus oxadiazine insecticide and has no antidote. Consumption of this insecticide will lead to Methemoglobinemia.

His blood sample was drawn and was dark muddy brown in colour. The patient's blood sample was sent to check the methemoglobin levels in the blood.

After the confirmation of methemoglobin levels in blood which is 80.06%, the patient was administered methylene blue injection at a dose of 1 mg/kg intravenously as an initial dose and later he was put on a maintenance dose of 1mg/kg, simultaneously the patient was also administered with Vitamin-C injection of 1gm dose. As the patient was in hypotension, he was started with inotropes. After the administration of methylene blue injection, the saturation was improved i.e., SpO2 from 40% to 85%.

Later, we observed that the patient's condition was improving and becoming hemodynamically stable and inotropes were stopped. On the second day of administration of the methylene blue injection, gradually the patient's saturation levels were improved to 93%. On Day -3 the patient's saturation levels were improved to 95% and then the patient was kept on the T-piece trail. His Arterial Blood Gas Analysis results were also improved as shown in the table given below. On Day-4, the patient was extubated as his saturation was maintained up to 95% and the patient's methemoglobin levels were found to be <5%. On Day-5, the patient was stable and the patient was discharged the next day.

Day	pH	pCO_2 (mmHg)	pO_2 (mmHg)	HCO_3^- (mmol/L)
Day 1	7.120	64.61	65.08	24.48
Day 2	7.179	48.24	82.19	15.05
Day 3	7.292	44.69	89.79	15.90
Day 4	7.354	39.09	92.58	22.36
Day 5	7.421	37.28	93.41	26.28

III. DISCUSSION:

Indoxacarb is a broad-spectrum oxadiazine pesticide that targets Lepidoptera such as beet armyworms, fire ants, and cockroaches. It is a "low-risk" insecticide which replaces organophosphate pesticides.

Methemoglobinemia is a potentially fatal medical condition where the oxygen-carrying ability of hemoglobin in the blood is reduced because iron in the hemoglobin is oxidized from its ferrous (Fe2+) state to the ferric (Fe3+) state. When iron is in the ferric state, it cannot effectively bind to or transport oxygen. This leads to a functional anemia due to elevated levels of methemoglobin.

Methemoglobinemia can present with varying levels of severity, ranging from mild to severe. The clinical signs of this condition can include cyanosis (a bluish tint to the skin), pallor, fatigue, weakness, headaches, CNS depression, metabolic acidosis, seizures, arrhythmias, coma, and, in extreme cases, death. The severity of symptoms is influenced by several factors, such as the amount of methemoglobin present, the speed at which it accumulates, the individual's ability to naturally reduce methemoglobin, and their overall health. Additionally, the length of exposure to oxidizing agents and the intensity of that exposure can also affect the severity of the symptoms.

Cyanosis may be visible with methemoglobin levels as low as 10%. The characteristic "chocolate brown blood" appearance can be seen in about 15% of cases.As methemoglobin levels rise to around 20%, patients might experience shortness of breath, fatigue, weakness, anxiety, dizziness, and headaches. When levels reach 30% to 50%, symptoms can include shortness of breath, confusion, and loss of consciousness. At levels of 50% and higher, there is a risk of rapid breathing, seizures, irregular heart rhythms, metabolic acidosis, central nervous system depression, and coma. Methemoglobin levels above 70% are often life-threatening.

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Methemoglobinemia treatment involves removing the causative agent and potentially using methylene blue (tetramethylthionine chloride) as an antidote. Administering high-flow oxygen through a non-rebreather mask helps enhance oxygen delivery to tissues and assists in the natural reduction of methemoglobin levels. Methylene blue is typically given at a dose of 1-2 mg/kg (0.1-0.2 mL/kg of a 1% solution) intravenously over 5 to 30 minutes. If methemoglobin levels remain above 30% or if symptoms are significant, an additional dose of up to 1 mg/kg can be administered one hour after the initial dose.Additional doses might be needed occasionally. Methylene blue contraindications include known hypersensitivity to methylene blue and/or methemoglobin reductase insufficiency. Methemoglobinemia can be reversed with methylene blue if the pentose phosphate pathway (PPP) and glucose-6-phosphate dehydrogenase (G6PD) are functional. G6PD can be detected via enzymatic activity or genetic testing (to detect known gene mutations). Patients with G6PD deficiency who are given methylene blue may experience hemolysis. Although G6PD enzyme deficiency may not be complete, most G6PD deficiency tests do not provide genetic information. Methylene blue is hence contraindicated in G6PD patients. These patients should be managed with ascorbic acid (Vitamin C), blood transfusions, and hyperbaric oxygen.

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