



CASE OF NEW COMPOUND CONSUMPTION: INDOXACARB

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ABSTRACT

We present a case of acute indoxacarb poisoning in a 40-year-old male. Indoxacarb, a widely used insecticide, has rarely been reported in human toxicity cases. This report highlights the clinical presentation, management, and outcome of indoxacarb poisoning to enhance awareness and guide treatment.

KEYWORDS : Indoxacarb, Insecticide Poisoning, Acute Toxicity, Supportive Care, Case Report.

INTRODUCTION

Indoxacarb is an oxadiazine pesticide extensively used in agriculture and pest control for both exterior and interior applications. It effectively controls lepidopteran pests, including cotton bollworm and native budworm in cotton. Additionally, it is sprayed on cotton, vegetables, and fruits. Poisoning can occur through ingestion, inhalation, physical contact, translaminar action, or during preening and rewetting of surfaces. Introduced in the United States in 2000, indoxacarb is considered a safer alternative to organophosphates.

Patient Information

The patient is a 60-year-old male, painter, residing in Kolhapur. He has no significant past medical history and is not on any regular medications.

Incident Details

The patient ingested approximately 50 ml of 14.5% indoxacarb pesticide while under the influence of alcohol in a suicide attempt. He presented to the emergency department within four hours of ingestion.

Mechanism of Action (MOA)

Indoxacarb is a neurotoxin that blocks sodium channels in the nervous system, leading to methemoglobinemia, hemolytic anemia, and allergic skin reactions.

Symptoms and Clinical Findings

The patient presented with nausea, vomiting, dizziness, headache, abdominal pain, dyspnea, and a general feeling of uneasiness. Upon admission, vital signs were recorded as follows: blood pressure was 140/100 mm Hg, heart rate was 116 bpm, respiratory rate was 30 breaths per minute, temperature was 36.4°C, and oxygen saturation was 84% on room air. The ingestion occurred 4 hours prior to presentation.

During the physical examination, the patient was found to be alert and oriented. Abdominal examination revealed an epigastric burning sensation since ingestion, with no guarding or rebound tenderness. Respiratory assessment showed equal bilateral air entry and clear auscultation, though the patient was tachypneic. Neurologically, there were no focal deficits, and reflexes were normal. Local examination revealed a bluish discoloration of the fingertips on both upper limbs and muddy brown-colored urine.

Diagnostic Assessment**Laboratory Tests**

- Complete Blood Count (CBC)
- Hemoglobin (Hb): 11.4 gm/dL
- Total Leukocyte Count (TLC): 11,000 /ccmm

- Platelets: 1.3 lakh/mm³
- Mean Corpuscular Volume (MCV): 90 fl
- **Electrolytes**
- Sodium (Na): 140 mmol/L
- Potassium (K): 3.5 mmol/L
- Chloride (Cl): 108 mmol/L
- **Liver Function Tests (LFT)**
- Total Bilirubin: 2.5 mg/dL
- Indirect Bilirubin: 1.8 mg/dL
- **Renal Function Tests (RFT)**
- Within normal limits
- **Urine**
- Color: Muddy brown or chocolate brown on spot test
- Routine Microscopy: No abnormalities, pigments, or RBCs
- **Arterial Blood Gas Analysis**

Parameter	On Admission	After Treatment
pH	7.34	7.46
pCO ₂	27.7 mmHg	25.1 mmHg
HCO ₃	14.4 mmol/L	19 mmol/L
pO ₂	70 mmHg	96 mmHg
AG	25 mmol/L	16 mmol/L
SO ₂	93%	98%
MethHb	9.2%	1.0%

- **Methemoglobin Levels**
- Day 1: 9.2%
- Day 3: 1.0%
- **Glucose-6-Phosphate Dehydrogenase (G6PD) Levels (Qualitative):** Not deficient

Imaging

- Chest X-ray: No abnormalities detected
- Ultrasound (USG) Abdomen: Altered echo-texture of liver, mild ascites
- High-Resolution CT (HRCT) Chest: No abnormalities detected

Management**Initial Management**

Upon presentation to the emergency department, the patient received gastric lavage within one hour using 2 liters of normal saline. Intravenous fluids were also initiated to ensure adequate hydration.

Supportive Care

The patient was given antacids and closely monitored throughout the treatment.

Definitive Treatment

Given the diagnosis of drug (poison)-induced methemoglobinemia, methylene blue and vitamin C were considered.

After ruling out glucose-6-phosphate dehydrogenase (G6PD) deficiency, methylene blue was administered at a dose of 1 mg/kg over 30 minutes (70 mg in this case). The maximum permissible dose is 7 mg/kg. The patient's oxygen saturation gradually increased from 84% to 88% over 4-6 hours. Due to persistent hypoxia (SpO₂ = 90%), this dose was repeated 12 hours later and the next morning.

Progression

Over the next 24 hours, the patient showed clinical improvement, with the bluish discoloration reversing. Other potential complications of the poisoning, such as seizures and arrhythmias, did not occur.

Psychiatric Evaluation

The patient underwent interpersonal counseling sessions and was advised on alcohol abstinence. He was encouraged to stay connected with loved ones and was prescribed antidepressants for 10 days until the next visit.

DISCUSSION

Indoxacarb is a broad-spectrum insecticide that exerts its toxic effects by blocking sodium channels in the nervous system, leading to paralysis and death. In humans, the exact mechanism of toxicity is not well understood, but symptoms can include gastrointestinal distress, dizziness, and, in severe cases, seizures and respiratory failure. There is no specific antidote for indoxacarb poisoning.

Methemoglobinemia typically presents with the abrupt onset of hypoxia following exposure to an oxidizing compound that induces methemoglobin formation. Symptoms generally begin when methemoglobin levels exceed 7-10%, with cerebral ischemia occurring at levels greater than 15% and lethality at levels over 60%. Cyanosis becomes apparent when methemoglobin levels reach 8-12%.

Cytochrome B5 reductase and glutathione are among the *in vivo* reducing agents involved in counteracting methemoglobinemia. Acquired methemoglobinemia is a medical emergency that requires a high degree of suspicion for accurate diagnosis. Key diagnostic clues include exposure to a known toxin, cyanosis disproportionate to pulse oximetry readings, a pulse oximetry SpO₂ of approximately 85% that does not improve with oxygenation, respiratory or neurological symptoms, and the presence of severely dark red or brownish-blue blood that does not turn red with oxygenation.

This case underscores the importance of early recognition and supportive management in cases of indoxacarb poisoning. Activated charcoal can be beneficial if administered promptly. Healthcare providers should be aware of the potential toxicity of indoxacarb and provide appropriate supportive care.

CONCLUSION

Acute indoxacarb poisoning is rare but can present with significant gastrointestinal and neurological symptoms. Prompt supportive care and close monitoring are essential for a favorable outcome. Further studies are needed to better understand the human toxicity profile of indoxacarb and develop specific treatment protocols.

Conflict of Interest: No conflict of interest.

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