



METHEMOGLOBINEMIA IS AN ATYPICAL MANIFESTATION OF INDOXACARB POISONING A CASE REPORT .

Emergency Medicine

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KEYWORDS

Indoxacarb is an oxadiazine insecticide, which acts by blocking sodium channels in the nervous system of insects and causes tremors, cessation of feeding, and death in a couple of hours. Contact with the substance can take place through ingestion, physical contact, transaminar action, during preening, and at rewetting of surfaces.

In our patient methemoglobinemia occurred following ingestion of indoxacarb. This case report highlights the importance of considering the possibility of methemoglobinemia in cases of exposure to indoxacarb and its early recognition and management.

Case Report:

A 30-year-old male farmer came to the emergency department 10 hours after ingestion of unknown insecticide poison followed by 6 episodes vomiting. Initially treated at local hospital with atropine. On examination, his Glasgow coma scale score was 10/15 and afebrile. On examination patient was drowsy and hypoventilating, pooling of oral secretions, gurgling sounds present, His SpO₂ was 85% on 15 lit / min of oxygen, B.P systolic 60 mmHg. Therefore, a decision was taken to intubate the patient and commence mechanical ventilation. After 1.5 litres of normal saline bolus B.P was 100/60 mmHg. He was shifted to General intensive Care Unit (GICU).

Routine and special investigations were carried out and the results were, LFT showed SGOT of 128 IU/L, SGPT of 109 IU/L; S. Creatinine was normal, Urine-pH-5.6, Specific gravity-1.016, no pus cells, no RBC, TLC-18,600.

the patient's blood was noticed to be muddy brown in colour, and peripheral cyanosis (bluish brown colour over tongue, lips) was noticed. An arterial blood gas analysis revealed a paO₂ of 142 mmHg with 15 lit/min O₂ and the SpO₂ was only 86%, SaO₂ was 99%.

After ruling out other poisons, and with a high degree of suspicion, Methemoglobinemia was considered which could be secondary to ingestion of indoxacarb. Blood was sent for methemoglobin level and co-oximetry. Methemoglobin level was found to be 33.4%. Immediately sterile inj. Methylene blue in 100 ml normal saline, was administered intravenously at a dose of 1.5 mg/kg. Methylene blue 60 mg IV BD and Inj. Vitamin-C 1 g IV were continued. The patient gradually started showing signs of improvement and his SpO₂ gradually improved to 92 - 95% on day two. The patient was gradually weaned off ventilatory support. The SpO₂ increased to 98% on day three. The patient was subsequently extubated on day three and continued to maintain a good gas exchange on face mask oxygen. On day four, the SpO₂ was 99% on face mask with 4 liters/min. of oxygen. On day four, the methemoglobin level was 1%. The patient was discharged from ICU on day five.

Thus our patient was managed successfully with methylene blue and other supportive/symptomatic treatment for methemoglobinemia which was probably secondary to ingestion of indoxacarb poisoning.

DISCUSSION:

Indoxacarb is an oxadiazine pesticide used for control of cotton bollworm and native budworm in cotton and soyabeans. Our Patient had developed peripheral cyanosis and had muddy brown

discoloration of blood. With an index of suspicion, after ruling out other poisonings, methemoglobinemia was diagnosed and treated. Methemoglobin is generated by oxidation of the heme iron moieties to ferric state, causes bluish-brown muddy color resembling cyanosis. It has got very high affinity to oxygen and oxygen is not delivered to the tissues (oxygen dissociation curve shifted to the left). It is suspected in patients with hypoxic symptoms who appear cyanotic but have a sufficiently high PaO₂. Muddy appearance of freshly drawn blood is a critical clue for diagnosis. Normal Meth-Hb levels are <1%.

The physiologic reduction of MethHb Fe³⁺ to Hb Fe²⁺ is mainly accomplished by red cell NADH-cytochrome b5 reductase.

It is manifested by Muddy brown discoloration of fresh blood, Bluish discoloration of skin and mucosa (at 1.5% or 10% of meth-hb), irritability, seizures. Cerebral ischemia occurs at >15% and >60% are lethal. It can be congenital or acquired due to drugs/chemicals. It can be diagnosed by co-oximetry, methemoglobin levels, ABG, SpO₂. In our patient methemoglobin was 33.4%.

Treatment includes ceasing the offending agent, correcting the metabolic abnormalities, administering methylene blue at a dose of 1-2 mg/kg loading dose q 30-60 min, to a maximum of 7 mg/kg, followed by 50-100 mg twice or thrice daily and other supportive measures as required. In our patient, we instituted treatment with 1.5 mg/kg of methylene blue as a loading dose, followed by 60 mg twice daily. Methylene blue gets reduced to leucomethylene blue which in turn reduces methemoglobin by NADPH reductase. Methylene blue reduces half-life of methemoglobin from 15-20 hours to 40-90 minutes. We also administered vitamin C 1 gm/day and dextrose containing fluids for supplementing NADH/ NADPH which is needed for reduction of methemoglobin by NADPH reductase enzyme. The patient's ventilation was supported mechanically for few days. He made an uneventful recovery and was discharged home after five days.

In our patient, methemoglobinemia had occurred following ingestion of indoxacarb, a pesticide, which was identified due to a high degree of suspicion and treated early with methylene blue. Literature search for methemoglobinemia secondary to indoxacarb did not retrieve any articles. Hence, this case report of methemoglobinemia secondary to indoxacarb poisoning.

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