



Letters to the Editor

Management of indoxacarb poisoning in a regional setting

Sir,

A 23-year-old male was admitted in our emergency room 3 h after suicidal ingestion of about 50 ml of unknown insecticide. His medical and family histories were negative for hereditary disorders. The patient was feeling lethargic and headache. On examination, he had stable vital signs. The heart and lung sounds were normal on auscultation. The skin and nail beds were grayish colored that did not respond to supplemental oxygen therapy. His oxygen saturation on pulse oximetry was 86% despite 6 L/min of supplemental oxygen. An arterial blood sample was obtained that appeared to be a dark brown color. The arterial blood gas (ABG) sample showed a pH of 7.37, an arterial oxygen pressure (PO₂) of 129 mm Hg, a PCO₂ of 44 mm Hg, bicarbonate of 24.9 mmol/L, and an oxygen saturation of 90%. His findings were suggestive for methemoglobinemia. The patient's relatives brought the poison container about 4 h after admission; and it was indoxacarb, an oxadiazine insecticide.

Methemoglobin is an abnormal type of hemoglobin in which oxygen-carrying ferrous ion (Fe²⁺) of heme is oxidized to the ferric state (Fe³⁺); hence, a decreased ability of the abnormal hemoglobin to release oxygen to the tissues causes hypoxia.^[1,2] Normally, <3% of hemoglobin is oxidized to methemoglobin each day, which is reduced with an NADH methemoglobin reductase (cytochrome b5 reductase) and to a lesser extent with the NADPH methemoglobin reductase, Vitamin C, and glutathione enzyme systems.^[3] Congenital methemoglobinemia is a consequence of diminished enzymatic reduction of intrinsic methemoglobin, whereas acquired methemoglobinemia results from exposure to drugs or toxins that cause oxidation of hemoglobin. When methemoglobin formation overwhelmed protective enzyme systems, its levels rise and cause diminished oxygen delivery to tissues.^[1] In patients with significant manifestations of tissue hypoxia,

besides administration of high-flow O₂, treatment with specific antidote, methylene blue, or Vitamin C, and hyperbaric oxygen should be considered.^[1,4]

Methemoglobinemia has been reported in a few cases of indoxacarb poisoning.^[5] Unfortunately, equipment was not available to measure methemoglobin level in our center. However, symptoms of headache and lethargy, combined with the clinical findings of grayish cyanosis and the brown color of the phlebotomized blood, without significant cardiopulmonary dysfunction were strong enough to reach a diagnosis of methemoglobin and initiate therapy. Since methylene blue, the choice of specific therapy, was not available in our center, treatment with ascorbic acid was considered.^[1,4]

The patient had no history of G6PD deficiency or hemolytic events; hence, he received 1500 mg of Vitamin C every 4 h intravenously. One hour after the third dose, another ABG sample showed a pH of 7.47, PO₂ of 67 mm Hg, a PCO₂ of 31 mm Hg, bicarbonate of 21.9 mmol/L, and an oxygen saturation of 91% and his oxygen saturation on pulse oximetry was 90%. After this dramatic response, the patient did not develop another episode of cyanosis.

In acute poisoning, methemoglobinemia as an important side effect should be considered if the patient presents with sudden onset of symptoms of hypoxia, and diminished oxygen saturation does not improve with supplemental oxygen. In such case, high arterial oxygen pressure despite clinical cyanosis is highly suggestive for methemoglobinemia.

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Conflicts of interest

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