Imidacloprid Poisoning

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Abstract

Imidacloprid is newer systemic insecticide, a nicotine analogue, acts on the nervous system. Patient can present with variable manifestations like irritability, labored breathing, emaciation, twitching and delirium. Here we report a case presented with sever neuropsychiatric symptoms with respiratory failure following self ingestion of poison. Patient recovered with supportive and symptomatic treatment.

Introduction

Imidacloprid is a relatively new insecticide in the chloronicotinyl nitroguanidine class. It was first registered for use as a pesticide in the U.S. in 1994 and was the first insecticide in its chemical class to be developed for commercial use.[1] Imidacloprid has a wide variety of uses; it is used on cotton and vegetable crops, turf grass and ornamental plant products, in indoor and outdoor cockroach control products and in termite control products. It is also used in products for pets, home lawn and garden use including some, like potting soil, which may not always be easily recognized as pesticides.

Imidacloprid acts as a competitive inhibitor at nicotinic acetylcholine receptors in the nervous system.[2] It effectively blocks the signals induced by acetylcholine at the post-synaptic membrane, resulting in impairment of normal nerve function.[2,4] Imidacloprid has a higher binding strength to insect nerve receptors than to mammalian receptors.[2]

Poisoning with imidacloprid has been reported to have very low toxicity. We are reporting a case of self poisoning with imidacloprid poisoning leading to severe psychiatric symptoms and respiratory failure.

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Case Report

A forty one year male patient was brought to emergency with alleged history of self ingestion of 75ml 70% imidacloprid three hours before admission. He had developed nausea, vomiting, abdominal cramps, muscle twitching and difficulty in breathing within 30 minutes of ingestion of poison. He had no significant co-morbid medical illness or any addiction. On arrival in emergency room he was found to be drowsy and dyspnoeic. On physical examination his temperature was 98°F with heart rate 115/min, blood pressure 150/90 mmHg, respiratory rate 45/min and oxygen saturation of 60%. Muscle twitching was present. There was no pallor, cyanosis or injury marks. There were scattered coarse crepitations on chest auscultation. On neurological examination he was drowsy with Glasgow Coma Scale (GCS) of 12/15 (E5, M6, V1) with no focal neurological deficit. Rest of the systemic examination was unremarkable.

Investigations showed that he had mild leucocytosis with normal hemoglobin level, RBC and platelet count. Serum electrolytes, random blood sugar, renal, liver and thyroid function was found normal. His serum cholinesterase and CPK level was normal. Chest x ray and ECG did not reveal any abnormality. Initial arterial blood gas showed metabolic acidosis (pH 7.2, HCO_3 15mmol/L, $PaCO_2$ 29, PaO_2 132) which normalized after 24 hours.

He was immediately resuscitated with endotracheal intubation and ventilated with ambu bag. Gastric lavage was

done and sample was collected for toxicological analysis. After he was stabilized he was shifted to medical ICU and put on invasive ventilator support. Literature was reviewed after obtaining the poison container. As there was no antidote available, the patient was treated symptomatically with IV fluids, antibiotics as prophylaxis against aspiration pneumonia and supportive care provided with mechanical ventilation and general nursing care. After 12 hours of ventilation the patient was fully conscious and started developing neuropsychiatric manifestations like agitation and delirium. Due to severe agitation he self extubated so he was sedated and reintubated. After 96 hours, neuropsychiatric manifestations subsided and he was weaned off from the ventilator and extubated on day five. Patient was shifted to the ward on the next day and after psychiatric counseling he was discharged. After a week the patient was followed up in out patient department, he was found fit to resume his work.

Discussion

Imidacloprid was developed in 1985 with the aim of combining compounds with high potency against insects with low mammalian toxicity and favorable persistence. On the basis of animal studies, it is classified as a "moderate toxic" (class II by WHO and toxicity category II EPA).[1] It is not banned, restricted, canceled, or illegal to import in any country.[3]

A few cases of significant human toxicity due to imidacloprid have been reported in medical literature. In a prospective human case series of 68 cases, the majority of the cases developed mild gastrointestinal symptoms and only one case required mechanical ventilation for respiratory failure.[4]

Our patient during the clinical course of toxicity developed gastrointestinal irritation, respiratory failure and severe neuropsychiatric symptoms. With symptomatic and supportive care our patient recovered completely. Till date neuropsychiatric symptoms in imidacloprid poisoning has been reported in one case with inhalation exposure,[5] mainly due to central nicotinic stimulation. Biochemical abnormality like metabolic acidosis in these cases may develop due to acidic metabolites such as 6-chloronicotinic acid and other metabolite.[4] Cardiovascular manifestations were also described in different case reports like tachycardia, bradycardia, arrhythmia, cardiac arrest[6] but were not present in our case.

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