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Case Report

Imidacloprid poisoning: a modern foe

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ABSTRACT

Imidacloprid is a relatively new insecticide in the chloronicotinyl nitroguanidine class. 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. Imidacloprid acts as a competitive inhibitor at nicotinic acetylcholine receptors in the nervous system resulting in impairment of normal nerve function. Scientific literature on human imidacloprid poisoning has been relatively sparse. We report three subjects who presented with imidacloprid poisoning.

Keywords: Imidacloprid poisoning, Government medical college, Patiala

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.[1] Imidacloprid is mainly used on cotton and vegetable crops, ornamental plant, indoor and outdoor cockroach control products and in termite control products.

Imidacloprid is designed to be effective by contact or ingestion.[2] Imidacloprid acts on several types of postsynaptic nicotinic acetylcholine receptors in the nervous system.[3,4] Following irreversible binding to the receptors, nerve impulses are spontaneously discharged at first, followed by failure of the neuron to propagate any signal.[5,6] Sustained activation of the receptor results from the inability of acetyl cholinesterases to break down the pesticide.[4] Mammalian nicotinic receptors are made up of a number of subtypes and are present at neuromuscular junctions as well as in the central nervous system in contrast to insects.[6] However, the binding affinity of imidacloprid at the nicotinic receptors in mammals is much less than that of insect nicotinic receptors.[7]

Poisoning with imidacloprid has been reported to have very low toxicity. We are reporting three cases of poisoning with imidacloprid poisoning.

CASE REPORT

Case 1

A forty four year old male patient was brought to emergency with alleged history of self-ingestion of unknown substance three hours before admission. He had developed nausea, vomiting, abdominal cramps and difficulty in breathing within 30 minutes of ingestion of poison. He had no significant co-morbid medical illness. He was a chronic alcoholic and smoker. On arrival in emergency room he was found to be drowsy and dyspneic. There was no smell of alcohol. On physical examination his temperature was 98.4°F with heart rate 122/min, blood pressure 130/90 mmHg, respiratory rate 36/min and oxygen saturation of 78%. There was no pallor, cyanosis or injury marks. There were scattered coarse crepitations on auscultation of chest. On neurological examination patient was drowsy with

Glasgow Coma Scale (GCS) of 10/15 (E4, M5, V1) with no focal neurological deficit. Rest of the systemic examination was unremarkable.

Investigations showed mild leucocytosis with normal hemoglobin level, WBC and platelet count. Serum electrolytes, random blood sugar, renal, liver and thyroid function was found normal. His serum cholinesterase and CPK levels were normal. Chest X ray and ECG was normal.

He was immediately resuscitated with endotracheal intubation and was given ambu bag ventilation. After he was stabilized and he was managed at high dependency unit. Poison container was made available by the relatives 3 hours into admission, which revealed 70% imidacloprid to be the content. As there was no antidote available, the patient was treated symptomatically with IV fluids, antibiotics as prophylaxis against aspiration pneumonia and supportive care was provided. Four hours into admission patient was conscious and was extubated.

After 12 hours the patient was fully conscious. Patient was shifted to the ward on the next day and was he was discharged next day.

Case 2

Twenty eight year old male presented to emergency department, with history of self-ingestion of 50ml of imidacloprid 17.8% SL four hours prior to admission (identified form the poison container). He developed nausea, vomiting and was treated with gastric lavage at a peripheral health centre before transfer to the study centre. He had no co-morbid medical conditions or addictions. At admission, he was conscious, oriented but anxious. On physical examination his temperature was 99.1°F with heart rate 112/min, blood pressure 140/90 mm Hg, respiratory rate 28/min and oxygen saturation of 94%. There was no pallor, cyanosis or injury marks. Cardiovascular and repiratory system examination were normal. On neurological examination patient was alert, attentive with Glasgow Coma Scale (GCS) of 15/15 (E4, M6, V5) with no focal neurological deficit. Rest of the systemic examination was unremarkable.

Investigations showed that he had mild leucocytosis with normal hemoglobin level, WBC and platelet count. Serum electrolytes, random blood sugar, renal, liver and thyroid function was found normal. His serum cholinesterase and CPK levels were normal. Chest X ray and ECG was normal. Poison container was made available by the relatives 2 hrs into admission. As there was no antidote available, the patient was treated symptomatically with IV fluids, and supportive nursing care was provided. 24 hours later, patient was shifted to the ward from high dependency unit and was he was discharged next day.

Case 3

A 54-year-old male farmer was brought to the emergency medicine ward in a state of drowsiness with history of having had an acute episode of intractable vomiting and watery diarrhea for about four hours. As per his relatives, he was spraying some pesticides (which was later found to be 70% imidacloprid) in his fields, and after an hour, he felt unwell and had difficulty breathing. Later on he developed nausea, vomiting, abdominal cramps, and muscle twitching for which he had taken treatment prescribed by a local practitioner, and was further referred to higher centre due to persistence of symptoms. Upon arrival, he was drowsy, had dyspnea and was unable to stand unsupported. There was no significant co-morbid medical illness, and relatives also denied consumption of any drugs, poison or medications except for the use of pesticide fumigation. The relatives also showed the empty container of insecticidal spray which was being used by the patient. Upon inspection of the spray, it mentioned the constituent of the insecticide as being imidacloprid 17.80 SL.

On physical examination his temperature was 98.2°F with heart rate 106/min, blood pressure 140/90 mm Hg, respiratory rate 28/min and oxygen saturation of 72%. There was no pallor, cyanosis or injury marks. On neurological examination he was drowsy, having Glasgow Coma Scale (GCS) of 7/15 (E2, M3, and V2) with no focal neurological deficit. Investigations showed that he had mild leukocytosis with normal hemoglobin levels, RBC and platelet counts. Serum electrolytes, random blood sugar, and renal function were found to be normal but liver functions were deranged with SGOT & SGPT levels as 79 and 67 IU/ L, respectively. Chest X-ray and ECG were normal. The patient was treated symptomatically along with good supportive and general nursing care in the absence of any specific antidote. On the second day his respiratory function and oxygen saturation improved. Patient was kept under observation for 48 hours and was discharged with stable hemodynamic and biochemical parameters.

Figure 1: Imida prime: systemic insecticide.

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]

Imidacloprid acts as a competitive inhibitor at nicotinic acetylcholine receptors in the nervous system resulting in impairment of normal nerve function.[8] It has a higher binding strength to insect nerve receptors than to mammalian receptors and is reported to have very low toxicity to human beings. It generally demonstrates low human lethality even in large ingestions. Few case reports of attempted suicides have been described.[9,10] Symptoms range from neuropsychiatric symptoms, cardiovascular manifestations like tachycardia, bradycardia, arrhythmia, and cardiac arrest.[9] There is a paucity of information about human toxicity.

It is not banned, restricted, canceled, or illegal to import in any country.[11] 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 from Srilanka, the majority of the cases developed mild gastrointestinal symptoms and only one case required mechanical ventilation for respiratory failure and no mortality was reported. It was demonstrated that imidacloprid self-poisoning resulted in mostly minor toxicity and was nonfatal. It seems favorable compared with outcomes with other insecticides, particularly organophosphorus compounds which commonly have a case fatality between 5% and 30%.[12,13]

One patient during the clinical course of toxicity developed gastrointestinal irritation, and respiratory failure. With symptomatic and supportive care all the three recovered completely.

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