Case Report

Imidacloprid Poisoning: An Emerging Cause of Potentially Fatal Poisoning

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Abstract

There are a variety of pesticides that are used to control the pests in agricultural lands and other places. Newer pesticides, developed as an alternative to highly toxic organophosphates such as imidacloprid including other neonicotinoid compounds, are being increasingly used considering their less harmful effects in case of human exposures. Though it is considered relatively safer to human beings, it can lead to potentially life-threatening complications and acute poisoning with these compounds may be fatal in large ingestion. We report a case of poisoning with imidacloprid compound presenting with a variety of systemic features including respiratory failure and patient's improvement with conservative management.

Keywords: Imidacloprid, neonicotinoid, respiratory arrest, suicide, ventilatory support

Introduction

Acute pesticide poisoning is a major health problem worldwide; organophosphorus poisoning accounts for most fatalities, and therefore alternative insecticides are used, hoping them less harmful to humans but potent enough to control pests. Imidacloprid belongs to neonicotinoid class, which selectively acts on nervous system of pests via nicotinic acetylcholine receptors (specifically a4b2 subtype), resulting in their favorable toxicological profile in case of human exposures.[1] We report a case of imidacloprid poisoning with suicidal intention who developed a variety of manifestations including paroxysmal atrial fibrillation, hypokalemia, central nervous system (CNS) depression, and respiratory arrest requiring mechanical ventilation and who recovered subsequently with supportive care.

Case Report

A 53-year-old male presented to our hospital with history of intentional consumption of 200 ml imidacloprid compound (70% WP) one hour prior to admission. On admission, he had abdominal discomfort, three episodes of vomiting and no other significant complaints. His pulse was irregularly irregular – 96/min, blood pressure (BP) – 110/70 mmHg, respiratory rate – 18/min, SpO_2 – 99% with room air, congested oral mucosa, and detailed systemic examination was normal. Electrocardiography (ECG) confirmed atrial fibrillation, and treatment with anticoagulation and diltiazem was planned, but before it was started, sinus rhythm was restored without any intervention and treatment was withheld. Investigations demonstrated normal hemogram, serum creatinine – 0.98 mg%, serum sodium – 134 mEq/L, serum potassium – 4.1 mEq/L, serum calcium – 9.2 mg%, phosphorous – 3.8 mEq/L, random blood sugar – 129 mg%, serum cholinesterase – 3987.5, and serum creatine phosphokinase-MB – 35 IU/L. Over the next 3 h, the patient became progressively drowsy and developed respiratory arrest for which he was intubated and ventilatory support was initiated. Repeat examination revealed Glasgow Coma Scale (GCS) score – 4/15, generalized hypotonia, no respiratory secretion, normal Doll's eye reflex, and no papilledema. Cardiac monitoring showed sinus tachycardia with normal BP, arterial blood gas analyzer (ABGA) showed pH – 7.21, PaO_2 – 65, SpO_2 – 88, pCO_2 – 64, and HCO_3 – 27. Repeat electrolytes and sugar were within the normal range.

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Ventilatory support of the patient was continued while FiO_2 was decreased to 21%. After 24 h, the patient had spontaneous respirations and sensorium improved over the next 24 h. Detailed neurological examination showed normal muscle strength and no focal neurological deficit. On the 4th day, the patient had hemorrhagic aspirate in Ryle's tube and was given cold saline lavage with 80 mg intravenous pantoprazole bolus followed by infusion at 8 mg/h for 24 h. Repeat coagulation parameters were within the normal limits and bleeding was controlled. Meanwhile, weaning process was started and the patient was weaned off from ventilatory support. Next day, the patient was asymptomatic, but repeat electrolytes showed hypokalemia (K^+ - 2.53 mEq/l) in the absence of muscle weakness and ECG signs of hypokalemia. Further evaluation showed urine potassium – 10.8 mMol/l (25–125), urine sodium – 336 mMol/l (40–250), urine pH – 7.0, urine osmolarity – 800 mOsm/kg (300–900), serum osmolarity – 271 mOsm/l (275–290) with transtubular potassium concentration gradient – 1.25, and ABGA showed normal acid–base balance. Hypokalemia was corrected with KCl infusion. The patient was evaluated with echocardiography and thyroid function tests which were normal and was discharged on the 7th day after psychiatric evaluation.

Discussion

Imidacloprid belongs to neonicotinoids compounds, chemically similar to nicotine, and other members of neonicotinoid class include acetamiprid, clothianidin, thiacloprid, dinotefuran, nitenpyram, and thiamethoxam.[2] Imidacloprid is the first neonicotinoid compound commercialized with widespread use and is most common among human intoxications due to neonicotinoids.[2] It is classified as moderately hazardous (Class-II WHO; toxicity category-II EPA) based on animal studies.[3] These compounds can be absorbed via ingestion, dermal or inhalational route, and there is more severe poisoning with oral ingestion than other routes.[1] Neonicotinoids are agonists at nicotinic acetylcholine receptors and interfere with transmission of impulses by increased activation, leading to fatigue and paralysis.[4] Receptor stimulation affects CNS as well as autonomic nervous systems. Selective toxicity to insects as compared to mammals is because of different structures and compositions of receptor subunits. CNS stimulation causes dizziness, drowsiness, disorientation, and coma while autonomic nervous system stimulation causes sweating, dilated pupils, tachycardia, and hypertension which may lead to coronary spasm and cardiac ischemia and therefore with the risk of arrhythmia, hypotension, and bradycardia.[5] The literature of imidacloprid poisoning in humans is scarce and mostly includes case reports and few studies.[2] A multicentric study from Sri Lanka, Portugal, and Australia by Mohamed *et al*. revealed that plasma concentration of imidacloprid is not useful in the clinical management of imidacloprid poisoning, whereas features such as dyspnea, apnea, coma, and mydriasis indicated severe poisoning.[3] A case report by Todani *et al*. reported paroxysmal atrial fibrillation lasting for 11 h with acetamiprid poisoning and additionally the patient in their case had CNS depression (GCS-8), hypotension, nausea, vomiting, and hyperglycemia, and improved with supportive care.[6] Huang *et al*. reported a case of ventricular fibrillation within 2 h following ingestion of imidacloprid and this patient presented with altered sensorium with diaphoresis, vomiting, tachycardia, and later developed apnea, cyanosis, and ventricular fibrillation and died.[7] Another case report by Agha *et al*. reported leukocytoclastic vasculitis with hepatic and kidney dysfunction with imidacloprid and stated that imidacloprid toxicity should be considered in the differential diagnosis of multiorgan failure following unknown poisoning.[8] A retrospective analysis of neonicotinoid exposures at Taiwan National Poison Center revealed mortality of 2.85% (2/70) and the causes of mortality were aspiration and respiratory failure and stated ventilatory support as the most important in clinical management.[9] In another case report, Panigrahi *et al*. reported a successful outcome of respiratory arrest treated with ventilatory support while Agarwal and Srinivas reported neuropsychiatric manifestations and rhabdomyolysis with imidacloprid and both patients improved with supportive management.[10,11] Viradiya and Mishra also reported severe neuropsychiatric symptoms with respiratory failure following ingestion of imidacloprid and recovery with symptomatic treatment.[12] Karatas reported a case of imidacloprid ingestion who presented with disorientation, increased salivation, and improvement with supportive management.[13] Shadnia and Moghaddam reported fatal imidacloprid poisoning despite adequate supportive care.[14] Our patient presented with gastrointestinal symptoms followed by transient atrial fibrillation with raised cardiac enzymes and later on developed CNS depression with respiratory arrest. A review of the available literature indicates that imidacloprid poisoning can involve gastrointestinal, cardiorespiratory, and nervous systems or it can be multisystem and can be life threatening. Treatment of imidacloprid poisoning largely remains supportive in the absence of effective antidote.[3] Our patient was treated with supportive care and he recovered without any sequelae. As neonicotinoids are considered relatively less toxic, here with this case report and with the review of literature, we want to stress that there should be close monitoring of poisoning due to imidacloprid and these patients should be managed with supportive care.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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