

## Imidacloprid Associated Convulsions: A Rare Case Report

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**How to cite this article:** Shashidhara KC, Savitha V, Jerin Joseph Abraham et al. Imidacloprid Associated Convulsions: A Rare Case Report. Indian Journal of Forensic Medicine and Toxicology/Volume 18 No. 3, July - September 2024.

### Abstract

**Background:** Acute pesticide poisoning is a global health concern, with organophosphorus compounds contributing significantly. Neonicotinoids, like imidacloprid, have emerged as alternative insecticides due to their selective action on pest nervous systems. This case report explores imidacloprid poisoning, emphasizing clinical manifestations and the ongoing search for safer pesticides.

**Methods:** A 46-year-old male, chronic smoker, and alcoholic, presented with suicidal imidacloprid poisoning. The patient experienced seizures, central nervous system (CNS) depression, hyponatremia, and respiratory arrest, necessitating mechanical ventilation. The case details the comprehensive evaluation, including imaging studies, and presents vital signs, laboratory results, and physical examinations. The patient received supportive care, and the discussion outlines the treatment strategy, highlighting the challenges and successes in managing imidacloprid toxicity.

**Conclusion:** This case underscores the complexity of imidacloprid poisoning, particularly noting convulsions secondary to hyponatremia. It stresses the need for further research into imidacloprid's metabolic effects and advocates for vigilant monitoring and supportive care in such cases. The report calls for increased reporting to enhance understanding and knowledge sharing regarding the potential toxic effects of imidacloprid, contributing to better management strategies for pesticide poisoning.

**Keywords:** Imidacloprid, Convulsions, Dyselectrolytemia

### Introduction

Acute pesticide poisoning is a major health problem worldwide; organophosphorus poisoning accounts for most fatalities, and therefore alternative

insecticides are used, hoping they are less harmful to humans but potent enough to control pests. Acute pesticide toxicity is extremely common in developing countries of the Asia-Pacific region, particularly

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**Submission date:** December 29, 2023

**Revision date:** January 22, 2024

**Published date:** July 17, 2024

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in setting of low education and poor regulatory frameworks. India is an agricultural country with a large rural population (60–80%), where pesticides are freely available and are used extensively and quite frequently for self-poisoning.

Imidacloprid belongs to neonicotinoid class, which selectively acts on nervous system of pests via nicotinic acetylcholine receptors (specifically  $\alpha 4\beta 2$  subtype), resulting in their favourable toxicological profile in case of human exposures<sup>1</sup>. Despite its lower toxicity, several cases have been reported with a range of serious complications, including neuropsychiatric sequelae, rhabdomyolysis resulting in acute kidney injury, ischemic and metabolic encephalopathy, ventricular fibrillation, multiorgan failure, and even death after exposure to imidacloprid.

We report a case of imidacloprid poisoning with suicidal intention who developed a variety of manifestations including hyponatremia, convulsions, central nervous system (CNS) depression, and respiratory arrest requiring mechanical ventilation and who recovered subsequently with supportive care.

### Case Report

A 46-year-old male patient who is smoker and chronic alcoholic, 65kgs in weight and previously in good health, farmer by occupation from rural area of Karnataka with no past history of Seizure disorder, head injury or any previous cerebrovascular events, presented to the Emergency department with H/o accidental inhalational contact with FIPROCIL-40% + IMIDACLOPRID 40%. Patient was exposed to the insecticide as he was spraying it without any protective gear. Imidacloprid belongs to neonicotinoids compounds, chemically similar to nicotine, and other members of neonicotinoid class include acetamiprid, clothianidin, thiacloprid, dinotefuran, nitenpyram, and thiamethoxam. Patient presented with an episode of seizure and was drowsy and disoriented. Patient was intubated in view of low GCS score-8. CT BRAIN showed essentially normal study.

Upon the initial physical examination, His vital signs were as follows: a blood pressure reading of 130/82 mmHg, a pulse rate of 114 beats per minute, a

respiratory rate of 16 breaths per minute, and a body temperature of 96.6°F. Pupils were of equal size and reacted promptly to light. Clear breath sounds were heard bilaterally, and the heartbeat was regular, with no audible murmurs. The abdomen appeared soft, flat, and free from tenderness.

The laboratory results showed the following values: haemoglobin level at 13.9 mg/dL, white blood cell count at 12070/mm<sup>3</sup>, platelets at 231000/mm<sup>3</sup>, prothrombin time at 12.1/0.88 seconds, activated partial thromboplastin time at 28.8 seconds, albumin at 4.5 g/dL (within the normal range of 3.5–5.3 g/dL), glucose at 101 mg/dL (normal range: 70–105 mg/dL), aspartate aminotransferase (AST) at 47 U/L (normal range: 0–36 U/L), alanine aminotransferase (ALT) at 21 U/L (normal range: 0–36 U/L), blood urea nitrogen at 9 mg/dL (normal range: 6–21 mg/dL), creatinine at 0.65 mg/dL (normal range: 0.6–1.2 mg/dL), sodium at 115mEq/L (normal range: 134–148 mEq/L), potassium at 4.4mEq/L (normal range: 3.0–4.8 mEq/L), and calcium at 7.4 mg/dL (normal range: 7.9–9.9 mg/dL).

Urinalysis was unremarkable. An electrocardiogram (EKG) showed sinus tachycardia. The chest radiograph showed no radiological abnormality. 2d echo showed mitral valve prolapse with EF-55%. Ultrasound abdomen and pelvis showed raised cortical echoes of bilateral kidneys with mildly globular left kidney, minimal interloop free fluid. The patient was started on prophylactic antibiotics, antiepileptics and other supportive care was given.

### Discussion

In this instance, the individual was exposed to Imidacloprid. Initially, the patient presented drowsy and disoriented along with an episode of seizure. Patient was intubated in view of low GCS score and was given supportive care. Hyponatremia and Hypocalcemia were corrected. IV Antiepileptics were administered. He was extubated and post extubation vitals were stable.

Imidacloprid is the first neonicotinoid compound commercialized with widespread use and is most common among human intoxications due to neonicotinoids. It is classified as moderately

hazardous (Class-II WHO; toxicity category-II EPA) based on animal studies<sup>2</sup>. These compounds can be absorbed via ingestion, dermal or inhalational route, and there is more severe poisoning with oral ingestion than other routes. Neonicotinoids are agonists at nicotinic acetylcholine receptors and interfere with transmission of impulses by increased activation, leading to fatigue and paralysis<sup>3</sup>. 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<sup>4</sup>.

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. 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<sup>2</sup>.

### Conclusion

This case discusses the first reported case of Imidacloprid causing convulsions secondary to hyponatremia. It highlights the limited availability of case reports regarding the toxic metabolic effects of Imidacloprid. The article underscores

the need for further studies and case reports to gain a better understanding of Imidacloprid toxic effects on various systems in humans. As a result, careful patient observation is essential in cases of Imidacloprid poisoning to gather more information about its potential toxic effects.

Informed consent has been taken from the patient in their understandable language.

**Conflicts of Interest:** Nil

**Source of funding:** Self

**Ethical Clearance:** INSTITUTIONAL ETHICS COMMITTEE,

IEC Registration ECR/387/  
INST/KA/2013/RR-19, 30  
Oct' 2023.

Ref No: JSSMC/  
IEC/27102023/ 49  
NCT/2023-24

### References

1. Tomizawa M, Casida JE. Neonicotinoid insecticide toxicology: Mechanisms of selective action. *Annu Rev Pharmacol Toxicol.* 2005;45:247-68.
2. Mohamed F, Gawarammana I, Robertson TA, Roberts MS, Palangasinghe C, Zawahir S, et al. Acute human self-poisoning with imidacloprid compound: A neonicotinoid insecticide. *PLoS One.* 2009;4:e5127.
3. Zhang A, Kayser H, Maienfisch P, Casida JE. Insect nicotinic acetylcholine receptor: Conserved neonicotinoid specificity of [(3)H] imidacloprid binding site. *J Neurochem.* 2000;75:1294-303.
4. Lin PC, Lin HJ, Liao YY, Guo HR, Chen KT. Acute poisoning with neonicotinoid insecticides: A case report and literature review. *Basic Clin Pharmacol Toxicol.* 2013;112:282-6.