



## SELF POISONING BY IMICON – A CASE REPORT


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### ABSTRACT

The agricultural use of organophosphorus, organochlorine, carbamate, pyrethroid, and nitrogen-based insecticides to protect crops from insects has increased enormously in recent years. In India the ready availability of these insecticides has led to a huge increase in human poisoning. The insecticide imidacloprid, 1- [(6-chloro-3-pyridinylmethyl)-4, 5-dihydro-N-nitro-1H-imidazol- 2-amine, is often misused for homicidal or suicidal purposes. 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. We report a case of self-poisoning with imidacloprid with severe neuropsychiatric symptoms, respiratory failure leading to death.

**Key words:** Self-poisoning, Imidacloprid, Neonicotinoid, Insecticides.

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### INTRODUCTION

The neonicotinoids are a new major class of highly potent insecticides, insecticides within this class includes imidacloprid, acetamiprid, clothianidine, and thiacloprid. 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.

Uses for individual products containing imidacloprid vary widely. The neonicotinoids are a new major class of highly potent insecticides that are used for crop protection and flea control [2].

Imidacloprid has a higher binding strength to insect nerve receptors than to mammalian receptors. Animal studies indicate relatively low toxicity to mammals because they have resistant nicotinic receptor subtypes compared to insects, as well as protection of the central nervous system by the blood brain barrier. Despite wide usage, human exposure experience resulting in toxicity is quite limited [3]. On the basis of animal studies it is classified as moderately hazardous (Class II WHO; toxicity category II EPA) [4,5]. Data on human exposure to imidacloprid is limited to occupational exposure [6,7,8,9].

Imidacloprid acts as a competitive inhibitor at nicotinic acetylcholine receptors in the nervous system. It effectively blocks the signals induced by acetylcholine at the post-synaptic membrane, resulting in impairment of normal nerve function [3].

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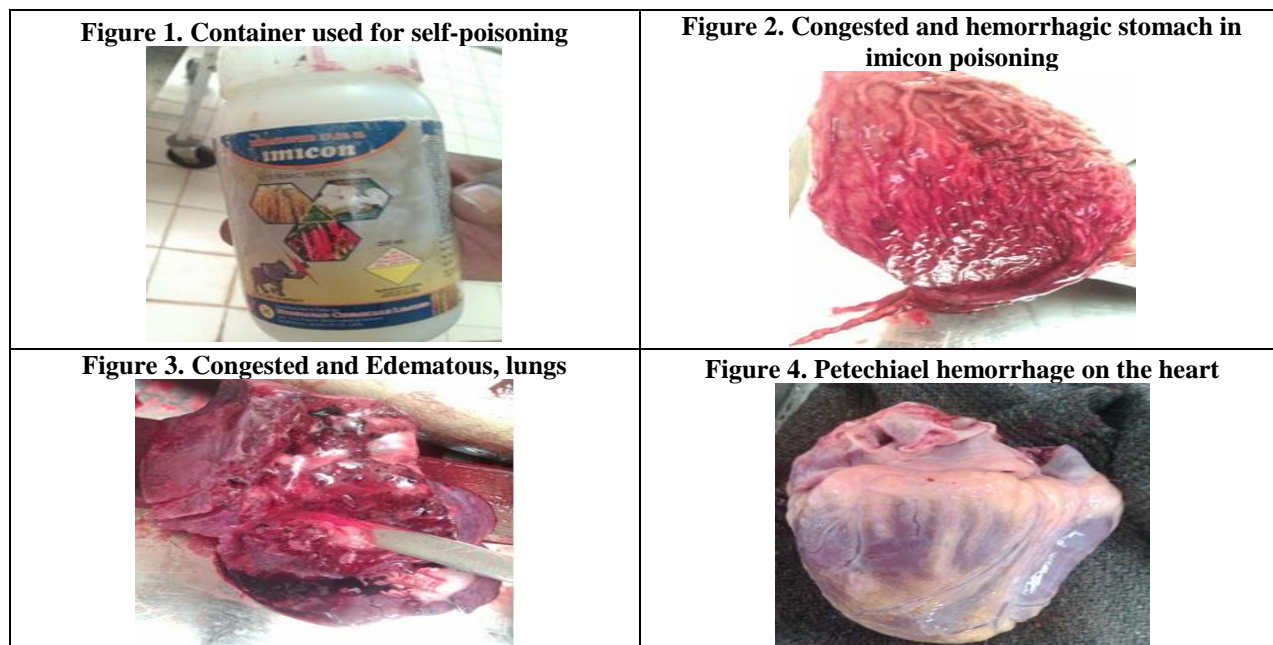
Mild clinical effects such as tachycardia, hypertension, mydriasis, nausea and vomiting occur, but more serious sequelae including respiratory failure, seizures [9,11,13,14] and even death [10,12,15] are reported. This raises serious doubts about its assumed superior safety profile over older insecticides. This group of insecticides is often misused for homicidal or suicidal purposes. We are reporting a case of self poisoning with imidacloprid poisoning leading to death.

**CASE REPORT**

A 19 year old female patient was brought to emergency department of MVJMC&RH, Hosakote, Bangalore, with alleged history of self ingestion of 250ml 70% imidacloprid. Her parents gave history of this substance used for killing the bee hives at their residence. She complained of nausea, vomiting, abdominal cramps and difficulty in breathing within 30 minutes of ingestion of poison. On arrival in emergency room she was found to be drowsy and dyspnoeic. On physical examination temperature was 98.7°F, HR 120/min, BP 150/90 mmHg, respiratory rate 50/min and oxygen saturation of 50%. There was no pallor, cyanosis or injury marks. There were scattered coarse crepitations on chest auscultation. On neurological examination she 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 mild leucocytosis with normal hemoglobin level, RBC and platelet count. Gastric lavage was done and sample was collected for toxicological analysis. After she was stabilized she

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. She received an atropine infusion at 1.2 mg/hour and prophylactic cefuroxime 750 mg 8 hourly and metronidazole 500 mg every 8 hours for suspected pulmonary aspiration. After 16 hours of ventilation the patient started developing neuropsychiatric manifestations like agitation, delirium and went into respiratory arrest, she was resuscitated with endotracheal intubation using atracurium 25 mg and midazolam 5 mg. But she did not recover after resuscitation and died. Patient was shifted to the mortuary for autopsy, on external examination no significant changes were seen except for white coloured froth oozing from nostrils and mouth, finger nail beds were discoloured bluish, ecchymosis of varying size and shape were present on both upper limbs and over the loin region on left side. On internal examination, both the lungs were congested, on cut section blood mixed with froth oozed out. Petechial hemorrhages were seen on the surface of the heart. Stomach contained 50ml of dark reddish brown coloured fluid, mucosa congested and haemorrhagic, with no peculiar smell. Other organs were congested. Organs were collected and sent for chemical analysis, and presence of Imidacloprid (neonicotinamide) was confirmed by the chemical examiners report in the viscera sent.



## DISCUSSION AND CONCLUSION

Imidacloprid is a systemic neonicotinoid insecticide produced by the German chemical firm Bayer CropScience and sold under such trade names as Gaucho, Admire, Merit, Advantage, Confidor, Provado, and Winner. Imidacloprid was first registered in the United Kingdom in 1993 and in the United States and France in 1994. Imidacloprid has been suspected of being associated with honeybee colony collapse disorder in man made hives. It has not however been directly linked to this disorder and research in recent years now points at other causes such as miticide sprays used by bee keepers to control mite parasites that are directly sprayed on the hive, a virus, and a fly known to parasitize American bumble bees and wasps has recently been discovered parasitizing California honey bees, causing bees to abandon their colonies in the night. 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) [4,5]. It is not banned, restricted, canceled, or illegal to import in any country [16]. A few cases of significant human toxicity due to imidacloprid have been reported in medical literature. Persons who might orally ingest acute amount show emesis, diaphoresis, drowsiness and disorientation.

This would need to be intentional since a large amount would need to be ingested to experience a toxic reaction. Imidacloprid is quickly and almost completely absorbed from the gastrointestinal tract, and eliminated via urine and feces (70-80% and 20-30%, respectively, of the 96% of the parent compound administered within 48 hours). The most important metabolic steps include the degradation to 6-chloronicotinic acid, a compound that acts on the nervous system as described above. Blood imidacloprid concentrations may be measured to confirm diagnosis in hospitalized patients or to establish the cause of death in postmortem investigations.

Our patient during the clinical course of toxicity developed gastrointestinal irritation, respiratory failure and severe neuropsychiatric

symptoms. With symptomatic and supportive care patient could not be resuscitated and died eventually. Till date neuropsychiatric symptoms in imidacloprid poisoning has been reported in one case with inhalation exposure [9], mainly due to central nicotinic stimulation. Tachycardia and hypertension have usually been reported in previous cases, and recurrent ventricular fibrillation was the reported cause of death in a 69 year-old woman with coronary artery disease [12]. There are no specific antidotes for neonicotinoid poisoning in mammals [17]. Treatment with oximes such as pralidoxime is expected to be either ineffective or contraindicated. Oximes in the absence of organophosphorus pesticides have a weak inhibitory effect on acetylcholinesterase activity and therefore might increase nicotinic effects (tachycardia, hypertension, muscle weakness). This study demonstrates that an acute ingestion of 70% SL formulations of imidacloprid, even following large ingestions in patients with self-poisoning, is relatively unsafe. Therefore, it may be not be advantageous to promote the use of imidacloprid or similar pesticides in areas where the incidence of self-poisoning is high. This will require careful consideration by independent regulatory authorities. Imidacloprid pesticides appear to be of low toxicity to humans causing only mild symptoms such as vomiting, abdominal pain, headache and diarrhoea in the majority of cases. Large ingestions may lead to sedation and respiratory arrest.

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**CONFLICT OF INTEREST:** The authors declare that they have no conflict of interest.

## STATEMENT OF HUMAN AND ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

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