



# New Generation Insecticide Intoxication; Imidacloprid

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## Abstract

Pesticides are chemical substances used to reduce the destructive effects of living organisms such as insects, rodents, herbs and fungi that live on and around human and animal bodies and plants and that damages or decreases the nutrient level of nutrient sources during their production, storage and consumption. They may lead to fatal intoxication in their intake for suicide purposes. Imidacloprid is a new generation insecticide agent and is accepted to have a side effect potential lower than the other insecticides. In our presentation, we aimed to share the clinic evolution that appears during and after the intake of imidacloprid with suicide purpose in 3 different individuals at different places and times.

**Keywords:** Insecticide; Intoxication; Imidacloprid

## Introduction

Insecticides include herbicides and rodenticides. Imidacloprid is new generation insecticide from the chloronicotinil nitroguanidine class with very low toxicity level for humans [1]. It is rapidly metabolized upon the intake and is released at 70% to 80% by urine and 20% to 30% by feces. Imidacloprid acts as a nicotinic acetylcholine receptor agonist in central nervous system and leads to neuromuscular paralysis [2]. As imidacloprid presents a higher binding affinity to insects nervous receptors compared to mammalian receptors, human damage is quite limited in spite of their extensive use [3]. However, serious intoxications including rhabdomyolysis, neuropsychiatric symptoms, ventricular fibrillation and death [4,5]. In our presentation, we aimed to discuss the clinic levels of 3 cases who attempted suicide using imidacloprid at different places and times.

## Case Series

### Case 1

It has been determined that the 48 year-old male patient who has been taken to our emergency department drank half glass [approximately 50 cc] of agricultural product containing imidacloprid (comprador) 4 h ago. The patient has no other complaint except from nausea; his general situation was good, oriented-cooperated, and he did not present any neuromuscular deficit. The measurements were as follows: arterial blood pressure 159/93 mmHg, pulse; 94/min, respiratory rate; 22/min, body temperature; 36.3°C. In arterial blood gas, the values were pH: 7.35, pCO<sub>2</sub>: 36.7 mmHg, pO<sub>2</sub>: 83 mmHg, SaO<sub>2</sub>: 99%, HCO<sub>3</sub>: 20.3 mEq/L. A treatment with 0.9% NaCl 150 cc/h and ranitidine hydrochloride (ulcuran) ampoule 2x1 intravenous has been started for the patient with normal laboratory results. The patient who did not develop any pathology and who presented stable vital observations has been discharged at the end of the 4<sup>th</sup> day.

### Case 2

A 67 year-old male patient who was suffering from psychiatric diseases for two years has been taken to another health care institute for stomach lavage and active coal treatment after the intake of an agricultural product for suicide and then transferred to our emergency department. His general situation was good, his conscience was open and he presented stomachache and nausea complaints. It has been determined that he drank approximately 100 cc of a substance containing imidacloprid (comprador) used against potatoes harms 2 h ago. In his examination, no pathological observation has been determined. The measurements were as follows: Blood pressure: 100/63 mmHg, pulse: 102/min, blood sugar: 160 mg/dl, respiratory rate: 25/min, body temperature 36.9°C. Hemogram and biochemistry results were normal. In arterial blood gas; pH: 7.40, pO<sub>2</sub>: 98 mmHg, pCO<sub>2</sub>: 42 mmHg, SaO<sub>2</sub>: 98%, HCO<sub>3</sub>: 21.1 mEq/L. A treatment with 0.9% NaCl 100 cc/h and ranitidine hydrochloride (ulcuran) ampoule 2x1 intravenous has been started for the patient who will be subject to symptomatic follow-up. The agitations that developed at the 8<sup>th</sup> h of the critical care unit have been taken under control using a single dose of midazolam 2 mg (dormicum) intravenous.

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At the 4<sup>th</sup> day of the follow-up and treatment, the patient has been discharged because his general situation and his laboratory results were stable.

### Case 3

A 42 year-old male patient who took an agricultural product for suicide purpose has been transferred to us for the necessity of a follow-up in a high level health care institute. It has been determined that the patient took the agricultural product Imidacloprid (comprador<sup>®</sup>) at an unknown time and dose. It has been established that our patient was living alone and that he has been found by his relatives in an unconscious way. He was subjected to stomach lavage and active coal treatment after the removal of his clothes in the health care institute. Moreover, it has been determined that 1 gm pralidoxin HCl and a total of 5 mg of atropine sulphate at different interval have been administrated for organophosphate poisoning risk and liquid has been performed. With the increase of the disturbance at the 6<sup>th</sup> h of the follow-up and treatment, the patient has been transferred to our hospital upon development of confusion. In the evaluation performed in our emergency department, the general situation was bad and the patient has been determined to be non-cooperated-non-oriented. The Glasgow Coma Scale (GCS): 11 (E3V3M5), he had no pathologic reflexes. No pathology except from the sensibility determined in abdomen palpation has been observed. Vital observations were as follows; blood pressure: 158/78 mmHg, pulse: 73/min, respiratory rate: 26/min, body temperature: 36°C, oxygen saturation: 93%. The electrocardiography was at normal sinus rhythm. 100% oxygen has been administrated at 6 L/min upon monitorization. The patient who has been hospitalized in emergency intensive care unit has been submitted to a treatment with 0.9% NaCl 150 cc/h and ranitidine hydrochloride (ulcuran<sup>®</sup>) ampoule 2x1 intravenous has been started for the patient to be subject to symptomatic follow-up and treatment. In arterial blood gas, the values were; pH: 7.32, pO<sub>2</sub>: 76 mmHg, pCO<sub>2</sub>: 18 mmHg, HCO<sub>3</sub><sup>-</sup>: 15.4 mEq/L. In total blood count, the results were as follows; hemoglobin: 16.2 g/dL, leucocytes: 25.900/mm<sup>3</sup>, hematocrit: 45.8%, thrombocyte: 301.000 /mm<sup>3</sup>. In biochemistry analysis, the results were: glucose: 128 mg/dL, urea: 30 mg/dL, creatinine: 1.95 mg/dL, chlorine: 101 mmol/L, potassium: 3.6 mmol/L. At the biochemical control at the 6<sup>th</sup> h of the follow-up, urea was 36 mg/dL, creatinine was 2.02 mg/dL. The patient whose conscience closed at the 8<sup>th</sup> h of hospitalization (GCS: 6), the results were as follows; blood pressure: 76/54 mmHg, respiratory rate: 10/min, pulse: 143/min. The patient has been intubated and mechanical ventilator has been installed. At the 9<sup>th</sup> h of the hospitalization, the patient underwent a cardiopulmonary arrest, and then he did not respond to a 60 seconds cardiopulmonary resuscitation and thus has been considered as exitus.

### Discussion

Imidacloprid shows its effects on the nicotinic acetylcholine receptors of the central nervous system. It has an agonist effect on the nicotinic acetylcholine receptors of insects that paralyzes them and then leads to their death. With the agonistic effect, first it stimulates the receptors and the excessive stimulation of the receptors prevents the transmission of neuronal stimuli [6]. In humans, the toxic effect may lead to vertigo, somnolence, orientation disorder, coma, tachycardia, increase in blood pressure, coronary spasm, cardiac ischemia, arrhythmia, hypotension, bradycardia, muscular cramps, neuropsychiatric symptoms, gastro esophageal erosion and ulcer, hemorrhagic gastritis, productive cough, fever,

leucocytosis, hyperglycemia, glycosuria and micro-hematuria [7]. In a study of Mohamed F et al. performed on 68 patients, most of the cases presented slight gastrointestinal symptoms and mechanic ventilation was required for one patient [8]. In our first two cases, patients presented nausea and stomachache, while the 3<sup>rd</sup> case applied with loss of consciousness and required mechanic ventilation. While the first two cases applied within 4 h, the time of substance intake of the third case was unknown. It has been proposed that toxic effects leading to respiratory insufficiency developed in case of a long exposure time.

The general approach is to remove the substance from the patient and the patient from the substance after the exposure to the toxic substance. First, the clothes of the patient shall be taken off. The external toxins are removed from the body of the patient with a bath, while orogastric lavage allows the removal of the corporal toxins. The severity of the intoxication is not proportional to the neo-nicotinoid concentration of plasma. Thus, there is no way of hemi perfusion for the elimination of neo-nicotinoid from the body [9]. Supportive treatment management is sufficient for all the patients intoxicated with neo-nicotinoid. After acute intoxication, the treatment is symptomatic and supportive as decontaminative, protective for respiratory ways, blockers of H<sub>2</sub> receptors, oxygen and liquids and does not present any antidote [2]. Differently from organophosphates, oximes are not efficient in the treatment. In absence of organophosphorous pesticides, oximes have a slight inhibitor effect on the action of acetyl cholinesterase, and thus may increase their nicotinic effects [tachycardia, hypertension, muscle weakness] [8]. In our case, in spite of the application of oximes, no response has been obtained. Especially the absence of information about the time period after the intake and the absence of a known antidote have increased the toxic effects of the drug.

The definitive mechanisms of imidacloprid intoxication are not known [7] and some respiratory, cardiovascular and neurological observations about the patient (dyspnea/apnea, coma, tachycardia, hypotension, midriasis and bradycardia) constitute indications for severe imidacloprid intoxication. In two important studies, the rate of death associated with imidacloprid intoxication was quite lower by 0% to 2.9% compared to the other insect killing insecticides [8,9]. In our two cases, there was a slight version of it and our patients have been discharged upon follow-up. In imidacloprid intoxication, death occurs within 4 h to 24 h after the lethal dose. In a case that ended with death, the patient died within few days with evolutive renal dysfunction without any renal replacement treatment [2]. In our case that ended with death, we observed that the renal functions were impaired but hemodialysis has not been performed due to the dramatic impairment of the clinic situation of the patient. At the 9<sup>th</sup> h of his hospitalization, the blood pressure of the patient dropped off, his heart rate increased, the urine output decreased, then cardiopulmonary arrest occurred and the patient died not responding to cardiopulmonary resuscitation procedure.

### Result

There is no specific symptom in intoxication with imidacloprid which is a new generation insecticide, a large choice of clinic observations from slight gastrointestinal symptoms, serious neurologic and cardiac observations and also death. The treatment is supportive and symptomatic and oximes are not necessary. In last year's, with the recommendations of safe agriculture products, the increase of cases of intoxication experienced with the increase of

utilization of insecticides of imidacloprid group. In cases coming to the hospital for suspicion of organophosphate intoxication, in spite of the low toxicity, it is possible to observe death and we wanted to draw attention to imidacloprid intoxication due to the difference of the treatment approach.

## References

1. Viradiya K, Mishra A. Imidacloprid poisoning. *J Assoc Physicians India*. 2011;59:594-5.
2. Shadnia S, Moghaddam HH. Fatal intoxication with imidacloprid insecticide. *Am J Emerg Med*. 2008;26(5): 634 e1-4.
3. Iyyadurai R, George IA, Peter JV. Imidacloprid poisoning-newer insecticide and fatal toxicity. *J Med Toxicol*. 2010;6(1):77-8.
4. Yeh IJ, Lin TJ, Hwang DY. Acute multiple organ failure with imidacloprid and alcohol ingestion. *Am J Emerg Med*. 2010;28(2):255.e1-3.
5. Agarwal R, Srinivas R. Severe neuropsychiatric manifestations and rhabdomyolysis in a patient with imidacloprid poisoning. *Am J Emerg Med*. 2007;25(7):844-5.
6. Matsuda K, Shimomura M, Kondo Y, Ihara M, Hashigami K, Yoshida N, et al. Role of loop D of the alpha7 nicotinic acetylcholine receptor in its interaction with the insecticide imidacloprid and related neonicotinoids. *Br J Pharmacol*. 2000;130:981-6.
7. 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(4):282-6.
8. 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(4):e5127.
9. Phua DH, Lin CC, Wu ML, Deng JF, Yang CC. Neonicotinoid insecticides: An emerging cause of acute pesticide poisoning. *Clin Toxicol*. 2009;47:336-41.