Fipronil Poisoning Presenting as Sinus Bradycardia - A Rare Case Report

Abhijit Wadekar[1], Sreekarthik Pratapa[2], Maharshi Patel[3], Shilpa Gaidhane[4], Nazli Khatib[5],

[1, 2, 3, 4] Department of Medicine, Acharya Vinoba Bhave Rural Hospital, Sawangi, Wardha, Maharashtra, India.
[5] Department of Physiology, Acharya Vinoba Bhave Rural Hospital, Sawangi, Wardha, Maharashtra, India.

INTRODUCTION

Fipronil is an N-phenylpyrazole insecticide, a second-generation insecticide which is relatively new and now commonly used in cotton growing community of rural Central India. Farmer suicide and deliberate self-poisoning is menace to the Vidarbha region of rural Central India. There is paucity of research published on fipronil poisoning, clinical features, complications and treatment data. It is scarcely documented worldwide.

Agricultural insecticides are common household items in rural areas of developing countries. Because of their easy availability, insecticides became a major source of deliberate self-poisoning. As per World Health Organization (WHO), around 3 million poisoning cases with around 0.2 million deaths are noted annually in the world.[1] About 99 % of these deaths occur in developing countries. Insecticide poisoning is an important public issue in India. Around 168,000 deaths occurred from pesticide self-poisoning which totals to almost 19.7 % of the global suicides.[2] The most common cause of self-poisoning in Central India is ingestion of organophosphorus compounds (OPC). Poisoning with organophosphorus compound insecticides has high mortality rate.[1]

In the last 20 years, there is development of a new class of insecticides in view of mortality due to accidental exposures of organophosphorus and organochlorine compounds. Fipronil being one of these chemicals, is considered less harmful to humans. According to WHO classification fipronil is class II moderately hazardous pesticide.[3] Fipronil, an N-phenylpyrazole with a trifluoromethyl sulfinyl substituent, one of the first second generation insecticides, acts at the γ-aminobutyric acid (GABA) receptor and blocks the chloride channel. Fipronil is often used to control pests, fleas and ticks on pets. There is, however, very less evidence or research on human beings on their toxicity; thus, post marketing surveillance is important to detect any undesirable health issues associated with these chemicals. Until now only 3 case reports are published regarding fipronil poisoning, two cases form India and others from Sri Lanka. Hence, we are reporting a case of acute fipronil poisoning which presented as acute gastritis and sinus bradycardia.

Corresponding Author:
Dr. Shilpa Gaidhane,
Department of Medicine,
Acharya Vinoba Bhave Rural Hospital,
Sawangi, Wardha, Maharashtra, India.
E-mail: dr.shilpagaidhane@gmail.com

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PRESENTATION OF CASE

A 30-year-old female patient, homemaker was brought by her sister to our emergency department with an alleged history of consumption of insecticide (fipronil 5 %) of around one and half glass (nearly 250 ml). Post consumption, patient had two episodes of vomiting which were foul smelling liquid, non-bilious, yellow in colour and containing food particles. She has consumed the insecticide due to dispute with her husband. Patient did not report any other complaints. On examination she was conscious and oriented to time place and person. Her heart rate was - 56 / min, regular, with respiratory rate of 18 / min and blood pressure of 120 / 70 mm of Hg. Pupils were normal in size, reacting to light, on per abdominal examination, she had epigastric tenderness. On examination of central nervous system, higher function, cranial nerve function and motor nerve function were found to be normal. Her blood investigations revealed haemoglobin 9.8 g / dl, leucocyte count 9100 / cumm, platelets were 299,000 / cumm, peripheral blood smear was normal. Her blood urea was 19 mg / dl, serum creatinine was 0.7 mg / dl, serum bilirubin 0.9 mg / dl, ALT:14 U / L, AST:28 U / L coagulation parameters were normal, urine examination showed: urine albumin-nil, urine sugar-nil, epithelial cells-1-2 cells / hpf. Blood fipronil level was in view of bradycardia, her thyroid profile and echocardiography were done and found to be normal. Patient was monitored daily for seizures, hepatotoxicity coagulation profile and kidney function test. All were found to be normal throughout the stay of five days. Her heart rate increased gradually without any medications. Patient psychiatric counseling and couple counseling was done.

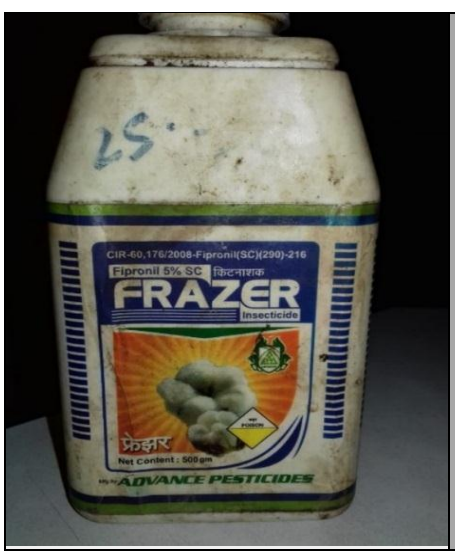

Figure 1. Container of Fipronil 5% Insecticide Presented by Patient

DISCUSSION

Epidemiology

So far only three research articles have been published regarding fipronil poisoning. With increasing use of second-generation insecticide in the cotton growing community of rural Central India, there is need to document the various presenting features of fipronil poisoning, treatment and outcome of fipronil poisoning. Here we are presenting an case of fipronil poisoning which presented as acute gastritis with sinus bradycardia.

Fipronil (CAS 120068-37-3, MW 437.16) is a relatively recent trifluoromethyl sulfonyl-mounted insecticide. Fipronil is used in liquid termiticides, as well as in agriculture, in granular turf products, seed treatments, topical pet care products.

It is used on crops to combat locusts, grasshoppers, fleas, ticks, cockroaches, ants, termites, weevils, and other insects. Cases of fipronil poisoning are not very common and very few cases are reported so far worldwide. Fipronil being newer compound, more research and reports are necessary.[3]

Mechanism of Action

Fipronil acts on Gaba channels restricting chloride ions to pass through these GABA-gated chloride channels, which results in hyperexcitability. Fipronil is toxic to insect by ingestion or contact. It inhibits Gaba aminobutyric acid gated chloride channels and glutamate-gated channels which causes excess neuronal activation thus, causing significant neurotoxicity. Fipronil shows differential affinity to receptors, affinity to insect receptors being higher than the mammalian receptors.[4,5] Fipronil sulfone, metabolite of fipronil has six times potent action in blocking vertebrate GABA gated channels. Active metabolites of fipronil are fipronil sulfide, disulfinyl, and sulfone. Another mechanism of action of fipronil involves metabolic enzymatic systems which usually has sulfhydryl groups. Fipronil disrupts these systems resulting in uncoupling of oxidative phosphorylation in mitochondria which subsequently causes ischemia, resulting in hypoxia in vital organs leading to death [6,7]

Clinical Features of Fipronil Poisoning

Post exposure presentation of fipronil poisoning in humans vary in terms of symptoms, which include sweating, gastritis symptoms like nausea and vomiting, pain in abdomen, neurological symptoms like dizziness, agitation, headache, and seizures which are mostly tonic clonic.[3,8] Clinical features of exposure to fipronil are usually reversible and they tend to resolve spontaneously. Our case had mild symptoms like nausea, vomiting and dizziness which was resolved after symptomatic management, there were no symptoms after 2 days of treatment. According to literature available regarding fipronil poisoning, most patients showed mild clinical symptoms which were temporary and comprised most commonly neurological symptoms like paraesthesia, dizziness and headache, other less commonly involved systems were gastrointestinal, respiratory and ocular and renal.[9]

Treatment and Outcome

Our study indicates that fipronil poisoning can present as mild and temporary undesirable effects on human body systems. Signs and symptoms of fipronil poisoning are generally reversible and resolve spontaneously with supportive treatment.

Few cases in the literature which were presented with seizure, were managed with benzodiazepines as standard practice first line management, along with supportive management of airway protection, oxygen supplementation.

This was followed by phenobarbital infusion in case of status epilepticus. Gastric lavage as of gastric emptying procedure is of no value as fipronil has low toxicity and gastric lavage showed poor efficacy.[12]

Our patient did not develop neurological symptoms, hepatotoxicity and acute kidney injury, this may be due to the fact that patient has consumed less amount of insecticide. And was brought to emergency department promptly.

Author	Presentation	Treatment	Outcome
Present study	Acute gastritis with sinus bradycardia	Gastric lavage supportive management	Complete recovery
Gutta S, et al.[10]	Acute neurotoxicity and delayed hepatotoxicity	Antiepileptics (phenytoin, levetiracetam, clobazam) atypical antipsychotics (quetiapine)	Complete recovery by third week
Yadla M, et al.[11]	Status epilepticus anuria	Haemodialysis, conservative measures.	AKI partially reversed. serum creatinine
Mohamed F, et al.[3]	Case-1. case 3, case 4, case 5, case 7: asymptomatic presentation	Supportive management	Complete recovery
	Case-2. Drowsy, sweating profusely, vomiting GTCS lasting for 1 minute.	Diazepam 10 mg IV,	Complete recovery
	Case-6. Acute gastritis	Forced emesis	Complete recovery
	Case-8. Status epilepticus	Intubation, benzodiazepines, phenobarbital	Pneumonia and died without regaining consciousness

Table 1. Documented Presentations of Fipronil Poisoning

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