

# Cardiac Conduction Disturbance Due To Prallethrin (Pyrethroid) Poisoning

Emmanuel M. Bhaskar · Swathy Moorthy ·  
Gaurav Ganeshwala · Georgi Abraham

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**Abstract** Pyrethroids are common household insecticides. Even though they are less toxic to humans, reports of accidental and suicidal poisoning are not uncommon. Cardiotoxicity due to pyrethroid poisoning is rare. We report a case of cardiac conduction disturbance due to a pyrethroid, prallethrin. A 28-year-old female presented after a suicidal consumption of prallethrin. Her clinical and laboratory parameters were normal during the first 24 h of hospital stay. On the second hospital day, she developed metabolic acidosis and sinus arrest with escape junctional rhythm. Despite correction of metabolic acidosis, the sinus arrest persisted for 3 days. She reverted back to sinus rhythm with bradycardia after this period and was discharged on the seventh hospital day. Her follow-up was uneventful. Pyrethroid poisoning can affect the gastrointestinal, respiratory, and nervous system. Most serious effects of the toxin in humans are seizures and coma. Mechanism of pyrethroid neurotoxicity is believed to be due to its ability to modify sodium, chloride, and calcium channels of the neurons. Our case raises the possibility that cardiac arrhythmia due to pyrethroid poisoning can occur due to its effect on sodium channels in the heart.

**Keywords** Pyrethroid poisoning · Prallethrin poisoning · Metabolic acidosis · Sinus arrest

## Introduction

Prallethrin (synthetic pyrethroid) is an insecticide, which is quite often used against household pests like mosquitoes, houseflies, and cockroaches [1]. Allethrin, was the first pyrethroid pesticide which was identified as early as 1949 [2]. Pyrethroid insecticides are classified as type-I (having a cyclopropane structure) and type-II (having a cyano group) and are about 2,250 times more toxic to insects compared to mammals [2, 3]. The class of pyrethroids includes about 42 compounds with varying chemical structure [3]. Humans and other mammals rapidly metabolize pyrethroid compounds to non-toxic substances [3], making them a preferred and approved insecticide for household use.

However, reports of accidental and intentional poisoning due to pyrethroid compounds are not uncommon [4, 5]. Pyrethroids are known for their neurotoxicity in vertebrates and humans [3]. It is unclear if certain clinical features are unique to poisoning due to a particular member of the pyrethroid family. We report a case of suicidal poisoning due to prallethrin (pyrethroid) with cardiac conduction disturbance but with no neurological toxicity.

## Case Report

A 28-year-old female presented to our emergency department 3 h after suicidal consumption of 20 ml of prallethrin, a pyrethroid insecticide. The formulation contained prallethrin (1.6%) and piperonyl butoxide (5%). History was notable for multiple family conflicts over the preceding month. There was no history suggestive of other drug or toxin co-ingestion. On initial questioning, she complained of mild throat pain and epigastric discomfort. On examination, her vital signs were stable, oral cavity was normal, and systems were unremarkable. Her blood gases and electro-

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E. M. Bhaskar (✉) · S. Moorthy · G. Ganeshwala · G. Abraham  
Department of Medicine,  
Sri Ramachandra Medical College and Research Institute,  
Porur, Chennai, India PIN-600116  
e-mail: drmebchennai@rediffmail.com

lytes were normal. She received a gastric lavage and was transferred to our intensive care unit. She was closely monitored with frequent clinical examination, continuous cardiac monitoring, and periodic blood gas analysis. Her condition was stable during the first 24 h, beyond which she developed acute metabolic acidosis (blood gases showed a pH of 7.21 and a bicarbonate of 15 meq/L). Corresponding serum electrolytes (Na-132 meq/L, K-4.2 meq/L, Hco<sub>3</sub>-17 meq/L, and Cl-100 meq/L) showed a high-normal anion-gap of 15 meq/L. Serum calcium was 9.6 mg/dL, phosphorus 3.2 mg/dL, and magnesium 2.2 mg/dL. Her serum albumin was 4 g/dL. Concomitantly, she developed intermittent episodes of sinus arrest with escape junctional rhythm (Fig. 1). The cardiac conduction disturbance was initially attributed to the metabolic acidosis. Infusion of normal saline was stopped to prevent worsening of metabolic acidosis. But her metabolic acidosis continued to deteriorate. The sinus arrest with escape junctional rhythm which was initially intermittent became persistent (Fig. 2). A repeat electrolyte panel (including calcium, phosphorus, and magnesium) was normal except for a serum bicarbonate of 14 meq/L. Osmolal gap was within normal range. Serial estimation of lactate levels was normal from admission.

She received 250 meq of intravenous sodium bicarbonate (7.5%) over a period of 12 h which normalized the metabolic acidosis (pH 7.34 and Hco<sub>3</sub> 22 meq/L). No further sodium bicarbonate was administered and her blood gases were normal during follow-up measurements. Despite the correction of metabolic acidosis, her sinus arrest with escape junctional rhythm persisted for 3 days. The junctional rhythm reverted back to sinus rhythm after this period but with a heart rate of 54-60/min. She was discharged on the seventh hospital day with a sinus rhythm and a heart rate of 66/min. She reviewed with us after 4 weeks when her rhythm was observed to be sinus with a

rate of 80/min. She had no neurological symptoms or signs during hospital stay or during follow-up.

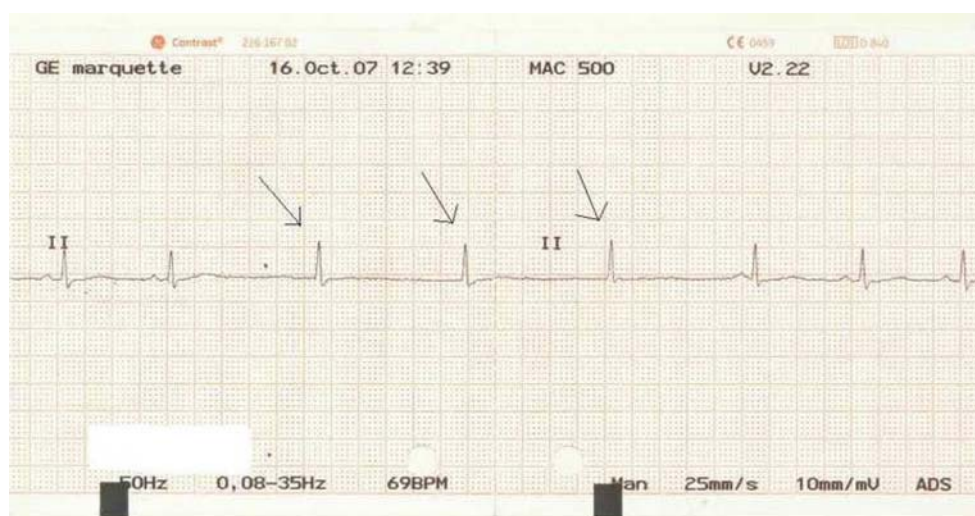
## Discussion

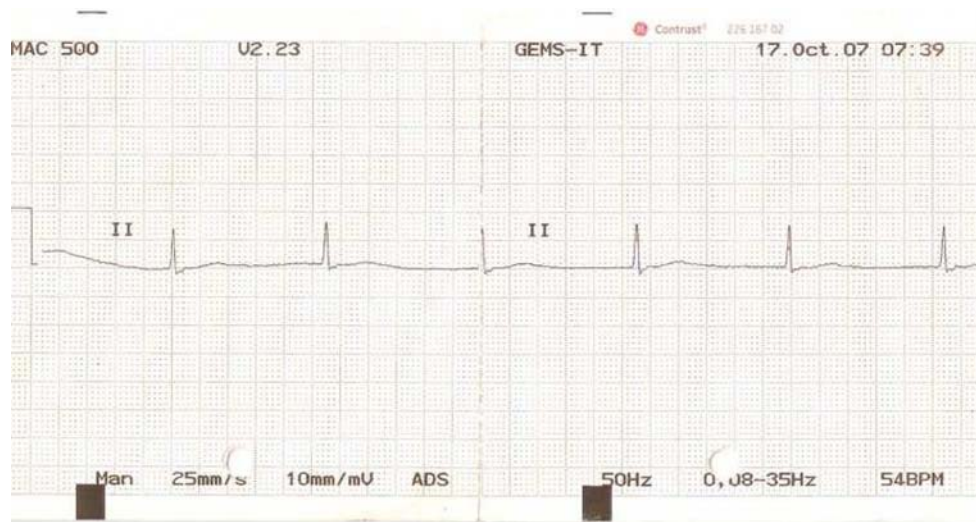
Poisoning due to pyrethroid insecticides can occur due to dermal exposure, inhalational exposure, and oral consumption [3]. Intentional pyrethroid poisoning often occurs due to oral consumption. Pyrethroid poisoning due to oral consumption is more severe than poisoning due to dermal exposure since the bioavailability of pyrethroids through gastric absorption is 36% while its bioavailability due to dermal absorption is only 1% [6].

The poisoning syndromes of pyrethroid compounds are familiarly called (1) T syndrome (due to type-I pyrethroids), characterized by severe fine tremor, marked reflex hyperexcitability, sympathetic activation, paresthesia and (2) CS syndrome (due to type-II pyrethroids), characterized by choreoathetosis, salivation, coarse tremor, increased extensor tone, moderate reflex hyperexcitability, sympathetic activation, parasthesia, and seizures[6]. The systemic manifestations of pyrethroid poisoning occur in 4-48 h and death due this toxin is very rare [2].

Yang et al. [7] analyzed the clinical features of 48 patients (38 intentional and 10 accidental) with poisoning due to insecticide formulations containing permethrin, xylene, and surfactant. In their observation, gastrointestinal symptoms and signs were most common (73%), which included sore throat, mouth ulceration, dysphagia, epigastric pain, vomiting, and malena. Central nervous system involvement was present in 33% which included confusion, seizures, and coma. Pulmonary involvement in the form of aspiration pneumonia and pulmonary edema were present in 29% of the patients. Mild renal dysfunction (10%) and hepatic dysfunction (6%) were

**Fig. 1** ECG shows transient sinus arrest with escape junctional rhythm. Arrow indicates escape junctional rhythm





**Fig. 2** ECG shows persistent junctional rhythm

also observed. Arrhythmias were observed in 4% (two cases) but the study has not explained the nature of the observed arrhythmias. Only one of the 48 patients died in their study. Since type-II compounds are more potent as insecticides [due to their cyano group], poisoning due to type-II pyrethroids are more common than type-I compounds [6].

Prallethrin is a type-I pyrethroid since it does not have a cyano group and the World Health Organization classifies it as moderately hazardous insecticide [8]. Our patient had mild gastrointestinal symptoms, metabolic acidosis, and prolonged sinus arrest with an escape junctional rhythm. The occurrence of metabolic acidosis in our patient due to infusion of normal saline (NS) is less likely since the patient received only 1.5 l of NS during the first 24 h and there was no relative hyperchloremia during this period. Moreover, alternative causes for metabolic acidosis like elevated lactate, sepsis, diarrhea, renal failure, hyperglycemic crisis, and alcohol intake were ruled out. This makes prallethrin the most likely cause of metabolic acidosis. The short duration of metabolic acidosis can be explained by the fact that pyrethroids get rapidly excreted. In a study on human volunteers, intake of different doses of a pyrethroid (cypermethrin) resulted in 75% excretion of the drug in the first 24 h irrespective of the initial dose and no detectable pyrethroid metabolites were found in their urine after 2 days [3].

The occurrence of a prolonged sinus arrest in pyrethroid poisoning is yet to be reported in the literature. In our patient, no other alternative cause for the prolonged sinus arrest like hypoxia, potassium imbalance, or drugs could be identified. The possibility of bicarbonate therapy producing sodium influx and prolonging the sinus arrest is less likely since sinus arrest is not a common complication of its administration [9]. Bicarbonate therapy is associated with

negative effects like hypervolemia, hyperosmolality, hypernatremia, hypotension, hypoxemia, impaired oxygen delivery to tissues, and hyperlactemia [9]. Decreased ionized calcium is also an adverse effect of bicarbonate therapy and is associated with ventricular depression and not with sinus node dysfunction [9]. Furthermore, beneficial effects of bicarbonate therapy on junctional bradyarrhythmia due to sodium channel-blocking drugs like citalopram has been reported [10]. We administered bicarbonate to this patient with the hope that normalization of arterial pH may achieve a sinus rhythm. The fact that the sinus arrest persisted for 72 h after correction of metabolic acidosis, makes prallethrin the probable cause of sinus node dysfunction in this patient. Pyrethroids modify gating characteristics of voltage sensitive sodium channels to delay their closure and cause protracted sodium influx [2]. This prolonged sodium influx lowers the action potential threshold and causes repetitive neuronal firing [2]. This is the proposed mechanism for paresthesia caused due to pyrethroids. While a higher concentration of pyrethroids can produce a very high influx of sodium and may prevent further action potential generation and result in conduction block in the neurons [2]. It is possible that the same mechanism can produce sinus arrest.

Spencer et al. [11], in their animal experiments, observed that tefluthrin (type I pyrethroid), fenpropathrin, and cypermethrin (type II pyrethroid) had cardiac arrhythmogenic potential while tetramethrin (type-I pyrethroid) had little effect on the heart. This indicates that cardiac arrhythmia could be a class effect, unique to certain pyrethroids.

In conclusion, our case depicts the fact that metabolic acidosis and cardiac conduction disturbance in the form of prolonged sinus arrest is possible with pyrethroid poisoning. Whether this is a class effect is not known.

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