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Seizure as a Manifestation of_Fenvalerate Poisoning in a Geriatric Patient A Case Report with Unique Electroencephalographic Findings

[1]Dr. Aby Eapen C, [2]Dr. Junu Henry, Dr Vishnu Venugopal
[1]DNB (Anaesthesia), DrNB Resident in Critical Care Medicine, [2]MDS Prosthodontics, Reader, [3]DNB Neurosurgery, MBBS MS Surgery
[1,3]Ananthapuri Hospital & Research Institute Trivandrum
[2]Sree Mookambika Institute Of Medical Sciences Padanilam, Kulasekharam, Tamil Nadu 629161

***Corresponding Author:**
Dr. Aby Eapen C
DNB (Anaesthesia), DrNB Resident in Critical Care Medicine,
Ananthapuri Hospital & Research Institute
Address: Thiruvananthapuram, Kerala, India

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Abstract

This case report details the rare occurrence of a seizure in a 64-year-old male following a deliberate ingestion of fenvalerate, a Type II pyrethroid insecticide. Fenvalerate poisoning typically presents with mild to moderate symptoms, and severe neurological sequelae, such as convulsions, are uncommonly reported in adults. The patient was initially managed with gastric decontamination and symptomatic care. A critical aspect of the case was the presence of a clinical seizure that occurred despite electroencephalogram (EEG) findings showing only a "mild degree diffuse non specific disturbance" with no definitive epileptiform abnormalities. The patient's clinical status improved significantly with supportive management, including the administration of anticonvulsants, leading to a full recovery and discharge. This case is presented to highlight the potential for severe neurotoxicity from fenvalerate and to document the unusual discrepancy between the clinical and electrophysiological findings. It provides valuable insights for clinicians in the diagnosis and management of severe pyrethroid poisoning.

Keywords: Fenvalerate, Pyrethroid Poisoning, Neurotoxicity, Seizure, Case Report, Electrophysiology

Introduction

Synthetic pyrethroids, a class of insecticides developed from natural pyrethrins, are widely used in agriculture, public health, and domestic settings due to their potent insecticidal activity and generally low mammalian toxicity.1, 2, 3 Fenvalerate, a Type II pyrethroid, is characterized by the presence of an α-cyano group, which significantly enhances its neurotoxic potency compared to Type I pyrethroids.1, 2 While pyrethroid poisoning is a common presentation in toxicology and emergency departments, particularly in regions where these pesticides are readily available, severe systemic and neurological complications are rare.1, 3, 4 The rapid metabolism and excretion of these compounds in the human body are the primary reasons for their favorable safety profile.2, 3

Common clinical features of pyrethroid exposure, especially through ingestion, include local irritation of the throat, nausea, vomiting, abdominal pain, and systemic symptoms such as paraesthesiae (a tingling sensation, often affecting the face), dizziness, headache, and fatigue.1, 2, 5 The most critical life-threatening features, although infrequent, are central nervous system (CNS) manifestations such as convulsions and coma.1, 5 The rarity of these severe neurological events, particularly seizures, in adult patients makes each documented case an important

contribution to the clinical literature. This report documents a case of fenvalerate poisoning in a geriatric patient that resulted in a seizure and highlights the subsequent management and positive clinical outcome. The case is particularly notable for the unusual finding on the patient's electroencephalogram (EEG), which did not show typical epileptiform activity despite the overt clinical seizure. This discrepancy raises important questions about the pathophysiology and diagnostic challenges of severe pyrethroid toxicity and underscores the educational value of this report for a broad clinical audience, including those in emergency medicine, toxicology, and neurology.

Case Presentation

A 64-year-old male was admitted to the hospital with a history of intentional ingestion of a bottle of fenvalerate insecticide at approximately 7:45 PM on May 26, 2025. Following the ingestion, he was initially taken to a local hospital (KTCT) where he received preliminary treatment with Inj. Pantop and was subsequently referred for higher care.

Upon arrival at the second hospital, the patient had already experienced two episodes of vomiting. His initial physical examination revealed that he was conscious but restless. No bleeding manifestations were observed, and the physical examination of the cardiovascular, respiratory, and gastrointestinal systems did not reveal any specific abnormalities (CVS- S1S2+, RS- B/L NVBS+, GIT- SOFT, NT). Despite the initial stable appearance, the patient's clinical course rapidly progressed, and he developed a seizure, which necessitated admission to the Multidisciplinary Intensive Care Unit (MDICU) for close monitoring and management.

Diagnostic investigations were performed to assess the extent of the toxic insult and the patient's overall physiological status. The patient's laboratory values are summarized in Table 1 below.

Test Name	**Result**	**Unit**	**Normal Range**
Complete Haemogram			
Haemoglobin	13.3	gm/dl	14.0–18.0
Total Count	6,450	Cells/ul	4000–11000
Neutrophil %	58	%	40–75
Lymphocyte %	32	%	20–45
Eosinophil %	06	%	1–6
Monocyte %	04	%	0–10
Basophil %	00	%	0–0
Platelet Count	1.97	lakh/ul	1.5–4.5

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Packed Cell Volume	39.7	%	40–54
ESR	17	mm/hr	<15
Biochemistry			
C-Reactive Protein (Quantitative)	29.8	mg/L	<10
Random Blood Glucose (RBGS)	117, 173	mg/dL	0–0
PT / INR			
PT	13.9	Seconds	11–16
PT Control	12.6	Seconds	
INR	0.94		0.8–1.2
Renal Function Test			
Urea	28	mg/dl	15–45
Creatinine	0.8	mg/dl	0.6–1.3
Sodium (Na)	140	mEq/L	135–145
Potassium (K)	4.2	mEq/L	3.5–5.1
Liver Function Tests			
Bilirubin Total	1.1	mg/dl	0.4–1.2
Bilirubin Direct	0.2	mg/dl	
SGOT / AST	28	U/L	5–40
SGPT / ALT	36	U/L	up to 50

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Alkaline Phosphatase	55	IU/L	5–120

A critical diagnostic finding was from the electroencephalogram (EEG) performed after the seizure episode. The report stated that the "EEG showed mild degree diffuse non specific disturbance of electrophysiological function, no epileptiform abnormalities were seen". This is a noteworthy observation given the patient's overt clinical seizure.

The patient was immediately given a Ryle's tube and administered 50 grams of activated charcoal to prevent further absorption of the toxic agent from the gastrointestinal tract. A multidisciplinary approach was adopted, with consultations sought from Neurology, Gastroenterology, Cardiology, and Nephrology departments. For the seizure episode, the patient was treated with intravenous Inj. Lorazepam and a loading dose of Inj. Levipil 500 mg twice daily. The patient also received psychiatric consultation and was prescribed Nexito and T. Lonazep upon discharge, indicating a clear psychological component to the poisoning.

The patient's clinical condition steadily improved with supportive care. His vitals stabilized, and repeat blood workup showed continued improvement. The patient was deemed clinically better and was discharged after a full recovery. The chronological sequence of events is summarized in Table 2.

Date	**Event**	**Diagnostic/Management Details**	**Patient Condition**
May 26, 2025	Ingestion of fenvalerate insecticide at 7:45 PM	Initial treatment at KTCT hospital with Inj. Pantop. Referred to AHRI.	Conscious, restless, with vomiting.
Upon Admission	Admission to hospital, two episodes of vomiting	Ryles tube inserted, 50g activated charcoal given. Critical care consultation sought.	Conscious, restless.
Post-Admission	Seizure episode	Inj. Lorazepam given. EEG performed.	Seizure.
Subsequent Days	Continued hospital course	Admitted to MICU. Administered Inj. Levipil 500 mg BD. Consultations with Neurology, Gastroenterology, Cardiology, and Nephrology.	Vitals stable. Clinically better.

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May 29, 2025	Continued monitoring	Blood workup done.	Patient becoming clinically better, vital signs stable, and blood workup improving.
Date of Discharge	Discharge	Advised Nexito and T. Lonazep.	Clinically better, vitals stable, blood workup improved. Discharged.

Discussion

Fenvalerate is a Type II pyrethroid, and its mechanism of action is primarily neurotoxic.2, 6 These compounds act on voltage-gated sodium channels in the nerve membranes, causing a persistent prolongation of the transient increase in sodium permeability during excitation.2 This results in spontaneous depolarization and repetitive discharges in the central and peripheral nervous systems, leading to hyperexcitability, muscle fasciculation, and, in severe cases, convulsions and paralysis.2 The presence of a cyano group in Type II pyrethroids makes them more potent neurotoxicants than their Type I counterparts, increasing the likelihood of these severe manifestations.2, 6

The patient's clinical presentation of a seizure is a rare but documented consequence of severe pyrethroid poisoning. A review of 196 cases of fenvalerate intoxication from Chinese medical literature noted that convulsions and coma were reported among the more severely poisoned individuals.5 Another case report described an adult male who developed status epilepticus after ingesting a pyrethroid-based insecticide, further confirming that this severe presentation, while uncommon, can occur.3 The patient's positive clinical outcome, with full recovery and discharge, is consistent with the generally favorable prognosis of pyrethroid poisoning, with most patients recovering within a week with symptomatic and supportive care.1, 4 The preservation of the patient's liver and renal function, as evidenced by the normal LFT and RFT results, supports the understanding that pyrethroids are efficiently metabolized and excreted, preventing widespread systemic organ damage.2

The most compelling aspect of this case is the incongruence between the patient's clinical seizure and the EEG findings, which reported a non-specific disturbance but no epileptiform activity. This observation is a significant contribution to the literature, as it highlights a potential diagnostic challenge in managing such cases. While some pesticide poisoning studies have reported specific EEG abnormalities, others have noted diffuse, non-specific changes.7, 8 The absence of classic epileptiform spikes on the EEG could be attributed to several factors. First, the EEG may have been performed after the acute convulsive phase had resolved or after the patient had received anticonvulsant medication (Lorazepam and Levipil), which can suppress electrical activity even if the underlying neurotoxic process is ongoing. Second, it is possible that the neurotoxic mechanism of fenvalerate, while sufficient to cause a clinical seizure, does not produce the kind of cortical hyperexcitability that manifests as epileptiform discharges on a surface EEG. This finding underscores that a normal or non-specific EEG does not rule out severe CNS involvement in pyrethroid poisoning and that clinical suspicion remains paramount.

The management strategy employed in this case, which involved prompt gastric decontamination with activated charcoal and vigorous symptomatic and supportive care, proved highly effective. There is no specific antidote for pyrethroid poisoning,9 so a targeted approach to managing the clinical symptoms is the cornerstone of treatment. The use of a multidisciplinary team, including neurologists, cardiologists, and other specialists, was essential in navigating the complexities of the case and ensuring all potential systemic complications were addressed.

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The patient's full recovery exemplifies the importance of early diagnosis and aggressive supportive care in achieving a positive outcome, even in the presence of severe neurological symptoms.

Conclusion

This case report documents a rare and educationally valuable instance of a seizure as a major manifestation of fenvalerate poisoning in a geriatric patient. While pyrethroid insecticides are generally considered to have low mammalian toxicity, severe neurotoxicity can occur, and clinicians should be aware of this potential. The case is particularly instructive due to the clinical-electrophysiological dissociation observed, where the patient's clear convulsive episode was not mirrored by definitive epileptiform activity on the EEG. This finding reinforces the notion that management should be guided by the clinical picture rather than relying solely on non-specific diagnostic tests. The successful outcome in this patient highlights the efficacy of early gastric decontamination and comprehensive symptomatic and supportive care. This report contributes to the body of knowledge on pyrethroid toxicity by detailing an unusual presentation and providing a critical learning point on the diagnostic complexities associated with severe neurotoxic events.

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