Case Report

Bull Emerg Trauma 2026;14(3):2-6.

Bulletin of Emergency And Trauma



Prompt Response of Pyrethroid-Induced Status Epilepticus to Hemoperfusion: A Case Report

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Received: April 28, 2025
Revised: August 05, 2026
Accepted: August 12, 2026

ABSTRACT

Pyrethroids can induce seizures by prolonging the opening of voltage-sensitive Na^+ channels and blocking the GABA_A receptor channels. Hemoperfusion is an extracorporeal removal method that is particularly effective for lipophilic xenobiotics. The case report describes a 48-year-old man who presented with drowsiness beginning 3 hours before admission. He had ingested Clonazepam (120 mg), LANDER^{Ò} insecticide liquid containing tetramethrin and cypermethrin; ≈400 mL), methadone (20 mg), and Aleraguard^{Ò} syrup (an herbal cough mixture; 120 mL). One hour later, his condition significantly deteriorated, presenting with hypotension, tachycardia (HR=180), generalized spasms, mydriatic pupils, and generalized tonic-clonic seizures that were unresponsive to benzodiazepine and phenobarbital. Consequently, he was endotracheally intubated and received continuous infusion of midazolam (10 mg/h), sodium thiopental (100 mg/h), and fentanyl (25 µg/h). At 26 hours, he remained irritable and experienced generalized or localized spasm upon stimulation, which was uncontrolled despite increasing the sodium thiopental dose to 150 mg/h. Therefore, he underwent a 6-hour session of hemoperfusion. Six hours after the completion of hemoperfusion, the sodium thiopental infusion was tapered off. On the 3rd day, he had markedly improved, was weaned from mechanical ventilation, and was successfully extubated. Hemoperfusion might be a life-saving treatment for severe pyrethroid poisoning.

Keywords: Pyrethrins; Hemoperfusion; Pesticide; Poisoning

Please cite this paper as:
Dorooshi GA, Arabizadeh N, Etemad L, Moshiri M, Taghavi AR. Prompt Response of Pyrethroid-Induced Status Epilepticus to Hemoperfusion: A Case Report. *Bull Emerg Trauma*. 2026;14(3):2-6. doi:10.30476/beat.2026.106669.1602.

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Introduction

Pyrethroids are a class of pesticides that bind to the voltage-sensitive Na^+ channels, inducing repetitive or "burst" discharges following a single stimulus. They are divided into two types, with type II being more potent due to the prolonged opening of Na^+ channels, which results in a more extended period of depolarization and stimulation. Moreover, the activation of voltage-sensitive Ca^{2+} channels by pyrethroids leads to neurotransmitter release [1]. Indeed, at high concentrations, pyrethroids, especially type II, block the inhibitory effect of γ-aminobutyric acid type A (GABA-A) receptors, which may contribute to seizures following severe poisoning [2].

Although pyrethroids are readily hydrolyzed into inactive compounds in the gastrointestinal tract [2], they still exhibit an oral bioavailability of 40-60% [3]. Since pyrethroids are lipophilic, they can penetrate tissue with high lipid content, such as the nervous system and adipose tissue [2]. However, only a very small proportion of orally absorbed deltamethrin is found in the brain; it is largely present in plasma and ultimately accumulates in fat, skin, and muscle. Pyrethroids have a high plasma protein-binding capacity (approximately 90%) [4]. In cases of high-dose poisoning, the proportion of protein binding decreases significantly. Pyrethroids are metabolized by the liver [3], and their inactive metabolites are excreted in the urine. They are typically eliminated via first-order kinetics within 12-24 hours. However, the elimination half-life of oral or intravenous permethrin in rats from the hippocampus, medulla oblongata, frontal cortex, and sciatic nerve was much higher than that in plasma [5].

Hemoperfusion is an extracorporeal removal method used in poisoning cases, particularly for nonpolar xenobiotics. In contrast to hemodialysis, hemoperfusion could remove compounds with high protein-bound and lipophilic properties [6]. Although there are some case reports and cross-sectional studies on the use of hemodialysis or hemoperfusion in severe pesticide poisoning [7-10], we could not find any reports of hemoperfusion in severe pyrethroid poisoning.

In this study, we reported a case of severe pyrethroid poisoning presenting with status epilepticus that showed prompt response to hemoperfusion.

Case Presentation

A 48-year-old man presented to the Clinical Toxicology Department of Khorshid Hospital, affiliated with Isfahan University of Medical Sciences, Isfahan, Iran (CTD-KH-IUMS), with drowsiness and dizziness that had begun 3 hours prior to admission. He reported ingesting clonazepam (120 mg), methadone (20 mg), LANDER® insecticide liquid spray (≈400 mL, containing tetramethrin, cypermethrin, and piperonyl butoxide), and Aleraguard® syrup (an herbal cough mixture; 120 mL). His medical history was significant for heavy smoking (two packs per day), psychiatric disorders, and a previous suicide attempt.

Upon arrival at the CTD-KH-IUMS, his vital signs were as follows: blood pressure (BP)=140/70 mmHg, temperature (T)=36.5°C, heart rate (HR)=80 beats per minute (bpm), respiratory rate (RR)=16 breaths/min, and oxygen saturation (SpO_2)=91%. His pupils were normal in size and reactive to light. Bilateral flexor plantar responses were observed, and his limb muscle strength was within normal limits. Cardiopulmonary examination was unremarkable. Bowel sounds were normoactive, with no evidence of ileus or hyperactivity. No tremors or fasciculations were observed. An electrocardiogram (ECG) revealed normal sinus rhythm.

Gastrointestinal decontamination was performed with the administration of 70 g (1 g/Kg) of activated charcoal, followed by 30 mL of magnesium hydroxide syrup. The initial laboratory test results are summarized in Table 1. Urine toxicology screening confirmed the presence of benzodiazepine and methadone. The patient underwent cardiac monitoring and received supportive therapy.

One hour after admission, the patient's condition deteriorated significantly, presenting with hypotension (BP=102/72 mmHg), tachycardia (HR= 180 BPM), generalized spasms, and mydriatic pupils, followed by a generalized tonic-clonic seizure. Consequently, after airway protection, oxygen therapy, and proper positioning, he received intravenous midazolam (10 mg) to control the seizure. Due to ongoing seizure activity, this was followed by intravenous diazepam (30 mg), and a phenobarbital infusion (1400 mg) was initiated. Following continued seizure activity and generalized spasms, he required endotracheal intubation. He was intubated using a rapid sequence intubation (RSI) protocol with atracurium (35 mg) and etomidate (25 mg), and subsequently transferred to the intensive care unit (ICU). He was treated with continuous infusions of midazolam (10 mg/h), sodium thiopental (100 mg/h), and fentanyl (25 µg/h). After intubation, the vital signs were as follows: (HR=74 bpm, RR=15 breaths/min, SpO_2=96%, and BP=105/71 mmHg). To evaluate the seizure, a portable electroencephalogram (EEG) was requested. However, it could not be conducted due to the unavailability of the facilities at that time. The patient received balanced dextrose/electrolyte solutions, and an additional 50 g of activated charcoal was administered via gavage.

After 14 hours, on the 2nd day of hospitalization, the patient's vital signs stabilized (HR=60 BPM, BP=125/80 mmHg, SPO_2=100% on mechanical ventilator, RR=16 breaths /min). As the patient exhibited irritability and generalized or localized spasms in response to stimuli (such as painful stimuli or oral/tracheal suctioning), the sodium thiopental dose was increased to 150 mg/h.

Table 1. Initial laboratory findings at admission.

Laboratory parameter	Patient value	Reference range	Unit
ALP	215	40–129	U/L
ALT	19	7–55	U/L
AST	26	8–48	U/L
Blood glucose	242	70–140	mg/dL
BUN	8	8–24	mg/dL
Hematocrit	39.2	38.3–48.6	%
Hemoglobin	13.6	13.2–16.6	g/dL
Platelet count	263×10^3	$135–317 \times 10^3$	/µL
RBC count	4.88×10^6	$4.35–5.65 \times 10^6$	/µL
RDW	13.1	11.8–14.5	%
WBC count	10.5×10^3	$3.4–9.6 \times 10^3$	/µL
CPK	70	39–308	U/L
Creatinine	75	65–119	µmol/L
LDH	329	122–222	U/L
Potassium	3.9	3.6–5.2	mmol/L
Sodium	139	135–145	mmol/L
INR	1.36	0.9–1.1	—
PT	16.9	9.4–12.5	s
aPTT	31	25–37	s
Venous blood gas			
pH	7.36	7.31–7.41	—
HCO_3^-	29.7	23–29	mmol/L
$PvCO_2$	52.7	41–51	mmHg
Base excess	3.1	−3 to +3	mmol/L

ALP; Alkaline phosphatase, ALT; Alanine aminotransferase, aPTT; activated partial thromboplastin time, AST; Aspartate aminotransferase, BUN; Blood urea nitrogen, CPK; Creatine phosphokinase; HCO_3^-; Bicarbonate, INR; International normalized ratio, LDH; Lactate dehydrogenase, PT; Prothrombin time, $PvCO_2$; Venous partial pressure of carbon dioxide, RBC; Red blood cell, RDW; Red cell distribution width, VBG; venous blood gas, WBC; white blood cell.

Subsequently, no tremors or fasciculations were observed. Laboratory tests indicated elevated serum creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) (CPK= 731 µg/L, LDH=432 U/L). A portable chest X-ray (CXR) revealed right lower lobe consolidation. Due to fever and suspected aspiration pneumonia, he was treated with ceftriaxone (1 g every 12 hours) and clindamycin (600 mg every 8 hours). Other conservative management included subcutaneous heparin (5000 units twice daily) and intravenous pantoprazole (40 mg once daily).

Twenty-six hours after admission, due to a suboptimal response to the high-dose sedation and to increase xenobiotic removal, the patient underwent hemoperfusion. The procedure was performed via the right femoral vein using an A320 cartridge for 6 hours. The gavage was held during hemoperfusion. Six hours after the completion of the procedure, the sodium thiopental was reduced to 50 mg/h until tapering off. No fasciculations or seizures were observed.

On the 3rd day, the patient showed marked improvement with normal vital signs (BP=116/78 mmHg, HR= 73 BPM, RR=12 breaths/min, SpO_2= 100% on mechanical ventilator). The sedative infusions were tapered off, and he exhibited no tremors, fasciculations, or seizures. He was successfully weaned from mechanical ventilation and extubated. On the 4th day, he was transferred to the general ward, and on the 5th day, he was discharged in stable condition and referred to the psychiatry department.

Discussion

We reported a case of severe pyrethroid poisoning with uncontrolled status epilepticus and tremors, requiring mechanical ventilation and high-dose sedation, which was effectively treated by hemoperfusion.

Pyrethroids, such as cypermethrin, deltamethrin, and permethrin, induce sodium-channel depolarization and block the GABA_A receptors at high concentrations, resulting in tremors, hyperreflexia, altered mental status, coma, and seizures [2]. Pyrethroid-induced prolonged seizures have been reported in several case studies [11-13]. Our patient also developed status epilepticus that required treatment with continuous infusions of high-dose midazolam and sodium thiopental [14].

To our knowledge, there are no previous reports on the use of hemoperfusion in pyrethroid poisoning. However, as mentioned previously, there are case reports and cross-sectional studies on hemoperfusion application in organophosphate poisoning [15, 16]. Additionally, a meta-analysis of 11 randomized controlled trials involving 811 patients demonstrated that hemoperfusion is a safe and effective treatment

protocol for organophosphate poisoning [8]. Since both classes of pesticides have a similar chemical structure, it is suggested that hemoperfusion could be as effective in severe pyrethroid poisoning as it is in organophosphate poisoning [8].

The elimination time of pyrethroids at non-toxic doses is 12-24 hours, whereas pyrethroid -induced seizure could last up to 72 hours [12, 17]. In the current case, anticonvulsant therapy was still required on the second day, prompting the use of hemoperfusion. The patient responded promptly to the procedure and was weaned from the ventilator and extubated on the 3rd day.

The patient also ingested Methadone (20 mg) and Aleraguard® syrup (120 mL). Aleraguard© syrup is a mixture of herbal medicines containing extracts of garlic, fennel (Foeniculum vulgare), Heracleum persicum, cloves (Syzygium aromaticum L.), Zataria multiflora Boiss, opium tincture, and Satureja hortensis. Although Aleraguard© syrup is prescribed for respiratory issues such as cough, almost all of its herbal compounds possess anticonvulsant properties [18-22]. Although there is a case report of an epileptic seizure induced by fennel essential oil in a 38-year-old woman with epilepsy [23], some researchers believed that fennel could not induce seizures in healthy individuals [24].

Unfortunately, we could not measure the serum levels of the xenobiotics, specifically before and after hemoperfusion. Furthermore, a portable EEG could not be performed due to the unavailability of the equipment at the time.

In conclusion, a patient with pyrethroid poisoning and persistent, refractory convulsions was successfully treated with hemoperfusion. Thus, we suggest that hemoperfusion might be a life-saving treatment for severe pyrethroid poisoning. However, as this is the first case of hemoperfusion performed for this indication, these findings require further research.

Declaration

Ethical Approval and Consent to Participate: He signed the informed consent form for publication of his medical history. The report was approved by the Ethics Committee of the Alzahra Research Center, Isfahan University of Medical Sciences, Isfahan, Iran (IR.ARI.MUI.REC.1402.262).

Consent for Publication: The data that support the findings of this study are available on request from the corresponding author.

Funding: The authors declare that no funding was received for this study.

Conflict of Interest: All authors declare no financial/commercial conflicts of interest.

Data Availability: None.

Authors' Contribution: The conception and design of the study: MM and GD; Acquisition of data, or analysis and interpretation of data: NAZ and MM; Drafting the article: NAZ, LE and AT; Revising draft critically for important intellectual content: MM, GD and AT; Treatment of patient: MM and GD; All authors have approved the final version to be submitted.

Acknowledgment: The authors would like to thank the patient for consenting to the publication of this report. We would also like to thank the staff of the general ward and the intensive care unit of CTD-KH-IUMS for their kind cooperation.

References

1. Gheshlaghi F, Dorooshi G, Samsam-Shariat S, Eizadi-Mood N, Etemad L, Pirmoradian P, et al. Low dose atropine triggered atrial fibrillation in chlorpyrifos plus cypermethrin poisoning, a rare case report and review of literature. *Journal of Iranian Medical Council*. 2024;**7**(3):588-96.
2. Holland MG. Insecticides: Organic Chlorines,Pyrethrins/Pyrethroids, And Insect Repellents. In: Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, R.S. H, editors. Goldfrank's Toxicologic Emergencies, Eleventh Edition. New York: Mc Graw Hill 2019. p. 1514-28.
3. Mallick P, Moreau M, Song G, Efremenko AY, Pendse SN, Creek MR, et al. Development and Application of a Life-Stage Physiologically Based Pharmacokinetic (PBPK) Model to the Assessment of Internal Dose of Pyrethroids in Humans. *Toxicol Sci*. 2020;**173**(1):86-99.
4. Sethi P, Bruckner JV, Mortuza TB, Cummings BS, Muralidhara S, White CA. Plasma Protein and Lipoprotein Binding of Cis- and Trans-Permethrin and Deltamethrin in Adult Humans and Rats. *Drug Metab Dispos*. 2019;**47**(9):941-8.
5. National Research Council Subcommittee to Review Permethrin Toxicity from Military U. Health Effects of Permethrin-Impregnated Army Battle-Dress Uniforms. Washington (DC): National Academies Press (US); 1994.
6. Abramson S. Chapter 51 - Extracorporeal Treatment of Poisonings. In: Himmelfarb J, Sayegh MH, editors. Chronic Kidney Disease, Dialysis, and Transplantation (Third Edition). Philadelphia: W.B. Saunders; 2010. p. 700-19.
7. Bo L. Therapeutic efficacies of different hemoperfusion frequencies in patients with organophosphate poisoning. *Eur Rev Med Pharmacol Sci*. 2014;**18**(22):3521-3.
8. Zhang M, Zhang W, Zhao S, Tian X, Fu G, Wang B. Hemoperfusion in combination with hemofiltration for acute severe organophosphorus pesticide poisoning: A systematic review and meta-analysis. *J Res Med Sci*. 2022;**27**:33.
9. Moshiri M, Darchini-Maragheh E, Balali-Mood M. Advances in toxicology and medical treatment of chemical warfare nerve agents. *Daru*. 2012;**20**(1):81.
10. Moshiri M, Mousavi SR,

Etemad L. Management of 2, 4-dichlorophenoxyacetic acid intoxication by hemodialysis: A case report. *Iranian Journal of Toxicology*. 2016;**10**(1):53-5.
11. Giampreti A, Lampati L, Chidini G, Rocchi L, Rolandi L, Lonati D, et al. Recurrent tonic–clonic seizures and coma due to ingestion of type I pyrethroids in a 19-month-old patient. *Clin Toxicol (Phila)*. 2013;**51**(6):497-500.
12. Panwar M, Usha G, Kumath M. Status epilepticus: An association with pyrethroid poisoning. *Indian J Crit Care Med*. 2013;**17**(2):119-20.
13. Rangaraju S, Webb A. Status epilepticus following inhalational exposure to bifenthrin, a Type II pyrethroid. *Clin Toxicol (Phila)*. 2013;**51**(9):906.
14. Menezes RG. The anticonvulsant of choice in pyrethroid induced convulsions. *Indian J Crit Care Med*. 2005;**18**(4):258-9.
15. Yokoyama K, Ogura Y, Kishimoto M, Hinoshita F, Hara S, Yamada A, et al. Blood purification for severe sarin poisoning after the Tokyo subway attack. *JAMA*. 1995;**274**(5):379.
16. Li Z, Wang G, Zhen G, Zhang Y, Liu J, Liu S. Application of hemoperfusion in severe acute organophosphorus pesticide poisoning. *Turk J Med Sci*. 2017;**47**(4):1277-81.
17. Park S, Kim DE, Park SY, Gil HW, Hong SY. Seizures in patients with acute pesticide intoxication, with a focus on glufosinate ammonium. *Hum Exp Toxicol*. 2018;**37**(4):331-7.
18. Zolfagharian F, Razavi BM, Hosseinzadeh H. Anticonvulsant effect of Satureja hortensis aerial parts extracts in mice. *Avicenna J Phytomed*. 2016;**6**(3):305-12.
19. Savairam VD, Patil NA, Borate SR, Ghaisas MM, Shete RV. Evaluation of antiepileptic activity of ethanolic extract of garlic containing 3.25% allicin in experimental animals. *Pharmacological Research - Modern Chinese Medicine*. 2023;**8**:100289.
20. Saeidi F, Azhdari Zarmehri H, Alimohammadi B, Erami E. The effect of hydroalcoholic extract of Heracleum persicum on pentylenetetrazol induced seizure in mice. *Journal of Zanjan University of Medical Sciences and Health Services*. 2013;**21**(86):45-55.
21. Nubed LN, Taiwe GS, Simons CR, Mbah JA. Natural products with anticonvulsant potentials from Syzygium aromaticum (Myrtaceae). *GSC Biological and Pharmaceutical Sciences*. 2023;**22**(1):001-14.
22. Shamsizadeh A, Fatehi F, Baniasad FA, Ayoobi F, Rezvani ME, Roohbakhsh A. The effect of Zataria multiflora Boiss hydroalcoholic extract and fractions in pentylenetetrazole-induced kindling in mice. *Avicenna J Phytomed*. 2016;**6**(6):597-603.
23. Skalli S, Soulaymani Bencheikh R. Epileptic seizure induced by fennel essential oil. *Epileptic Disord*. 2011;**13**(3):345-7.
24. Tisserand R, Young R. The nervous system. In: Tisserand R, Young R, editors. Essential Oil Safety: A Guide for Health Care Professionals, 2nd Edition St. Louis: Churchill Livingstone; 2014. p. 131-46.

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