

First Fatal Human Case of Beta-Cyfluthrin Intoxication in Türkiye: A Case Report

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Abstract

Cyfluthrin, a type II synthetic pyrethroid, is extensively used in agricultural and veterinary practices. While generally considered to have low mammalian toxicity, significant exposure to concentrated formulations can lead to systemic toxicity and multiorgan failure. Human fatalities due to cyfluthrin are rare and not well-characterized in the literature. We report the case of a 67-year-old female shepherd with no known comorbidities who presented to the emergency department approximately 24 hours after dermal and possible inhalational exposure to a veterinary insecticide containing 25 g/L beta-cyfluthrin in emulsifiable concentrate form. She had applied the formulation directly to livestock without protective equipment. Within hours, she developed acute retrosternal chest pain, vomiting, and transient loss of consciousness. Upon arrival to the emergency department, she was hypotensive, bradycardic, and hypoxic. Laboratory analysis revealed severe metabolic acidosis, hyperglycemia, markedly elevated hepatic and cardiac enzymes, acute kidney injury, and coagulopathy. Imaging showed a moderate pericardial effusion without tamponade. Despite intensive supportive care, including high-dose sodium bicarbonate, N-acetylcysteine, and fluid resuscitation, her condition deteriorated, culminating in disseminated intravascular coagulation, shock, and death on ICU day 8. This case illustrates the potential lethality of beta-cyfluthrin exposure in humans and underscores the importance of early recognition, protective practices, and aggressive management in suspected pyrethroid poisoning.

Keywords: Cyfluthrin, insecticides, multiorgan failure, pesticide poisoning, rural population

Introduction

Synthetic pyrethroids are a widely used class of insecticides derived from natural pyrethrins, known for their high efficacy against pests and relatively low mammalian toxicity. Among them, cyfluthrin, a type II pyrethroid containing a cyano group, is extensively applied in agricultural, veterinary, and public health settings for the control of a broad spectrum of insects (1,2). Its mechanism of action involves prolonged activation of voltage-gated sodium channels in nerve cells, resulting in sustained depolarization and repetitive neuronal firing, ultimately leading to paralysis in target species (3).

Despite their widespread perception as “safe” insecticides for humans, pyrethroids—particularly in their concentrated forms—may cause significant systemic toxicity through dermal, inhalational, or oral exposure. Human poisoning typically results in mild neurological and gastrointestinal symptoms; however, more severe presentations including seizures, pulmonary edema, shock, and multiorgan dysfunction have been reported, particularly following large or prolonged exposures to type II pyrethroids such as cyfluthrin and beta-cyfluthrin (4–6). The severity of

toxicity depends on multiple factors including formulation concentration, route and duration of exposure, and individual susceptibility.

Beta-cyfluthrin is a more potent stereoisomer-enriched variant of cyfluthrin used in many commercial emulsifiable concentrate products for livestock and crop protection. In Türkiye, these formulations are commonly used in rural settings with limited awareness of personal protective measures. Literature on fatal human exposures to cyfluthrin is sparse, with most case reports describing full recovery after supportive care (5,6). A fatal case of human beta-cyfluthrin intoxication following dermal exposure has not been previously documented in the medical literature.

We present a rare and fatal case of presumed cyfluthrin intoxication in a shepherd who developed progressive lactic acidosis, ischemic hepatitis, coagulopathy, and ultimately death following unprotected dermal and inhalational exposure to a 25 g/L beta-cyfluthrin emulsifiable concentrate preparation. This report highlights the potential lethality of improperly handled pyrethroid insecticides and underscores the importance of early recognition and aggressive supportive care in similar presentations.

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Case Report

A 67-year-old female shepherd with no known medical history was brought to the emergency department (ED) by ambulance after collapsing in a remote pasture area. According to family members, approximately 24 hours earlier, the patient had used an insecticidal veterinary preparation containing 25 g/L beta-cyfluthrin in emulsifiable concentrate form to treat her goats for fleas. The product was applied without gloves or respiratory protection in an open-air setting. She reportedly complained of retrosternal chest pain, fatigue, and vomiting several hours later, and then became increasingly confused and weak over the next day.

Upon presentation to the ED, her vital signs were: blood pressure 65/45 mmHg, heart rate 65 bpm, respiratory rate 22 breaths per minute, oxygen saturation 88% on room air, and temperature 36.5°C. She was pale, diaphoretic, and mildly lethargic, but oriented to person and place. Cardiac auscultation revealed muffled heart sounds; bilateral basal crackles were heard on lung examination. Her abdomen was soft and non-tender. No obvious dermal lesions or rashes were noted, though her hands and arms had a faint chemical odor. Neurological examination showed no focal deficits; GCS was 13 (E4V4M5).

Initial laboratory evaluation revealed severe metabolic acidosis (pH: 6.95, bicarbonate: 14 mmol/L), hyperglycemia (glucose: 420 mg/dL), elevated liver enzymes (AST: 2913 U/L, ALT: 2205 U/L, LDH >2726 U/L, and cardiac injury (troponin T: 62 ng/L). Renal function was impaired (creatinine: 2.86 mg/dL; urea: 148 mg/dL), and coagulation parameters were abnormal (INR: 1.51; fibrinogen: 244 mg/dL; D-dimer: 6400 ng/mL). Electrocardiography revealed sinus bradycardia. Transthoracic echocardiography demonstrated a 2–3 cm circumferential pericardial effusion without tamponade physiology. Chest radiograph and thoracoabdominal CT revealed mild bilateral pleural effusions and pericardial fluid, with no signs of pulmonary embolism.

Due to the lack of available toxicology screening for pyrethroids at our center, the diagnosis of cyfluthrin poisoning was made clinically, based on the exposure history, symptom profile, and exclusion of alternative causes. She was admitted to the intensive care unit with a presumptive diagnosis of pyrethroid-induced lactic acidosis, ischemic hepatitis, and developing multiorgan dysfunction syndrome. Initial therapy included high-dose intravenous sodium bicarbonate infusion, N-acetylcysteine, broad-spectrum antibiotics, and aggressive fluid resuscitation. Despite supportive care, she remained hypotensive, oliguric, and showed signs of progressive hepatic injury.

By ICU day 2, she developed disseminated intravascular coagulation with thrombocytopenia (platelets: $55 \times 10^3/\mu\text{L}$), elevated D-dimer (>21,000 ng/mL), and worsening liver enzyme levels. Hemodialysis could not be initiated due to

hemodynamic instability. Multidisciplinary consultations concluded that her deterioration was consistent with a systemic inflammatory response syndrome secondary to pesticide intoxication. On day 8, the patient experienced pulseless electrical activity arrest. Cardiopulmonary resuscitation and multiple doses of epinephrine failed to achieve return of spontaneous circulation. She was declared dead after 45 minutes of advanced life support.

Discussion

Cyfluthrin is a widely used type II synthetic pyrethroid that targets voltage-gated sodium channels, resulting in repetitive neuronal firing and eventual paralysis in insects. While considered to have low mammalian toxicity due to hepatic detoxification pathways, significant dermal or inhalational exposure to concentrated formulations—such as emulsifiable concentrates (ECs)—may lead to serious systemic effects in humans (1,2).

In this case, exposure occurred during unsupervised outdoor application of a veterinary insecticide containing 25 g/L beta-cyfluthrin, a formulation commonly marketed in Türkiye under various trade names. The patient's symptomatology developed rapidly and progressed over the subsequent 24 hours, suggesting substantial percutaneous and/or inhalational absorption. Though gastrointestinal ingestion was not confirmed, nausea, vomiting, and central nervous system symptoms are consistent with systemic absorption and early toxicity (3,4).

Previous reports in the literature describe largely mild-to-moderate symptoms following cyfluthrin exposure—often paresthesia, dizziness, nausea, or tremors (1,3). However, more severe cases have been documented, including seizures, pulmonary edema, and shock, particularly after ingestion or exposure to high-concentration commercial products (5,6). Isbister et al. reported a series of concentrated pyrethroid exposures in which some patients developed metabolic acidosis and cardiovascular compromise (6). Similar to our case, these presentations required intensive care, but fatality was rare—highlighting the exceptional severity of our patient's outcome.

The development of pericardial effusion in this patient is particularly noteworthy, as it is infrequently reported in pyrethroid poisoning. Its etiology may involve toxin-induced pericardial inflammation or secondary capillary leak syndrome. The profound hepatic injury observed is consistent with ischemic hepatitis and has been attributed in animal models to mitochondrial dysfunction, oxidative stress, and direct hepatocellular apoptosis following cyfluthrin exposure (7). The metabolic acidosis (pH 6.95) was life-threatening and likely multifactorial-related to tissue hypoperfusion, lactic acidosis, and impaired clearance by the liver and kidneys.

A major limitation was the absence of confirmatory toxicological analysis, including serum or urinary cyfluthrin or metabolite levels (e.g., 3-phenoxybenzoic acid). However, this diagnostic barrier is common in rural or resource-limited centers, where diagnosis is typically clinical. Additionally, product retrieval and chemical analysis were not possible due to lack of labeling and disposal before admission. The establishment of regional toxicology reference laboratories capable of detecting pyrethroid metabolites would greatly enhance diagnostic accuracy in such rural poisonings. This case highlights the critical need for training programs on pesticide safety among rural agricultural workers and livestock handlers.

Conclusion

This case underscores that beta-cyfluthrin, while perceived as low-risk, may be fatal under certain exposure scenarios. The delayed presentation—approximately 24 hours post-exposure—likely contributed to diagnostic and therapeutic delay. Notably, no antidote exists for cyfluthrin poisoning, and management remains entirely supportive. Early recognition, organ function monitoring, and aggressive critical care are essential. Education on pesticide handling and mandatory labeling may reduce the risk of such fatalities in rural areas.

Conflict of interest: The authors declare no conflicts of interest.

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