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THE EFFECT OF PYRETHROID INGESTION ON BUTYRYLCHOLINESTERASE LEVEL

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ABSTRACT

We report a 45-year-old man who experienced dyspnoea, severe chest pain followed by decreased level of consciousness, and seizure-like symptoms requiring sedation and intubation. The level of high-sensitivity troponin was elevated. The patient was diagnosed in an outside institution with acute coronary syndrome along with infectious meningitis based on studies performed on cerebrospinal fluid (CSF). The patient was transferred to our hospital for further management. Cardiac ultrasound showed dilated cardiomyopathy with decreased ejection fraction. Significant coronary stenosis was excluded by coronary angiography and a polymerase chain reaction neuro-panel on the CSF came back negative. After exclusion of ACS and meningitis, detailed history taking showed that the patient´s office at work had been treated with an insecticide. The product used turned out to be deltamethrin, a type II pyrethroid. The butyrylcholinesterase (BChE) level was low. Studies have shown that deltamethrin produces neurotoxicity and cardiovascular complications; however, the effect of deltamethrin on the BChE level and the development of non-ischemic cardiomyopathy remains unclear.

KEYWORDS

Pyrethroid toxicity, butyrylcholinesterase level, acetylcholinesterase level, organophosphate comparison, toxicological effects

LEARNING POINTS

- Pyrethroid intoxication can lead to significant cardiotoxic effects, which necessitate prompt recognition and management.
- Although pyrethroids primarily act on sodium channels, they may indirectly affect cholinesterase levels, complicating the diagnostic process.
- Effective management of pyrethroid poisoning relies on early diagnosis, supportive care, and targeted treatment of both neurological and cardiovascular manifestations.

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INTRODUCTION

Pyrethroids are synthetic analogues of natural pyrethrins (insecticidal compounds from chrysanthemum flowers). They are widely used in agriculture, public health, and household insecticides due to their high insecticidal activity and relative safety in mammals. However, they can still cause toxicity, especially at high level of exposure. Pyrethroids are typically classified into two main types based on their chemical structure and neurotoxic effects: type I pyrethroids and type II pyrethroids. Type I pyrethroids (permethrin, resmethrin, tetramethrin, allethrin, bifenthrin) lack an alpha-cyano moiety at the alpha-position, while type II pyrethroids (cypermethrin, deltamethrin, fenvalerate, lambda-cyhalothrin) have an alpha-cyano moiety at the phenyl benzyl alcohol position. Type I pyrethroids cause the "T syndrome", manifested as restlessness, muscle twitches, hyperexcitability, fine tremors, sympathetic activation, and paraesthesia. Whereas type II pyrethroids cause the "CS syndrome", choreoathetosis (C) and salivation (S). CS syndrome is characterized by symptoms such as hyperactivity, a hunched posture, excessive salivation, tremors, seizure and lack of coordination, which may progress to serpentine or writhing movements[1]. Under typical exposure conditions, deltamethrin demonstrates low toxic impact on human subjects. Nevertheless, toxic levels of deltamethrin can have significant impact on humans. This case reports neurotoxicity, cardiotoxicity, and a reduced BChE level following accidental exposure to deltamethrin. It underscores the importance of examining how deltamethrin affects BChE levels and its potential contribution to the onset of non-ischemic cardiomyopathy.

CASE DESCRIPTION

A previously healthy 45-year-old man was referred from another facility to our hospital after being sedated and intubated with an unclear diagnosis of his acute presentation (dyspnoea, heavy chest pain, decreased level of consciousness, and seizure-like activity). Based on his clinical picture, meningitis was highly suspected and therefore lumbar puncture was performed. Further workup revealed elevated troponin suggestive of acute coronary syndrome requiring administration of dual antiplatelet therapy and prophylactic low molecular weight heparin.

Upon arrival to our emergency department, the patient was intubated and sedated, with drooling saliva noted. Extubation was performed once the patient regained consciousness and exhibited resistance to the endotracheal tube. A brain computed tomography (CT) scan ruled out ischemic and haemorrhagic etiology. Further workup *(Table 1)* revealed elevated D-dimer; hence, acute pulmonary embolism and aortic dissection were ruled out by chest CT angiography. Nonspecific electrocardiogram changes with elevated troponin raised the suspicion of ischemic cardiomyopathy. Urgent cardiac ultrasound showed dilated left ventricle with a decrease in ejection fraction (48-50%). The patient was continued on dual antiplatelet therapy, and coronary angiography was planned. Repeat cerebrospinal fluid (CSF) studies performed on the sample received from the outside institution was still suggestive of infectious meningitis *(Table 2)*. The patient was kept nil per OS and 2 l 0.9% normal saline solution was administered intravenously (IV) q24hrs (for adequate hydration), ceftriaxone 2 g IV q12hrs, vancomycin 1.25 g IV q12hrs, and acyclovir 750 mg IV q8hrs pending the result of the polymerase chain reaction (PCR) neuro-panel. Multidrug urine toxicology screening performed was positive for benzodiazepine (the patient was sedated on midazolam in the outside institution). The BChE level was low. The patient was admitted to the intensive care unit (ICU) for monitoring, further investigation, and management.

The PCR neuro-panel came back negative. Vancomycin and acyclovir were stopped, Ceftriaxone was adjusted to 2 g IV q24hrs, due to aspiration pneumonia (secondary to

On admission	Patient's results	Reference range
White blood cell count (10^3/µl)	12.13	4-10
Neutrophils (%)	91.2	40-67
D-dimer (ng/ml)	3,516	<500
Serum Creatinine (mg/dl)	1.2	0.6-1.3
CPK (IU/l)	2171	20-205
CPK-MB (ng/ml)	27.4	0-5.2
Troponin-I (ng/ml)	0.105	0-0.03
NT-pro BNP (pg/ml)	360.4	< 125
Butyrylcholinesterase (U/l)	2,401	5,900-12,220
CRP (mg/l)	134	0-5

Table 1. *Blood test results. Abbreviations: CPK, creatine phosphokinase; CPK-MB, creatine phosphokinase-myocardial band; pro BNP, N-terminal pro-B-type natriuretic peptide; CRP, C-reactive protein.*

On admission	Patient's results	Repeat results	Reference range
WBC (/µl)	106	21	
RBC (/µl)	44,000	2,000	
Neutrophils (%)	84	82	
Lymphocytes (%)	16	18	
Glucose (mg/dl)	111	46	40-80
Protein (mg/dl)	58	54	15-45
Albumin (mg/dl)	-	308	100-300
PCR: E. coli K1, H. Influenzae, Listeria monocytogenes, Mycobacterium Tuberculosis, Mumps virus, Enterovirus, CMV, EBV, HSV-1, HSV-2, HHV-6, Human Parechovirus, VZV, HHV-7, Cryptococcus neoformans, Neisseria meningitides, Streptococcus pneumoniae, Streptococcus agalactiae	Not detected		

Table 1. *CSF results.*
Abbreviations: CSF, cerebrospinal fluid; WBC, white blood cells; RBC, red blood cells; PCR, polymerase chain reaction; CMV, cytomegalovirus; EBV, Epstein-Barr virus; HSV, herpes simplex virus; HHV, human herpes virus; VZV, varicella-zoster virus.

loss of consciousness and seizure-like activity), and was administered for a total of 7 days. Brain magnetic resonance imaging performed ruled out encephalitis, and electroencephalography ruled out seizure. Coronary angiography revealed abnormal origin of right coronary artery; however, coronary artery stenosis was excluded.

Detailed history taking showed that the patient's office at work had been treated with an insecticide. The insecticide used turned out to be deltamethrin, a type II pyrethroid. Hence, Deltamethrin intoxication was confirmed based on the results of repeated serum BChE testing that were severely diminished.

The patient improved on a daily basis, was transferred to regular floor 48hours after ICU admission, and was then discharged home.

The pyrethroid intoxication was managed only through monitoring, observation, and supportive treatment.

DISCUSSION

The primary mechanism of toxicity for pyrethroids differs from that of organophosphates and carbamates. Pyrethroids act mainly on voltage-gated sodium channels. They prolong the opening of these sodium channels after activation, leading to sustained sodium influx, delayed repolarization and repetitive nerve firing. This extends depolarization and triggers excitotoxicity of the neurons[2]. Academic research indicates that such chemicals should not lead to measurable degradation of cholinesterase activity.

Cholinesterases are of two main types: acetylcholinesterase (AChE) and butyrylcholinesterase (BChE). AChE, also known as choline esterase I, RBC cholinesterase, or erythrocyte cholinesterase, is an enzyme found primarily in red blood cells, neuromuscular junctions and in the central and peripheral nervous system. AChE breaks down the neurotransmitter acetylcholine (ACh). BChE, also known as pseudocholinesterase or plasma cholinesterase, is an enzyme found primarily in the liver and also in plasma. BChE preferentially breaks down esters like butyrylcholine and propionylcholine. Organophosphate and carbamate pesticides inhibit AChE and BChE by covalently binding to serine residues in the active site of the enzyme at the synaptic cleft. This binding leads to phosphorylation of the enzyme, rendering it inactive[1]. Organophosphates are known to inhibit both AChE and BChE; Whereas, pyrethroids indirectly decrease BChE by diminishing the synthetic activity of hepatocytes. Hence, a decrease in BChE level associated with pyrethroid toxicity should be considered biomarker of liver dysfunction[3]. Pyrethroids have been shown to suppress BChE activity in animal studies. However, earlier studies did not observe a decrease in BChE level in humans, whereas this case demonstrated a significant reduction that remained consistent across serial measurements[2]. The unexpected drop-in cholinesterase activity in this patient suggests an alternative mechanism, possibly due to:

1. *Indirect enzyme modulation:* Scientific evidence reveals that while pyrethroids do not prevent cholinesterase inhibition directly, they produce oxidative damage that affects cholinesterase activity through autonomic dysfunction. The high oxidative stress might disrupt enzyme synthesis and boost enzyme breakdown leading to lower cholinesterase levels[4].
2. *Delayed cholinergic dysregulation:* High-level pyrethroid exposure has been linked to disruptions in cholinergic neurotransmission. Deltamethrin, in particular, interferes with calcium homeostasis, which may secondarily affect cholinesterase expression or function[5].

3. *Liver dysfunction:* Through pyrethroid induced genotoxicity (direct DNA damage) or Indirect DNA damage by generating ROS and initiating apoptotic signals that diminish the synthetic activity of hepatocytes[3].

Hence organophosphate intoxication lowers both AChE and BChE; whereas pyrethroids lower only BChE. As the primary mechanism of toxicity for pyrethroids differs from that of organophosphates and carbamates, there is no role in administering pralidoxime and toxic exposure to pyrethroids is managed only through monitoring, observation, and supportive treatment.

The development of non-ischemic cardiomyopathy warrants special attention, especially given the patient's lack of prior cardiac history. Although cardiomyopathy was confirmed by cardiac ultrasound, coronary angiography showed no evidence of obstructive coronary artery disease.

The research demonstrates that type II pyrethroid compounds including deltamethrin might cause cardiac dysfunction by several different processes[6]. The potential mechanisms for deltamethrin-induced cardiomyopathy include:

1. *Autonomic dysregulation:* Pyrethroids autonomic heart control function results in bradycardia, hypotension, and cardiac dysfunction. The impairment of the autonomic nervous system in patients may also account for other symptoms of toxicity such as persistent salivation, altered mental state, and seizure activity, as observed during this patient's initial presentation.
2. *Oxidative stress and arrhythmogenic effects:* Deltamethrin exposure in rats resulted in increased deoxyribonucleic acid damage, decreased cardiac myocyte membrane fluidity, increased cholesterol content, protein and lipid oxidation in cardiac myocytes, and elevated levels of inflammatory cytokines. An in vitro study in fish suggested that deltamethrin is associated with arrhythmogenic effects on the fish heart, with irregularities in rate and rhythm of atrial beating and strong reductions in the force of atrial contraction, and that these effects may be caused by alterations in sodium ion channel function[6].
3. *Sodium and calcium channel disruption:* Research shows that deltamethrin disrupts calcium regulation in cardiac myocytes which might lead to ventricular arrhythmias and impaired contractions[7]. The abnormalities in intracellular calcium regulation provide a potential explanation for the detected ventricular enlargement together with hypokinesia.
4. *Takotsubo cardiomyopathy:* Although the pathogenesis is not well understood, a number of features suggest that it may be caused by diffuse catecholamine-induced microvascular spasm or dysfunction, resulting in myocardial stunning[8].

Past case reports have documented bradycardia and cardiac arrhythmias after pyrethroid exposure yet dilated cardiomyopathy is as a very uncommon and unclear consequence of such poisoning[6]. This case suggests that acute deltamethrin poisoning could be a potential cause of non-ischemic rapid-onset dilated cardiomyopathy, warranting further clinical evaluation.

CONCLUSION

The case described highlights two important, yet rarely reported, characteristics of deltamethrin toxicity that impact BChE activity and cause cardiac toxicity. Past research has established the neurotoxic and cardiotoxic properties of pyrethroids but decreased BChE activity discovered in this case indicates a previously unidentified cholinergic effect of deltamethrin in humans. The development of non-ischemic cardiomyopathy in a patient with no other cardiac health issues should cause concern about exposure to pyrethroid chemicals.

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