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CASE REPORT

Tetrodotoxin-induced involuntary movements: a case report

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Abstract

Background: Tetrodotoxin (TTX) is a potent neurotoxin found in pufferfish that blocks voltage-gated sodium channels and leads to flaccid paralysis. Although its effects are typically limited to the peripheral nervous system, rare central neurological manifestations may be under-recognized. **Case**: A 70-year-old male presented to the emergency department approximately 10 h after consuming pufferfish stew. The initial symptoms included slurred speech, facial weakness, and general fatigue without respiratory compromise. Vital signs and laboratory evaluations, including cardiac enzyme levels, blood gas analysis, and toxicology screening, were within normal limits. Brain computed tomography and electrocardiogramrevealed no acute abnormalities. Shortly after arrival, the patient developed progressive involuntary side-to-side head movements and repetitive swaying of the upper body. Comprehensive toxicological screening revealed negative results for other neurotoxins. Supportive care was administered and no antidotes were available. Over the course of 12 to 24 h, the involuntary movements gradually subsided and fully resolved by 48 h post-ingestion, with complete neurological recovery. **Conclusion**: This case report describes a rare presentation of tetrodotoxin poisoning involving involuntary motor activity, suggesting potential central nervous system involvement. This challenges the conventional understanding that tetrodotoxin toxicity is confined to the peripheral nervous system. Clinicians should be aware of such atypical features to ensure a timely diagnosis and appropriate supportive care.

Keywords

Tetrodotoxin; Nervous system diseases; Poisoning; Involuntary movements

1. Introduction

Tetrodotoxin (TTX) is a potent neurotoxin found primarily in pufferfish and other marine organisms [1, 2]. It can cause severe neurological paralysis in humans and can lead to death. Although TTX poisoning is not prevalent globally, it continues to occur in specific regions and cultural contexts, making it a relevant concern in the field of public health [3]. TTX poisoning is most frequently reported in the Asia-Pacific region [3, 4]. Japan has historically recorded the highest number of TTX poisoning cases, mostly attributed to the consumption of fugu (pufferfish) [4]. During the 1960s, Japan reported several hundred cases annually with a case fatality rate of approximately 6–7% [5]. However, the implementation of strict licensing systems and regulatory policies for pufferfish preparation has led to a dramatic decline, with fewer than one or two cases being reported annually in recent years. Similarly, countries such as China, the Philippines, and Thailand have reported multiple poisoning incidents linked to the consumption of pufferfish or other TTX-containing seafood [6–8].

Sporadic cases of TTX poisoning have been reported in regions such as the Middle East and Africa, particularly along the Red Sea coast in countries such as Egypt and Sudan, where toxic pufferfish are consumed. In North America, TTX poisoning is rare, but has occurred in areas such as Hawaii, California, and Florida, typically associated with imported seafood or traditional cooking practices within immigrant communities [8, 9].

While pufferfish (family Tetraodontidae) are the most well-known carriers of TTX, other marine species, such as blue-ringed octopuses, sea urchins, certain gastropods, and polychaete worms, may also contain the toxin. TTX tends to peak during the breeding season of pufferfish, typically from spring through summer, leading to a seasonal clustering of poisoning incidents [9].

The case fatality rate for TTX varies by region and access to medical care, ranging from 7% to 60%. Early diagnosis and timely respiratory support are critical for improving patient outcomes [10]. Countries such as Japan have implemented stringent controls on the sale and preparation of pufferfish,

and the World Health Organization (WHO) has issued advice regarding the risks associated with TTX-containing foods [11].

In summary, although TTX is geographically concentrated, it remains a significant food safety and toxicological concern, requiring continued surveillance, education, and regulatory action at both national and international levels [12]. TTX is a potent neurotoxin that exerts its effect by selectively blocking voltage-gated sodium channels in excitable tissues, such as peripheral nerves and skeletal muscles [13, 14]. This blockade inhibits the initiation and propagation of action potentials, ultimately resulting in flaccid paralysis [14].

TTX poisoning typically occurs following the ingestion of pufferfish and other marine organisms containing toxins [15]. Clinical symptoms usually develop rapidly, within 10 min to 3 h after ingestion, with more severe cases associated with higher doses of toxin exposure [14, 15].

The primary mechanism of tetrodotoxin toxicity involves its high-affinity binding to the extracellular pore region of voltage-gated sodium channels (Nav1.1–Nav1.7), particularly in neuronal and skeletal muscle membranes [16]. By occluding the sodium-ion pathway, TTX prevents the influx of sodium ions, which is essential for the generation of action potentials. As a result, depolarization of the nerve or muscle membrane is inhibited, leading to failure of nerve signal transmission and subsequent flaccid paralysis [14, 16].

Importantly, TTX does not readily cross the blood-brain barrier; therefore, central nervous system function remains relatively intact, which explains why many patients retain full consciousness even in the presence of profound paralysis [16, 17]. This unique feature makes TTX clinically distinguishable from other forms of neurotoxicity or central nervous system depressants [17]. However, a 59-year-old man who ingested pufferfish toxin experienced loss of consciousness and fixed dilated pupils. Electroencephalogram (EEG) examination revealed mild cortical dysfunction. This indicates that cases of central nervous system dysfunction have been reported in patients with TTX poisoning [18].

Cardiac muscle is generally less affected than skeletal muscle owing to differences in sodium channel subtypes; however, high doses of TTX can also impair cardiac conduction, resulting in bradycardia or fatal arrhythmias. Clearance of the toxin occurs through hepatic metabolism and renal excretion, although the exact pharmacokinetics in humans remains incompletely characterized [13, 17]. The clinical course of TTX can be divided into three progressive stages (mild, moderate, and severe). In the early (mild) stages, patients commonly report perioral paresthesia, including numbness or tingling of the lips, tongue, and face, which may extend to the extremities. This may be accompanied by dizziness, headache, nausea, vomiting, abdominal discomfort, and diarrhea [17].

As the condition progresses to a moderate stage, neurological symptoms become more prominent. These symptoms include dysarthria (slurred speech), hypersalivation, progressive muscle weakness, and ataxia [11, 14, 17]. Paralysis typically ascends from the lower to upper extremities, and early signs of respiratory involvement, such as dyspnea or chest tightness, may begin to manifest.

Respiratory muscle paralysis can occur in severe cases, leading to respiratory failure. Notably, patients often remain conscious and alert during paralysis, producing a clinical state similar to “locked-in syndrome”. Autonomic dysfunction may also develop, including bradycardia, hypotension, and cardiac arrhythmia. Without prompt medical intervention, death usually occurs within 4–6 h post-ingestion due to respiratory arrest [17]. Currently, there are no specific antidotes for the use of TTX. Management is supportive and primarily focuses on securing the airway, providing oxygen, and initiating mechanical ventilation when needed. In cases where respiratory support is promptly initiated, patients often recover fully within 24 h, as the toxin is eventually cleared from the body. However, the mortality rate can exceed 50% in cases that lack adequate and timely intervention, underscoring the importance of rapid recognition and emergency treatment.

2. Case report

A 70-year-old male who consumed pufferfish stew at approximately 5:30 PM. At approximately 8:00 PM on the same day, he began experiencing slurred speech, facial paralysis, and dizziness. As the symptoms worsened, he presented to the emergency department at approximately 3:30 AM. The patient stated that he had consumed approximately one whole pufferfish and had eaten its raw gonads. A review of the patient’s medical history revealed no evidence of epilepsy, Parkinson’s disease, or metabolic disorder.

Upon arrival, the patient was fully alert, with a Glasgow Coma Scale (GCS) score of 15. And the patient’s blood pressure was 190/100 mmHg, heart rate was 69 beats per minute, respiratory rate was 20 breaths per minute, body temperature was 36.9 °C, and oxygen saturation was 99%. Physical examination revealed dysarthria, while motor strength testing of all four limbs showed intact function with a motor grade of five points. However, the patient complained of general weakness and was unable to walk properly. In addition, the patient complained of heart burning. Arterial blood gas analysis revealed a pH of 7.397, base excess of −0.3 mmol/L, lactate level of 1.9 mmol/L, and hematocrit of 52.8%. Hemoglobin was measured at 17.2 g/dL, arterial carbon dioxide ($PaCO_2$) at 39.9 mmHg, and arterial oxygen (PaO_2) at 76.6 mmHg. Other electrolyte levels were within the normal limits.

Cardiac enzyme tests revealed normal findings: Creatine Kinase-MB, 1.59 ng/mL, Troponin I, 4.5 ng/L, and N-terminal pro-B-type natriuretic peptide, 48.9 pg/mL. Blood urea nitrogen (BUN), creatinine was 0.92 mg/dL, aspartate aminotransferase (AST) were 19 mg/dL, 0.92, 32 U/L, and alanine aminotransferase (ALT) was 38 U/L, respectively. In particular, the patient’s CRP (C-Reactive Protein) level was measured at 2.6 mg/dL.

An electrocardiogram taken upon arrival is shown in Fig. 1. The initial electrocardiogram (ECG) revealed a normal sinus rhythm without evidence of ischemic changes, arrhythmia, or conduction abnormalities.

To differentiate stroke from the patient’s complaint of dysarthria, a brain computed tomography (CT) scan was performed, as shown in Fig. 2. Initial brain CT was performed upon arrival to rule out acute cerebrovascular events. The radiological report confirmed no evidence of intracranial hemorrhage, infarction, or space-occupying lesions. Overall,

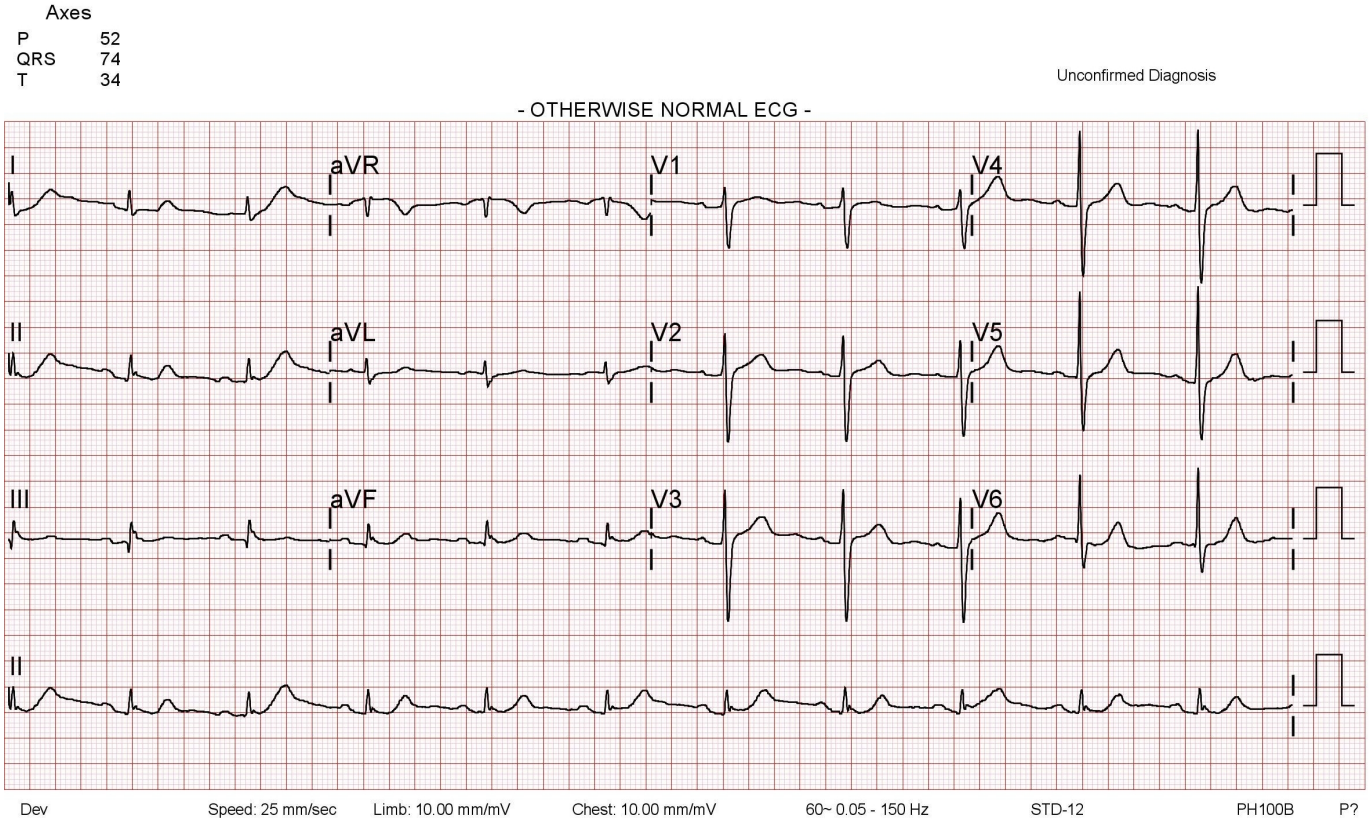

FIGURE 1. Initial electrocardiographic findings. The initial 12-lead electrocardiogram showed a normal sinus rhythm with no ischemic or arrhythmic changes. P, P wave; QRS, QRS complex; T, T wave; aVR, augmented Vector Right (lead aVR); aVL, augmented Vector Left (lead aVL); aVF, augmented Vector Foot (lead aVF); ECG, electrocardiogram; STD, ST-segment depression.

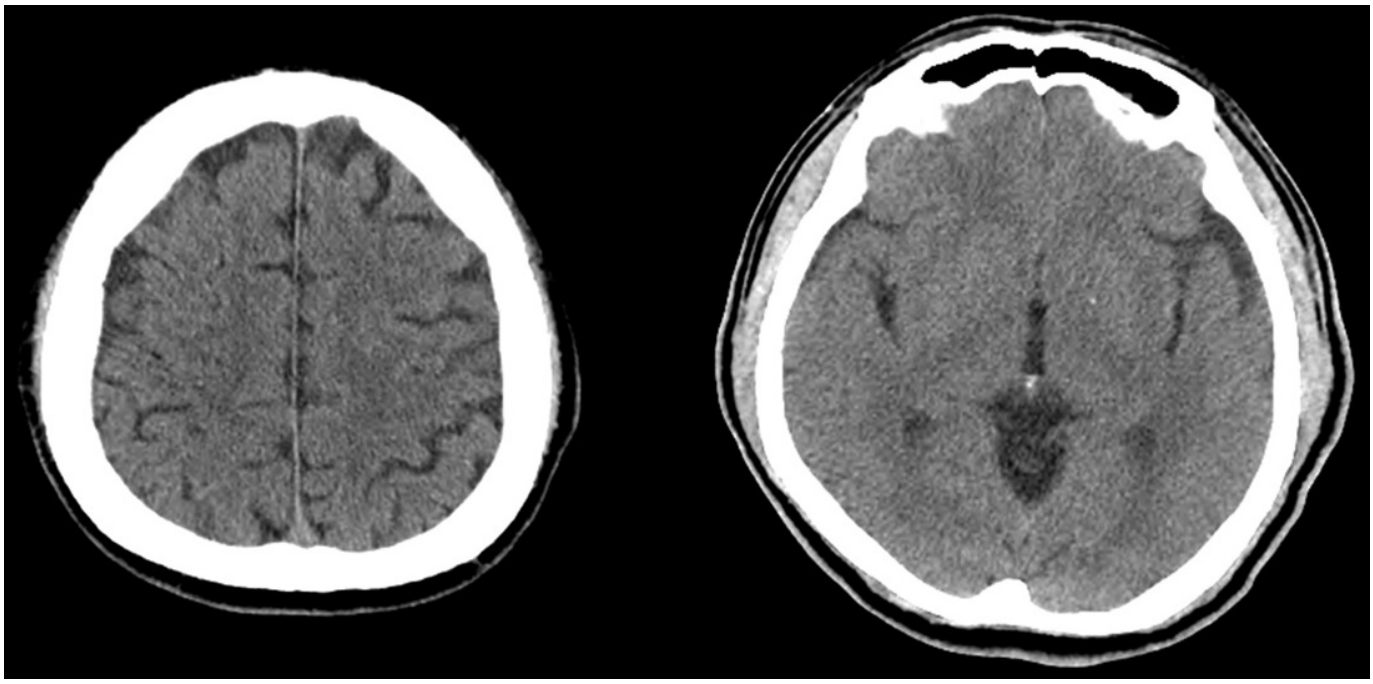

FIGURE 2. Initial brain CT findings. Brain CT image obtained upon presentation showing no evidence of intracranial hemorrhage or acute infarction.

the CT findings were within the normal limits.

Approximately 20 min after arriving at the emergency department, the patient began to exhibit nonspecific neurological symptoms. The patient complained of discomfort due to involuntary side-to-side head movements that progressively worsened over time. Initially, he began to move his head side to side intermittently but eventually started shaking his head more vigorously and simultaneously exhibited severe involuntary back and forth movements of his body. The involuntary movements exhibited by patients are shown in Fig. 3.

Additional drug testing was performed to identify the cause of these involuntary movements. Tests for amphetamines, methamphetamines, barbiturates, benzodiazepines, cocaine, cannabinoids, opiates, tricyclic antidepressants, and methadone yielded negative results. Supportive care was initiated promptly to manage the patient's condition. Despite the involuntary movements, his vital signs remained stable, and he maintained adequate oxygen saturation without respiratory compromise. Continuous monitoring was conducted in the emergency observation unit, and intravenous hydration and symptomatic management were provided to the patient.

Given the absence of structural abnormalities on imaging and the negative results from the comprehensive toxicology screen, TTX poisoning was strongly suspected to be the primary cause of neurological symptoms.

Currently, no specific antidotes are available for TTX. Nevertheless, continuous infusion of Plasma-Lyte was provided for fluid resuscitation, along with the administration of activated charcoal. As the patient weighed 68 kg, 68 grams of activated charcoal were diluted and administered. A total of 68 g of activated charcoal was diluted in 450 mL of water and placed in a paper cup, allowing the patient to self-administer the mixture. Although the patient exhibited involuntary movements, he remained alert and was able to ingest the activated charcoal voluntarily without difficulty. Therefore, administration of antipsychotic medications or sedatives was not required. The treatment focused on close observation and supportive therapy. A neurological consultation was conducted to identify the cause of the patient's dizziness and involuntary movements. To evaluate possible central nervous system involvement, the neurology department conducted lumbar punctures in the brain.

Cerebrospinal fluid (CSF) analysis obtained via lumbar puncture demonstrated results within normal physiological ranges. Cerebrospinal fluid (CSF) analysis obtained via lumbar puncture revealed findings consistent with bacterial infection. The CSF appeared turbid upon gross examination. Laboratory results revealed a markedly elevated white blood cell (WBC) count of 1240 cells/μL with predominant neutrophilic pleocytosis, an elevated protein concentration of 185 mg/dL, and a significantly decreased glucose level of 24 mg/dL (with a concurrent serum glucose level of 92 mg/dL), suggestive of bacterial metabolism within the CSF. CSF Gram staining revealed the presence of numerous polymorphonuclear leukocytes and gram-negative bacilli. CSF culture subsequently yielded growth of Escherichia coli, a known pathogen in Central Nervous System infections. The opening pressure was elevated to 260 mmH_2O. As part of the initial empirical treatment for suspected bacterial meningitis, the patient was administered ceftriaxone intravenously at a dose of 2 g every 12 h (2 g IV q12h).

Over the next 12 h, the patient's involuntary movements and dizziness gradually diminished in both frequency and intensity. At 24 h post-ingestion, dizziness and abnormal motor activity completely resolved. The patient also reported a significant improvement in general weakness and was able to ambulate without assistance. Repeat neurological examinations confirmed the absence of dysarthria, facial weakness, or abnormal movements. He was discharged in stable condition after 48 h of observation, with no residual neurological deficits.

This case highlights the importance of early supportive care and vigilant monitoring of suspected tetrodotoxin poisoning, particularly when atypical symptoms, such as involuntary movements, are present.

3. Discussion

TTX is a well-characterized, heat-stable neurotoxin that exerts its toxic effects through the selective blockade of voltage-gated sodium channels (Nav1.1–Nav1.7), primarily affecting excitable tissues such as peripheral nerves, skeletal muscles, and

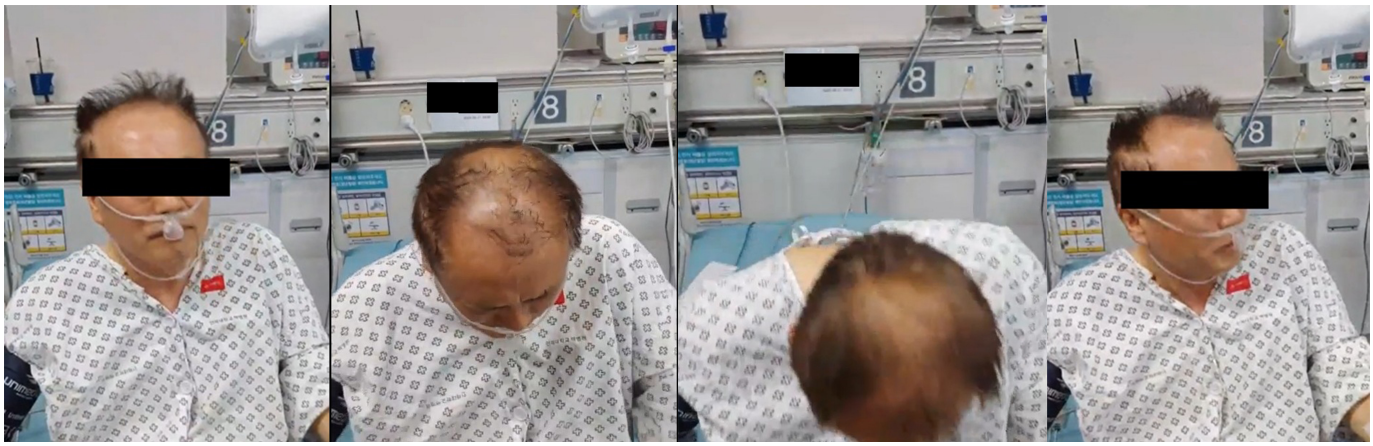

FIGURE 3. Involuntary movements observed after tetrodotoxin poisoning. Following pufferfish (tetrodotoxin) poisoning, the patient exhibited involuntary movements including side-to-side head shaking and back-and-forth swaying of the body.

cardiac conduction pathways [18]. The mechanism of the toxin involves binding to the extracellular pores of sodium channels, thereby preventing sodium influx, disrupting the generation and propagation of action potentials, and ultimately leading to flaccid paralysis [2, 18]. Clinically, the initial symptoms of TTX poisoning typically appear within 10 minutes to 3 hours after ingestion, depending on the toxin dose and individual sensitivity [3, 11, 19]. The earliest and most characteristic signs include perioral paresthesia, tingling, and numbness around the lips and tongue [20]. These symptoms often progress to involve the face, extremities, and, sometimes, the trunk. This peripherally dominant presentation is consistent with the pharmacological properties of TTX, which poorly penetrates the blood-brain barrier [19, 20].

Despite the identification of abnormal cerebrospinal fluid (CSF) parameters suggestive of bacterial meningitis, including turbid appearance, elevated white blood cell count with neutrophilic predominance, increased protein levels, and reduced glucose concentration, the interpretation remains complex. While these findings may indicate a concurrent or secondary bacterial CNS infection, the temporal correlation with tetrodotoxin (TTX) exposure and the presence of atypical neurological symptoms, such as progressive involuntary movements, raises the possibility of TTX-associated central nervous system involvement [21, 22]. Moreover, although Escherichia coli was isolated from the CSF culture, the lack of systemic signs of sepsis and unusual neurologic presentation warrants consideration of alternative or synergistic mechanisms. Emerging evidence provides potential mechanistic insights into how TTX exposure, in the context of systemic inflammation, may contribute to central neurotoxicity. Several studies have reported that inflammatory cytokines released from the peripheral nervous system can disrupt the tight junction and transcytosis pathways of the blood-brain barrier (BBB), leading to increased permeability. Other studies have shown that peripheral inflammation (such as burns or trauma) can elevate the levels of Interleukin-6 (IL-6) and Interleukin-1β (IL-1β), thereby increasing BBB permeability, activating central immune cells, and potentially causing neurodegeneration as well as cognitive and neurological dysfunction [23].

In this case, the markedly elevated C-reactive protein (CRP) level (26 mg/L) may reflect an acute-phase systemic inflammatory response. Although CRP is a nonspecific marker, its elevation supports the hypothesis that TTX toxicity may induce a broader immunological or inflammatory cascade beyond its known peripheral neurotoxic effects. Previous studies have suggested that systemic inflammation can disrupt the integrity of the blood-brain barrier through cytokine-mediated pathways such as IL-1β or IL-6, thereby facilitating the entry of circulating neurotoxins into the CNS. Although direct evidence for this mechanism in human TTX poisoning is limited, this case underscores the need to further explore the role of inflammation and BBB permeability in mediating central neurological manifestations following TTX exposure [21–23].

Based on this evidence, the involuntary muscle movements observed in the patient were understood to be a result of TTX infiltration due to central nervous system infection. The disappearance of involuntary movements following the administration of activated charcoal and the detoxification process are also considered to be supporting evidence. In addition, the dizziness reported by the patient was interpreted as peripheral vertigo caused by TTX, consistent with the findings of previous studies [24, 25].

Therefore, the early and moderate stages of TTX poisoning are classically characterized by peripheral paralysis that occurs with full preservation of consciousness. However, if TTX crosses the blood-brain barrier due to a central nervous system infection, involuntary movements may occur. Such atypical symptoms should be recognized as possible exceptions in the clinical presentation of TTX, particularly in emergency settings [26]. As toxicity progresses, the muscle weakness becomes more pronounced. Patients may begin to experience fatigue, difficulty in walking, or a sensation of limb heaviness. Additional neurological symptoms including dizziness, diplopia, and dysarthria may also occur. In advanced stages, flaccid paralysis may ascend from the lower extremities and involve the respiratory muscles, potentially leading to respiratory failure, a common cause of death in severe cases of TTX [19].

Interestingly, the case presented here demonstrated an atypical neurological course. In addition to dysarthria and facial weakness, the patient developed dizziness and progressive involuntary movements of the head and trunk, including rhythmic and repetitive motions, suggestive of central motor pathway involvement. Such a presentation is rare and, to our knowledge, represents the first reported case of tetrodotoxin-associated involuntary movements potentially implicating central nervous system dysfunction [18]. This observation raises the possibility that TTX, under certain physiological or pathological conditions, may exert more extensive effects than previously appreciated, possibly due to individual variability in blood-brain barrier permeability, toxin dose, or other cofactors.

There is no known antidote to TTX poisoning. Management is primarily supportive and focuses on the prompt recognition of symptoms and aggressive stabilization of the airway, breathing, and circulation. The cornerstone of treatment is respiratory support. In moderate-to-severe cases, mechanical ventilation may be required for hours to days, depending on the degree of respiratory muscle paralysis. In our case, the patient did not require intubation; however, this finding may not be generalizable.

For patients presenting within an hour of ingestion, activated charcoal may be administered to reduce the gastrointestinal absorption of the toxin. However, its effectiveness beyond the first hour is unclear, and its use must be weighed against the risk of aspiration, particularly in drowsy or neurologically impaired individuals. Gastric lavage is no longer routinely recommended but may be considered in cases of massive recent ingestion in conscious patients.

Various adjunctive therapies have been explored, including pyridoxine, sodium bicarbonate, and atropine; however, data regarding their efficacy are limited and primarily anecdotal. No randomized clinical trial has demonstrated a clear benefit. In this context, the role of early supportive care, especially airway protection and hemodynamic stabilization, cannot be overstated. The prognosis of TTX poisoning largely depends on the time to medical intervention and availability of respiratory support. Without prompt airway management, mortality

rates can reach 50–60%, especially in settings without access to intensive care. However, when supportive care is adequate and timely, most patients make a full recovery within 24 to 48 h, as the toxin is metabolized by the liver and excreted renally.

The patient described in this report exhibited full clinical recovery with only symptomatic treatment despite presenting with unusual involuntary motor activity. This outcome underscores the reversibility of TTX and the importance of monitoring for atypical neurological manifestations. It also calls for greater clinician awareness of the broader symptomatology beyond the classic signs of peripheral nerve and respiratory involvement. This case expands the current clinical understanding of TTX by introducing previously unreported symptoms of involuntary central motor activity. This raises the possibility that TTX may, in rare cases, exert central effects that are not solely explained by its classical pharmacodynamics. Further investigation is needed to determine whether such symptoms represent an outlier or previously under-recognized manifestation. Neuroimaging and electrophysiological studies in similar future cases may help elucidate the pathophysiological basis of these findings. Additionally, there is a need for the development of specific antidotes or pharmacological antagonists of TTX. The unique mechanism of sodium channel blockade provides a targeted pharmacological niche that can be explored for therapeutic intervention. Until such agents become available, clinical vigilance, public education, and regulatory oversight of pufferfish preparation remain essential strategies for prevention and risk mitigation.

4. Limitation

This case report has several limitations. First, because it describes a single patient, the findings cannot be generalized, and the atypical presentation of involuntary movements may represent either a rare phenomenon or an under-recognized aspect of tetrodotoxin (TTX) poisoning. Additional cases are required to determine the broader clinical significance of these observations. Second, the interpretation of the patient's neurological symptoms is complicated by the presence of concurrent bacterial meningitis. The abnormal cerebrospinal fluid findings and confirmed Escherichia coli infection make it difficult to clearly distinguish whether the involuntary movements resulted from TTX-induced central effects, from the CNS infection itself, or from a synergistic interaction between both processes. This overlap limits the ability to attribute causality to TTX alone. Third, advanced neurodiagnostic tools—such as brain MRI, electroencephalography, electromyography, or quantitative TTX level measurements—were not performed. The absence of these objective assessments restricts the ability to elucidate the underlying neurophysiological mechanisms and confirm central nervous system involvement. Despite these limitations, this report provides important insight into a potentially atypical manifestation of TTX toxicity and underscores the need for further investigation.

5. Conclusion

Although geographically limited, TTX poisoning remains a significant public health concern in regions in which pufferfish consumption is culturally prevalent, particularly in Asia. Typical presentations of TTX poisoning predominantly involve peripheral nervous system impairment with symptoms such as facial paralysis and dysarthria, while central nervous system involvement is considered uncommon because of the toxin's limited ability to cross the blood-brain barrier.

This case report highlights a rare but noteworthy manifestation of TTX poisoning, such as involuntary movements of the head and body, potentially suggesting involvement of the central nervous system. While the early symptoms were mild, including slurred speech and facial weakness, the patient progressed to exhibit pronounced, repetitive, and involuntary motor activity. These findings expand the clinical spectrum of TTX and suggest that the central effects may be under-recognized. Currently, there is no specific antidote for tetrodotoxin, and its treatment relies on rapid recognition and aggressive supportive care, especially respiratory support. Although the patient in this case fully recovered with symptomatic management, the unique neurological presentation underscores the need for clinicians to be vigilant of atypical signs of TTX poisoning. Further research is warranted to elucidate the full range of neurological effects associated with TTX and develop standardized treatment protocols. Additionally, improved awareness and regulation of pufferfish preparation and distribution remains essential to prevent future cases.

AVAILABILITY OF DATA AND MATERIALS

The datasets used and/or analyzed in the present study are available by the corresponding author on reasonable request.

AUTHOR CONTRIBUTIONS

JHK—the first author developed the initial concept of the case report, collected all relevant clinical data, conducted a comprehensive literature review, and drafted the initial manuscript including the case description and early discussion. YDJ—the corresponding author, supervised the entire study process, refined the study design, validated the clinical and neurological interpretations, prepared and finalized all figures, and critically revised the manuscript for intellectual depth and clarity. SIK, SMH—contributed to the clinical investigation, supported the acquisition of diagnostic information, and provided important comments that strengthened the relevance of the findings. EKJ, SMH—assisted in interpreting cerebrospinal fluid results and inflammatory markers, contributed additional literature related to blood–brain barrier disruption, and reviewed the manuscript for scientific accuracy. SJK—participated in reviewing the patient's neurological course, ensured consistency between clinical documentation and manuscript content, and provided critical revisions to improve readability and clinical applicability. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. The study was approved by institutional review board of Inje paik Hospital, Ethics Approval Number: 2024-031. Written informed consent was obtained from the patient for publication of this study and accompanying images.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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