Case Report

Journal of Emergency Medicine Case Reports

Myoclonus Developing After High-Dose Pregabalin Intake: A Rare Clinical Case

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Abstract

Pregabalin has become a widely prescribed drug worldwide in recent years. There has been an increase in its accidental, suicidal, and even abusive use. It induces anxiolytic, sedative, or euphoric effects when taken together with substances such as alcohol. It is associated with many adverse effects, and neurologic side effects may also occur. Here, a 17-year-old male patient who had myoclonic seizures after taking high doses of pregabalin with alcohol without any known additional drug intake is presented because it is a rare clinical finding. A 17-year-old male patient was brought to the pediatric emergency department due to a fall after drinking alcohol. His triage vital signs were stable. His Glasgow Coma Scale score was 15. In the 30th minute of his stay in the PED, the patient had myoclonic jerks in his arms and legs, and their frequency began to increase. The patient's history was questioned again. His brother stated that the patient might have taken pregabalin. The patient stated that he had taken seven pregabalin pills (1 pill = 300 mg) with alcohol. In laboratory tests, there was no pathology other than respiratory acidosis. COHb: 7.2, lactate: 7. The blood ethanol level was 10 mg/dL. Cranial and cervical tomography were normal. The serum pregabalin level was determined to be 300 mg/L using the LC-MS/MS method at the reference hospital. Other toxic analyses were negative. The patient was discharged after one day of supportive treatment. Alcohol and high-dose pregabalin intake may rarely lead to the development of neurologic adverse effects such as myoclonus. Given the increasing prescription rates, pregabalin poisoning and related clinical findings will be frequently seen in emergency departments. Therefore, especially in cases with alcohol consumption, additional medication intake should be questioned, pregabalin exposure should be considered, and appropriate management strategies should be implemented.

Keywords: Adverse effects, alcohol, myoclonus, pregabalin

Introduction

Pregabalin (S-(+)-3-isobutyric acid) (Lyrica®) is a lipophilic analog of γ-aminobutyric acid (GABA). It has a similar chemical structure to gabapentin but is a more potent antiepileptic than gabapentin. It is used in many diseases, such as partial seizures, central and peripheral neuropathic pain, and anxiety disorder (1,2). It binds to the α2-δ subunit of the voltage-gated calcium channel in the brain and spinal cord and reduces the release of neurotransmitters, including glutamate, norepinephrine, and Substance P (3). Pregabalin does not bind to plasma proteins and readily crosses the blood-brain barrier. It is cleared unchanged by renal excretion with an elimination half-life of approximately 6 hours. This period is increased in patients with renal failure, depending on creatinine clearance (4).

Myoclonus is a clinical symptom characterized by brief, shock-like, involuntary movements caused by muscle contractions or inhibitions. Drugs that can cross the blood-brain barrier, such as pregabalin, cause myoclonus (5). The mechanisms for myoclonus induced by drugs or toxins are poorly defined (6). In addition, it is unclear why myoclonus occurs in some individuals and not others. However, as the frequency of use of these drugs increases, the likelihood of experiencing adverse effects and emergency department visits will increase.

Multifocal, isolated myoclonus and isolated seizures have been reported as neurologic findings in high-dose gabapentin or pregabalin use. The incidence of myoclonus is higher in patients with impaired renal function. Increased muscle rigidity, clonus, and hyperreflexia are not expected (7-10). Here, a 17-year-old male patient who had myoclonic seizures after taking high doses of pregabalin with alcohol without any known additional drug intake is presented because it is a rare clinical finding.

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Received: 05.05.2025 • **Revision:** 21.07.2025 • **Accepted:** 17.08.2025
DOI: 10.33706/jemcr.1692249

Cite this article as: Derinoz Guleryuz O. Myoclonus Developing After High-Dose Pregabalin Intake: A Rare Clinical Case. Journal of Emergency Medicine Case Reports. 2025;16(4): 134-136

Case Report

A 17-year-old male patient was brought to the Pediatric Emergency Medicine (PED) by ambulance due to a fall and head trauma after drinking alcohol in a park. His medical history revealed that he had drunk 7-8 glasses of whiskey and 2-3 glasses of beer, approximately 3hours before admission, then fell and hit his head on a parked car after losing his balance. He had no history of taking any other drugs or substances. The patient's triage vital signs were stable, and his Glasgow Coma Scale (GCS) score was 15. His speech was difficult to understand, but his history could be obtained. The patient was taken to the resuscitation room and placed on a backboard; a cervical collar was placed. His vital signs were stable. In the physical examination, there were no pathologic findings other than abrasions and soft tissue edema in the right frontal and zygomatic regions. Fingertip blood sugar was recorded as 114 mg/dL. The patient was monitored, and oxygen therapy was started with a non-rebreathing oxygen mask with a reservoir. Isotonic fluid was given at 20 cc/kg. Blood gas, laboratory, and imaging tests were planned due to the history of alcohol consumption and head trauma.

Approximately 30 minutes into the application, the patient had myoclonic jerks in his arms and legs, and their frequency began to increase during follow-up. With the assumption that alcohol consumption would not cause myoclonic seizures, the patient's history was questioned again. When his brother stated that the child's friends used pregabalin and that his brother might have used it as well, it was thought that the clinical symptoms might be related to pregabalin. The patient stated that he consumed seven pregabalin pills (1 pill=300 mg) with alcohol. During this time, the patient's myoclonic seizures began to increase.

The patient was administered midazolam to stop his myoclonus before being sent for tomography. He had a short-term respiratory arrest. He recovered quickly with bag-mask ventilation, and spontaneous respiration occurred.

The patient's venous blood gas showed respiratory acidosis (PH: 7.31, PCO2: 48.6, PO2: 49.2, SO2: 82.9%, HCO3: 19, BE:-7). COHb: 7.2, Lactate: 7. Although he stated that he did not smoke, the patient was considered to have CO poisoning and continued oxygen therapy. Complete blood count, liver function tests, and kidney function tests were normal. Serum paracetamol and salicylic acid levels were negative. The blood alcohol level (ethanol) was determined as 10 mg/dL (normal range: 0-10 mg/dL). Cranial and cervical tomography was normal. His serum pregabalin level was studied using the LC-MS/MS method at the reference hospital, which was determined as 300 mg/L (Detection limit: 0.41 mg/L). Other toxic analyses were negative. The patient's legal report was written. The Department of Child and Adolescent Psychiatry was consulted due to substance abuse. The patient was admitted to the Pediatric Intensive Care Unit for follow-up. The patient received supportive treatment for 1 day and was discharged after his myoclonus regressed during follow-up.

Discussion

Pregabalin has become a widely prescribed drug worldwide in recent years. Therefore, there has been an increase in its accidental, suicidal, and even abusive use (1). This case report is presented to discuss myoclonus, a rare clinical condition following high-dose pregabalin intake. Although myoclonus has rarely been reported with isolated pregabalin intake in the literature, our case is presented to emphasize that a rare neurologic symptom, such as myoclonus, may occur with high-dose pregabalin intake with alcohol.

Pregabalin is a drug used in many diseases, such as partial seizures, central and peripheral neuropathic pain, and anxiety disorders (1,2). It has been assumed that pregabalin has euphoric effects due to its direct or indirect impact on the dopaminergic reward system. Patients increasingly use higher doses of pregabalin than recommended to achieve these euphoric effects (11). It has also been reported that pregabalin induces anxiolytic, sedative, or euphoric effects when taken together with substances such as alcohol, benzodiazepines, opioids, cannabis, and amphetamines (12-16). In the study by Isoardi et al.(8), 488 presentations of 418 patients with pregabalin exposure were evaluated retrospectively. When the clinical findings of the cases, ranging in age from 15 to 89 (median age: 41) years, were assessed, 16% of all exposures were found to have coma, 5% had hypotension, and 2% had seizures. The median pregabalin dose of the cases with documented pregabalin dose was reported as 1200 mg. Eighty-eight percent of all pregabalin exposures were concomitant with drug intake, and concomitant alcohol intake was also detected in 18%. Of the 58 patients who received isolated pregabalin, one was found to have coma, one had hypotension, one had agitation, and three had seizures. No respiratory depression respiratory depression was observed in any patients. As a result, pregabalin is often taken in polypharmacy, and isolated intakes are pretty rare. Coma is not associated with isolated intakes but with intakes together with other sedative drugs (8).

According to the results of this study, myoclonus was not detected in any of the patients. These data show that myoclonus is a rare clinical condition. Therefore, this case emphasizes that myoclonus may occur rarely and that pregabalin exposure should be considered in cases with unexplained clinical findings. In a study evaluating patients developing myoclonus with gabapentin or pregabalin, 38 patients aged between 23 and 79 were examined. It was reported that 12 (32%) of these patients developed pregabalin-related myoclonus (7). One reported case was of a 30-year-old patient with renal failure who developed

DOI: 10.33706/jemcr.1692249
Journal of Emergency Medicine Case Reports. 2025;16(4): 134-136

myoclonus when the pregabalin dose was increased (17). Unlike that case, ours involved myoclonus without renal failure following high-dose pregabalin. Isolated pregabalin intakes are usually benign, and patients can be monitored with supportive treatment (8).

Pregabalin has a low volume of distribution (approximately 0.5 L/kg), a low molecular weight (approximately 159 Da), and does not bind to protein (18). Due to these properties, drug elimination can be achieved using extracorporeal methods such as hemodialysis and/or hemofiltration in the treatment. It has been reported in the literature that a 30-year-old patient who was on hemodialysis due to renal failure and had myoclonus after their pregabalin dose was increased was successfully treated with hemodialysis again (17). Our patient had no known renal disease, and despite the high serum pregabalin level, he was discharged after being monitored with supportive treatment and successfully managed. Patient management after taking high doses of pregabalin and alcohol together may require a multidisciplinary approach. Given the increasing pregabalin prescription rates, it is likely that such cases will be managed more frequently in emergency departments.

Conclusion

As a result, alcohol and high-dose pregabalin intake may rarely lead to the development of neurologic adverse effects such as myoclonus. Given the increasing prescription rates, pregabalin poisoning and related clinical findings will be frequently seen in emergency departments. Therefore, especially in cases with alcohol consumption, additional medication intake should be questioned, pregabalin exposure should be considered, and appropriate management strategies should be implemented.

Consent was obtained from the patient for this study. The authors declared no conflict of interest.

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