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Case Report

Internal Medicine Section

Pregabalin-induced Positive Myoclonus Mimicking a Stroke: A Case Report

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ABSTRACT

Pregabalin is often prescribed to relieve neuropathic pain and is largely tolerated very well. Myoclonus as an adverse effect of pregabalin use is an infrequent occurrence that may go unrecognised. This case describes a 62-year-old female patient who developed myoclonus shortly after dose escalation of pregabalin from 75 mg twice daily to 150 mg twice daily, raising concerns about an acute underlying neurological condition. An extensive evaluation revealed no evidence of a structural, metabolic, infectious, epileptic, endocrine or toxic cause. The close temporal relationship between the onset of positive myoclonus and the use of pregabalin, along with the return of the patient to her baseline physical and neurological state following discontinuation of the drug, supported the diagnosis of pregabalin-induced positive myoclonus. Physicians should perform a thorough medication review for the identification of potential adverse drug reactions, exclude other possible diagnoses, and withdraw the use of pregabalin in order to ensure that the patient has a complete return to a normal neurological state.

Keywords: Adverse drug reactions, Drug-related side-effects, Electroencephalography, Lorazepam, Renal Insufficiency

CASE REPORT

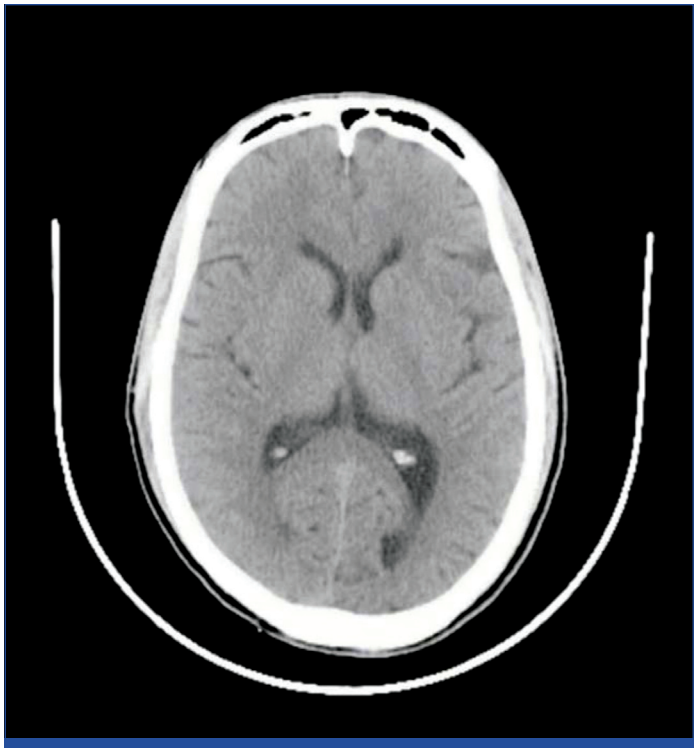
A 62-year-old female presented to the emergency department with an 8-hour history of sudden-onset involuntary jerking movements involving both upper limbs, associated with transient slurred speech, unsteadiness, and difficulty performing fine motor tasks. Her past medical history was significant for type 2 diabetes mellitus, hypertension since six years, and chronic low back pain secondary to degenerative spinal disease since two years. The movements were irregular, occurred in short bursts while the patient was at rest, and occasionally affected the lower extremities also. The jerks were multifocal, predominantly involved the distal upper limbs with occasional proximal and truncal involvement, were asynchronous. They were not stimulus-sensitive on clinical examination. There was no evidence of asterixis, tremor, chorea, dystonia or fasciculations. The patient remained fully conscious throughout the episodes. Myoclonus during sleep could not be assessed because the movements resolved before prolonged observation during sleep could be performed. The patient had a brief episode of slurred speech, felt "wobbly", and reported difficulty with fine motor tasks during these eight hours. The patient denied any loss of consciousness, tongue biting, urinary or faecal incontinence, headache, vision disturbances, fever, recent illness, head injury, alcohol use or illegal drug use.

The patient had been treated with pregabalin 75 mg twice daily for neuropathic pain for three months with adequate pain control before this episode. The dose of pregabalin was increased to 150 mg two times daily four days before presentation. She confirmed taking all prescribed medications exactly as directed and denied missed doses, self-adjustment of doses, or use of herbal preparations or other over-the-counter medications. In addition to pregabalin, she was on metformin 500 mg twice daily, telmisartan 40 mg once daily, atorvastatin 20 mg once daily and vitamin B12 1500 µg once daily. She denied starting any new prescription or over-the-counter medications. The patient had no previous history of seizures, strokes, movement disorders, or mental illness.

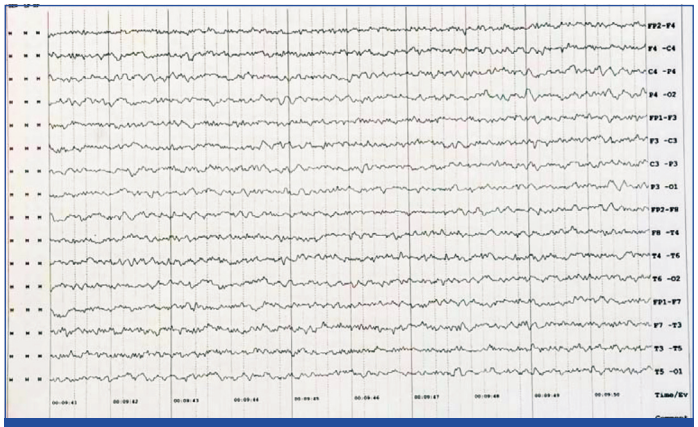
The patient's examination revealed that she was afebrile, with a blood pressure of 134/82 mmHg, pulse rate of 82 beats/min, respiratory rate of 16 breaths/min, oxygen saturation of 98% on room air, and capillary blood glucose of 118 mg/dL. She was alert and oriented to time, place, and person, and cooperative as evidenced by her Glasgow Coma Scale score being 15/15. The cranial nerve examination was unremarkable. Involuntary, frequent multifocal myoclonic jerks of both arms occurred without any alteration in mental status. Evaluation of muscle tone, strength, deep tendon reflexes, cerebellar function, gait and sensory function was normal. There were no focal neurologic deficits, meningeal signs, or evidence of extrapyramidal dysfunction.

Given the acute onset of transient neurologic symptoms and the presence of involuntary movement, the differential diagnosis included acute ischemic stroke, focal seizures, metabolic encephalopathy, drug or toxin-induced myoclonus, electrolyte disturbances, endocrine disease, and central nervous system infection. Immediate Non-Contrast Computed Tomography (NCCT) demonstrated neither intracranial bleeding nor ischaemia [Table/Fig-1]. The absence of focal neurological deficits on serial examinations, together with a normal CT brain, made acute stroke unlikely. An electroencephalography demonstrated a normal study without any epileptiform activity [Table/Fig-2]. A normal electroencephalography without epileptiform discharges and preserved awareness during episodes argued against an epileptic seizure disorder. Routine laboratory investigations, including complete blood count, serum electrolytes, renal and liver function tests, calcium, magnesium, thyroid function tests, inflammatory markers and blood glucose, were within normal limits, excluding metabolic, endocrine and infectious causes.

A detailed medication review found a clear temporal relationship between the dose of pregabalin increasing from 75 mg twice daily to 150 mg twice daily and the appearance of the patient's symptoms. The complete absence of structural, epileptic, metabolic, endocrine, infectious and toxic aetiologies excluded these as possible causes and led to the most likely diagnosis of pregabalin-induced myoclonus. Pregabalin was withdrawn immediately, and the patient received intravenous lorazepam 1 mg, followed by a second 1 mg dose due to continued jerking movements. The frequency and intensity of the myoclonic jerks diminished significantly within two hours after beginning intravenous lorazepam and resolved completely within twenty-four hours. The patient remained neurologically stable during her hospitalisation without any recurrence of symptoms and was discharged after forty-eight hours on 30 mg of duloxetine daily as an alternative treatment for neuropathic pain. A formal causality



[Table/Fig-1]: Non-Contrast Computed Tomography (NCCT) showing a normal study.



[Table/Fig-2]: Electroencephalography showing a normal study.

assessment using the Naranjo Adverse Drug Reaction Probability Scale yielded a score of 7, indicating a "probable" adverse drug reaction. When the patient returned for a review two weeks later, she was completely asymptomatic with no recurrence of myoclonic jerks or other neurological manifestations.

DISCUSSION

Pregabalin is structurally similar to γ-Aminobutyric Acid (GABA) and binds to the α2δ subunit of voltage-gated presynaptic calcium channels, resulting in decreased calcium influx and decreased release of excitatory neurotransmitters, like glutamate, norepinephrine, and substance P. Although pregabalin is generally well tolerated, it has a documented history of neurotoxic side-effects. While myoclonus is an uncommon side-effect of pregabalin, it is clinically significant and often presents as an acute neurological emergency [1-3]. The exact mechanism of action of pregabalin-induced myoclonus is still unclear. Potential mechanisms suggested include paradoxical cortical hyperexcitability and altered inhibitory neurotransmission. Although pregabalin is eliminated almost exclusively by the kidneys, neurotoxicity has occasionally been reported in patients with normal renal function, suggesting that individual susceptibility, altered blood-brain barrier permeability, genetic variability in α2δ receptor expression, or idiosyncratic neuronal hypersensitivity may contribute to the development of myoclonus independent of impaired drug clearance [1-3].

Zaidi S and Lyden PD reported positive myoclonus with transient focal neurological deficits resulting from an accidental increase in the dosage of pregabalin in a patient who had normal renal function [4]. In this case, the patient had normal renal function and an unremarkable metabolic workup, and symptoms resolved with discontinuation of pregabalin and treatment with lorazepam. Unlike the present case, the patient reported by Zaidi S and Lyden PD presented with transitory neurological deficits and was designated as a stroke alert, whereas the present case manifested only with positive myoclonus [4].

Desai A et al., in a case series involving seven patients, noted that myoclonus from pregabalin and gabapentin most often presented with multifocal upper-extremity jerking. They also noted that when pregabalin was discontinued, the patient returned to normal [5]. This patient demonstrated a similar pattern of multifocal upper-limb myoclonus but differed by having preserved renal function, thereby expanding the spectrum of patients in whom pregabalin neurotoxicity should be considered.

Pregabalin causes two types of movement disorders, positive myoclonus and negative myoclonus. Positive myoclonus results from sudden muscle contraction, while negative myoclonus results from a brief interruption of sustained muscle activity. Negative myoclonus can be subtle and may be confused with weakness, tremor or unexplained falls [6,7].

Park KD et al., described an individual who was experiencing confusion and mild renal impairment shortly after starting pregabalin, and who developed negative myoclonus [7]. In contrast, the current case developed positive myoclonus. Both patients exhibited complete clinical recovery after discontinuing the medication. A case of de novo absence status epilepticus following the start of pregabalin in a patient with end-stage renal failure receiving maintenance haemodialysis has been reported by Hussain K et al, which resolved after discontinuation of the drug [8]. Collectively, these reports indicate that pregabalin-related neurotoxicity encompasses a spectrum ranging from isolated movement disorders to altered consciousness and epileptic syndromes, with reversibility following drug discontinuation being a consistent clinical feature.

Myoclonus due to medication is diagnosed mostly by ruling out other possible causes including acute ischemic stroke, seizures, metabolic encephalopathy, toxic syndromes and other neurological conditions. A systematic evaluation, including appropriate laboratory tests, imaging studies and careful review of the most recent medication history, is necessary for diagnosis [3,5,6]. Furthermore, formal causality assessment using the Naranjo Adverse Drug Reaction Probability Scale classified the reaction as "probable", providing additional objective evidence supporting pregabalin as the causative agent [9,10].

Pregabalin can also cause neurologic manifestations other than movement disorder, including encephalopathy and epilepsy. Careful exclusion of alternative aetiologies, comprehensive review of recent medication use, prompt discontinuation of pregabalin, and supportive care can help prevent unnecessary investigations or inappropriate treatment [3,7,11].

The similarities and distinguishing features of the present case compared with previously reported cases of pregabalin-induced neurotoxicity are summarised in [Table/Fig-3].

CONCLUSION(S)

Pregabalin-induced positive myoclonus is a rare but reversible adverse drug reaction that may mimic acute neurological emergencies, including stroke and seizure disorders, even in patients with preserved renal function. This case highlights the importance of obtaining a detailed medication history and considering pregabalin-induced neurotoxicity after excluding structural, metabolic, infectious, and epileptic causes. Prompt

Study	Age/Gender	Renal function	Clinical presentation	Pregabalin exposure	Management	Outcome
Present case	62 years/Female	Normal	Multifocal positive myoclonus without encephalopathy or persistent focal deficits	Dose escalation from 75 mg BD to 150 mg BD	Drug withdrawal + lorazepam	Complete recovery within 24 hours; no recurrence at 2-week follow-up
Zaidi S and Lyden PD (2026) [4]	64 years/Female	Normal	Positive multifocal myoclonus with transient word-finding difficulty, diplopia, and brain fog	Inadvertent dose increase (200 mg to 300 mg nightly)	Pregabalin withdrawal + lorazepam	Complete resolution within 6 hours
Desai A et al., (2019) [5]	51 years/Male, 68 years/Male, 54 years/Female	Impaired in two cases, normal in one case	Positive, negative, and rest myoclonus	Therapeutic doses	Drug withdrawal ± haemodialysis	Complete resolution following drug discontinuation
Hussain K et al., (2019) [8]	65 years/Male	End-stage renal disease	Absence status epilepticus	Initiation of therapy (75 mg)	Drug withdrawal	Complete recovery
Park KD et al., (2018) [7]	80 years/Male	Mild transient renal impairment	Negative myoclonus with gait instability, falls, confusion, and memory impairment	Therapeutic initiation (150 mg/day for 2 days)	Pregabalin withdrawal + intravenous lorazepam	Complete recovery

[Table/Fig-3]: Comparison of the present case with previously reported cases of pregabalin-induced myoclonus [4,5,7,8].

recognition, discontinuation of the offending drug, and appropriate supportive treatment can result in rapid and complete neurological recovery. Clinicians should maintain a high-index of suspicion for pregabalin-induced myoclonus in patients presenting with new-onset involuntary movements, particularly following recent initiation or dose escalation of pregabalin, to avoid unnecessary investigations and inappropriate treatment.

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