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CASE REPORT

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Gabapentin-Induced Myoclonus in an Elderly Patient with End-Stage Renal Disease: A Case Report

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Abstract

Gabapentin is frequently prescribed for neuropathic pain and restless leg syndrome, particularly in patients with end-stage renal disease (ESRD). While generally well tolerated, rare adverse effects such as myoclonus can occur, particularly in those with impaired renal clearance. We report the case of a 72-year-old female with ESRD on maintenance hemodialysis who developed generalized myoclonus following a recent increase in her gabapentin dose. Neurological investigations ruled out structural or infectious causes. Her symptoms resolved completely after discontinuation of gabapentin and supportive care. This case highlights the importance of cautious dosing of renally excreted drugs like gabapentin and monitoring for neurotoxic side effects in dialysis patients.

1. Introduction

Gabapentin is a gamma-aminobutyric acid (GABA) analogue commonly used for the treatment of neuropathic pain, partial seizures, and restless leg syndrome (Nwankwo et al., 2024). Its widespread use in patients with chronic kidney disease (CKD) and ESRD is due to its efficacy and favorable safety profile (Raouf et al., 2020)transcending all chronic disease states. Patients with end-stage renal disease (ESRD. However, gabapentin is primarily excreted unchanged via the kidneys, and accumulation in patients with impaired renal function can lead to neurotoxicity, manifesting as dizziness, ataxia, altered mental status, and rarely, myoclonus (Nwankwo et al., 2024; Quintero, 2017).

Myoclonus, defined as sudden, involuntary muscle jerks, can be cortical, subcortical, spinal, or peripheral in origin (Kojovic et al., 2011). Drug-induced myoclonus is an underrecognized phenomenon in ESRD patients, with gabapentin being a documented but infrequent cause (Desai et al., 2019; Rissardo et al., 2025). The mechanism is thought to involve gabapentin-induced changes in inhibitory neurotransmission, which may be potentiated in the setting of reduced clearance. Here, we present a case of gabapentin-induced myoclonus in a hemodialysis-dependent patient, with symptom resolution upon cessation of the drug.

2. Case Report

A 72-year-old female with a history of ESRD secondary to long-standing type 2 diabetes mellitus, hypertension, and diabetic neuropathy presented with involuntary jerking movements of her arms and face for three days. The symptoms began insidiously and progressively worsened, leading to functional impairment and sleep disturbance. She denied headache, fever, visual disturbances, or recent head trauma. There was no recent infection or change in

her dialysis regimen. Notably, she had recently complained of worsening neuropathic pain, for which her gabapentin dose was increased from 100 mg once daily to 300 mg twice daily two weeks prior to presentation (Table 1).

On physical examination, she was alert and oriented, but intermittent facial twitching and upper limb myoclonus were observed. No focal deficits, rigidity, or signs of encephalopathy were noted. She had no asterixis, and her neurological exam was otherwise unremarkable.

Brain MRI was unremarkable. Electroencephalogram (EEG) showed no epileptiform activity. No evidence of infection, metabolic encephalopathy, or structural abnormalities was found. Gabapentin was identified as the likely etiology based on temporal association, symptom pattern, and known renal pharmacokinetics. The medication was discontinued, and conservative measures including hydration and continued dialysis were employed. Over the next 72 hours, the patient experienced complete resolution of myoclonic activity without need for anticonvulsants.

Table 1. Laboratory Investigations at Presentation

Test	Result	Reference Range	Interpretation
WBC	7.1 ×10^3/μL	4–11 ×10^3/μL	Normal
Hemoglobin	10.8 g/dL	11.5–16.0 g/dL	Mild anemia (ESRD-related)
BUN	41 mg/dL	7–25 mg/dL	Elevated (ESRD)
Creatinine	5.3 mg/dL	0.6–1.2 mg/dL	Elevated (ESRD)
Sodium	137 mmol/L	135–145 mmol/L	Normal
Calcium	8.2 mg/dL	8.5–10.5 mg/dL	Mildly low
Magnesium	2.1 mg/dL	1.5–2.5 mg/dL	Normal
Serum ammonia	24 μmol/L	15–45 μmol/L	Normal

3. Discussion

Gabapentin-induced neurotoxicity is a rare but recognized phenomenon, particularly in patients with ESRD (Zand et al., 2010). As gabapentin is renally excreted unchanged, impaired clearance results in drug accumulation and potential neurotoxic manifestations (Lal et al., 2012). Myoclonus, although rare, has been described in case reports and small series in ESRD patients receiving excessive doses (Desai et al., 2019).

According to Ibrahim et al., 2017, even therapeutic doses of gabapentin may lead to toxicity in dialysis patients due to inter-individual variability in drug clearance. A study by Goodman et al. demonstrated that gabapentin is not efficiently removed by hemodialysis, necessitating careful dose adjustment and patient monitoring (Lal et al., 2012). The half-life of gabapentin can be extended up to 132 hours in ESRD without dialysis, compared to 6–8 hours in normal renal function (Koncicki et al., 2015).In our case, the patient tolerated a low dose (100 mg/day) for several months, but escalation to 600 mg/day (300 mg BID) likely exceeded her individual clearance threshold, triggering neurotoxicity. Importantly, this occurred despite normal ammonia, calcium, and magnesium levels, and in the absence of infection or structural brain pathology.

In our case, the patient tolerated a low dose (100 mg/day) for several months, but escalation to 600 mg/day (300 mg BID) likely exceeded her individual clearance threshold, triggering neurotoxicity. Importantly, this occurred despite normal ammonia, calcium, and magnesium levels, and in the absence of infection or structural brain pathology.

Given the increasing use of gabapentin in neuropathy and restless leg syndrome in CKD/ESRD patients, this case underscores the importance of initiating at the lowest possible dose and considering renal-adjusted maintenance schedules. Symptoms such as myoclonus should prompt immediate medication review.

4. Conclusion

This case highlights gabapentin-induced myoclonus as a potential and reversible cause of neurologic symptoms in patients with ESRD. Clinicians should be cautious with dosing in dialysis-dependent patients and remain vigilant for neurotoxicity, even at doses considered therapeutic in the general population. Early recognition and drug withdrawal typically result in full recovery without the need for invasive intervention.

Declarations

Ethics approval statement

No ethical approval was required for the current study as it did not deal with any human or animal samples.

Consent to participate

Not applicable

Consent to publish

Not applicable

Data Availability Statement

The data are available from the corresponding author upon reasonable request

Competing Interests

The authors declare that they have no conflict of interest

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Author contribution

Conceptualization, Data curation, Investigation, Formal analysis, Writing—review and editing: Md.A.R.F. All authors have read and agreed to the published version of the manuscript

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