Received: 16 June 2024 | Revised: 17 August 2024 | Accepted: 14 September 2024

DOI: 10.1002/ccr3.9477

CASE REPORT

Clinical Case Reports Open Access WILEY

Hypocalcemia-induced seizures in a peritoneal dialysis (PD) patient: A case report

Mohammed Taha[1] | **Ayman Azhary**[2,3] | **Abdallah Elssir Ahmed**[3] | **Abubakar Abdelbagi**[4] | **Mohammed Elfatih Hamida**[5] | **Mohammed Elfatih Hussein Ournassеir**[2]

[1]Department of Internal Medicine, University of Gezira, Wad Madani, Sudan

[2]Department of Medical Microbiology, Faculty of Medical Laboratory Sciences, Omdurman Islamic University, Khartoum, Sudan

[3]Molecular Biology Unit, Sirius Training and Research Centre, Khartoum, Sudan

[4]Department of Medical Biochemistry and Biophysics, Umeå University, Umeå, Sweden

[5]Orotta College of Medicine and Health Sciences, Unit of Microbiology, Asmara, Eritrea

Correspondence
Ayman Azhary, Department of Medical Microbiology, Faculty of Medical Laboratory Sciences, Omdurman Islamic University, Khartoum, Sudan.
Email: azhryaymn18@gmail.com

Key Clinical Message

A thorough evaluation is necessary for seizures caused by hypocalcemia, both during the patient's presentation and following their recovery from the postictal seizure episode. This is because the underlying cause of hypocalcemia must be ruled out in order to ensure the best possible clinical outcome from calcium and vitamin D therapy.

Abstract

Patients with multiple systemic issues, including neurological involvement and seizure development, are not uncommon among nephrologists. Both the central and peripheral neural systems can be impacted by kidney disease. The main symptoms are myopathy, cranial or peripheral neuropathy, cognitive impairment, and seizures. A 22-year-old female with an unusual medical history who had been known to have end-stage kidney disease (ESKD) for a year and regularly had CAPD (continuous ambulatory peritoneal dialysis) suffered from two episodes of tonic-colonic seizures 2 weeks apart. On physical examination, symptoms of tongue biting, decreased vision in the left eye, and mild bilateral pulmonary air entry were notable. Upon examination, there was evidence of severe hypocalcemia, hyponatremia, a high renal profile (urea and creatinine), anemia, and a severe vitamin D deficiency. Her peritoneal dialysis (PD) prescription was reviewed, her seizures ceased, and she was released from the hospital after the hypocalcemia was treated with intravenous calcium and high doses of vitamin D. The issue of hypocalcemic seizures should be carefully evaluated both at the presentation and after the patient recovers from the postictal stage. By following this, seizure episodes can be prevented with good success if patients strictly adhere to the medication for which they are responsible.

KEYWORDS

CAPD (continuous ambulatory peritoneal dialysis), end-stage kidney disease (ESKD), hypocalcemia, seizures

1 | INTRODUCTION

The body's organ systems and numerous other organs communicate with the kidneys. When compared to the other organs, the brain might be the one that sufferers of renal illnesses experience the most and most varied effects from.[1] An increased risk of cerebrovascular problems, such as stroke, seizures, white matter illnesses, intracerebral microbleeds, and cognitive decline, is linked to chronic kidney disease (CKD). This has also been reported in patients with mild-to-moderate chronic kidney disease (CKD).[2]

A group of neuronal cells in the brain experiences abnormal, excessive, or simultaneous neuronal activity, resulting in temporary symptoms and signs known as seizures.[3] Normal serum calcium (Ca) concentrations are kept within an extremely small range, which is necessary for both good extracellular and intracellular functions. The maintenance of calcium homeostasis as well as the development and maintenance of bones depend on vitamin D.[4] Decreased renal synthesis of 1, 25-dihydroxyvitamin D and hyperphosphatemia lead to hypocalcemia, which can be quite severe in chronic kidney disease (CKD) patients.[5] A potentially fatal consequence of hypocalcemia is arrhythmia, heart failure, and seizures.[4]

In this communication, we are presenting a case of hypocalcemia-induced seizures in a peritoneal dialysis (PD) patient. We will discuss the clinical and laboratory investigations that were conducted to better understand this case and highlight the importance of an accurate diagnosis and appropriate management strategies.

2 | CASE HISTORY AND PRESENTATION

A 22-year-old female, known for end-stage kidney disease (ESKD) for about 1 year on regular CAPD (continuous ambulatory peritoneal dialysis), presented to Omdurman Military Hospital complaining of two attacks of generalized tonic colonic convulsions 2 weeks apart. She has been a known case of chronic kidney disease (CKD) since she was 6 years old, after she presented with hematuria and generalized body swelling. At that time, her abdominal ultrasound showed small kidneys, and she was admitted for several months but never required dialysis. She did not receive calcium antagonists. She was on furosemide tabs at a dose of 120 mg per day and angiotensin-converting enzyme (ACE) inhibitors with regular follow-up for 12 years. Throughout her illness, the patient sought medical advice from several hospitals and centers. By the time she arrived at our facility, she had established chronic kidney disease (CKD), for which a renal biopsy was not recommended. Chronic glomerulonephritis is supposed to be the cause of the renal disease.

She stopped taking her medications after 12 years, and her follow-up was lost. After seeking medical attention multiple times for recurrent urinary tract infections (UTIs) and weight loss 2 years prior, it was discovered that she had glycosuria with normal blood glucose and HbA1C. She was then referred to the nephrology department for further evaluation. She was informed about her CKD and was given supplements of calcium, vitamin D, and folic acid. When she went for another nephrology opinion 2 months later, she was informed that she was getting close to end-stage kidney disease (ESKD) and was encouraged to get a preemptive kidney transplant.

She traveled to Egypt 3 months later, where she was given the same diagnosis and treatment plan. Following her return from Egypt, she sought a second opinion. This time, the recommendation was for her to begin peritoneal dialysis as a transition to transplantation, which she declined. She began emergency hemodialysis using a temporary right jugular hemodialysis catheter (HD) 2 months later after presenting with uremic symptoms and anemia. Her hemothorax required the placement of a chest tube and an intensive care unit (ICU) hospitalization. Her urine output was 2.9 L per day when she began peritoneal dialysis about a month later, requiring two exchanges of 1 L of the yellow solution (1.36) each day. She was kept on calcium, 1 alpha-calcidol, and erythropoetin, and her condition remained stable for the entire year.

Her illness began approximately 2 weeks before she presented. She experienced a generalized tonic colonic convulsion that lasted for roughly 5 min and was accompanied by tongue biting; there was no loss of sphincteric control, and no anuria, headache, fever, or trauma had preceded it. She sought medical help, was diagnosed with hypoglycemia, underwent a dextrose infusion, and was discharged from home. She experienced a similar type of widespread, tonic colonic convulsion 2 weeks later. She disclaimed having any signs related to cranial nerves, motor, sensory, or sphincteric disturbances, or higher functions. Her urine was normal in amount, color, and frequency. Her systemic review revealed no unusual findings, and she did not exhibit any constitutional symptoms. There was no family history of a similar condition; there is no family history of hypertension (HTN) or diabetes mellitus (DM). There was no significant family history of renal disease. There is no important drug or social history.

3 | DIFFERENTIAL DIAGNOSIS, INVESTIGATIONS, AND TREATMENT

The differential diagnoses of hypocalcemia-induced seizures included primary epilepsy, structural central nervous system (CNS) lesion, and metabolic encephalopathy. Her clinical examinations revealed a Glasgow coma scale (GCS) of 15, oriented to time, place, and person. Vitals: heart rate (HR): 92 beats/min regular, blood pressure (BP): 160/100, respiratory rate (RR): 18 cycles/min, and temperature: 37.1. Mouth: signs of tongue biting. Jugular venous pressure (JVP) was not raised. The neurological exam was clear, apart from decreased visual acuity in the left eye. Chest exam: mildly decreased air entry in both lung bases. Her abdominal ultrasound revealed small kidneys, mild ascites, and bilateral pleural effusion, otherwise unremarkable. Her brain MRI and electroencephalogram (EEG) findings were normal. Her laboratory investigations showed a high renal profile, hyponatremia, normal potassium levels, and severe hypocalcemia (5.72 mg/dL). Unfortunately, her effluent calcium and phosphate were not determined, because it was technically unfeasible in our center, but we tried to overcome that by frequent monitoring of her blood values (Table 1). Urine analysis: on January 19, 2023: specific gravity: 1005, sugar +++, protein +++, blood +, pus 4–6, and RBCs 6–8. CBC: Hb: 8.2 gm, TWBCs: 7.7×10^9/L, PLT: 192/mm^3, CRP: 39.1 mg/dL, LFTs: normal, apart from albumin of 3.1 gm/dl and her RBS of 126 mg/dL. Her serum magnesium was 2.1 mg/dL, PTH level was 281 pg/mL (reference range: 10–55 pg/mL), PO4 was 2.6 mg/dL (reference range: 2.8–4.5 mg/dL), and 25-OH-vitamin D was 6 ng/mL (reference range: 20–40 ng/mL).

Regarding management and treatment, her dialysis prescription was modified to four exchanges per day of 1.36 solution and 4 h of dwell time (with recording of the in and out fluids in addition to the urine output). She received IV phenytoin for 2 days, followed by levetiracetam tables (500 mg), with no further convulsions. She received intravenous calcium gluconate and therapeutic vitamin D orally (50.000 IU weekly for 6 weeks, then reassessment). She was seen by a dentist and was put on amoxicillin, metronidazole, and mouthwash. Furthermore, she was started on amlodipine (10 mg PO) to regulate her blood pressure.

4 | OUTCOME AND FOLLOW-UP

She displayed signs of hypertensive retinopathy and impaired visual acuity in both eyes, coupled with evidence of retinal hemorrhage. For additional care, she was referred to a specialist. The patient's visual disturbance was due to hypertensive retinopathy, and regarding the impact and modification of treatment, we did neurology and ophthalmology consultations, and we focused on controlling her blood pressure in addition to scheduling regular follow-up visits with the mentioned departments. Her hypocalcemia was treated with intravenous calcium gluconate and high doses of vitamin D; her PD prescription was reviewed, her seizures stopped, and she was discharged from the hospital.

5 | DISCUSSION

One well-known but rarely occurring electrolyte imbalance that can cause seizures is hypocalcemia. While there are a few reports of hypocalcemic seizures in children, adults rarely experience them. Supplementing with calcium and vitamin D serves as the cornerstone of care.[6,7]

Parathyroid hormone (PTH) and vitamin D are essential for preserving the body's calcium balance. It has been demonstrated in multiple studies that the prevalence of vitamin D deficiency in the general population is between 50% and 94%.[6] When a patient presents with seizures, hypocalcemia should be promptly detected and treated. When treating such patients, underlying reasons such as vitamin D deficiency, among other problems, must be taken into account.[6]

In this case report, we present a case of hypocalcemia-induced seizures in a 22-year-old female diagnosed with

TABLE 1 Renal profile during the admission period.

Date	Blood urea (RR: 6–24 mg/dL)	Serum creatinine (RR: 0.6–1.1 mg/dL)	S. Na (135-145 mmol/L)	S. K (RR: 3.5–5.5 mmol/L)	S. Ca (RR: 8.5–10.5 mg/dL)	S. phosphate (2.8–4.5 mg/dL)
19/1/2023	128 mg/dL	13.1 mg/dL	NA	NA	5.72 mg/dL	2.6 mg/dL
22/1/2023	133 mg/dL	13 mg/dL	125 mmol/L	3.7 mmol/L	NA	NA
23/1/2023	135 mg/dL	13 mg/dL	118 mmol/L	4.2 mmol/L	NA	NA
24/1/2023	144 mg/dL	11.7 mg/dL	127 mmol/L	3.7 mmol/L	7.2 mg/dL	2.9 mg/dL
25/1/2023	137 mg/dL	11.9 mg/dL	130 mmol/L	3.6 mmol/L	6.9 mg/dL	2.9 mg/dL
26/1/2023	122 mg/dL	12.5 mg/dL	134 mmol/L	3.7 mmol/L	6.8 mg/dL	3.1 mg/dL

end-stage kidney disease (ESKD) characterized by evidence of tongue biting, decreased visual acuity in the left eye, and somewhat diminished air in both lungs, anemia, significant azotemia with a raise in blood urea nitrogen (BUN) and creatinine, hyponatremia, severe hypocalcemia, and severe vitamin D deficiency in Sudan. The patient was successfully managed and treated with intravenous calcium gluconate and vitamin D, leading to cessation of seizures and stabilization of her condition.

In terms of management of anemia, hypocalcemia, and vitamin D deficiency, we optimized our patient's iron status and maintained her on an erythropoiesis-stimulating agent after excluding other causes of anemia or erythropoietin resistance, and concerning hypocalcemia and vitamin D deficiency, we started her on replacement therapy for both in addition to optimizing her peritoneal dialysis and ensuring adequacy. For this patient, the rationale for choosing peritoneal dialysis over hemodialysis is that it was the patient's choice after consulting about the modalities of renal replacement therapy, taking into account that she is young and she prefers more mobility and home-based treatment, in addition to the lesser dietary and fluid restrictions and, above all, the preservation of her residual kidney function. Her electrolyte disturbances are not caused directly by being on peritoneal dialysis.

A steady decline in kidney function is the hallmark of chronic kidney disease (CKD). According to the Kidney Disease Improving Global Outcomes (KDIGO) program, chronic kidney disease (CKD) is defined as a problem with kidney function or structure that lasts longer than 3 months. Individuals with CKD have a greater probability of seizure occurrence. It is estimated that approximately 10% of patients with advanced CKD experience seizures.[8] In advanced chronic kidney disease (CKD) and end-stage renal disease (ESRD), hypocalcemia is frequently observed.[9] Restoring this to the normal range through intravenous administration of calcium gluconate and oral vitamin D is crucial for effective management. Alkaline phosphatase, vitamin D, parathyroid hormone, and phosphate levels should be measured periodically in accordance with the stage of chronic kidney disease (CKD). Regular monitoring is necessary to identify any abnormalities and to take prompt action to avoid complications.

Seizures brought on by hypocalcemia have received a lot of clinical attention. Clinical observation has often shown that hypocalcemia contributes to the onset of seizures and other general excitability processes, including tetany, bronchospasm, and Chvostek's sign.[10] Regarding the management of hypocalcemia, IV Calcium Gluconate (10%) in 10 mL over 10 min is administered rather than calcium chloride because of the decreased risk of extravasation-induced tissue necrosis. Vitamin D supplements taken orally should be started as soon as possible in patients with chronic renal failure and continued even after they are discharged.[11]

For the benefit of physicians, researchers, patients, and their families, a framework for classifying seizures, epilepsies, and epileptic syndromes has been established by the 2017 ILAE classification system. It expands upon recently developed definitions of epilepsy and seizures. The first step in classifying seizures is figuring out whether they are focal or generalized in their early symptoms.[12]

In short, this case report of seizures in an ESKD patient caused by hypocalcemia highlights the importance of timely and accurate diagnosis, appropriate treatment and follow-up, and patient adherence to treatment plan to achieve optimal clinical outcome (i.e., cessation of seizures, as observed in our case) from therapeutic calcium and vitamin D administration. Furthermore, this case emphasizes the significance of monitoring and managing calcium and phosphate levels in peritoneal dialysis patients, especially those with risk factors like vitamin D deficiency and secondary hyperparathyroidism, to prevent severe complications such as seizures.

To the best of our knowledge, there are few published reports of hypocalcemia-induced seizures in ESKD patients. This case report will be valuable in adding knowledge for diagnosing and managing hypocalcemic seizures.

6 | CONCLUSION

Frequent neurological symptoms of hypocalcemia include delirium, tetany, and seizures, indicating a link between hypocalcemia and the emergence of neurological disorders. The majority of reviews recommend starting with 10% intravenous calcium gluconate in 10 mL spread out over 10 min. It is also recommended that adolescents with ESKD who have seizures be closely watched for signs of hypocalcemia and vitamin D deficiency after being discharged from the hospital. This is seen as reasonably effective in preventing seizures, provided that the patient follows their prescribed medication schedule.

AUTHOR CONTRIBUTIONS

Ayman Azhary: Conceptualization; data curation; investigation; writing – original draft. **Mohammed Taha:** Conceptualization; data curation; formal analysis; investigation; validation. **Abdallah Elssir Ahmed:** Conceptualization; data curation; writing – original draft. **Abubakar Abdelbagi:** Conceptualization; data curation; formal analysis. **Mohammed Elfatih Hamida:** Writing – original draft; writing – review and editing. **Mohammed Elfatih Hussein Ournasseir:** Investigation; methodology; writing – review and editing.

FUNDING INFORMATION
None.

CONFLICT OF INTEREST STATEMENT
All authors declare that they have no conflicts of interest.

DATA AVAILABILITY STATEMENT
The data that supports the findings of this study are available in the main manuscript of this article.

CONSENT
Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID
Mohammed Taha https://orcid.org/0009-0006-2730-7469
Ayman Azhary https://orcid.org/0000-0002-2804-1408
Abdallah Elssir Ahmed https://orcid.org/0000-0001-8572-3942
Mohammed Elfatih Hamida https://orcid.org/0000-0002-5337-399X
Mohammed Elfatih Hussein Ournasseir https://orcid.org/0000-0003-2424-9647

REFERENCES
1. Gungor O, Aydin Z, Inci A, Oguz EG, Arici M. Seizures in patients with kidney diseases: a neglected problem? *Nephrol Dial Transplant*. 2023;38(2):291-299. doi:10.1093/ndt/gfab283
2. Chillon JM, Massy ZA, Stengel B. Neurological complications in chronic kidney disease patients. *Nephrol Dial Transplant*. 2016;31(10):1606-1614. doi:10.1093/ndt/gfv315
3. Sarmast ST, Abdullahi AM, Jahan N. Current classification of seizures and epilepsies: scope, limitations and recommendations for future action. *Cureus*. 2020;12(9):e10549. doi:10.7759/cureus.10549
4. Aihara S, Yamada S, Kondo M, et al. Severe hypocalcemia and resulting seizure caused by vitamin D deficiency in an older patient receiving phenytoin: eldecalcitol and Maxacalcitol ointment as potential therapeutic options for hypocalcemia. *Case Rep Nephrol*. 2019;2019:3653276. doi:10.1155/2019/3653276
5. Al-Shebani T, Azeem M, Elhassan E. A case of advanced chronic kidney disease with severe hypocalcemia, how to safely manage and dialyze? *Saudi J Kidney Dis Transpl*. 2019;30(5):1166-1170. doi:10.4103/1319-2442.270275
6. Sagar A, Datta G, Jayaraman M. Vitamin D deficiency presenting as seizures. *Med J Armed Forces India*. 2024:S0377123723002150. doi:10.1016/j.mjafi.2023.11.010
7. Manappallil RG, Krishnan R, Veetil PP, et al. Hypocalcemic seizure due to vitamin D deficiency. *Indian J Crit Care Med*. 2020;24(9):882-884. doi:10.5005/jp-journals-10071-23586
8. Wu HHL, Wang CCY, Chhetri SK, Ponnusamy A, Warwicker P. Seizures and chronic kidney disease: an in-depth review. *Adv Neurol*. 2023;2(2):314. doi:10.36922/an.314
9. O'Neill WC. Targeting serum calcium in chronic kidney disease and end-stage renal disease: is normal too high? *Kidney Int*. 2016;89(1):40-45. doi:10.1016/j.kint.2015.10.001
10. Han P, Trinidad BJ, Shi J. Hypocalcemia-induced seizure: demystifying the calcium paradox. *ASN Neuro*. 2015;7(2):1759091415578050. doi:10.1177/1759091415578050
11. Poh Y. Breakthrough seizure secondary to Hypocalcaemia in an adolescent with chronic kidney failure: a case report. *J Anesth Intensive Care Med*. 2018;8(1):555729-555734. doi:10.19080/JAICM.2018.08.555729
12. Pack AM. Epilepsy overview and revised classification of seizures and epilepsies. *Contin Minneap Minn*. 2019;25(2):306-321. doi:10.1212/CON.0000000000000707

How to cite this article: Taha M, Azhary A, Ahmed AE, Abdelbagi A, Hamida ME, Ournasseir MEH. Hypocalcemia-induced seizures in a peritoneal dialysis (PD) patient: A case report. *Clin Case Rep*. 2024;12:e9477. doi:10.1002/ccr3.9477