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(CASE REPORT)

Refractory convulsive status epilepticus secondary to cefepime-induced neurotoxicity in a patient with chronic kidney disease

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Abstract

Background: Cefepime-induced neurotoxicity is an important adverse drug reaction in patients with impaired or rapidly changing renal function. The initial manifestations are often nonspecific, which may delay recognition until seizures or coma develop.

Case presentation: We report a 61-year-old man with stage 4 chronic kidney disease who was admitted with severe *Pseudomonas aeruginosa* pneumonia. Cefepime was prescribed at 2 g intravenously every 12 hours after a 2-g loading dose and was not reduced when his creatinine clearance declined from approximately 22 to 14 mL/min. On treatment day 4, disorientation and multifocal myoclonus progressed to recurrent generalized tonic-clonic seizures despite lorazepam and a full levetiracetam loading dose. Continuous electroencephalography demonstrated 2-2.5 Hz generalized periodic discharges and recurrent electrographic seizures. Structural neuroimaging and metabolic testing did not identify another sufficient cause. A cefepime concentration obtained 8 hours after the last dose was 52.4 mg/L. Cefepime was stopped, continuous propofol and midazolam were used for seizure control, and two hemodialysis sessions were completed. Electrographic seizures ceased within 18 hours; the patient was extubated at 60 hours and recovered without recurrent seizures during 90 days of follow-up.

Conclusion: Renal dose adjustment at the start of therapy does not protect against later accumulation when kidney function deteriorates. Daily reassessment of renal clearance, prompt review of new neurologic symptoms, early electroencephalography, immediate withdrawal of cefepime, and hemodialysis in severe cases can shorten exposure and support full neurologic recovery.

Keywords: Cefepime; Neurotoxicity; Refractory status epilepticus; Chronic kidney disease; Electroencephalography; Hemodialysis

1. Introduction

Cefepime is a fourth-generation cephalosporin used for severe infections caused by Gram-negative organisms, including *Pseudomonas aeruginosa*. Approximately 80%-85% of an administered dose is eliminated unchanged in urine. As glomerular filtration falls, the elimination half-life lengthens and systemic exposure increases. Cefepime also reduces gamma-aminobutyric acid-mediated inhibition, providing a pharmacologic basis for cortical hyperexcitability and seizures [1].

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The clinical presentation ranges from slowed thinking, inattention, aphasia, agitation, and somnolence to myoclonus, seizures, nonconvulsive status epilepticus, and coma. Recognition is difficult in hospitalized patients because sepsis, uremia, hypoxemia, and delirium can produce similar findings. In a systematic review, renal dysfunction was present in nearly nine of every ten affected patients, and symptoms typically appeared four days after cefepime was started [2]. Neurotoxicity has nevertheless been reported with regimens judged appropriate for renal function, particularly when clearance changes quickly or when serum creatinine provides an imprecise estimate of current filtration [3,4].

Electroencephalography may show diffuse slowing, triphasic-wave morphology, or generalized periodic discharges between 1 and 3 Hz. These findings often fall on the ictal-interictal continuum, so the distinction between toxic-metabolic encephalopathy and nonconvulsive status epilepticus depends on the evolution of the pattern, accompanying clinical signs, and response to antiseizure medication [5,6]. Persistent convulsive activity after an adequate benzodiazepine and a second-line antiseizure drug meets the practical definition of refractory status epilepticus and usually requires intubation, continuous anesthetic therapy, and continuous electroencephalographic monitoring [7,8].

Diagnosis remains clinical. A compatible exposure, recognized risk factors, exclusion of a more convincing alternative, and improvement after withdrawal together establish the causal argument. Cefepime concentrations can strengthen that assessment, but no isolated value reliably separates all toxic from nontoxic exposures [9,10]. In patients with advanced renal impairment and severe neurologic manifestations, hemodialysis can reduce drug exposure more rapidly than supportive care alone [11]. This report describes a documented episode of refractory convulsive status epilepticus associated with cefepime accumulation in a patient with chronic kidney disease and acute renal deterioration.

2. Case presentation

2.1. Clinical history and comorbidities

A 61-year-old man, employed as a merchant and independent in basic and instrumental activities of daily living, was admitted for a severe respiratory infection. His history included type 2 diabetes mellitus for 18 years, hypertension, stage 4 chronic kidney disease attributed to diabetic kidney disease, and anemia of chronic kidney disease. During the preceding six months, serum creatinine had remained between 2.7 and 3.0 mg/dL, corresponding to an estimated glomerular filtration rate of 21-24 mL/min/1.73 m^2. He had no personal or family history of epilepsy, traumatic brain injury, stroke, cognitive decline, or harmful alcohol use.

His usual medications were insulin glargine, amlodipine, carvedilol, calcium carbonate, and weekly erythropoietin. He was not receiving gabapentinoids, tramadol, baclofen, or another drug with a recognized neurotoxic risk in renal impairment. He reported no drug allergies and had not received cefepime previously.

Five days before admission, he developed fever, productive cough with green sputum, progressive dyspnea, poor oral intake, and functional decline. A hospitalization for a complicated urinary tract infection two months earlier increased concern for a resistant Gram-negative pathogen. His family had noticed greater drowsiness during the previous 24 hours, although he remained coherent, recognized relatives, and walked without assistance. There had been no headache, photophobia, vomiting, transient loss of consciousness, or involuntary movements before the respiratory illness.

2.2. Physical examination at admission

At presentation, the patient was somnolent but arousable to voice. He was oriented to person, place, and time and followed two-step commands. Blood pressure was 102/64 mmHg, heart rate 112 beats/min, respiratory rate 28 breaths/min, temperature 38.6 °C, and oxygen saturation 88% on room air. The mucous membranes were mildly dry and capillary refill was 3 seconds. There were no signs of venous congestion. Inspiratory crackles and diminished breath sounds were present at the right lung base; cardiac rhythm was regular without murmurs. The abdomen was soft and nontender, and mild bilateral ankle edema was noted.

The Glasgow Coma Scale score was 14 because eye opening required verbal stimulation. Pupils were equal and reactive, extraocular movements were intact, and no facial asymmetry was observed. Strength was 5/5 in all extremities, deep tendon reflexes were symmetric, and plantar responses were flexor. Neck stiffness, sensory loss, dysmetria, asterixis, and myoclonus were absent. This examination established a neurologic baseline before the later deterioration.

2.3. Initial laboratory and imaging studies

Initial studies showed a leukocyte count of 17.8 × 10^9/L with 89% neutrophils, hemoglobin 9.6 g/dL, platelets 286 × 10^9/L, C-reactive protein 186 mg/L, and procalcitonin 6.8 ng/mL. Serum creatinine was 3.4 mg/dL and blood urea nitrogen was 64 mg/dL. Creatinine clearance calculated with the Cockcroft-Gault equation at a body weight of 75 kg was approximately 22 mL/min. Sodium, corrected calcium, magnesium, glucose, and liver biochemical tests did not reveal an abnormality sufficient to account for the altered alertness. Arterial blood gas analysis while receiving supplemental oxygen showed pH 7.36, $PaCO_2$ 34 mmHg, PaO_2 68 mmHg, and lactate 2.4 mmol/L.

Chest radiography showed right basilar consolidation. Computed tomography of the chest confirmed an air bronchogram and a small nonloculated pleural effusion. Two blood culture sets and a tracheal aspirate were obtained. Severe pneumonia with risk factors for multidrug-resistant Gram-negative infection was suspected, and vancomycin plus cefepime was started. After a 2-g loading dose, cefepime was continued at 2 g intravenously every 12 hours. This dose exceeded the expected regimen for the estimated clearance and remained unchanged while creatinine rose to 4.6 mg/dL, urine output fell to 0.35 mL/kg/h, and calculated creatinine clearance declined to 14 mL/min over the next 48 hours. The findings were interpreted as acute kidney injury superimposed on chronic kidney disease.

Table 1 Clinical and laboratory trends

Parameter	Admission	Day 2	Status epilepticus (day 4)	Post-HD
Leukocytes (×10^9/L)	17.8	14.2	12.6	9.7
Hemoglobin (g/dL)	9.6	9.1	8.8	9.2
Sodium (mmol/L)	136	137	138	139
Potassium (mmol/L)	5.1	5.3	4.9	4.3
Corrected calcium (mg/dL)	8.9	8.7	8.8	9.0
Magnesium (mg/dL)	2.1	2.2	2.3	2.0
Blood urea nitrogen (mg/dL)	64	86	101	54
Creatinine (mg/dL)	3.4	4.6	5.1	3.7
eGFR (mL/min/1.73 m^2)	19	13	11	17
CRP (mg/L)	186	112	68	32
Plasma cefepime (mg/L)	-	-	52.4	17.6

Note: eGFR = estimated glomerular filtration rate; CRP = C-reactive protein; HD = hemodialysis.

2.4. Neurologic deterioration and status epilepticus

The respiratory culture grew *Pseudomonas aeruginosa* susceptible to cefepime, piperacillin-tazobactam, and ciprofloxacin; blood cultures remained negative. Vancomycin was stopped after 48 hours. On the third cefepime treatment day, fever and oxygen requirements were improving, but the family observed delayed responses, poor sustained attention, and verbal perseveration. Early on day 4, action tremor, multifocal myoclonic jerks, and disorientation to time and place appeared. These changes were initially attributed to sepsis-associated delirium and uremia. Haloperidol 1 mg was given without benefit. Two hours later, forced deviation of the head and eyes to the left was followed by a generalized tonic-clonic seizure lasting more than five minutes.

Lorazepam 4 mg was administered intravenously and repeated once because convulsive activity continued. Motor activity stopped briefly but returned seven minutes later. Levetiracetam was loaded intravenously at 60 mg/kg, for a total dose of 4.5 g. A third generalized convulsion followed, and consciousness did not return between episodes. Refractory convulsive status epilepticus was diagnosed. The patient underwent rapid-sequence intubation, received a propofol bolus followed by continuous infusion, and was transferred to the intensive care unit. Recurrent electrographic seizures during initial propofol titration led to the addition of continuous midazolam.

2.5. Diagnostic assessment

Noncontrast head computed tomography showed no hemorrhage, edema, or ischemic lesion. Brain magnetic resonance imaging, obtained after hemodynamic stabilization, demonstrated chronic small-vessel ischemic changes without

diffusion restriction, meningeal enhancement, or another acute abnormality. Repeat testing showed glucose 128 mg/dL, sodium 138 mmol/L, corrected calcium 8.8 mg/dL, magnesium 2.3 mg/dL, and no relevant change in liver biochemical tests. Blood gas analysis showed neither hypercapnia nor severe acidemia. Central nervous system infection was considered less likely because there was no persistent fever, meningismus, bacteremia, immunosuppression, or suggestive imaging finding; an emergent lumbar puncture was therefore not performed.

Continuous electroencephalography revealed a disorganized background with 2-2.5 Hz generalized periodic discharges of triphasic morphology and four generalized-onset electrographic seizures during the first recording hour. Before each event, the discharges increased in frequency and amplitude and were accompanied by subtle facial twitching, supporting an ictal component. The timing of symptoms, cumulative cefepime exposure, progressive renal impairment, preceding myoclonus, and absence of an acute structural lesion made cefepime neurotoxicity the leading diagnosis. A plasma sample obtained eight hours after the final dose measured 52.4 mg/L, above the 36-mg/L concentration with the best discriminatory performance reported in a hospitalized cohort [9]. The Naranjo adverse drug reaction score was 8, consistent with a probable drug-related event.

2.6. Treatment

Cefepime was stopped immediately, without waiting for the concentration result, and was replaced with renal-adjusted piperacillin-tazobactam according to the susceptibility profile. Levetiracetam was continued and adjusted to 500 mg every 12 hours for renal function. Anesthetic infusions were directed toward electrographic seizure control rather than prolonged deep suppression. Propofol was titrated to 40 μg/kg/min and midazolam to 0.15 mg/kg/h with continuous electroencephalographic and hemodynamic monitoring. Phenytoin was not used because of hypotension risk and the response achieved with the selected regimen.

Urgent hemodialysis was recommended because severe neurotoxicity occurred in the setting of oliguria and progressive azotemia. A four-hour treatment was performed with a high-flux dialyzer, blood flow of 300 mL/min, and dialysate flow of 500 mL/min. Substantial ultrafiltration was avoided because respiratory fluid balance was stable. A second session was completed the following day to reduce the possibility of postdialysis rebound. After the first session, cefepime concentration fell to 17.6 mg/L. Seizures stopped, and generalized periodic discharges decreased to less than 1 Hz. Ictal control was achieved 18 hours after cefepime withdrawal. Midazolam was tapered over the next 24 hours, and propofol was stopped at 36 hours without electrographic recurrence.

2.7. Outcome and follow-up

At 48 hours, the patient opened his eyes spontaneously and followed simple commands. He was extubated at 60 hours. Mild cognitive slowing and word-finding difficulty persisted for two days, after which language, attention, immediate memory, and motor strength returned to baseline. Follow-up electroencephalography showed mild diffuse slowing without epileptiform discharges. Pneumonia continued to improve, and he completed ten days of antipseudomonal treatment. At discharge on hospital day 14, serum creatinine was 3.1 mg/dL, urine output had normalized, and no further dialysis was required.

Renal-adjusted levetiracetam was maintained for six weeks as a precaution. At the 30-day visit, the patient had resumed his previous activities and neurologic examination was normal. Levetiracetam was then tapered over two weeks without recurrence. At 90 days, he remained independent, had experienced no further confusion, myoclonus, or seizures, and serum creatinine remained close to his previous baseline.

Table 2 Clinical and therapeutic timeline

Time point	Findings	Clinical decisions
Day 0	Admission for severe pneumonia; stage 4 CKD and early AKI.	Cefepime 2-g loading dose followed by 2 g every 12 h; vancomycin.
Day 2	Creatinine 4.6 mg/dL; estimated clearance 14 mL/min.	Cefepime not readjusted; vancomycin discontinued.
Day 3	Psychomotor slowing, verbal perseveration, and multifocal myoclonus.	Initially attributed to delirium/uremia.

Day 4	Generalized tonic-clonic seizure >5 min with recurrence and no recovery.	Lorazepam 8 mg total, levetiracetam 4.5 g, and intubation.
Days 4-5	EEG: 2-2.5 Hz GPDs and recurrent electrographic seizures.	Propofol plus midazolam; cefepime withdrawal; urgent HD.
Day 5	Seizure cessation; post-HD cefepime 17.6 mg/L.	Second hemodialysis session; gradual anesthetic withdrawal.
Days 6-7	Recovery of consciousness; EEG without seizures.	Extubation at 60 h; renal-adjusted levetiracetam.
Day 14	Kidney function near baseline; no neurologic deficit.	Hospital discharge and outpatient follow-up.
Day 90	No seizure recurrence.	Levetiracetam successfully tapered and discontinued.

Note: CKD = chronic kidney disease; AKI = acute kidney injury; EEG = electroencephalography; GPDs = generalized periodic discharges; HD = hemodialysis.

3. Discussion

The temporal sequence in this patient was characteristic of cefepime neurotoxicity: several days of exposure, worsening renal function, progressive encephalopathy, myoclonus, convulsive and electrographic seizures, and recovery after the drug was withdrawn. The prescription was already excessive for the estimated clearance at admission and became increasingly inappropriate as acute kidney injury evolved. This is a recurrent problem in unstable renal function, because serum creatinine is not at steady state and commonly used equations may overestimate actual drug clearance [12].

The combination of encephalopathy, myoclonus, and seizures is consistent with beta-lactam interference at the GABA-A receptor. Reduced renal clearance prolongs plasma exposure, while inflammation and uremia may increase blood-brain barrier permeability. Clinical manifestations can therefore evolve along a continuum, beginning with impaired attention or aphasia and progressing to periodic discharges, nonconvulsive status epilepticus, or, less often, refractory convulsive status epilepticus [1,2]. In this setting, new myoclonus should not be dismissed as a nonspecific feature of critical illness; it may be the first clinically useful sign of drug accumulation.

Renal assessment also requires more than a single estimated glomerular filtration rate. Laboratory-reported eGFR is normalized to 1.73 m^2 and may not match the method used in antimicrobial dosing recommendations. The Cockcroft-Gault equation becomes less reliable when creatinine is changing rapidly, edema is present, or muscle mass is reduced. For this patient, the fall in estimated clearance from 22 to 14 mL/min, accompanied by oliguria and positive fluid balance, was more informative than any isolated value and should have prompted immediate reconsideration of both dose and interval.

Therapeutic drug monitoring can provide objective support, although availability remains limited. Lau et al. found a 6% incidence of cefepime neurotoxicity and identified 36 mg/L as the trough concentration with the best discriminatory ability [9]. Their subsequent toxicodynamic analysis showed a continuous exposure-response relationship rather than an absolute threshold [10]. The concentration of 52.4 mg/L in this patient was obtained eight hours after the last dose, implying greater exposure earlier in the dosing interval. When cefepime must be continued because no adequate alternative is available, Bayesian dose adjustment and concentration monitoring may help individualize treatment [13].

The differential diagnosis required careful consideration. Uremia and sepsis may both cause somnolence, but neither adequately explained the close relationship to cefepime exposure, recurrent seizures, and a markedly elevated drug concentration. The infection was already improving when neurologic symptoms became prominent. Computed tomography and magnetic resonance imaging excluded an acute cerebrovascular lesion, an important mimic when aphasia, gaze deviation, or focal deficits dominate [14,15]. Normal or near-normal glucose, sodium, calcium, magnesium, arterial blood gas values, and liver tests reduced the probability of another metabolic cause. Rapid improvement after cefepime withdrawal and dialysis provided a clear positive dechallenge.

Table 3 Differential diagnosis of neurologic deterioration

Diagnosis	Supporting findings	Findings reducing likelihood
Sepsis-associated encephalopathy	Severe infection at admission	Infection was improving when neurologic deterioration began; symptoms tracked cefepime exposure and improved after withdrawal.
Uremic encephalopathy	Azotemia and oliguria	May have contributed but does not independently explain recurrent seizures or the markedly elevated cefepime concentration.
Acute cerebrovascular event	Forced gaze deviation and initial focal features	CT and MRI showed no acute lesion; complete neurologic recovery.
Metabolic disturbance	CKD and critical illness	Glucose, sodium, calcium, magnesium, blood gases, and hepatic function did not show a causal abnormality.
Central nervous system infection	Fever and altered consciousness	No meningismus, bacteremia, or suggestive imaging findings; rapid improvement after drug withdrawal and HD.
Cefepime-induced neurotoxicity	Typical latency, renal impairment, myoclonus, GPDs/seizures, and plasma level 52.4 mg/L	Most coherent diagnosis; Naranjo score 8 and recovery after withdrawal and hemodialysis.

Note: CKD = chronic kidney disease; CT = computed tomography; MRI = magnetic resonance imaging; GPDs = generalized periodic discharges; HD = hemodialysis.

Electroencephalographic interpretation deserves separate emphasis. Generalized periodic discharges with triphasic morphology are common in cefepime neurotoxicity, but morphology alone does not determine whether a pattern is ictal. Current terminology weighs frequency, evolution, modifiers, and clinical or electrographic response to medication [5]. The proposed division between cefepime encephalopathy and nonconvulsive status epilepticus may be overly rigid because toxic dysfunction and ictal activity can coexist [16]. In this patient, recurrent generalized convulsions, evolving electrographic seizures, subtle facial twitching, and failure to recover consciousness justified aggressive treatment.

Status epilepticus was refractory because convulsive activity continued after two adequate lorazepam doses and a full levetiracetam load. Contemporary reviews support continuous anesthetic infusion with electroencephalographic monitoring when first- and second-line therapy fail [7,8,17]. Evidence does not establish a universally superior anesthetic. Propofol was selected for rapid control, and midazolam was added when electrographic seizures recurred. Once cefepime had been withdrawn and extracorporeal clearance completed, continuous monitoring allowed the infusions to be reduced without unnecessarily prolonging pharmacologic coma.

Removing the cause was as important as suppressing seizures. Cefepime should be stopped as soon as neurotoxicity is suspected; waiting for a concentration result prolongs cerebral exposure. Hemodialysis is particularly reasonable when severe symptoms occur with advanced renal impairment, high concentrations, or persistent ictal activity. Its low molecular weight, limited protein binding, and relatively small distribution volume make cefepime dialyzable. Delayed dialysis has been associated with prolonged manifestations in end-stage kidney disease [11]. Here, the decline from 52.4 to 17.6 mg/L paralleled electroencephalographic improvement. Although the separate effects of dialysis, anesthetic drugs, and antiseizure medication cannot be quantified, the pharmacokinetic fall and absence of recurrence during anesthetic withdrawal support a clinically meaningful contribution from extracorporeal clearance.

Published reports also caution against relying exclusively on a label of renal dose adjustment. Neurotoxicity has occurred despite regimens considered appropriate [3,4], and systematic reviews include many cases without an obvious dosing error [2,18]. Older age, preexisting cerebral disease, systemic inflammation, hypoalbuminemia, and sudden changes in volume of distribution may increase the unbound fraction or central nervous system exposure. Cases reported outside intensive care units further show that surveillance is relevant on general medical wards as well [19].

The ACORN randomized clinical trial offers broader context. Cefepime and piperacillin-tazobactam did not differ in the primary outcome of acute kidney injury or death, but neurologic dysfunction occurred more often among patients assigned to cefepime [20]. Cefepime remains an essential antimicrobial; the practical implication is not routine

avoidance, but deliberate selection of dose and interval according to current clearance, renal trajectory, indication, and microbiologic susceptibility, followed by de-escalation when possible.

Several preventive steps emerge from this clinical course. Baseline kidney function and the indication for cefepime should be documented before treatment. Dose and interval should be reviewed whenever creatinine, urine output, fluid balance, or kidney replacement modality changes, rather than only when the first prescription is entered. New confusion, aphasia, tremor, or myoclonus between treatment days 2 and 6 warrants immediate medication review and consideration of electroencephalography. Coordination among infectious diseases, pharmacy, nephrology, neurology, and intensive care can shorten delays in diagnosis, drug withdrawal, antiseizure treatment, and extracorporeal clearance.

This report has the limitations expected of a single-patient observation. It cannot estimate incidence or compare treatment strategies. Lumbar puncture was not performed because the clinical and imaging findings did not support central nervous system infection. Drug withdrawal, hemodialysis, anesthetic infusions, and antiseizure therapy occurred in close succession, so the contribution of each intervention cannot be isolated. Even so, the exposure history, elevated cefepime concentration, positive dechallenge, electroencephalographic course, and complete recovery form a coherent causal account.

4. Conclusion

Cefepime neurotoxicity should be considered when a patient develops new confusion, myoclonus, or seizures during treatment, especially when chronic kidney disease is accompanied by acute deterioration in renal function. A dose selected at admission may become excessive within hours. In severe presentations, management should combine immediate cefepime withdrawal, focused exclusion of alternative causes, protocol-based treatment of status epilepticus, continuous electroencephalographic monitoring, and early hemodialysis when drug accumulation is probable. The complete neurologic recovery in this patient shows the value of recognizing the prodromal symptoms before prolonged cerebral exposure leads to lasting injury.

Compliance with ethical standards

Informed consent

Written informed consent was obtained from the patient for publication of this case report and the de-identified clinical information contained in the manuscript. Patient confidentiality was maintained throughout manuscript preparation.

Disclosure of conflict of interest

The authors declare that they have no conflicts of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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