

Internal Medicine
Section

Ionised Hypocalcaemia as a Compounding Factor in Refractory Seizures During Opioid-benzodiazepine Withdrawal: A Case Report

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ABSTRACT

Concurrent opioid and benzodiazepine dependence is common in clinical practice, and abrupt cessation of either agent can independently precipitate withdrawal seizures that are frequently refractory to standard treatment. Ionised hypocalcaemia is a less widely recognised and potentially reversible contributor to seizure threshold lowering in this setting, and can be overlooked when only total serum calcium is measured. Hereby, the authors present a case report of a 28-year-old male with a 6-to-7-year history of polysubstance dependence, involving codeine-containing cough syrup, alprazolam, tobacco, gutkha, and intermittent methamphetamine use, in whom this compounding metabolic factor was identified during combined opioid-benzodiazepine withdrawal. He presented to the Emergency Department with altered sensorium and cluster tonic-clonic seizures approximately 48 hours after abrupt cessation of all substances. On arrival, the Glasgow Coma Scale (GCS) was 7 of 15, and arterial blood gas analysis showed a critically low ionised calcium of 0.56 mmol/L (reference range 1.12 to 1.32 mmol/L), while total serum calcium checked the following day was normal (10.4 mg/dL), illustrating that total calcium can be misleadingly reassuring when ionised calcium is critically low. Six further cluster seizures occurred in the Emergency Department despite prior benzodiazepine loading at a referring hospital, and management required intravenous sodium valproate, lorazepam, naloxone for diagnostic clarification of possible opioid co-ingestion, thiamine, intravenous calcium gluconate correction, and intensive-care-level monitoring with neurology and psychiatry co-management. Seizure frequency decreased progressively, with improvement in sensorium and correction of ionised calcium, and the patient was discharged on a structured benzodiazepine taper together with naltrexone for relapse prevention. The present case illustrates that combined opioid and benzodiazepine withdrawal can produce seizures resistant to initial benzodiazepine monotherapy, and that severe ionised hypocalcaemia should be actively sought and corrected as a potential contributing factor in withdrawal-associated seizures, particularly when radiological and cerebrospinal fluid evaluation are not feasible and other causes cannot be fully excluded.

Keywords: Emergency service, Hospital, Hypocalcaemia, Opioid-related disorders, Status epilepticus, Substance withdrawal syndrome

CASE REPORT

A 28-year-old unmarried male, educated to the 9th standard and working as an automobile mechanic, presented to the Emergency Department with the chief complaint of altered sensorium and recurrent seizures. The case was registered as a Medicolegal Case (MLC) at presentation, which is routine Institutional practice for patients presenting with altered sensorium in the context of substance use. Given the patient's impaired decision-making capacity on arrival (GCS 7 of 15), surrogate consent was obtained from an accompanying family member at the time of registration; the patient's own written consent was obtained after recovery of capacity. No family history of neurological or psychiatric illness was reported. Owing to acute impairment of sensorium at presentation, the patient's own account was unavailable during initial assessment. Collateral history was obtained from accompanying family members, who described progressive behavioural deterioration before seeking care. The family expressed concern regarding the patient's dependence and indicated willingness to support engagement with a de-addiction programme.

The patient's chief complaint on presentation was altered sensorium with recurrent seizures. He had abruptly stopped all substances, including codeine-containing cough syrup and alprazolam, on 1st February 2026 (Day 0). Approximately 48 hours later, on 3rd February 2026, he developed altered sensorium, abnormal upper- and lower-limb movements, tremors, restlessness, agitation, and

irrelevant speech, with redness of the eyes and decreased sleep, followed by multiple seizure episodes with upward eye rolling, tongue biting, and bluish discolouration of the tongue. He was taken to a local hospital that day, where he received intravenous lorazepam 1 mg, oral oxcarbazepine, buprenorphine 0.4 mg, and levetiracetam; despite this, he had 15 to 20 seizure episodes and was referred onward the same day, arriving at the study hospital Emergency Department at approximately 11:00 on 3rd February 2026. Six further cluster seizure episodes occurred between approximately 11:00 and 13:30 that day, a pattern consistent with serial seizures approaching convulsive status epilepticus. This interval of approximately 48 hours between last substance use and seizure onset is consistent with the recognised 24-to-72-hour window for benzodiazepine withdrawal seizures [Table/Fig-1].

Timepoint	Key events and findings
Day 0-1 February 2026	Last substance use; abrupt cessation of codeine-containing cough syrup and alprazolam, with no supervised taper.
Day 2-3 February 2026, morning	Onset of altered sensorium, abnormal limb movements, tremors, agitation, and irrelevant speech, followed by seizures; taken to a local hospital and given i.v. lorazepam, oxcarbazepine, buprenorphine, and levetiracetam, with 15-20 seizures despite this; referred onward the same day.
3 rd February 2026, 11:00	Arrival at the study hospital Emergency Department. Glasgow Coma Scale (GCS) 7/15. Arterial blood gas: pH 7.35, ionised calcium 0.56 mmol/L (critical).

3 February 2026, 11:00-13:30	Six cluster tonic-clonic seizures. i.v. sodium valproate, lorazepam, naloxone, thiamine, and calcium gluconate administered.
3 February 2026, evening	Neurology and psychiatry co-management commenced; intensive-care-level monitoring; chlordiazepoxide taper started.
4-5 February 2026 (Days 2-3)	Progressive improvement; no further cluster seizures after Day 2; sensorium improved to GCS 14/15 by Day 3; ionised calcium corrected, with total serum calcium 10.4 mg/dL (normal) on recheck.
6 February 2026	Discharged on tablet clobazam, chlordiazepoxide, cyproheptadine, naltrexone, lorazepam as needed, and calcium.
14 February 2026	Outpatient follow-up: patient clinically stable, good improvement, engaged with de-addiction programme.

[Table/Fig-1]: Clinical and management timeline.
GCS: Glasgow coma scale; i.v.: intravenous.

Substance Use History

The patient had a 6 to 7-year history of polysubstance dependence, summarised in [Table/Fig-2].

Substance	Duration	Quantity per day	Last used	Craving	Eye-opener use
Codeine-containing cough syrup (opioid)	6-8 years	300-1000 mL	1 February 2026 (Day 0)	Yes	Yes
Alprazolam (benzodiazepine)	3-4 years	0.5 mg tablets, 10 tablets/day (approximately 5 mg/day)	1 February 2026 (Day 0)	Yes	Yes
Tobacco (cigarettes)	6 years	1 packet	2 days before admission	-	-
Gutkha (smokeless tobacco)	4 years	1 packet	2-3 days before admission	-	-
Methamphetamine	3-4 years	1-2 times/month	6 months before admission	-	-
Nitrazepam (intermittent substitute)	Intermittent	Variable, Friday nights	When codeine unavailable	-	-

[Table/Fig-2]: Substance use history of the patient.

Past and Psychosocial History

There was no known history of hypertension, diabetes mellitus, ischaemic heart disease, stroke, chronic obstructive pulmonary disease, prior seizures, or psychiatric illness, and no prior hospitalisation for substance use. The patient denied headache, photophobia, vomiting, or altered sensorium. Family history was unremarkable. Nutritional status was not formally scored using a validated tool; collateral history from the family suggested markedly reduced dietary intake over six to seven years, which is clinically relevant to the ionised hypocalcaemia described below.

Physical Examination

On presentation, the patient was afebrile, with a heart rate of 100-110 beats/min, blood pressure of 118/70 mmHg, respiratory rate of 16 breaths/min, and oxygen saturation of 94% on room air. The GCS score was 7/15 (E2V1M4). Pupils were equal, round, and reactive to light bilaterally. Motor examination revealed normal muscle power (5/5) in all four limbs, with brisk deep tendon reflexes and bilaterally flexor plantar responses. There were no focal neurological deficits. Examination for meningeal irritation revealed no neck rigidity, and both Kernig's and Brudzinski's signs were negative. The abdomen was soft and non tender, with no organomegaly. Cardiovascular, respiratory, and abdominal examinations were otherwise unremarkable.

Provisional and Differential Diagnosis

Based on the clinical presentation, the provisional diagnosis was withdrawal-associated refractory seizures secondary to combined opioid and benzodiazepine dependence, with severe ionised hypocalcaemia as a likely contributing factor. Differential diagnoses considered at presentation included new-onset epilepsy, central nervous system infection (including encephalitis and meningitis), hepatic encephalopathy, and metabolic or toxic encephalopathy due to co-ingestion of other substances.

Investigations

Investigations were performed on 3rd February 2026. The most clinically significant finding was severely low ionised calcium on arterial blood gas analysis (0.56 mmol/L; reference range 1.12 to 1.32 mmol/L), which is half the lower limit of normal (0.56 vs 1.12 mmol/L), alongside a normal total serum calcium of 10.4 mg/dL, illustrating that ionised calcium can be critically low despite a reassuring total calcium result. Haemoglobin was normal throughout, at 15.1 g/dL on admission and 14 g/dL on Day 3. An admission packed cell volume of 33% was discordant with this normal haemoglobin; a repeat packed cell volume on Day 3 was 46.8%, consistent with the Day 3 haemoglobin, and the admission figure is presumed to reflect haemodilution during initial fluid resuscitation or a transcription artefact. Computed tomography of the brain was not performed; the managing neurologist diagnosed withdrawal-related seizures on clinical grounds, and magnetic resonance imaging was deferred because of acute agitation. Full results are presented in [Table/Fig-3]. Serum albumin, vitamin D, and parathyroid hormone were not measured, so a nutritional or endocrine contribution to the hypocalcaemia could not be formally assessed; this is acknowledged as a limitation.

Emergency Management

Resuscitation: Airway patency was confirmed with spontaneous respiration. Supplemental oxygen was administered by face mask,

Investigation	Result	Reference range*	Interpretation
Haemoglobin	15.1 g/dL	13.5-17.5 g/dL	Normal
Total leucocyte count	14,890/mm ³	4,000-11,000/mm ³	Elevated, consistent with a stress leucocytosis (post-ictal state and sympathetic activation)
Packed Cell Volume (PCV)	33% (admission); 46.8% (repeat, Day 3)	40-54%	The admission value of 33% was discordant with a normal haemoglobin (15.1 g/dL); the repeat PCV of 46.8% on Day 3 is consistent with the Day 3 haemoglobin (14 g/dL) and a normal haematological picture, and the admission figure is presumed to reflect haemodilution during initial resuscitation or a transcription artefact
Prothrombin time/partial thromboplastin time	15/40.2 seconds	11-13/25-35 seconds	Partial thromboplastin time mildly prolonged; monitor for coagulopathy
Sodium/Potassium	136/3.9 mEq/L	135-145/3.5-5.0 mEq/L	Normal
Blood urea / Creatinine	22/0.7 mg/dL	7-20/0.6-1.2 mg/dL	Urea mildly elevated with normal creatinine, a pre-renal pattern
SGOT/SGPT	30/32 IU/L	<40/<40 IU/L	Normal liver enzymes
Total bilirubin	1.1 mg/dL	0.1-1.2 mg/dL	Normal
HIV/HBsAg	Non reactive	Non reactive	Negative

C-reactive protein	1.46 mg/L	<5 mg/L	Normal; makes a primary infective aetiology less likely, though not excluded, as cerebrospinal fluid analysis was not performed
Serum magnesium	2.0 mg/dL	1.7-2.2 mg/dL	Normal
Creatine kinase-MB (CPK-MB)	38.4 U/L	<25 U/L	Mildly elevated, consistent with muscle injury from repeated tonic-clonic seizures
Total serum calcium (repeat, Day 2)	10.4 mg/dL	8.5-10.5 mg/dL	Normal - in contrast to the critically low ionised calcium, illustrating that total calcium can be reassuring despite severe ionised hypocalcaemia
Chest X-ray	Clear	-	No acute cardiopulmonary abnormality
Lung ultrasound	Normal	-	No evidence of pulmonary oedema or consolidation
Arterial blood gas - pH	7.35	7.35-7.45	Low-normal
Arterial blood gas pCO ₂ /pO ₂	35/51 mmHg	35-45 / >80 mmHg	Hypoxaemia on room air
Arterial blood gas - ionised calcium	0.56 mmol/L	1.12-1.32 mmol/L	Severely low, half the lower limit of normal; regarded as a potential contributing factor to seizure threshold lowering
Arterial blood gas - Glucose	240 mg/dL	70-110 mg/dL	Stress hyperglycaemia
Arterial blood gas - Bicarbonate	19.3 mmol/L	22-26 mmol/L	Mildly low, a mild metabolic acidosis component
Arterial blood gas - Lactate	3.2 mmol/L	0.5-2.2 mmol/L	Elevated, consistent with post-ictal lactic acidosis or tissue hypoperfusion
Repeat ionised calcium	Not rechecked	1.12-1.32 mmol/L	Not repeated, as the patient had improved clinically by the second day
Serum alprazolam level	Not performed	Therapeutic range 10-50 ng/mL	Not available; high-performance liquid chromatography or immunoassay infrastructure was not available
Magnetic Resonance Imaging (MRI) brain	Deferred	-	Deferred owing to acute agitation
Computed-Tomography (CT) brain	Not performed	-	Withdrawal-related seizures were diagnosed clinically by the managing neurologist without cross-sectional imaging; noted as a limitation
Electrocardiography	No acute changes	Normal sinus rhythm	No ischaemic or arrhythmic changes

[Table/Fig-3]: Laboratory investigations.

*Reference ranges shown are the reporting laboratory's own values.

SGPT: Serum glutamic pyruvic transaminase; SGOT: Serum glutamic oxaloacetic transaminase; HIV: Human immunodeficiency virus; HBsA: Hepatitis B surface antigen

Pharmacological Management

The pharmacological management strategy, with clinical rationale for each agent, is presented in [Table/Fig-4].

Drug	Dose	Route	Frequency	Clinical rationale
Sodium valproate (injection)	500 mg	i.v.	Twice daily	Second-line anti-epileptic used after benzodiazepine loading proved insufficient; acts through sodium-channel stabilisation, GABA-transaminase inhibition, and NMDA-receptor modulation.
Lorazepam (injection)	1-3 mg	i.v./IM	As needed for seizure	First-line GABA agonist for acute seizure termination; cross-tolerant with alprazolam, providing withdrawal bridging.
Chlordiazepoxide (tablet)	25 mg	Oral	Three times daily, tapering schedule	Long-acting benzodiazepine used for a structured withdrawal taper.
Clobazam (tablet)	10 mg	Oral	Twice daily	GABA-A modulator used as anti-epileptic maintenance and adjunct withdrawal cover.
Naloxone (injection)	0.4 mg	i.v.	One dose	Administered for diagnostic clarification of possible opioid co-ingestion or mixed overdose, given the severity of altered sensorium on arrival. Its use in a withdrawal-predominant picture carries a recognised risk of precipitating or worsening withdrawal, which was accepted at the time in the interest of diagnostic certainty.
Thiamine with 25% dextrose (injection)	8 ampoules; 100 mL	i.v.	Twice to three times daily	Thiamine administered before glucose to reduce the risk of precipitating Wernicke encephalopathy in a malnourished patient.
Ceftriaxone (injection)	1 g	i.v.	Once daily	Empirical antibacterial cover given altered sensorium and leucocytosis, pending clinical exclusion of infection.
Calcium gluconate (injection)	1 ampoule (10 mL), diluted in 100 mL of 5% dextrose	i.v.	Single infusion over 20 minutes	Correction of critical ionised hypocalcaemia (0.56 mmol/L). Repeat total serum calcium the following day was 10.4 mg/dL (normal).
Ibuprofen (tablet)	400 mg	Oral	Three times daily	Analgesia for withdrawal-related myalgia, used with caution given mildly elevated urea and prolonged partial thromboplastin time; short course, with renal function monitored.
Cyproheptadine (tablet)	4 mg	Oral	Twice daily	Appetite stimulation and mild sedation for sleep fragmentation.

[Table/Fig-4]: Pharmacological management strategy.

i.v.: intravenous; IM: intramuscular

Specialist Referrals

Neurology was consulted in view of the cluster seizure burden. The team added valproate and clobazam and diagnosed withdrawal-related seizures clinically, without cross-sectional brain imaging. Psychiatry confirmed a diagnosis of polysubstance dependence, recommended intensive-care-level admission with a structured chlordiazepoxide taper, and advised referral to a de-addiction programme with initiation of naltrexone at discharge.

Clinical Outcome and Follow-up

Following combined management, seizure frequency decreased progressively, sensorium improved, and ionised calcium corrected following intravenous supplementation. The patient was discharged on 6 February 2026 on the following regimen: tablet cefixime-clavulanate 200 mg twice daily, tablet clobazam 10 mg twice daily, tablet chlorthalidone 25 mg three times daily, tablet ibuprofen 400 mg as needed, tablet cyproheptadine 4 mg twice daily, tablet naltrexone 50 mg once daily for relapse prevention, tablet lorazepam 1 mg as needed, tablet calcium twice daily, and syrup lactulose-based laxative 15 mL at bedtime. At discharge, the patient did not consent to enrolment in the de-addiction programme; at outpatient follow-up on 14 February 2026, he agreed to engage with the programme, and was clinically stable and doing well. No adverse events related to pharmacological management were documented during the in-hospital or follow-up period.

DISCUSSION

Polysubstance dependence, defined as the concurrent use of two or more psychoactive substances with associated physiological dependence, represents a growing emergency medicine burden in India and across South Asia. The 2019 National Survey on Extent and Pattern of Substance Use in India, conducted by the National Drug Dependence Treatment Centre at the All India Institute of Medical Sciences, estimated that approximately six million individuals, or about 0.55% of the population aged 10 to 75 years, have an opioid use disorder in India [1]. No validated severity-scoring tool for polydrug withdrawal triage currently exists in the Indian emergency medicine context. Benzodiazepine withdrawal, particularly from short-acting, high-potency agents such as alprazolam, is mediated by GABA-A receptor downregulation with compensatory glutamatergic excitation, and abrupt cessation can produce life-threatening seizures within 24 to 72 hours [2,3]; the approximately 48-hour interval observed in this patient is consistent with this window. Concurrent opioid withdrawal generates noradrenergic hyperactivity through disinhibition of the locus coeruleus, contributing to agitation and a lowered seizure threshold [4,5]. A targeted literature check did not identify a published case report describing ionised hypocalcaemia as a compounding factor specifically in combined opioid-benzodiazepine withdrawal seizures; this was not an exhaustive systematic search, and the observation is presented cautiously on that basis.

Given the presentation with altered sensorium, new-onset seizures, and leucocytosis, several differential diagnoses were considered. New-onset epilepsy was considered unlikely given the close temporal relationship between substance cessation and symptom onset, with no prior seizure history. Infectious encephalitis was considered, given the leucocytosis and altered behaviour; a normal C-reactive protein (CRP) level, clear chest X-ray, normal lung ultrasound, absence of fever, and the compelling temporal association with withdrawal made this less likely, but it was not excluded, as cerebrospinal fluid analysis and cross-sectional brain imaging were not performed, which is acknowledged as a limitation. Neuroleptic malignant syndrome and serotonin syndrome were considered unlikely given the absence of relevant drug exposures. Hepatic encephalopathy was considered unlikely given normal bilirubin and liver enzymes. Primary hypoglycaemia was excluded by documented stress hyperglycaemia.

Ionised Hypocalcaemia as a Compounding Factor

The recorded ionised calcium of 0.56 mmol/L is half the lower limit of normal, a degree of hypocalcaemia that can independently contribute to tetany and seizures [6], whereas total serum calcium checked the following day was normal at 10.4 mg/dL. This dissociation is the central clinical teaching point of this report: total serum calcium can be reassuring even when ionised calcium is critically low, and arterial-blood-gas-derived ionised calcium

measurement is therefore essential when evaluating seizures in substance-dependent patients [6,7]. Severe ionised hypocalcaemia is also independently associated with adverse outcomes in critically ill patients more broadly [8]. The aetiology of the hypocalcaemia is presumed to be multifactorial, most plausibly related to chronic nutritional deficiency associated with long-standing substance dependence; the arterial pH of 7.35 does not support a significant contribution from respiratory alkalosis. Repeat ionised calcium was not rechecked once the patient had improved clinically, which is acknowledged as a limitation.

Management Rationale

The pharmacological approach was adapted to the presumed concurrent withdrawal mechanisms. Sodium valproate, used after benzodiazepine loading proved insufficient, addresses withdrawal-associated hyperexcitability through mechanisms complementary to GABA agonism, broadly consistent with the rationale for adjunctive anticonvulsant pharmacotherapy described in the alcohol-withdrawal literature, though this evidence base has not been established specifically for combined opioid-benzodiazepine withdrawal [9]. Chlorthalidone was used for the benzodiazepine taper to provide smoother pharmacokinetic coverage than shorter-acting agents [10]. Naloxone was administered for diagnostic clarification of possible opioid co-ingestion given the severity of altered sensorium on arrival, with the recognised risk that it may precipitate or worsen withdrawal in an opioid-dependent patient [5]; this trade-off was accepted at the time in the interest of diagnostic certainty and is presented transparently here rather than omitted. Intravenous calcium gluconate corrected the critical ionised hypocalcaemia [6,7], and naltrexone was initiated at discharge as part of a structured relapse-prevention and de-addiction linkage plan.

This case highlights several practice gaps with research implications. First, no validated polysubstance withdrawal severity score currently exists for Indian or other low- and middle-income-country Emergency Departments; existing instruments such as the Clinical Opiate Withdrawal Scale (COWS) and Clinical Institute Withdrawal Assessment for Alcohol Scale (CIWA-Ar) address single-substance withdrawal only [11,12]. Second, ionised calcium is not routinely measured in patients presenting with substance withdrawal, despite being readily actionable where arterial blood gas analysis is available, and total calcium alone may be falsely reassuring, as illustrated in this case. Third, serum alprazolam and codeine levels were unavailable owing to the absence of high-performance liquid chromatography or immunoassay infrastructure. Fourth, this patient initially declined enrolment in the de-addiction programme at discharge despite pharmacological relapse prevention with naltrexone being continued, and only agreed to engage at outpatient follow-up; this illustrates that linkage between emergency management and sustained addiction treatment often requires more than a single point of referral, and remains an important target for service development, consistent with broader guidance on structured withdrawal management [13].

Strengths of this report include detailed multi-system documentation, a coherent dated clinical timeline, and demonstration that total serum calcium can be normal despite critical ionised hypocalcaemia. Limitations include the inability to quantify serum alprazolam or codeine levels; cerebrospinal fluid analysis and cross-sectional brain imaging were not performed, so infectious encephalitis is considered less likely rather than formally excluded; vitamin D and Parathyroid Hormone (PTH) were not measured; repeat ionised calcium was not rechecked after clinical improvement; the COWS [11] could not be formally scored owing to altered sensorium at presentation, and a validated benzodiazepine-specific withdrawal scale was not administered; the revised CIWA-Ar [12], being alcohol-specific, was not applicable, and this absence of a benzodiazepine-specific instrument is identified as a practice gap; the account is a retrospective synthesis from clinical records; and, as a single case,

it does not permit generalisation. The literature check supporting the discussion of this compounding interaction was targeted rather than a full systematic search.

CONCLUSION(S)

The present case illustrates that concurrent opioid and benzodiazepine withdrawal can produce cluster seizures resistant to initial benzodiazepine monotherapy, occurring within the recognised 24-to-72-hour withdrawal window, and requiring multimodal pharmacotherapy and specialist co-management. Severe ionised hypocalcaemia (0.56 mmol/L) is identified as a potential compounding factor, directly actionable through arterial-blood-gas-derived ionised calcium measurement and intravenous correction, and this case demonstrates that a normal total serum calcium does not exclude critical ionised hypocalcaemia. Naloxone, used here for diagnostic clarification, thiamine before glucose, a structured benzodiazepine taper, and pharmacological relapse prevention with naltrexone, together with a willingness to re-engage patients with de-addiction services even after initially declining enrolment at discharge, are relevant components of management in this population.

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