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Research

CASE REPORT: GENERALISED TONIC-CLONIC SEIZURE 2⁰ SEVERE HYPONATREMIA

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Check for updates	Abstract
Published on: 22 Nov 2025	A 26-year-old man was admitted complaining of two days of abrupt, jerky limb movements, a three-to-four-minute period of unconsciousness, tongue biting, and mouth foam. Despite not receiving regular antiepileptic medication, the
Published by: Futuristic Publications	patient had experienced a seizure episode a year prior. Upon inspection, the patient had minor cyanosis during convulsions and was sleepy but arousable. Hyponatremia (Na+ 118 mmol/L) and mild metabolic acidosis on ABG tests
2025 All rights reserved.	were found in laboratory examinations, indicating electrolyte imbalance-induced seizures. The generalized tonic-clonic epilepsy caused by severe hyponatremia was confirmed by the generalized spike and wave pattern on the EEG.
Creative Commons Attribution 4.0 International License.	Keywords: Generalized tonic-clonic seizure, Hyponatremia, Electrolyte imbalance, Metabolic acidosis, Epilepsy, Generalized spike-and-wave pattern, Electroencephalography (EEG), Cyanosis, Postictal drowsiness.

INTRODUCTION

One of the most striking manifestations of neurological dysfunction is generalized tonic-clonic seizures (GTCS), which are marked by convulsive movements, a sudden loss of consciousness, and generalized muscle rigidity [1]. Although epilepsy is a prevalent cause, metabolic problems, especially electrolyte imbalances, can sometimes trigger seizures in people who have never had one before [2]. One of the most common electrolyte abnormalities seen in clinical practice is hyponatremia, which is defined as a blood sodium content below 135 mmol/L [3]. Cerebral edema brought on by severe hyponatremia (usually less than 120 mmol/L) can cause neurological symptoms such disorientation, altered sensorium, and seizures. Seizures due to hyponatremia are often generalized

and can be the first or only presenting feature of the underlying electrolyte disturbance [4]. Recognizing this association is crucial, as management differs fundamentally from that of primary seizure disorders; prompt correction of sodium imbalance can rapidly resolve neurological symptoms and prevent recurrence [5].

Here, we present a case of a patient who developed a generalized tonic-clonic seizure secondary to severe hyponatremia, highlighting the diagnostic approach, pathophysiological basis, and importance of timely electrolyte correction.

CASE PRESENTATION

A 26 yrs old male patient presented to emergency department with chief complaints of suddenly jerky limb movement, loss of conscious for 3-4 minutes, tongue bite and fronthing mouth and post ictal confusion and headache. He take alcohol occasionally and he had a history 1 episode of seizure 1 year back but not on regular medication

PHYSICAL AND SYSTEMIC EXAMINATION

He was presented with drowsy but arousable presence of mild cyanosis during seizure

	DAY 1	DAY 2	DAY 3	DAY 4
Temperature (F)	99.1	98.7	98.4	98.2
Blood pressure (mmHg)	160/100	148/90	130/90	120/80
Pulse (bpm)	128	110	96	88
CVS	Tachycardia	S1S2+	S1S2+	S1S2+
Respiratory system	BAE+	BAE+	BAE+	BAE+
P/A	SOFT&NT	SOFT&NT	SOFT&NT	SOFT&NT

LAB INVESTIGATIONS

HAEMATOLOGICAL PATTERNS

Parameters	Results
Hb[13-17] g/dl	14.2
RBC[4.0-5.5] millions/cumm	4.9
WBC[4000-10000] cells/cumm	7,800
Lymphocytes [15-30]%	26
Eosinophils [1-6]%	2
Monocytes [2-10]%	3
ESR [0-10]mm 1st hour	8
Platelets[1-4] lakhs/cum	4.2
PCV[35-40]%	41

ELECTROLYTES

Parameters	Results
Na [136-146]mmol/l	118*
K[3.5-5.0]mmol/l	3.8
Cl [98-107]mmol/l	92

OTHER INVESTIGATIONS

ABG ANALYSIS - Metabolic acidosis

EEG (electroencephalography)

IMPRESSION; Generalised spike & wave pattern tonic clonic epilepsy

TREATMENT CHART

S.NO	TRADE NAME	GENERIC NAME	DOSE	ROA	FRQ	CLASS	INDICATION
1	INJ.LORAZEPAM	lorazepam	4mg	IV	STAT	Benzodiazep ine	First-line emergency management of active generalized tonic-clonic seizure (status epilepticus) due to its rapid anticonvulsant action.
2	INJ.MIDAZOLAM	midazolam	2mg/hr	IV	CONTI NUE	Benzodiazep ine	Sedation and control of recurrent seizures / status epilepticus.
3	INJ.PHENYTOIN	phenytoin sodium	100mg	IV	TID	Hydantoin anticonvulsa nt	Prevents further episodes of generalized tonic-clonic seizures. Used when seizure is due to electrolyte imbalance like hyponatremia.
4	TAB.LEVIPIL	levetiracetam	500mg	РО	BD	SV2A modulator antiepileptic	Long-term seizure prophylaxis in generalized

							tonic-clonic epilepsy.
5	IV 3%NACL	Hypertonic saline	100ml	IV	4HRS	Electrolyte / Hypertonic saline	Primary treatment for severe symptomatic hyponatremia (<120 mEq/L)
6	INJ.PANTOP	pamtoprazole	40mg	IV	OD	Proton pump inhibitor	Prevents gastritis/ulcer due to IV medications and stress response.
7	TAB.FOLITAS	Folic acid	5mg	PO	OD	Vitamin (B-complex)	Prevention of folate deficiency due to chronic antiepileptic therapy (phenytoin affects folate metabolism)

DISCUSSION

Severe hyponatremia can lead to generalized tonic-clonic seizures, as illustrated by a case of a 26-year-old man with serum sodium levels of 118 mEq/L. His seizure symptoms included sudden jerky movements and post-ictal confusion, with EEG results indicating electrolyte-induced seizures rather than primary epilepsy. The rapid drop in sodium caused cerebral Edema, raising the seizure threshold. Treatment with hypertonic saline was initiated immediately, ensuring sodium correction did not exceed 8 mEq/L within 24 hours. Concurrently, acute seizures were managed with IV lorazepam and midazolam, followed by oral levetiracetam for long-term control. Patient education emphasized adherence to medications, hydration, and avoidance of alcohol. This case highlights the importance of identifying metabolic triggers in new-onset seizures to avoid inappropriate treatment.

CONCLUSION

This case emphasizes how crucial it is to identify severe hyponatremia as a treatable cause of generalized tonic-clonic seizures. Complete neurological recovery without complications was achieved through early detection, controlled correction of serum sodium levels, and timely administration of suitable antiepileptic drugs.

In order to prevent misdiagnosis and needless long-term antiepileptic therapy, the case highlights the necessity of requiring electrolyte evaluation in all patients presenting with new-onset seizures. To stop recurrence, patient education about lifestyle changes, medication adherence, and electrolyte imbalance monitoring is still crucial.

In metabolic seizure emergencies, prompt interprofessional intervention greatly improves outcomes.

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