



Case Report: Hypomagnesemia Presenting as Altered Sensorium and Delayed-Onset Atrial Fibrillation in a Patient with Multiple Comorbidities

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Abstract

Introduction:-

Hypomagnesemia is an under recognized electrolyte disorder that can present with neurological symptoms and cardiac arrhythmias. Elderly patients with diabetes, chronic kidney disease (CKD), and polypharmacy are particularly vulnerable. A 78-year-old male with type 2 diabetes (on irregular oral hypoglycemic agents), dyslipidemia, osteoarthritis on NSAIDs, and CKD presented with altered sensorium (GCS:- E4V3M5). No trauma, seizure, or focal deficit was noted. Differential diagnoses included tropical fever, meningitis, and uremic encephalopathy, all of which were excluded. Initial investigations revealed severe hypomagnesemia (0.6 mg/dL) with otherwise unremarkable electrolytes and stable renal function. On Day 2, the patient developed new-onset atrial fibrillation (AF). He was managed with intravenous magnesium sulfate and amiodarone infusion. Serum magnesium normalized to 1.9 mg/dL by Day 7, coinciding with resolution of altered sensorium and rhythm stabilization.

Discussion:-

Magnesium plays a crucial role in neuronal excitability and cardiac electrophysiology. Severe deficiency may mimic metabolic or infectious encephalopathy and predispose to arrhythmias, including AF. Hypomagnesemia is common in CKD and contributes to higher cardiovascular mortality and delirium due to neurological manifestation. Timely recognition and correction are essential, as seen in our patient who showed rapid neurological and cardiac recovery after magnesium repletion.

Conclusion:-

Hypomagnesemia should be considered in elderly patients presenting with altered mental status or new arrhythmias, with CKD and polypharmacy. Early detection and treatment can lead to dramatic improvement.

Keywords: Hypomagnesemia, Atrial fibrillation, Altered sensorium, Chronic kidney disease

Introduction

Hypomagnesemia is an often-overlooked electrolyte disturbance with neurological and cardiovascular implications. In elderly patients with chronic comorbidities, polypharmacy, and renal impairment, the risk of magnesium deficiency is higher. We present a case of a 78-year-old male who initially presented with altered sensorium and subsequently developed atrial fibrillation (AF), both attributable to hypomagnesemia.

Case Report:-

A 78-year-old male, a known case of type 2 diabetes mellitus (on irregular oral hypoglycemic agents), dyslipidemia, osteoarthritis (on regular NSAIDs), and chronic kidney disease (CKD) on medical management, was admitted with altered sensorium and decreased responsiveness for one day.

On admission, his Glasgow Coma Scale (GCS) was E4V3M5. No history of seizures, trauma, or focal neurological deficit was present. Vitals were stable, and systemic examination revealed reduced sensorium without focal deficits.

Differentials considered at presentation included tropical fever, meningitis, and uremic encephalopathy. Investigations showed:

1. Lumbar puncture: Normal, excluding meningitis.
2. Renal function tests: Stable, not suggestive of uremic encephalopathy.
3. Infective markers and tropical fever panel: Negative.
4. MRI Brain :- Nil significant findings.

Laboratory Investigations

Parameter	Day 1	Day 7	Reference Range
Blood Urea	58 mg/dL	77 mg/dL	10–50 mg/dL
Serum Creatinine	1.7 mg/dL	1.7 mg/dL	0.6–1.2 mg/dL
Sodium	139 mmol/L	137 mmol/L	135–145 mmol/L
Potassium	4.6 mmol/L	4.2 mmol/L	3.5–5.0 mmol/L
Calcium (total)	8.0 mg/dL	8.4 mg/dL	8.5–10.5 mg/dL
Phosphorus	3.3 mg/dL	4.4 mg/dL	2.5–4.5 mg/dL
Magnesium	0.6 mg/dL	1.9 mg/dL	1.7–2.2 mg/dL

On day 2 of admission, the patient developed new-onset atrial fibrillation with an irregularly irregular pulse. ECG confirmed AF with rapid ventricular response.

Management included:

1. Intravenous magnesium sulfate (MgSO_4) replacement,

2. Amiodarone bolus and infusion for rhythm stabilization,
3. Supportive care and close monitoring.

A psychiatry consultation was obtained for suspected delirium; delirium secondary to infection was considered but excluded.

Following correction of hypomagnesemia, the patient showed drastic improvement in sensorium and

restoration of rhythm stability, correlating with magnesium normalization to 1.9 mg/dL by Day 7.

Discussion:-

Magnesium is an essential electrolyte involved in numerous biochemical processes that regulate cellular function, nerve conduction, and other vital physiological activities. Hypomagnesemia refers to a disturbance characterized by a serum magnesium level below 1.46 mg/dL. The condition is usually asymptomatic until magnesium levels drop below 1.2 mg/dL (0.5 mmol/L).

Magnesium is essential for neuronal excitability, neurotransmitter release, and cardiac conduction. Severe hypomagnesemia can present with encephalopathy-like symptoms and predispose to arrhythmias such as AF(1,2). Other manifestations are tremors, tetany including positive Trousseau and Chvostek signs, Choreoathetosis, Seizures, Vertical nystagmus, Delirium, Depression, Psychosis, ECG changes including widening of the QRS complex, peaked T waves (with mild to moderate deficiency), prolongation of the PR interval, and diminution of the T wave (with severe deficiency) and Atrial fibrillation and ventricular premature beats, Increased risk of digoxin toxicity by inhibiting Na-K-ATPase and depleting intracellular potassium.

In this patient, multiple comorbidities (diabetes, CKD, chronic NSAID use) likely contributed to magnesium loss. Hypomagnesemia is particularly common in CKD and is associated with higher cardiovascular morbidity and mortality(3,4). Neurological manifestations, including delirium and altered sensorium, have also been documented in hypomagnesemic states(5).

The development of AF on Day 2 further complicated the clinical picture. Low serum magnesium is an independent predictor of AF in community and hospitalized cohorts (6,7). Importantly, intravenous magnesium has been shown to aid both in rhythm stabilization and as adjunctive therapy in AF management (8)

This case highlights the importance of routinely checking magnesium levels in elderly patients with altered sensorium or unexplained arrhythmias, especially in those with CKD and polypharmacy.

Conclusion

Hypomagnesemia can masquerade as infectious or metabolic encephalopathy and can precipitate atrial fibrillation. Prompt recognition and correction not only reverse neurological symptoms but also stabilize cardiac rhythm. This case underscores the need for vigilance in elderly patients with chronic comorbidities.

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