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Severe Hypomagnesaemia with Refractory Hypokalaemia, Bradyarrhythmia, and Normal Anion Gap Metabolic Acidosis: A Case of Suspected Proximal Renal Tubular Dysfunction

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Abstract

Severe hypomagnesaemia is an important but frequently under-recognised electrolyte disturbance that may result in refractory hypokalaemia, metabolic abnormalities, and clinically significant cardiac arrhythmias. We report the case of an 85-year-old woman who presented with profound hypomagnesaemia, hypokalaemia, bradyarrhythmia, and normal anion gap metabolic acidosis. Electrocardiography demonstrated atrial fibrillation with a slow ventricular response and QT prolongation. Biochemical findings raised suspicion for renal tubular acidosis, and the presence of trace glycosuria suggested a possible proximal tubular process, including Type 2 renal tubular acidosis or partial Fanconi syndrome. The patient was treated with intravenous and oral magnesium, potassium supplementation, and sodium bicarbonate therapy, with subsequent clinical and biochemical improvement. This case highlights the critical role of magnesium in potassium homeostasis and cardiac electrophysiology and underscores the importance of considering renal tubular disorders in patients with normal anion gap metabolic acidosis and electrolyte wasting.

Keywords: Severe hypomagnesaemia; Electrolyte; Hypokalaemia

Background

Hypomagnesaemia is frequently overlooked in clinical practice despite its central role in electrolyte homeostasis and cardiac stability. Magnesium is essential for intracellular potassium regulation and modulates myocardial ion channels. Severe magnesium deficiency may lead to refractory hypokalaemia, conduction abnormalities, QT prolongation, and increased risk of malignant arrhythmias [1,2]. Normal anion gap metabolic acidosis in the presence of hypokalaemia should prompt evaluation for renal tubular acidosis. Proximal renal tubular acidosis may occur in isolation or as part of a broader proximal tubular disorder such as Fanconi syndrome [3,4]. In elderly patients, these conditions may be multifactorial and frequently under-recognised.

Case Presentation

An 85-year-old woman was admitted for evaluation of significant electrolyte abnormalities identified during routine testing. She reported generalised weakness, fatigue, and reduced oral intake. There was no history of diarrhoea, vomiting, or other gastrointestinal losses. On examination, she was haemodynamically stable. However, during early admission, she developed marked bradycardia, with heart rates recorded between 30 and 40 beats per minute. She did not report syncope or chest pain but was considered at high risk for arrhythmic complications.

Investigations

Initial laboratory investigations demonstrated severe electrolyte abnormalities. Serum magnesium was markedly reduced, ranging from 0.30 to 0.50 mmol/L. Serum potassium ranged from 3.0 to 3.3 mmol/L. Serum bicarbonate was reduced at 17–20 mmol/L,

and the anion gap was normal. Serum sodium ranged from 134 to 136 mmol/L. Renal function was relatively preserved, with an estimated glomerular filtration rate of approximately 50–60 mL/min/1.73 m². Serum lactate was normal. Venous blood gas analysis demonstrated a pH of 7.27, pCO_2 of 48 mmHg, bicarbonate of 22 mmol/L, and base excess of −5 mmol/L, consistent with metabolic acidosis. Venous blood gas demonstrating acidemia with reduced bicarbonate and negative base excess consistent with metabolic acidosis (Tables 1-3).

Electrocardiography demonstrated atrial fibrillation with a slow ventricular response. Subsequent tracings showed QT prolongation, with a corrected QT interval of approximately 512 milliseconds (Figure 1). A prior transthoracic echocardiogram demonstrated atrial fibrillation, mildly reduced left ventricular systolic function (LVEF 51%), regional wall motion abnormalities, and moderate biatrial enlargement. Right ventricular function was preserved. Urinalysis demonstrated a urine pH of 6, no proteinuria, and trace glycosuria. There was no evidence of infection. The presence of glycosuria in the absence of significant hyperglycaemia raised suspicion for proximal tubular dysfunction (Figure 2).



Figure 1: *ECG – Atrial fibrillation with slow ventricular response.*

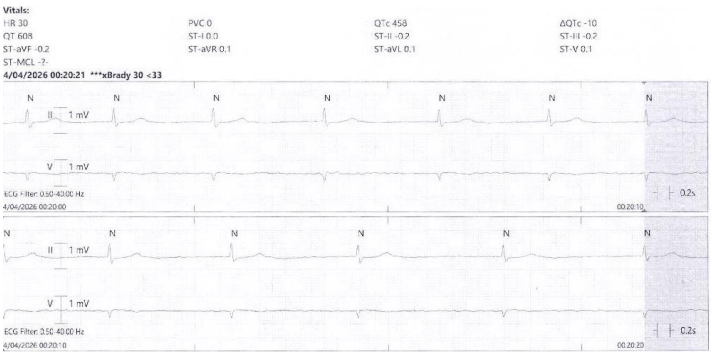


Figure 2: *Telemetry – QT prolongation.*

Differential Diagnosis

The differential diagnosis included proximal (Type 2) renal tubular acidosis, distal (Type 1) renal tubular acidosis, renal magnesium wasting syndromes, drug-induced tubular dysfunction, and gastrointestinal bicarbonate loss. The coexistence of normal anion gap metabolic acidosis, hypokalaemia, severe hypomagnesaemia, and trace glycosuria strongly suggested a renal tubular process. The presence of glycosuria supported a proximal tubular defect, including possible Type 2 renal tubular acidosis or partial Fanconi syndrome. Distal renal tubular acidosis was considered less likely in the absence of persistently alkaline urine or nephrolithiasis. Gastrointestinal bicarbonate loss was considered unlikely given the absence of relevant clinical features.

Diagnostic Approach

Diagnostic Algorithm for patient with Metabolic acidosis

Step 1: Confirm metabolic acidosis with low pH and low bicarbonate
Step 2: Calculate anion gap → normal
Step 3: Assess potassium → hypokalaemia
Step 4: Differentiate renal vs gastrointestinal causes using urine anion gap
• Negative → GI bicarbonate loss
• Positive → Renal cause (RTA)
Step 5: Measure urine pH
• >5.5 suggests distal RTA
• <5.5 suggests proximal RTA after steady state
Step 6: Evaluate for proximal tubular dysfunction
• Glycosuria
• Phosphaturia
• Uric acid wasting
Step 7: Assess urinary magnesium losses
Conclusion: Hypokalaemia + NAGMA + Mg wasting + glycosuria
Findings in this case are most consistent with proximal tubular dysfunction (possible Type 2 RTA / Fanconi)

Treatment

The patient was treated with intravenous magnesium replacement followed by oral supplementation. Potassium replacement was administered concurrently. Sodium bicarbonate therapy was initiated to correct metabolic acidosis. Continuous cardiac monitoring was maintained due to the risk of bradyarrhythmia and ventricular arrhythmias associated with electrolyte abnormalities and QT prolongation. A medication review was undertaken to exclude drug-induced causes.

Outcome and follow-up

Following treatment, there was progressive improvement in serum magnesium and potassium levels. The patient's heart rate improved, with resolution of bradycardia. Acid–base status stabilised with bicarbonate therapy. Clinically, she reported

improvement in symptoms, including reduced fatigue and improved appetite. Further outpatient evaluation was planned to clarify the underlying tubular disorder. This included urine electrolyte studies, urine anion gap, fractional excretion of bicarbonate, and a Fanconi screen.

***Table 1*:** *Laboratory Trends During Admission.*

Parameter	7 Apr 2026	9 Apr 2026	12 Apr 2026	15 Apr 2026	Reference Range
Magnesium (mmol/L)	0.30	0.55	0.53	0.69	0.7–1.0
Potassium (mmol/L)	3.0–3.2	3.6	4.7	4.5	3.5–5.0
Sodium (mmol/L)	134–136	139	136	136	135–145
Bicarbonate (mmol/L)	17–20	22	23	27	22–28
Creatinine (µmol/L)	~80	74	69	73	60–110
eGFR (mL/min/1.73 m²)	50–60	~65	70	65	>60

Table 2*: Venous Blood Gas Trends.*

Date	pH	pCO_2	HCO_3^-	Base Excess	Lactate
7 Apr	7.27	48	22	-5	1.5
9 Apr	7.29	41	20	-7	0.9
12 Apr	7.37	42	24	-1.1	2.5
15 Apr	7.38	48	28	2.5	1.3

Table 3*. Comparison of Renal Tubular Acidosis Subtypes.*

Feature	Type 1 (Distal RTA)	Type 2 (Proximal RTA)	Type 4 RTA
Primary defect	Impaired H^+ secretion	Impaired HCO_3^- reabsorption	Aldosterone deficiency/resistance
Serum potassium	Low	Low	High
Urine pH	>5.5 (persistently)	Variable (<5.5 after steady state)	<5.5
Anion gap	Normal	Normal	Normal
Bicarbonate loss	Mild	Significant	Mild
Nephrolithiasis	Common	Rare	Rare
Fanconi features	Absent	May be present	Absent
Magnesium wasting	Uncommon	Possible	Rare
Likelihood in this case	Less likely	Most likely	Unlikely

Discussion

This case highlights the complex interplay between magnesium deficiency, potassium homeostasis, renal tubular function, and cardiac electrophysiology. Magnesium deficiency leads to increased renal potassium wasting and refractory hypokalaemia by affecting distal tubular potassium channels [2]. Consequently, potassium replacement alone is often ineffective until magnesium is corrected. Hypomagnesaemia also has significant cardiac effects. It predisposes to bradyarrhythmias, QT prolongation, and ventricular arrhythmias [5]. In this case, the presence of underlying structural heart disease likely increased susceptibility to these electrophysiological disturbances. The presence of normal anion gap metabolic acidosis with preserved renal function suggests renal tubular acidosis [3]. The finding of trace glycosuria raises suspicion for proximal tubular dysfunction, as glycosuria in the absence of hyperglycaemia indicates impaired proximal tubular reabsorption [4,6]. When considered alongside electrolyte wasting, this supports a diagnosis of proximal tubular dysfunction, including possible Type 2 renal tubular acidosis or partial Fanconi syndrome. However, definitive classification requires further investigations, including urine anion gap, urine pH during acidosis, and bicarbonate handling studies. In the absence of these data, the diagnosis should be described as suspected proximal tubular dysfunction.

Conclusion

This case describes severe hypomagnesaemia with refractory hypokalaemia, bradyarrhythmia, and normal anion gap metabolic acidosis in an elderly woman. The findings are most consistent with renal tubular dysfunction, likely involving a proximal tubular process such as possible Type 2 renal tubular acidosis or partial Fanconi syndrome. Early recognition and correction of magnesium deficiency were essential in preventing potentially life-threatening complications.

Learning points

- Severe hypomagnesaemia is a key cause of refractory hypokalaemia.
- Magnesium deficiency can lead to bradyarrhythmia and QT prolongation.
- Normal anion gap metabolic acidosis should prompt evaluation for renal tubular acidosis.
- Trace glycosuria may indicate proximal tubular dysfunction.
- Definitive classification requires targeted urinary investigations.

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