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# Fluctuating hypokalemia with late-onset hypomagnesemia and evolving acid–base disturbance: a case report

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**Case Report**

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## Abstract

I report a 20-year-old female with hypovolemic shock and profound hypokalemia (1.7 mmol/L). Initial labs mimicked distal renal tubular acidosis, but low urinary magnesium (1.2 mg/dL) confirmed gastrointestinal losses. Refractory potassium levels stabilized only after magnesium correction. This case highlights the importance of serial monitoring in complex electrolyte disturbances.


## Key Clinical Message

Hypokalemia may follow a fluctuating course rather than linear correction. Late-onset hypomagnesemia can impair potassium stabilization, and evolving acid–base disturbances highlight the importance of serial monitoring.

## Introduction

Hypokalemia is a common electrolyte abnormality resulting from gastrointestinal losses, renal wasting, or intracellular shifts.[1,6,7] Severe hypokalemia may lead to cardiac arrhythmias and neuromuscular dysfunction.[1,7] Hypomagnesemia is a recognized contributor to refractory hypokalemia due to impaired renal potassium conservation.[3,5,8] This case is notable for the temporal evolution of electrolyte disturbances and acid–base status, illustrating the interplay between hypokalemia, hypomagnesemia, and physiological compensation.

## Case Presentation

A 20-year-old female presented on 21 March 2026 with persistent vomiting, generalized weakness, and near syncope. She reported prolonged constipation for approximately 15 days and a recent febrile illness treated with oral cefuroxime.

On admission, she was hypotensive (80/40 mmHg) and tachycardic, consistent with hypovolemic shock. Initial laboratory evaluation revealed severe hypokalemia (1.7 mmol/L) and metabolic acidosis.

**Arterial blood gas (21 March 2026):**
pH 7.405, $PCO_2$ 16.1 mmHg, $HCO_3^-$ 10.1 mmol/L — consistent with **primary metabolic acidosis with respiratory compensation**.

Serial electrolyte monitoring demonstrated a fluctuating potassium course (Table 1). Serum potassium improved initially but declined again on 24 March before stabilizing following magnesium correction.

Hypomagnesemia (1.3 mg/dL) was identified on 24 March and corrected to 2.3 mg/dL on 25 March, after which potassium levels stabilized.

**Repeat arterial blood gas (26 March 2026):**
pH 7.524, $PCO_2$ 17.9 mmHg, $HCO_3^-$ 14.4 mmol/L — demonstrating **alkalemia with persistent metabolic**

**acidosis and superimposed respiratory alkalosis**, consistent with an evolving mixed acid–base disorder.

Hemoglobin declined from 12.5 g/dL on admission to 8.6 g/dL on 26 March, consistent with hemodilution and systemic illness.

**Table 1.** Serial laboratory values

| Date & Time | $Na^+$ (mmol/L) | $K^+$ (mmol/L) | $HCO_3^-$ (mmol/L) | Mg (mg/dL) | Hb (g/dL) |
|---|---|---|---|---|---|
| 21.03 | 142 | 1.7 | 9 | — | 12.5 |
| 22.03 (Morning) | 144 | 1.7 | 10 | — | 11.1 |
| 22.03 (Evening) | 148 | 2.1 | 13 | — | — |
| 23.03 (Morning) | 143 | 2.7 | 12 | — | 9.9 |
| 23.03 (Evening) | 141 | 3.3 | 14 | — | — |
| 24.03 (Morning) | 141 | 2.9 | 11 | — | — |
| 24.03 (Evening) | 140 | 3.4 | 15 | 1.3 | — |
| 25.03 | 141 | 3.2 | 20 | 2.3 | — |
| 26.03 | — | — | 14.4 | — | 8.6 |

Abbreviations: $Na^+$, sodium; $K^+$, potassium; $HCO_3^-$, bicarbonate; Mg, magnesium; Hb, hemoglobin; —, not available.

# Discussion

This case demonstrates a dynamic electrolyte disorder characterized by severe hypokalemia with a fluctuating course. The transient decline in potassium following initial correction suggests ongoing gastrointestinal losses and intracellular redistribution.[6,7,8]

Hypomagnesemia emerged during the later phase and coincided with difficulty in maintaining potassium stability, underscoring its role in potassium homeostasis rather than the primary etiology.[3,5,8]

The acid–base profile evolved significantly during hospitalization. Initial arterial blood gas analysis demonstrated metabolic acidosis with appropriate respiratory compensation.[9,10,12] Subsequent analysis revealed alkalemia with persistently low bicarbonate and further reduction in $PCO_2$, indicating a mixed acid–base disorder with superimposed respiratory alkalosis.[9,12] This evolution likely reflects physiological adaptation during recovery, including hyperventilation and changing metabolic demands.

Distal renal tubular acidosis (Type 1 RTA) was initially considered; however, subsequent findings supported gastrointestinal loss as the primary etiology. Low urinary magnesium (1.2 mg/dL) despite

hypomagnesemia indicated appropriate renal conservation, arguing against renal wasting. The initial positive urinary anion gap was likely misleading due to excretion of unmeasured organic anions, such as ketoacids from starvation and vomiting.[3,8,9,10,12]

The gradual decline in hemoglobin is consistent with hemodilution and systemic inflammation rather than acute blood loss.

# Conclusion

Severe hypokalemia may exhibit a non-linear correction pattern. Late-onset hypomagnesemia can impair potassium stabilization. Recognition of evolving electrolyte and acid–base disturbances is essential for effective management.

# Declarations

### Consent

Written informed consent was obtained from the patient.

### Conflict of Interest

None declared.


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### AI Statement

AI tools were used for language editing and formatting assistance. All clinical interpretation remains the responsibility of the authors.

### Author Contributions

Khandaker Abdul Sadib contributed to all aspects of the work, including data collection, analysis, manuscript drafting, and final approval.

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## Figures

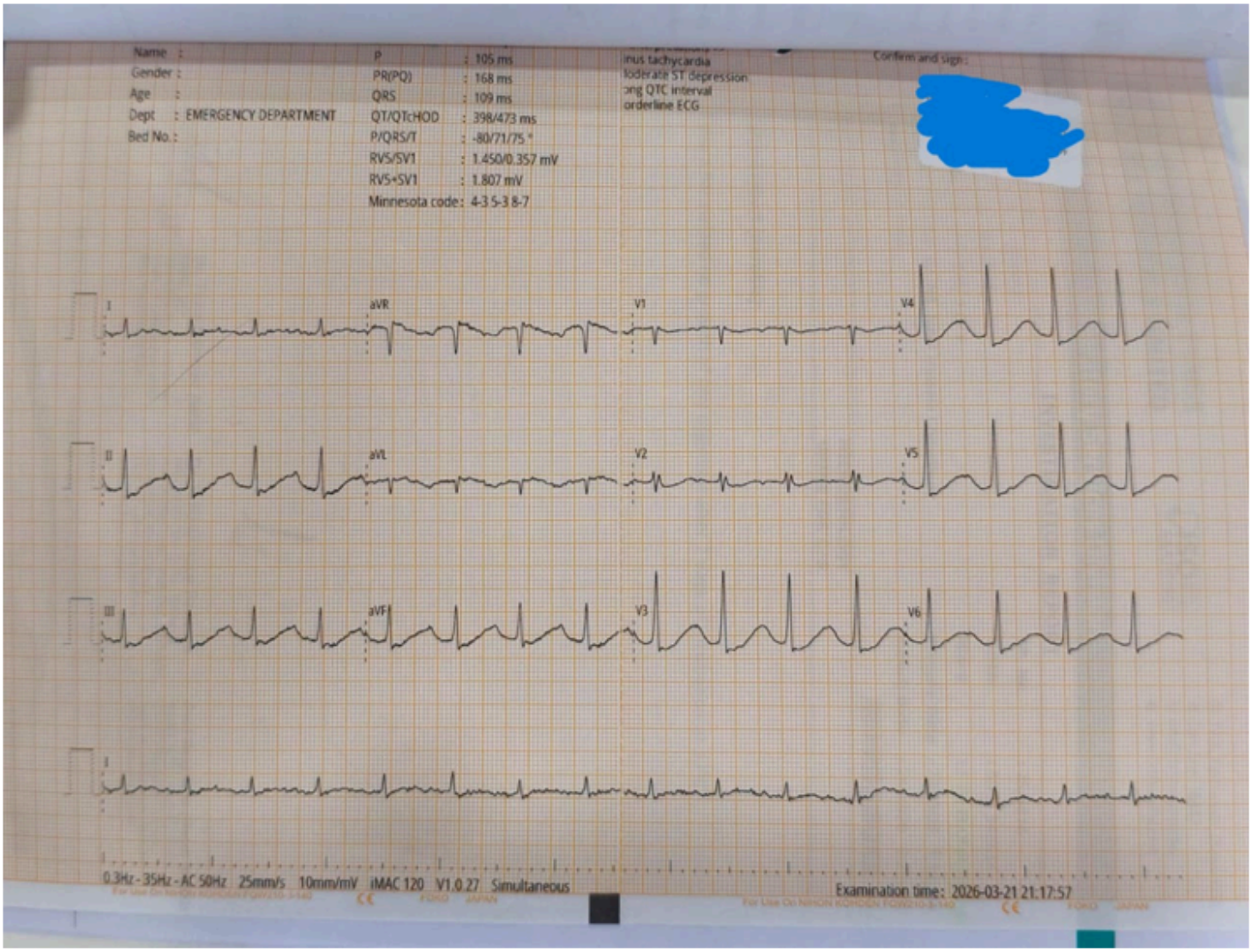


**Figure 1**

Admission electrocardiogram

Electrocardiogram obtained on admission demonstrating sinus tachycardia, ST-segment depression, and QTc prolongation (~473 ms), as shown in Figure 1. These findings are consistent with severe hypokalemia and reflect increased arrhythmogenic risk.