CASE REPORT

Rare case of refractory hypokalaemia in a patient with acute myelomonocytic leukaemia (AML M4): A case report

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Abstract

A 39-year-old man, who was recently diagnosed with Acute Myelomonocytic Leukaemia (AML M4 subtype), presented at the Emergency Department of Ayub Teaching Hospital, Abbottabad, with all the common symptoms of AML, including anaemia, bleeding, and generalised weakness. What makes this case report interesting is the presence of severe refractory hypokalaemia. He was admitted to the Medical D unit of Ayub Teaching Hospital, Abbottabad, on January 14, 2024. His serum potassium remained low at 2.4mmol/L, (n=3.5-5.1 mmol/L) despite aggressive potassium replacement. Urinary electrolyte analysis showed renal potassium wasting, likely due to activation of the renin-angiotensin-aldosterone system (RAAS). Elevated serum renin levels supported this mechanism. In addition, the patient also had acute kidney injury and electrolyte abnormalities potentially caused by excessive lysozyme secretion by monocytes, as described in a prior case report. Refractory hypokalaemia in AML M4 is rare and has a poor prognosis. Early recognition and management of hypokalaemia is crucial to prevent complications and improve patient outcomes. This case highlights the importance of monitoring electrolytes and the need for further research on ideal management protocols for refractory hypokalaemia in AML M4.

Keywords: Acute Myelomonocytic Leukaemia, Acute kidney injury, Renin-angiotensin-aldosterone system, Severe refractory hypokalaemia, Urinary electrolyte analysis.

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Introduction

Acute Myeloid Leukaemia (AML) is a neoplasia of the bone marrow, characterised by the rapid proliferation of the bone marrow cells, particularly of the myeloid lineage.[1] As a result of bone marrow failure and ineffective red blood cell production, patients with AML often present with a variety of signs and symptoms such as anaemia, excessive bleeding, generalised weakness, fatigue, shortness of breath, and easy bruising. In addition, the onset of these symptoms is fairly rapid, often presenting within days to weeks.[2]

Hypokalaemia, or low serum potassium level is a relatively common electrolyte abnormality particularly seen in patients with AML.[3] Hypokalaemia can occur as a result of various mechanisms, including gastrointestinal losses, activation of the renin-angiotensin-aldosterone system (RAAS), or direct renal potassium wasting.[3] It can result in life-threatening complications that can include various systems of the body, including (but not limited to) the cardiovascular, musculoskeletal, and respiratory systems.

In this case report, we demonstrate a rare case of a 39-year-old man with a recently diagnosed Acute Myelocytic Leukaemia of the M4 subtype (Acute Myelomonocytic Leukaemia), presenting with severe refractory hypokalaemia in addition to other vague symptoms commonly associated with AML (high-grade fever, generalised body weakness, muscle cramps, abdominal pain, and constipation) for about eight days. This report aims to highlight the importance of recognising and managing hypokalaemia in patients with AML at its earliest. Additionally, we hope to contribute to the limited literature on refractory hypokalaemia in patients with the M4 subtype of AML.

Case Report

A 39-year-old man with a history of hypertension for the last four years, presented to the Emergency Department (ED) of Ayub Teaching Hospital, Abbottabad, on January 14, 2024, with an eight-day history of high-grade fever, generalised body weakness, muscle cramps, abdominal pain and constipation. He had been diagnosed as a case of Acute Myeloid Leukaemia (AML) five months earlier with a bone marrow biopsy, which showed 77% blast cells, of which 50% had monocytoid morphology, indicating a diagnosis of AML M4 subtype (Table 1).

The patient had undergone a total of three chemotherapy sessions with the last session carried out 20 days prior to his admission. Following his last chemotherapy session

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(which included Doxorubicin and Cytosar) he had intermittent fever spikes (up to 102^{O}F) with rigors and chills, and had developed multiple boils on his lower abdomen, indicating a possible infection. In addition, he also reported that he had difficulty performing his routine tasks since last week due to extreme fatigue and body aches.

On physical examination, the patient's vital signs were notable for a blood pressure of 110/80 mm of Hg, a pulse rate of 104 beats per minute, and a temperature of 102^{O}F. He appeared pale, with significant abdominal examination findings, including multiple boils on his lower abdomen (largest measuring 3×3cm), and tenderness at the left hypochondrium reaching up to the umbilicus. In addition, the spleen was palpable at three finger breadths below the costal margin, liver span measuring 13cm, and decreased bowel sounds on auscultation. Neurological examination revealed a power of 4/5 in the lower limbs. The rest of the systemic examination was unremarkable.

Table-1: Laboratory Results.

Test	Result	Reference value
RBC Count	3.37x10^{12}/L	4.7-6.1
TLC	278x10^{3}/Ul	4-11
Platelets	36x10^{3}/Ul	150-400

RBC=Red Blood Cell TLC= Total Leucocyte Count

Table-2: Peripheral Blood Film Findings.

Finding	Description
RBC	Normocytic Normochromic
WBC	N10%, L 3%, Myelocytes 10% , Blasts 77% (50% Blasts have Monocytoid Morphology)
Platelets	Low on Film
Reticulocytes	0.1%
Conclusion	Acute Leukaemia Morphologically suggestive of Acute Myelomonocytic Leukaemia

RBC=Red Blood Cells WBC= White Blood Cells

Table-3: Serum and Urine Chemistry Studies.

Test	Result	Reference value
TLC	126×10^{3}/uL	4-11
HB	6.8g/dl	11.5-17.5
MCV	89.6fL	76-96
CRP	156mg/L	<5.0
LDH	414U/L	<248
Sodium	139mmol/L	135-150
Potassium	2.5mmol/L	3.5-5.1
Chloride	104mmol/L	96-106
Urine Analysis		
Glucose	NILL	
Proteins	Trace	
Pus cells	2-3	
RBC	Nill	

TLC= Total Leucocyte Count HB= Haemoglobin MCV= Mean Corpuscular Volume
CRP= C Reactive Protein LDH= Lactate De-Hydrogenase RBC= Red Blood Cell

Table-4: Urine Electrolyte Studies.

Electrolyte in Urine	Result	Reference value
Potassium	30.9 mEq/L	20
Sodium	73 mEq/L	20

Table-5: Additional Testing for Hypokalaemia.

Test	Result	Reference value
Serum Renin	48.65	3.11-41.2uIU/ml

The patient's laboratory investigations showed severe leukocytosis, normocytic anaemia, and thrombocytopenia, in addition to raised C-reactive protein and Lactate dehydrogenase levels (Tables 2 and 3). Notably, his symptoms like muscle cramps, fatigue, and constipation were well explained by his low potassium level of 2.4 mmol/L (n=3.5-5.1 mmol/L) representing severe hypokalaemia (Table 3).

Despite aggressive and continuous intravenous and oral potassium replacement, the patient's potassium levels did not improve significantly. Consequently, a workup to look for the underlying cause for potassium loss was initiated, including urinary electrolytes, which demonstrated renal loss of potassium (Table 4). Suspecting activation of the renin-angiotensin system as the cause of renal potassium loss, the patient's serum renin level was sent, which was noted to be elevated (Table 5). It was planned to start potassium-sparing diuretics, but unfortunately, the patient died on day five of his hospital admission.

This case highlights the rare occurrence of severe refractory hypokalaemia presenting in a patient diagnosed with Acute Myelomonocytic Leukaemia, likely due to a number of underlying causes, including the activation of renin-angiotensin-aldosterone system (RAAS).

Discussion

Hypokalaemia is a relatively common electrolyte abnormality associated with AML. However, this report highlights a rare case of severe refractory hypokalaemia, which makes it noteworthy.

The patient's laboratory investigations showed elevated white blood cell count, normocytic anaemia, thrombocytopenia, and elevated inflammatory markers, all consistent with the investigations likely to be seen in a patient with AML. The diagnosis of AML M4 subtype was confirmed with a bone marrow biopsy, in addition to the presence of monocytoid blast cells on the peripheral smear. The patient's signs and symptoms, including fatigue, constipation, and muscle cramps, were all consistent with his severely low serum potassium levels (2.4mmol/L) (n=3.5-5.1 mmol/L).

Despite aggressive management with intravenous and oral

potassium replacement, the patient's serum potassium remained low, indicating a rare case of severe refractory hypokalaemia. Renal potassium wasting was seen on the urinary electrolyte analysis, which indicated a possibility of the activation of the renin-angiotensin-aldosterone system (RAAS). Monocytes can produce renin-like substances that can contribute to urinary potassium losses.[3]

In a recent case report, Pérez-Pinzón et al. described the case of a patient diagnosed with myelomonocytic type of AML, presenting with acute kidney injury (AKI) and electrolyte abnormalities, including hypokalaemia due to high levels of lysozyme accumulated in the tubular cells of the kidneys. This rare complication of AML is characterised by excessive secretion of lysozyme by macrophages and monocytes.[4]

The management of a patient with refractory hypokalaemia is complicated with a relatively poor prognosis.[4] Often a multifaceted approach is required. In addition, the use of potassium sparing diuretics may mitigate the effects of aldosterone and reduce the urinary potassium loss. However, in the present case, the patient unfortunately died before this approach could be initiated.

This case emphasises the importance of early detection and effective management of hypokalaemia in patients with AML. This can help prevent life-threatening complications and improve patient outcome. Additionally, this case also contributes to the limited literature available on patients with M4 subtype of AML having severe refractory hypokalaemia, highlighting the importance of further research to explain the underlying mechanism and strategise ideal management protocols.

Conclusion

This case report presents a rare case of severe refractory hypokalaemia in a patient with M4 subtype of AML. The cause appears to be multifactorial, however, excessive activation of RAAS and secretion of lysozyme seems to be the major contributing factors in the development of refractory hypokalaemia. Early recognition and ideal management plans for the electrolyte abnormalities are crucial in patients with AML to prevent long-term complications and improve the outcomes of the patient.

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Author Contribution:
SS, MMK, AHS & AW: Concept, data analysis, interpretation, drafting, reviewing and final approval.
MB: Concept, drafting, reviewing, data interpretation and final approval.