To Treat or Not to Treat? A Case of Pseudohyperkalemia in Chronic Lymphocytic Leukemia
Urvi V. Patel, DO, MPH, Mingran Yu, DO, Sara Khan, MD, Department of Medicine, Reading Hospital.

INTRODUCTION

Pseudohyperkalemia is a well understood but under-recognized phenomenon of falsely elevated potassium levels that is not a true reflection of in vivo potassium. This finding may occur in the context of any extreme leukocytosis or thrombocytosis and has been reported in patients with chronic lymphocytic leukemia (CLL).

CASE DESCRIPTION

A 69-year-old male with relapsed CLL treated two months prior with bendamustine and rituximab presented to the hospital with a two-week history of fatigue and diffuse pain. Associated complaints included chills and diaphoresis. Patient denied fevers, dyspnea, abdominal pain, sick contacts or bleeding diatheses.

Vitals: Temp 36.9 °C, HR 101 bpm, BP 135/89 mmHg, RR 30 bpm, SpO2 92%.

Physical Exam: Patient was alert and oriented with palpable anterior cervical, submandibular, and inguinal lymphadenopathy.

Initial Laboratory Results:

<table>
<thead>
<tr>
<th>Calcium</th>
<th>8.5 mg/dL</th>
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<tbody>
<tr>
<td>Phosphorus</td>
<td>4.4 mg/dL</td>
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<tr>
<td>Uric acid</td>
<td>11.2 mg/dL</td>
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<tr>
<td>Lactate</td>
<td>203 IU/L</td>
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<tr>
<td>Dehydrogenase</td>
<td>156</td>
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A subsequent whole blood potassium was ordered which also showed a potassium of 10.1 mmol/L. However, electrocardiogram (EKG) did not show any evidence of hyperkalemia (Figure 1).

Due to discrepancy between serum chemistry and EKG an i-STAT EG+ was obtained that showed a serum potassium of 4.9 mmol/L.

- **All subsequent electrolyte studies were obtained utilizing point of care testing.**
- Patient was treated with prophylaxis for high risk of tumor lysis syndrome (TLS) with rasburicase.
- He was admitted for further evaluation and management of CLL and ultimately treated with bendamustine and rituximab.

CLINICAL COURSE

Laboratory studies showed a significant decrease in WBC count over subsequent days with serum chemistries showing accurate measurement of potassium after the WBC had descended to less than 100,000/uL (Figure 2).

DISCUSSION

Leukocytic burden and the fragility of leukemic cells seen in CLL makes mechanical shearing of cells common during blood draws with vacuum containers and pneumatic transport of blood samples, both of which induce mechanical stress resulting in pseudohyperkalemia.

Centrifugation of heparinized tubes also causes in vitro cell destruction and release of potassium.

Serum potassium levels should be confirmed with laboratory studies that optimize cellular integrity.

- Point-of-care venous blood testing minimizes potential for premature cell lysis and coagulation therefore providing accurate assessment of in-vivo potassium levels.

CONCLUSION

Differentiation between true hyperkalemia and pseudohyperkalemia is crucial to avoid unnecessary overcorrection of potassium levels that can lead to life-threatening iatrogenic hypokalemia.

**REFERENCES**


DO, MPH, Mingran Yu, DO, Sara Khan, MD, Department of Medicine, Reading Hospital.

<table>
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<tr>
<td>Other</td>
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<tr>
<td>Collection Technique</td>
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<td>Cellular Shifts</td>
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<tr>
<td>Decreased Urinary Excretion</td>
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<td>Increased K &quot;Production&quot;</td>
</tr>
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</table>

**Figure 1.** EKG shows normal sinus rhythm without evidence of prolonged QRS, QT interval or peaked T waves.

**Figure 2.** Graph depicting potassium levels and WBC count of the patient.

**Table 1. Causes of hyperkalemia**

- Hyperkalemia
- Causes of Pseudohyperkalemia
- Causes of true Hyperkalemia
- Other
- Collection Technique
- Cellular Shifts
- Decreased Urinary Excretion
- Increased K "Production"

**Hyperkalemia**

- Hypoparathyroidism
- Diabetic ketoacidosis
- Hyporeninemic hypoaldosteronism
- Mitochondrial disorders
- Hereditary caudal regression syndrome
- Myopathies
- Myoglobinuria
- Treatment with renin-angiotensin system inhibitors
- Drug-induced hyperkalemia

**Pseudohyperkalemia**

- Release of potassium from red blood cells
- Heparin-induced pseudohyperkalemia
- Mechanical trauma
- Prolonged in vitro transport
- WBC release K+ during heparinization
- WBC fragility
- Released from FCT during clotting process

**Decreased Urinary Excretion**

- Diabetes insipidus
- Aldosterone deficiency
- Renal failure
- Kidney transplantation
- Corticosteroid therapy
- Diuretic therapy
- Acute renal failure

**Increased K "Production"**

- Insulin deficiency
- Acidosis
- Urinary tract obstruction
- Myoglobinuria
- Extrarenal potassium production
- Increased aldosterone levels
- Increased renin levels