

CLINICAL VIGNETTE

Pseudohyperkalemia Related to Essential Thrombocytosis

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Introduction

Pseudohyperkalemia is a condition caused by leakage of potassium from cellular elements often due to hemolysis but also occurring in patients with excess hematological cells.¹ Essential thrombocytosis (ET), polycythemia vera (PCV), chronic lymphocytic leukemia (CLL) are hematological conditions that contribute to falsely elevated serum potassium levels.¹⁻³ Clinical suspicion for pseudohyperkalemia should be raised with high abnormal complete blood count results, especially if changes in the potassium level track with rising platelet, red blood cell counts or white blood cell counts.^{3,4} The clinical method to correct for pseudohyperkalemia is to draw a plasma potassium, which excludes these cellular elements. By excluding these elements, a more accurate estimate of actual blood potassium is obtained from a patient's laboratory specimen.²

We present a case of pseudohyperkalemia in a patient with severe thrombocytosis with platelet counts of 950-1000 x 10³/ul. The difference between the serum potassium and plasma potassium was striking at 2mmol/L. The difference between serum and plasma potassium was severe enough to raise concerns that the patient may have had hypokalemia that appeared to be normokalemia or hyperkalemia due to the underlying platelet disorder.

Case Report

An 85-year-old male with history of hypertension, iron deficiency anemia, essential thrombocytosis and chronic kidney disease stage 3 presented for routine follow up. His hypertension was well controlled on amlodipine 7.5 mg once daily. His iron deficiency anemia was due to prior gastrointestinal bleeding, which had resolved. He was taking ferrous sulfate once daily and omeprazole once daily. His essential thrombocytosis was managed by hematology. He previously had taken aspirin, which was discontinued after gastrointestinal bleed.

His lab tests were remarkable for elevated potassium at 5.9 mmol/L. His prior potassium levels had been in the low 5's mmol/L. Other labs were notable for platelet of 1,004 x10E3/uL, previously elevated in 900's x10E3/uL, hemoglobin of 12.3 g/dL and iron was 33 mcg/dL.

A plasma potassium was also checked at his hematologist's request to exclude any effect of essential thrombocytosis and returned 3.9 mmol/L.

Discussion

The usual rise in serum potassium in essential thrombocytosis is estimated to be 0.07-0.15 mmol/L for every 100,000 increase in platelets beyond 450,000 platelets/ul. In this case the patient's platelets of 950,000 – 1,000,000 platelets/ul would have predicted a rise in serum potassium at most of about 0.75 mmol/L.¹ However, the discrepancy between the plasma and serum potassium was much larger at 2 mmol/L. Whereas the equation would have predicted a plasma potassium of 5.2 mmol/L at the peak level of serum potassium of 5.9 mmol/L, the finding of plasma potassium of 3.9 mmol/L suggests that patient was normokalemic or possibly hypokalemic. This highlights the risk of treating apparent hyperkalemia due to pseudohyperkalemia from hematological abnormalities. Plasma potassium should be checked in any patient with abnormally high red blood cells, white blood cells and/or platelets prior to treatment of apparent hyperkalemia. In this patient, treatment with sodium polystyrene, patiomer, or zirconium cyclosilicate (ZCS) may have resulted in dangerous levels of hypokalemia.⁵

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