



Case Report

Clinical Implication of Pseudohyperkalemia in Patients With Leukocytosis - A Case Report

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ABSTRACT

Potassium has critical role in cellular homeostasis. Hyperkalemia is a potentially life threatening electrolyte imbalance that needs to be addressed promptly to avoid imminent complications. Sometimes its diagnosis is complicated by factitious elevations in serum potassium levels that were clinically unexplained. Since the workup of pseudohyperkalemia consumes valuable health resources and can affect patient safety, it is essential to identify the variables causing this. The following case report emphasizes the need to recognize pseudohyperkalemia in patients with leukocytosis.

KEYWORDS: Pseudohyperkalemia, leukocytosis, hyperkalemia.

INTRODUCTION

Extreme leukocytosis can be associated with spurious elevation of blood potassium levels [1]. It is essential to promptly diagnose pseudohyperkalemia to avoid inappropriate treatment. Evaluation of plasma potassium levels usually determines the correct diagnosis. The following case report emphasized the clinical implication of pseudohyperkalemia in a patient with acute myeloid leukemia.

CASE REPORT

A 71 year old male with history of acute myeloid leukemia, presented to the emergency department complaining of fatigue and weakness in lower extremities for several weeks. He had no signs and symptoms suggestive of vaso-occlusive crisis. His white count was 185,000 cells per microliter. Peripheral blood smear revealed cells highly suspicious for myeloblasts, favoring the diagnosis of acute myeloid leukemia. Subsequently a bone marrow aspirate and biopsy with flowcytometry and cytogenetics was performed confirming the diagnosis of AML-M7.

During the hospital course patient had potassium of 6.2 mEq/L (Normal range 3.5-5 mEq/L). The test was repeated and reported as 6.4 mEq/L.

The electrocardiogram did not reveal any changes related to hyperkalemia. Patient was treated with insulin, bicarbonate and kayexelate. However, the repeat potassium level 2 hours after the treatment remained high at 6.1 mEq/L. His fatigue and muscle weakness was attributed to hyperkalemia. Despite the above treatment measures neither his symptoms nor potassium level changed.

This raised our suspicion for the possibility of pseudohyperkalemia that is not accurately reflecting the in-vivo serum potassium levels because of high WBC count. Then plasma based potassium levels (free of cellular elements) were ordered and found to in normal limits (4.4 mEq/L). Also, serum levels of potassium levels gradually trended down when WBC count decreased after starting treating for AML.

DISCUSSION

Pseudohyperkalemia should be considered if serum potassium is greater than plasma level by at least 0.4 mEq/l is encountered with normal electrocardiogram, absence of signs and symptoms and cannot be explained by any medication or underlying illness [2]. Because of elevated WBCs in this patient, there is increased lysis of cells. It could be due to shortage of metabolic fuels that causes impaired sodium-potassium adenosine triphosphatase (ATP) activity that in turn increases the expulsion of potassium from cells. This is an in-vitro phenomenon that occurs during clotting process [3].

It is usually confirmed by measuring plasma potassium that reflects accurate levels as seen in this patient rather than serum levels [4]. However, there has been a debate regarding the most reliable sample to differentiate true from spurious hyperkalemia [5]. Analysis of plasma has been recommended at some premier institutions [6].

Pseudohyperkalemia has been reported in patients with normal blood counts. Other causes have been attributed such as mechanical injury to cellular components during sample handling and delay in analysis for over six hours [7]. At our institution repeat blood samples will be drawn with extra precautions as per the lab protocol. Therefore the sample handling could not be a factor in our case as the repeat serum potassium level did not change in spite of utmost care taken during second blood draw. High index of suspicion is needed to diagnose this disorder. The clinical implication is to obtain both serum and plasma potassium in the patients with suspected pseudohyperkalemia.

CONCLUSION

Many in-vivo and in-vitro related factors could contribute to the phenomenon of pseudohyperkalemia. Therefore, the possible factors associated with the development of this process needs to be excluded. It is always prudent to have the hospital system flag the potassium results in patients with elevated WBC counts indicating the possible relationship between pseudohyperkalemia and extreme leukocytosis. However, high index of suspicion is needed to

diagnose pseudohyperkalemia to avoid unnecessary investigation and potentially harmful treatments.

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