

Pseudohyperkalemia From a Pneumatic Tube Transport System: Case Report and Literature Review

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Abstract

Objective: To report a case of pseudohyperkalemia due to a pneumatic tube transport system.

Case Summary: A 75-year-old male presented to the emergency medicine department with chest pain and intermittent vision loss over the previous 2 days. Laboratory studies revealed a potassium value of 9.6 mEq/L and a white blood cell (WBC) count of $262 \times 10^9/L$. An electrocardiogram did not reveal changes consistent with hyperkalemia. Emergent treatment for the hyperkalemia was instituted. Repeat plasma potassium values obtained after treatment for the hyperkalemia remained significantly elevated. It was eventually recognized that the hyperkalemia was due to the combination of undiagnosed leukemia causing a significantly elevated WBC count and transport of the patient's specimen to the laboratory via a pneumatic tube transport system. Manual transport of the specimen to the laboratory repeatedly revealed normal or hypokalemic values.

Discussion: Hyperkalemia is a potentially fatal electrolyte abnormality that must be differentiated from pseudohyperkalemia. Pseudohyperkalemia is defined as a spurious elevation of potassium levels usually due to mechanical trauma during venipuncture resulting in hemolysis and release of potassium from the cellular elements of blood. Pneumatic tube transport systems should be listed in the scientific literature as another potential cause of pseudohyperkalemia, especially in patients with high WBC and/or platelet counts.

Conclusion: Pharmacists and other health care providers should be aware of pneumatic tube transport systems potentially causing pseudohyperkalemia, because regular treatments for hyperkalemia for this problem may cause patient harm.

Key Words—pneumatic tube transport systems, pseudohyperkalemia

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Hyperkalemia is a potentially life-threatening electrolyte derangement that must be recognized and corrected. Cardiovascular and neurological dysfunctions are the primary clinical manifestations of hyperkalemia. Pseudohyperkalemia is defined as a spurious elevation of potassium levels usually due to mechanical trauma during venipuncture resulting in hemolysis and release of potassium from the cellular elements of blood. Other rare causes include rheumatoid arthritis, mononucleosis, hereditary spherocytosis, and familial pseudohyperkalemia. We present a case of pseudohyperkalemia due in part to a pneumatic tube transport system in a patient who at the time had undiagnosed leukemia. Our objective in

reporting this case is to alert pharmacists of the potential for pneumatic tube transport systems to cause pseudohyperkalemia in leukemic patients with markedly elevated platelet or white blood cell (WBC) counts.

CASE PRESENTATION

A 75-year-old male presented to the Department of Emergency Medicine with chest pain and intermittent loss of vision for the preceding 2 days. His past medical history was significant only for mild untreated hypertension. On presentation he was hemodynamically stable and complained of intermittent loss of vision, which he described as “curtain down,”

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lasting for a few seconds with each episode. His review of systems was negative apart from the aforementioned symptoms. At the time of presentation, his physical examination was normal. Computed tomography (CT) scan of the brain was read as normal. Other pertinent labs included a WBC count of $262 \times 10^3/\mu\text{L}$ ($262 \times 10^9/\text{L}$) with multiple immature forms; blood urea nitrogen and creatinine of 26 mg/dL and 1.3 mg/dL, respectively; and serum potassium of 9.6 mEq/L. A diagnosis of chronic lymphocytic leukemia was made based on the patient's history, high WBC count that was predominantly lymphocytes, and a review of the patient's peripheral blood smear by a hematologist. A 12-lead electrocardiogram (ECG) did not reveal changes consistent with hyperkalemia such as T-wave peaking or widening of the QRS complex (Figure 1).

The patient was treated with intravenous calcium gluconate, 50% dextrose, insulin, and oral sodium polystyrene sulfonate and then was transferred to the intensive care unit. A repeat plasma potassium level was 5.6 mEq/L and, as previously mentioned, no ECG changes consistent with hyperkalemia were noted. A follow-up potassium sample 1 hour later revealed a potassium value of 7.6 mEq/L. Intravenous 50% dextrose and insulin were repeated, and a higher dose of sodium polystyrene sulfonate was given. After this therapy, a repeat potassium value of 7.8 mEq/L was

obtained. It was at this time that the clinical team suspected that this patient may have pseudohyperkalemia due to the extremely elevated WBC count. Trauma during pneumatic tube transport was also suspected. To confirm this suspicion, 2 blood samples obtained with one venipuncture were sent for analysis utilizing 2 different transportation modes. Sample A was hand delivered and Sample B was sent through routine pneumatic tube transport system to the Department of Laboratory Medicine. Sample A revealed potassium of 3.4 mEq/L and Sample B revealed a potassium of 7.8 mEq/L (Table 1). It was determined that the extreme leukocytosis and mechanical trauma from pneumatic tube transportation were the most likely causes of pseudohyperkalemia.

DISCUSSION

Hyperkalemia is a potentially life-threatening clinical problem encountered frequently by pharmacists during routine clinical patient monitoring. Pseudohyperkalemia is a clinical condition in which there is an artifactual elevation of the serum potassium level due to a release of intracellular potassium. It was first described by Hartmann and Mellinoff in 1955.¹ Pseudohyperkalemia has been reported in clinical settings such as in vitro hemolysis due to mechanical trauma during venipuncture, severe thrombocytosis and/or leukocytosis, and familial hyperkalemia. Before

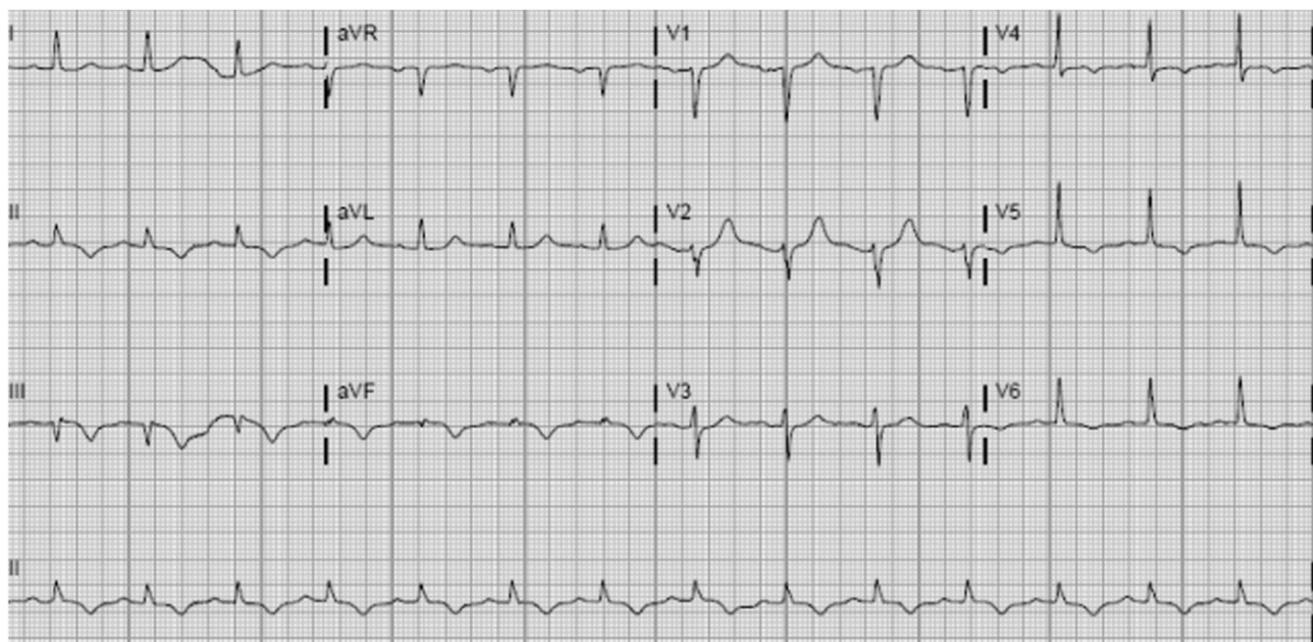


Figure 1. ECG: Sinus rhythm normal axis, Q waves in inferior leads, no changes significant for hyperkalemia.

Table 1. Descriptive of time and mode of sample delivery and corresponding potassium levels

Time/Mode of delivery	Plasma potassium
0:14 hours/Pneumatic tube system	9.6 mEq/L
3:05 hours/Pneumatic tube system	5.6 mEq/L
4:57 hours/Pneumatic tube system	7.6 mEq/L
7:00 hours Sample A/Pneumatic tube system	7.8 mEq/L
7:30 hours Sample B/Hand delivered	3.4 mEq/L

Note: 1 mEq/L = 1 mmol/L.

the routine heparinization of collected blood specimens, this phenomenon was attributed to the clotting process inducing an in vitro release of potassium from leukocytes as they were destroyed. Because the potassium value is now measured in plasma or heparinized tubes, clotting was an unlikely cause in our case.

Other causes for pseudohyperkalemia apart from the ones mentioned above include fist clenching during phlebotomy, which may result in small rise in the potassium value.² Placement of the sample on ice or a delay in specimen processing may lead to a shortage of metabolic fuels causing a progressive rise in potassium levels.^{3,4} Our patient specimens were properly collected, and there was no delay in specimen processing. A case series reported on 4 chronic lymphocytic leukemia patients with WBC counts greater than $200 \times 10^3/\mu\text{L}$ ($200 \times 10^9/\text{L}$) that were reported to have pseudohyperkalemia attributed to use of vacuum tubes, which may directly lyse WBCs releasing potassium.^{5,6} However, a case-control study by Kellerman et al showed no difference in plasma potassium levels when the phlebotomy with performed with a vacutainer versus a syringe, provided that the method of transporting the sample to the laboratory was similar.⁷ In our case, vacutainers were used to collect the blood sample, but only transport by the pneumatic tube system resulted in the apparent rise in potassium.

Another cause of pseudohyperkalemia is a rare genetic entity known as *familial pseudohyperkalemia*. This disease is an autosomal dominant disorder that maps to the same locus (16q23-ter) as hereditary xerocytosis.⁸ This familial condition is characterized by temperature-dependent loss of potassium from red blood cells and is attributed to abnormally high potassium permeability of the red cell membrane⁸⁻¹¹ either through Na^+ , K^+ - ATPase pump,

or $\text{Na}^+\text{K}^+\text{2Cl}^-$ co-transport system or passive diffusion.

The case-control study by Kellerman et al,⁷ additional case reports found in the literature,¹²⁻¹⁴ and our case report indicate that pneumatic tube transport systems have been responsible for significantly elevated serial potassium results. It would appear that spurious results utilizing pneumatic tube transport systems are especially likely to occur in patients with severe leuko- or thrombocytosis. Prior studies examining the effect of pneumatic tube transport systems on the rise in serum potassium values in patients with a normal WBC count showed no evidence of clinically important potassium elevations.^{15,16} The disparate results noted in our case when 2 specimens were sent to the laboratory using 2 different methods of transport strongly suggested trauma and cellular lysis of WBCs. Both samples were collected utilizing a vacutainer method with heparin and lithium as diluents and were transported immediately after collection. However, Sample A was transported through the pneumatic tube system while Sample B was manually delivered to the chemistry laboratory yielding potassium values of 7.8 mEq/L and 3.4 mEq/L, respectively.

Hospitals utilizing pneumatic tube transport systems should develop policies concerning what materials are appropriate to transport via these systems. These policies should take into consideration appropriate transport based on recommendations from the manufacturer of the system as well as published literature that may discover problems, as in our case report, after widespread transport system use.

We conclude that because pneumatic tube systems are common methods of transporting phlebotomy specimens to the clinical laboratory, pharmacists should be aware that pseudohyperkalemia is a potentially important problem in patients with either leuko- or thrombocytosis. Recognition of this problem is necessary to prevent iatrogenic hypokalemia due to treatment of spuriously elevated potassium values.

CONCLUSION

Pseudohyperkalemia should be suspected in patients with extremely elevated WBC and/or platelet counts who lack clinical and ECG evidence of hyperkalemia. In patients with either extreme leuko- or thrombocytosis, we recommend avoiding the transport of blood specimens intended to be analyzed for potassium via pneumatic tube transport systems. In addition, we recommend that specimen transport systems be listed as important causes of this problem in susceptible patients.

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AUTHOR QUERIES

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There are no queries in this article.