Reporte de Caso

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High pot but no T

Potasio elevado, sin T picuda

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ABSTRACT

Introduction*. The identification and treatment of patients with hyperkalemia is necessary to prevent the development of arrhythmias. Pseudohyperkalemia is most commonly due to specimen haemolysis and is often recognised by laboratory scientists who subsequently report test results with cautionary warnings. The authors present a case of pseudohyperkalemia in a patient with chronic lymphocytic leukaemia.* ***Report case****: the technical factors and method of transport are a potential cause of pseudohyperkalemia. Pseudohyperkalemia has been associated with hyperleukoctosis, in cancer patient populations, more commonly in CLL in adults, but also acute lymphoblastics leukemia in children. This places the patient at risk of unnecessary and potentially dangerous treatments.* ***Conclusion****: Physicians should consider pseudohyperkalemia as the underlying cause of elevated potassium levels in patients with malignant leucocytosis who do not have signs or symptom of systemic hyperkalemia.*

Keywords: *Potassium; Electrocardiography; Hyperkalemia; Leukemia, Lymphocytic; Chronic* ***(Fuente****: DeCS-BIREME).*

RESUMEN

Introducción. La identificación y el tratamiento de pacientes con hiperpotasemia son necesarios para prevenir el desarrollo de arritmias. La pseudohiperpotasemia se debe más comúnmente a la hemólisis de la muestra y a menudo es reconocida por los laboratoristas que posteriormente informan los resultados de las pruebas con advertencias de precaución. Los autores presentan un caso de pseudohiperpotasemia en un paciente con leucemia linfocítica crónica. **Reporte de caso:** los factores técnicos y el método de transporte son una causa potencial de pseudohiperpotasemia. La pseudohiperpotasemia se ha asociado también con hiperleucoctosis, en poblaciones de pacientes con cáncer, más comúnmente en Leucemia linfocítica crónica en adultos, pero también con leucemia linfoblástica aguda en niños. Esto pone al paciente en riesgo de tratamientos innecesarios y potencialmente peligrosos. **Conclusión:** Los médicos deben considerar la pseudohiperpotasemia como la causa subyacente de los niveles elevados de potasio en pacientes con leucocitosis maligna que no presentan signos o síntomas de hiperpotasemia sistémica

Palabra Clave: Potasio; Electrocardioma; Hiperkalemia; Leucemia; Linfoma (**Source:** *DeCS-BIREME*).

INTRODUCTION

Intracellular and extracellular fluid compartments differ by their electrolyte concentrations. The extracellular fluid contains a large amount of sodium but only a small amount of potassium[(1)].

The fact that over 98 per cent of the total body potassium is intracellular and only 2 per cent is in the extracellular fluid is a challange to regulation. Normally the intracellular potassium concentration is approximately 140 mmol/L compared with 4 to 5 mmol/L in the extracellular fluid[(2)]. This precise control is necessary because many cell functions are very sensitive to changes in extracelular fluid potassium concentration.

Hyperkalemia is defined by a serum potassium level >5.0 mmol/l (5.0 mEq/l). Levels > 6.0 mmol/l (6.0 mEq/l) can induce fatal arrhythmias[(3)] as well as elevated T waves. Thus, the British mnemonic “high pot(assium), high T, low pot low T has been introduced into clinical practice.

Sponataneous hyperkalemia is a rare in healthy individuals because the cellular and renal potassium

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metabolism prevent significant potassium accumulation in the extracellular fluid. Hyperkalemia is seen frequently in renal failure and metabolic acidosis.

An understanding of potassium physiology is helpful when approaching patients with hyperkalemia. The identification and treatment of patients with hyperkalemia is necessary to prevent the development of arrhythmias. Hyperkalemia leads to increased myocyte excitability shortening of the myocyte action potential.

Excess potassium in the extracellular fluids can block conduction of the cardiac impulse from the atria to the ventricles through the A-V bundle. Elevation of potassium concentration to only 8 to 12 mEq/l—two to three times the normal value—can induce arrhythmia, cardiac failure and death[1]. The mechanism can be explained by the fact that a high potassium concentration in the extracelular fluids decreases the resting membrane potential in the cardiac muscle fibers. As the membrane potential decreases, as well as the action potential which significantly affects systolic contraction[1].

REPORT CASE

A moderately frail 80-year-old woman with a history of chronic lymphocytic leukaemia (CLL) diagnosed in 2003 and followed up without treatment presented in an emergency room with a 2 day history of upper respiratory symptoms, exercise-induces shortness of breath, a productive cough, wheezing, and tachypnea. Her past medical history was significant for hypertension, chronic obstructive pulmonary disease, obstructive sleep apnea, gastroesophageal reflux disease and macular degeneration.

On initial examination, the pulse rate was 111/min, her blood pressure was 139/63 mm Hg and the temperature was 38.1°C. Her respiratory rate was 27/min and the oxygen saturation was 85% (room air). Pulmonary auscultation revealed bilateral basal crackles. The remaining examination was normal. Blood cultures obtained at admission were negative. Sputum Gram stain did not reveal microorganisms. A respiratory viral panel from a nasopharyngeal swab was negative for Covid19. A CT scan of the chest obtained at admission revealed bilateral lower lobe infiltrates. She was admitted to the hospital with the diagnosis of community-acquired pneumonia, did not require ICU admission, and a course of antibiotic therapy with Ceftriaxone (7 days) was started.

The Patient was referred to the Geriatrische Klinik St. Gallen after nine days for geriatric therapy. On admission, her vital signs were within normal limits and the physical examination was unremarkable. The presented with a body temperature of 36.7° C, a blood pressure of 120/63mmHg and a regular heart rate of 84 bpm. Her oxygen saturation was 92% (room air). At admission she was treated with pantoprazole, budesonide/formoterol, lisinopril, hydrochlorothiazide and torasemide.

Her lab test showed a white blood cell count of 206.9 (4-10G/l), a haemoglobin 115 (120-160g/l) and a platelet count of 193 (150-300G/l). Sodium 131 (136-144mmol/l) and creatinine 56 (<95umol/l) were within normal ranges whereas potassium was 8.1 (3,5-5,1 mmol/l). Haemolysis was indicated in the accompanying report.

Given her history of CLL, additional lab work was done to rule out tumour lysis syndrome: uric acid 284 (150-360 mol/l), lactate dehydrogenese 447 (<265U/l), creatine kinase 37(<145U/l), phosphate 1.10 (0.8-1.5 mmol/l) and the calculated glomerular filtration rate 83 (>60ml/min/1,73m2) were not suggestive for an underlying tumor lysis syndrome. Based on the high potassium concentration we performed a resting ECG.

The ECG did not demonstrate elevated T waves, loss of P waves, prolonged QRS intervals, or evidence of high-grade blocks consistent with hyperkalemia.

On day 3, Labs were repeated and potassium remained elevated at 8,8mmol/l with normal renal function. Our patient remained asymptomatic. Over the next days, her potassium generally remained between levels 8 to 9 mmol/l. The patient never had clinical signs of hyperkalemia, (as shown in the table 1). Treatment for hyperkalemia wasn`t initiated. She experienced no cardiac events during the thirteen days of her hospitalization.

Based on these findings we suspected pseudohyperkalemia due to the transportation of our blood sample with a of pneumatic tube delivery system. A sample analyzed by an automated blood gas analyzer demonstrated a potassium of 3.6 mmol/l, thus confirming the diagnosis of pseudohyperkalemia.

Table 1. Potassium levels at different time points

	Day 1 K+(mmol/l)	Day 3 K+(mmol/l)	Day 4 K+(mmol/l)	Day 5 K+(mmol/l)	Day 6 K+(mmol/l)
Pneumatic transport-serum	8.1	8.8	8.9	8	
Automated analyzer					3.6
Patient symptoms	A	A	A	A	A

A: asymptomatic.

DISCUSSION

This case describes pseudohyperkaliemia attributed to

leukocytosis due to chronic lymphocytic leukemia. The technical factors and method of transport are a potential cause of pseudohyperkalemia.

The Initial evaluation of hyperkalemia requires a thorough history and physical examination, the review of all medications an assessment of the kidney function and an ECG. The kidneys regulate concentrations of hydrogen, sodium, potassium, phosphate, and other ions in the extracellular fluid. High potassium together with a normal creatinine can also help, as a normal creatinine can be predictive of pseudohyperkalemia[1].

In true hyperkalemia initial ECG manifestations include peaked precordial T waves and a shortened QT interval. With progression of hyperkalemia, lengthening of the PR interval, loss of the P wave, widening of the QRS complex, a sine wave pattern may occur. However, the ECG findings and the clinical context can help identify pseudohyperkalemia and avoid inappropriate treatment. Our Patient did not show any clinical signs of hyperkalemia and the ECG did not show electrocardiographic signs suggestive for hyperkalemia.

A potentially life threatening electrolyte disorder is acute tumor lysis syndrome. With the lysis of cells potassium, uric acid, and phosphate are released into the circulation. In our patients these electrolyte abnormalities, were within normal range.

However, given the presence of chronic lymphocytic leukemia with leucocytosis and otherwise normal clinical findings, the suspicion of pseudohyperkalemia was high.

Pseudohyperkalemia is commonly seen secondary to red cell or white blood cell damage leading to hemolysis. In our patient lysis induced and increase in extracellular potassium concentration. As leucocytes were destroyed, large amounts of potassium contained in the cells were released into the extracellular compartment. It is of note that the patient had a slightly elevated lactate dehydrogenase (LDH) level of 447U/l. LDH is often used as a marker of tissue breakdown as LDH is abundant in blood cells and can function as a marker for hemolysis.

Common causes of pseudohylerkaliemia include mechanical stressors. The most common causes are related to the technique of blood drawing and can involve the following mechanisms, traumatic venepuncture, tourniquet usage and use of vacuum tubes can result in the release of potassium from red cells and a characteristic reddish tint of the serum due to the concomitant release of haemoglobin. Red serum can also represent severe intravascular hemolysis rather than a haemolysed specimen[2]. Issues related to specimen transport (Pneumatic systems), handling (delay), and processing (centrifugation) which might have led to extensive blasts destruction as opposed to whole blood potassium samples and may lead to falsely elevated plasma potassium levels[4].

Pseudohyperkalemia in patients with very high white blood cell counts due to leukaemia or lymphoma has also been reported after mechanical disruption of white blood cells during transport of blood samples via pneumatic tube systems. High white blood cell counts (>120 000/microl) caused by chronic lymphocytic leukaemia can lead to falsely elevated potassium concentrations due to cell fragility.

Centrifugation of a heparinized tube causes in vitro cell destruction and release of potassium as these cells are freely suspended in plasma[2]. Pseudohyperkalemia has been associated with hyperleukoctosis, in cancer patient populations, more commonly in CLL in adults, but also acute lymphoblastics leukemia in children. (a case of hyperkalemia versus pseudohyperkalemia in CLL)[5].

Lauren K. et. al. demonstrated that a considerable proportion of plasma samples in patients with CLL and leucocytosis have high potassium[6].

Moustafa et.al. conduced a study in which they described several causes of pseudohyperkalemia in CLL patients. The negative pressure in a vacutainer causing destruction of the fragile leukemic blasts, the large number of leukocytes might amplify the leakage of potassium due to coagulation from cells in non-heparinized specimens, centrifugation of serum samples can lead to extensive blasts destruction as opposed to whole blood potassium samples[7]. Guiheneuf et al presented two leukemia cases with pronounced leukocytosis who each showed a profound elevation in serum potassium when the specimens were transported through pneumatic tube, but who showed a normal serum potassium when the specimens were transported by a person[8].

We think that a combination the same factors increased the serum potassium concentration in our patient.

Ranjitkar et. al. recommended to obtaining a whole blood potassium from a blood gas syringe. In their study they demonstrated that leucocytosis considerably elevated plasma potassium, observing a 0.6mEq/L increase for every 100x109 cells/L increment in leukocyte count across all patients[9]. Using this approach we found a potassium within normal range.

Huang reported pseudohyperkaliemia in a 83year old patient with a long history of asymptomatic CLL This patient was initially treated for hyperkalemia with insulin plus glucose and sodium polystyrene (10). Due to the ECG findings and an another analytic method we

could avoid such a potentially dangerous therapy.

This case highlights the importance of considering pseudohyperkalemia in cases with a very high leucocyte count due to CLL. Technical factors as pneumatic tube delivery systems and vacuum tubes are a potential cause of WBC lysis and therefore pseudohyperkalemia. The ability to differentiate true hyperkalemia from pseudohyperkalemia is crucial for determining the appropriate interventions and to avoid potentially harmful therapies.

CONCLUSIONS

Physicians should consider pseudohyperkalemia as the underlying cause of elevated potassium levels in patients with malignant leucocytosis who do not have signs or symptom of systemic hyperkalemia. As physicians, we need to consider clinical presentation before interpreting the lab value in day-to-day practice.

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