Pseudohyperkalemia due to Thrombocytosis: A Diagnostic and Therapeutic Case Report of an Elderly Patient

Ren Anping
General Department, Taiping Town Central Hospital, Conghua District, Guangzhou City
No.133 Taiping East Road, Taiping Town, Conghua District, Guangzhou City
Email: R462672808@outlook.com

ABSTRACT

An 87-year-old woman who was bedridden was brought in because she had been experiencing substernal pain for 10 days and right foot pain and swelling for more than a month. Laboratory tests revealed significant thrombocytosis (platelet count peaks at 2903 × 10^9/L) and recurrent severe hyperkalemia (serum potassium up to 7.58 mmol/L). In vitro pseudohyperkalemia caused by potassium release from lysed platelets was confirmed by a significant difference between serum potassium (6.37–7.58 mmol/L) and heparinized whole-blood potassium (3.9–4.41 mmol/L). This case emphasizes how crucial it is to differentiate between artifactual hyperkalemia and significant thrombocytosis in order to prevent needless procedures.

Keywords: Pseudohyperkalemia; Extreme Thrombocytosis; Elderly Patient; Diagnostic Pitfalls; Hematologic Disorders

Introduction

Pseudohyperkalemia, an artificial elevation unrelated to in vivo pathophysiology, can result in incorrect diagnosis and treatment, while hyperkalemia is a potentially fatal electrolyte disorder that needs immediate attention [1-3]. During sample processing, potassium is released from cellular components, most commonly from platelets in thrombocytosis (>500×10^9/L) [4-6], leukocytes in excessive leukocytosis (>200×10^9/L) [2], [7], [8], or hemolysis[9]. This results in pseudohyperkalemia. An especially underappreciated cause is extreme thrombocytosis (>1000 × 10^9/L), in which platelets release intracellular potassium during the development of a serum clot [10], [11], resulting in a difference between serum (higher) and plasma/normal whole blood potassium levels [5], [12], [13].

There are important clinical ramifications to this phenomena. In high-risk populations, up to 41.2% of cases of apparent hyperkalemia may really be pseudohyperkalemia [14], and 34.7% of cases result in iatrogenic hypokalemia as a result of improper treatment. When older patients have many comorbidities, the diagnostic problem is exacerbated because asymptomatic presentations [10], [15] and a lack of ECG abnormalities [16-18] can delay identification. Thrombocytosis-associated pseudohyperkalemia has been observed in solid malignancies [1], post-splenectomy states [19], and myeloproliferative diseases [6], [20].

Platelet counts in these patients range from 500 to 2404×10^9/L. Comparative examination of serum samples and heparinized plasma, which inhibits potassium release

linked to clotting, is necessary for laboratory confirmation[4], [5], [21]. According to recent research, this differentiation is especially important in critical care settings when quick potassium correction may be started [21], chronic renal failure [22], and hematologic malignancies [3], [8], [21]. This case highlights the importance of targeted testing in preventing iatrogenic injury from needless potassium-lowering medicines and exemplifies a diagnostic difficulty in geriatric care[13], [23], [24].

Case Presentation

Chief Complaints

An 87-year-old woman who had been bedridden for more than a year complained of erythema, edema, and discomfort in her right foot for a month, as well as ten days of ongoing substernal pain (after mechanical compression during movement).

History of Present Illness

The patient was primarily bedridden or wheelchair-dependent for almost a year due to their general weakness, frequent drooling, dysphagia (liquid aspiration), and urine/fecal incontinence. One month before to admission, she had increased discomfort, edema, and erythema in her right foot, but no fever. Ten days prior to admission, sternal tension during transfer caused persistent substernal pain that was non-radiating and did not cause dyspnea. She also reported intermittent left lumbar pain, which was a result of the fall. When she was initially admitted, her foot mass fluctuated and it seemed likely that she had gouty arthritis. Present state: regular urine and bowel movements, low appetite, alert but drowsy.

Past Medical History

With a peak systolic blood pressure of 180 mmHg and a 3-year history of hypertension, the patient's condition is poorly managed as a result of non-adherence to treatment. She has a history of lacunar infarction, a type of cerebrovascular disease, but no lingering impairments. Analgesics have been used to treat sporadic bouts of gouty arthritis, and no preventative urate-lowering medication has been used. She also suffers from persistent left lumbar pain following a fall. She denies having a history of blood transfusions, diabetes, coronary artery disease, renal disease, cancer, surgery, any food or medication allergies.

Physical Examination

At admission, the patient's vital signs were as follows: temperature of 37.8°C, pulse of 104 bpm, respiration rate of 20/min, and blood pressure of 160/80 mmHg. She was wheelchair-bound, underweight, cooperative during the examination, and generally alert but sluggish. In addition to erythema, warmth, and edema, the first metatarsophalangeal joint of the right foot showed a 2×2 cm fluctuant and sensitive mass, while the left foot had mild pitting edema. There was no rash, edema, or jaundice on the skin or mucosa. Bibasilar coarse breath sounds, right basal crackles, and a grade 3/6 holosystolic murmur were detected by auscultation of the aortic and pulmonary areas. A neurological test revealed strong motor strength and no focal deficits (5/5).

Laboratory Tests

Upon admission, laboratory tests revealed the following findings: C-reactive protein (CRP) was 46.8 mg/L (reference range <5 mg/L), uric acid was 676 μmol/L (reference range 150–357 μmol/L for females), white blood cells (WBC) were 22.8 × 10^9/L (reference range 4.0–11.0 × 10^9/L), platelets were 2267 × 10^9/L (reference range 150–450 × 10^9/L), and random blood glucose was 6.8 mmol/L.

Important follow-up conclusions included: uric acid reaching 758 μmol/L, at which point febuxostat was started; platelets peaking at 2903 × 10^9/L, WBC at 31.91 × 10^9/

L, and malignant growth factor at 70.94 U/mL (elevated); the first recorded discrepancy, in which serum potassium was 7.54 mmol/L versus heparinized whole-blood potassium at 3.9 mmol/L; a follow-up showing serum potassium at 7.58 mmol/L versus heparinized whole-blood potassium at 4.41 mmol/L; and a head CT scan that showed bilateral centrum semiovale, right basal ganglia, and corona radiata, as well as cerebral atrophy and bilateral internal carotid artery cavernous segment sclerosis.

Treatment and Outcome

A bone marrow biopsy was declined by the patient's family, but the final diagnosis included pseudohyperkalemia due to extreme thrombocytosis (with platelets exceeding 2500 × 10^9/L), a skin abscess on the right foot that was resolved after drainage, and a suspected myeloproliferative neoplasm, which was supported by refractory thrombocytosis, leukocytosis, and an elevated tumor marker. Stage 3 hypertension (extremely high risk), gouty arthritis, recurrent pneumonia, senile cardiac valve disease, sleep disorders, and post-stroke sequelae (dysphagia, functional dependency) were among the comorbidities.

Ceftazidime was started for infection management after the right foot abscess was cut and drained upon admission. Comorbidities were treated with analgesics (lornoxicam, tramadol), antihypertensives (amlodipine, irbesartan-hydrochlorothiazide), and allopurinol. Aggressive treatment, including furosemide, insulin-glucose infusions, sodium bicarbonate, and oral sodium zirconium cyclosilicate (made by the family themselves), was started for persistent hyperkalemia (serum potassium 5.5–7.54 mmol/L).

Hyperkalemia persisted throughout the duration, and platelet counts increased further, reaching a peak of 2903 × 10^9/L. Interestingly, even with serum potassium >7.0 mmol/L, no electrocardiographic alterations characteristic of hyperkalemia were seen (ECGs revealed T-wave inversion). All potassium-lowering medications were stopped when a significant difference between serum and heparinized whole-blood potassium levels suggested pseudohyperkalemia, which was verified by additional testing.

Ceftriaxone was used instead of ceftazidime as an antibiotic for pneumonia. A nasogastric tube was placed for dysphagia, vitamin D2 supplementation was administered for deficiency (25-hydroxyvitamin D 17.3 ng/mL), aminophylline was used for wheezing, and an air mattress was used to prevent pressure ulcers. Analgesics for multisite pain (foot, sternum, and lumbar region), together with ongoing antihypertensives and urate-lowering medication (febuxostat), were used to control the symptoms.

After 55 days, the patient was released with clear lung fields, a cured foot infection, and stable vital signs (blood pressure 132/70 mmHg, heart rate 78 bpm). Serum potassium levels were artifactually higher at discharge (6.56 mmol/L) than heparinized whole blood levels (3.90 mmol/L), and platelets were still increased (2804 × 10^9/L) without bleeding or thrombosis. The family chose palliative assistance over additional hematology workup and post-discharge medicines.

Discussion

This case underscores three key points that align with current understanding of pseudohyperkalemia in thrombocytosis. As for the mechanism, extreme thrombocytosis (>500×10^9/L) causes platelet lysis during serum clot formation, which releases intracellular potassium and artificially raises serum levels while plasma potassium stays normal. This

is known as pseudohyperkalemia, and it is a characteristic diagnostic feature [1], [10], [11]. As demonstrated here and in other cases [4], [5], heparinized whole-blood testing avoids this in vitro artifact, with the 3-4 mmol/L serum-plasma discrepancy being pathognomonic [10]. In cases of essential thrombocythemia, when there is noticeable thrombocytosis but no actual hyperkalemia, the phenomena is well-documented [6], [20], [22].

Furthermore, there is a significant risk of mismanagement because pseudohyperkalemia can result in improper potassium-lowering treatments that could be fatal [1], [12], [13]. Since genuine hyperkalemia usually presents with peaked T-waves or QRS widening, the lack of ECG alterations despite elevated serum potassium (>7.0 mmol/L) should arouse suspicion [16]. As observed here and in earlier reports [23], [25], individuals who receive unnecessary treatment run the risk of developing conditions such intestinal necrosis from kayexalate or hypoglycemia from insulin [26], [30], which is especially harmful for elderly individuals [10].

Lastly, the etiology of thrombocytosis needs to be assessed. Prolonged extreme thrombocytosis indicates clonal illnesses such as essential thrombocythemia [27-29], but reactive thrombocytosis from situations such as infection seldom above 1500×10^9/L [30]. As seen by this case, when platelet counts continue to be noticeably high, essential thrombocythemia—a philadelphia-negative myeloproliferative neoplasm characterized by megakaryocytic hyperplasia [28], [31]—should be taken into consideration. This case emphasizes the necessity of striking a compromise between patient preferences and diagnostic rigor in geriatric care, even while bone marrow biopsies are still diagnostic [29], [32].

Conclusion

Extreme thrombocytosis-induced pseudohyperkalemia is a serious diagnostic hazard, particularly in older patients with intricate comorbidities. To prevent needless interventions, clinicians should link serum potassium with heparinized whole-blood levels and clinical observations (such as no ECG alterations). Although workup may be limited by patient or family wishes, unexplained severe thrombocytosis necessitates evaluation for hematologic malignancy.

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