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Case Report

Biochemistry Section

Laboratory Detection of Pseudohyperkalaemia in a Trauma Patient: A Case Report

MS SUDHARSHAN[1], P RENUKA[2], A RAJA MYDEEN[3], KOTHAI GNANAMOORTHY[4], VM VINODHINI[5]

ABSTRACT

Pseudohyperkalaemia refers to a spuriously elevated serum potassium concentration resulting from laboratory artefacts and frequently poses a diagnostic and therapeutic challenge in routine clinical practice. Its significance is heightened in acute care settings, where timely and appropriate clinical decisions are critical. The present report describes the case of a 53-year-old male who developed pseudohyperkalaemia following polytrauma with extensive lower limb injuries, including a crush injury to the left foot. Initial potassium levels were within the normal range; however, subsequent elevations were observed in the absence of Electrocardiographic (ECG) changes or clinical features suggestive of true hyperkalaemia. Further evaluation revealed thrombocytosis, a recognised cause of pseudohyperkalaemia due to potassium release from activated platelets during clot formation. The present case is notable because the diagnostic dilemma occurred in a critically injured trauma patient, where differentiating true hyperkalaemia from pseudohyperkalaemia had significant therapeutic implications. Prompt recognition prevented unnecessary administration of potassium-lowering therapy, which could have resulted in iatrogenic hypokalaemia. Correlation of laboratory findings with clinical assessment, along with the use of plasma rather than serum samples, aided in confirming the diagnosis. The present report highlights the importance of maintaining a high index of suspicion for pseudohyperkalaemia in patients with thrombocytosis or leukocytosis, particularly in trauma and critical care settings. Awareness of this phenomenon can prevent mismanagement, enhance patient safety, and guide rational treatment decisions.

Keywords: Crush injury, Electrolyte imbalance, Extensive lower limb injuries, Infection, Thrombocytosis

CASE REPORT

A 53-year-old male presented to the Emergency Department following a Road Traffic Accident (RTA). The patient had not been receiving any regular medications prior to the event and had no significant past medical or surgical history. There were no known co-morbidities, including diabetes mellitus, hypertension, or chronic kidney disease. On arrival, his vital signs were as follows: pulse rate 108 beats/min, blood pressure 118/76 mmHg, respiratory rate 24 breaths/min, oxygen saturation 96% on room air, and he was afebrile. General examination revealed pallor, with no evidence of icterus, cyanosis, or pedal oedema. Systemic examination of the cardiovascular, respiratory, abdominal, and central nervous systems was unremarkable.

Initial laboratory investigations demonstrated Haemoglobin (Hb) of 11.4 g/dL, total leukocyte count of 17,200/mm^3, and platelet count of 2.23 lakh/mm^3. Renal and liver function tests were within normal limits. Serum potassium at admission was 4.7 mmol/L. Radiological assessment revealed a comminuted fracture of the right femur and a non-salvageable crush injury of the left foot. No intra-abdominal or thoracic injuries were detected on Focused Assessment with Sonography for Trauma (FAST) examination or chest radiography. Serial laboratory monitoring during hospitalisation showed a progressive increase in serum potassium levels, which coincided with marked thrombocytosis, along with the development of anaemia and intermittent leukocytosis [Table/Fig-1]. Despite potassium levels rising to 6.3 mmol/L, the patient remained asymptomatic and exhibited no ECG changes consistent with hyperkalaemia. This discrepancy raised suspicion of pseudohyperkalaemia.

The diagnosis was confirmed by demonstrating normal potassium levels in plasma samples obtained from heparinised tubes (ranging from 4.2 to 4.5 mmol/L), in contrast to elevated values in serum samples. The temporal association with thrombocytosis further supported this diagnosis.

Over the ensuing 30 days, the patient was stabilised with intravenous fluids and blood transfusions as indicated. Empirical broad-spectrum antibiotic therapy was initiated with intravenous ceftriaxone 1 g twice daily and metronidazole 500 mg three times daily for 10 days, later adjusted according to wound status and culture sensitivity reports. Analgesia was maintained with intravenous paracetamol 1 g every eight hours during the acute phase, followed by oral formulations as tolerated. Supportive care included nutritional supplementation and meticulous wound management.

Date	Hb (g/dL)	T.WBC (cu.mm)	Platelet (cu.mm)	Potassium (mmol/L)
7/5/23	11.4	17,200	223,000	4.7
8/5/23	9.8	11,180	191,000	4
11/5/23	8.8	8,840	252,000	4.5
13/5/23	8.8	14,230	334,000	4
14/5/23	9.8	16,010	441,100	4.5
15/5/23	8.9	20,480	521.000	4
20/5/23	8.5	12,170	1,105,400	6.2
23/5/23	8.7	13,300	1,266,400	6.3
25/5/23	9	12,990	1,040,400	5.9
26/5/23	9.1	12,960	974,100	5.9
27/5/23	9.2	11,550	857,100	5.7
29/5/23	9.6	8,900	660,100	4.8
31/5/23	8.7	9,230	434,100	4.6
1/6/23	8.2	10,020	456,000	4.6
Reference values	13-17	4,000-11,000	150,000-450,000	3.6-5.2

[Table/Fig-1]: Laboratory findings.
T.WBC: Total white blood cells

The patient underwent multiple staged surgical interventions tailored to his clinical progression. Initially, a Chopart's amputation of the left foot was performed due to extensive non-salvageable injury. Temporary stabilisation of the femoral fracture was achieved using skeletal traction via a right upper tibial pin. Definitive fixation was subsequently performed with Open Reduction and Internal Fixation (ORIF) using plating. Owing to progressive necrosis and infection, a revision below-knee amputation of the left lower limb was required.

The patient later underwent serial wound debridement, stump revision, and Split-Thickness Skin Grafting (STSG) of the left medial thigh to achieve adequate soft tissue coverage.

Pseudohyperkalaemia was managed conservatively with close monitoring of plasma potassium levels and serial ECGs. Daily ECGs were performed between 20/5/23 and 27/5/23 during periods of elevated serum potassium, all of which demonstrated no features of hyperkalaemia or T-wave abnormalities. Potassium-lowering therapy was deliberately avoided, preventing iatrogenic hypokalaemia. Management remained focused on surgical stabilisation, infection control, wound care, and supportive therapy. The patient's condition improved steadily with multidisciplinary care. He was mobilised with physiotherapy in the postoperative period and was discharged after 30 days of hospitalisation. On follow-up, he remained clinically stable, with normalisation of Hb, platelet counts, and potassium levels.

DISCUSSION

In the present case of polytrauma, the patient developed spuriously elevated serum potassium levels during hospitalisation. The rise in potassium coincided with progressive thrombocytosis, while the patient remained asymptomatic and exhibited no ECG changes suggestive of true hyperkalaemia. The diagnosis was confirmed by demonstrating normal plasma potassium concentrations, thereby establishing pseudohyperkalaemia.

Pseudohyperkalaemia, first described as a spurious elevation in potassium unrelated to total body potassium, is clinically significant because misinterpretation as true hyperkalaemia can result in inappropriate treatment, leading to iatrogenic hypokalaemia and potentially fatal arrhythmias [1,2].

The present case underscores the diagnostic challenge of distinguishing true hyperkalaemia from pseudohyperkalaemia, particularly in critically injured patients, where inappropriate therapy could have serious consequences. The differential diagnosis of hyperkalaemia is summarised in [Table/Fig-2] [3].

Condition	Serum K+	Plasma K+
Hyperkalaemia	High	High
Pseudohyperkalaemia	Falsely high	Normal
Reverse pseudohyperkalaemia	Normal	Falsely high

[Table/Fig-2]: Differential diagnosis of hyperkalaemia.

A difference of at least 0.4 mmol/L between serum and plasma potassium concentrations has been suggested as a useful diagnostic indicator of pseudohyperkalaemia [4]. A related entity, termed pseudonormokalaemia, may obscure underlying hypokalaemia, further complicating clinical interpretation [5].

Serum potassium concentration is a critical determinant of neuromuscular function and is routinely measured using techniques such as flame photometry and ion-selective electrodes. While these methods provide rapid and reliable results, accurate interpretation requires awareness of potential preanalytical errors [6,7]. The intracellular compartment serves as the primary reservoir of potassium, with concentrations approximately 40 times higher than in the extracellular space [7]. Consequently, in-vitro potassium elevation may occur due to leakage from erythrocytes during or after sample processing, or occasionally as a result of improper phlebotomy technique [8,9].

Several pre-analytical factors during blood collection and handling can affect serum potassium measurement accuracy, including prolonged tourniquet application, vigorous fist clenching before phlebotomy, haemolysis, thrombocytosis, and leukocytosis [10]. The various causes of pseudohyperkalaemia described in the literature are summarised in [Table/Fig-3] [11].

An increased risk of leukocyte related pseudohyperkalaemia has been reported in samples with white blood cell counts exceeding

Causes	Description
Prolonged tourniquet application	Applying a tourniquet for too long before drawing blood can cause blood cells to rupture.
Haemolysis during venipuncture	If red blood cells rupture during blood collection, potassium inside the cells leaks out.
Samples undergoing centrifugation prior to clot formation	Blood samples need to clot before being centrifuged. If centrifuged before clotting, cellular material can contaminate the serum, potentially leading to inaccurate test results.
Leukocytosis	(WBC) count of 50,000 - 100,000 /µL (cu. mm) or greater
Thrombocytosis	Platelet counts exceeding 400,000 - 500,000 /µL (cu. mm)
Familial pseudohyperkalaemia	A rare genetic condition where a person naturally has high potassium levels inside their red blood cells. However, the total amount of potassium in their body is normal.
Hereditary Spherocytosis (HS)	In HS, a genetic mutation affects the structure of the red blood cell membrane. This makes the red blood cells more fragile and prone to rupture

[Table/Fig-3]: Causes and laboratory finding in pseudohyperkalaemia [11].

50,000-100,000/µL [12,13]. Similarly, thrombocytosis associated pseudohyperkalaemia is typically observed when platelet counts exceed 400,000-500,000/µL [14,15].

In the present case, the maximum white blood cell count reached 20,480/µL, which is below the range commonly associated with leukocyte induced pseudohyperkalaemia. In contrast, the markedly elevated platelet count of 1,266,400/µL far exceeded the threshold linked to thrombocytosis related pseudohyperkalaemia. Potassium release from platelets during clot formation is a well recognised mechanism underlying this phenomenon [16], strongly suggesting thrombocytosis as the primary contributor in the patient.

Numerous studies have documented the frequent association between infections and reactive thrombocytosis [17,18], reflecting its secondary nature in response to inflammatory conditions such as infection, malignancy, or trauma [19]. In patient of the current case, severe soft tissue injury combined with superimposed infection likely precipitated thrombocytosis, thereby contributing to pseudohyperkalaemia.

CONCLUSION(S)

The present case highlights thrombocytosis as the principal cause of pseudohyperkalaemia in a polytrauma patient, while leukocytosis remained below levels typically implicated in the condition. The reactive thrombocytosis likely developed secondary to trauma and infection. Recognising pre-analytical and haematological contributors to spurious hyperkalaemia is essential to prevent misdiagnosis and unnecessary potassium-lowering therapy. Clinicians should consistently correlate laboratory findings with clinical presentation and confirm potassium levels using plasma samples when pseudohyperkalaemia is suspected.

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PARTICULARS OF CONTRIBUTORS:
1. Postgraduate Student, Department of Biochemistry, SRM Medcial College Hospital and Research Centre, Chengalpattu, Tamil Nadu, India.
2. Professor, Department of Biochemistry, SRM Medcial College Hospital and Research Centre, Chengalpattu, Tamil Nadu, India.
3. Postgraduate Student, Department of Pharmacology, SRM Medical College Hospital and Research Centre, Chengalpattu, Tamil Nadu, India.
4. Associate Professor, Department of Internal Medicine, ESI Medical College and Hospital, Chennai, Tamil Nadu, India.
5. Professor and Head, Department of Biochemistry, SRM Medcial College Hospital and Research Centre, Chengalpattu, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
P Renuka,
Professor, Department of Biochemistry, SRM Medcial College Hospital and Research Centre, Chengalpattu, Tamil Nadu, India.
E-mail: renukap@srmist.edu.in

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