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Platelet count and serum potassium: An overlooked link in pediatric thrombocytosis

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ABSTRACT

Objective: Thrombocytosis, particularly in children, can lead to spurious biochemical findings such as pseudohyperkalemia, which may result in unnecessary interventions if unrecognized. This study aimed to evaluate the effect of platelet count on serum potassium levels in children with thrombocytosis and to analyze the association between potassium levels and the etiological factors of thrombocytosis.

Materials and Methods: In this retrospective study, 1899 pediatric patients with platelet counts >600×10^9/L were analyzed over one year. After excluding conditions that could affect serum potassium (e.g., essential thrombocytosis, anemia, polycythemia/leukocytosis, renal or hepatic disease, and hyperbilirubinemia), 1210 cases of reactive thrombocytosis were included. Patients with available serum potassium measurements and follow-up data at 3 and 6 months were assessed.

Results: The mean age of the 1210 patients was 2.7±3.4 years, and 54.8% were male. Thrombocytosis was primarily due to infections (71.8%) and surgery, burns, or trauma (28.2%). Platelet counts ranged from 600×10^9/L to 2.193×10^9/L, and serum potassium levels from 2.47 to 9.3 mEq/L. Hyperkalemia occurred in 20.4% of patients, increasing with higher platelet counts (16.7% for 600–900×10^9/L, 29.1% for 900–1000×10^9/L, and 33.5% for >1000×10^9/L). Significant decreases in platelet and potassium levels were observed at 3- and 6-month follow-ups ($p<0.001$). A weak positive correlation was observed between platelet and potassium levels ($r=0.187$), more pronounced in infection-related cases ($r=0.218$). Potassium increased by 0.06 mEq/L for every 100×10^9/L rise in platelet count, with a significant correlation only above 1000×10^9/L ($r=0.196$, $p=0.038$).

Conclusion: Pseudohyperkalemia is a common yet overlooked phenomenon in pediatric thrombocytosis. Recognizing the quantitative platelet–potassium relationship can prevent diagnostic confusion and unnecessary treatment.

Keywords: Hyperkalemia, thrombocytosis, platelet count, pseudohyperkalemia

Introduction

Potassium is an essential electrolyte with a narrow physiological range, and both elevated and decreased levels can necessitate urgent medical attention (1,2). Obtaining accurate and reliable measurements is therefore critical (3). Several pre-analytical factors can lead to erroneous potassium results that do not accurately reflect in vivo concentrations (4,5). Such incorrect readings are often classified as pseudohyperkalemia (PHK) or pseudonormokalemia (PNK) (6–8). Hemolysis during sample collection and transport, as well as leukocytosis, can contribute to PHK or PNK (9). In addition, thrombocytosis represents another significant cause of such spurious results (10). This phenomenon, first described by Hartman in 1955, demonstrated significantly higher serum potassium levels compared to plasma levels in the absence of clinical symptoms. It was attributed to the release of potassium from platelets during aggregation and degranulation in test tube (11).

In patients with thrombocytosis, spuriously elevated serum potassium levels can lead to inappropriate administration of potassium-lowering therapies, potentially resulting in unrecognized iatrogenic hypokalemia or life-threatening cardiac arrhythmias. Similarly, in cases of PNK, where potassium levels appear within the normal range, true hypokalemia may remain

undetected (10,12,13). The most reliable method to identify PHK and PNK is to analyze the sample in a lithium-heparin tube after centrifugation.

In adult populations, previous investigations reported platelet count thresholds of 500×10^9/L and 598×10^9/L for PHK and PNK, respectively, based on serum versus plasma potassium measurements. Furthermore, these studies revealed a strong association between platelet counts and serum potassium concentrations, while plasma potassium levels were unaffected.

In this study, we investigate the impact of platelet count on serum potassium levels in children with thrombocytosis of various etiologies and examine the relationship between potassium concentrations and the underlying causes of thrombocytosis.

Materials and Methods

In this retrospective analysis, patients younger than 18 years with platelet counts greater than 600×10^9/L on complete blood count were evaluated between January 1, 2021, and January 1, 2022. Patients with thrombocytosis who had concurrent serum potassium measurements were included in the analysis. When available, platelet and potassium levels obtained at 3- and 6-month follow-up visits were also recorded. Ankara Bilkent City Hospital, Department of Pediatrics, a large tertiary pediatric center in Türkiye, identified 1899 patients with platelet counts $>600\times10^9$/L within one year. Of these patients, 1594 had simultaneous serum potassium measurements available. To minimize potential confounding factors, newborns and patients with anemia, polycythemia, primary thrombocytosis, leukocytosis, renal or hepatic disease, or hyperbilirubinemia were excluded, as these conditions could influence potassium homeostasis. The remaining patients were categorized into two etiological groups: those with reactive thrombocytosis due to infections and those with thrombocytosis related to soft tissue injury caused by surgery, burns, or trauma. After applying the exclusion criteria, a total of 1210 patients with reactive thrombocytosis were eligible and included in the final analysis. Patients were categorized based on their platelet counts as follows: moderate thrombocytosis for platelet counts between $600\text{-}900\times10^9$/L, severe thrombocytosis for counts between $900\text{-}1000\times10^9$/L, and very severe thrombocytosis for counts above 1000×10^9/L. The process of patient selection, including inclusion and exclusion criteria and final cohort composition, is summarized in Figure 1.

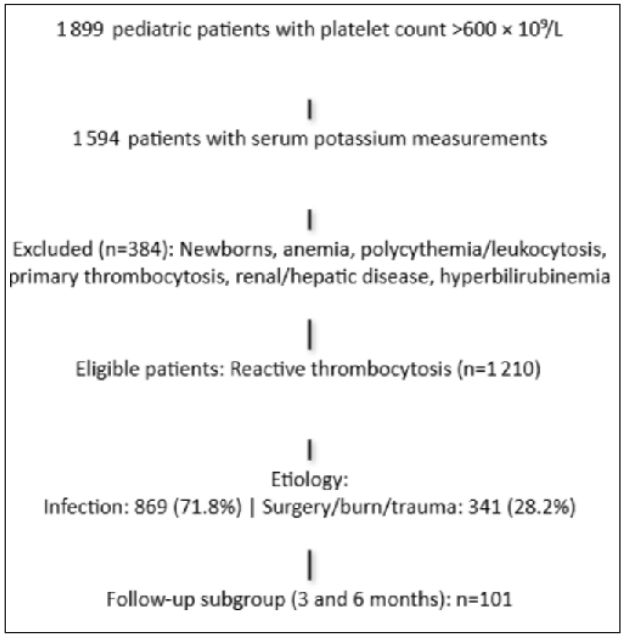

Figure 1: Flowchart of patient selection and study population

Venous blood samples for complete blood count analysis were drawn into 3 mL EDTA tubes and processed using an automated hematology analyzer (Advia 2120, Siemens, Helatneer, Germany). Venous blood samples for serum potassium concentration were collected in 5 mL serum separator tubes. The samples were centrifuged at 4000 rpm for 10 minutes and analyzed using an automated chemistry analyzer (Atellica, Siemens, Helatneer, Germany). All automated analyzers are subjected to daily internal quality control and monthly external proficiency testing to maintain analytical accuracy and consistency. In our institution, hemolyzed samples are automatically flagged by the laboratory information system as part of routine quality control and only data from non-hemolyzed samples were included in the analysis. The reference interval for serum potassium was defined as 3.5–5.5 mEq/L.

This retrospective study was conducted using anonymized data obtained from the hospital's electronic medical records. As the study did not involve direct contact with patients and no identifiable personal information was used, individual informed consent was not required.

Statistical analysis

The normality of distribution for continuous variables was confirmed with the Kolmogorov-Smirnov test. Categorical variables were expressed as numbers and percentages, whereas continuous variables were summarized as mean and standard deviation and as median and IQR where appropriate. To evaluate the change in the potassium and platelet levels obtained in the time interval (baseline, 3rd month, 6th month) Friedman Test was applied. The Bonferroni adjusted Wilcoxon signed-rank test was used for multiple comparisons of times. The relationship between serum potassium concentrations and platelet counts was evaluated using the Pearson or Spearman correlation coefficient, based on the normality of the data. Simple linear regression analyses were performed to derive equations for predicting potassium levels from platelet counts in the entire cohort and in diagnostic subgroups. The R^2 values of the regression models were calculated, and scatter plots with corresponding regression lines were produced to visually represent the associations. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA), and R software, Version 4.1.1 (R Foundation for Statistical Computing, Vienna, Austria). Graphical visualizations were created using the ggplot2 and cowplot packages in R.

Results

The mean age of the 1210 patients was 2.7±3.4 years, of whom 663(54.8%) were male and 547 (45.2%) were female. Demographic characteristics and diagnostic details are summarized in Table I. Etiologies of thrombocytosis were categorized, with 869 patients (71.8%) presenting with infections and 341 patients (28.2%) associated with surgery, burns, or trauma. The serum potassium concentrations of the patients ranged from 2.47 to 9.3 mEq/L, while their platelet counts ranged from 600×10^9/L to 2193×10^9/L. Among the 1210 patients, 908 (75%) had moderate thrombocytosis, 141 (11.6%) had severe thrombocytosis, and 161 (13.4%) were classified as very severe.

Of the 1210 patients with thrombocytosis, 20.4% had hyperkalemia (serum potassium >5.5 mEq/L). Hyperkalemia prevalence increased with platelet count: 16.7% in moderate, 29.1% in severe, and 33.5% in very severe thrombocytosis. Platelet and potassium values for each subgroup are summarized in Table I. A total of 101 patients had complete blood count and serum potassium measurements at both 3- and 6-month follow-ups, showing a statistically significant decrease in median platelet counts and potassium levels over time (p<0.001 for both) (Table II).

Platelet counts showed a weak positive correlation with serum potassium levels in the overall cohort (r=0.187), which was slightly stronger in patients with infections (r=0.218). Simple linear regression was used to estimate potassium from platelet counts in the total sample and diagnostic subgroups, with statistically significant equations and R^2 values presented in Table III.

The relevant scatter plots with regression lines are shown in Figure 2. For every 100×10^9/L increase in platelet count, serum potassium rose by approximately 0.06 mEq/L in the total sample, 0.05 mEq/L in the surgery-burn-trauma subgroup, and 0.08 mEq/L in the infection subgroup. When analyzed by platelet subgroups, no significant correlation was observed for counts of 600–900×10^9/L (r=0.008, p =0.819) or 900–1.000×10^9/L (r=0.030, p=0.141). A weak positive correlation emerged for platelet counts exceeding 1.000×10^9/L (r=0.196, p=0.038) (Table III).

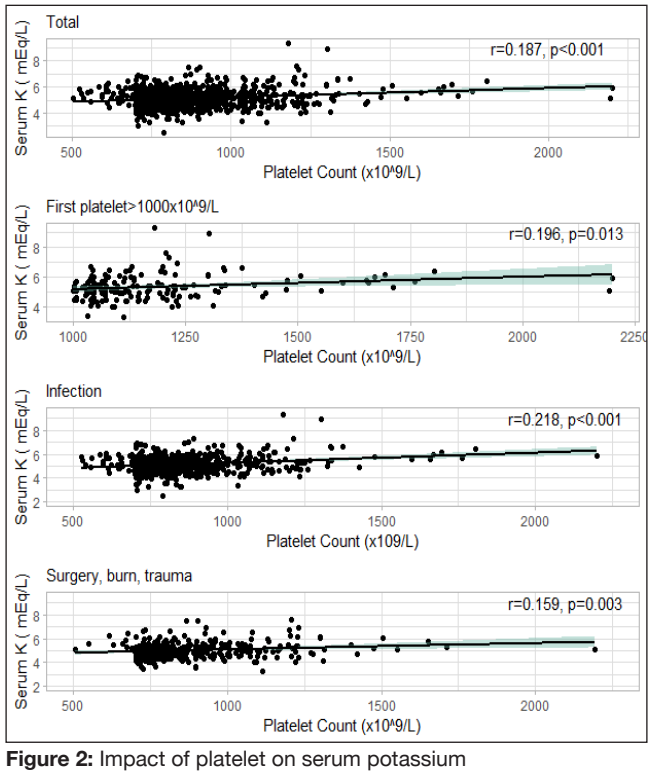

Figure 2: Impact of platelet on serum potassium

Table I: Demographical Characteristics, diagnosis, platelet count, and serum potassium concentration of patients (n=1210)

Variable	Value
Age (year)*	2.7±3.4; 1.0 (1.0–3.0)
Gender†	
Male	663 (54.8)
Female	547 (45.2)
Diagnosis†	
Infection	869 (71.8)
Surgery–burn–trauma	341 (28.2)
Platelet Count (×10^9/L)*	
Infection	830.1±160.1; 782.0 (731.0–879.5)
Surgery–burn–trauma	877.5±193.2; 814.0 (744.5–954.5)
Serum Potassium (mEq/L)*	
Infection	5.1±0.6; 5.1 (4.7–5.5)
Surgery–burn–trauma	5.0±0.7; 4.9 (4.6–5.4)

***:** *mean±SD;median (IQR),* **†:** *n(%)*

Discussion

Reactive thrombocytosis is common in children, particularly among hospitalized patients, and is clinically significant due to its potential impact on serum potassium levels. Elevated platelet counts can lead to PHK, which may result in misinterpretation of laboratory results and unnecessary interventions. Recognizing this phenomenon is crucial to prevent inadvertent iatrogenic hypokalemia and associated cardiac complications. Our study demonstrates that serum potassium levels increase with rising platelet counts, with the relationship influenced by the underlying cause of thrombocytosis. These findings highlight the importance of careful potassium monitoring in children with marked thrombocytosis to guide appropriate clinical management.

Delgado et al. (14) reported PHK and PNK in 0.14% of all serum potassium measurements when compared with plasma values, whereas Thurlow et al. (15) found the incidence of PHK to be as high as 16% in patients with platelet counts exceeding 500×10^9/L. In our study, hyperkalemia was observed in 20.4% of patients with moderate to severe reactive thrombocytosis, and its prevalence increased with rising platelet counts. However, the exact frequency of PHK could not be determined, as simultaneous plasma potassium measurements, the gold standard for differentiating true hyperkalemia from PHK, were not available.

Spurious hyperkalemia has been highlighted in several studies for its clinical significance and risk of misdiagnosis (16). Moderate-to-strong correlations between platelet counts and serum potassium have been reported, particularly in adults with myeloproliferative disorders (17,18). In a large retrospective study by Makale et al. (19), the correlation was

Table II: Changes in platelet count and serum potassium concentration over time

	Baseline*,†	3rd month †	6th month	p*	p†	p‡	p §
Platelet (n=101)	820.0 (768.8-966.5)	598.5 (521.0-700.8)	423.0 (364.8-488.3)	<0.001	<0.001	<0.001	<0.001
Potassium (n=101)	5.2 (4.7-5.6)	4.8 (4.4-5.3)	4.5 (4.2-4.9)	<0.001	<0.001	<0.001	<0.001

**: Overall p value, †: Baseline vs. 3rd month, ‡: Baseline vs. 6th month, §: 3rd vs. 6th month. Data are presented as median (IQR). Comparisons among the three time points were performed using the Friedman test. Post hoc pairwise comparisons were conducted using the Wilcoxon signed-rank test with Bonferroni correction.*

Table III: Linear regression analysis predicting serum potassium level from platelet count

	B	SE B	β	t	p	95% CI for B	R^2
Total sample	0.06×10^{-2}	0.01×10^{-2}	0.187	6.603	<0.001	0.04×10^{-2}-0.09×10^{-2}	0.035
Baseline platelet≥1000x10^9/L	0.08×10^{-2}	0.03×10^{-2}	0.196	2.523	0.013	1.80×10^{-4}-1.00×10^{-3}	0.038
Diagnosis							
Infection	0.08×10^{-2}	0.01×10^{-2}	0.218	6.568	<0.001	5.98×10^{-4}–1.00×10^{-3}	0.047
Surgery, burn, trauma	0.05×10^{-2}	0.01×10^{-2}	0.159	2.973	0003	0.01×10^{-2}-0.09×10^{-2}	0.025

***B:** Unstandardized regression coefficient, **SE:** Standard error, **β:** Standardized regression coefficient, **CI:** Confidence interval. Platelet count was used as the independent variable, and serum potassium level was used as the dependent variable.*

0.155, likely due to limited preanalytical corrections and lack of etiological categorization. The highest correlation (r=0.82) was observed in patients with polycythemia vera (20). A study comparing different etiologies suggested higher PHK incidence and stronger correlations in primary thrombocytosis, although mild reactive cases were underrepresented (18). Primary thrombocytosis is extremely rare in children and is known to affect serum potassium levels; therefore, patients with primary thrombocytosis were excluded from our study.

In our large pediatric cohort, weak correlations were observed in both infection and surgery-burn-trauma subgroups. These correlations were lower than those reported in primary thrombocytosis, where higher and more stable platelet counts and altered platelet fragility contribute to a stronger association. No correlation was detected in our cohort for platelet counts below 1.000×10^9/L. While previous smaller studies suggested a threshold of $500–600\times10^9$/L for PHK/PNK, our findings indicate that this threshold is higher in children with reactive thrombocytosis. The stronger correlations reported in patients with essential thrombocytosis or myeloproliferative disorders suggest additional factors influencing PHK that are absent in reactive thrombocytosis.

This study has several limitations. One important factor affecting PHK in the preanalytical process is platelet elevation; however, other influencing variables could not be fully excluded and should be addressed in future prospective studies. Patients with essential thrombocytosis were not included in comparative analyses due to their limited number. Furthermore, the absence of plasma potassium measurements prevented the determination of a definitive platelet cutoff for identifying PHK. Additionally, multivariate adjustments for potential confounders such as age and sex were not performed when evaluating the correlation between platelet counts and serum potassium levels. Finally, the single-center design may limit the generalizability of our findings to broader pediatric populations and diverse healthcare settings.

Limitations

Despite its limitations, the study's key strengths lie in its large pediatric sample, the analysis of diverse causes of reactive thrombocytosis, and the generation of practical, clinically relevant findings that can help clinicians accurately recognize pseudohyperkalemia and avoid inappropriate or potentially harmful treatments.

Conclusion

Thrombocytosis-related PHK is a clinically significant in vitro phenomenon that may lead to misinterpretation of laboratory results and inappropriate treatment, especially in pediatric patients. Our study demonstrates that serum potassium levels increase proportionally with platelet counts in children with reactive thrombocytosis, particularly due to infections and surgery-burn-trauma, even in the absence of comorbidities. Clinicians should consider simultaneous platelet counts when evaluating elevated potassium results, as unrecognized PHK may mask true hypokalemia. This awareness can prevent unnecessary interventions and iatrogenic complications. While our findings support the need for cautious interpretation of potassium levels in thrombocytosis, further prospective studies are warranted to develop standardized correction approaches and broader clinical guidelines.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Ankara Bilkent City Hospital (18.09.2024, reference number: TABED 2-24-494).

Contribution of the authors

Concept: BD, NY; Design: BD, NY, DK; Formal analysis and investigation: BD, NY, EA; Analysis of data: SPY, BD; Literature search; BD, NY, EA; Writing – original draft preparation: BD, NY; Editing: NY; Supervision: NY

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Conflict of interest

The authors declare that there is no conflict of interest.

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