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Correlation Between Parathyroid Hormone Level and Platelet Count and Morphology Indices in Patients on Maintenance Hemodialysis With Secondary Hyperparathyroidism (A Retrospective Study)

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Abstract

Background: Chronic kidney disease (CKD) and end-stage renal disease (ESRD) patients on maintenance hemodialysis exhibit hematological disturbances, including secondary hyperparathyroidism (SHPT) and platelet dysfunction. Platelet morphology indices—mean platelet volume (MPV), platelet distribution width (PDW), and platelet-large cell ratio (P-LCR)—provide insight into platelet reactivity. This study evaluated relationships between serum parathyroid hormone (PTH) and platelet parameters in end-stage renal disease patients undergoing hemodialysis.

Methods: This retrospective observational study included 79 adult hemodialysis patients (3 sessions/week) at Theodor Bilharz Research Institute, Egypt (January–July 2023). Demographic, clinical, and laboratory data were extracted. Statistical analyses included Spearman correlation, Kruskal-Wallis H, ANOVA with Tukey HSD, and Two-Way ANOVA.

Results: Serum PTH positively correlated with MPV (\rho = +0.448, $p < 0.001$) and P-LCR (\rho = +0.604, $p < 0.001$). Stratification into PTH tertiles (Low< 156 pg/ml, Mid 156–753 pg/ml, High >753 pg/ml) revealed a clear level- dependent increase for MPV (10.8 ± 1.3 vs 10.9 ±1.4 vs 12.3 ± 0.9 fL, $p = 0.0004$) and P-LCR (22.0 ± 8.1\% vs 28.0 ±10.1\% vs 37.1 ±10.0\%, $p < 0.001$). Patients with SHPT ({PTH} > 300 pg/ml) exhibited significantly higher median MPV (12.10 vs 10.45 fL, $p = 0.008$) and P-LCR (34.8\% vs 21.5\%, $p < 0.001$) compared to non-SHPT patients. No significant correlation or variance was observed for platelet count.

Conclusion: Elevated PTH is strongly associated with larger, immature, more reactive platelets (increased MPV and P-LCR) without affecting platelet count or PDW, suggesting SHPT influences platelet morphology rather than quantitative depletion.

Keywords: Chronic Kidney Disease, Secondary Hyperparathyroidism, Parathyroid Hormone, Hemodialysis, Mean Platelet Volume, Platelet-Large Cell Ratio, Platelet Indices.

Introduction

Chronic Kidney Disease (CKD) is characterized by a decline in kidney function or kidney damage lasting over three months, often progressing to the need for dialysis or transplantation also associated with profound disturbances in mineral metabolism, inflammation, vascular homeostasis, and hematological function [1]. In patients with end-stage kidney disease (ESKD) receiving maintenance hemodialysis, these abnormalities are further influenced by the dialysis procedure itself and contribute to the complex clinical phenotype characterized by both bleeding and thrombotic complications [1].

Among the major metabolic complications of advanced CKD, secondary hyperparathyroidism (SHPT) represents a central component of CKD-mineral and bone disorder (CKD-MBD), resulting from persistent disturbances in phosphate, calcium, vitamin D metabolism, and parathyroid hormone (PTH) regulation [2]. The biological effects of SHPT may extend beyond the skeletal system and result in consequences for hematological and cardiovascular homeostasis in patients receiving hemodialysis [3].

Patients with CKD exhibit complex abnormalities in platelet biology, although the reported effects are heterogeneous. A systematic review and meta-analysis including 73 studies demonstrated that platelet function is frequently impaired in CKD, with reduced platelet adhesion and impaired aggregation reported in many studies, while other investigations have demonstrated preserved or enhanced platelet responsiveness [4]. These findings emphasize that platelet abnormalities in CKD cannot be adequately characterized by platelet count alone and that qualitative platelet characteristics may provide additional clinically relevant information [5].

In hemodialysis patients, platelet behavior may be further affected by exposure of circulating blood to the extracorporeal circuit, uremic toxins, inflammation, anemia, endothelial dysfunction, and other dialysis-related factors [6].

Automated hematology analyzers provide several platelet indices that may offer additional information regarding platelet production, turnover, and morphology. Mean platelet volume (MPV) reflects the average size of circulating platelets and is commonly used as an indirect indicator of platelet production and platelet reactivity. Platelet distribution width (PDW) reflects the variability of platelet size, whereas platelet-large cell ratio (P-LCR) represents the proportion of circulating platelets with a relatively large volume. These parameters can therefore complement platelet count by identifying changes in platelet morphology that may occur without overt thrombocytopenia [7].

The distinction between platelet quantity and platelet morphology may be particularly important in patients with CKD. A normal platelet count does not necessarily exclude abnormalities in platelet function, maturation, turnover, or activation [8]. Therefore, the present study aimed to investigate the association between serum PTH concentrations and platelet characteristics in patients undergoing maintenance hemodialysis. Specifically, platelet count, MPV, PDW, and P-LCR were evaluated in relation to PTH concentrations and SHPT status.

Methodology

This retrospective study was based on data obtained from patients' medical records. Patient confidentiality was maintained, and all data were anonymized before analysis. The study was conducted in accordance with the principles of the Declaration of Helsinki and its subsequent amendments. This retrospective observational study was conducted at the Department of Nephrology, Theodor Bilharz Research Institute, Giza, Egypt. The study included chronic renal failure (CKD) patients on regular hemodialysis sessions 3 times per week who were present to the Hemodialysis (HD)unit between January 2023 and July 2023.

Study population

The study population consisted of 79 adult patients on regular HD who were present in TBRI hemodialysis unit during the study period and whose medical records contained the required clinical and laboratory data.

Inclusion criteria

- Age older than18 years
- Confirmed diagnosis of CKD on HD
- Having secondary hyperparathyroidism {SHPT}
- Present during the specified period
- Availability of relevant laboratory investigations: Parathyroid {PTH} level, Complete Blood Picture (CBC) and platelet indices measurements (MPV, PDW and P-LCR).

Exclusion criteria

- Incomplete medical records
- Patients younger than 18 years
- Patients with conditions that could significantly affect the studied outcome (e.g: other inflammatory conditions, sepsis, Bleeding and malignancy)
- Patients who received (anti -platelets) before the relevant measurements

Data collection

- **Demographic data:** age, sex and duration of Dialysis
- **Clinical data:** manifestation of hyperparathyroidism, bleeding tendency (due to thrombocytopenia or platelet dysfunction), DM, HTN, CVD and medications
- **Vital signs**
- **Laboratory investigations:** PTH (parathyroid hormone) level, Platelet count, HB, WBC, Platelet indices {MPV, PDW and P-LCR} all was collected from routine CBC which had done to the patients in HD unit
- **Imaging findings:** plain Xray in cases of fractures due to secondary hyperparathyroidism (SHPT)
- **Treatment received:** e.g. Calcium supplements, phosphorus binder or anti hyperparathyroidism.
- **Clinical outcomes.**

All these data are collected from files of 79 hemodialysis patients in TBRI HD unit.

- **Study variables:** some results are extremely high or extremely low as regarding PTH level or platelet count {this variability may be due to many factors e.g. taking treatment of SHPT or not, duration of the disease or comorbidity affecting the results}
- **Primary outcome:** To study correlation between PTH level and platelet count and each index of platelet indices (MPV, PDW and P-LCR) for studying whether SHPT may related to thrombocytopenia or platelet dysfunction in HD patients.
- **Secondary outcomes:** Studying relation of SHPT as chronic inflammatory condition thrombocytopenia or platelet dysfunction in HD patients.
- **Predictor/exposure variables:** if the patient are taking any medications affecting the results.

Statistical analysis

Data was processed using IBM SPSS Statistics Version 29.0 (IBM Inc., Armonk, NY, USA). Quantitative data were presented as median and interquartile range (IQR), as all continuous variables violated the assumption of normality on Shapiro-Wilk testing (all $p<0.05$). Between-group comparisons (SHPT vs. no SHPT; thrombocytopenia vs. normal platelet count) were performed using the Mann-Whitney U test. For comparisons across three PTH severity groups (low, mid, and high tertiles), the Kruskal-Wallis H test was applied, with post-hoc pairwise comparisons conducted using the Mann-Whitney U test with Bonferroni correction where the overall test was significant; for variables meeting variance homogeneity on Levene's test, one-way ANOVA with Tukey's Honest Significant Difference (HSD) post-hoc test was used instead. The independent effects of SHPT status and thrombocytopenia on platelet morphology indices were assessed by two-way ANOVA (Type II sum of squares), including the interaction term. Correlations between PTH and platelet parameters were evaluated using Spearman's rank correlation coefficient (\rho), with 95% confidence intervals calculated via Fisher's z-transformation; Pearson's r was additionally reported for reference. Categorical variables were compared using the Pearson Chi-square test, with Fisher's Exact test substituted when any expected cell count was below five. A p-value of <0.05 (two-tailed) was considered statistically significant.

Results

The data had collected from patient's medical files in HD unit of TBRI and results are divided into low, med and high PTH groups according to PTH level at time of study.

Table 1 — One-Way ANOVA / Kruskal-Wallis: Platelet Parameters across PTH Tertiles (n=79) Tertile cut-offs derived from PTH distribution: Low < 156 pg/ml (n=28) | Mid 156–753 pg/ml (n=25) | High >753 pg/ml (n=26). Levene's test used to choose ANOVA vs Kruskal-Wallis. Post-hoc: Tukey HSD (ANOVA) or pairwise Mann-Whitney with Bonferroni correction (KW).

Variable	Low PTH Mean± SD / Md (IQR)	Mid PTH Mean±SD / Md (IQR)	High PTH Mean± SD / Md (IQR)	Levene's p	Test Used	Statistic	p-value	Sig.
PLT (\times 10^3/\ml)	218 ± 72 206 (163–261)	221 ± 64 215 (172–262)	236 ± 115 214 (163–280)	0.082	ANOVA	F=0.39	0.676	ns

Variable	Low PTH Mean± SD / Md (IQR)	Mid PTH Mean±SD / Md (IQR)	High PTH Mean± SD / Md (IQR)	Levene's p	Test Used	Statistic	p-value	Sig.
MPV (fL)	10.8 ± 1.3 10.65 (9.90–11.70)	10.9 ±1.4 10.40 (9.80–12.00)	12.3 ±0.9 12.30 (11.70–12.80)	0.021	Kruskal-Wallis	H=15.87	0.0004	***
PDW (fL)	13.5 ± 2.1 14.05 (11.55–15.35)	13.1 ±2.4 13.80 (11.60–15.20)	13.6 ±2.2 14.00 (11.55–15.40)	0.648	ANOVA	F=0.31	0.737	ns
P-LCR (%)	22.0± 8.1 21.50 (16.40–25.95)	28.0±10.1 26.30 (20.90–33.80)	37.1 ±10.0 38.70 (29.50–44.55)	0.392	ANOVA	F=18.94	<0.001	***

By dividing patients into 3 groups according to PTH level (low PTH, mid PTH and high PTH) and studying correlation with platelet count, MPV, PDW and P-LCR we found significant positive correlation between PTH level and MPV {P value: 0.0004 and P-LCR {P value: <0.001} without any significant correlation with Platelet count or PDW.

Table 2 — Post-Hoc Pairwise Comparisons: MPV & P-LCR (Tukey HSD / Bonferroni) Applied only to significant ANOVA results. Tukey HSD controls family-wise error rate (\alpha=0.05). Pairwise Mann-Whitney with Bonferroni correction applied for Kruskal-Wallis (MPV).

MPV (fL) — Kruskal-Wallis Post-hoc (Bonferroni)

Comparison	Mean Diff	p (adjusted)	Sig.	Interpretation
Low vs Mid PTH	+0.22	0.841	ns	No difference
Low vs High PTH	-1.54	0.001	***	High PTH \ larger MPV
Mid vs High PTH	-1.76	<0.001	***	High PTH \ larger MPV

P-LCR (%) — ANOVA Post-hoc (Tukey HSD)

Comparison	Mean Diff	p (Tukey)	Sig.	Interpretation
Low vs Mid PTH	-6.04	0.027	*	Mid PTH \ higher P-LCR
Low vs High PTH	-15.10	<0.001	***	High PTH \ markedly higher P-LCR
Mid vs High PTH	-9.06	0.003	**	High PTH \ higher P-LCR

As higher PTH level in cases of SHPT, as higher MPV and P-LCR level indicating increase number of large immature platelets due to increase and rapid rate of production of platelets to compensate high level of destruction which occurs with chronic inflammatory condition as SHPT.

Table 3 — Two-Way ANOVA: Platelet Indices ~ SHPT \times Thrombocytopenia Type II SS ANOVA. Tests independent and combined effects of SHPT status and thrombocytopenia on platelet morphology indices.

Outcome Variable	SHPT Main Effect F (p)	Thrombocytopenia F (p)	Interaction SHPT\times Thrombocytopenia F (p)	Key Conclusion
MPV (fL)	F=7.84\ (0.007**)	F=0.18\ (0.673\ {ns})	F=0.02\ (0.893\ {ns})	SHPT independently affects MPV; no thrombocytopenia effect; no interaction
P-LCR (%)	F=29.11\ (<0.001***)	F=0.04\ (0.843\{ns})	F=0.11\ (0.738\ {ns})	SHPT strongly affects P-LCR; independent of thrombocytopenia
PDW (fL)	F=0.29\ (0.593\ {ns})	F=0.52\ (0.474\t {ns})	F=0.33\ (0.566\{ns})	No significant effects on PDW
PLT (\times 10^3/\mul)	F=0.21\ (0.648) {ns})	F=20.52\ (<0.001***)	F=0.12\ (0.734\ {ns})	Only thrombocytopenia status predicts PLT (by definition); SHPT has no effect

- SHPT independently \ MPV without any effect of thrombocytopenia with no interaction between SHPT and thrombocytopenia.
- SHPT strongly \ P-LCR; independent of thrombocytopenia.

By increasing level of PTH both MPV and P-LCR are increased indicating effect of hyperparathyroidism on increasing number of large immature platelets after increase destruction.

Summary of Full Dataset Findings (n=79 with PTH):

- **PTH vs PLT count** — not significant (\rho=+0.086, p=0.451). PTH has no meaningful relationship with the number of platelets. Thrombocytopenia prevalence is actually highest in the lowest PTH tertile (28.6%), confirming the earlier finding that SHPT does not drive thrombocytopenia.
- **PTH vs MPV** — significant *** (\rho=+0.448, p<0.001). A moderate positive correlation: as PTH rises, platelets become larger. SHPT patients (PTH>300) had significantly higher MPV than non-SHPT patients (median 12.10 vs 10.45 fL, p=0.008). Kruskal-Wallis confirms a dose-response across tertiles (10.65 \ 10.40 \ 12.30 fL, H=15.87, p=0.0004).
- **PTH vs PDW** — not significant (\rho=+0.095, p=0.407). Platelet size variability (distribution width) is not related to PTH level.
- **PTH vs P-LCR** — significant *** and the strongest finding (\rho=+0.604, p<0.001). A strong positive correlation — the proportion of large platelets rises markedly with PTH. The dose-response is unmistakable: 21.5% ,26.3% ,38.7% across low/mid/high PTH tertiles (H=25.24, p<0.001). SHPT patients have nearly double the P-LCR of non-SHPT patients (34.8% vs 21.5%, p<0.001).

Clinical interpretation: PTH/SHPT in hemodialysis does not affect platelet count but does affect platelet morphology — specifically promoting larger, more activated platelets (MPV) with a higher proportion of large immature platelets (P-LCR). This suggests PTH may influence megakaryocyte maturation or platelet formation rather than thrombopoiesis per se.

Complete dataset baseline metrics:

- **79:** HD Patients with PTH measurements (n= 79 total)
- **347 pg/ml:** PTH median (IQR 156–753)
- **59.5%:** SHPT prevalence, n=47)
- **16.5%:** Thrombocytopenia ({PLT}<150, n=13)

Discussion

The present study showed a significant moderate positive correlation between PTH and MPV indicating that higher PTH concentrations were associated with larger circulating platelets. This finding was further supported by the comparison between patients with and without SHPT, in whom the median MPV was significantly higher among patients with SHPT (12.10 vs. 10.45 fL, p=0.008) and these findings was similar to Erken 2020 [9] and Zhu Y 2018 [10], while some studies demonstrated opposite results Li J 2018[11].

Moreover, patients with SHPT had markedly higher P-LCR values than those without SHPT (34.8% vs. 21.5%, p<0.001). The significant differences between all three PTH tertiles in post-hoc analysis indicate a consistent gradient rather than an isolated difference between extreme groups. Thus, P-LCR appeared to be the platelet parameter most strongly associated with PTH in this cohort [12].

Larger platelets are generally considered to have greater metabolic and hemostatic activity than smaller platelets. Therefore, the observed association between elevated PTH and both MPV and P-LCR may indicate an alteration in platelet production, maturation, or activation in patients with SHPT.

Analysis according to PTH tertiles also showed no significant difference in platelet count (p=0.308). These findings indicate that the relationship between PTH and platelets in this population is unlikely to be mediated primarily through changes in the number of circulating platelets [13].

Interestingly, thrombocytopenia was actually more prevalent among patients in the lowest PTH tertile than among those in the highest tertile (28.6% vs. 7.7%, respectively; p=0.040). This observation supports the absence of a direct relationship between PTH elevation and thrombocytopenia. Although the platelet count is an indicator of the megakaryocyte reserve in the bone marrow the numbers in the peripheral blood are influenced by many factors, including splenic elimination, inflammatory conditions, and anemia [14]. In this study, even if the platelet counts were low, it was sufficient to maintain hemostatic purposes. It is known that in advanced CKD, the function of platelets is affected rather than numbers in the peripheral blood. In line with our findings, there are studies in the literature that reported lower platelet counts in advanced CKD and heamodialysis patients Lokesh 2016 [15]. PDW showed no significant association with PTH (\rho=0.095, p=0.407), and no significant differences were detected across PTH tertiles or according to SHPT status. These findings are different from Berna and Seda study that found statistically significantly higher PDW in patients with PHPT compared to control group and postoperative 3rd month values (16). In our study the lack of change in PDW despite the marked increase in MPV and P-LCR is noteworthy. It suggests that PTH-associated platelet changes may preferentially involve platelet size and the relative proportion of larger platelets rather than producing a generalized increase in platelet size heterogeneity.

Several potential mechanisms may explain the observed relationship. PTH excess in CKD is part of the complex mineral and bone disorder of chronic kidney disease and may coexist with systemic inflammation, oxidative stress, endothelial dysfunction, and altered calcium-phosphate metabolism. These processes can influence megakaryopoiesis, platelet turnover, and platelet activation. It is therefore biologically plausible that the uremic and endocrine environment accompanying SHPT could alter the characteristics of newly released platelets [17].

The clinical relevance of these findings is important because platelet indices are routinely available as components of the complete blood count and do not require specialized testing. The strong association between PTH and P-LCR, together with the moderate association with MPV, raises the possibility that platelet indices could provide inexpensive surrogate markers of altered platelet biology in patients with CKD and SHPT. However, the present study does not establish their diagnostic or prognostic utility. In particular, larger prospective studies would be required to determine whether MPV or P-LCR can predict clinically relevant outcomes such as cardiovascular events, thrombosis, bleeding, or response to treatment of SHPT [18].

Study limitations

First, the sample size was relatively modest, with 79 patients having available PTH measurements, which may limit statistical power and generalizability. Second, the observational design precludes determination of causality; therefore, it cannot be concluded that elevated PTH directly causes increases in MPV or P-LCR.

Conclusion

Among patients undergoing hemodialysis, higher PTH levels were significantly associated with larger platelets and a greater proportion of large circulating platelets, as reflected by increased MPV and P-LCR. P-LCR demonstrated the strongest relationship with PTH, showing a clear stepwise increase across PTH severity tertiles. Conversely, PTH was not significantly associated with platelet count or PDW, and SHPT was not associated with thrombocytopenia. These findings suggest that SHPT in hemodialysis patients may be linked predominantly to qualitative changes in platelet characteristics and related to platelet dysfunction rather than quantitative platelet depletion. Further prospective and mechanistic studies are warranted to clarify the biological basis of this association and to determine whether MPV and P-LCR have clinical or prognostic value in CKD patients with SHPT.

Recommendations

More studies are needed to determine causes and risk factors of platelet dysfunction in hemodialysis patients with SHPT to avoid complications of bleeding tendency and CVD risk in these patients.

All authors had full Access to all of the data in this study and take complete responsibility for the integrity of the data and accuracy of the data analysis.

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Abbreviations:

SHPT: Secondary hyperparathyroidism
PLT: Platelet
MPV: Mean platelet volume
PWD: platelet distribution width
P-LCR: Platelet-Large Cell Ratio
HD: Hemodialysis

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