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Research Article

Effects of Parathyroidectomy on Renal Anemia in End-Stage Renal Disease

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Abstract

Objectives: This study aimed to assess postoperative changes in renal anemia parameters and erythropoietin-stimulating agent (ESA) requirements. The focus was on dialysis patients who underwent parathyroidectomy (PTx) for secondary hyperparathyroidism (SHPT).

Methods: This retrospective study included 22 dialysis patients who underwent PTx for SHPT. Demographic, biochemical, and hematological data were recorded preoperatively and at 1, 3, 6, and 12 months postoperatively. Changes in hemoglobin (Hgb), platelet (Plt) levels, and ESA doses were analyzed using appropriate parametric and nonparametric tests, and correlations between variables were assessed using Pearson's or Spearman's coefficients.

Results: The mean preoperative PTH level decreased from 2248±308 pg/mL to 468±165 pg/mL at 3 months ($p<0.001$). The mean Hgb level increased from 9.3±1.5 g/dL to 11.7±2.0 g/dL at 12 months ($p<0.05$). The ESA requirement decreased by 48%, from 22,500±13,000 IU/month to 11,700±10,400 IU/month ($p<0.05$). A moderate negative correlation was observed between increases in Hgb levels and decreases in ESA doses ($p<0.05$). In the subgroup with a biochemical response, the platelet count significantly increased at 12 months ($p<0.035$).

Conclusion: Parathyroidectomy enhances hematological parameters by increasing erythropoietin sensitivity in dialysis patients with SHPT. The reduction in ESA requirements and the increase in Hgb levels reflect improved postoperative erythropoiesis.

Keywords: Chronic kidney disease, erythropoietin resistance, erythropoiesis, parathyroidectomy, renal anemia, secondary hyperparathyroidism

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Many organ systems can be negatively affected by metabolic disorders that develop after chronic kidney disease (CKD).[1] Although various complications of the hematopoietic system develop, renal anemia is one of the most common and earliest-onset findings.[2,3] Multifactorial causes have been implicated in the development of anemia associated with CKD.[3,4] It has been suggested that many factors, including a shortened erythrocyte lifespan, chronic blood loss, increased erythropoiesis inhibitors, uremic toxins, iron and vitamin deficiencies, hemolysis, and hyperparathyroidism, contribute to anemia. Still, the most crucial factor is decreased erythropoietin production.[5,6] In these patients, elevated parathyroid hormone (PTH) levels have been shown to suppress bone marrow function, leading to osteitis fibrosa cystica and bone marrow fibrosis and thereby decreasing erythrocyte mass.[7–9] Additionally, a significant increase in hemoglobin levels and a decrease in ESA requirements were reported after parathyroidecto-

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my (PTx) in patients receiving dialysis treatment and using erythropoietin-stimulating agents (ESAs).[10]

Renal anemia not only reduces quality of life but can also lead to increased cardiovascular morbidity and mortality, as well as significant clinical problems.[11,12] Given these risks, the effectiveness of different approaches to anemia treatment in patients with CKD can directly determine prognosis.[12] Despite the importance of this issue, clinical data regarding changes in anemia parameters and their effects on erythropoietin sensitivity after PTx are limited. To address this gap, our study aimed to evaluate changes in renal anemia parameters and ESA doses during a 12-month follow-up of patients with CKD receiving dialysis treatment after PTx.

Methods

This study was approved by the İnönü University Health Sciences Non-Interventional Clinical Research Ethics Committee (Ethics Committee No. 2018-B17/18.09.2018). The study was conducted in accordance with the principles of the Declaration of Helsinki. In our study, we retrospectively analyzed patients with end-stage renal disease (ESRD) who underwent hemodialysis (HD) or peritoneal dialysis (PD) treatment in our clinic between January 2009 and December 2018. Patient data were retrieved from an electronic database. Patients with ESRD aged ≥18 years who underwent PTx for secondary or tertiary hyperparathyroidism, had renal anemia, and were using ESAs were included in the study. Anemia was diagnosed based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria (hemoglobin <13.5 g/dL in men and <12 g/dL in women).[13] Transferrin, ferritin, B12, folate, and thyroid hormone levels were measured to rule out other causes of anemia, and patients with etiologies other than renal anemia were excluded from the study. Additionally, patients aged <18 years, those with a history of kidney transplantation before or after PTx, those with dialysis insufficiency, those with a diagnosis of malignancy, and those receiving immunosuppressive therapy for any reason were excluded. Dialysis adequacy was assessed using the Kt/V ratio, which is based on urea clearance (K), dialysis duration (t), and the patient's urea volume of distribution (V). Dialysis insufficiency was defined as Kt/V<1.2 in HD patients and Kt/V<1.7 in PD patients.

Patient demographics, dialysis duration, dialysis adequacy ratio, CKD etiology, and comorbidities were recorded. ESA doses, PTH, hemoglobin (Hgb), mean corpuscular volume (MCV), mean corpuscular hemoglobin content (MCH), platelet (Plt) levels, and ESA doses were measured before PTx and at 1, 3, 6, and 12 months after PTx. To ensure the standardization of ESA doses, the formula 1 µg darbepoetin alfa=200 IU epoetin alfa was used for patients receiving darbepoetin alfa. Patients who were converted from peritoneal dialysis to hemodialysis were evaluated based on their dialysis modality at the time of surgery. Additionally, transferrin, ferritin, B12, folic acid, and thyroid hormone levels were determined.

Statistical Analysis

Based on the sample size analysis, a minimum of 16 patients would be sufficient to achieve 95% confidence (α=0.05) and 80% power (β=0.20). The 22 patients included in the study exceeded this value, demonstrating that the sample had sufficient statistical power. Data are expressed as the mean±standard deviation or median (min–max). Normality was assessed using the Shapiro–Wilk test. The Mann–Whitney U test, dependent- and independent-samples t-tests, Wilcoxon and Kruskal–Wallis tests, one-way analysis of variance (ANOVA), and Pearson or Spearman correlation coefficients were used when appropriate. Statistical significance was accepted at $p<0.05$. Analyses were performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 22 patients were included in the study. Of these, a subgroup of 18 patients was defined based on the achievement of an adequate biochemical response at 1 month after PTx; specifically, four patients were excluded from the subgroup because their PTH levels remained above 300 pg/mL at that time point. Fifteen patients received HD, and seven received PD. No significant differences were found in pre- and post-PTx Hgb levels or ESA doses according to dialysis type ($p>0.05$) (Table 1).

Total PTx was performed in eight patients, total PTx plus autotransplantation in six, and subtotal PTx in eight. The mean preoperative PTH level was 2248±308 pg/mL. It dropped sharply in the first month after surgery, reaching its lowest level of 468±165 pg/mL by month 3, and remained stable thereafter. PTH declines were statistically significant at all follow-up points compared with preoperative values ($p<0.001$). The mean pre-PTx Plt count was 206±15/mm^3, rising to 249±22/mm^3 at month 12, but this increase was not significant ($p>0.05$). The mean pre-PTx Hgb level was 9.3±1.5 g/dL. It increased significantly during follow-up, reaching 11.7±2.0 g/dL at month 12 ($p<0.05$). ESA doses decreased from 22,500±13,000 U/month before surgery to 11,700±10,400 U/month at month 12. This 48% reduction was significant ($p<0.05$) (Table 2). A moderate negative correlation was found between the increase in Hgb levels after PTx and the decrease in ESA doses ($p<0.05$) (Fig. 1).

In the 18-patient subgroup, PTH levels remained significantly lower than preoperative values at all follow-up points

Table 1. Changes in Hgb and ESA doses by dialysis type (n=22)

Dialysis type	Parameter	Preoperative	1 month	3 months	6 months	12 months	p
PD (n=7)	Hgb (g/dL)	9.5±1.3	9.1±2.3	9.6±1.8	10.1±1.6	10.3±1.7	>0.05
	EUA (U/month)	24,000±13,000	13,000±17,000	15,000±13,000	14,000±12,000	10,000±10,000	
HD (n=15)	Hgb (g/dL)	9.1±1.5	9.9±1.5	11.0±1.8	11.3±2.1	12.0±1.9	>0.05
	EUA (U/month)	18,000±12,000	18,000±10,000	16,000±9,000	16,000±11,000	15,000±10,000	

Hgb: Hemoglobin; ESA: Erythropoiesis-stimulating agent; PD: Peritoneal dialysis; HD: Hemodialysis; EUA: Emergency use authorization.

(p<0.001). Platelet counts significantly increased at 12 months compared with pre-PTx values (p<0.035) and were higher at month 12 than at months 1, 3, and 6 (p<0.007, p<0.048, and p<0.016, respectively). Hgb levels were higher at months 3, 6, and 12 than before surgery (p<0.02, p<0.004, and p<0.001, respectively) but showed no significant change at month 1 (p>0.05). ESA doses decreased significantly at months 3, 6, and 12 after PTx (p<0.011, p<0.02, and p<0.003, respectively) (Table 3). There was no significant correlation between Hgb levels and ESA doses at months 1 and 12 (p>0.05), but there was a moderate negative correlation at months 3 and 6 (p<0.029 and p<0.014, respectively) (Fig. 2).

Discussion

Although medical treatment methods are primarily preferred for secondary hyperparathyroidism (SHPT) in patients with chronic kidney disease (CKD), unresponsiveness can develop over time.[14,15] Therefore, PTx stands out as an effective treatment method for refractory cases.[14,15] In our study, patients with ESRD who underwent PTx due to SHPT unresponsive to medical treatment were examined. PTH levels decreased significantly in the early postoperative period. Postoperatively, PTH reached its lowest level in the first month and remained stable thereafter. A significant decrease in ESA requirements was observed in parallel with significant increases in Hgb levels at months 3, 6, and 12. Furthermore, a significant increase in Plt was observed at month 12 in the subgroup with a biochemical response. The negative correlation between Hgb levels and ESA doses suggests that PTx enhances the hematopoietic response by increasing erythropoietin sensitivity. Furthermore, the significant increase in Plt observed at 12 months

Table 2. Changes in PTH, Hgb, Plt and ESA doses before and after parathyroidectomy (n=22)

Timepoint	PTH (pg/mL, Mean±SD)	Hgb (g/dL, Mean±SD)	Plt (/mm³, Mean±SD)	ESA (U/month, Mean±SD)	p (PTH)	p (Hgb)	p (Plt)	p (ESA)
Preoperative	2248±308	9.3±1.5	206±15	22,500±13,000	—	—	—	—
1 mo	616±164	9.8±1.85	214±16	17,300±12,600	<0.001	>0.05	>0.05	>0.05
3 mo	468±165	10.6±1.95	215±12	15,000±12,600	<0.001	<0.02	>0.05	<0.003
6 mo	550±150	11.0±2.0	222±17	14,900±12,000	<0.001	<0.01	>0.05	<0.006
12 mo	642±172	11.7±2.0	249±22	11,700±10,400	<0.001	<0.01	>0.05	<0.001

PTH: Parathyroid hormone; Hgb: Hemoglobin; Plt: Platelet; ESA: Erythropoiesis-stimulating agent; SD: Standard deviation; mo: Month.

Table 3. Changes in PTH, Hgb, Plt counts and ESA doses before and after parathyroidectomy (n=18)

Timepoint	PTH (pg/mL, Mean±SD)	Hgb (g/dL, Mean±SD)	Plt (/mm³, Mean±SD)	ESA (U/month, Mean±SD)	p (PTH)	p (Hgb)	p (Plt)	p (ESA)
Preoperative	2248±308	9.4±1.4	213±81	21600±12800	–	–	–	–
1 mo	41±13	9.95±1.45	199±77	17200±12800	<0.001	>0.05	>0.05	>0.05
3 mo	70±27	10.5±1.8	218±57	14900±13300	<0.001	<0.02	>0.05	<0.011
6 mo	70±24	10.8±1.7	226±86	14800±12500	<0.001	<0.004	>0.05	<0.02
12 mo	66±27	11.6±1.8	260±108	11600±11000	<0.001	<0.001	<0.035	<0.003

PTH: Parathyroid hormone; Hgb: Hemoglobin; Plt: Platelet; ESA: Erythropoiesis-stimulating agent; SD: Standard deviation; mo: Month.

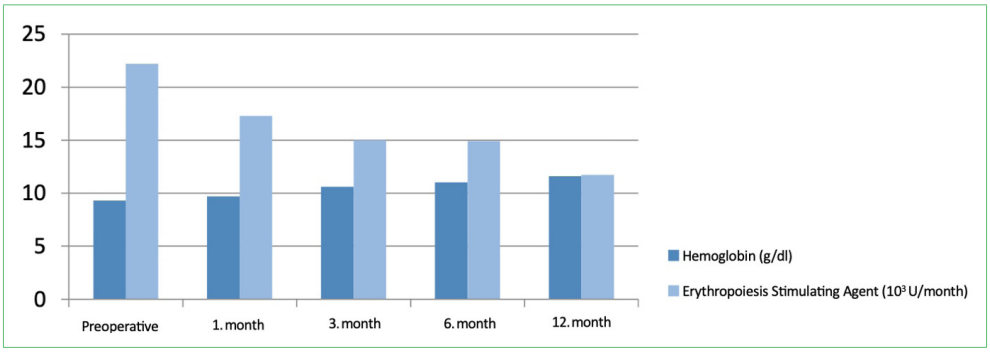

Figure 1. Relationship between hemoglobin levels and erythropoiesis -stimulating agent doses (n=22).

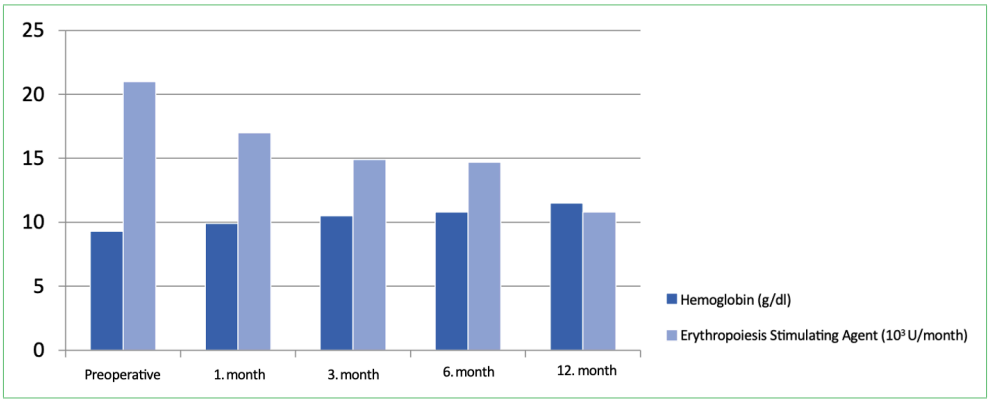

Figure 2. Relationship between hemoglobin levels and erythropoiesis-stimulating agent doses (n=18).

in the subgroup that developed a biochemical response supports the persistence of this effect at the hematological level. These findings demonstrate that PTx not only improves calcium-phosphorus balance but also positively impacts erythropoiesis dynamics, highlighting the systemic benefits of surgery.

The biochemical efficacy of PTx in treating SHPT, as evidenced by the achievement of target PTH levels, has been demonstrated in many studies.[14,15] In a study of 80 hemodialysis patients who underwent PTx, the preoperative median PTH level was 1239 pg/mL. This decreased to 11.2 pg/mL at postoperative month 1 and remained stable at 18.0 and 22.6 pg/mL at months 6 and 12, respectively ($p<0.001$).[15] Another study of 257 patients who underwent PTx for ESRD found a significant decrease in PTH levels, which remained stable until month 12.[14] In our study, in line with the literature, we observed a significant decrease in PTH levels at month 1 after surgery. These values remained stable from month 3 onward. The current findings show that PTx for SHPT in patients with ESRD can effectively control PTH levels. It also plays an important role in maintaining long-term biochemical balance. This significant and sustained reduction in PTH levels can be considered not only a biochemical success but also the underlying mechanism for improved hematological parameters.

Studies examining hematological parameters after PTx show that a decrease in PTH levels positively affects erythropoiesis and mineral-metabolic balance.[16–18] A study examining 81 cases of ESRD reported that although no significant increase in Hgb levels was found after PTx, ESA requirements decreased from 41.6 IU/kg/week to 10.3 IU/kg/week ($p=0.008$).[18] This study also found a statistically significant decrease in ESA use, particularly in patients with Hgb≤10 g/dL (OR: 20.1, 95% CI: 4.71–125, $p<0.001$) and transferrin saturation<25% (OR: 12.8, 95% CI: 2.51–129, $p<0.001$).[16] In another study examining 37 patients with ESRD and SHPT, serum PTH levels decreased from 1,871±236 pg/mL to 172±29 pg/mL ($p<0.001$) after PTx. In contrast, weekly erythropoietin doses decreased from 10,086±1,721 U to 3,514±620 U ($p<0.05$).[17] These results suggest that although some studies have not observed a significant increase in Hgb levels, substantial reductions in ESA doses improve erythropoiesis and increase erythropoietin sensitivity after PTx. Indeed, in our study, the preoperative ESA requirement of 22,500 IU/month decreased to 11,700 IU/month at 12 months, and Hgb levels increased

significantly from 3 months onward. Our study is one of the limited number of clinical studies demonstrating that reductions in ESA doses are sustained, the negative correlation between Hgb levels and ESA doses persists, and PTx enhances the functional response of erythropoiesis.

Fibroblast growth factor 23 (FGF-23) is an important factor in the development of anemia. It reduces erythropoietin production and receptor expression.[18] FGF-23 increases in CKD when phosphate levels rise and inhibits renal 1α-hydroxylase activity, leading to SHPT. High FGF-23 levels have been linked to resistant hyperparathyroidism. Conversely, patients with low levels respond better to calcitriol treatment.[19–21] After PTx, FGF-23 levels decrease significantly.[22] Although our study did not examine FGF-23 levels, we believe that PTx reduces FGF-23 levels. This reduction may remove its suppressive effect on erythropoietin, leading to increased Hgb levels and decreased ESA doses.

Studies examining changes in hematological parameters after parathyroidectomy (PTx) have reported varying results for platelet counts. For example, in a prospective study examining 29 patients with ESRD receiving HD, the platelet count was $17.8\pm4.8\times10^4/mm^3$ before PTx, $21.0\pm7.6\times10^4/mm^3$ at week 2, $20.2\pm5.0\times10^4/mm^3$ at month 3, and $21.2\pm6.2\times10^4/mm^3$ at month 12; this increase was reported to be statistically significant at all time intervals ($p<0.01$).[23] Similarly, another series of 11 patients reported that bone marrow function returned to normal after total PTx, with a positive effect on Plt.[24] In contrast, a prospective study examining 213 cases found that Plt was $202\times10^3/mm^3$ before PTx but decreased to $193\times10^3/mm^3$ at postoperative follow-up ($p=0.033$).[25] In our study, Plt levels increased in the postoperative period. We believe that the discrepancy in findings in the literature may be due to varying levels of bone marrow suppression in the preoperative period and individual differences in the rate of reversal of the chronic effects of PTH on the hematopoietic system.

Limitations

The relatively small number of patients and the retrospective design limit the generalizability of our results. Additionally, the absence of biomarker assessments, such as FGF-23, complicates conclusions about the mechanisms by which PTx improves hematological outcomes. Nevertheless, the completeness of the patient data, regular follow-up for 12 months after PTx, and assessment of both biochemical and hematological parameters are strengths. These findings significantly contribute to the literature on the long-term effects of PTx on the hematopoietic system and support future prospective studies.

Conclusion

PTx in patients with SHPT resistant to medical treatment can significantly reduce PTH levels. This leads to biochemical stabilization. The reduction in the negative effects of PTH on hematological parameters results in higher Hgb levels and lower ESA requirements. The current findings indicate that PTx improves the management of renal anemia in patients with chronic kidney disease (CKD). However, prospective studies evaluating more parameters and including larger patient groups are needed to confirm our results and clarify the mechanisms underlying its beneficial effects on anemia.

Disclosures

Ethics Committee Approval: This study was approved by the İnönü University Health Sciences Non-Interventional Clinical Research Ethics Committee (Ethics Committee No: 2018-B17).

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