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Research Article

Frequency and Risk Factors of Persistent Hyperparathyroidism Post Kidney Transplant in Sudanese

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Abstract:

Background: Persistent secondary hyperparathyroidism after renal transplantation is common, and risk factors for post-transplant secondary hyperparathyroidism are pre-transplant levels of PTH and calcium, time spent on dialysis before transplantation, and nodular hyperplasia of the parathyroid glands.

Purpose: This study aimed to determine the frequency and risk factors of persistent hyperparathyroidism one-year post-kidney transplant.

Methodology: Cross-Sectional Hospital-based study was conducted in a period from September 2018 to March 2019 in Ibn Sina Hospital and Ahmed Gasim Hospital and covered 81 study participants.

Results: Pre-kidney transplantation PTH level, (58%) reported mild increase in PTH level, (37.1%) reported moderate increase while only (4.9%) had a severe increase. Post-kidney transplant PTH, (60.5%) reported normal levels of PTH, (34.6%) a mild increase while (4.9%) reported a moderate decrease in PHT.

After three months of PKT, serum calcium was high in (38.3%) and low phosphate (2.5%). After one year of PKT, serum calcium was high in (8.6%) and low phosphate (2.5%). a significant reduction in the PTH after KT compared with PTH levels before KT (p-value < 0.001) and the association between PKT level of PTH with age, gender and duration of dialysis before KT (P value < 0.05).

Conclusion: Obtaining a level of PTH adequate to the graft function and to normalize levels of calcium, and phosphorus as a primary clinical objective for patients with secondary HPT after renal transplantation in Sudan.

Risk factors such as age, duration of dialysis, and pre-transplantation level of PTH should be taken into account in the management policy after renal transplantation.

Keywords: frequency; risk; factors; persistent; Hyperparathyroidism; post-kidney; transplant

Introduction

One of the most common complications in cases with chronic renal failure is secondary hyperparathyroidism (HPT). Several pathways have been linked as the means by which HPT is initiated and sustained in renal failure, notwithstanding the gaps in our current understanding.

The disturbance of the endocrine parathyroid hormone – vitamin D axis has been linked to abnormalities in both calcium/ phosphate and vitamin D metabolism. 3Furthermore, secondary hyperparathyroidism is a nearly universal finding in cases with end- stage renal complaint. undressed

secondary hyperparathyroidism causes osteoporosis, pathological fractures, and order monuments [1].

These symptoms and the necessity of dialysis have serious counteraccusations on case's quality of life. order transplantation remains the treatment of choice in renal failure and is reported to resolve numerous of the endocrine and metabolic imbalances of hyperparathyroidism [2]

In the transplant community, it's presently standard procedure to stay a full-time following transplant before considering parathyroidectomy.

This is grounded on earlier exploration showing that, following a successful order transplant, post-transplant hypercalcemia generally goes down in time. Consequently, a vigilant waiting approach is generally employed for asymptomatic cases with elevated parathyroid hormone situations in the time following transplantation. [3] Successful order transplantation corrects the endocrine and metabolic imbalances and the main abnormalities responsible for secondary HPT in the first months. however, parathyroid hormone (PTH) affair doesn't always return to normal after the original positive events. In cases with satisfactory renal function, elevated complete PTH (iPTH) situations have been seen in further than 25 of cases one-time following transplantation. [2]

Only many data are available in the literature on the long- term natural history of HPT after successful renal transplantation. This can be explained by the lack of a dependable and precise iPTH assay until the late 1980s. Prior to this, estimates of physiologically active PTH were squishy, inconsistent, and non-uniform due to the use of colourful PTH fractions in radioimmunoassay for iPTH. [3] The preface of an immunoradiometric assay (IRMA) to measure complete PTH in the late 1980s redounded in an important enhancement in the study of HPT in both non-uraemic and uraemic individualities.[3]

It's well honoured that cases with end stage renal conditions (ESRD) have altered bone mineral metabolism and hyperplastic parathyroid glands. phosphorus, and calcitriol a drop in serum parathyroid hormone (PTH)

occurs by about 1 time after successful renal transplantation. [3] still, some renal transplant donors with indeed good renal function indeed by one time may continue to have elevated position of PTH and patient hypercalcemia [3]. In some studies, deficient resolution of hyperparathyroidism is reported in further than 50 of cases despite excellent allograft function. This may be due to deficient normalization of renal function and/ or to a deficient resolution of there-transplant parathyroid hyperplasia. Although some enhancement has passed, threat factors for this morbidity remain inadequately understood. [3].

When treating a patient with chronic kidney disease (CKD) the clinician is handling one of the most challenging tasks in clinical practice. Few failing organs have such a widespread impact on the organism as the kidney. Patients with end stage renal disease (ESRD) have a mortality risk 10-20 times higher than patients with normal renal function, and the major cause of death is cardiovascular disease. [4] Patients with ESRD also develop bone disease and have a higher risk of fractures compared to the general population. The complex relation between the kidney, the bones and vascular calcifications led to the definition in 2005 of chronic kidney disease – Mineral and Bone Disorder (CKD-MBD) by Kidney Disease International Improving Global Outcome (KDIGO). [4] Secondary hyperparathyroidism (sHPT), with increased volume of the parathyroid glands and increasing levels of parathyroid hormone (PTH), is a major element of CKD-MBD and it develops over time in the vast majority of patients with CKD. Vascular and bone-related outcomes as well as increased mortality are a direct effect of sHPT. [5] After renal transplantation, renal function increases and the mineral metabolism dramatically improves. Renal function increases and mineral metabolism significantly improves following renal transplantation. The understanding of sHPT following renal transplantation has grown in the last several years. Nevertheless, there are currently no target PTH levels for recipients of renal transplants, and treatment recommendations are still ambiguous. [4] In addition, there is disagreement over whether PTX should be done prior to or following renal transplantation. Research has indicated that performing PTX following transplantation results in a decline in renal function. The benefits of PTX on mortality rates observed in dialysis patients have not been demonstrated in patients receiving a kidney transplant. [4, 5] First Point. The endocrine system comprises four small glands called parathyroid glands, which are typically located behind the thyroid's left and right lobes. Supernumerary glands are occasionally present and are typically located in the thymus tissue, though they can also be found in other places. [6] Chief cells and oxyphil cells are the two cell types that make up the parathyroid gland. Parathyroid hormone is produced and released by chief cells. Although oxyphil cells proliferate in CKD and express a large number of genes found in chief cells, their exact role is still unknown. [6] The preservation of a healthy calcium homeostasis is the parathyroid gland's primary duty. When calcium is present, calcium-sensing receptors (CaSR)16 on the surface of chief cells cause PTH to be produced and released. The preservation of normal calcium homeostasis is the parathyroid gland's primary job. When calcium levels fall below a specific threshold, calcium-sensing receptors (CaSR)16 on the surface of chief cells trigger the synthesis and release of PTH. PTH release is inhibited by high calcium levels. Three mechanisms exist for parathyroid hormone to mobilize calcium. The first method involves triggering

osteoblasts, which then trigger osteoclasts, causing the skeleton to release calcium into the net. [7]. The second method involves boosting calcium reabsorption in the kidney's distal tubuli. The activation of vitamin D, which in turn acts to increase calcium absorption in the intestine, is the third way PTH increases calcium. PTH release is also stimulated by elevated phosphate levels, though the exact mechanism is unknown. [7]. So, throughout this context, this study aimed to measure the frequency and risk factors of persistent hyperparathyroidism post-kidney transplant in Sudanese patients.

Material and Methods: This was a descriptive, cross-sectional hospital-based study conducted at Ibn Sina Hospital and Ahmed Gasim Teaching Hospital, during the period from September 2018 to March 2019. Ibn Sina Hospital is a tertiary hospital, located in the Khartoum capital of Sudan in Mohamed Najeeb Street and Ahmed Gasim Hospital in Khartoum North. These are the two hospitals in Sudan where kidney transplantation operations are done. In Ibn Sina Hospital there is a separate kidney transplant refer clinic per week with an average of 60 patients in each clinic, while in Ahmed Gasim Hospital there are two refer clinics.

The study populations were patients who underwent kidney transplants attending Ibn Sina

and Amed Gasim hospitals and had measured parathyroid hormone levels before kidney transplantation. Inclusion criteria, recipients of first renal transplant, no history of parathyroidectomy before transplant, and availability of pre-transplant parathyroid hormone measurement in the 2 months preceding transplantation the study excluded suboptimal renal function post-transplantation (creatinine more than 1.3 mg/dl.

The participants were interviewed about age, gender, time of transplantation duration of hemodialysis before kidney transplantation, baseline creatinine Parathyroid hormone level before and one year after transplantation, Calcium, phosphorus, and alkaline phosphatase levels 3month and one year after transplantation.

Sampling techniques via simple random sampling, it was included all patients with available elevated parathyroid hormone levels before transplantation, statistical analysis was performed using SPSS 25 software (SPSS, Chicago, IL, USA), and variables were compared using Student's t-test (for paired data) or post hoc test, Mann–Whitney U test for non-parametric data. For categorical data, comparisons were performed using the chi-square test (X2) or Fisher's exact test, as appropriate. Statistical significance was set at $P < 0.05$.

Ethical clearance was obtained from the ethical committee of the Sudan Medical Specialization Board, Council of Internal Medicine. An official agreement from the general managers of Ibn Sina &Ahmed Gasim Teaching Hospital preceded the study. Ethical considerations were taken, presented to the ethics review committee, and approved, data was handled with a high degree of confidentiality throughout the study, and written informed consent was obtained from all participants in the study.

Results

During the study period, a total of 81 study participants were included. Two-thirds of them (66.7%) were less than 40 years in age and more than half of them (55.6%) were males with a male: female ratio of about 1: 1.3. Table (1)

Demographic	Frequency	Percent
Age – years		
20_29	17	21.0
30_39	37	45.7
40_49	15	18.5
50_60	12	14.8
Gender		
Male	45	55.6
Female	36	44.4
Total	81	100.0

Table 1: Sociodemographic characteristics of the study participants (n = 81)

Concerning the post-kidney transplant OTH, our study found that more than half of them (60.5%) reported normal levels of PTH, (34.6%) a mild increase while (4.9%) reported a moderate decrease in PHT. After three months of PKT, serum calcium was high among only (38.3%) and low phosphate was reported among (2.5%). After one year of PKT, serum calcium was high among only (8.6%) and low phosphate was reported among (2.5%) as detailed in Table (2).

Investigation			Frequency	Percent
Parathyroid hormone		**Normal (≤ 65 pg/ml)**	49	60.5
		Mild increase (66_ 200 pg/ml)	28	34.6
		Moderate increase (201_400 pg/ml)	4	4.9
Calcium	**Three months PKT**	**Low (< 8.5 mg/dL)**	0	0.0
		Normal (8.5 – 10.2 mg/dL)	50	61.7
		High (> 10.2 mg/dL)	31	38.3
	One-year PKT	**Low (< 8.5 mg/dL)**	0	0.0
		Normal (8.5 – 10.2 mg/dL)	74	91.4
		High (> 10.2 mg/dL)	7	8.6
Phosphate	**Three months PKT**	**Low (< 2.5 mg/dL)**	2	2.5
		Normal (2.5 – 4.5 mg/dL)	79	97.5
		High (>4.5 mg/dL)	0	0.0
	One-year PKT	**Low (< 2.5 mg/dL)**	2	2.5
		Normal (2.5 – 4.5 mg/dL)	79	97.5
		High (>4.5 mg/dL)	0	0.0

Table 2: the distribution of the study participants according to their post-transplant investigation results (n = 81)

The study reported a significant reduction in the PTH after KT compared with PTH levels before KT (p-value < 0.001) as detailed in Table (3).

Parathyroid hormone level	Before		After	
	Freq.	Percent	Freq.	Percent
Normal (≤ 65 pg/ml)	0	0.0	49	60.5
Mild increase (66_ 200 pg/ml)	47	58.0	28	34.6
Moderate increase (201_400 pg/ml)	30	37.0	4	4.9
Severe increase (> 401_ pg/ml)	4	4.9	0	0.0
Total	81	100.0	81	100.0

Table 3: comparison between the parathyroid level before and after kidney transplant (n = 81)

P - value < 0.001 Our study found that there is a significant association between PKT level of PTH with age, gender, and duration of dialysis before KT (P value < 0.05) as detailed in Table (4-5).

General characteristics		Post-kidneytransplant parathyroid hormone level								P value
		Normal (n = 49)		Mild (n = 28)		Moderate (n = 4)		Total (n = 81)		
		Freq.	%	Freq.	%	Freq.	%	Freq.	%	
Age– years	20_29	16	32.7	1	3.6	0	0.0	17	21.0	< 0.001
	30_39	28	57.1	8	28.6	1	25.0	37	45.7	
	40_49	4	8.2	10	35.7	1	25.0	15	18.5	
	50_60	1	2.0	9	32.1	2	50.0	12	14.8	
Gender	Male	21	42.9	20	71.4	4	100.0	45	55.6	0.010
	Female	28	57.1	8	28.6	0	0.0	36	44.4	

Table 4: The relation between the post-kidney transplant parathyroid hormone level with demographical characteristics (n = 81

Table (4) The relation between the post-kidney transplant parathyroid hormone level with demographical characteristics (n = 81)

Moreover, the levels of calcium and phosphate were also related to the level of parathyroid hormone after KT (p-value < 0.05) as shown in Table (6-7).

Our study showed that nearly two-thirds of the (64.2%) had been transplanted within the period 2015 – 2017 and the majority Table (5) The relation between the post-kidney transplant parathyroid hormone level with demographical characteristics (n = 81)

Duration of PKT dialysis	Post-kidney-transplant parathyroid hormone level								P value
	Normal (n = 49)		Mild (n = 28)		Moderate (n = 4)		Total (n = 81)		
	Freq.	%	Freq.	%	Freq.	%	Freq.	%	
< 2	17	34.7	0	0.0	0	0.0	17	21.0	< 0.001
2_4	32	65.3	21	75.0	0	0.0	53	65.4	
> 4	0	0.0	7	25.0	4	100.0	11	13.6	

Table 7: the relation between the post-kidney transplant parathyroid hormone level with Phosphate level (n = 81)

(89%) was on hemodialysis for less than four years before the transplantation Figure (1) and (2).

Calcium level		Post-kidney transplant parathyroid hormone level								P value
		Normal (n = 49)		Mild (n = 28)		Moderate (n = 4)		Total (n = 81)		
		Freq.	%	Freq.	%	Freq.	%	Freq.	%	
Three months PKT	Low	0	0.0	0	0.0	0	0.0	0	0.0	< 0.001
	Normal	43	87.8	7	25.0	0	0.0	50	61.7	
	High	6	12.2	21	75.0	4	100.0	31	38.3	
One-year PKT	Low	0	0.0	0	0.0	0	0.0	0	0.0	0.002
	Normal	48	98.0	24	85.7	2	50.0	74	91.4	
	High	1	2.0	4	14.3	2	50.0	7	8.6	

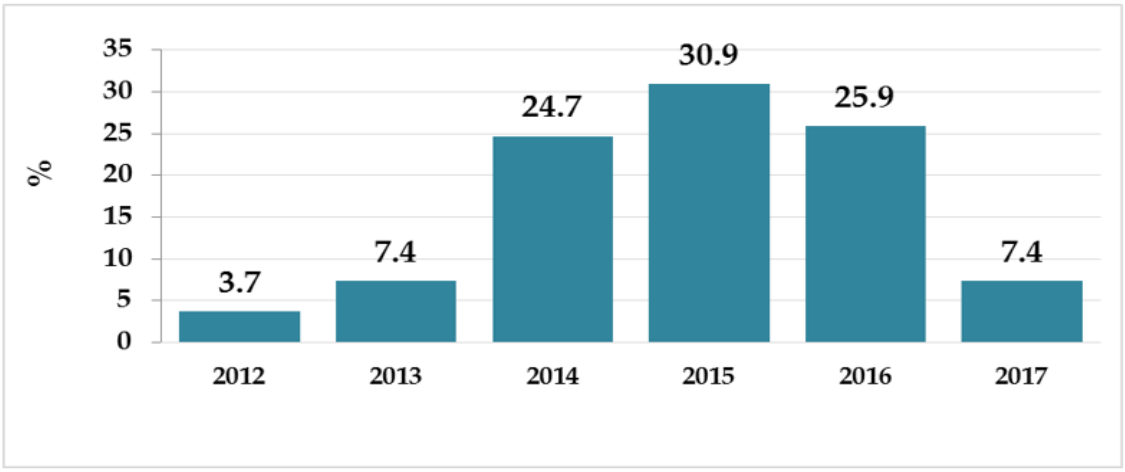

Figure 1: the distribution of the study participants according to their time of transplantation (n = 81)

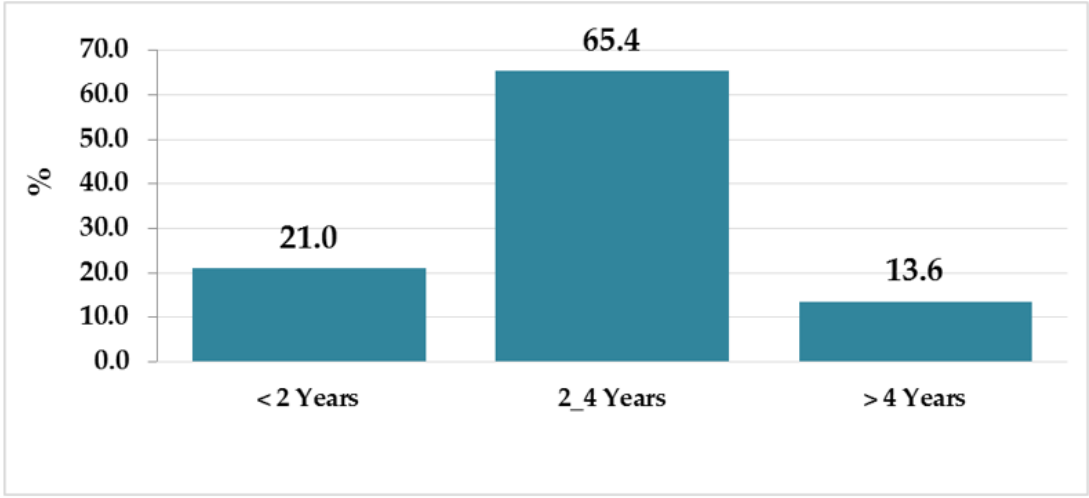

Figure 2: Distribution of the study participants according to their duration of hemodialysis before transplantation (n = 81)

Study participants reported normal levels of serum creatinine on the baseline. Regarding the pre-kidney transplantation PTH level, more than half of them (58%) reported a mild increase in PTH level, (37.1%) reported a moderate increase while only
(4.9%) had a severe increase Figure (3).

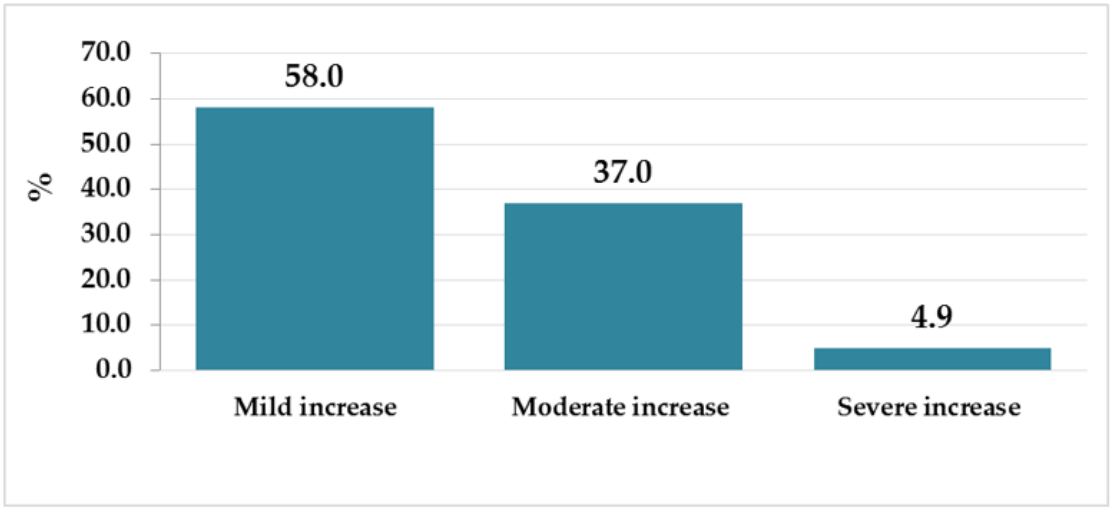

Figure 3: Distribution of the study participants according to their pre-transplant Parathyroid hormone (n = 81)

Discussion

This study aimed to know the frequency and risk factors of persistent hyperparathyroidism one-year post-kidney transplant and covered 81 study participants. Two-thirds of them (66.7%) were less than 40 years in age and more than half of them (55.6%) were males with male: female ratio of about 1: 1.3. In the context of this issue. In Taiwan, Cheng sp etal stated that female gender is associated with an increased risk of parathyroid nodular hyperplasia and parathyroidectomy rate in dialysis patients Women with secondary hyperparathyroidism had worse bone mineral metabolism before parathyroidectomyand they stated that additional research regarding target organ susceptibility to hyperparathyroidism in different genders is warranted. [8] in United States, Lou I. etal claimed that many parameters have been studied as possible risk factors for predicting persistent post-transplant hyperparathyroidism, including female gender, elevated pre-transplant parathyroid hormone (PTH) and hypercalcemia, but none have been prospectively validated. [9]

Our study reported that the majority (89%) was on hemodialysis for less than four years before the transplantation. Correspondingly, In UK, Alireza H. etal reported that patients were on dialysis for an average period of 17.4 months prior to transplantation. [10]

Regarding the pre kidney transplantation PTH level, more than half of them (58%) reported mild increase in PTH level, (37.1%) reported moderate increase while only (4.9%) had severe increase. Similar findings were reported in Norway, by Gøransson LG et al who found that one hundred and three patients (80%) had an elevated level of PTH prior to kidney transplantation. [11]

Concerning the Post-kidneytransplant PTH, our study found that more than half of them (60.5%) reported normal level of PTH, (34.6%) mild increase while (4.9%) reported moderate decrease in PHT. Likewise, In UK, Alireza H. etal found tha hyperparathyroidism was in 12 (9.9%) and 7 (5.7%) patients three months and one year after transplantation respectively. [10] In more depth, in Belgium, Evenepoel P etal add that post-transplant iPTH levels correlated significantly with transplant kidney function. [12] Furthermore in United States, Wolf M etal found THAT The results did not differ across the low and high PTH strata, and rates of persistent hyperparathyroidism remained higher than 40% when defined using a higher

PTH threshold greater than 130 pg/mL and confirmed that persistent hyperparathyroidism is common after kidney transplantation. [13] Further studies should determine if persistent hyperparathyroidism or its treatment influences long-term post transplantation clinical outcomes.[13]

After three months PKT, serum calcium was high among only (38.3%) while after one-year PKT, serum calcium was high among only (8.6%). A higher proportion was reported in Norway, by Gøransson LG etal (23%) had hypercalcaemia. [9] Moreover, in UK, Alireza H. etal, confirmed that an increase in the serum Ca and a decrease in serum P and iPTH level was seen in the patients after transplantation (P< 0.001).[10]

Our study reported that low phosphate was reported among (2.5%) after three months and also after one-year PKT. A differ findings, reported in US by Wolf M, who stated that in its most severe form, disordered mineral metabolism after kidney transplant is characterized by hyperphosphatemia, and markedly elevated levels of parathyroid hormone (PTH) .[16]Moreover, hypophosphatemia present clinicians with management challenges because they may jeopardize graft function and bone health and exacerbate fracture and cardiovascular risk. [13]

The study reported a significant reduction in the PTH after KT compared with PTH levels before KT (p value < 0.001). Within similar context, In Italy, Messa P etal the severity of pre-existing secondary hyperparathyroidism is the main factor determining its persistence after renal transplantation.[14]

Moreover, similar results were reported in Norway, by Gøransson LG etal who concluded that patients transplanted pre-emptively had a statistically significantly lower iPTH level compared with patients who had received dialysis. [9] Furthermore, In United States, Jahromi AH, etal found that (26.6%) attained normal PTH between 1 and 2 years, with the remaining (43.1%) categorized as having HPT. [15] likewise, In France, Bonarek H etal found that the basal PTH decreased from 195 +/- 54 pg/ml before KT to 70 +/- 12 pg/ml six months later (P < 0. 005). [16]

Our study found that there was a significant association between PKT level of PTH with age, gender and duration of dialysis before KT (P value < 0.05) and with levels of calcium and phosphate were also related with the level of parathyroid hormone after KT (p value < 0.05). Similarly, In UK, Alireza H. etal found that elderly patients and patients with longer duration on dialysis had an increased risk of developing post-transplant hyperpara-thyroidism and hypercalcemia in the first year post transplant (P< 0.05) and they confirmed that the age and duration on dialysis before transplantation seems to be important risk factors for post-transplant hyperparathyroidism [[10] Other differ set of factors was reported In United States, by Jahromi AH, etal who claimed that comparing normalization of PTH by 2 years to HPT patients, obesity (P<0.001), months on dialysis (P<0.001), and delayed graft failure (P=0.006) were predictive of non-normalization of PTH. [15] Moreover, In Belgium, Evenepoel P etal relrealized that patient with persistent HPT had significantly elevated serum levels of iPTH, calcium and phosphorus at the time of RT, and had spent a longer time on dialysis. [12]

Our study had some limitations. The relatively limited number of study participant (81 study participants from limited number of study areas - Ibn Sina hospital and Ahmed Gasim hospita) may affect negatively the probability of finding significant relationships between different factors and characteristics with HPTH among Sudanese patients who had been renal transplanted.

Conclusion

The study reported a significant reduction in the PTH after KT compared with PTH levels before KT and there was a significant association between PKT level of PTH with age, gender, and duration of dialysis before KT and with levels of calcium and phosphate were also related with the level of parathyroid hormone after KT.

This study concluded that the age of presentation is less than 40 -years, with males more than females, and there is a mild and moderate increase of parathyroid hormone in pre-kidney transplantation PTH level.

our study found that post-kidney transplant OTH more than half reported normal levels of PTH, with a one-third mild increase while small reported a moderate decrease in PHT.

After three months of PKT, serum calcium was high among only two-fifths, and low phosphate was reported in lower cases.

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Consent: All participants provided informed consent.

Approval: Ethical clearance and supportive letters were obtained from the Sudan Medical Specialization Board (SMSB) and& the Educational Development Centre (EDC). Written permission was obtained from Ibn Sina and Ahmed Gasim hospitals.

Author contribution

All authors contributed to the manuscript writing of the manuscript.

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No potential conflicts of interest were reported by the authors.

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Data Sharing

The authors agreed on the date-sharing policy of this journal.

References

1. Yuen, N. K., Ananthakrishnan, S., & Campbell, M. J. (2016). Hyperparathyroidism of renal disease. The Permanente Journal, 20(3).
2. Lou, I., Foley, D., Odorico, S. K., Leverson, G., Schneider, D. F., Sippel, R., & Chen, H. (2015). How well does renal transplantation cure Hyperparathyroidism? Annals of Surgery, 262(4), 653-659.
3. David, D. S., Sakai, S., Brennan, B. L., Riggio, R. A., Cheigh, J., Stenzel, K. H., Rubin, A. L., & Sherwood, L. M. (1973). Hypercalcemia after renal transplantation. New England Journal of Medicine, 289(8), 398-401.
4. Gupta, G., Unruh, M. L., Nolin, T. D., & Hasley, P. B. (2010). Primary care of the renal transplant patient. Journal of General Internal Medicine, 25(7), 731-740.
5. Liu, W., & Wynne, J. J. (2019). Figure 12 from: Liu WX, Wynne JJ (2019) cave millipede diversity with the description of six new species from Guangxi, China. Subterranean biology 30: 57-94.
6. Medas, F., Erdas, E., Loi, G., Podda, F., Barca, L., Pisano, G., & Calò, P. G. (2019). Intraoperative parathyroid hormone (PTH) testing in patients with primary hyperparathyroidism and PTH levels in the normal range. BMC Surgery, 18(1).
7. Tang, E., Bansal, A., Novak, M., & Mucsi, I. (2018). Patient-reported outcomes in patients with chronic kidney disease and kidney transplant—Part 1. Frontiers in Medicine, 4.
8. Cheng, S., Yang, T., Lee, J., Chen, H., Wu, C., Liu, T., & Liu, C. (2011). Gender differences among patients with secondary Hyperparathyroidism undergoing

Parathyroidectomy. Journal of Surgical Research, 168(1), 82-87.

9. Lou, I., Foley, D., Odorico, S. K., Leverson, G., Schneider, D. F., Sippel, R., & Chen, H. (2015). How well does renal transplantation cure Hyperparathyroidism? Annals of Surgery, 262(4), 653-659.
10. Salehipour, M., Jalaeian, H., Salahi, H., Bahador, A., Davari, H., Nikeghbalian, S., Sagheb, M., Raiss-Jalali, G., Roozbeh, J., Behzadi, S., Janghorban, P., Sepas, H., & Malek-Hosseini, S. (2007). Are large nonfunctional kidneys risk factors for Posttransplantation urinary tract infection in patients with end-stage renal disease due to autosomal dominant polycystic kidney disease? Transplantation Proceedings, 39(4), 887-888.
11. Gøransson, L. G., & Bergrem, H. (2007). Renal transplant and secondary hyperparathyroidism. Scandinavian Journal of Urology and Nephrology, 41(6), 553-557.
12. Evenepoel, P., Claes, K., Kuypers, D., Maes, B., Bammens, B., & Vanrenterghem, Y. (2004). Natural history of parathyroid function and calcium metabolism after kidney transplantation: A single-centre study. Nephrology Dialysis Transplantation, 19(5), 1281-1287.
13. Wolf, M., Weir, M. R., Kopyt, N., Mannon, R. B., Von Visger, J., Deng, H., Yue, S., & Vincenti, F. (2016). A prospective cohort study of mineral metabolism after kidney transplantation. Transplantation, 100(1), 184-193.
14. Messa, P., Sindici, C., Cannella, G., Miotti, V., Risaliti, A., Gropuzzo, M., Di Loreto, P. L., Bresadola, F., & Mioni, G. (1998). Persistent secondary hyperparathyroidism after renal transplantation. Kidney International, 54(5), 1704-1713.
15. Lou, I., Foley, D., Odorico, S. K., Leverson, G., Schneider, D. F., Sippel, R., & Chen, H. (2015). How well does renal transplantation cure Hyperparathyroidism? Annals of Surgery, 262(4), 653-659.
16. Bonarek, H., Merville, P., Bonarek, M., Moreau, K., Morel, D., Aparicio, M., & Potaux, L. (1999). Reduced parathyroid functional mass after successful kidney transplantation. Kidney International, 56(2), 642-649.

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