อุบัติการณ์และปัจจัยที่เกี่ยวข้องกับภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไต

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Incidence and Factors Associated with Hyperparathyroidism Following Kidney Transplantation

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Abstract

Background: Kidney transplantation (KT) can reverse renal function and improve patient survival while the intact parathyroid hormone (iPTH) level persists high in some kidney transplant recipients (KTRs). Thus, we examined the incidence and factors associated with post-KT hyperparathyroidism. Objectives: We aimed to examine the incidence and factors associated with post-KT hyperparathyroidism. Methods: A single center, retrospective study of 312 KTRs between January 1997 and December 2020 was investigated. We included KTRs having serum iPTH level after 6 months post-KT with stable renal function. KTRs without post-KT iPTH level were excluded. We defined hyperparathyroidism (HPT) according to estimated glomerular filtration rate (eGFR); iPTH >65 and >130 pg/mL for eGFR ≥60 and <60 mL/min/1.73 m^2, respectively. We divided patients into groups of post-KT HPT and those without HPT. We analyzed the incidence, associated factors and compared between groups. STATA version 15.1 was used for statistic analyses. Results: The data revealed 144 KTRs, we found that incidence of post-KT HPT was 85 (59.0%). Among these, there were 67 patients having pre-transplant iPTH level and 33 (49.3%) were defined as persistent HPT. Factors associated with post-KT HPT were female and high pre-transplant iPTH level. We found one case of biopsy-proven nephrocalcinosis, might be the sequelae of post-KT HPT. Neither report of death nor cardiovascular events relating to post-KT HPT. Conclusion: Our study showed that the incidence of hyperparathyroidism following kidney transplantation accounted for 59.0% despite the recovery of renal function. Female and high pre-transplant iPTH level were its associated factors.

Keywords: Kidney transplantation (KT), Hyperparathyroidism (HPT), Hyperparathyroidism following kidney transplantation

บทคัดย่อ

ภูมิหลัง: การปลูกถ่ายไตเป็นวิธีการรักษาผู้ป่วยโรคไตเรื้อรังระยะสุดท้ายให้การทำงานของไตกลับสู่สภาวะใกล้เคียงปกติ และเพิ่มอัตราการรอดชีวิตของผู้ป่วย แต่ยังพบระดับฮอร์โมนพาราไทรอยด์สูงได้ในระยะหลังปลูกถ่ายไต ผู้วิจัยจึงต้องการศึกษาอุบัติการณ์และปัจจัยที่เกี่ยวข้องกับภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไต วัตถุประสงค์: ผู้วิจัยจึงต้องการศึกษาอุบัติการณ์และปัจจัยที่เกี่ยวข้องกับภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไต วิธีการ: ผู้วิจัยทำการศึกษาแบบย้อนหลังในผู้ป่วยจำนวน 312 ราย ที่ได้รับการปลูกถ่ายไตในช่วงเดือนมกราคม พ.ศ. 2540 ถึงเดือนธันวาคม พ.ศ. 2563 ในโรงพยาบาลราชวิถี โดยคัดเลือกผู้ป่วยที่มีการทำงานของไตคงที่และมีผลตรวจระดับฮอร์โมนพาราไทรอยด์หลังการปลูกถ่ายไต 6 เดือนขึ้นไป นิยามของภาวะฮอร์โมนพาราไทรอยด์สูงผิดปกติขึ้นกับการทำงานของไต โดยระดับฮอร์โมนพาราไทรอยด์มากกว่า 65 พิโคกรัมต่อมิลลิลิตร เมื่ออัตราการกรองของไต 60 มิลลิลิตรต่อนาทีขึ้นไป และระดับ

ฮอร์โมนพาราไทรอยด์มากกว่า 130 พิโคกรัมต่อมิลลิลิตรเมื่ออัตราการกรองของไตน้อยกว่า 60 มิลลิลิตรต่อนาที ผู้วิจัยทำการแบ่งกลุ่มผู้ป่วยตามภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไต ทำการวิเคราะห์และหาปัจจัยที่เกี่ยวข้อง การวิเคราะห์สถิติโดยใช้โปรแกรม STATA 15.1 **ผล:** ผู้วิจัยพบผู้ป่วยจำนวน 144 ราย เข้าเกณฑ์การศึกษาและพบอุบัติการณ์ของภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไตร้อยละ 59 ในจำนวนนี้มีผู้ป่วย 67 ราย ที่มีผลตรวจระดับฮอร์โมนพาราไทรอยด์ก่อนปลูกถ่ายไตและเข้าได้กับภาวะฮอร์โมนพาราไทรอยด์สูงต่อเนื่องหลังปลูกถ่ายไตจำนวน 33 (ร้อยละ 49.3) ปัจจัยที่เกี่ยวข้องกับภาวะฮอร์โมนพาราไทรอยด์สูงต่อเนื่องหลังปลูกถ่ายไต คือ ผู้ป่วยเพศหญิงและมีระดับฮอร์โมนพาราไทรอยด์สูงก่อนปลูกถ่ายไต มีรายงานพบภาวะ nephrocalcinosis จากผลตรวจพยาธิชิ้นเนื้อไตที่ปลูกถ่ายในผู้ป่วย 1 ราย ซึ่งน่าจะเป็นผลจากภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไต การศึกษานี้ไม่พบผู้ป่วยภาวะแทรกซ้อนระบบหัวใจหลอดเลือดหรือการเสียชีวิตที่เกี่ยวเนื่องจากภาวะนี้ **สรุป:** ภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไตพบได้บ่อย ร้อยละ 59 แม้ว่าการทำงานของไตจะดีขึ้นโดยปัจจัยสำคัญคือเพศหญิงและมีระดับฮอร์โมนพาราไทรอยด์สูงก่อนปลูกถ่ายไต

คำสำคัญ: การปลูกถ่ายไต, ภาวะฮอร์โมนพาราไทรอยด์สูง, ภาวะฮอร์โมนพาราไทรอยด์สูงหลังปลูกถ่ายไต

Background

The secondary hyperparathyroidism is one of a major character of chronic kidney disease and mineral bone disease (CKD-MBD), it can occur early in the course of CKD, and its prevalence increases as kidney function declines, particularly at the estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2.[1] Thus the secondary hyperparathyroidism is a common disorder found in most of the patients with end-stage renal disease (ESRD). It develops as a consequence of the inability of the failing kidney to maintain the complex homeostasis between the serum parathyroid hormone (PTH), calcium (Ca), phosphate (P), and vitamin D. This dysregulation of parathyroid function is associated with the serious bone abnormalities and cardiovascular effects[2] such as the osteitis fibrosa, adynamic bone disease, calciphylaxis, impaired erythropoiesis leading to anemia, and coronary artery diseases. In CKD patients, the parathyroid hormone (PTH) levels are used as a surrogate marker of CKD-MBD treatment, complying with the compliance of diet restriction.[2] High PTH levels can aggravate the development of autonomy of the parathyroid glands.

Most kidney transplant recipients (KTRs) have improved their renal function after transplantation, resulting in decline in serum PTH back to the normal level within 6 months post-transplantation.[3] Some of them still have high PTH levels up to 15 years post kidney transplantation[4]. The definition of post-KT hyperparathyroidism (post-KT HPT) varies according to the cut-off level of serum PTH and the time interval post-transplantation of which each study employed. Overall, prevalences of post-KT HPT range from 14% to 89%.[3-19] KTRs who have both pre-KT and post-KT HPT, are defined as persistent hyperparathyroidism. Risk factors associated with post-KT HPT include old age, long dialysis vintage, high level of serum PTH pre-transplantation, high serum alkaline phosphatase (ALP), parathyroid gland hyperplasia, history of taking cinacalcet before transplantation, and poor graft function.[1, 7, 8, 12, 13, 19] Moreover, previous study[9] by Prakobsuk, et al. in Thai population showed that hyperparathyroidism and high fractional excretion of phosphate were predictors of death-censored graft loss in KTRs. Similarly, Joke, et al.[1] showed that hyperparathyroidism was a risk factor for death-censored graft loss. Therefore, the post-KT HPT should be properly recognized, identified and corrected to reduce the risk of complications.

Treatment of post-KT HPT has been reported in several studies.[13, 18] Fortunately, the intact PTH (iPTH) levels usually decrease over time post kidney transplantation. Majority of KTRs receive conservative managements to maintain the serum levels of calcium and phosphate within the normal range. Cases with more severe post-KT HPT require aggressive treatments, including calcimimetics and surgery. Surgical parathyroidectomy is effective, and usually indicated for those KTRs with failed medication and/or parathyroid adenoma.[18]

Thus, we aimed to examine the incidence and factors associated with post-KT hyperparathyroidism.

Materials and methods

We conducted a retrospective cohort study. The data were obtained from medical records at Rajavithi Hospital from October 2021 to February 2022. Patients aged more than 18 years old, who received kidney transplantation from January 1997 to December 2020, had follow-up visits at the kidney transplantation

clinic, Rajavithi Hospital, had stable kidney function, and had blood tests for iPTH level after 6 months post-transplantation, were enrolled. Those who received simultaneous multi-organ transplantation were excluded. Informed consent was waived due to a retrospective nature of this study. This study was approved by local ethical committee, Rajavithi Hospital. The IRB approval number 209/2564 on September 8, 2021.

Pre-KT demographic data were collected. These included age, gender, co-morbidities, mode of preceding dialysis, dialysis vintage, and type of kidney donor. To circumvent fluctuation in laboratory data values during the pre-KT period, only those which were measured within 6 months prior to transplantation were included for analysis. These included serum iPTH level, creatinine, calcium; normalized to a serum albumin concentration of 4.0 mg/dL, phosphate, computed calcium-phosphate product, alkaline phosphatase (ALP), vitamin D; 25(OH)D level, and hemoglobin. Previous treatment of the pre-KT HPT was collected as name and dosage of drugs, or type of surgery for treatment of pre-KT HPT were also collected.

Post-KT laboratory data were evaluated at 6-month post-transplantation. These included serum iPTH level, creatinine, eGFR; calculated by CKD-EPI, calcium; normalized to a serum albumin concentration of 4.0 mg/dL, phosphate, computed calcium-phosphate product, alkaline phosphatase (ALP), vitamin D; 25(OH)D level, and hemoglobin. Treatment associated with parathyroid hormone, calcium, phosphate and vitamin D level after KT were collected as name of the drug, its dosage and history of post-KT parathyroidectomy.

We defined hyperparathyroidism according to estimated glomerular filtration rate (eGFR); iPTH >65 and >130 pg/mL for eGFR ≥60 and <60 mL/min/1.73 m^2, respectively.[20] If HPT persisted through to a period of 6 months post-KT, post-KT HPT was diagnosed. KTRs having both pre- and post- KT HPT were defined as persistent hyperparathyroidism.

Among those who had pre-transplant iPTH data, we classified them into 2 group; those with or without post-KT HPT. Factors associated post-KT HPT were evaluated by using univariate and multivariate analyses.

The complications and any events occurred after KT were collected, including symptomatic coronary artery disease; symptomatic CAD, stroke; ischemic or hemorrhagic, peripheral artery disease, or death from any causes.

Statistical Analysis

Continuous data were analyzed, using mean and standard deviation for normal distribution, and using median and interquartile range (IQR) for a skewed distribution. Categorical data were compared using chi-square or Fisher's exact test. Comparing the medians, we used Wilcoxon rank sum tests. Logistic regression analysis was used to obtain odds ratios (OR) and adjusted odds ratios (aOR). The stepwise backward logistic regression was used to select final model. Multivariate models were developed by adjusting for covariates with $p < .1$ in univariate models. STATA version 15.1 (StataCorp LLC) was used for statistic analyses.

Results

A total of 312 patients had undergone KT between January 1997 and December 2020, and 144 patients were eligible in this study. No cases were excluded due to simultaneous multi-organ transplantation. Of these patients, 64 (44.4%) were female. Median age at the time of KT was 41 (IQR 33.5-47.0) years old. Types of transplanted kidney were 56 (38.9%) and 88 (61.1%) of living donor and deceased donor KT, respectively. 136 patients (94.4%) received HD prior to transplantation and 93 (64.6%) had dialysis vintage ≥2 years. Median pre-transplant iPTH level was 401 (155-713) pg/mL (n=67 cases), and 9 (6.3%) underwent parathyroidectomy prior to KT. 3 (2.1%) patients received parathyroidectomy after KT due to hyperparathyroidism with parathyroid gland hyperplasia and failed medication. Post-KT biochemical characteristics included serum creatinine 1.4 (1.1-1.8) mg/dL, eGFR 55.5 (41-68.5) mL/min/1.73 m^2, serum calcium 9.8 (9.4-10.3) mg/dL, serum phosphate 3 (2.5-3.6) mg/dL, the calcium-phosphate product 30.1 (25.5-34.7) mg/dL, alkaline phosphatase 78.5 (56.5-93) U/L, 25(OH)D level 23.2 (15.4-29.3) ng/mL, and hemoglobin 12.2 (11-13.6) g/dL. Median post-transplant iPTH level was 118.5 (74-214.5) pg/mL. Characteristics were summarized in **Table 1**.

Compared to those without post-KT HPT, patients with post-KT HPT were more likely to be female, receive HD prior to KT, have higher GFR, lower phosphate level, lower CaP product post-transplantation. Additionally,

patients with post-KT HPT had higher PTH levels pre-KT.

We found that 85 (59%) patients had high PTH and defined as post-KT HPT, which 50 (34.7%) had iPTH > 65 pg/mL with eGFR 60 mL/min/1.73m^2, and 35 (24.3%) had iPTH > 130 pg/mL with eGFR 60 mL/min/1.73m^2.

Among 144 eligible cases, 67 patients had pre-transplant iPTH levels, of which 33 cases (49%) had both pre-and post-KT HPT, so called persistent HPT. In patients with persistent HPT, 13 (39.4%) of 33 had hypercalcemia. We also found that 4 (6%) of 67 patients were new-onset of post-KT HPT, and there was no difference of serum calcium between patients with persistent HPT and those with new-onset HPT.

To evaluate the factors associated with hyperparathyroidism following KT, all of 144 KTRs were divided into post-KT HPT and non post-KT HPT groups according to the post-transplant iPTH levels by eGFR status. On univariate analyses, We found that patients with post-KT HPT were more likely to be female (p=.01, Odds ratio (OR) 2.69), having hemodialysis prior to KT (p=.06, OR 4.7), having lower CaP product; <30 mg/dL (p=.03, OR 2.18) and lower phosphate; <2.5 mg/dL (p=.01, OR 4.51) post-trasnplantation. On multivariate analysis, we found that female (p=.006, OR 2.75), and lower post-KT phosphate; <2.5 mg/dL (p=.01, OR 4.65) were significant associated with post-KT hyperparathyroidism. There was no significant associated for dialysis vintage, longer duration post-transplant, hypocalcemia, hypophosphate between groups. The results of univariate and multivariate analyses were summarized in **Table 2**.

There was a report of 1 (0.7%) patient having documented symptomatic CAD and the final diagnosis defined by cardiac catheterization was double vessel disease. Five KTRs died, and cause of death was infection for all of them. There was 1 patient presented with late allograft dysfunction and diagnosed biopsy-proven nephrocalcinosis. Post-KT complications and events are summarized in **Table 3**. To evaluate between post-KT HPT and non post-KT HPT groups, there was no significant complications and events occurred.

Table 1: Characteristics of patients with Post-KT hyperparathyroidism (n = 144)

Patient Characteristics	Overall (n=144)	Post-KT HPT (n=85)	No post-KT HPT (n=59)	p-value
Female, n (%)	64 (44.4)	46 (54.1)	18 (30.5)	.01*[a]
Age at KT (year) (IQR)	41 (33.5-47.0)	41 (34.0-47.0)	41 (33.0-47.0)	.73[b]
Donor type, n (%)	LDKT 56 (38.9) DDKT 88 (61.1)	LDKT 30 (35.3) DDKT 55 (64.7)	LDKT 26 (44.1) DDKT 33 (55.9)	.29[c]
Mode of RRT prior to KT, n (%)	HD 136 (94.4) PD 8 (5.6)	HD 83 (97.7) PD 2 (2.4)	HD 53 (89.8) PD 6 (10.2)	.04*
Dialysis vintage prior to KT >2 years, n (%)	93 (64.6)	58 (68.2)	35 (59.3)	.27[c]
Pre-KT iPTH (pg/dL) (n=67) (IQR)	401 (155-713)	467 (236-782)	231 (104-556)	.03*[b]
Pre-KT parathyroidectomy, n (%)	9 (6.3)	4 (4.7)	5 (8.5)	.36[c]
Post-KT Parathyroidectomy, n (%)	3 (2.1)	3 (3.5)	0	.27[a]

Table 1: Characteristics of patients with Post-KT hyperparathyroidism (n = 144) (Continue)

Patient Characteristics	Overall (n=144)	Post-KT HPT (n=85)	No post-KT HPT (n=59)	p-value
Biochemical characteristics, post-transplantation				
Serum creatinine (mg/dL) (IQR)	1.4 (1.1-1.8)	1.2 (1.1-1.5)	1.7 (1.3-2.0)	<.01*[b]
eGFR (ml/min/1.73^2) (IQR)	55.5 (41-68.5)	62 (48-73)	44 (38-59)	<.01*[b]
Serum calcium (mg/dL) (IQR)	9.8 (9.4-10.3)	9.9 (9.5-10.3)	9.7 (9.2-10.1)	.05[b]
Serum phosphate (mg/dL) (IQR)	3 (2.5-3.6)	2.9 (2.5-3.5)	3.2 (2.8-3.8)	<.01*[b]
CaP product (mg/dL) (IQR)	30.1 (25.5-34.7)	28.8 (25.1-33.5)	31.2 (27.0-36.7)	.01*[b]
ALP (U/L) (IQR)	78.5 (56.5-93)	77.0 (58.5-97.0)	78.5 (53.5-91.5)	.53[b]
25(OH)D level (ng/mL) (IQR)	23.2 (15.4-29.3)	29.3 (20.9-33.6)	19.8 (14.9-25.4)	.14 [b]
Hemoglobin (g/dL) (IQR)	12.2 (11-13.6)	12.3 (10.9-13.6)	11.9 (11.2-14.0)	.72 [b]
iPTH (pg/dL) (IQR)	118.5 (74-214.5)	186 (123-255)	69 (53-97)	<.01*[b]

[a] Fisher's exact test, [b] Wilcoxon rank sum test, [c] Chi-square test

* Statistically significant differences, p-value < .05.

KT = Kidney transplantation, HPT = Hyperparathyroidism, LD = Living donor, DD = Deceased donor,
HD = Hemodialysis, PD= Peritoneal dialysis, iPTH = Intact parathyroid hormone, eGFR = Estimated glomerular filtration rate,
CaP = Calcium-phosphate, ALP = Alkaline phosphatase

Table 2: Factors associated with post-KT hyperparathyroidism

Factors	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Sex; Female	2.69 (1.33, 5.41)	.01	2.75 (1.34-5.64)	.006
Age at KT	0.85 (0.43, 1.65)	.62		
Donor type; deceased donor	1.44 (0.73, 2.85)	.29		
Mode of RRT before transplantation; hemodialysis	4.7 (0.91, 24.15)	.06		
Dialysis vintage before KT; 2 years	1.47 (0.74, 2.94)	.27		
Pre-KT parathyroidectomy	0.53 (0.14, 2.08)	.37		
Post-KT serum calcium; > 10.5 mg/dL	1.19 (0.48, 2.94)	.71		
Post-KT CaP product; < 30 mg/dL	2.18 (1.11, 4.31)	.03		
Post-KT serum phosphate; < 2.5 mg/dL	4.51 (1.46, 13.9)	.01	4.65 (1.47-14.67)	.01
Hemoglobin (g/dL)	0.96 (0.8, 1.14)	.62		
Post-KT Duration; 5 years	0.56 (0.27, 1.17)	.13		

OR = Odds ratio, aOR = adjusted odds ratio, CI = Confidence interval, KT = Kidney transplantation,
eGFR = Estimated glomerular filtration rate, iPTH = Intact parathyroid hormone, CaP = Calcium-phosphate, ALP = Alkaline phosphatase

Table 3: Post-KT complications and events in patients with hyperparathyroidism (n = 144)

Post-KT complications and events	n (%)	Remarks
• Symptomatic CAD	1 (0.7)	Double vessel disease
• troke	-	
• PAD	-	
• Bone fracture	-	
• Death from any causes	5 (3.5)	5 patients died due to infection
• Nephrocalcinosis	1 (0.7)	Biopsy-proven diagnosis of kidney allograft

CAD = Coronary artery disease, PAD = Peripheral artery disease

Discussion

In this single-center cohort, we found the incidence of post-KT HPT were significantly high (59%) among Thai KTRs compared to the previous studies.[3, 4, 10, 17] Among those patients having pre-transplant iPTH levels, we found the high incidence (49.3%) of patients with persistent hyperparathyroidism, who had both pre- and post-KT HPT.

Successful kidney transplantation can improve the mineral metabolism and hyperparathyroidism. It usually takes up to 6 months post-transplantation for the time of iPTH recovery. While over 6-month post-transplantation, post-KT hyperparathyroidism is still frequently observed, as shown in our study. The major cause of post-KT HPT might be pre-existing pre-KT HPT, particularly in cases with iPTH level > 800 pg/mL. Severe hyperparathyroidism usually correlates with parathyroid gland hyperplasia and/ or tertiary hyperparathyroidism, which is usually refractory to medical management and needs calcimimetic drugs and/ or surgical intervention. Some patients with severe HPT while on the transplant waitlist have to postpone the transplant operation due to an extra time needed for correction of the HPT problem.

Our study demonstrated that half of KTRs still had high iPTH levels after 6-month post-transplantation despite the recovery of renal function. Moreover, half of them were persistent hyperparathyroidism, who had HPT since pre-transplant period. We assumed that those patients with post-KT HPT might have parathyroid gland hyperplasia and/or adenoma, however, we did not have evidences such as imaging to confirm those conditions. There were 6% developed new-onset hyperparathyroidism post-transplantation, which could imply those conditions might be from pre-transplant HPT as in the persistent HPT over primary hyperparathyroidism. Two reasons supported pre-transplant HPT in new-onset HPT, including; firstly, some patients with hyperparathyroidism might not show the high iPTH level due to the unsteady iPTH level with the aggressive PTH suppression during the treatment of secondary hyperparathyroidism. Secondly, there was no significant hypercalcemia, which commonly found in primary hyperparathyroidism.

Factors associated with post-KT HPT were female patients and high iPTH level pre-transplant as shown in the previous studies.[8, 10, 12, 14, 17] Our study found that longer dialysis vintage (> 2 years) did not correlate with post-KT HPT, which implied to longer time on dialysis affecting some indicators especially residual renal function, that could be the significant risk for developing HPT. We also found that post-KT hypophosphatemia (<2.5 mg/dL) was another associated factor, which could be from phosphaturia due to the effect of parathyroid hormone. However, we did not measure the 24-hour urinary phosphate excretion to confirm this postulation. To evaluate the association between post-KT HPT and post-transplant duration, we found no difference between patients receiving KT within 5 years and those with longer duration. While there was uncommon for HPT persisting after 12 months post-transplantation.

As for the impact of post-transplant HPT, our study did not find any complications, such as cardiovascular, or fracture events occurred. There were 5 patients died due to infection, which might not correlate with HPT. There was one case of biopsy-proven nephrocalcinosis, which the patient presented with late allograft dysfunction and

post-KT HPT was the proposed cause of nephrocalcinosis without any other causes of allograft dysfunction, however, we did not have any other evidences such as pre-implantation (donor) or previous allograft biopsy to strongly confirm or exclude the other causes.

In contrast to our findings, others have demonstrated an association between post-KT HPT and adverse transplant outcomes.[1, 9, 21, 22] Although we found the high incidence of hyperparathyroidism following KT without significant adverse impact on patient and graft survival, however, there was variable time post-KT at which PTH levels were collected, and there was no protocol biopsy or other tools to confirm the evidences of allograft dysfunction other than serum creatinine.

The strengths of our study include we have reported the high incidence of HPT after 6 months post kidney transplantation and pre-transplant high iPTH levels as the associated factor of. We also found the persistent HPT after KT longer than 6-12 months. Therefore, we would recomend monitoring and management post-KT HPT appropriately to prevent the long term effects.

There were several limitations of this study. Firstly, we conducted in a single center. Secondly, the retrospectively review, there were lots of missing data, in particular, the pre-transplant iPTH level, so the number of patients having complete data were only one-fifth of the total number (67 of 312). Thirdly, we did not have data those might be the associated factors, including residual renal function prior to KT, parathyroid gland size, vitamin D level, history of cinacalcet use, dialysis prescription, and imaging of parathyroid gland, to clarify the risk factors of pre-transplant HPT those might affect.

Conclusions

In conclusion, hyperparathyroidism following kidney transplantation occurs frequently (59.0%) despite the recovery of renal function. Female and high pre-transplant iPTH level are its associated factors.

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