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Abstract

Background: Pre-emptive kidney transplantation (PEKT) is beneficial for patients, improves graft survival and minimizes the complications associated with chronic kidney disease. However, reports on pediatric PEKT are limited, and little is known about the parathyroid hormone (PTH) abnormalities and calcium-phosphorus disorders (CPD) in this condition. This study was the first to report on mineral disorders in pediatric PEKT patients during a one-year period.

Methods: We conducted a comparative examination of the abnormalities in calcium, phosphorus, calcium-phosphorus products and PTH before and one year after living donor kidney transplantation among PEKT and Non-PEKT patients.

Results: Thirty three patients were included. The patients were divided into two groups: PEKT (n=13, five months in CKD stage 4-5) and non-PEKT (n=20, 23.5 months in dialysis). Mean age at transplantation was 9.6 ± 4.9 years. Hypercalcemia and hyperphosphatemia were observed before and after transplantation in the PEKT and non-PEKT groups, and 15% of patients in each groups exhibited bone disorders and

ectopic calcifications associated with mineral disorders. The mineral disorders were present for approximately 3 months after transplantation in both treatment groups.

Conclusions: No significant differences in PTH or CPD were noted; moreover, normalization of abnormal values did not differ between the PEKT and non-PEKT groups. Compared to non-PEKT, PEKT did not improve the course of mineral metabolism disorders. Mineral and bone disorder (MBD) treatment was likely insufficiently provided to pediatric PEKT patients. To obtain the maximum advantage of PEKT, calcium and phosphorus levels should be strictly controlled before kidney transplantation.

Keywords

Calcium, parathyroid hormone, pediatric, Phosphorus, Pre-emptive kidney transplantation,

Introduction

Secondary hyperparathyroidism is a frequently occurring complication in patients with kidney failure. In patients with kidney transplantation, high levels of parathyroid hormone (PTH) have various effects during the perioperative period, such as systemic calcification [1-2], graft loss [3], and cardiovascular disease (CVD) [4-6]. Furthermore, after kidney transplantation, the improvement in kidney function leads to hypercalcemia and hypophosphatemia [7-10] because of the prolonged hyperparathyroidism that occurs. Therefore, strict control of calcium and phosphorus levels is required to prevent the progression of Chronic Kidney Disease mineral and bone disorder (CKD-MBD) and CVD. A limited number of reports have examined the abnormalities in bone mineral metabolism in pediatric kidney transplant patients [11-13]. Pre-emptive kidney transplantation (PEKT) often provides good results [14-15] post-transplantation. As the incidence of pediatric PEKT is expected to increase, studies on the pre- and postoperative management of the abnormalities in bone mineral metabolism are very important.

We conducted a comparative examination of the changes in calcium, phosphorus, and PTH levels in two groups of pediatric patients: those undergoing PEKT and those receiving peritoneal dialysis prior to kidney transplantation. This study is the first to examine the abnormalities in mineral metabolism of pediatric PEKT patients during a one-year period. The results demonstrated abnormalities in PTH and calcium-phosphorus disorders (CPD) during the perioperative period of pediatric PEKT.

Methods

We studied 33 patients who underwent living donor kidney transplantation at Toho University Omori Medical Center, Tokyo, Japan, between April 2008 and November 2011. This center accepts pediatric kidney transplant patients from all over Japan. The patients were divided into two groups (PEKT and non-PEKT) based on the use of peritoneal dialysis prior to kidney transplantation. The PEKT group was defined as patients who did not use dialysis immediately before to transplantation. All treatment was conducted by continuous cycler peritoneal dialysis (CCPD) or CCPD + continuous ambulatory peritoneal dialysis (CAPD). Medical records were retrospectively examined

to obtain the following information: treatment before transplantation; levels of serum calcium, phosphorus, intact parathyroid hormone (iPTH), and calcium and phosphorus products before and after transplantation; and complications associated with calcium and phosphorus. Normal ranges for age (calcium, phosphorus, iPTH and calcium and phosphorus products) were based on standard laboratory data in Japan [16] and the criteria for chronic kidney disease (CKD) stage 4-5 in the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines. Chest and abdominal X-rays, abdominal ultrasonography, electrocardiography, and echocardiography were obtained for all patients before kidney transplantation. The estimated glomerular filtration rate (eGFR) was calculated using the Schwartz method. Unpaired two-tailed t-test, Welch t-test, Mann-Whitney U test and Fisher's exact test were used for the statistical analysis. P-values less than 0.05 were considered statistically significant. All statistical analyses were conducted using StatMate version 4.01 (Advanced Technology for Medicine & Science, Tokyo, JAPAN). This study was approved by the Ethics committee of our institution (No. M16227).

Results

Patient population

We studied 23 males and 10 females who underwent kidney transplantation during a period of 3 years and 8 months. The patients were divided into two groups, PEKT (n = 13) and non-PEKT (n = 20), based on their use of peritoneal dialysis prior to kidney transplantation (Table 1). The mean age at transplantation of all patients was 9.6 ± 4.9 years.

Primary disease

In this population, hypodysplastic kidney was the most frequent cause of CKD (11 patients; 33%), followed by shock kidney (4 patients; 12%) and juvenile nephronophthisis (3 patients; 9%), as shown in Table 2. The number of cases of hypodysplastic kidney was significantly higher in the PEKT group than the non-PEKT group ($p < 0.01$).

Kidney function

The eGFR value immediately before transplantation was 17.7 ± 10.6 ml/min/1.73 m^2 in the PEKT group. The eGFR values after kidney transplantation were equivalent in both groups. Specifically, the eGFR 2-4 months after transplantation was 88.0 ± 24.8 ml/min/1.73 m^2 in the PEKT group and 90.6 ± 24.4 ml/min/1.73 m^2 in the non-PEKT group (p = 0.7742). The eGFR at 10-12 months after transplantation was 80.4 ± 14.8 ml/min/1.73 m^2 in the PEKT group (n = 12) and 84.7 ± 19.7 ml/min/1.73 m^2 (n = 19) in the non-PEKT group (p = 0.5225). (2-4 months after transplantation indicate 3.1±0.5 months: average ± standard deviation, 10-12 months after transplantation indicate 11.9 ±0.4 months: average ± standard deviation.)

Medical treatment

Calcium and phosphorus abnormalities were medically treated in 30 patients (91%) by administering calcium carbonate, vitamin D, or phosphorus binders (calcium carbonate and vitamin D: 16 patients, vitamin D: 7 patients, calcium carbonate: 4 patients, phosphorus binders: 2 patients, phosphorus binders and vitamin D: 1 patient). Three patients (9%) with calcium and phosphorus abnormalities were not medically treated

from the previous hospital; 2 of these patients were in the PEKT group. We concluded that treatment was necessary for three patients. Oral administration of cinacalcet hydrochloride and parathyroidectomy were not performed in either group. Immunosuppressive agents are one of the four types of drugs used for kidney transplantation [17]. Specifically, the following immunosuppressive agents were used for ABO incompatible kidney transplantation: calcineurin inhibitors (cyclosporine 18 patients, tacrolimus 14 patients, tacrolimus hydrate 1 patient), antimetabolites (mycophenolate mofetil 31 patients, mizoribine 2 patients), methylprednisolone sodium succinate 33 patients, basiliximab 32 patients, and rituximab 4 patients. Transfusion required the use of a hydrating solution (Na 77 mEq/L, K 0 mEq/L, Cl 77 mEq/L) and lactated Ringer solution (Na 131 mEq/L, K 4 mEq/L, Ca 3 mEq/L) in all patients. Immediately after transplantation, diuretics were used in 28 patients (PEKT 9 patients, non-PEKT 19 patients), and antihypertensive agents were used in 6 patients (PEKT 3 patients, non-PEKT 3 patients).

Duration of dialysis

The PEKT group included three patients who underwent peritoneal dialysis during the course of treatment. Two of them in the PEKT group experienced a long dialysis duration. Patient No. 16 experienced bacterial peritonitis six times, and the peritoneal dialysis catheter was removed twice. Patient No. 17 underwent peritoneal dialysis for five years before first kidney transplantation. A second kidney transplantation was performed after 10 years due to renal dysfunction. In the non-PEKT group, the dialysis length prior to transplantation was 1 year and 11 months (range, 2 months to 7 years and 3 months).

The duration from CKD stage 4-5 (or the start of peritoneal dialysis) to kidney transplantation was five months (range, 1 to 28 months) in the PEKT group (n = 11) and 31.5 months (range, 3 to 87 months) in the non-PEKT group (n = 20) ($p < 0.001$). As an exception, one patient who underwent second kidney transplantation had CKD stage 3a, and one patient was classified with CKD stage 4 immediately before transplantation in the PEKT group.

Changes in calcium levels

Changes in calcium levels before and after kidney transplantation in both groups are presented in Figure 1. Before transplantation, hypercalcemia was observed in 10 patients in both groups, including 3 patients in the PEKT group. On the kidney transplant day (day 0), a significant difference in calcium levels was not noted in either group. Although temporary, hypocalcemia was observed in both the PEKT and non-PEKT groups immediately after transplantation; the calcium concentration soon returned to normal levels. After transplantation, temporary hypercalcemia was observed in 16 patients (48%), including 5 patients in the PEKT group ($p = 0.284$). Furthermore, 8 patients (24%) demonstrated chronic hypercalcemia more than 3 months after transplantation. Of these, 1 patient belonged to the PEKT group ($p = 0.082$), and the serum calcium level at 2-4 months after transplantation was not significantly higher in the PEKT group than in the non-PEKT group. In addition, five patients continued to indicate chronic hypercalcemia at 1 year, including 1 patient in the PEKT group ($p = 0.331$).

Changes in phosphorus levels

Changes in phosphorus levels before and after kidney transplant in both groups are presented in Figure 2. Before transplantation, hyperphosphatemia was observed in 15 patients in all patients, including 4 patients in the PEKT group ($p = 0.157$). A significant difference in phosphorus was not noted on the day of kidney transplantation (DAY 0) in either group. Although temporary, hypophosphatemia was observed in both the PEKT and non-PEKT groups immediately after transplantation; the phosphorus levels soon returned to normal. The minimum serum phosphorus level after transplantation was 2.0 ± 0.8 (mg/dl) in all patients. The minimum serum phosphorus level occurred at 4.2 ± 2.7 days and 4.3 ± 2.8 days after transplantation in the PEKT and non-PEKT groups, respectively ($p = 0.94$). Serum phosphorus levels returned to normal (2.5 mg/dl) after 1 day (range, 1 to 21 days) and 4 days (range, 1 to 131 days) in the PEKT and non-PEKT groups, respectively.

One patient (non-PEKT), who had the longest dialysis period (87 months) of all patients, demonstrated chronic hypophosphatemia (≤ 2.0 mg/dl) at 1 year after transplantation. Some patients had hypercalcemia, hyperphosphatemia and hyperparathyroidism before kidney transplantation.

Changes in PTH levels

The maximum PTH level immediately before transplantation was 445 pg/ml (26–2104 pg/ml) in all patients, and the PTH levels decreased in both groups within 3 months after transplantation (Figure 3). Overall, the PTH level immediately before transplantation was 283 pg/ml (56–954 pg/ml) in patients with hypercalcemia for longer than 3 months after transplantation (n = 8) and 196 pg/ml (6–2104 pg/ml) in patients with hypercalcemia for less than 3 months after transplantation (n = 25) (not significant).

Changes in calcium-phosphorus products

Before transplantation, calcium and phosphorus product levels of 55 mg^2/dl^2 or greater were observed in 24 patients (73%), including 10/13 (77%) patients in the PEKT group and 14/20 (70%) patients in the non-PEKT group. Extremely high levels of calcium and phosphorus products (≥100mg^2/dl^2) were observed in the PEKT group before transplantation. Immediately before transplantation, calcium and phosphorus product levels of 55 mg^2/dl^2 or greater were observed in 14 patients (42%), including 4/12 (33%)

patients in the PEKT group and 10/20 (50%) patients in the non-PEKT group. The calcium and phosphorus product levels declined immediately after transplantation and then gradually recovered. In contrast, after transplantation, calcium and phosphorus products levels of 55 mg^2/dl^2 or more were observed in the non-PEKT group (Figure 4), though there was no statistically significant difference.

Complications

Calcium gluconate hydrate was used in 7 patients (PEKT 4 patients, non-PEKT 3 patients) to treat asymptomatic hypocalcemia. Acute tubular necrosis immediately after transplantation was observed in two patients (PEKT 1, non-PEKT 1). Moreover, other complications were noted in 7 patients (cerebral hemorrhage 1, sepsis 1, congestive heart failure 1, anemia 1, cyclosporine a-induced toxicity 1, cytomegalovirus infection 1, convulsions 1).

Calcification

Before transplantation, ectopic calcification associated with abnormalities in bone mineral metabolism was observed in 5 patients in all patients (Table 1). Of these, 1

patient (non-PEKT) did not show hypercalcemia and hyperphosphatemia before transplantation. In addition, hyperparathyroidism was observed in two patients (PEKT 1, non-PEKT 1).

(Calcification of PEKT)

Systemic osteosclerosis, kidney calcification, and cardiovascular calcification were observed in 2 patients in the PEKT group. Specifically, patient No. 4 had a hypodysplastic kidney and exhibited hypercalcemia (max 14.3 mg/dl) until kidney transplantation. This patient had already acidemia, osteosclerosis of the skull and short stature from visiting our center. The duration from CKD stage 4-5 to kidney transplantation was three months. On the Other hand, Patient No. 25 had a shock kidney and had previously developed hyperphosphatemia (10.4 mg/dl) and high levels of calcium and phosphorus products ($\geq 100 mg^2/dl^2$). This patient had cardiovascular calcification and gallstones. Hyperphosphatemia and hypercalcemia were not corrected until kidney transplantation. The duration from CKD stage 4-5 to kidney transplantation was three months.

(Calcification of Non-PEKT)

Skull hyperostosis, pancreatic calcification and rickets were observed in 3 patients in the non-PEKT group. Patient No. 8 had nephronophthisis without hypercalcemia. This patient had pancreatic calcification but no vascular calcification. Patient No. 9 had a shock kidney and had previously developed hypercalcemia (11.4 mg/dl) and skull hyperostosis. The patient's hypercalcemia was improved until transplantation. Patient No. 10 had a bilateral multicystic dysplastic kidney with hyperparathyroidism (PTH 1571 pg/ml, ALP 1860 IU/l), rickets and deformity of the upper and lower limbs. Peritoneal dialysis was started at eight months of age, and the patient was given low phosphorus milk by tube feeding due to frequent vomiting. The duration from the start of peritoneal dialysis to kidney transplantation was 18 months (Patient No. 8), 52months (Patient No. 9) and 20 months (Patient No. 10).

Discussion

Although there are some reports on patient and graft survival, on the other hand, there are few reports on mineral disorders in pediatric PEKT. The present study revealed that the frequency of mineral disorders was similar in the PEKT and non-PEKT groups. Furthermore, mineral bone disorder (MBD) treatment was likely insufficiently provided to pediatric PEKT patients, although duration of chronic renal failure was short.

Calcium and phosphorus abnormalities and hyperparathyroidism are also significant complications during the perioperative period in PEKT patients.

PEKT presently accounts for 20–25% of all transplantations in Japan. PEKT minimizes the occurrence of complications associated with chronic kidney failure and demonstrates a satisfactory patient survival and kidney graft survival rate [14, 18-21]. PEKT has also attracted attention due to the cost of dialysis [22]. Furthermore, PEKT provides a chance to increase the final height of children [23-24]. The progression of CKD is associated with increased CVD [25-26]. In this study, 13 patients (39%) opted for PEKT, possibly due to the recent interest in this procedure among medical institutions and patients' families. However, treatment with PEKT requires continuous

medical management of chronic kidney failure immediately before transplantation and involves difficulties in managing high blood pressure, acidosis, uremia, and abnormalities in mineral metabolism.

Hypercalcemia caused by hyperparathyroidism was noted in both groups before kidney transplantation. Moreover, hyperphosphatemia due to reduced phosphorus excretion was observed in both groups before kidney transplantation. The post-transplantation hypercalcemia observed in this study is believed to be caused by prolonged hyperparathyroidism [9, 27], which systemically increases the risk of ectopic calcification. In this study, iPTH levels were extremely high in both groups before transplantation; high iPTH levels are considered to be a cause of hypercalcemia one year after transplantation. Hypercalcemia and hypophosphatemia after kidney transplantation are also associated with the duration of dialysis before transplantation [10]. Prolonged hyperparathyroidism has been reported in adults for at least 1 year after transplantation [28], and high levels of iPTH along with a history of dialysis are known to trigger pre-transplantation factors [29]. Levels of iPTH are reported to decrease by 3–6 months after transplantation and to gradually return to normal [30-31]; however, the

normalization of iPTH at day 90 is greater in PEKT than in non-PEKT patients [13]. In this study, no marked difference was observed in iPTH levels between the PEKT and non-PEKT groups before transplantation, but the levels observed in both groups were extremely high. This result suggests that the medical management of abnormalities in mineral metabolism was not sufficient in the PEKT group. Furthermore, before transplantation, we found that many patients exceeded the level of iPTH (100–200 pg/ml) considered to avoid complications in patients with peritoneal dialysis [32]; however, patients in both groups returned to normal levels within 3 months after transplantation.

The known complications of CKD in childhood include developmental delays, decreased final height, and renal osteodystrophy [33]. Dietetic treatment and phosphorus binders or vitamin D are commonly used to treat CKD-MBD in children [34]. In this study, 11 patients (85%) in the PEKT group and 19 (95%) in the non-PEKT group were treated with calcium carbonate and vitamin D or phosphorus binders. A relatively low ratio of CKD-MBD management in the PEKT group suggests the difficulty of providing early interventions, including dietetic treatment (e.g., phosphorus-restricted

diet). In general, when hypercalcemia or secondary hyperparathyroidism continues after transplantation despite medical treatment, the administration of cinacalcet hydrochloride or parathyroidectomy is considered. However, reductions in the function of the transplanted kidney have been reported after the initiation of such treatments [30, 35-36]. Furthermore, the administration of cinacalcet hydrochloride for a short period of time aggravates the levels of serum calcium and PTH after discontinuation, and the effect on growth plates, which may express calcium sensitive receptors, remains unclear. Thus, the administration of these drugs should be carefully considered in children. CKD-MBD is known to persist after kidney transplantation [37-38], and high levels of calcium and phosphorus at the time of and after transplantation increase the risk of prolonged secondary hyperparathyroidism [30] and loss of function in the transplanted kidney [39]. Therefore, calcium and phosphorus levels should be controlled before transplantation as much as possible in both PEKT and non-PEKT patients. In this study, no levels of hypercalcemia were observed that would directly lead to a decrease in the kidney function of the transplant; however, an increased cardiovascular risk has been reported for CKD-MBD [40], and cardiovascular calcification was

observed in one patient in the PEKT group in this study. This finding indicates that the cardiovascular complications associated with CKD-MBD may occur in PEKT patients.

As previously reported, similar to adult patients, hypocalcemia immediately after transplantation [41] was noted in both groups in this study. Furthermore, hypophosphatemia was observed 4 days after transplantation on average; however, no significant difference was observed in the minimum serum phosphorus levels after transplantation. Acute hypophosphatemia of ≤ 2.5 mg/dl results in a risk of respiratory or heart failure. In this study, the PEKT group was slightly superior in terms of recovery of phosphorus level. In this study, patients with extremely high serum phosphorus levels contributed to the high pre-transplantation calcium and phosphorus product levels in the PEKT group. In the non-PEKT group, continuous hypercalcemia after transplantation was noted in all patients with prolonged calcium-phosphorus product levels of 55 mg^2/dl^2 or greater after transplantation. We were not able to determine whether hypercalcemia and hypophosphatemia occurred exclusively in patients with hyperparathyroidism at one year after transplantation.

This study suggests the need to closely examine patients undergoing PEKT for reactive hypercalcemia until 3 months after transplantation, similar to the monitoring of patients who have undergone peritoneal dialysis prior to transplantation.

The limited occurrence of CKD-associated complications is considered an advantage of PEKT in general. However, in this study, abnormalities in mineral metabolism were also present in the PEKT group, particularly within 3 months after transplantation. This finding is similar to the abnormalities observed in the non-PEKT group, and the medical management of both groups was complex. Secondary hyperparathyroidism, phosphorus reabsorption disorder due to the use of calcineurin inhibitors for immunosuppression, and the use of steroids cause hypophosphatemia after transplantation [41]. Control of pre-transplantation serum phosphorus is also particularly important in PEKT patients. The calcification of organs and cardiovascular structures in patients before transplantation suggests that the limitations of medical management prior to PEKT should be clearly understood. Two points were considered as reasons why there is no clear difference between PEKT and non-PEKT patients. First, PEKT patients and parents may be less aware of oral medication and dietary

therapy compared to dialysis patients. Second, there is a possibility that waiting in chronic renal failure with insufficient treatment if the patients adhere to PEKT.

The limitations of this study include that it was a retrospective study, the sample size was small, the data were incomplete, and data on $1,25(OH)_2D$, 25(OH)D and FGF23 were not available. Accordingly, a long-term prospective study should be performed using well-matched groups of patients to confirm our findings.

In conclusion, calcium and phosphorus abnormalities and hyperparathyroidism were significant complications during the perioperative period in pediatric PEKT patients, as well as in patients who underwent peritoneal dialysis. Moreover, hyperparathyroidism was not normalized earlier in PEKT patients than in non-PEKT patients in this study because treatment for mineral disorders was likely insufficiently implemented in pediatric PEKT patients. This study demonstrates that careful medical management along with consultation with transplant doctors and medical institutions at the appropriate time is very important to avoiding prolonged abnormalities in mineral metabolism and to maximize the benefits of pediatric PEKT.

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Author contributions

K.H, O.M., S.S., A.A. contributed to study conception and design. O.M., S.S. and A.A. gave conceptual advice. K.H. collected clinical data and records. K.H. wrote the manuscripts and performed the statistical analysis. All authors read and approved the final version of the manuscript.

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Figure legends

Figure 1: Changes in calcium before and after kidney transplantation in PEKT and Non-PEKT. There was no significant in the two groups at any time. PEKT, Pre-emptive Kidney Transplantation

Figure 2: Changes in phosphorus before and after kidney transplantation in PEKT and Non-PEKT. There was no significant in the two groups at any time. PEKT, Pre-emptive Kidney Transplantation

Figure 3: Changes in parathyroid hormone before and after kidney transplantation in PEKT and Non-PEKT. There was no significant in the two groups at any time. PEKT, Pre-emptive Kidney Transplantation

Figure 4 Changes in calcium-phosphorus products before and after kidney transplantation in PEKT and Non-PEKT. There was no significant in the two groups at any time. PEKT, Pre-emptive Kidney Transplantation

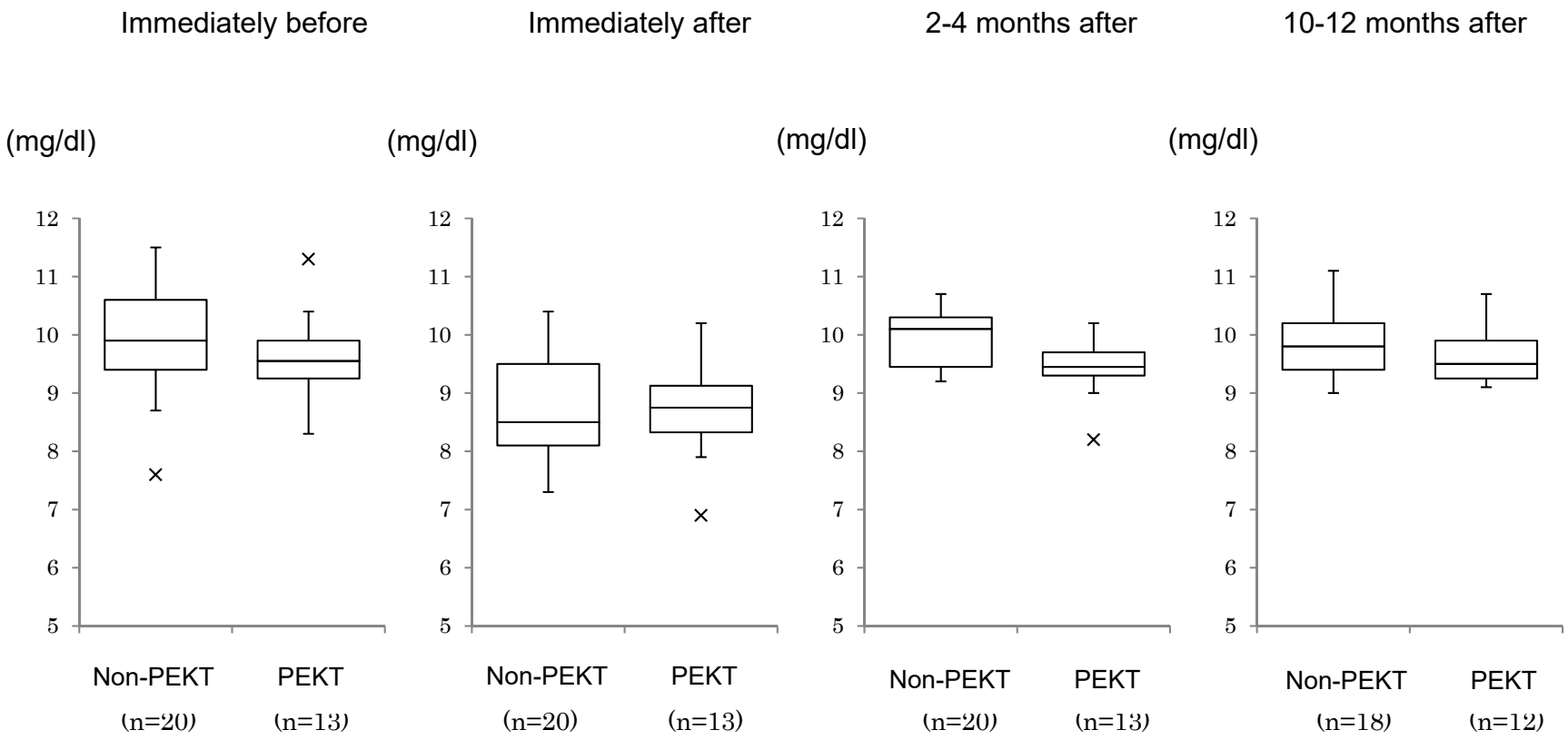
Immediately before
Immediately after
2-4 months after
10-12 months after
(mg/dl)
(mg/dl)
(mg/dl)
(mg/dl)
12
11
10
9
8
7
6
5
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=18)
(n=12)

Figure.1

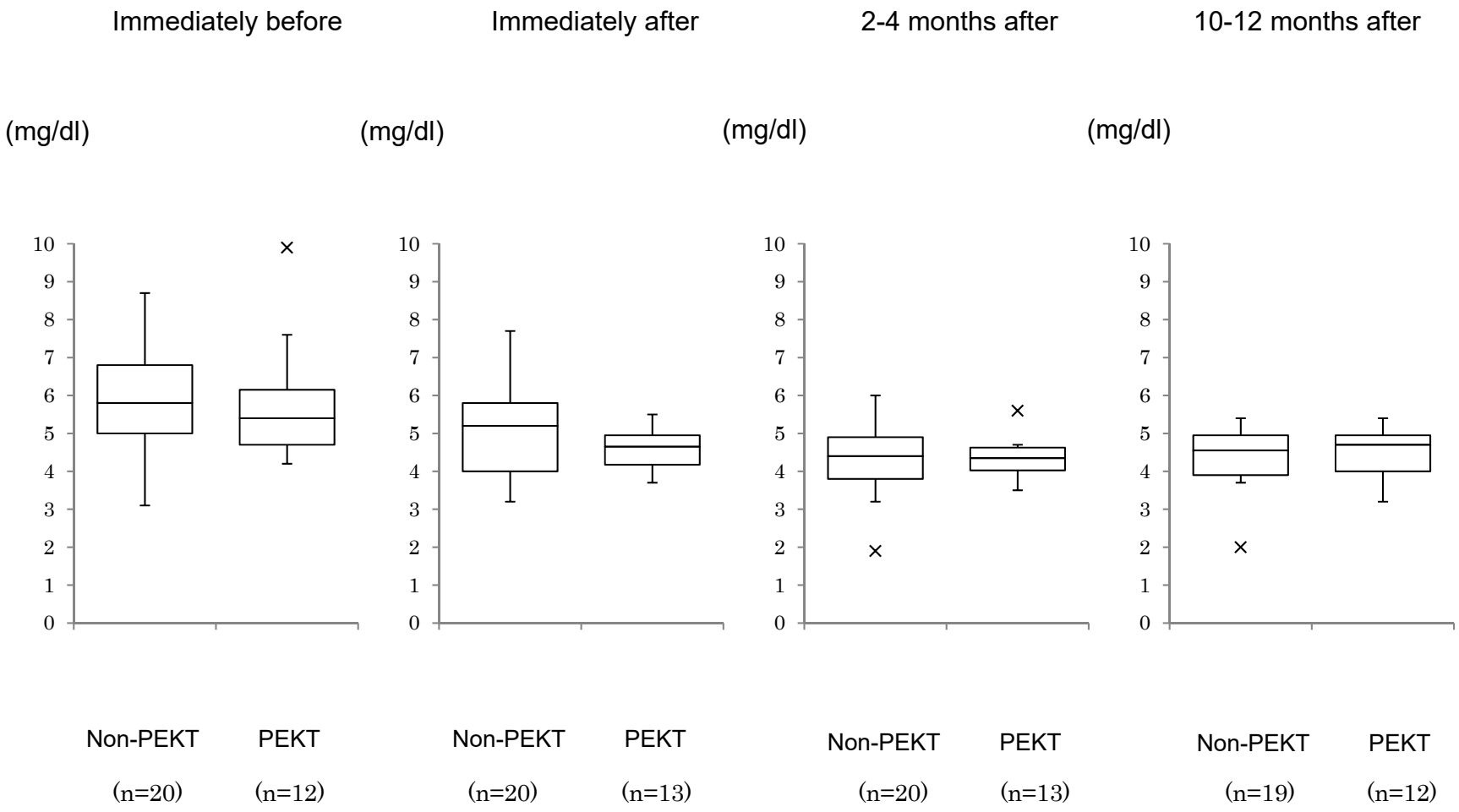
Immediately before
Immediately after
2-4 months after
10-12 months after
(mg/dl)
(mg/dl)
(mg/dl)
(mg/dl)
10
9
8
7
6
5
4
3
2
1
0
Non-PEKT
PEKT
(n=20)
(n=12)
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=19)
(n=12)

Figure.2

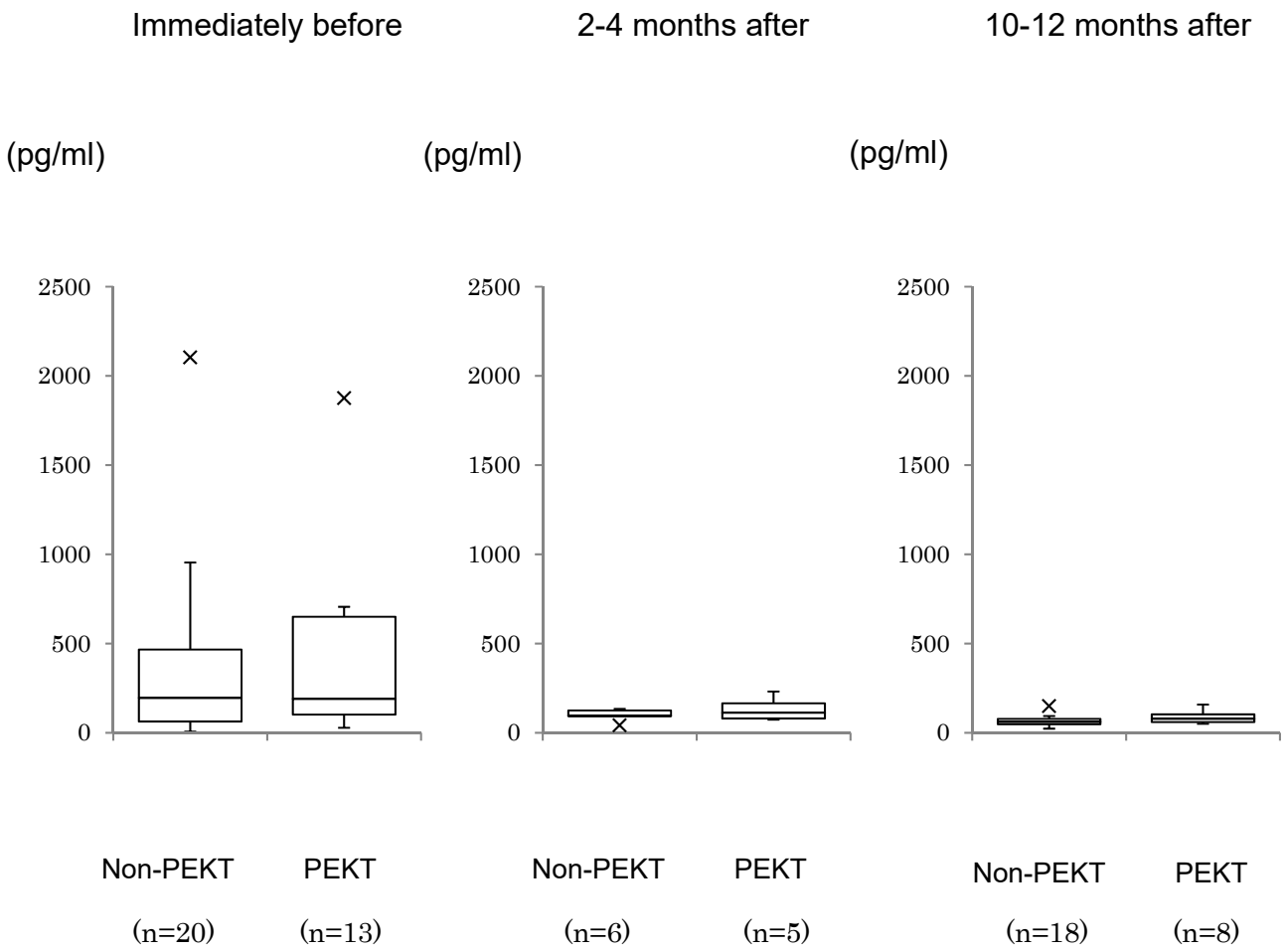
Immediately before
2-4 months after
10-12 months after
(pg/ml)
(pg/ml)
(pg/ml)
2500
2000
1500
1000
500
0
Non-PEKT
PEKT
(n=20)
(n=13)
(n=6)
(n=5)
(n=18)
(n=8)

Figure.3

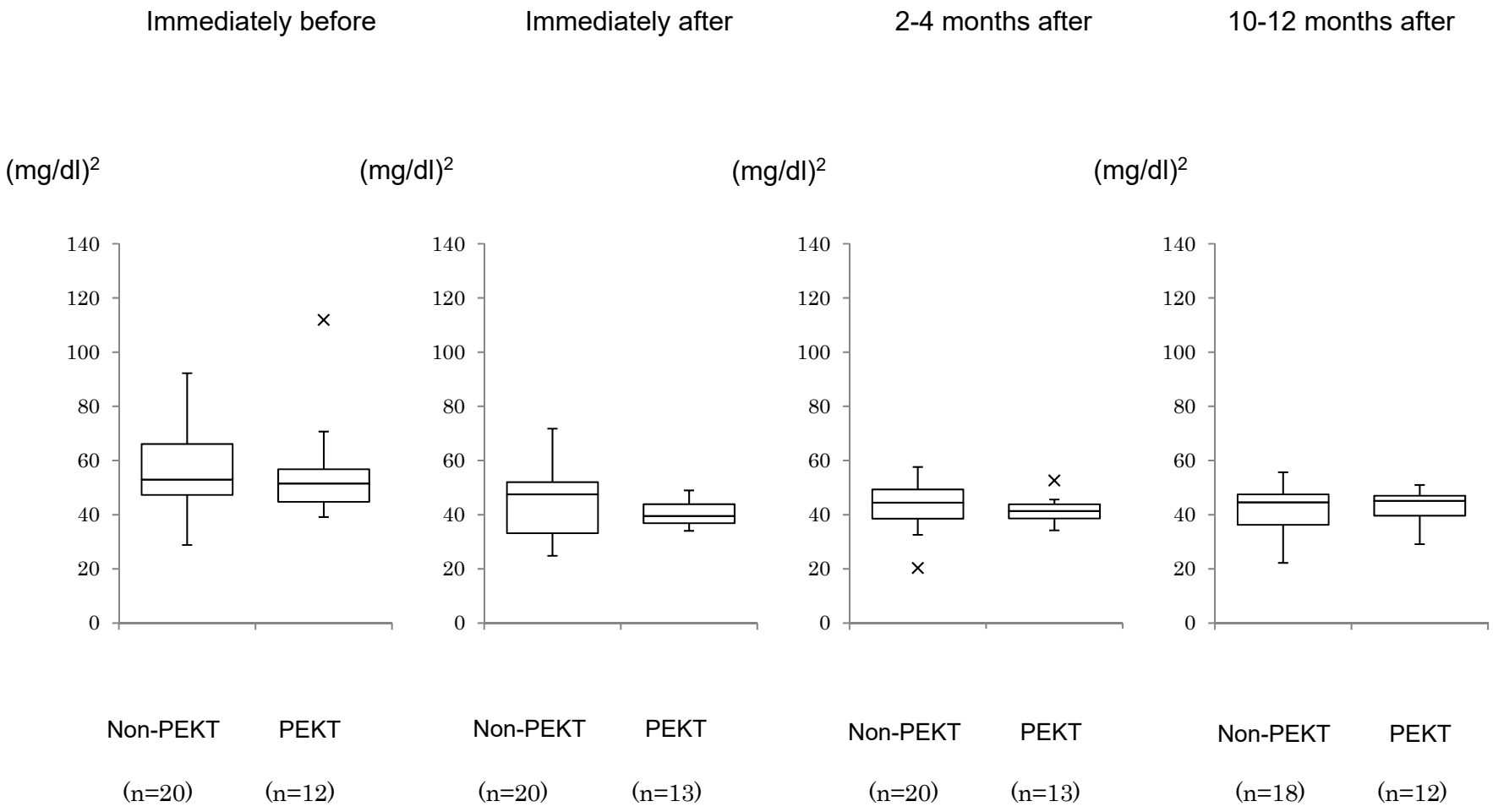
Immediately before
Immediately after
2-4 months after
10-12 months after
(mg/dl)2
(mg/dl)2
(mg/dl)2
(mg/dl)2
140
120
100
80
60
40
20
0
Non-PEKT
PEKT
(n=20)
(n=12)
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=20)
(n=13)
Non-PEKT
PEKT
(n=18)
(n=12)

Figure.4

Table 1. Cause of Chronic kidney disease

Disease	Pre-emptive Transplantation (n=13)	Non pre-emptive Transplantation (n=20)
Congenital nephrotic syndrome	0	1
Crescentic glomerulonephritis	0	1
Diffuse mesangial screlosis	0	1
Focal segmental glomerulonephritis	0	1
Hemolytic uremic syndrome	0	1
Henoch-Schönlein purpura nephritis	0	1
Hypodysplastic kidney	8	3
Maturity-Onset Diabetes of the Young 5	1	0
Membranoproliferative glomerulonephritis	0	1
Multicystic dysplastic kidney	0	1
Multicystic dysplastic kidney + Hypodysplastic kidney	1	1
Nephronophthisis	1	2
Neurogenic bladder (myelomeningocele)	1	0
Reflux nephropathy	0	1
Shock kidney	1	3
Steroid resistant nephrotic syndrome	0	1
Torg-winche syndrome	0	1

Table 2. Characteristics of the study population

Charcteristics	Pre-emptive Transplantation (n=13)	Non pre-emptive Transplantation (n=20)	p value
Sex of recipients (% male)	69	70	0.67
Episode of dialysis (%)	23	100	<0.01
Duration of dialysis (months)	0 (0-62)	23.5 (2-87)	0.36
Age of transplantation (yr)	10 (4-15)	9 (1-20)	0.50
Treatmet of abnormalities in Calcium and phosphorus (%)	85	95	0.34
Transient hypercalcemia after Transplantation (%)	38	55	0.28
Prolonged hypercalcemia more than 3 months after transplantation (%)	8	35	0.08
The minimum hypophosphatemia after transplantation (mg/dl)	2.3±1.0	1.8±0.5	0.11
The maximum of PTH before transplantation (pg/ml)	679 (95-1876)	374.5 (26-2104)	0.30
bone disorders and ectopic Calcifications (%)	15	15	0.85

PTH,parathyroid hormone