Original Article

Super-high levels of serum intact-parathyroid hormone and bone turnover markers descended with recuperating allograft function and a short-term high-dose methylprednisolone during preoperative period of renal transplantation: a retrospective cohort study

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Background: Secondary hyperparathyroidism is an important factor of chronic kidney disease-mineral and bone disorder (CKD-MBD), which frequently results in maintenance dialysis patients having super-high levels of serum intact-parathyroid hormone (iPTH) and bone turnover markers (BTMs). This study aimed to investigate the immediate changes of iPTH and BTMs levels after renal transplantation during the perioperative period, and to explore the allograft function rapid recovery and the effect of high-dose glucocorticoids on serum iPTH and BTMs.

Methods: Between April 2018 and August 2021, a total of 346 Chinese kidney transplantation (KT) recipients (median age, 34.0 years; 236 males and 110 females; median dialysis duration, 12 months) were enrolled in this retrospective cohort study. The included patients had been undergoing maintenance dialysis for at least three months before transplant, and all of them accepted short-term high-dose methylprednisolone (MP) to prevent allograft rejection in the perioperative period. Allograft functions were evaluated and divided into different groups accorded to the CKD staging on the postoperative fifth day. Serum beta C-terminal crosslinking telopeptide of type I collagen (β-CTX), type 1N-terminal propeptide (P1NP), osteocalcin (OC), and iPTH were measured from fasting morning blood samples before surgery and on the postoperative fifth day with an electro-chemiluminescence immunoassay analyzer (2012; Roche Diagnostics).

Results: Among the participants, the graft functions were in CKD-II (n=134), CKD-III (n=137), CKD-IV (n=24), and CKD-V (n=51) after the postoperative fifth day. The changes of P1NP level [−95.8 (−84.0 to −2.4) ng/mL] and the OC level [−88.0 (−96.9 to −42.9) ng/mL] were significantly greater than those of the β-CTX level [−62.3 (−73.6 to 0) pg/mL] and the iPTH level [−57.6 (−15.6 to 11.9) pg/mL] (P<0.001). In the CKD-V group, the changes of β-CTX level [−0.7 (−43.15 to 0) pg/mL (+15.7%, P=0.61)] and the iPTH level [−8.69 (226.73 to 17.79) pg/mL (−22.8%), P=0.36] were less than those of the CKD-II group (P<0.001). β-CTX, P1NP, and OC levels related with iPTH (r=0.413, 0.459, 0.482, respectively, P<0.001), and iPTH level with estimated glomerular filtration rate (eGFR; r=−0.474, P<0.001).

Conclusions: The super-high levels of BTMs and iPTH rapidly descended with recuperating allograft function during the short-term, indicating that improvement of current dialysis equipment to achieve clean up iPTH could more favorably decrease BMTs and improve CKD-MBD. Osteogenesis markers P1NP and

OC still decreased and were not affected in CKD-V group, indicating that high-dose glucocorticoids might strongly inhibit osteoblast activity.

Keywords: Allograft function; bone turnover markers (BTMs); glucocorticoid; osteocalcin (OC); renal transplantation

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Introduction

End-stage renal disease (ESRD) is often combined with mineral and bone metabolism derangement, secondary hyperparathyroidism, multiple renal osteodystrophy and angiostenosis, known as chronic kidney disease-mineral and bone disorder (CKD-MBD) (1,2). Patients with maintenance dialysis (CKD-G5D) often have a super-high level of intact-parathyroid hormone (iPTH) and biochemical bone turnover markers (BTMs) (3-6). This situation may cause a higher incidence of osteoporosis, fracture, osteonecrosis, and renal osteodystrophy, and may lead to deteriorated quality of life and increased risk of mortality for ESRD patients (7,8). The super-high iPTH level is the central feature of CKD-MBD, and the increased BTMs could lead to osteolytic osteopathy, which is correlated with hyperparathyroidism.

Serum BTMs, as markers that can be detected non-invasively, have been used in clinical research to evaluate bone turnover, predict fracture risk, and monitor the osteoporosis treatment response (9,10). Among the BTMs, serum C-terminal telopeptide of type I collagen (CTX-I; a bone resorption marker) and procollagen type 1N-terminal propeptide (P1NP; a bone formation marker) have been recommended by the International Osteoporosis Foundation (IOF) and the International Federation of Clinical Chemistry (IFCC) Bone Marker Standards Working Group for clinical use (11,12).

CKD-MBD is important influence factor for ESRD patient survival quality. IPTH and BTMs are the leading factors resulting in and compounding or aggravating mineral abnormality and bone disorder. How to reduce iPTH levels in CKD-G5D patients has always been a difficult problem closely monitored by kidney disease clinicians. Many reports have highlighted the relationship between the BTMs, iPTH, and osteocalcin (OC) in long-term follow up after kidney transplantation (KT). Currently, these targets (BTMs, iPTH) cannot be improved by the existing equipment and technique of dialysis. However, bone loss after KT has been shown to vary at different timepoints (9,13). It is currently unclear which post-KT changes in CKD-G5D patients result in super-increased iPTH and BTMs levels. It is necessary to discover a way to resolve the difficult situation of CKD-MBD. If the underlying relationship between iPTH and BTMs levels in the perioperative period is identified, measures can be taken to improve the life quality of CKD-MBD patients and reduce the risk of mortality in ESRD patients.

This study retrospectively investigated the immediate changes of iPTH and BTMs levels before and after KT, explored the effect of allograft function rapid recovery

Highlight box

Key findings

- The super-high levels of bone turnover markers (BTMs) and intact-parathyroid hormone (iPTH) rapidly descended with recuperating allograft function during the short-term postoperative period. Osteogenesis markers type 1N-terminal propeptide (P1NP) and osteocalcin (OC) still decreased and were not affected in the chronic kidney disease (CKD)-V group.

What is known and what is new?

- Secondary hyperparathyroidism is important factor of CKD-mineral and bone disorder (MBD), which often results in maintenance dialysis patients having super-high levels of iPTH and BTMs.
- The super-high levels of BTMs and iPTH rapidly descended with recuperating allograft function during the short-term; the osteogenesis markers P1NP and OC decreased in the CKD-V group.

What is the implication, and what should change now?

- This study suggested that improved dialysis equipment and regulation of iPTH could be more helpful to decrease BMTs and improve CKD-MBD. High-dose glucocorticoids might intensely inhibit osteoblast activity.

and high-dose glucocorticoids on serum iPTH and BTMs levels, and attempted to discover a way to resolve the difficult situation of CKD-MBD. We present this article in accordance with the STROBE reporting checklist (available at https://tau.amegroups.com/article/view/10.21037/tau-24-398/rc).

Methods

Participants

This was a retrospective cohort study. Between April 2018 and August 2021, 346 KT recipients were enrolled consecutively from the Kidney Disease Center, The First Affiliated Hospital, Zhejiang University, School of Medicine. Before KT, these patients had been undergoing dialysis (peritoneal dialysis or hemodialysis) for at least three months, and all of them had been matched with a donor kidney, consented to the KT operation, methylprednisolone (MP) pulse therapy and the other antirejection therapy, and had agreed to undergo biochemical BTMs and the other biochemical indicator assessment. The exclusion criteria were a dialysis duration of less than three months before operation, primary hyperparathyroidism, previous parathyroidectomy, undergoing diphosphonate therapy, and lacking adequate laboratory data. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional ethics committee of the First Affiliated Hospital, College of Medicine, Zhejiang University (Medical Ethical Committee, No. IIT20221208A) and written informed consent was taken from all the patients.

Treatment and groups

All renal recipients accepted a short-term high-dose MP intravenous injection on the operative day and preoperative first day at a dose of: 10 mg/kg/d and a follow-up 5 mg/kg/d for 2 days, and from the fifth postoperative day, oral prednisone was continued at 10 mg/kg/d and decreased every day to 10–15 mg/day. Meanwhile, oral mycophenolate was given on the operative day and oral tacrolimus was administered postoperatively to prevent allograft rejection.

On the fifth postoperative day, allograft functions were evaluated by the estimated glomerular filtration rate (eGFR; creatinine-based eGFR) and use of eGFR to confirm CKD staging (14). All recipients were divided into different groups according to the CKD staging: CKD-II (eGFR 60–90 mL/min/1.73 m^2), CKD-III (eGFR 30–59 mL/min/1.73 m^2), CKD-IV (eGFR 15–29 mL/min/1.73 m^2), and CKD-V (eGFR <15 mL/min/1.73 m^2) groups.

Biochemical examinations

Before surgery and on the postoperative fifth day, fasting blood samples were collected at 7:00–8:30 am and detected within 24 hours of admission. Serum beta C-terminal crosslinking telopeptide of type I collagen (β-CTX), type 1N-terminal propeptide (P1NP) (total P1NP), OC, and iPTH were measured using an electro-chemiluminescence immunoassay analyzer (Roche Diagnostics GmbH, Mannheim, Germany). The serum C-terminal telopeptide of type I collagen (CTX-I; a bone resorption marker) and P1NP (a bone formation marker) recommended by the IOF and the IFCC Bone Marker Standards Working Group for clinical use (15,16). The CTX is mainly measured by electrochemiluminescence immunoassay analyzer (ECLIA) using the β-CrossLaps kit (Roche Diagnostics) (11). As bone ages, α-aspartic amino acid converts to β-aspartic acid, so detecting β-CTX is indicative of resorption of matured bone (12). Serum creatinine, total calcium (serum calcium), phosphate, and albumin, among others, were measured by photometric assays *in vitro* using an automated clinical chemistry analyzer (Olympus Diagnostica GmbH, Hamburg, Germany).

Data collection

The following clinical data of the participants were collected at baseline and postoperatively: gender, age, Kinsfolk renal, peritoneal dialysis or hemodialysis, dialysis duration, and the biomarkers such as eGFR, serum albumin, hemoglobin, serum total calcium, serum phosphates, serum total alkaline phosphatase (TAP), 25(OH)D3, iPTH, β-CTX, P1NP, OC, parathyroid hyperplasia, and extraosseous calcification.

Statistical analysis

Categorical variables were expressed as absolute numbers with percentages. The percentage data were compared by chi-square test. The median (interquartile range) values were presented by the Explore Test for non-normally distributed variables and the absolute changes from preoperative value to postoperative value in β-CTX, P1NP, OC, and iPTH concentrations were compared,

Table 1 Baseline characteristics of study population before kidney transplantation operation (n=346)

Index	Data
Gender (male/female)	236/110
Age, years	34.0 (18 to 68)
Kinsfolk renal	234 (67.6)
No kinsfolk renal	112 (32.4)
Peritoneal dialysis	129 (37.3)
Hemodialysis	217 (62.7)
Dialysis duration, months	12 (2 to 141)
eGFR, mL/min/1.73 m^2	5.39±1.99
Serum albumin, g/L	42.08±5.83
Hemoglobin, g/L	109.56±15.61
Serum total calcium, mmol/L	2.27±0.19
Serum phosphates, mmol/L	1.95±0.53
TAP, U/L	84.67±38.45
25(OH)D3, nmol/L	44.61±26.17
iPTH, pg/mL	378.01±294.00
β-CTX, pg/mL	3,139.50±1,440.20
P1NP, ng/mL	459.43±317.56
OC, ng/mL	197.40±67.87
Parathyroid hyperplasia	95 (27.9)
Extraosseous calcification	73 (21.2)

Values are presented as mean ± SD, number (percentage), or medians (range). eGFR, estimated glomerular filtration rate; TAP, serum total alkaline phosphatase; 25(OH)D3, 25hydroxyvitamin D; β-CTX, beta C-terminal crosslinking telopeptide of type I collagen; P1NP, procollagen type 1N-terminal propeptide; OC, osteocalcin; iPTH, intact-parathyroid hormone.

and the interclass data were evaluated by nonparametric tests using the Kruskal-Wallis test for K independent samples. Continuous variables were presented as mean standard deviation (SD) for normally distributed variables (at each group) and the differences were compared by an independent samples *t*-test. The correlation of BTMs and influencing factors were analyzed by Spearman's correlation analysis and or linear regression analysis. A P value less than 0.05 was considered statistically significant, and SPSS statistical software for Windows (version 22; IBM Corp., Armonk, NY, USA) was used for statistical analysis. Graphs were made using GraphPad Prism for Windows version 8.0.2 (GraphPad Software, La Jolla, CA, USA).

Results

Baseline characteristics

A total of 346 KT recipients were enrolled in this study. The baseline data before operation are displayed in *Table 1*; the serum phosphates, iPTH, β-CTX, P1NP, and OC levels exceeded normal reference values (*Table 1*). Spearman correlation analysis showed that β-CTX was significantly correlated with P1NP (r=0.592, P<0.001) and OC (r=0.473, P<0.001). P1NP correlated with OC (r=0.449, P<0.001). β-CTX, P1NP and OC all correlated with iPTH (r=0.399, P<0.001; r=0.320, P<0.001; and r=0.464, P<0.001, respectively).

BTMs levels and absolute changes

The serum levels of P1NP, OC, β-CTX, and iPTH had significantly decreased on the postoperative fifth day in all recipients. The changes of P1NP levels [−95.8 (−84.0 to −2.4) ng/mL] (−99.6%, P=0.001), OC levels [−88.0 (−96.9 to −42.9) ng/mL) (−94.8%, P=0.001), β-CTX levels [−62.3 (−73.6 to 0) pg/mL] (−60.8%, P<0.001), and iPTH levels [−57.6 (−15.6 to 11.9) pg/mL] (−66.7%, P<0.001) were significant (*Table 2*). The changes of P1NP levels and the OC levels were greater than that of the β-CTX levels (P<0.001, *Table 2*), whereas the changes of iPTH levels were comparable to those of the β-CTX levels (P=0.462, *Table 2*).

BTMs in allograft groups

At the postoperative fifth day, the allograft function reverted to CKD-II [134 (38.7%)], CKD-III [137 (39.6%)], CKD-IV [24 (6.9%)], and remained CKD-V [51 (14.7%)]. Before surgery, the β-CTX, iPTH, P1NP, and OC levels were not statistically different in all groups. After the postoperative fifth day, these BTMs levels were increased in the CKD-IV and CKD-V groups compared with the CKD-II group. The changes of P1NP and OC levels were more patent than the β-CTX and iPTH levels in every allograft group (P<0.001). The changes of β-CTX and iPTH levels decreased along with renal function fall-off, up to the CKD-V group, the β-CTX level [−0.7 (−43.15 to 0) pg/mL] (+15.7%, P=0.61) and the iPTH level [−25.54 (225.24 to 17.79) pg/mL] (−22.8%, P=0.36), compared to the CKD-II group, P<0.001

Table 2 Compared β-CTX, P1NP, OC and iPTH levels and absolute changes before and after kidney transplantation

Values	β-CTX (pg/mL)	P1NP (ng/mL)	OC (ng/mL)	iPTH (pg/mL)
Preoperative	2,263.25 (889.8 to 6,011.0)	391.45 (41.33 to 1,230.0)	100.68 (41.32 to 320.0)	270.82 (20.2 to 2,172.0)
Postoperative	854.32 (234.8 to 6,011.0)	16.39 (6.45 to 1,200.00)	12.03 (1.27 to 182.8)	108.95 (22.6 to 1,834.0)
Change	−62.3 (−73.6 to 0) (−60.8%)*	−95.8 (−84.0 to −2.4) (−99.6%)*	−88.0 (−96.9 to −42.9) (−94.8%)*	−57.6 (−15.6 to 11.9) (−66.7%)*
P value[a]		<0.001	<0.001	0.46

Values are presented as median (interquartile range) and percentages. Absolute changes from baseline were calculated (postoperative value minus preoperative value) and the drop rate was analyzed by chi-square test. [a], compared the drop rate with β-CTX. *, P<0.001 (difference between before and after operation). β-CTX, beta C-terminal crosslinking telopeptide of type I collagen; P1NP, procollagen type 1N-terminal propeptide; iPTH, intact-parathyroid hormone; OC, osteocalcin.

(*Table 3*, *Figure 1*).

Spearman correlation analysis showed that after operation, β-CTX significantly correlated with serum iPTH (r=0.413, P=0.001); P1NP correlated with iPTH (r=0.459, P<0.001); OC correlated with iPTH (r=0.482, P=0.001), and the iPTH correlated with the eGFR (r=−0.474, P<0.001).

Influencing factors for iPTH

Linear regression analysis showed that the iPTH levels correlated with the dialysis duration levels and parathyroid hyperplasia before operation (P<0.001, P=0.013, respectively, *Table 4*). After surgery, the iPTH levels correlated with parathyroid hyperplasia and eGFR (P<0.001, P<0.001, respectively, *Table 4*).

Discussion

In this study, KT recipients had super-high levels of serum BTMs before surgery in maintenance dialysis state, these BTMs related with each other, and all participants had highly elevated levels of iPTH. These results are consistent with those of previous reports (9,10,12,13). Parathyroid hormone (PTH) is basic single-stranded polypeptide hormone that is secreted by parathyroid chief cells, with a 9,500 dalton molecular weight (13). To date, it has not been possible to remove PTH by peritoneal dialysis and hemodialysis in uremia patients, so super-high levels of iPTH have resulted in the excess increase of BTMs, causing bone metabolism derangement (9,10,12,13).

In this study, along with allograft function recovery, serum iPTH and BTMs levels quickly descended. These results confirmed that increased PTH might be cleared by normal renal function, and heightened BTMs correlated with PTH might be improved (13,17).

Our findings indicated that if the technique of peritoneal dialysis and hemodialysis could remove the major cause of CKD-MBD, iPTH, metabolic bone disorders might improve, and the survival quality of ESRD patients would be enhanced. Although KT recipients had overcome the effects of secondary hyperparathyroidism (SHPT)—the leading cause of CKD-MBD in the maintenance dialysis period, they were prone to being affected by the tertiary hyperparathyroidism after transplant, which induced hypercalcinemia. osteoporosis, fracture, angiostenosis, or led to some recipients having to undergo parathyroidectomy. Future research should include correlation study about tertiary hyperparathyroidism after KT (18).

In this study, the levels of osteoblast markers P1NP and OC rapidly decreased on the postoperative fifth day, not only in the CKD-II and CKD-III groups, but also in the CKD-IV and CKD-V groups, and their changes were greater than those of β-CTX and iPTH. This indicated that rapid decreases in P1NP and OC during the preoperative period were not affected by the allograft function (12,19), which might be the action of the short-term high-dose MP for osteoblasts (20). Through the inhibition of osteoblast activity, MP can lead to a decrease of bone formation markers (20,21). The survival rate of the recipient depends on survival rate of the graft, so the improved graft survival may help patients improve their quality of life (22). Previous research by Sablik *et al.* found that the addition of MP to intravenous immunoglobulins can significantly improve the graft survival in KT patients (23). At the same time, the changes of osteoclast marker β-CTX correlated with iPTH; their rate of decline were decremented alone with allograft eGFR, demonstrating that short-term high-dose MP did

Table 3 Compared BTMs levels and absolute changes before and after operation in every groups

Values	CKD-II group (n=134)	CKD-III group (n=137)	CKD-IV group (n=24)	CKD-V group (n=51)
Gender (male/female), n	86/48	101/36	15/9	34/17
P value†		0.058	0.523	0.446
Age (range), years	31 (18–62)	34 (20–68)	38.5 (27–61)	45 (26–65)
P value†		<0.001	<0.001	<0.001
Dialysis duration (months)	18.16±24.84	24.92±29.48	41.06±37.63	59.81±31.13
P value†		0.042	0.008	<0.001
β-CTX (pg/mL)				
Preoperative	2,121.00 (934.50–6,003.00) (3,219.50)	2,003.50 (889.80–6,005.00) (2,865.00)	3,105.75 (1,360.00–6,001.00) (3,097.00)	2,132.00 (1,026.00–6,011.00) (2,296.00)
Postoperative	687.58 (234.80–3,267.00) (942.45)	663.00 (307.00–3,032.0) (1,026.00)	1,144.25 (842.40–3,513.00) (1,538.50)	2,117.00 (583.30–6,011.00) (2,657.00)
Change				
Descend-rate	−67.58 (−74.87 to −45.58) (−70.7%)*	−66.91 (−65.49 to −49.51) (−64.2%)*	−63.16 (−38.06 to −41.46) (−50.3%)*	−0.70 (−43.15 to 0) (+15.7%)
P value[a]		0.289	0.525	0.099
P value[b]		0.230	<0.001	<0.001
P1NP (ng/mL)				
Preoperative	383.33 (62.49–1,230.00) (360.85)	359.15 (41.33–1,208.00) (349.00)	524.57 (138.40–1,200.00) (342.85)	388.50 (114.40–1,201.00) (359.90)
Postoperative	10.85 (6.45–226.10) (15.66)	10.76 (7.16–379.30) (15.60)	13.53 (9.98–86.71) (23.26)	33.12 (13.06–1,200.00) (44.63)
Change				
Descend-rate	−97.17 (−89.68 to −81.62) (−95.7%)*	−97.00 (−82.68 to −68.60) (−95.5%)*	−97.42 (−92.79 to −92.77) (−93.2%)*	−91.47 (−88.58 to −0.08) (−87.6%)*
P value[a]		0.801	0.914	0.963
P value[b]		0.903	0.006	<0.001
OC (ng/mL)				
Preoperative	102.00 (41.32–320.00) (210.40)	96.55 (43.30–303.00) (204.00)	121.75 (93.06–302.00) (185.75)	118.40 (53.57–301.00) (210.20)
Postoperative	8.79 (1.27–106.00) (9.99)	7.6 (2.49–182.80) (9.14)	29.44 (3.45–113.70) (11.98)	42.80 (4.10–126.50) (45.29)
Change				
Descend-rate	−91.38 (−96.93 to −66.88) (−95.3%) *	−92.13 (−94.25 to −39.67) (−95.5%)*	−75.82 (−96.29 to −62.35) (−93.6%)*	−63.8 (−92.35 to −57.97) (−78.5%) *
P value[a]		0.558	0.660	0.807
P value[b]		0.876	0.024	<0.001
iPTH (pg/mL)				
Preoperative	247.25 (39.90–2,172.00) (291.05)	237.15 (54.30–1,179.00) (281.10)	657.18 (125.80–1,715.00) (320.60)	387.70 (20.2–1,557.00) (409.40)
Postoperative	57.42 (22.60–740.00) (81.75)	70.10 (29.80–730.50) (93.40)	130.72 (56.60–447.00) (145.75)	288.70 (66.00–1,834.00) (316.20)

Table 3 (*continued*)

Table 3 (*continued*)

Values	CKD-II group (n=134)	CKD-III group (n=137)	CKD-IV group (n=24)	CKD-V group (n=51)
Change				
Descend-rate	−76.78 (−96.92 to −41.10) (−71.9%)*	−70.44 (−45.12 to −38.04) (−66.8%)*	−80.11 (−55.01 to −73.93) (−54.5%)*	−25.54 (225.24 to 17.79) (−22.8%)
P value[a]		0.66	0.31	0.02
P value[b]		0.01	<0.001	<0.001

Values are presented as mean ± SD, median (range) (percentages) or median (range) (interquartile range). Interclass data were compared by Kruskal-Wallis test. Absolute changes from baseline were calculated (postoperative value minus preoperative value) and drop rates were compared by chi-square test. †, compared with the CKD-II group. *, P<0.001 (change of before and after operative). [a], compared preoperative value with CKD-II group. [b], compared postoperative value with the CKD-II group. β-CTX, beta C-terminal crosslinking telopeptide of type I collagen; P1NP, procollagen type 1N-terminal propeptide; iPTH, intact-parathyroid hormone; OC, osteocalcin; CKD, chronic kidney disease; BTM, bone turnover marker; SD, standard deviation.

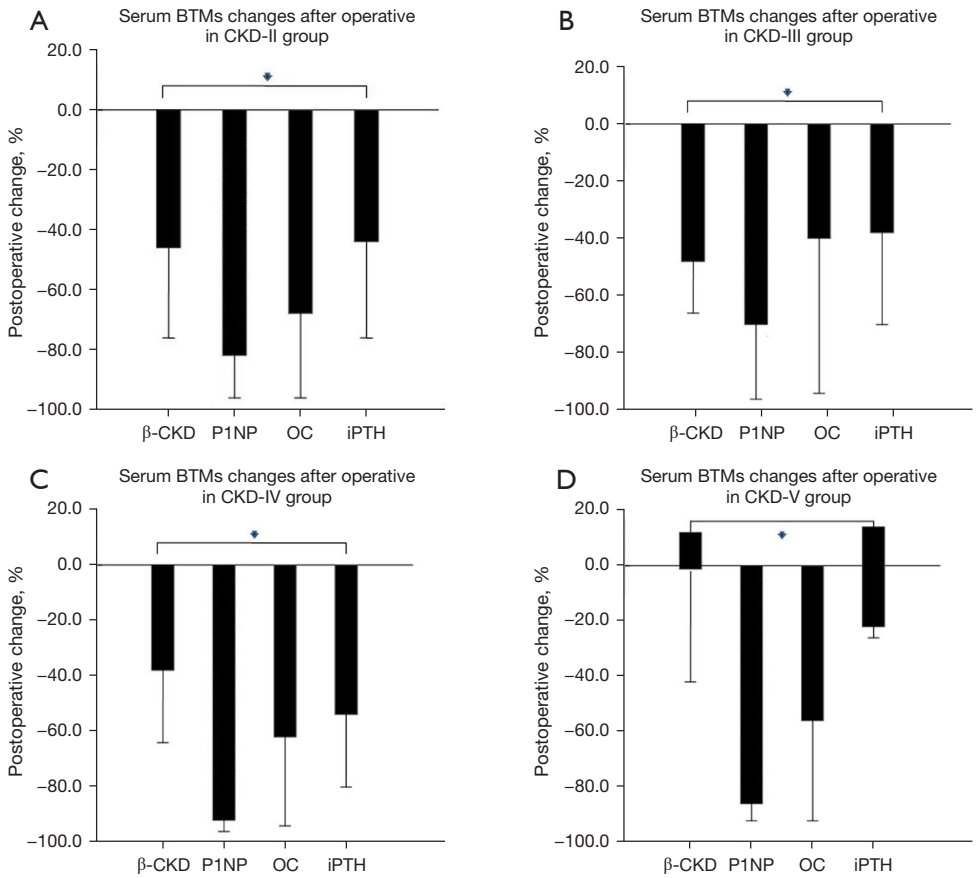

Figure 1 After the post-operative fifth day, the changes of P1NP and OC reflected that every allograft CKD group had attained patency, and the changes of β-CTX and iPTH levels were lower in every CKD group, especially the CKD-V group. (A) In the CKD-II group, the rates of decrease in P1NP and OC were more pronounced than those of β-CTX and iPTH (P=0.001); (B) in the CKD-III group, the rates of decrease of P1NP and OC were more pronounced than those of β-CTX and iPTH (P=0.001); (C) in the CKD-IV group, the rates of decrease of P1NP and OC were more pronounced than those of β-CTX and iPTH (P=0.001); (D) in the CKD-V group, the rates of decrease of P1NP and OC were more pronounced than those of β-CTX and iPTH (P=0.001). β-CTX, beta C-terminal crosslinking telopeptide of type I collagen; iPTH, intact-parathyroid hormone; OC, osteocalcin; CKD, chronic kidney disease; P1NP, type 1N-terminal propeptide; BTM, bone turnover marker.

Table 4 The influencing factor for iPTH levels preoperatively and postoperatively in regression linear analysis

iPTH	Model B	Unstandardized standard error	Coefficients beta	Standardized t	Coefficients Sig
Preoperative					
Dialysis duration	2.693	0.444	0.291	6.069	<0.001
Parathyroid hyperplasia	79.924	32.054	0.122	2.493	0.01
Postoperative					
Parathyroid hyperplasia	144.497	21.244	0.317	6.802	<0.001
eGFR	−3.138	0.378	−0.387	−8.302	<0.001

eGFR, estimated glomerular filtration rate; iPTH, intact-parathyroid hormone.

not inhibit osteoclast activity during the KT perioperative period.

Previously, arterial spin labeling and blood oxygen level-dependent imaging was used to evaluate the effectiveness of renal transplantation function due to their non-invasive nature (24). Since the patients who underwent KT in this study had already provided blood samples in clinical practice, our study analyzed the existing blood samples, minimizing secondary trauma to the patients.

Limitations

A limitation of this study is that it was a single-center study. Moreover, the effect of allograft function and high-dose glucocorticoids on BTMs were not studied by setting a KT-only group and a group using high-dose glucocorticoids, which should be added into future studies.

Conclusions

The super-high levels of iPTH and BTMs rapidly descended with recuperating allograft function during the short-term, indicating that improving current dialysis equipment to regulate iPTH could more efficiently decrease BMTs and improve CKD-MBD. That the osteogenesis markers P1NP and OC levels still decreased in CKD-V group indicated that short-term high-dose glucocorticoids during kidney grafting in the perioperative period might increase the inhibition of osteoblast activity.

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None.

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://tau.amegroups.com/article/view/10.21037/tau-24-398/rc

Data Sharing Statement: Available at https://tau.amegroups.com/article/view/10.21037/tau-24-398/dss

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional ethics committee of the First Affiliated Hospital, College of Medicine, Zhejiang University (Medical Ethical Committee, No. IIT20221208A) and written informed consent was taken from all the patients.

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