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Incidence and Impact of Hypophosphatemia on Renal Function in Kidney Transplant Recipients: A Single-center Study

Zeki Toprak[1], Umut Kasapoğlu[2], Fatih Gökhan Akbay[3], Emre Akar[4], Süheyla Apaydın[5]

[1]*University of Health Sciences Turkey, Ümraniye Training and Research Hospital, Clinic of Nephrology, İstanbul, Turkey*
[2]*University of Health Sciences Turkey, Bakırköy Dr. Sadi Konuk Training and Research Hospital, Clinic of Nephrology, İstanbul, Turkey*
[3]*University of Health Sciences Turkey, Gülhane Training and Research Hospital, Clinic of Nephrology, İstanbul, Turkey*
[4]*University of Health Sciences Turkey, Bakırköy Dr. Sadi Konuk Training and Research Hospital, Clinic of Internal Medicine, İstanbul, Turkey*
[5]*İstanbul Göztepe Medical Park Hospital, Clinic of Nephrology, İstanbul, Turkey*

Abstract

Objective: Hypophosphatemia is a common complication of kidney transplantation. However, the relationship between hypophosphatemia and renal function in patients undergoing kidney transplantation remains uncertain. This study aimed to evaluate the relationship between serum phosphate levels and graft function in patients undergoing renal transplantation within the first 3 months after transplantation.

Methods: We conducted a retrospective cohort study included patients who underwent kidney transplantation between 2016 and 2020. Data on patient demographics and clinical and laboratory findings, such as serum creatinine, phosphate, calcium, hemoglobin and parathormone levels, were collected from the hospital database.

Results: Hypophosphatemia was observed in 59 (47.5%), 41 (33.06%) and 32 (25.8%) patients at the 1st week, 1st month and 3rd month after transplantation. The post-transplant median creatinine levels decreased to 1.36 (1.01-1.58) mg/dL, 1.22 (1.04-1.5) mg/dL, and 1.20 (1.0-1.49) mg/dL at week 1, month 1 and month 3. The median phosphate level before transplantation was 5.1 (4.8-5.7) mg/dL. This value decreased to 2.5 (1.8-3.27) mg/dL, 2.82 (2.05-3.55) mg/dL, and 3.01 (2.30-3.73) mg/dL at week 1, month 1 and month 3. There was no significant difference in serum creatinine and estimated glomerular filtration rate between the hypophosphatemic and normophosphatemic groups at week 1 (p=0.839, p=0.931), month 1 (p=0.453, p=0.441) and month 3 (p=0.592, p=0.570). The causes of end-stage renal disease were chronic glomerulonephritis in 20 patients (16.1%), hypertension in 35 (28.2%), diabetes mellitus in 18 (14.5%) (17 type 2 and 1 type 1), and secondary amyloidosis in 5 (4%). Nephrolithiasis, autosomal dominant polycystic kidney disease, vesicoureteral reflux, and no identifiable cause were found in 7 (5.6%), 4 (3.2%), 16 (12.9%) and 19 (15.3%), patients respectively.

Conclusion: Hypophosphatemia is common after kidney transplantation. No correlation was identified between hypophosphatemia and functional performance of the transplanted kidney.

Keywords: Hypophosphatemia, kidney transplant, graft function

INTRODUCTION

As chronic kidney disease progresses, fibroblast growth factor-23 (FGF-23) and parathyroid hormone (PTH) levels increase and calcitriol levels decrease and this contributes to hyperphosphatemia (1,2). After successful kidney transplantation, blood levels of certain molecules may rapidly change in the presence of a functioning graft, potentially resulting in hypophosphatemia (3-5). Hypophosphatemia has been reported in approximately 22% to 85% of patients following successful kidney transplantation (3,5-11). However, the relationship between hypophosphatemia and renal function in patients undergoing renal transplantation remains unclear.

Address for Correspondence: Zeki Toprak, University of Health Sciences Turkey, Ümraniye Training and Research Hospital, Clinic of Nephrology, İstanbul, Turkey
Phone: +90 554 563 14 00 **E-mail:** zktprk@gmail.com **ORCID ID:** orcid.org/0000-0002-7411-3628

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The objective of this study was to assess the correlation between serum phosphate levels and graft function in patients undergoing renal transplantation during the initial 3 months after transplantation.

METHODS

We conducted a retrospective cohort study included patients who underwent kidney transplantation at our hospital between 2016 and 2020. Initially, 127 participants were enrolled in this study. The inclusion criteria for this study were that the participant had undergone their first kidney transplantation for at least one year prior. The exclusion criteria for this study were early post-transplant death (n=2), primary non-functioning transplanted kidney (n=0), loss to follow-up within 3 months after transplantation (n=1), age below 18 years, and history of parathyroidectomy before transplantation. The remaining 124 participants were followed from the date of transplantation until the end of the study (January 31, 2021).

Following the study's objectives and protocol, which were aligned with the ethical standards outlined in the "Declaration of Helsinki" and sanctioned by the Ethics Committee of University of Health Sciences Turkey, Bakırköy Dr. Sadi Konuk Training and Research Hospital (decision number: 2020-23-07, date: 16.11.2020), the patients' records were retrospectively examined using the hospital information system.

Patient demographics and clinical and laboratory findings were collected from the hospital database. These included age, sex, dialysis type, donor age, and sex; serum creatinine, phosphate, calcium, hemoglobin, PTH, and tacrolimus levels, as well as follow-up records. We collected data on immunological and clinical factors that may impact the outcomes. These factors include preemptive kidney transplantation (defined as transplantation without prior dialysis treatment), donor status (living or deceased), presence of donor-specific antibodies, and type of immunosuppressive drug administered.

During the first year after transplantation, patients were monitored, and routine laboratory tests were conducted to record data on serum creatinine, phosphate, calcium, albumin, and PTH levels. These tests were conducted before transplantation as well as during the first week, first, and third months after transplantation. Post-transplant hypophosphatemia was defined as a serum phosphate level below 2.3 mg/dL. The assessment of renal allograft function was performed by calculating the estimated glomerular filtration rate (eGFR) using the chronic kidney disease - epidemiology collaboration creatinine 2021 equation.

If necessary, the patients received anti-thymocyte globulin at a dose of 1.5 mg/kg daily for 5 days. Following a total of 1500 mg of intravenous methylprednisolone, the patient was switched to oral prednisolone at 40 mg/day. The prednisolone dosage was gradually reduced to 30 mg/day after one week, 20 mg/day after two weeks, and 5 mg/day after one month.

During the maintenance phase, patients were prescribed a calcineurin inhibitor (tacrolimus or cyclosporin) in two divided doses, along with an antiproliferative agent (mycophenolate mofetil up to 2 g/day or mycophenolate sodium up to 1440 mg/day) in addition to prednisolone. Calcineurin inhibitor dosage was adjusted as necessary to maintain target blood levels.

Renal biopsy was performed in cases of acute rejection. Treatment was administered according to the Banff criteria, including pulse methylprednisolone, anti-thymocyte globulin, plasmapheresis, and intravenous immunoglobulin alone or in combination.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics (Version 23.0; IBM Corp., Armonk, NY). The Mann-Whitney U test was used to compare the findings between the hypophosphatemic and normophosphatemic groups for each time period. Pearson's correlation analysis was used to assess the linear association between serum phosphate levels and other variables. The data are presented in two formats: median interquartile range, and percentages (%), as appropriate. A p-value of less than 0.05 was considered to be statistically significant.

RESULTS

In the study group, females constituted 37.9% (n=47) and males 62.1% (n=77) of the 124 patients, respectively. The median age was 40.00 (30.25-52.00) years, and the median follow-up period was 29 (ranged, 12 to 45) months. Table 1 presents the characteristics of dialysis type, number of human leukocyte antigen mismatches, graft type, and induction and maintenance immunosuppressive treatment.

The causes of end-stage renal disease were chronic glomerulonephritis in 20 patients (16.1%), hypertension in 35 (28.2%), diabetes mellitus in 18 (14.5%) (17 with type 2 and 1 with type 1), and secondary amyloidosis in 5 (4%) patients respectively. Nephrolithiasis, autosomal dominant polycystic kidney disease, vesicoureteral reflux, and no identifiable cause were found in 7 patients (5.6%), 4 patients (3.2%), 16 patients (12.9%), and 19 patients (15.3%), respectively.

During the follow-up period, acute allograft rejection was observed in 24 (19.3%) patients. The median phosphate level before transplantation was 5.1 (4.8-5.7) mg/dL. This value decreased to 2.5 (1.8-3.27) mg/dL, 2.82 (2.05-3.55) mg/dL, and 3.01 (2.30-3.73) mg/dL at 1st week, 1st month and 3rd month, respectively.

Hypophosphatemia was observed in 59 (47.5%), 41 (33.06%), and 32 (25.8%) patients at the 1st week, 1st month and 3rd month after transplantation, respectively. Post-transplant median creatinine levels decreased significantly to 1.36 (1.01-1.58) mg/dL, 1.22 (1.04-1.5) mg/dL, and 1.20 (1.0-1.49) mg/dL at 1st week, 1st month and 3rd month, respectively. Table 2 shows patients' characteristics according to serum phosphate levels at 1 week, 1. and 3. month post-transplant.

Table 1. Clinical characteristics of the cohort

Variables	
Number	124
Recipient gender • Female, n (%) • Male, n (%)	 47 (37.9%) 77 (62.1%)
Donor gender • Female, n (%) • Male, n (%)	 67 (54.03%) 57 (45.97%)
Age, year (median, IQR)	40.00 (30.25-52.00)
Type of dialysis, n (%) • Preemptive • Hemodialysis • Peritoneal dialysis	 67 (54.03%) 55 (44.35%) 2 (1.61%)
Donor type • Living • Deceased	 114 (91.93%) 10 (8.07%)
Miss match count, n (%) • 0 MM • 1 MM • 2 MM • 3 MM • 4 MM • 5 MM • 6 MM	 4 (3.22%) 11 (8.87%) 16 (12.90%) 41 (33.06%) 23 (18.54%) 15 (12.09%) 14 (11.29%)
Follow-up time, (month)	29 (ranged, 12 to 45)
Induction treatment, n (%) • ATG • None	 88 (70.96%) 36 (29.04%)
Maintenance treatment • Tac + MMF • Tac + MFNa • Cyc + MMF • Cyc + MFNa	 109 (87.90%) 14 (11.29%) 1 (0.80%) 0
Acute rejection, n (%)	24 (19.3%)

IQR: Interquartile range, MM: Miss match, ATG: Anti-thymocyte globulin, Tac: Tacrolimus, MMF: Mycophenolate mofetil, MFNa: Mycophenolate sodium, Cyc: Cyclosporine

The median PTH level before transplantation was 275.5 (156.7-474.75) ng/L. This value decreased to 155.4 (96.7-254.50) ng/L at 1st month and 113.42 (86.7-174.50) ng/L at 1st year. There were no significant differences in PTH levels between the normo- and hypophosphatemic patients.

Significant differences were found between the pre-transplant laboratory parameters and the post-transplant parameters at the 1st week, 1st month, and 3rd month ($p<0.05$). Table 3 presents the laboratory parameters before and after transplantation.

No significant difference was found between the serum creatinine and eGFR values of the hypophosphatemic group and normophosphatemic group at 1 week ($p=0.839$, $p=0.931$), 1st month ($p=0.453$, $p=0.441$), and 3rd month ($p=0.592$, $p=0.570$), respectively.

There was no significant correlation between serum phosphate and creatinine levels at 1 week ($r=0.063$, $p=0.488$), 1st month ($r=0.058$, $p=0.527$), and 3rd month ($r=0.43$, $p=0.642$). Similarly, no significant correlation was found between serum phosphate and eGFR levels at 1st week ($r=-0.031$, $p=0.732$), 1st month ($r=-0.048$, $p=0.600$), and 3rd month ($r=-0.064$, $p=0.485$).

DISCUSSION

Hypophosphatemia is a common electrolyte disorder after successful kidney transplantation and can be observed in up to 85% of patients in the early post-transplant period (11). In the early post-transplant period, hypophosphatemia is often observed, particularly in the first year (4). This is caused by an increase in renal phosphate excretion capacity, which is attributed to high levels of FGF-23 and PTH following the return of renal function (4). In the current study, hypophosphatemia was observed in 41 (33.06%) in the first month after transplantation. A previous study reported hypophosphatemia in 43.6% of patients during the third month after transplantation (12). Our study obtained similar results.

In our study, no significant correlation or difference was found between the serum creatinine and eGFR values of the hypophosphatemic and normophosphatemic group at 1st week, 1st month, and 3rd month, respectively. Similar to our findings, a study by Kim et al. (13) found no relationship between hypophosphatemia and graft outcomes.

In contrast to our study, a study by Nakai et al. (14) on 90 kidney transplant patients found that hypophosphatemia was an independent predictor of good kidney survival at the 1st and 3rd months post-transplant but not at the 12th month post-transplantation (14). Similarly, in contrast to our study, van Londen et al. (15) showed that graft failure was lower

in patients who developed hypophosphatemia after kidney transplantation than in those who did not develop hypophosphatemia. Also, Işıktaş Sayılar (16) found that hypophosphatemia following kidney transplantation was correlated with better kidney function.

After kidney transplantation, it has been demonstrated that elevated PTH levels decrease during the first 3 months (17). In the first 3 months after kidney transplantation, FGF-23 and PTH levels decrease rapidly because of increased 1,25-dihydroxyvitamin D production (18). In our study, no significant difference was found between the patient groups with and without hypophosphatemia regarding PTH levels. This finding is consistent with those of similar studies in the literature (9,19).

Table 2. Patients' characteristics according to serum phosphate levels at 1 week, 1. and 3. month post-transplant

Variables	Hypophosphatemia in 1st week		Hypophosphatemia in 1st month		Hypophosphatemia in 3rd month	
	+	-	+	-	+	-
n (%)	59 (47.58%)	65 (52.42%)	41 (33.06)	83 (66.94)	32 (25.80%)	92 (74.20%)
Age (years) median (IQR 25-75)	42 (30-53)	39 (31-50)	43 (30-54)	38 (31-51)	45.5 (29.25-54.75)	39 (31-49)
Male recipient, n (%)	35 (59.3)	42 (64.6)	27 (65.9)	50 (60.2)	19 (59.4)	58 (63)
Body mass index median (IQR 25-75)	24.67 (20.93-27.42)	24.74 (21.46-28.17)	24.69 (21.46-27.22)	24.65 (21.27-28.58)	26.28 (22.65-30.27)	24.12 (20.76-26.64)
Living donor, n (%)	55 (93.2)	59 (90.8)	37 (90.2)	77 (92.8)	29 (90.6)	85 (92.4)
Preemptive transplantation, n (%)	33 (55.9)	34 (52.3)	23 (56.1)	44 (53)	20 (62.5)	47 (51.1)
Donor age (years) median (IQR 25-75)	49 (42-58)	48 (38-59)	48 (38.5-56.5)	48 (40-58)	47 (34.5-53.75)	51.5 (40.25-59)
Male donor, n (%)	27 (45.8)	30 (46.2)	19 (46.3)	38 (45.8)	15 (46.9)	42 (45.7)
Drug use, n (%)						
· CNIs, n	5	65	0	8	32	92
· Cyclosporin	0	1 (1.5)	0	1 (1.2)	0	1 (1.1)
· Tacrolimus	59 (100)	64 (98.5)	41 (100)	82 (98.8)	32 (100)	91 (98.9)
· ATG, n	34	37	27	44	24	64
· Antiproliferative agent, n	59	65	41	83	32	92
· Mycophenolate mofetil	49 (83.1)	61 (93.8)	36 (87.8)	74 (89.2)	28 (87.5)	82 (89.1)
· Mycophenolate sodium	10 (16.9)	4 (6.2)	5 (12.2)	9 (10.8)	4 (12.5)	10 (10.9)
Preoperative laboratory median (IQR 25-75)						
· Phosphate, mg/dL	5.3 (4.9-5.7)	5.02 (4.8-5.6)	5.4 (4.9-5.7)	5.1 (4.8-5.6)	5.1 (4.8-5.67)	5.1 (4.8-5.7)
· Pre-transplant PTH, ng/L	265 (190-456)	277 (151-506)	291 (157.5-443.5)	265 (156-490)	281.5 (156.75-583)	275.5 (156.75-462.5)
Laboratory in 3. month median (IQR 25-75)						
· Phosphate, mg/dL	2.4 (1.8-3.27)	3.24 (2.76-4.1)	2.19 (1.75-2.78)	3.3 (2.79-3.96)	1.8 (1.6-2.16)	3.28 (2.82-3.95)
· Calcium, mg/dL	9.4 (9.1-9.83)	9.1 (8.74-9.6)	9.3 (9.1-9.8)	9.2 (8.8-9.8)	9.2 (9-9.75)	9.3 (8.92-9.8)
· eGFR, mL/min/1.73 m^2	68 (50.75-87.25)	69 (52.25-86.5)	72 (55.5-87.5)	66 (52-85)	72 (53-88)	68 (52-85)
· Serum creatinine mg/dL	1.2 (1-1.51)	1.23 (1.09-1.48)	1.2 (1-1.48)	1.23 (1.08-1.55)	1.21 (0.93-1.51)	1.23 (1.07-1.5)

n: Number, IQR: Interquartile range, CNIs: Calcineurin inhibitors, ATG: Anti-thymocyte globulin, PTH: Parathyroid hormone, eGFR: Estimated glomerular filtration rate

Table 3. Laboratory parameters before and after transplantation				
Variables (median IQR 25-75)	Pre-transplant	Post-transplant		
		1st Week	1st Month	3rd Month
Phosphate level (mg/dL)	5.1 (4.8-5.7) mg/dL	2.5 (1.8-3.27) mg/dL	2.82 (2.05-3.55) mg/dL	3.01 (2.30-3.73) mg/dL
Creatinine levels (mg/dL)	5.48 (4.92-6.92) mg/dL	1.36 (1.01-1.58) mg/dL	1.22 (1.04-1.5) mg/dL	1.20 (1.0-1.49) mg/dL
eGFR, mL/min/1.73 m^2	10.60 (7.50-12.00)	65.00 (50.00-81.00)	68.50 (52.00-87.00)	72.50 (57.00-85.00)
Calcium (mg/dL)	8.2 (7.94-8.60)	8.6 (8.21-9.00)	9.1 (8.63-9.60)	9.2 (9.00-9.80)
PTH ng/L	275.5 (156.7-474.75) ng/L	-	155.4 (96.7-254.50) ng/L	113.42 (86.7-174.50) ng/L
IQR: Interquartile range, eGFR: Estimated glomerular filtration rate, PTH: Parathyroid hormone				

Drugs such as high-dose steroids and tacrolimus used in immunosuppressive regimens are believed to cause renal phosphate loss (20). Although hypophosphatemia is frequently observed after kidney transplantation, it is not as common in patients undergoing lung transplantation, for which similar and usually higher doses of immunosuppressive drugs are used (21). In our study, while the majority of patients were on the same immunosuppressive regimen, some developed hypophosphatemia. Therefore, although immunosuppressive drugs may cause urinary phosphate loss, they are unlikely to be the main cause of hypophosphatemia.

Study Limitations

Our study has major limitations, including its retrospective design and single-center nature, small sample size, lack of data on dietary phosphate intake, absence of evaluation of fractional phosphate excretion, and absence of evaluation of FGF-23 and 25-hydroxyvitamin D vitamin levels.

CONCLUSION

In conclusion, hypophosphatemia is common after kidney transplantation. No correlation was identified between hypophosphatemia and functional performance of the transplanted kidney. Further prospective, larger, controlled, multicenter studies are needed to determine the effects of phosphate levels on graft function.

Footnote

Ethics Committee Approval: Following the study's objectives and protocol, which were aligned with the ethical standards outlined in the "Declaration of Helsinki" and sanctioned by the Ethics Committee of University of Health Sciences Turkey, Bakırköy Dr. Sadi Konuk Training and Research Hospital (decision number: 2020-23-07, date: 16.11.2020).

Informed Consent: Since the study was designed retrospectively, no written informed consent forms were obtained from the patients.

Authorship Contributions

Concept: Z.T., U.K., E.A., Design: Z.T., U.K., S.A., Data Collection or Processing: Z.T., U.K., F.G.A., Analysis or Interpretation: F.G.A., S.A., Literature Search: Z.T., U.K., S.A., Writing: Z.T.

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