Original Clinical Research Quantitative

Assessment of the Correlation Between Serum Phosphate Level and Muscle Strength as Measured by Handgrip Strength in Patients Treated With Hemodialysis

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Abstract

Background: Sarcopenia, commonly observed in patients treated with hemodialysis, correlates with low serum phosphate levels. Although normophosphatemia is desired, dietary phosphate restriction is difficult to achieve and may result in undesirable protein restriction.

Objective: We aimed to evaluate whether hyperphosphatemia is associated with higher muscle strength in patients receiving hemodialysis treatment.

Design: A single-center prospective observational study.

Setting: Ambulatory prevalent patients undergoing hemodialysis treatments in a dialysis unit of a tertiary hospital.

Patients: Participants included prevalent patients treated with hemodialysis. All patients were above 18 years. Only patients with residual kidney function below 200 mL/24 hours were included to avoid bias.

Measurements: Muscle strength was measured by handgrip strength (HGS). Each patient repeated 3 measurements, and the highest value was recorded. Handgrip strength cutoffs for low muscle strength were defined as <27 kg in men and <16 kg in women. Biochemical parameters, including serum phosphate level, were driven from routine monthly blood tests. Hyperphosphatemia was defined as serum phosphate above 4.5 mg/dL.

Methods: Handgrip strength results were compared to nutritional, anthropometric, and biochemical parameters—in particular phosphate level. Long-term mortality was recorded.

Results: Seventy-four patients were included in the final analysis. Handgrip strength was abnormally low in 33 patients (44.5%). Patients with abnormal HGS were older and more likely to have diabetes mellitus and lower albumin and creatinine levels. There was no correlation between HGS and phosphate level ($r = 0.008$, $P = .945$). On multivariable analysis, predictors of higher HGS were body mass index and creatinine. Diabetes mellitus and female sex predicted lower HGS. Hyperphosphatemia correlated with protein catabolic rate, blood urea nitrogen, and creatinine. On multivariable analysis, predictors of hyperphosphatemia were higher creatinine level, normal albumin level, and heart failure. During mean follow-up time of 7.66 ± 3.9 months, 11 patients died. Mortality was significantly higher in patients with abnormally low HGS compared with normal HGS (odds ratio = 9.32, $P = .02$).

Limitations: A single-center study. All measurements were performed at one time point without repeated assessments. Direct dietary intake, degree of physical activity, and medication compliance were not assessed.

Conclusion: Hyperphosphatemia correlated with increased protein intake as assessed by protein catabolic rate in patients treated with hemodialysis; however, neither correlated with higher muscle strength as measured by HGS.

Trial registration: MOH 202125213

Abrege

Contexte: La sarcopénie, qui est fréquemment observée chez les patients traités par hémodialyse, est corrélée à de faibles taux sériques de phosphate. Dans ce contexte, la normophosphatémie est souhaitée, mais la restriction alimentaire en phosphate est difficile à réaliser et peut entraîner une restriction indésirable en protéines.

Objectif: Notre objectif était de déterminer si l'hyperphosphatémie est associée à une plus grande force musculaire chez les patients qui reçoivent un traitement par hémodialyse.

Conception: Étude observationnelle prospective monocentrique.

Cadre: Le service de dialyse d'un hôpital de soins tertiaires.

Sujets: Des patients prévalents âgés de plus de 18 ans qui recevaient des traitements d'hémodialyse en ambulatoire dans le service de dialyse de l'hôpital. Afin de limiter les biais, seuls les patients avec une fonction rénale résiduelle inférieure à 200 ml/24 heures ont été inclus.

Mesures: La force musculaire a été mesurée par le test de force de préhension (HGS - handgrip strength). Trois mesures ont été faites pour chaque patient et la valeur la plus élevée a été enregistrée. Les seuils de faible force musculaire à l'HGS ont été établis à < 27 kg pour les hommes et à < 16 kg pour les femmes. Les paramètres biochimiques, notamment le taux de phosphate sérique, ont été déterminés à partir des analyses sanguines mensuelles des patients. L'hyperphosphatémie a été définie par une concentration sérique en phosphate supérieure à 4,5 mg/dl.

Méthodologie: Les résultats de l'HGS ont été comparés aux paramètres nutritionnels, anthropométriques et biochimiques — plus particulièrement au taux de phosphate. La mortalité à long terme a été enregistrée.

Résultats: Soixante-quatorze patients ont été inclus dans l'analyse finale. Les résultats de l'HGS étaient anormalement faibles chez 33 patients (44,5 % des sujets). Les patients qui avaient obtenu un résultat anormal à l'HGS étaient plus âgés, plus susceptibles de souffrir de diabète, et présentaient des taux d'albumine et de créatinine plus faibles. Aucune corrélation n'a été observée entre le résultat à l'HGS et le taux sérique de phosphate ($r=0.008$; $p=0.945$). Dans l'analyse multivariée, l'indice de masse corporelle et le taux de créatinine étaient des prédicteurs d'un résultat plus élevé à l'HGS, alors que le diabète et le fait d'être une femme étaient prédictifs d'un résultat inférieur à l'HGS. L'hyperphosphatémie a été corrélée au taux de catabolisme des protéines, à l'urée et au taux de créatinine. Dans l'analyse multivariée, un taux de créatinine plus élevé, un taux d'albumine normal et une insuffisance cardiaque étaient des facteurs prédictifs d'une hyperphosphatémie. Au cours de la période moyenne de suivi (7,66 ± 3,9 mois), 11 patients sont décédés. La mortalité était significativement plus élevée chez les patients qui présentaient un résultat anormalement faible à l'HGS par rapport à la normale (RC: 9,32; $p = 0,02$).

Limites: L'étude a été menée dans un seul centre. Toutes les mesures ont été effectuées à un moment donné sans évaluations répétées. L'apport alimentaire direct, le degré d'activité physique et l'observance des médicaments n'ont pas été évalués.

Conclusion: Chez des patients traités par hémodialyse, l'hyperphosphatémie est corrélée à une augmentation de l'apport en protéines évalué par le taux de catabolisme des protéines, mais ni l'une ni l'autre n'est corrélée à une plus grande force musculaire mesurée par HGS.

Introduction

Sarcopenia, defined as loss of skeletal muscle and reduced muscle strength, is common among patients undergoing dialysis and correlates with morbidity and mortality in this patient population.[1] The incidence of sarcopenia increases with age and is associated with protein energy wasting (PEW), low serum phosphate level, and low albumin in patients treated with dialysis.[2,3] Although muscle strength does not measure muscle mass directly, previous studies have demonstrated a positive correlation between them.[4] Handgrip strength (HGS) is used to estimate muscle strength. It is recommended by National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (KDOQI) clinical practice guidelines as a reliable method to evaluate muscle function in dialysis patients, which can be additionally used to assess nutritional status in patients treated with dialysis.[5]

Abnormal phosphate metabolism and retention is common among patients with chronic kidney disease (CKD), aggravated as glomerular filtration rate (GFR) decreases.[6,7] Hyperphosphatemia is considered a major risk factor for cardiovascular mortality in patients with CKD.[8-10] However, the interaction between serum phosphate level and survival in patients receiving dialysis has a U-shaped configuration, with increased mortality either with low phosphate level (<4.0 mg/dL) or high phosphate level (>5.5 mg/dL).[11] Phosphate level of 4.4 mg/dL was associated with the lowest

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mortality risk in a multicenter observational study in patients treated with hemodialysis (HD).[12] Many efforts are made to normalize serum phosphate in patients with CKD, using phosphate-restricted diet, phosphorus binders, longer or more frequent dialysis sessions to facilitate phosphate removal, and novel drug therapies.[13,14] However, in daily practice, dietary phosphate restriction is difficult to achieve and may result in undesirable protein restriction, leading to PEW.[15] In fact, a more liberal phosphate diet can assist in reaching sufficient protein requirements. It may also be associated with improved survival in specific patient groups.[16]

We hypothesized that hyperphosphatemia in patients treated with HD may be associated with higher muscle strength due to a more liberal protein intake.

Methods

This is a single-center prospective observational study, comparing muscle strength as measured by HGS to serum phosphate level in patients treated with HD. The study was approved by institute ethics committee, trial number 0310-21-TLV, and all patients gave informed consent.

Prevalent patients treated with HD were enrolled if they were above 18 years old, were able to sign informed consent, and comply with study procedure. To avoid bias of phosphate urinary loss in patients with significant residual kidney function (RKF), only patients with RKF of less than 200 mL/24 hours were included. Patients were excluded if they underwent dialysis treatments less than 3 months or if they were hospitalized 30 days prior to enrollment.

Outcome Measures

The primary outcome was to test the correlation between serum phosphate level and muscle strength, estimated by HGS. Secondary outcomes were to assess the correlation between serum phosphate level and HGS to other nutritional and anthropometric parameters: normalized protein catabolic rate (nPCR), body mass index (BMI), and additional biochemical parameters.

Muscle Strength Measurement

After recruitment, HGS was measured using a Handgrip Dynamometer (Baseline Hydraulic Hand Dynamometer) in the dominant hand, performed before dialysis session. Each patient repeated 3 measurements, and the highest value was recorded. Handgrip strength cutoffs for low muscle strength were defined as <27 kg in men and <16 kg in women, according to the definition of the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) cutoffs for low muscle strength by HGS.[17,18]

Clinical and Biochemical Parameters

Biochemical parameters were driven from routine monthly blood tests and included serum phosphate, calcium, albumin, hemoglobin, creatinine, blood urea nitrogen (BUN), total cholesterol, parathyroid hormone (PTH), and C-reactive protein (CRP). Hyperphosphatemia was defined as serum phosphate above 4.5 mg/dL, and hypoalbuminemia was defined as serum albumin level below 38 g/L.

Daily protein consumption was estimated using nPCR according to urea kinetics calculations.[19] It was calculated for actual body weight (BW) as well as for adjusted body weight, calculated as adjusted BW = ideal BW + [(actual BW − ideal BW) × 0.25]. Ideal BW was calculated according to BMI 23.[5] BMI was calculated as weight in kilograms divided by the square of height in meters. Dialysis adequacy was calculated by single pool Kt/Vurea (spKt/V) using Daugirdas formula.[20]

Medical History Was Retrieved From Medical Charts

Medications including active vitamin D analogues and phosphate binders were retrieved from the medical chart. Phosphate binders included calcium-based and non-calcium-based chelators. Phosphate binding equivalent dose (PBED) was calculated to compare between the different agents.[21] The use of Oral Nutritional Supplements (ONS) was also documented.

Handgrip strength, and clinical and biochemical parameters were completed at baseline, thereafter no more study procedures were performed except for patients' mortality which was recorded.

Sample Size Calculation

Sample size was tested for a cross-sectional study, estimating means, by using WINPEPI software. Since muscle strength has greater variance than phosphate, HGS was the limiting factor in the calculation. We expected to find a difference of approximately 3 to 4 kg in muscle strength between the normal versus abnormal phosphate level according to previous studies[22] ($\alpha = 0.05$). Considering a cross-sectional study, we expected less than 2% dropouts. Consequently, the calculated sample size was 48 to 112 participants. We included 74 participants in the analysis, a sample that had the power to detect a difference of 3 kg between groups with variance of 9% with a confidence level of 95%.

Statistical Analysis

Continuous variables were first tested for normal distribution using the Q-Q plots and histogram. Normally distributed
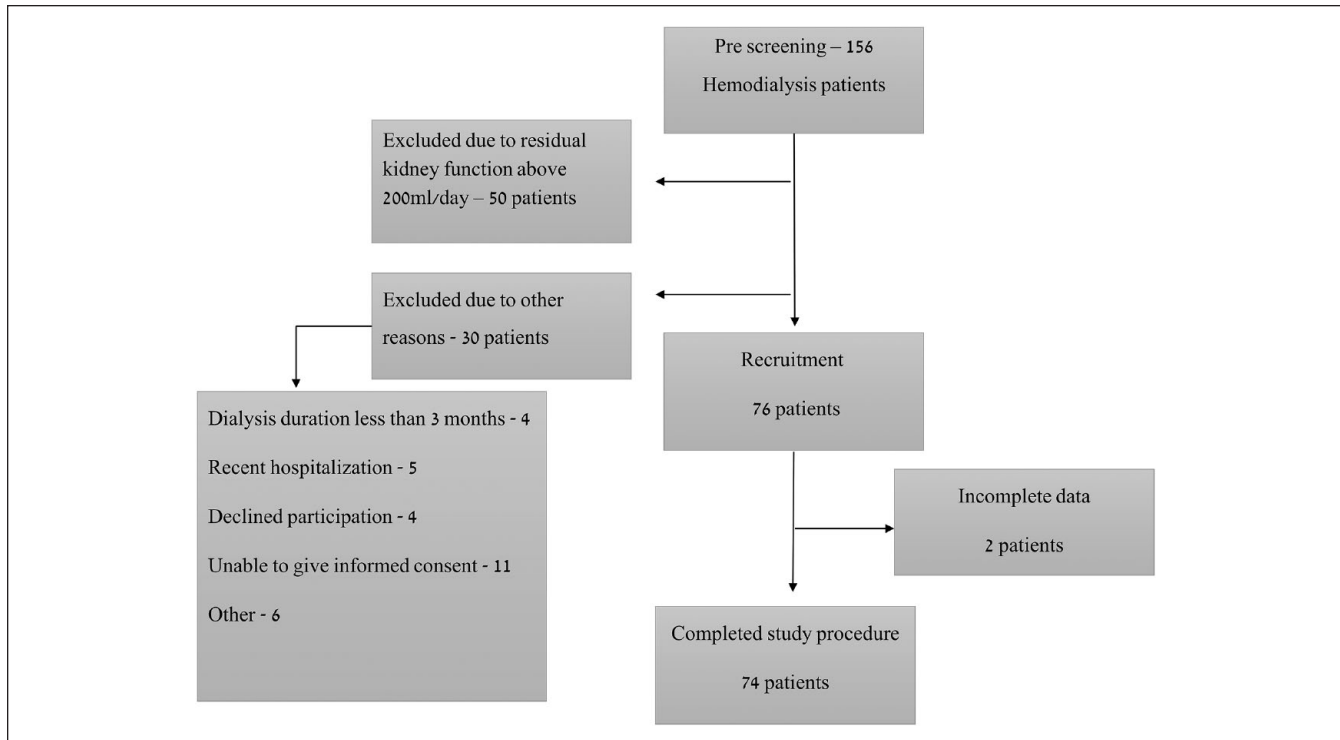
Figure 1. Study consort.

variables were summarized and displayed as mean (standard deviation, SD) and include age, HGS, weight, BMI, PCR, phosphate, creatinine, albumin, BUN, and hemoglobin. Non-normally distributed variables are presented as median (IQR, interquartile range) and include dialysis vintage, spKt/V, PTH, calcium, and CRP.

Continuous variables were compared by using a *t* test if normally distributed or by Mann-Whitney test if not normally distributed.

For all categorical variables, the chi-square statistic was used to assess the differences between groups.

A correlation of the different variables to HGS as a dichotomous parameter (normal/abnormal) was calculated by Pearson's correlation coefficient. Nonparametric variables were also assessed using Spearman's correlation. Categorical and dichotomous variables were tested using chi-square. Parameters with significance <0.3 in the univariable analysis were included in a multivariable logistic regression analysis, including age, creatinine, albumin, calcium, CRP, malignancy, diabetes mellitus, and heart failure. A stepwise forward regression was used.

Thereafter, HGS was analyzed as a continuous variable, using linear regression. Continuous variables normally distributed were analyzed using Pearson's correlation, categorical variables were assessed using Spearman's correlation, and dichotomous and nonparametric variables were assessed using *t* test.

The same process was performed for phosphate level, as a categorical variable (normal ≤4.5 mg/dL or hyperphosphatemia >4.5 mg/dL) and as a continuous variable.

Univariate Kaplan-Maier and multivariate Cox proportional regression was used to identify predictors of mortality.

A 2-tailed *P* value <.05 was considered statistically significant for all analyses. IBM SPSS Statistics for Windows, version 22 (IBM Corp., Armonk, New York) was used for all statistical analyses.

Results

Seventy-six patients treated with HD were recruited between December 2021 and February 2023. Two patients had missing data; therefore, 74 were included in the final analysis (Figure 1). Mean age was 69.2 ± 14.3, and 24 (32.5%) were females. All patients received phosphate binders, 17.6% were calcium-based and 82.4% non-calcium based (Sevelamer Carbonate 55.4%, Lanthanum Carbonate 18.9%, and Sucroferric Oxyhydroxide 8.1%). Median PBED was 3.20 (IQR, 1.75-5.40). Active Vitamin D analogues were given to 60.8% of patients. Eleven patients (14.9%) received ONS. Mean HGS was 27.4 ± 8.9 kg in men and 17.2 ± 6.6 kg in women. Handgrip strength was abnormally low in 33 (44.5%). Patients' baseline characteristics according to HGS status are presented in Table 1. Patients with abnormal HGS

Table 1. Patients' Baseline Characteristics According to Handgrip Status.

	All N = 74	Normal HGS N = 41	Low HGS N = 33
Age (y) (mean, SD)	69.2 ± 14.3	65.1 ± 14.6	74.2 ± 12.2
Sex, female (n, %)	24 (32.5%)	14 (34%)	10 (30%)
HGS, kg (mean, SD)	24.1 ± 9.4	29.5 ± 8	17.3 ± 6.5
Dialysis vintage (y) (median, IQR)	4.5 (2.6-7.5)	4.3 (2.5-7.9)	4.6 (2.5-7.6)
Dialysis access AVF (n, %)	41 (55.4%)	24 (58.5%)	17 (51.5%)
Comorbidities (n, %)			
Active malignancy	6 (8%)	2 (5%)	4 (12%)
Diabetes mellitus	33 (44.5%)	11 (27%)	22 (67%)
Heart failure	20 (27%)	8 (19.5%)	12 (36.5%)
Liver disease	6 (8%)	3 (7%)	3 (9%)
Anthropometric measurements			
Actual weight (mean, SD)	74.1 ± 19.5	75 ± 21.7	73 ± 16.6
Ideal weight (mean, SD)[a]	64.9 ± 7.7	65.3 ± 7.8	64.5 ± 7.4
Adjusted weight (mean, SD)[b]	67.2 ± 9.1	67.8 ± 9.8	66.6 ± 8.3
BMI	26.2 ± 5.9	26.3 ± 6.6	26 ± 5
PCR			
PCR (mean, SD)	82.4 ± 27.8	83.2 ± 29.1	81.4 ± 26.5
nPCR adjusted (mean, SD)	1.2 ± 0.4	1.2 ± 0.3	1.2 ± 0.3
nPCR actual (mean, SD)	1.2 ± 0.3	1.1 ± 0.3	1.1 ± 0.3
spKt/V (median IQR)	1.3 (1.2-1.8)	1.4 (1.2-1.8)	1.3 (1.2-1.7)
Blood test			
BUN, mg/dL (mean, SD)	61.1 ± 18.3	60.8 ± 18.9	61.6 ± 17.8
Creatinine, mg/dL (mean, SD)	7.5 ± 2.1	8.2 ± 2	6.6 ± 1.8
Albumin, g/L (mean, SD)	39.7 ± 3.2	40.5 ± 3	38.6 ± 3.2
Calcium, mg/dL (median, IQR)	8.4 (8.1-9.8)	8.3 (8-8.9)	8.6 (8.2-8.8)
Phosphor, mg/dL (mean, SD)	5.1 ± 1.5	5.1 ± 1.3	5.1 ± 1.6
PTH, pg/mL (median, IQR)	333 (184.2-592.5)	375 (170-631)	323 (209.5-561)
CRP (median, IQR)	10.0 (2.6-19.6)	7.4 (2-17.5)	11.2 (3.8-24.2)
Hemoglobin, g/dL (mean, SD)	10.9 ± 1.3	10.9 ± 1.3	10.9 ± 1.4

Abbreviations: HGS = handgrip strength; AVF = arteriovenous fistula; BMI = body mass index; PCR = protein catabolic rate; nPCR = normalized protein catabolic rate; spKt/V = single pool Kt/V; PTH = parathyroid hormone; CRP = C-reactive protein.
[a]Ideal weight was calculated according to BMI 23.
[b]Adjusted weight is equal to ideal weight + [(actual weight − ideal weight) × 0.25].

were older and more likely to have diabetes mellitus and lower albumin and creatinine levels.

There was no correlation between HGS and phosphate level ($r = -0.049$, $P = .677$) (Figure 2).

On multivariable regression analysis, predictors of higher HGS were BMI and a trend to higher creatinine. Diabetes mellitus and female sex predicted lower HGS (Table 2).

Mean phosphate level was 5.1 mg/dL (± 1.5). Phosphate level correlated with BUN ($r = 0.273$, $P = .019$) and creatinine ($r = 0.338$, $P = .003$) levels and negatively correlated with hypoalbuminemia ($r = -0.299$, $P = .010$).

Hyperphosphatemia correlated with PCR ($r = 0.266$, $P = .022$), nPCR ($r = 0.294$, $P = .011$), BUN ($r = 0.324$, $P = .005$), and creatinine ($r = 0.323$, $P = .005$). On multivariate logistic regression, predictors of hyperphosphatemia were higher creatinine level, normal albumin level, and heart failure (Table 3).

Mortality

During mean follow-up time of 7.66 ± 3.9 months, 11 patients died. Five patients died from cardiovascular causes, 3 as a result of malignancy, 2 due to infection, and 1 patient died from an undetermined cause. The Kaplan-Meier curve indicates that mortality was significantly higher in patients with abnormally low HGS compared with normal HGS (odds ratio [OR] = 9.32, $P = .02$, Figure 3). Heart failure was also associated with increased mortality risk (OR = 7.07, $P = .008$).

On Cox regression, abnormally low HGS was associated with a higher mortality risk (hazard ratio [HR] = 21.69, $P = .027$). Low calcium was protective (HR = 0.17, $P = .048$). Phosphate level did not significantly predict mortality; however, there was a trend to reduced mortality rate with low phosphate level (HR = 0.44, $P = .059$; Table 4).
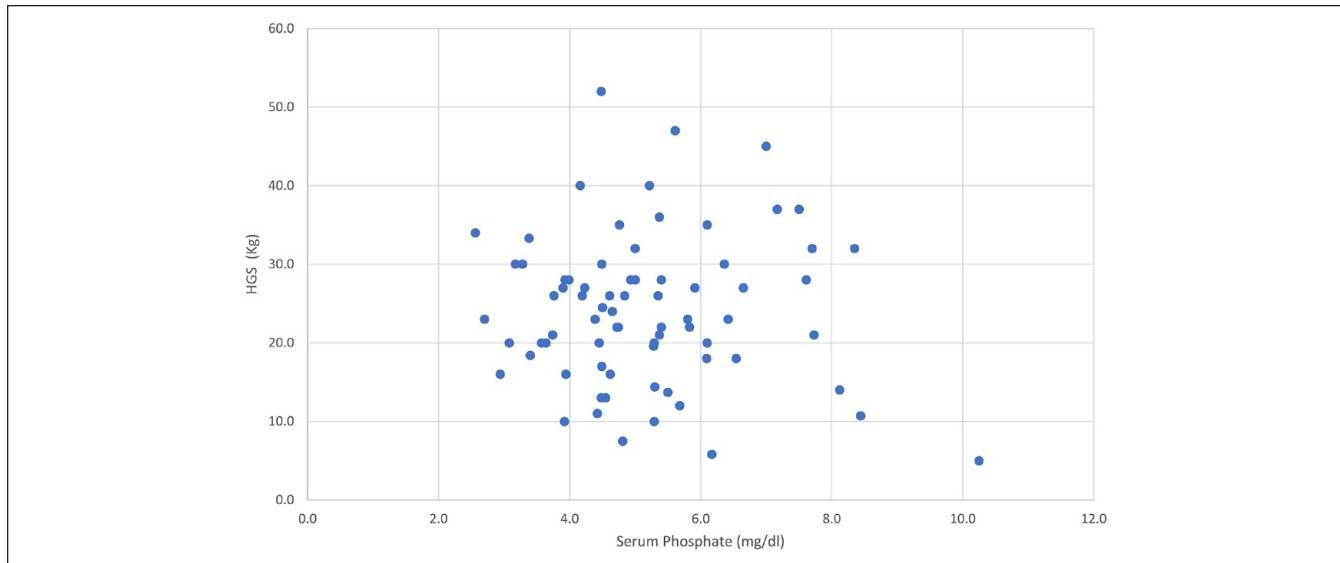
Figure 2. Correlation between handgrip strength and phosphate level.

Table 2. Multivariable Analysis of Predictors of Higher Handgrip Strength.

Variable	B	OR	Significance	95% CI Lower	95% CI Upper
BMI	0.438	0.275	0.019	0.076	0.801
Diabetes mellitus	−4.008	−0.212	0.036	−7.748	−0.268
Female sex	−9.699	−0.482	<0.001	−13.586	−5.812

Abbreviation: BMI = body mass index.

Table 3. Multivariable Analysis of Predictors of Normal Phosphate.

Variable	B	OR	Significance	95% CI Lower	95% CI Upper
Creatinine	0.396	4.405	0.036	1.027	2.150
Normal albumin[a]	−1.658	5.384	0.020	0.47	0.773
Heart failure	1.625	4.191	0.041	1.072	24.064

[a]Albumin above 38 g/L.

Discussion

In this prospective study performed in patients treated with HD, higher muscle strength, measured by HGS, did not correlate with serum phosphate level.

While phosphate-restricted diet can significantly lower serum phosphate level in patients receiving dialysis after 3 months,[23] the long-term consequences of this strategy need to be elucidated as it may compromise intake of other nutrients, especially protein, leading to malnutrition.[16] Therefore, we hypothesized that higher phosphate levels will be associated with increased daily protein intake and, perhaps, increased muscle strength. Hyperphosphatemia had a positive correlation with nPCR, suggesting higher daily protein intake in these patients, as well as with normal albumin level; however, there was no association between phosphate levels and HGS. Compliance to phosphate binders and phosphate dietary source was not tested, and may have affected results, as phosphate gastrointestinal absorption from animal- and plant-based food is lower than the absorption of inorganic
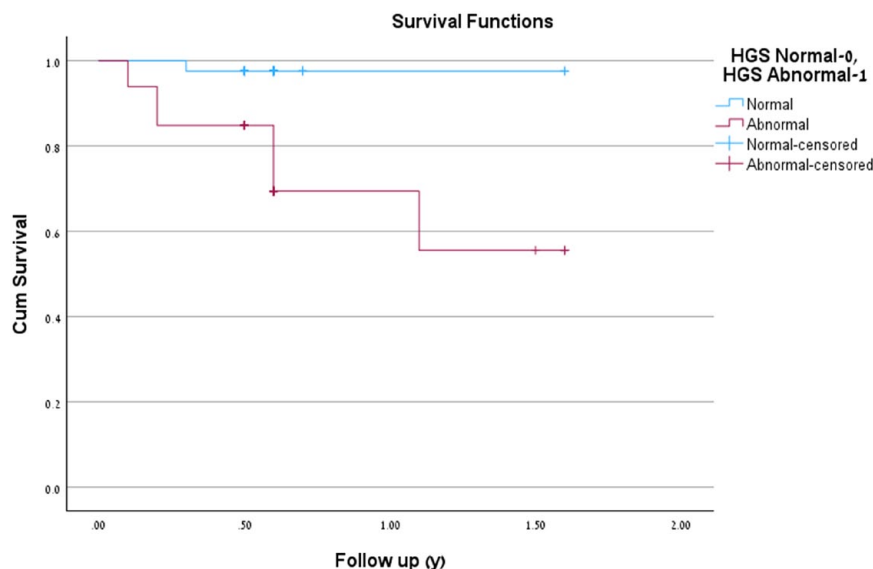
Figure 3. Mortality according to HGS groups.

Table 4. Cox Regression Analysis for All-Cause Mortality.

Variable[a]	B	OR	*P* value	95% CI Lower	95% CI Upper
HGS, abnormal	3.08	21.69	.027	1.414	332.735
Calcium	−1.76	0.17	.048	0.030	0.984
Phosphate	−0.82	0.44	.059	0.189	1.032
Creatinine	−0.53	0.59	.073	0.330	1.050
BUN	0.07	1.07	.062	0.996	1.158

Abbreviations: HGS = handgrip strength; BUN = blood urea nitrogen.
[a]Variables in the equation: age, dialysis vintage, heart failure, HGS, calcium, phosphate, creatinine, BUN, albumin, C-reactive protein, and spKt/V.

phosphorus from phosphate additives in processed food.[24] Furthermore, HGS and nPCR did not correlate in our cohort. Previous observational studies identified physical exercise as an important determinant of muscle strength.[25,26] Since physical activity was not assessed in the current study, it is plausible that findings are influenced by its existence, among other factors. Our findings suggest that high protein consumption by itself is insufficient to enhance muscle strength in patients treated with dialysis.

In anuric patients, pre-dialysis creatinine level reflects muscle mass and creatinine generation, combined with dietary protein consumption. It is therefore an indicator of nutritional status.[5] Chronically low creatinine levels generally indicate reduced muscle mass as well as protein intake. In the Hemodialysis (HEMO) study, mean creatinine level was 10.3 ± 2.9 mg/dL, significantly exceeding the mean creatinine in our study, 7.5 ± 2.1 mg/dL. The HEMO study found a significant negative correlation between serum creatinine and mortality, with 13% mortality reduction for every 1 mg/dL elevation in creatinine level above baseline.[27] In our study, we identified higher pre-dialysis creatinine level as a predictor of both increased HGS and hyperphosphatemia; however, it did not predict mortality.

Handgrip strength has many advantages for muscle strength assessment and subsequently sarcopenia in patients with CKD.[28] It is a simple, non-expensive, validated objective tool, correlates with other methods of body composition assessment and not influenced by hydration status.[29] While the EWGSOP2 defined sarcopenia cutoff points for low muscle strength by HGS according to sex as less than 27 kg for men and less than 16 kg for women, patients' age is not a criterion.[18] In our study, low HGS was relatively common,

identified in 44.5% of our study population. Previous studies in patients receiving hemodialysis reported a higher incidence of 51.5% to 66%.[28,30,31] In the current study, HGS, but not phosphate level, was a predictor of mortality. Handgrip strength has been shown to predict mortality in patients receiving dialysis in previous studies. A meta-analysis demonstrated 5% mortality risk reduction for every 1 kg increase in HGS.[32] In the current study, we demonstrated 9.32 times higher risk of all-cause mortality in patients with abnormally low HGS compared with those with normal results. This finding highlights the importance of HGS as part of the routine physical examination of patients treated with dialysis to assess prognosis. Nutritional interventions along with resistance training can improve HGS,[33] although additional research is necessary to determine the impact of increase in HGS on mortality.

The study has several limitations. Being a single-center study, it is subjected to bias. All measurements were performed at 1 time point, at study entrance, without repeated assessments. While we reviewed phosphate-binders' dosage and use of active vitamin D analogues, both an important determinant of serum phosphate level, medication compliance was not assessed. Since we chose to evaluate protein intake according to nPCR and did not use tools such as 24-hour food recalls or food frequency questionnaires, we could not calculate dietary phosphate intake, as well as differentiate the different sources of phosphate. We also did not have information on degree of physical activity, a factor that could influence muscle strength. Sample size was 74 participants. It is possible that a larger sample would have revealed additional correlations; however, it was adequate to detect a significant difference between groups according to data from previous studies.

Future research should focus on defining age and sex-based cut off values for HGS in different populations in order to better characterize patients at risk for low muscle mass and sarcopenia.

Conclusion

Limiting phosphate intake may limit protein intake in patients treated with hemodialysis. However, while hyperphosphatemia is associated with increased protein intake as evaluated by nPCR, it did not show a correlation with muscle strength in this patient's population.

Ethics Approval and Consent to Participate

The study was approved by the institute's ethics committee, trial number 0310-21-TLV, trial number in Ministry of Health 202125213.

Consent for Publication

All authors reviewed the manuscript and provided consent for publication.

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