Clinical Kidney Journal, 2023, vol. 16, no. 12, 2597–2604

https://doi.org/10.1093/ckj/sfad202
Advance Access Publication Date: 25 August 2023
Original Article

ORIGINAL ARTICLE

Trunk muscle quality and quantity are associated with renal volume in nondiabetic people

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ABSTRACT

Background. Renal disease is a major problem in terms of community health and the economy. Skeletal muscle is involved in crosstalk with the kidney. We therefore investigated the relationship between muscle quality and quantity, and renal parenchymal volume (RPV).

Methods. The association between the parameters of skeletal muscle and RPV/body surface area (BSA) was analyzed by computed tomography in 728 middle-aged participants without kidney disease or diabetes mellitus in a cross-sectional study. A retrospective cohort study of 68 participants was undertaken to analyze the association between changes in RPV/BSA and muscle parameters. Parameter change was calculated as follows: parameter at the follow-up examination/parameter at the baseline examination. The normal attenuation muscle (NAM) and low attenuation muscle (LAM) were identified by Hounsfield Unit thresholds of +30 to +150, and –29 to +29, respectively.

Results. Positive correlations were found between estimated glomerular filtration rate and RPV/BSA ($r = 0.451$, $P < .0001$). Multiple regression analyses revealed that the NAM index was positively related to RPV/BSA ($\beta = 0.458$, $P < .0001$), whereas the LAM index was negatively related to RPV/BSA ($\beta = –0.237$, $P < .0001$). In this cohort study, a change in the LAM index was independently associated with a change in RPV/BSA ($\beta = –0.349$, $P = .0032$).

Conclusion. Both trunk muscle quantity and quality were associated with renal volume related to renal function in nondiabetic people. An increase in low quality muscle volume might be related to a decrease in renal volume.

Keywords: kidney volume, muscle quality, muscle quantity

INTRODUCTION

Renal disease is a major worldwide health and economic burden [1]. Chronic kidney disease (CKD) can evolve to end-stage kidney disease with the burden of dialysis, and is associated with cardiovascular disease [2]. The kidney itself can be divided into renal parenchyma, the renal sinus containing renal pelvis, calyces, renal vessels, nerves, lymphatics, perinephric fat and renal sinus fat, and cysts. In addition, the glomerular filtration rate (GFR) shows a strong correlation with the renal parenchymal volume (RPV) [3, 4]. The renal volume is precisely determined by computed tomography (CT) with errors of 3% or less [5]. CT can also assess the RPV and total renal volume separately.

Received: 18.3.2023; Editorial decision: 20.7.2023

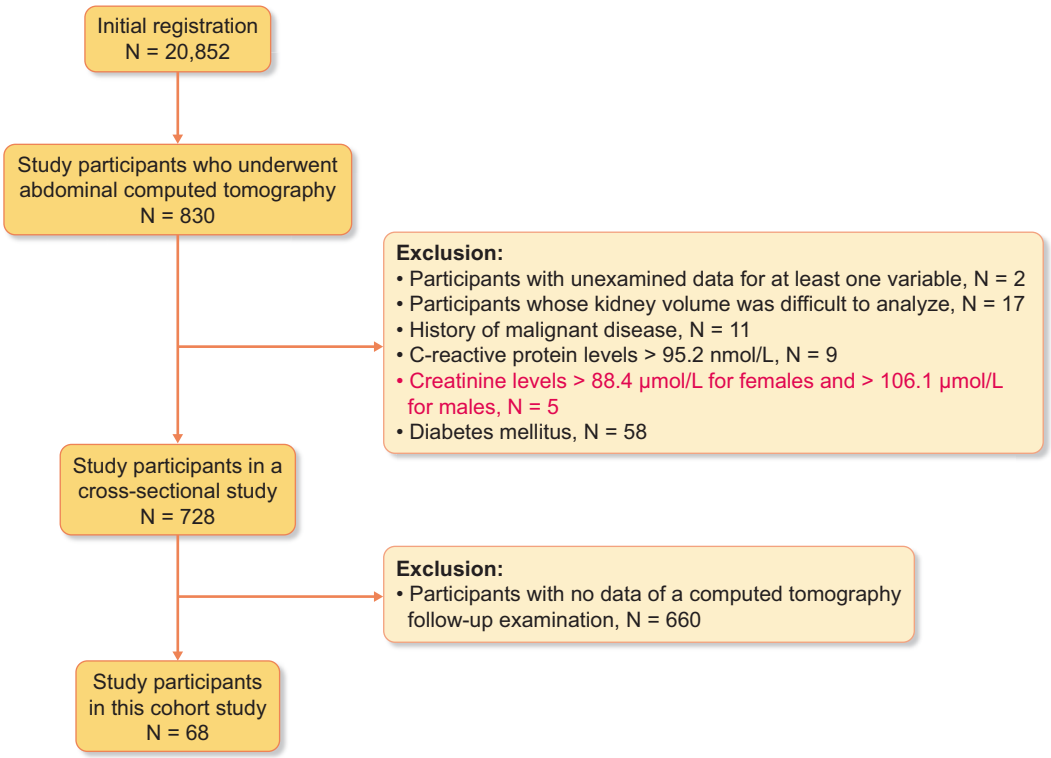

Figure 1: Flowchart of inclusion and exclusion criteria for study participants. Abdominal CT was not included in basic examination items; however, this was conducted upon a participant's request.

Skeletal muscle is involved in crosstalk with other organs [6]. In a mouse model, skeletal muscle hypertrophy reduced renal damage, independently of exercise [7]. In contrast, CKD was linked to reduced skeletal muscle mass [8].

Low density muscle indicates muscle's lipid accumulation, and plays an important role in nonalcoholic fatty liver disease (NAFLD) or metabolic syndrome [9–11]. Therefore, low density muscle is considered low quality muscle.

With regard to findings, our hypothesis was that skeletal muscle was associated with the RPV. This led us to investigate the link between skeletal muscle quality and quantity, and the RPV in middle-aged Japanese without kidney disease. Moreover, we investigated the relationship between the amount of change in the RPV and the amount of change in muscle mass.

MATERIALS AND METHODS

Participants and study design

The Nishimura Health Survey was used as previously described [9–11]. A cross-sectional cohort study was performed with a mean follow-up period of 2.0 years in study participants who had received medical health check-ups. This study included a total of 20 852 participants who were given physical check-ups between 1 April 2013 and 31 March 2020. We excluded study participants with incomplete data, or for whom we had difficulty analyzing kidney volume by CT. Participants that had a history of high creatinine levels, diabetes mellitus, high C-reactive protein (CRP) levels or malignant disease (Fig. 1) were also excluded from the study since systemic inflammation, kidney dysfunction and active infection can affect nutritional and metabolic statuses. Levels of CRP were considered to be high if >95.2 nmol/L. Levels of serum creatinine were considered to be high if ≥88.4 μmol/L for females and ≥106.1 μmol/L for males. People were also excluded from the investigation if a lack of data was available on follow-up CT examinations. The research ethics committee of the Kyoto Prefectural University of Medicine (ERB-C-1017-1) approved this study, which was conducted according to the Declaration of Helsinki. Written, informed consent was obtained from all study participants. Study data was freely available to all authors who also edited and gave approval for the final version of the manuscript.

Data collection and measurements

Biomarkers and demographic data were evaluated as previously described [9–11]. Such biomarkers included: CRP, creatinine, fasting plasma glucose, hemoglobin A1c (HbA1c), high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and triglycerides. The body surface area (BSA) was calculated using Du Bois and Du Bois BSA equation: BSA = 0.007184 × height 0.725 × weight 0.425. The estimated GFR (eGFR) was calculated using the equation of the Japanese Society of Nephrology: eGFR = 194 × Cre – 1.094 × age – 0.287 (mL/min/1.73 m^2). For females, a correction factor of 0.739 was used to multiply the eGRF. Moreover, we also used an equation to estimate GFR that is adjusted for muscle mass. A previous study demonstrated that the muscle mass–based eGFR (MMB-eGFR) equation displayed better performances than equations based on demographics [12].

Computed tomography

Unenhanced transverse CT investigations were undertaken with an Activion 16 CT Scanner (Toshiba Medical Systems, Otawara,

Japan). The CT data set was acquired with 120 kV of tube potential, and all participants underwent CT scans with the same parameters. A cross-sectional area of skeletal muscle was assessed with a CT scan slice at the third lumbar vertebra, as previously described [9–11]. Images were segmented using the specialized software SliceOmatic (v5.0, TomoVision, Montreal, Canada), and were semi-automatically calculated by a well-trained technician. The skeletal muscle was identified and quantified by Hounsfield Unit (HU) thresholds, and the normal attenuation muscle (NAM) and low attenuation muscle (LAM) were identified and quantified by HU thresholds of +30 to +150 and –29 to +29, respectively. The intermuscular adipose tissue (IMAT) area was identified and quantified by HU thresholds of –190 to –30. Each cross-sectional area of skeletal muscle, NAM, LAM or IMAT was normalized to the height squared (cm^2/m^2), and these were labeled the skeletal muscle index (SMI), NAM index, LAM index and IMAT index, respectively. The skeletal muscle density (SMD), as assessed by CT, was described for the entire muscle area at the third lumbar vertebra level. Cross-sectional renal areas were manually drawn, and were used to calculate the RPV by summing all areas with a slice thickness of 5.0 mm. For the renal parenchymal area, the tracing excluded blood vessels, sinus fat and cysts [3]. We also calculated BSA-adjusted RPV (RPV/BSA): RPV/BSA = RPV/BSA (cm^3/m^2). A change in RPV/BSA was calculated as follows: change in RPV/BSA = RPV/BSA at a follow-up examination/RPV/BSA at a baseline examination. A change in muscle parameter was calculated as follows: change in muscle parameter = muscle parameter at the follow-up examination/muscle parameter at the baseline examination. The intraclass correlation coefficients for skeletal muscle parameters and the RPV from 100 random subset samples were all >0.95.

Definitions

According to the guidelines of the American Diabetes Association, the definition of diabetes mellitus (DM) was a fasting plasma glucose level ≥126 mg/dL (7.0 mmol/L), HbA1c level ≥6.5% (48 mmol/mol) and/or a history of diabetes [13]. The definition of hypertension was a diastolic blood pressure of >90 mmHg or systolic blood pressure of >140 mmHg, as well as a history of hypertension. The definition of dyslipidemia was combined high-density lipoprotein cholesterol of <1.03 mmol/L, low-density lipoprotein cholesterol of ≥4.14 mmol/L or triglyceride level of ≥1.69 mmol/L, as well as a history of dyslipidemia. When study participants performed any kind of sports at least 30 min/day regularly, they were categorized as regular exercisers.

Statistical analysis

Categorical parameters are shown as numbers, and continuous parameters are shown as the mean ± standard deviation.

Student's *t*-test or chi-squared test were conducted to assess the statistical significance of differences between two groups.

A Pearson correlation coefficient was used to determine a linear association between RPV/BSA and several parameters. Originally showing a skewed distribution, CRP levels were analyzed after log transformation.

The association between RPV/BSA and the IMAT index, NAM index, LAM index, or total SMI and total SMD was determined by multiple regression analyses. The independent variables used were: age; alcohol consumption; body mass index (BMI); exercise habits; smoking status; hypertension; dyslipidemia; sex; and HbA1c, creatinine and CRP levels. In addition, multiple regression analyses explored the association between the IMAT index, NAM index, LAM index, total SMI or total SMD and several parameters. The following parameters were designated independent variables: age; sex; BMI; exercise habits; alcohol consumption; smoking status; hypertension; dyslipidemia; and HbA1c, creatinine, CRP and RPV/BSA levels. We have performed analysis of variance inflation in linear regression model to detect multicollinearity, and all values did not exceed 5.0.

In a cohort study, multiple regression analyses explored the association between a change in the RPV/BSA and changes in the total SMI, IMAT index, LAM index, NAM index or total SMD. To avoid excessive overfitting, the following parameters were designated independent variables: age; follow-up duration; and sex. $P < .05$ was considered statistically significant. JMP version 11.0 software (SAS Institute Inc., Cary, NC, USA) was used for statistical analyses.

RESULTS

Table 1 shows the clinical characteristics of participants of the study.

No correlations were found between BMI and RPV/BSA (Table 2). Positive correlations between eGFR, or MMB-eGFR and RPV/BSA were found. Positive correlations were found between NAM index, total SMI or total SMD and RPV/BSA, and negative correlations were found between IMAT index or LAM index and RPV/BSA.

In a cross-sectional study, multiple regression analyses revealed that the IMAT index ($\beta = –0.157$, $P < .0001$), NAM index ($\beta = 0.458$, $P < .0001$) and LAM index ($\beta = –0.237$, $P < .0001$) were independently associated with RPV/BSA (Table 3). When both total SMI and SMD were included in the same model, both total SMI ($\beta = 0.240$, $P < .0001$) and SMD ($\beta = 0.328$, $P < .0001$) were independently associated with RPV/BSA.

In addition, after adjustment for age, BMI, sex, exercise, alcohol consumption, smoking status, hypertension, dyslipidemia, HbA1c, creatinine and CRP levels, RPV/BSA was independently associated with the IMAT index ($\beta = –0.135$, $P < .0001$), total SMI ($\beta = 0.173$, $P < .0001$), NAM index ($\beta = 0.231$, $P < .0001$), LAM index ($\beta = –0.137$, $P < .0001$) and total SMD ($\beta = 0.234$, $P < .0001$) (Table 4).

We performed a retrospective cohort study of 68 participants to analyze the relationship between the RPV/BSA change and IMAT index, NAM index, total SMI, LAM index or total SMD change during a follow-up period of 2.0 ± 0.9 years. Mean changes in the RPV/BSA, total SMI, IMAT index, LAM index, NAM index and total SMD were 0.999, 0.998, 1.442, 1.034, 0.987 and 0.985, respectively. After adjustment for age, sex and follow-up duration, only the LAM index change ($\beta = –0.349$, $P = .0032$) was independently associated with a change in RPV/BSA (Table 5).

DISCUSSION

Our study yielded four main findings. First, we found positive correlations between eGFR, or MMB-eGFR and RPV/BSA. Second, correlations were found between several muscle parameters and RPV/BSA. The LAM associated with muscle's lipid accumulation was found to be negatively related to RPV/BSA, whereas NAM was found to be positively related to RPV/BSA. Moreover, SMD was found to be positively related to RPV/BSA. Third, in this cross-sectional study, muscle quality and quantity were independently linked to RPV/BSA. Fourth, in this cohort study, an increase in low quality muscle volume was independently associated with a decrease in the RPV. Altogether, these findings

Table 1: Clinical characteristics of study participants.

Male				
RPV/BSA (cm^3/m^2)	<119.5	119.5–133.7	>133.7	P
N	151	151	152	
Age (years)	53.2 ± 11.2	50.8 ± 9.7	50.0 ± 10.0	.0163
BMI (kg/m^2)	24.1 ± 3.6	23.1 ± 2.8	23.4 ± 2.7	.0127
BSA (m^2)	1.84 ± 0.16	1.79 ± 0.13	1.79 ± 0.12	.0012
Waist circumference (cm)	86.4 ± 9.0	82.5 ± 7.5	83.1 ± 7.6	<.0001
Regular exercise (–/+)	101/50	101/50	113/39	.2665
Alcohol consumption (–/+)	108/43	97/54	106/46	.3645
Smoking (–/+)	130/21	119/32	101/51	.0002
Hypertension (–/+)	100/51	111/40	102/50	0.3277
Dyslipidemia (–/+)	92/59	113/38	96/56	.0230
HbA1c (%)	5.5 ± 0.3	5.5 ± 0.3	5.5 ± 0.3	.9702
Creatinine (μmol/L)	81.3 ± 10.1	75.9 ± 9.1	71.0 ± 8.7	<.0001
eGFR (mL/min/1.73 m^2)	69.7 ± 10.3	76.0 ± 10.8	82.4 ± 12.9	<.0001
MMB-eGFR (mL/min/1.73 m^2)	91.4 ± 13.2	100.8 ± 13.5	108.8 ± 15.8	<.0001
CRP (nmol/L)	9.2 ± 11.8	6.1 ± 8.5	6.2 ± 8.3	.0074
IMAT index (cm^2/m^2)	0.7 ± 0.7	0.5 ± 0.5	0.5 ± 0.4	<.0001
Total SMI (cm^2/m^2)	50.4 ± 7.1	51.3 ± 7.4	52.0 ± 6.5	.1637
NAM index (cm^2/m^2)	37.0 ± 7.1	39.7 ± 7.7	40.3 ± 6.7	.0002
LAM index (cm^2/m^2)	13.4 ± 4.2	11.6 ± 4.5	11.7 ± 4.4	.0004
Total SMD (HU)	42.7 ± 5.5	45.5 ± 6.0	45.7 ± 5.8	<.0001
RPV (cm^3)	200.0 ± 22.1	227.2 ± 18.6	262.7 ± 22.4	<.0001
RPV/BSA (cm^3/m^2)	108.8 ± 8.3	126.8 ± 4.1	147.1 ± 11.2	<.0001
Female				
RPV/BSA (cm^3/m^2)	<114.4	114.4–131.2	>131.2	P
N	91	91	92	
Age (years)	51.8 ± 11.0	48.4 ± 11.1	47.8 ± 10.3	.0253
BMI (kg/m^2)	21.8 ± 4.1	20.7 ± 2.6	21.3 ± 3.5	.1281
BSA (m^2)	1.52 ± 0.14	1.51 ± 0.10	1.52 ± 0.12	.6943
Waist circumference (cm)	77.4 ± 9.7	75.7 ± 7.6	76.4 ± 8.3	.3905
Regular exercise (–/+)	73/18	73/18	78/14	.6528
Alcohol consumption (–/+)	89/2	83/8	84/8	.1199
Smoking (–/+)	89/2	85/6	86/6	.3038
Hypertension (–/+)	74/17	80/11	76/16	.4382
Dyslipidemia (–/+)	68/23	67/24	71/21	.8505
HbA1c (%)	5.6 ± 0.3	5.5 ± 0.3	5.5 ± 0.3	.0356
Creatinine (μmol/L)	59.6 ± 7.1	56.1 ± 6.8	52.0 ± 6.7	<.0001
eGFR (mL/min/1.73 m^2)	72.8 ± 10.6	79.6 ± 12.4	86.8 ± 13.0	<.0001
MMB-eGFR (mL/min/1.73 m^2)	93.2 ± 15.1	101.6 ± 14.7	111.5 ± 17.7	<.0001
CRP (nmol/L)	7.2 ± 10.0	5.1 ± 9.1	6.0 ± 11.8	.3703
IMAT index (cm^2/m^2)	1.3 ± 1.3	1.3 ± 1.0	1.2 ± 0.9	.7652
Total SMI (cm^2/m^2)	39.0 ± 6.0	39.0 ± 4.6	39.9 ± 5.1	.4285
NAM index (cm^2/m^2)	25.9 ± 6.7	27.2 ± 5.4	28.2 ± 5.9	.0419
LAM index (cm^2/m^2)	13.1 ± 4.5	11.8 ± 3.0	11.7 ± 3.3	.0178
Total SMD (HU)	37.8 ± 6.9	39.8 ± 5.6	40.4 ± 6.3	.0127
RPV (cm^3)	158.4 ± 17.3	184.7 ± 14.0	220.7 ± 21.8	<.0001
RPV/BSA (cm^3/m^2)	104.4 ± 7.8	122.4 ± 4.8	144.9 ± 10.8	<.0001

Data are number of participants or mean ± standard deviation.
The study participants were divided into three subgroups according to RPV/BSA tertiles for men and women, respectively.

indicate that muscle quantity and quality as evaluated by CT are both independently associated with renal volume, which, in turn, is associated with renal function. In particular, the low quality of skeletal muscle is linked to decreased RPV.

In our study, participants with DM were excluded because a recent study demonstrated that participants with DM could develop both renal hypertrophy and atrophy [14]. Namely, DM itself might affect renal size. Tubular basement membrane thickening leads to renal hypertrophy, whereas renal atrophy results from tubular atrophy and fibrosis, or a decrement in blood supply by ischemic diseases [14]. Therefore, the relationship between DM and renal size might have influenced the relationship between muscle parameters and renal size in participants with DM.

Sonographic measurements of renal volume are reported to be inaccurate, whereas CT measurements are accurate [15–17]. Moreover, we measured RPV by CT, not by using total renal volume, which includes tissue that does not have a renal function. In addition, our findings are similar to those of a previous study showing that RPV had a strong correlation with renal function [3]. The previous study used GFR measured by iohexol clearance instead of estimating renal function.

To our knowledge, this study is the first to assess the link between RPV and muscle parameters. In participants without

Table 2: Correlations of clinical parameters with RPV.

	RPV/BSA	
Clinical parameter	*r*	P
Age	−0.092	.0129
BMI	−0.062	.0928
Waist circumference	−0.094	.0114
HbA1c	−0.036	.3270
Creatinine	−0.258	<.0001
eGFR	0.451	<.0001
MMB-eGFR	0.483	<.0001
CRP[a]	−0.145	<.0001
IMAT index	−0.133	.0003
Total SMI	0.125	.0007
NAM index	0.193	<.0001
LAM index	−0.154	<.0001
Total SMD	0.182	<.0001

A Pearson correlation coefficient was used to determine a linear association between RPV/BSA and several parameters.
[a]Values were analyzed after log transformation.

Table 3: Results from multiple regression analyses (adjusted β coefficient) of the RPV in non-diabetic participants.

	RPV/BSA			
	β	β	β	β
Age	−0.030	0.104*	0.015	0.137*
Male	0.501*	0.348*	0.516*	0.325*
BMI	0.027	−0.051	0.152*	0.027
Regular exercise	0.021	−0.030	0.005	−0.030
Alcohol consumption	0.009	0.015	0.019	0.024
Smoking	0.113*	0.091*	0.119*	0.097*
Hypertension	0.065	0.078*	0.072*	0.077*
Dyslipidemia	0.024	0.032	0.027	0.034
HbA1c	−0.007	−0.034	−0.010	−0.028
Creatinine	−0.708*	−0.788*	−0.703*	−0.792*
CRP	−0.087*	−0.063	−0.101*	−0.072*
IMAT index	−0.157*			
Total SMI				0.240*
NAM index		0.458*		
LAM index			−0.236*	
Total SMD				0.328*
Adjusted R^2	0.2750	0.3371	0.2828	0.3458

*$P < .05$.

DM, muscle quantity and quality were independently linked to RPV, and an increase in low quality muscle volume was associated with an RPV decrease. Skeletal muscle wasting is a common feature of CKD [18]; however, the association between kidney and muscle remains to be elucidated. Since glomerular number is fixed at birth, the RPV increases with an increase in body size during development due to the hypertrophy of nephrons [19–21]. The RPV varies presumably to accommodate increased metabolic demands. The conceivable mechanisms of this crosstalk between kidney and muscle may be explained by the following: the kidney has a pivotal role in maintaining body homeostasis by acting as a major endocrine organ. However, an injury to the kidney may lead to the release of substances in to the circulation that affect multiple organs including skeletal muscle [18, 22–25]. A previous study demonstrated crosstalk between kidney and muscle in people with CKD, where a soluble pro-cachectic factor, activin A, produced by injured kidney builds up in blood, reduces muscle homeostasis, and leads to muscle wasting [18]. Moreover, in catabolic conditions, including CKD, potential mediators of crosstalk, including insulin-like growth factor-I (IGF-I), myostatin, interleukin (IL)-6 and tumor necrosis factor-α, are activated to affect the growth and function of skeletal muscles [26–30]. The conceivable mechanism regarding the effect of muscle on the kidney is as follows: a previous study demonstrated that Akt1-mediated muscle growth reduces renal damage [7]. The myogenic activation of Akt upregulated serum levels of stromal cell-derived factor-1, IL-17 and IL-10, which can activate endothelial nitric oxide synthase 3 (eNOS). This renal improvement may be mediated by increased eNOS signaling in the kidney. Moreover, skeletal muscle can release IGF-I into the circulation, and IGF-I regulates skeletal muscle hypertrophy and repair [6, 31]. Intermittent IGF-I therapy in patients with advanced CKD was shown to preserve renal function [32]. Moreover, IGF-I regulates renal size and GFR [33]. The effect of muscle quality on the kidney is as follows: a previous study revealed an independent association between lower skeletal muscle density and mortality [34]. A significant relationship between myosteatosis and NAFLD has been reported [9]. Moreover, previous studies showed reduced levels of IGF-I, which is associated with renal size, in the case of NAFLD [35]. Altogether, a major association is likely between muscle quality and quantity, and renal volume. Moreover, recent reports showed that the association between loss of muscle mass and CKD, as well as the consequences of sarcopenia for morbidity and mortality in CKD [8, 36, 37].

The clinical relevance of skeletal muscle quality and quantity for renal function may be important. The association between skeletal muscle and RPV cannot be totally explained by the amount of skeletal muscle present. Our findings indicated that skeletal muscle quality was linked to RPV, independent of skeletal muscle quantity. The interesting role of skeletal muscle quality and quantity in RPV hints at the potential for developing novel preventive measures. However, because of the retrospective nature of this study, the interpretation of our study findings was limited. The correlation between skeletal muscle parameters and renal function needs to be investigated in large prospective studies. Age-related decreases in muscle thickness occur after 50 years of age; on the other hand, age-related changes in muscle quality occur after 30 years of age [38]. Therefore, it needs to be investigated whether resistance training, which has an effect on muscle hypertrophy and results in muscle quality improvement [38], leads to change in the RPV.

Several study limitations should be considered. First, our study participants were all Japanese so whether results can be generalized to other ethnicities is uncertain. However, ethnic background was found not to influence renal parenchymal volume—complementing autopsy data showing no ethnic-based differences in kidney weight [20]. Furthermore, because an abdominal CT was at each participant's request, this possibly introduced an inherent bias in study design. Second, although protein intake can influence renal size, data on food consumption were not available [39]. Third, while glomerular number correlates with kidney weight in healthy adults, it is fixed at birth, and can vary up to 8-fold [19, 40]. Therefore, this relationship could obscure compensatory renal hypertrophy. Fourth, causality was not demonstrated in the present study. An argument for causality would have been to have patients with CKD with a strong association between muscle parameters and kidney volume. However, we excluded study participants with renal dysfunction in our study. As an alternative, we performed a longitudinal cohort study. However, over a 2-year period, the magnitudes of varia-

Table 4: Results from multiple regression analyses (adjusted β coefficient) of several skeletal muscle parameters in non-diabetic participants.

	IMAT index β	Total SMI β	NAM index β	LAM index β	Total SMD β
RPV/BSA	–0.135*	0.173*	0.231*	–0.137*	0.234*
Adjusted R^2	0.3758	0.6970	0.6662	0.5855	0.6244

The analyses were adjusted for age, sex, BMI, exercise, alcohol consumption, smoking status, prevalence of hypertension and dyslipidemia, and HbA1c, creatinine and CRP levels.
*$P < .05$.

Table 5: Results from multiple regression analyses (adjusted β coefficient) of changes in the RPV in non-diabetic participants.

	Change in RPV/BSA				
	β	β	β	β	β
Age	–0.105	–0.142	–0.124	–0.148	–0.126
Male	–0.250	–0.114	–0.252	–0.233	–0.249*
Follow-up duration	–0.103	–0.146	–0.087	–0.036	–0.065
Change in IMAT index	0.130				
Change in total SMI		–0.200			
Change in NAM index			0.080		
Change in LAM index				–0.349*	
Change in total SMD					0.184

*$P < .05$.

tion of muscle parameters and kidney volume was not so large. Fifth, an organ area by CT can be calculated and converted to actual area from a standard grid printed on the roentgenogram, and the volume of the organ slice can be calculated as area times the width between slices. However, there is a 3% variability in the method. Sixth, the Pearson correlation coefficient between RPV/BSA and muscle parameters has a low value, although the values are significant. A weak correlation that is statistically significant suggests that that particular exposure has an impact on the outcome variable, but that there might be other important determinants as well. Finally, whether the results can be generalized to other muscle regions is uncertain. A study demonstrated that metabolic syndrome was associated with abdominal muscle thickness, but not with thigh muscle thickness [41].

CONCLUSION

Trunk muscle quantity and quality was associated with renal volume in study participants without DM. Specifically, an increase in low quality muscle volume was independently associated with a renal volume decrease.

ACKNOWLEDGEMENTS

We thank all staff members at the medical health checkup center at Nishimura Clinic.

FUNDING

No specific funding or any other benefit was received for this investigation or for publishing this manuscript.

AUTHORS' CONTRIBUTIONS

M.T. designed and conducted the statistical analysis and wrote the manuscript. M.F. and H.N. reviewed and edited the manuscript. H.O. and Y.H. contributed to the discussion. M.Y. and M.K. researched the data. All authors read and approved the final submitted manuscript.

DATA AVAILABILITY STATEMENT

Reasonable data requests from this study may be made to the corresponding author.

CONFLICT OF INTEREST STATEMENT

H.O. received grant support from the Japan Society for the Promotion of Science and received personal fees from MSD K.K., Mitsubishi Tanabe Pharma Corporation, Sumitomo Dainippon Pharma Co., Ltd, Novo Nordisk Pharma Ltd, Daiichi Sankyo Co., Ltd, Eli Lilly Japan K.K., Kyowa Hakko Kirin Company Ltd, Kissei Pharmaceutical Co., Ltd, Takeda Pharmaceutical Co., Ltd, Kowa Pharmaceutical Co., Ltd, Ono Pharmaceutical Co., Ltd and Sanofi K.K. Y.H. received grants from Asahi Kasei Pharma, and received personal fees from Novo Nordisk Pharma Ltd, Sanofi K.K., Takeda Pharmaceutical Co., Ltd, Ono Pharmaceutical Co., Ltd, Sumitomo Dainippon Pharma Co., Ltd, Mitsubishi Tanabe Pharma Corp., Daiichi Sankyo Co., Ltd and Kowa Company, Ltd outside the submitted work. M.F. received grants from Ono Pharma Co., Ltd, Oishi Kenko Inc., Yamada Bee Farm, Nippon Boehringer Ingelheim Co., Ltd, Kissei Pharma Co., Ltd, Mitsubishi Tanabe Pharma Corp., Daiichi Sankyo Co., Ltd, Sanofi K.K., Takeda Pharma Co., Ltd, Astellas Pharma Inc., MSD K.K., Kyowa Kirin Co., Ltd, Sumitomo Dainippon Pharma Co., Ltd, Kowa Pharma Co., Ltd, Novo Nordisk Pharma, Ltd, Sanwa Kagagu Kenkyusho Co., Ltd, Eli Lilly, Japan, K.K., Taisho Pharma Co., Ltd, Terumo Corp., Tejin Pharma, Ltd, Nippon Chemiphar Co., Ltd, Abbott Japan Co., Ltd, Johnson & Johnson K.K. Medical Co. and TERUMO CORPORATION, and received personal fees from Nippon Boehringer Ingelheim Co., Ltd, Kissei Pharma Co., Ltd, Mitsubishi Tanabe Pharma Corp., Daiichi Sankyo Co., Ltd, Sanofi K.K., Takeda Pharma Co., Ltd, Astellas Pharma Inc., MSD K.K., Kyowa Kirin Co., Ltd, Sumitomo Dainippon Pharma Co., Ltd, Kowa Pharma Co., Ltd, Novo Nordisk Pharma Ltd, Ono Pharma Co., Ltd, Sanwa Kagaku Kenkyusho Co., Ltd, Eli Lilly Japan K.K., Taisho Pharma Co., Ltd, Bayer Yakuhin, Ltd, AstraZeneca K.K., Mochida Pharma Co., Ltd, Abbott Japan Co., Ltd, Teijin Pharma, Ltd, Arkray Inc., Medtronic Japan Co., Ltd, Nipro Corp. and TERUMO CORPORATION, outside the submitted work. Sponsors were not involved in any data collection, analysis or its interpretation; the design of the study; or in the writing or final submission of this manuscript. Affiliated research foundations, or authors and their immediate families have not received any financial support or other benefits from any company associated with the subject matter of this manuscript. Although affiliated with a department that is financially supported by pharmaceutical companies, the authors have not received specific funding for this investigation, and this did not affect their

following of journal policies with regard to the sharing of data and materials. The other authors declare that they have no conflicts of interest to disclose.

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Received: 18.3.2023; Editorial decision: 20.7.2023