International Journal of Gerontology 17 (2023) 65–68
https://doi.org/10.6890/IJGE.202301_17(1).0013

International Journal of Gerontology

journal homepage: http://www.sgecm.org.tw/ijge/

Original Article

Effect of Sarcopenia on Post-Contrast Acute Kidney Injury in Patients with Abdominal Aortic Aneurism Treated with Endovascular Aneurysm Repair

Hakan Erkan [a *], Merve Erkan [b], Ahmet Özderya [c], Dursun Topal [a]

[a] *Department of Cardiology, Bursa City Hospital, University of Health Sciences, Bursa, Turkey,* [b] *Department of Radiology, Bursa Yuksek İhtisas Training and Research Hospital, University of Health Sciences, Bursa, Turkey,* [c] *Department of Cardiology, Ahi Evren Cardiovascular and Thoracic Surgery Research and Application Center, University of Health Sciences, Trabzon, Turkey*

ARTICLE INFO

Accepted 13 June 2022

Keywords:
sarcopenia,
endovascular procedures,
acute kidney injury

SUMMARY

Purpose: Sarcopenia is a syndrome presenting with progressive and generalized skeletal muscle loss and disability. Its incidence is higher among the elderly. Recent studies have reported an association between sarcopenia and postoperative acute kidney injury (AKI). However, the association between sarcopenia and post-contrast AKI (PC-AKI) remains uninvestigated. This study aimed to assess the association between sarcopenia and the development of PC-AKI in patients with abdominal aortic aneurysm who underwent endovascular aneurysm repair (EVAR).
Method: We included 109 patients in the study. PC-AKI was defined as a serum creatinine (SCr) concentration increase of ≥ 0.3 mg/dL or ≥ 1.5–1.9 times the baseline value within 72 hours after the EVAR procedure. Sarcopenia was defined as a skeletal mass index (SMI) of ≤ 52.4 cm^2/m^2 for men and ≤ 38.5 cm^2/m^2 for women. SMI was calculated by dividing the cross-sectional area of the skeletal muscle measured using preoperative abdominal computed tomography (CT) by the height in square meters.
Results: The baseline demographic, laboratory, and medication data were similar in patients with and without sarcopenia. PC-AKI developed in 10 (19.2%) and 7 patients (12.3%) in the sarcopenia and non-sarcopenia groups, respectively (p = 0.31). Multivariate logistic regression analysis showed that the baseline Scr value (odds ratio [OR], 4.075; 95% confidence interval [CI]), 1.277– 13.000; p = 0.01), and the use of angiotensin converting enzyme inhibitors or angiotensin receptor blockers (OR, 3.940; 95% CI, 1.191–13.085; p = 0.02) are independent determinants of PC-AKI.
Conclusion: Sarcopenia is not associated with the development of PC-AKI. Patients with sarcopenia should be evaluated for classical risk factors of PC-AKI.

1. Introduction

Post-contrast acute kidney injury (PC-AKI), previously known as contrast-induced nephropathy, is defined as a rapid impairment of renal function occurring within 24–72 hours of exposure to parenteral iodine contrast media.[1] PC-AKI is associated with serious adverse outcomes, including cardiovascular events, progression to renal failure, need for hemodialysis, prolonged hospital stay, increased medical cost, and mortality.[2,3]

Sarcopenia is the progressive loss of skeletal muscle mass and a decline in muscle strength and is primarily a geriatric condition.[4] Although sarcopenia is a musculoskeletal disease it is closely associated with other organ system disorders such as cardiovascular disease, respiratory disease, metabolic disorders, and diabetes.[5–8] Sarcopenia is also closely associated with renal disease.[9] Recent studies have shown that sarcopenia is associated with an increased incidence of postoperative AKI after donor hepatectomy and surgery for abdominal aortic aneurysm (AAA).[10,11] However, the association between sarcopenia and PC-AKI has not been investigated.

Percutaneous endovascular aneurysm repair (EVAR) is typically used to treat AAA and requires the use of a contrast medium. Because of its higher incidence in this population, we hypothesized that sarcopenia contributes to the development of PC-AKI in this population. Therefore, in this study, we aimed to evaluate the association between sarcopenia on the development of PC-AKI in patients who underwent EVAR for AAA.

2. Materials and methods

2.1. Study population

We included 109 consecutive patients (52 sarcopenia and 57 non-sarcopenia) with infrarenal AAA who were treated with EVAR at our hospital between 2017 and 2020. All clinical data were retrospectively collected from the patients' medical records. We excluded patients with a ruptured AAA, bleeding requiring blood transfusion, intraoperative hypotension (systolic blood pressure < 90 mmHg), renal artery thromboembolic complications, exposure to contrast media within 2 weeks before the procedure (including for computed tomography [CT] imaging), dialysis for chronic renal failure, and prominent accessory renal artery or renal artery stenosis. Fur-

* Corresponding author. Department of Cardiology, Bursa City Hospital, University of Health Sciences, Bursa, Turkey.
E-mail address: drhakanerkan@hotmail.com (H. Erkan)

thermore, patients exposed to nephrotoxic drugs such as aminoglycosides and nonsteroidal anti-inflammatory drugs were also excluded. The study was approved by the Bursa City Hospital Ethics Committee, and the need for written informed consent was waived given the retrospective study design.

2.2. Study procedures

Preoperative CT angiography was performed for all patients to determine their anatomic eligibility for EVAR. The aorta was evaluated from the descending thoracic aorta to the femoral artery with a 1- to 5-mm slice thickness. A scheduled elective EVAR procedure was performed in patients with an aneurysm diameter of at least 55 mm and a suitable anatomy for EVAR. According to our routine clinical practice, all patients received intravenous isotonic saline infusion at a rate of 1 mg/kg per hour starting 24 hours before the procedure and until 12 hours after the procedure. No specific reno-protective medication, such as sodium bicarbonate or N-acetylcysteine, was administered.

Endovascular procedures were performed in a hybrid operation room under fluoroscopic control. The study was carried out using a non-ionic, low-osmolality contrast agent. Peripheral venous blood samples were collected at baseline and at 24, 48, and 72 hours after contrast exposure to measure the levels of blood serum creatinine (SCr). In addition, the estimated glomerular filtration rate (GFR) was calculated before and after the procedure concurrently with SCr measurement in the study population, using the Modification of Diet in Renal Disease formula.[12] The highest SCr level within 72 hours after the procedure was used to diagnose PC-AKI.

2.3. Definition and measurement of sarcopenia

PC-AKI was defined an increase of > 0.3 mg/dL or ≥ 1.5–1.9 times the baseline value within 72 hours after the endovascular procedure as recommended by Contrast Media Safety Committee (CMSC) of the European Society of Urogenital Radiology.[1]

Sarcopenia was assessed using axial CT imaging, at the level of the third lumbar vertebrae (L3). Initially, the cross-sectional areas (CSA, in cm^2) of all skeletal muscles at this level (rectus abdominis, external and internal obliques, psoas, transverse abdominis, rectus abdominis, quadratus lumborum, and the erector spinae) were measured by using OsiriX software (Figure 1). The skeletal muscles were isolated using Hounsfield units (HU) of -29 to 150. Thus, the exact muscle area was obtained based on the HU, excluding vasculature and fat infiltration. Then, CSA measurements were normalized to patient size by dividing the CSA by height in square meters to obtain the skeletal muscle index (SMI; cm^2/m^2). Finally, sarcopenia was diagnosed when SMI was ≤ 52.4 cm^2/m^2 in men and ≤ 38.5 cm^2/m^2 in women, as previously defined.[13–16]

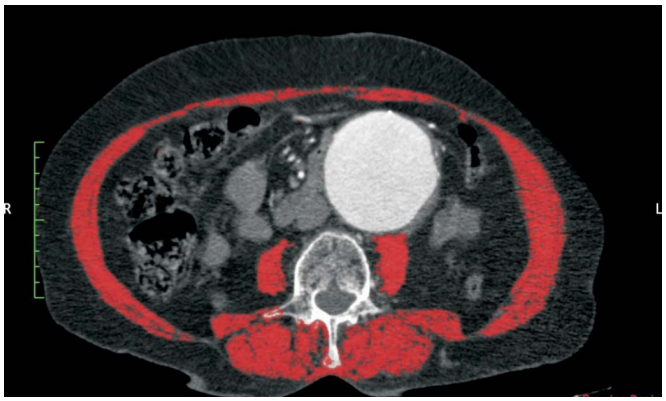

Figure 1. Measurement of the cross-sectional area of all skeletal muscles from the L3 vertebra level.

2.4. Statistical analysis

Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as numbers and percentages. The Kolmogorov-Smirnov test and histogram analysis were used to test the normality of the distribution of continuous variables. Continuous variables were analyzed using the independent-sample t test and/or the Mann-Whitney *U* test according to the normality of distribution. The chi-square and Fisher exact tests were used for categorical variables. A p value < 0.05 was considered statistically significant. All statistical analysis were performed using the SPSS 21 statistical software (SPSS Inc., Chicago, Illinois, USA).

3. Results

The demographic and laboratory findings including age, sex, hypertension, diabetes mellitus, baseline SCr, and contrast volume were similar between the sarcopenia and non-sarcopenia groups. SMI was expectedly lower in the sarcopenia group (p < 0.001). However, the prevalence of PC-AKI did not differ between the groups. PC-AKI developed in 10 (19.2%) and 7 (12.3%) patients in the sarcopenia and non-sarcopenia groups, respectively (p = 0.31; Table 1). Multivariate logistic regression analysis showed that baseline SCr (OR, 4.075; 95% CI, 1.277–13.000; p = 0.01) and the use of angiotensin-converting enzyme inhibitors or angiotensin receptor blockers (OR, 3.940; 95% CI, 1.191–13.085; p = 0.02) are independent determinants of PC-AKI (Table 2).

Table 1
Baseline demographic and laboratory data of study population.

	Patients with sarcopenia (n = 52)	Patients without sarcopenia (n = 57)	p
Age (years)	73 ± 6.0	72 ± 7	0.19
Gender (male), n (%)	45 (86.5%)	48 (84.2%)	0.73
BMI	27 ± 5	27 ± 4	0,35
Hypertension, n (%)	44 (84.6%)	46 (80.7%)	0.59
Diabetes mellitus, n (%)	10 (19.2%)	13 (22.8%)	0.64
Coronary disease, n (%)	19 (36.5%)	21 (36.8%)	0.97
Smoking, n (%)	33 (63,5%)	29 (50.9%)	0.18
Calcium antagonist, n (%)	11 (21.2%)	12 (21.1%)	0.99
Beta bloker, n (%)	10 (19.2%)	7 (12.5%)	0.33
ACEI/ARB, n (%)	27 (51.9%)	31 (54.4%)	0.79
Statin, n (%)	20 (38.5%)	20 (35.1%)	0.71
ASA	26 (50%)	21 (36%)	0,16
Basal creatinine (mg/dL)	0.9 ± 0.3	1.1 ± 0.4	0.10
GFR (ml/min)	73 ± 18	69 ± 20.	0.28
Albumin	3.58 ± 0.6	3.54 ± 0.4	0.23
Triglycerides (mg/dl)	150 ± 70	171 ± 80	0.26
LDL (mg/dl)	119 ± 38	125 ± 41	0.43
HDL (mg/dl)	40 ± 10	40 ± 13	0.89
Hematocrit	39 ± 5	38 ± 5	0.75
Contrast volume (ml)	188.4 ± 62.6	192.7 ± 54.1	0.70
SMI, cm^2/m^2	**39.7 ± 6**	**53.9 ± 5**	**< 0.001**
PC-AKI, n (%)	10 (19.2%)	7 (12.3%)	0.31

ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blockers, ASA: acetylsalicylic acid, BMI: bady mass index, GFR: glomerular filtration rate, HDL: high-density lipoprotein, LDL: low-density lipoprotein, PC-AKI: post-contrast acute kidney injury, SMI: skeletal mass index.

Table 2
Univariate and multivariable analysis for PC-AKI.

	Univariate analysis				Multivariable analysis			
	OR	95% CI		p	OR	95% CI		p
		Lower	Upper			Lower	Upper	
Age	1.033	0.955	1.118	0.41				
BMI	1.03	0.927	1.145	0.58				
Hypertension	1.01	0.262	3.96	0.98				
Diabetes mellitus	2.218	0.469	10.490	0.31				
Coronary disease	1.474	0.478	4.539	0.49				
Smoking	1.599	0.566	4.518	0.37				
ACEI/ARB	**3.262**	**1.062**	**10.018**	**0.03**	**3.940**	**1.191**	**13.085**	**0.02**
Baseline SCr	**3.305**	**1.199**	**9.110**	**0.02**	**4.075**	**1.277**	**13.000**	**0.01**
Contrast volume	1.001	0.992	1.010	0.87				
Sarcopeni	1.701	0.595	4.857	0.32				

ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blockers, BMI: bady mass index, CI: confidence interval, OR: odds ratio, PC-AKI: post-contrast acute kidney injury, SCr: serum creatinine.

4. Discussion

To our knowledge, this is the first study to assess the effect of sarcopenia on the onset of PC-AKI in patients with AAA who underwent EVAR. The incidence of PC-AKI did not differ between patients with and without sarcopenia. Thus, we conclude that sarcopenia does not affect the development of PC-AKI.

PC-AKI causes severe adverse outcomes and is a relatively common clinical consequence of diagnostic and interventional percutaneous procedures.[1] Because no effective treatment for PC-AKI has been developed, identifying at-risk individuals and taking preventive preoperative measures are critical. The risk factors identified for PC-AKI in earlier studies, such as advanced age and impaired kidney function, are frequently seen in patients with sarcopenia. For instance, the study by Foley et al. showed that sarcopenia is associated with a decline in GFR in community-dwelling individuals.[9] Similarly, Yang et al. reported that sarcopenia is associated with declining renal function among those without diabetes and those with type 2 diabetes.[17] However, we could not find a significant association between sarcopenia and PC-AKI although several risk factors were common. PC-AKI and sarcopenia are multifactorial disorders and pathogenesis of them were not clearly understood until today. We hypothesized that the development of PC-AKI in this population is likely associated with a multifactorial interaction of risk factors that can vary between individuals.

In contrast, in a recently published study by Bang et al. that examined the association between sarcopenia and postoperative AKI in patients with AAA, sarcopenia was associated with an increased incidence of postoperative AKI.[11] However, this study had some important differences from our study. First, the study included patients who underwent both open surgical repair (OSR) (55.9%) and EVAR (44.1%). Evidently, most of the study population was not exposed to contrast media as it is not used in OSR. Second, the definition AKI differed between the studies. The KDIGO AKI guideline is currently the most used guideline to diagnose AKI, which identifies AKI based on the change in SCr over the first 7 postoperative days. However, PC-AKI is defined according to CMSC of the European Society of Urogenital Radiology. Therefore, we diagnosed PC-AKI based on an increase in SCr within 72 hours after contrast media exposure in our study. Finally, the cut-off values used to establish sarcopenia also differed significantly between the studies. In our study, we used the cut-off values proposed by Prado et al. that are widely accepted for the diagnosis of sarcopenia. However, Bang et al. determined these cut-off values in women and men to be 28.6 and 39.6, respectively. They had reported their hypothesis that the cut-off points they used were more appropriate than the classically accepted cut off values for the diagnosis of sarcopenia in the Asian population. Therefore, the results of Bang's study cannot be directly compared with ours.

This study has some limitations. First, this is a retrospective study. Second, our study population included patients who underwent the EVAR procedure. Therefore, our study results may not represent the general population.

5. Conclusion

The incidence of PC-AKI is similar among patients with and without sarcopenia. Thus, patients should be assessed for classical and other risk factors for the development of PC-AKI.

Conflict of interest

There is not any commercial association that might pose a conflict of interest in connection with this manuscript.

References

1. Stacul F, van der Molen AJ, Reimer P, et al. Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines. *Eur Radiol*. 2011;21(12):2527–2541.
2. James MT, Samuel SM, Manning MA, et al. Contrast-induced acute kidney injury and risk of adverse clinical outcomes after coronary angiography: a systematic review and meta-analysis. *Circ Cardiovasc Interv*. 2013;6(1):37–43.
3. Hiremath S, Akbari A, Wells GA, et al. Areiso-osmolar, as compared to low-osmolar, contrast media cost-effective in patients undergoing cardiac catheterization? An economic analysis. *Int Urol Nephrol*. 2018; 50(8):1477–1482.
4. Cruz-Jentoft AJ, Bahat G, Bauer J. et al. Sarcopenia: revised European consensus on definition and diagnosis. *Age Ageing*. 2019;48(1):16–31.
5. Hong SH, Choi KM. Sarcopenic obesity, insulin resistance, and their implications in cardiovascular and metabolic consequences. *Int J Mol Sci*. 2020;21(2):494.
6. Bone AE, Hepgul N, Kon S, et al. Sarcopenia and frailty in chronic respiratory disease. *Chron Respir Dis*. 2017;14(1):85–99.
7. Dominguez LJ, Barbagallo M. The cardiometabolic syndrome and sarcopenic obesity in older persons. *J Cardiometab Syndr*. 2007;2(3):183–189.
8. Wang M, Tan Y, Shi Y, et al. Diabetes and sarcopenic obesity: Pathogenesis diagnosis, and treatments. *Front Endocrinol (Lausanne)*. 2020;11:568.
9. Foley RN, Wang C, Ishani A, et al. Kidney function and sarcopenia in the United States general population: NHANES III. *Am J Nephrol*. 2007;27(3): 279–286.
10. Sim JH, Kwon HM, Jun IG, et al. Association of skeletal muscle index with postoperative acute kidney injury in living donor hepatectomy: A retrospective single-centre cohort study. *Liver Int*. 2022;42(2):425–434.

11. Bang JY, Jun IG, Lee JB, et al. Impact of sarcopenia on acute kidney injury after infrarenal abdominal aortic aneurysm surgery: A propensity matching analysis. *Nutrients*. 2021;13(7):2212.
12. Levey AS, Coresh J, Greene T, et al. Using standardized serum creatinin values in the modification of diet in renal disease study equation for estimating glomerular filtration rate. *Ann Intern Med*. 2006;145(4):247–254.
13. Montano-Loza AJ, Meza-Junco J, Prado CM, et al. Muscle wasting is associated with mortality in patients with cirrhosis. *Clin Gastroenterol Hepatol*. 2012;10(2):166–173.
14. Gul DH, Kim MY, Seo YS, et al. Clinical usefulness of psoas muscle thickness for the diagnosis of sarcopenia in patients with liver cirrhosis. *Clin Mol Hepatol*. 2018;24(3):319–330.
15. Kumar V, Benjamin J, Shasthry V, et al. Sarcopenia in cirrhosis: Fallout on liver transplantation. *J Clin Exp Hepatol*. 2020;10(5):467–476.
16. Prado CM, Lieffers JR, McCargar LJ, et al. Prevalence and clinical implications of sarcopenic obesity in patients with solid tumours of the respiratory and gastrointestinal tracts: A population-based study. *Lancet Oncol*. 2008;9(7):629–635.
17. Yang R, Zhang Y, Shen X, et al. Sarcopenia associated with renal function in the patients with type 2 diabetes. *Diabetes Res Clin Pract*. 2016;118:121–129.