

RESEARCH LETTER

Is There a Correlation Between Sarcopenia and Aortic Aneurysm Morphometry?

Low muscle mass is common in patients with abdominal aortic aneurysm (AAA) and is associated with reduced post-operative survival,¹ frailty, and acute and chronic disease. AAA is due to connective tissue abnormalities influenced by enzymatic activity. Matrix metalloproteinases, which are implicated in this process, have also been shown to affect muscle development.² It is unclear, however, if low muscle mass is related to the anatomy and evolution of AAA. It was hypothesised that low muscle mass, measured by total psoas area (TPA), is associated with certain features of aneurysmal disease in the abdominal aorta, which may determine suitability for infrarenal endovascular aneurysm repair. A retrospective study was conducted to investigate whether patients with AAA and low muscle mass have different aortic anatomy compared with patients with AAA and normal muscle mass, and to explore the relationship, if any, between TPA and aortic anatomy.

Consecutive patients who underwent pre-operative computed tomography (CT) scans of the abdominal aorta in arterial phase between 1 January 2017 and 31 June 2018 were included. Patients with thoraco-abdominal, saccular, mycotic, isolated iliac, and post-dissection aneurysms were excluded. Also excluded were patients who had undergone previous aortic surgery. Anatomical features of the AAA (maximum AAA diameter, proximal aortic neck diameter, and length), AAA extent (infrarenal, juxtarenal, or

suprarenal), and TPA were assessed on axial and reconstructed CT images by trained operators (M.K., A.A., A.E.). TPA was measured on cross sections of the psoas muscles at the upper level of the fourth lumbar vertebra according to a previously described reproducible technique.³ Low muscle mass was defined as the lowest sex specific quartile of both psoas muscle areas on pre-operative CT scan. Aortic neck diameter (outer to outer) was measured in the first 10 mm below the lowest main renal artery. Neck length was the length of aorta below the lowest main renal artery of outer to outer maximum diameter not >32 mm, with $\leq 10\%$ gradient along a distance of 10 mm. AAAs were deemed suprarenal if extending above at least one main renal artery without involving the coeliac axis and thoraco-abdominal if extending to or beyond the coeliac axis. Measurements were performed on reconstructed cross sections. Continuous data were presented as mean \pm standard deviation or median (interquartile range), and tested with parametric or non-parametric tests, according to the underlying distributions. Multivariable analyses were performed when significant associations were found on univariable tests.

A total of 372 patients (318 men) were identified, with a mean age of 76 ± 7.4 years. Adequate TPA measurements were obtained in all but seven. Median AAA diameter was 65 (56–70) mm and mean TPA was 23 ± 6.0 mm². Thirty-nine had a juxtarenal and 25 a suprarenal AAA. Median body mass index was 27 (24–30) kg/m². Ninety-one (25%) had low muscle mass. In patients with infrarenal AAA, mean neck diameter was 24 ± 4.0 mm and median neck length was 28 (14–40) mm.

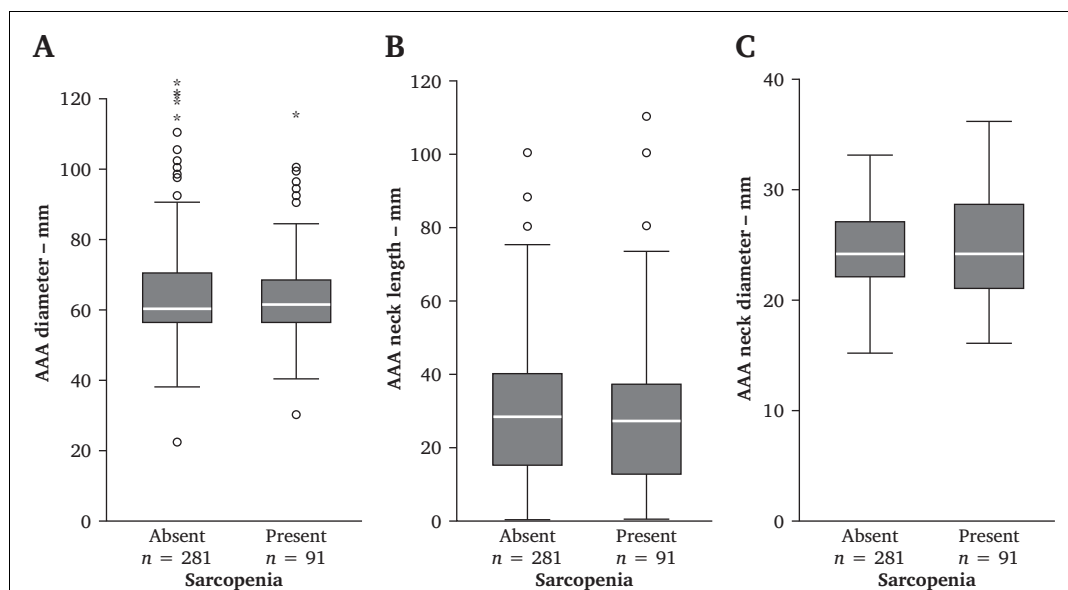


Figure 1. Abdominal aortic aneurysm (AAA) diameter (A), neck length (B), and neck diameter (C) in patients with and without low muscle mass ("sarcopenia"), defined as the lowest sex specific quartile of both psoas muscle areas. Ninety-one patients had low muscle mass and 281 did not.

TPA did not correlate with AAA diameter ($p = -.054$), neck length ($p = .049$), or neck diameter ($r = -.013$). There was no significant difference in AAA diameter, neck length, and neck diameter between patients with low muscle mass and those without (Fig. 1). Low muscle mass was commoner among patients with ruptured AAA (9/19 vs. 82/346; $p = .040$), but this difference disappeared after adjustment for age (odds ratio 2.4, 95% confidence interval 0.92–6.2; $p = .073$), as patients with ruptured AAA were significantly older (mean 81 ± 5.7 years vs. 76 ± 7.3 years; $p = .008$). The prevalence of low muscle mass in patients with infrarenal and those with juxta/suprarenal AAA was the same (25%).

The study did not demonstrate a relationship between TPA and aortic anatomy. The high prevalence of low muscle mass in (older) patients with ruptured AAA must be interpreted with caution, as age is known to be negatively associated with muscle mass. Furthermore, more liberal use of operative intervention in the emergency setting may have led to the inclusion of frailer patients in the rupture group. An adequately powered study on allcomers (including patients turned down for surgery) would be necessary to answer this question.

Like evolution of muscle mass, AAA formation is determined by genetic and environmental factors. It is unknown, however, why certain patients develop juxta- or suprarenal aortic aneurysms, as opposed to the commoner infrarenal variety, which is often treatable by standard endovascular techniques. It was hypothesised that yet unknown factors may influence both the development of muscle mass and the evolution of aortic anatomy in patients with AAA. If that were the case a relationship between anatomy of the aorta and TPA would be expected. In essence, the study does not support the hypothesis, and suggests that AAAs and low muscle mass do not share genetic or environmental aetiologies. The study has obvious limitations, including the retrospective design and the inclusion of selected patients with large AAA undergoing operative intervention. Nevertheless, these findings may be of interest to other researchers intending to explore a potential relationship between muscle mass and aortic disease.

CONFLICT OF INTEREST

None.

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