Original Article

HEALTH SCIENCES
MEDICINE

DOI: 10.32322/jhsm.1565696

J Health Sci Med. 2025;8(1):4-9

Evaluation of the relationship between body fat distribution and abdominal aorta calcified plaques with computed tomography

İsmail Taşkent[1], Ali Küpeli[2]

[1]Department of Radiology, Faculty of Medicine, Kastamonu Univesity, Kastamonu, Turkiye
[2]Department of Radiology, Trabzon Kanuni Training and Research Hospital, Trabzon, Turkiye

Cite this article as: Taşkent İ, Küpeli A. Evaluation of the relationship between body fat distribution and abdominal aorta calcified plaques with computed tomography. *J Health Sci Med.* 2025;8(1):4-9.

Received: 13.10.2024 ◆ **Accepted**: 13.11.2024 ◆ **Published**: 12.01.2025

ABSTRACT

Aims: In this study, we aimed to investigate the relationship between the development of calcified plaques in the abdominal aorta and the amount and distribution of abdominal fat tissue.

Methods: Between September 2021 and April 2024, we selected 69 patients with calcified plaques in the abdominal aorta and 165 control patients who underwent non-contrast abdominal computed tomography for suspected ureterorenal stones. Demographic characteristics, clinical features, subcutaneous, visceral, and total fat tissue areas, their ratios, and the diameter of the abdominal aorta lumen were recorded.

Results: Patients with abdominal aortic calcified plaques showed significantly higher visceral fat area, visceral fat ratio, hypertension, diabetes, and hepatosteatosis. In contrast, no significant differences were found between the two groups regarding height, weight, body-mass index, and total fat tissue area. Additionally, patients with aortic wall calcification had significantly larger aortic lumen diameters compared to those without.

Conclusion: Calcified atherosclerotic plaques in the abdominal aorta are particularly associated with visceral fat area.

Keywords: Atherosclerosis, computed tomography, body fat distribution

INTRODUCTION

Obesity is defined as the accumulation of an excessive amount of fat in the body. Clinically, it is usually assessed by an increase in body weight, and the most commonly used parameter is the body-mass index (BMI), which is calculated by dividing weight in kilograms by the square of height in meters.[1] BMI is the preferred tool for reporting the prevalence of obesity in the community; however, obesity is a highly heterogeneous condition.[1] Furthermore, different fat depots carry different metabolic risks.[2-5] Particularly, visceral adipose tissue is considered a unique pathogenic fat depot.[2,3,6] It is also linked to various pathological conditions such as increased insulin resistance, susceptibility to cancers, and higher mortality rates in hospitals.[3,7] The accumulation of visceral fat tissue also increases the risk of arterial hypertension and cardiovascular diseases.[3,8] Both visceral and subcutaneous adipose tissues can be measured using a specialized software through abdominal computed tomography (CT).

Although the underlying mechanisms of vascular calcification are not fully understood, known factors include diabetes, hypertension, smoking, aging, dialysis, and osteoporosis.[9] Abdominal aortic calcification correlates with an increased incidence of myocardial infarction, stroke, and peripheral artery disease.[10-15] Abdominal aortic calcification may be an indicator of more advanced atherosclerosis. CT and lateral abdominal X-Rays are used to assess aortic wall calcification.[2] Recent studies have increasingly focused on the impact of ectopic fat depots, particularly visceral fat tissue, on arterial calcification. Findings from these studies provide valuable insights into how abdominal fat distribution affects vascular health, enhancing our understanding of the pathophysiology of atherosclerosis.[15]

In this study, we evaluated the relationship between the amount and distribution of abdominal adipose tissue and the development of calcified atherosclerotic plaques in the abdominal aorta.

METHODS

The study was initiated with the approval of the Kastamonu University Faculty of Medicine Clinical Researches Ethics Committee (Date: 26.09.2024, Decision No: 2024/59). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Between

Corresponding Author: İsmail Taşkent, itaskent@kastamonu.edu.tr

September 2021 and April 2024, we recorded 69 patients with calcified atherosclerotic plaques in the abdominal aorta who had undergone non-contrast abdominopelvic CT for suspected urolithiasis. The control group included 165 patients who also underwent non-contrast abdominopelvic CT for suspected urolithiasis without evidence of calcified atherosclerotic plaques in the abdominal aorta in the clinical database. Age, sex, body weight (kg), and height (cm) were recorded for both patient and control groups. Additionally, BMI was calculated by dividing weight (kg) by height squared (m^2).

All CT examinations were conducted using a 64-detector CT scanner (Revolution EVO; GE Medical Systems, JAPAN) with axial images reconstructed at 1.25 mm thickness from the diaphragm to the pubic symphysis. Patients were scanned using a standard protocol without intravenous or oral contrast material. The CT protocol parameters were as follows: 120 kVp, 150-165 mAs tube current, maximum 2.5 mm collimation, 1.25 mm slice thickness, and 0.5 s rotation time. Images were reconstructed with multiplanar reformations.

Two radiologists re-evaluated the CT images to reach consensus. The presence of calcified atherosclerotic plaques in the abdominal aorta was identified from the aortic hiatus to the iliac bifurcation, and the abdominal aortic diameter was measured (Figure 1).

Figure 1. 46 year-old man with renal colic. Unenhanced abdominal CT image shows calcified atherosclerotic plaques in the abdominal aorta

CT: Computed tomography

Axial CT images at the level of the umbilicus were used to measure both visceral and subcutaneous fat areas. Measurements were performed using the workstation software (ADW 4.7, GE), applying regions of interest (ROI) (Figure 2). Total fat area (TFA=SFA+VFA), visceral fat percentage (VF%=VFA/TFA×100), and subcutaneous fat percentage (SF%=SFA/TFA×100) were calculated and recorded.

The liver attenuation of the right lobe was measured using a standard ROI of approximately 200 mm^2, and hepatosteatosis was defined as a liver attenuation value of 40 HU or less.[16-18] Additionally, the presence of hypertension and diabetes mellitus was recorded for both patient and control groups.

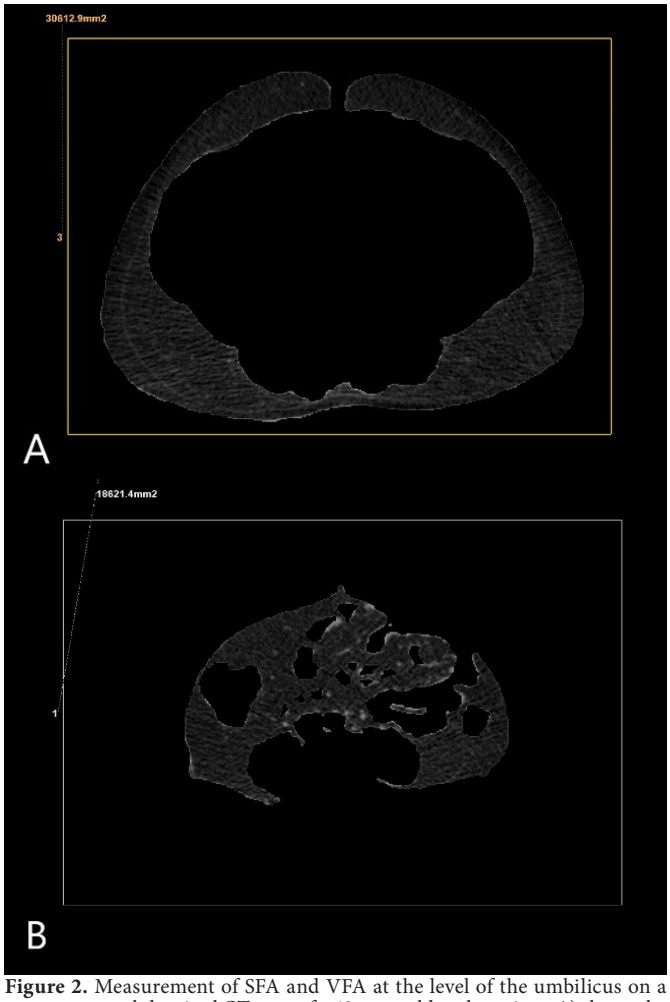

Figure 2. Measurement of SFA and VFA at the level of the umbilicus on a non-contrast abdominal CT scan of a 49-year-old male patient. A) shows the SFA measurement, recorded as 306.12 cm^2. B) shows the VFA measurement, recorded as 186.21 cm^2

SFA: Subcutaneous fat area, VFA: Visceral fat area, CT: Computed tomography

All data were analyzed using the Statistical Package for the Social Sciences (SPSS 20.0, SPSS Inc., Chicago, IL, USA) and MedCalc Statistical Software version 16.8 (MedCalc Software bvba, Ostend, Belgium). Means and ranges for age, abdominal aortic diameter, BMI, SFA, VFA, TFA, SF%, and VF% values were calculated for both groups. The Kolmogorov-Smirnov test was used to assess deviation from normal distribution. The Mann-Whitney U test and Student's t-test were used to compare the CT findings of the patient and control groups. The Mann-Whitney U test was used to analyze BMI. The student's t-test was used for the analysis of age, abdominal aortic diameter, VFA, SFA, TFA, SF%, and VF%. Additionally, chi-square tests were used to examine the differences in the frequencies of hepatosteatosis, hypertension, and diabetes mellitus between the two groups. Finally, univariate analysis was used to obtain odds ratios (ORs). Results with a p-value of less than 0.05 were considered statistically significant.

RESULTS

In this study, 69 patients (38 men, 31 women) with calcified atherosclerotic plaques in the abdominal aorta and 165 control patients (87 men, 78 women) were evaluated. The average ages of the patient and control groups were 63.96 (±11.95) and 61.61

(±7.36) years, respectively. The average heights (cm) and body weights (kg) of the patient and control groups were 168.77 (±10.05) cm and 164.92 (±7.09) cm, 83.57 (±14.01) kg and 84.51 (±14.87) kg, respectively. The average BMI (kg/m²) values for the patient and control groups were 29.63 (±6.00) and 31.17 (±5.53), respectively. No statistically significant difference was observed between the two groups in terms of average height, body weight, or BMI (p=0.06).

The average values of SFA, VFA, TFA, VF%, and SF% in the patient and control groups were as follows: SFA: 241.51 cm² and 325.23 cm²; VFA: 246.62 cm² and 162.41 cm²; TFA: 488.13 cm² and 487.64 cm²; VF%: 47.26 and 33.58; SF%: 52.72 and 66.42. No significant difference was observed in TFA (p=0.98). However, significant differences were detected between the two groups in terms of VFA, SF%, and VF% (p < 0.001) (Figure 3, 4) (Table 1). The average abdominal aortic diameters in the patient and control groups were 19.03 cm and 17.41 cm, respectively, with a significant difference between the two groups (p<0.001).

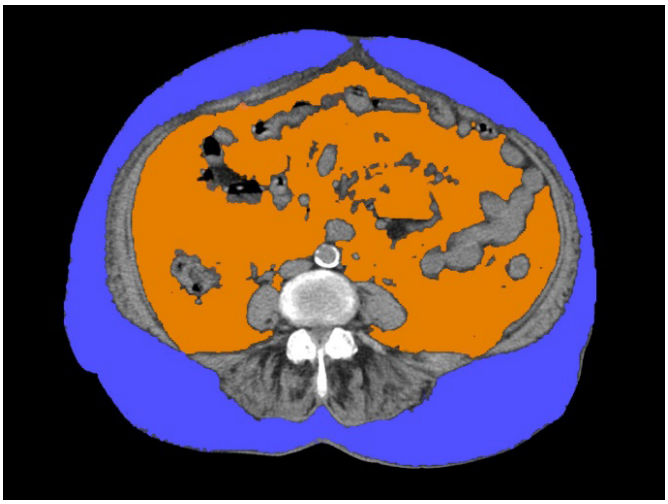

Figure 3. 46 year-old man with the calcified atherosclerotic plaques in the abdominal aorta shows increased VFA/TFA

VFA: Visceral fat area, TFA: Total fat area

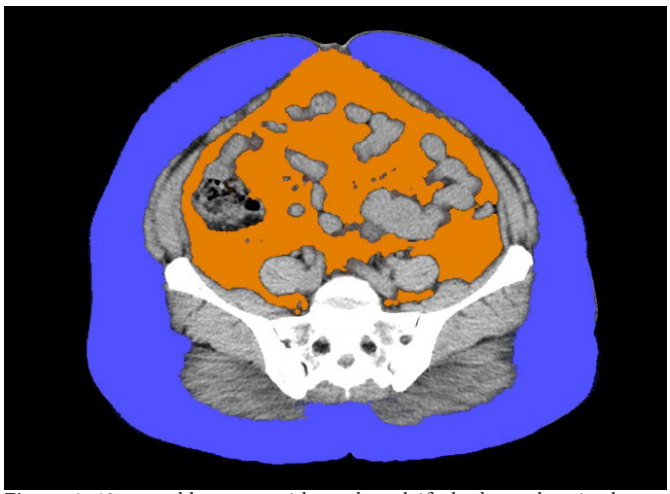

Figure 4. 48 year-old woman without the calcified atherosclerotic plaques in the abdominal aorta shows decreased VFA/TFA

VFA: Visceral fat area, TFA: Total fat area

Data on the presence of hepatosteatosis (HS), hypertension (HT), and diabetes mellitus (DM) are provided in Table 1. The rate of HS in patients with calcified atherosclerotic plaques in the abdominal aorta was statistically significantly higher compared to the control group (p<0.05), and the rates of HT and DM were observed to be significantly higher (p<0.001). OR analyses indicated that the presence of DM (OR=4.11), the presence of HT (OR=3.31), an increase in aortic diameter (OR=2.25), VFA greater than 160cm² (OR=4.04) and VF% (OR=4.47) were associated with the presence of calcified atherosclerotic plaques in the abdominal aorta (Table 2).

DISCUSSION

In this retrospective study, we found a strong association between calcified atherosclerotic plaques in the abdominal aorta and visceral obesity. Additionally, an increase in aortic diameter was identified as a factor associated with the presence of calcified atherosclerotic plaques in the abdominal aorta.

Obesity, defined as abnormal or excessive fat accumulation in the body, is typically assessed using BMI, which is the preferred tool for evaluating obesity prevalence within communities. However, obesity is a highly heterogeneous condition.[1] There is also a J-shaped relationship between BMI and the risk of morbidity and mortality, which is linked to hypertension, dyslipidemia, diabetes mellitus, cardiovascular diseases, and cancer.[19-21] Many studies in the literature have investigated abdominal fat distribution by using VFA and SFA, both evaluated by CT. VFA has been shown to correlate more strongly with cardiometabolic risk than SFA.[3,22] For example, Rosenquist et al.[23] demonstrated that an increase in VFA may elevate cardiometabolic risks.

Major clinical risk factors for calcified atherosclerotic plaques in the abdominal aorta include genetic predisposition, male gender, smoking, a sedentary lifestyle, metabolic syndrome, hypertension, and, notably, visceral obesity.[24-28] The mechanisms underlying aortic calcification are complex and often interconnected, with one factor potentially triggering multiple others in a cycle of adverse outcomes. Efe et al.[29] reported that a high levels of VFA increase the risk of aortic atherosclerosis. In our study, we selected a control group with no significant differences in height, weight, or BMI compared to the patient group. We observed that patients with calcified atherosclerotic plaques in the abdominal aorta had higher VFA and VF% than controls, while lower SFA and SF% values were noted in the patient group compared to the control group.

We also found that patients with calcified aortic plaques experienced hepatosteatosis, hypertension, and diabetes mellitus more frequently than those without, consistent with findings by Efe et al.[29] Our study identified DM, HT, VF% greater than 34%, and VFA over 160 cm² as the strongest factors associated with calcified aortic plaques.

The prevalence of calcified plaques in the abdominal aorta ranges from 2.1% to 14.7% depending on the age and characteristics of the selected population.[30-33] This condition becomes more common with age, and one study found it to be more prevalent among older patients with ischemic heart disease.[32] Calcified plaques in the abdominal aorta can be evaluated using lateral abdominal X-Rays, electron-

Table 1. Demographic and clinical characteristics of patients with and without calcified atherosclerotic plaques

	Total	Calcified atherosclerotic plaques in the abdominal aorta		p value
		Present±SD (Min- Max)	Absent±SD (Min- Max)	
Number	234	69	165	
Age (year)	62.30±9.00 (44-89)	63.96±11.95 (44-89)	61.61±7.36 (45-72)	0.132
Gender				
Male	125	38	87	0.85
Famele	109	31	78	
Height (cm)	166.06±8.24 (149-191)	168.77±10.05 (150-191)	164.92±7.09 (149-182)	0.16
Weight (kg)	84.23±14.60 (58-140)	83.57±14.01 (62-129)	84.51±14.87 (58.00-140.00)	0.64
BMI	30.72±5.70 (19.38-48.44)	29.63±6.00 (22.23-46.09)	31.17±5.53 (19.38-48.44)	0.06
Hepatosteatosis				
Present	92	36	56	0.014
Absent	142	33	109	
Diabetes mellitus				
Present	56	30	26	<0.001
Absent	178	39	139	
Hypertension				
Present	98	43	55	<0.001
Absent	136	26	110	
VFA (cm^2)	187.24±105.47 (37-610)	246.62±158.00 (56-610)	162.41±57.88 (37-373)	<0.001
VF%	37.61±13.43 (11-80)	47.26±15.81 (21-80)	33.58±9.84 (11-69)	<0.001
SFA (cm^2)	300.54±103.44 (85-601)	241.51±93.11 (85-464)	325.23±97.60 (140-601)	0.005
SF%	62.38±13.42 (20-89)	52.72±15.79 (20-79)	66.42±9.84 (31-89)	<0.001
TFA (cm^2)	487.79±148.19 (154-936)	488.13±200.54 (154-936)	487.64±120.52 (228-846)	0.98
Aortic diameter (mm)	17.89±2.19 (14.30-26.59)	19.03±2.83 (15.41-26.59)	17.41±1.65 (14.30-22.68)	<0.001

SD: Standard deviation, Min: Minimum, Max: Maximum, BMI: Body-mass index, VFA: Visceral fat area, VF%: Percentage of visceral fat, SFA: Subcutaneous fat area, SF%: Percentage of subcutaneous fat, TFA: Total fat area

Table 2. Odds ratio of the presence of calcified atherosclerotic plaques in the abdominal aorta

Variable	Odds ratio	95% CI
Male gender	1.41	0.81-2.63
Age >63 y	0.95	0.54-1.66
BMI (>30.4)	0.52	0.29-0.92
Hepatosteatosis	2.12	1.20-3.76
Hypertension	3.31	1.84-5.94
Diabetes mellitus	4.11	2.18-7.75
VFA (>160 cm^2)	4.04	2.19-7.48
VF% (>34)	4.47	2.40-8.32
SFA (<296 cm^2)	2.08	1.17-3.71
TFA (>476 cm^2)	0.98	0.56-1.72
Aort diameter (>17.5 mm)	2.25	1.26-4.03

BMI: Body-mass index, VFA: Visceral fat area, VF%: Percentage of visceral fat, SFA: Subcutaneous fat area, SF%: Percentage of subcutaneous fat, TFA: Total fat area

beam computed tomography, or plain CT, with each method having its own advantages and limitations. Plain CT, the most commonly used method, was also preferred in our study.

Our findings show that patients with calcified plaques in the abdominal aorta have significantly larger aortic diameters than those without. Visceral obesity may contribute to a larger aortic diameter, thicker aortic walls, and increased vascular thickness. This phenomenon can be attributed to several factors, including adaptation to elevated blood pressure due to hypertension, increased blood volume, and structural or functional abnormalities in the aorta specific to obesity.[24] Furthermore, visceral obesity accelerates vascular aging and raises the risk of future cardiovascular events.[34]

Visceral obesity is associated with various pathological conditions, such as abnormal glucose and lipid metabolism, insulin resistance, and increased risk of cardiovascular diseases. It also predisposes individuals to certain cancers and surgical complications.[7] As an active endocrine tissue, visceral fat releases atherogenic factors that promote atherosclerosis. Consequently, visceral obesity accelerates vascular aging and increases the risk of cardiovascular diseases, hypertension, and calcified plaques.[7] Additionally, increased visceral fat may contribute to dysfunctional subcutaneous fat tissue, resulting in excessive fat accumulation in ectopic locations like the heart, liver, skeletal muscle, pancreas, and gastrointestinal tract. This ectopic fat may be a cause of hepatosteatosis.[35] Despite the strong relationship between metabolic syndrome and visceral adiposity and hepatosteatosis, visceral adiposity and hepatosteatosis are not currently considered diagnostic criteria for metabolic syndrome.

Limitations

This study has several limitations. First, we evaluated the CT images based on consensus and did not assess inter-observer and intra-observer variability. Second, the sample size was relatively small. Third, we had no access to laboratory parameters such as lipid panels. Fourth, measurements of adipose tissue areas were obtained from a single CT slice at the level of the abdomen instead of volume calculations. Fifth, the single-center design, absence of long-term follow-up data, and lack of control for potential confounding factors such as lifestyle variables may limit the generalizability of our findings. Conducting larger, multicenter studies could enhance the generalizability of the findings and help validate the results across diverse population groups

CONCLUSION

Calcified atherosclerotic plaques in the abdominal aorta are associated with obesity, particularly visceral obesity. Patients with calcified atherosclerotic plaques in the abdominal aorta may have higher rates of hypertension, diabetes mellitus, increased VFA, VF%, and aortic diameter compared to those without. However, further studies are needed to better understand the relationship between calcified aortic plaques, diabetes mellitus, cardiovascular diseases, and metabolic syndrome.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Kastamonu University Faculty of Medicine Clinical Researches Ethics Committee (Date: 26.09.2024, Decision No: 2024/59)

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. *J Chronic Dis*. 1972;25(6):329-343. doi:10.1016/0021-9681(72)90027-6
2. McLaughlin T, Lamendola C, Liu A, Abbasi F. Preferential fat deposition in subcutaneous versus visceral depots is associated with insulin sensitivity. *J Clin Endocrinol Metabol*. 2011;96(11): E1756-E1760. doi:10.1210/jc.2011-0615
3. Fox CS, Massaro JM, Hoffmann U, et al. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham heart study. *Circulation*. 2007;116(1):39-48. doi:10.1161/circulationaha.106.675355
4. McLaughlin T, Abbasi F, Lamendola C, Reaven G. Heterogeneity in the prevalence of risk factors for cardiovascular disease and type 2 diabetes mellitus in obese individuals: effect of differences in insulin sensitivity. *Arch Int Med*. 2007;167(7):642-648. doi:10.1001/archinte.167.7.642
5. Karlsberg D, Steyer H, Fisher R, et al. 426 impact of visceral abdominal tissue on coronary artery disease as defined by quantitative computed tomographic angiography. *J Cardiovas Comp Tomograp*. 2022;16(4):S13.
6. Imai A, Komatsu S, Ohara T, et al. Visceral abdominal fat accumulation predicts the progression of noncalcified coronary plaque. *Atherosclerosis*. 2012;222(2):524-529.
7. Shuster A, Patlas M, Pinthus JH, Mourtzakis M. The clinical importance of visceral adiposity: a critical review of methods for visceral adipose tissue analysis. *Br J Radiol*. 2012;85(1009):1-10. doi:10.1259/bjr/38447238
8. Lamarche B, Lemieux S, Dagenais GR, Després JP. Visceral obesity and the risk of ischaemic heart disease: insights from the Québec cardiovascular study. *Growth Horm IGF Res*. 1998; 8(Suppl B):1-8. doi:10.1016/s1096-6374(98)80018-x
9. Golledge J. Abdominal aortic calcification: clinical significance, mechanisms and therapies. *Curr Pharmaceut Design*. 2014; 20(37):5834-5838. doi:10.2174/1381612820666140212195309
10. Yoon HE, Park BG, Hwang HS, et al. The prognostic value of abdominal aortic calcification in peritoneal dialysis patients. *Int J Med Sci*. 2013;10(5):617-623. doi:10.7150/ijms.5773
11. Ohya M, Otani H, Kimura K, et al. Vascular calcification estimated by aortic calcification area index is a significant predictive parameter of cardiovascular mortality in hemodialysis patients. *Clin Experiment Nephrol*. 2011;15(6):877-883. doi:10.1007/s10157-011-0517-y
12. Nielsen M, Ganz M, Lauze F, et al. Distribution, size, shape, growth potential and extent of abdominal aortic calcified deposits predict mortality in postmenopausal women. *BMC Cardiovasc Disord*. 2010;10(56):1-10.
13. Verbeke F, Van Biesen W, Honkanen E, et al. Prognostic value of aortic stiffness and calcification for cardiovascular events and mortality in dialysis patients: outcome of the calcification outcome in renal disease (CORD) study. *Clin J Am Soci Nephrol*. 2011;6(1):153-159. doi:10.2215/cjn.05120610
14. Parr A, Buttner P, Shahzad A, Golledge J. Relation of infra-renal abdominal aortic calcific deposits and cardiovascular events in patients with peripheral artery disease. *Am J Cardiol*. 2010; 105(6):895-899. doi:10.1016/j.amjcard.2009.10.067
15. Ordulj I, Šarić F, Tandara M, et al. Visceral and ectopic abdominal fat effect on the calcification of the abdominal aorta and its branches-an MSCT study. *Life (Basel)*. 2023;14(1):2.
16. Park YS, Park SH, Lee SS, et al. Biopsy-proven nonsteatotic liver in adults: estimation of reference range for difference in attenuation between the liver and the spleen at nonenhanced CT. *Radiology*. 2011;258(3):760-766. doi:10.1148/radiol.10101233
17. Kodama Y, Ng CS, Wu TT, et al. Comparison of CT methods for determining the fat content of the liver. *AJR Am J Roentgenol*. 2007;188(5):1307-1312. doi:10.2214/ajr.06.0992
18. Park SH, Kim PN, Kim KW, et al. Macrovesicular hepatic steatosis in living liver donors: use of CT for quantitative and qualitative assessment. *Radiology*. 2006;239(1):105-112. doi:10.1148/radiol.2391050361

19. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. *New Engl J Med*. 1995;333(11):677-685. doi:10.1056/nejm199509143331101
20. Whitlock G, Lewington S, Sherliker P, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet (London, England)*. 2009;373(9669):1083-1096. doi:10.1016/s0140-6736(09)60318-4
21. Lee H, Kim H, Jeon JS, Noh H, Lee EJ, Kwon SH. Association between abdominal perivascular adipose tissue quantity and quality assessed by CT and cardiometabolic risk. *Clin Nutrit (Edinburgh, Scotland)*. 2023;42(6):869-878. doi:10.1016/j.clnu.2023.04.003
22. Goodpaster BH, Krishnaswami S, Harris TB, et al. Obesity, regional body fat distribution, and the metabolic syndrome in older men and women. *Arch Int Med*. 2005;165(7):777-783. doi:10.1001/archinte.165.7.777
23. Rosenquist KJ, Pedley A, Massaro JM, et al. Visceral and subcutaneous fat quality and cardiometabolic risk. *JACC Cardiovas Imag*. 2013;6(7):762-771. doi:10.1016/j.jcmg.2012.11.021
24. Jayalath RW, Mangan SH, Golledge J. Aortic calcification. *Eur J Vasc Endovasc Surg*. 2005;30(5):476-488. doi:10.1016/j.ejvs.2005.04.030
25. Kim ED, Kim JS, Kim SS, et al. Association of abdominal aortic calcification with lifestyle and risk factors of cardiovascular disease. *Korean J Family Med*. 2013;34(3):213-220. doi:10.4082/kjfm.2013.34.3.213
26. Figueiredo CP, Rajamannan NM, Lopes JB, et al. Serum phosphate and hip bone mineral density as additional factors for high vascular calcification scores in a community-dwelling: the São Paulo Ageing & Health Study (SPAH). *Bone*. 2013;52(1):354-359. doi:10.1016/j.bone.2012.10.019
27. Sutton-Tyrrell K, Newman A, Simonsick EM, et al. Aortic stiffness is associated with visceral adiposity in older adults enrolled in the study of health, aging, and body composition. *Hypertension (Dallas, Tex : 1979)*. 2001;38(3):429-433. doi:10.1161/01.hyp.38.3.429
28. Jain SH, Massaro JM, Hoffmann U, et al. Cross-sectional associations between abdominal and thoracic adipose tissue compartments and adiponectin and resistin in the Framingham heart study. *Diabetes Care*. 2009;32(5):903-908. doi:10.2337/dc08-1733
29. Efe D, Aygün F, Acar T, Yildiz M, Gemici K. Investigation of relation between visceral and subcutaneous abdominal fat volumes and calcified aortic plaques via multislice computed tomography. *Vascular*. 2015;23(4):396-402. doi:10.1177/1708538114552012
30. Pleumeekers HJ, Hoes AW, van der Does E, et al. Aneurysms of the abdominal aorta in older adults. *Am J Epidemiol*. 1995;142(12):1291-1299. doi:10.1093/oxfordjournals.aje.a117596
31. Newman AB, Arnold AM, Burke GL, O'Leary DH, Manolio TA. Cardiovascular disease and mortality in older adults with small abdominal aortic aneurysms detected by ultrasonography: the cardiovascular health study. *Ann Int Med*. 2001;134(3):182-90. doi:10.7326/0003-4819-134-3-200102060-00008
32. Madaric J, Vulev I, Bartunek J, et al. Frequency of abdominal aortic aneurysm in patients >60 years of age with coronary artery disease. *Am J Cardiol*. 2005;96(9):1214-1216. doi:10.1016/j.amjcard.2005.06.058
33. Fornasari A, Kuntz S, Martini C, et al. Objective methods to assess aorto-iliac calcifications: a systematic review. *Diagnostics (Basel, Switzerland)*. 2024;14(10):1053. doi:10.3390/diagnostics14101053
34. O'Connor SD, Graffy PM, Zea R, Pickhardt PJ. Does nonenhanced CT-based quantification of abdominal aortic calcification outperform the Framingham risk score in predicting cardiovascular events in asymptomatic adults? *Radiology*. 2019;290(1):108-115. doi:10.1148/radiol.2018180562
35. Tchernof A, Després JP. Pathophysiology of human visceral obesity: an update. *Physiol Rev*. 2013;93(1):359-404. doi:10.1152/physrev.00033.2011