Computed tomography attenuation of periaortic adipose tissue in abdominal aortic aneurysms

Original Research

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Short Title: CT attenuation of periaortic adipose tissue in AAA

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Tweet: Higher CT attenuation of periaortic adipose tissue in symptomatic #AAA in aneurysm & non-aneurysm aorta than asymptomatic & controls

Declaration of research ethics

The study was conducted with research ethics committee approval (14/SS/0080, 21/ES/0044), with Caldicott approval (2021/0066) and was conducted in accordance with the Declaration of Helsinki.

Sources of funding

SD, MBJS, AJF, MRD, DEN, ROF and MCW are supported by the British Heart Foundation (PG/21/10461, FS/18/31/33676, FS/19/15/34155, FS/SCRF/21/32010, RE/18/5/34216, FS/ICRF/20/26002, CH/09/002/26360). MRD is the recipient of the Sir Jules Thorn Award for Biomedical Research 2015 (15/JTA). DEN is the recipient of a Wellcome Trust Senior Investigator Award (WT103782AIA). DD is funded by grants from the National Institutes of Health/National Heart, Lung, and Blood Institute (1R01HL148787-01A1 and 1R01HL151266) and a grant from the Winnick Foundation.

Disclosures

MRD has received speaker fees from Pfizer, Bristol Myers Squib, Radcliffe Cardiology, Edwards and Novartis. He has received consultancy fees from Novartis, Jupiter Bioventures, Beren and Silence therapeutics. DD has received software royalties from Cedars Sinai Medical Center and also has a patent. MCW has given talks for Canon Medical Systems, Siemens Healthineers and Novartis. SD, ET, MBJS, JN, AJF, DEN and ROF have no conflicts of interest to declare.

Word count

Abstract: 245 words

Text: 2523 words

Number of tables and figures: 1 tables | 3 figures

Summary Statement

Periaortic adipose tissue CT attenuation was increased in symptomatic abdominal aortic aneurysms, including in patients with acute aortic rupture.

Key Points

There was no difference in periaortic adipose tissue attenuation between asymptomatic patients and controls in the aneurysmal segment (-81.44 vs -83.27 HU; P=0.43) and the non-aneurysmal segment (-75.43 vs -78.81 HU; P=0.19).

Periaortic adipose tissue attenuation was higher in symptomatic patients within aneurysmal (-57.85 HU) and non-aneurysmal (-58.16 HU) segments compared with asymptomatic patients and control groups (P<0.001).

Keywords

Periaortic adipose tissue
Periaortic fat attenuation
Computed tomography angiography
Cardiovascular imaging
Abdominal aortic aneurysm

Abstract

Purpose: To assess periaortic adipose tissue attenuation on CT angiography in different abdominal aortic aneurysm disease states.

Materials and Methods: In a retrospective observational study from January 2018 to December 2022, periaortic adipose tissue attenuation was assessed on CT angiography in patients with asymptomatic or symptomatic (including rupture) abdominal aortic aneurysms, and control individuals without aneurysms. Adipose tissue attenuation was measured using semi-automated software in periaortic aneurysmal and non-aneurysmal segments of the abdominal aorta, and in subcutaneous and visceral adipose tissue. Periaortic adipose tissue attenuation values between the three groups was assessed using Students t-test and Wilcoxon rank sum test followed by a multi-regression model.

Results: Eighty-eight individuals (median age, 70 [IQR, 65-78] years; 78 male and 10 female) were included: 70 patients with abdominal aortic aneurysms (40 asymptomatic and 30 symptomatic including 24 with rupture), and 18 controls. There was no evidence of differences in the periaortic adipose tissue attenuation in the aneurysmal segment in asymptomatic patients versus controls ((-81.44±7 versus -83.27±9 HU, Hounsfield units, P=0.43) and attenuation in non-aneurysmal segments between asymptomatic patients versus controls (-75.43±8 versus -78.81±6 HU, P=0.08). However, symptomatic patients demonstrated higher periaortic adipose tissue attenuation in both aneurysmal (-57.85±7 HU, P<0.0001) and non-aneurysmal segments (-58.16±8 HU, P<0.0001) when compared with the other two groups.

Conclusions: Periaortic adipose tissue CT attenuation was not increased in stable abdominal aortic aneurysm disease. There was a generalised increase in attenuation

in patients with symptomatic disease, likely reflecting the systemic consequences of acute rupture.

1. Introduction

Perivascular adipose tissue refers to any adipose tissue surrounding a blood vessel, including both periaortic and pericardial deposits.[1] This tissue is metabolically active and secretes a number of bioactive substances termed ‘adipokines’. Excessive caloric intake causes adipose tissue remodelling through a process of adipocyte hyperplasia and hypertrophy, subsequent adipocyte dysfunction and apoptosis followed by inflammatory cell infiltration and fibrosis, which results in a chronic low-grade inflammatory state.[2]

Analysis of pericoronary adipose tissue attenuation has shown its ability to predict myocardial infarction and all-cause and cardiac mortality on retrospective analyses of coronary CT angiograms.[3, 4] This imaging biomarker demonstrates a difference in CT-measured adipose tissue attenuation (from more negative to less negative Hounsfield Unit [HU] values), purported to occur as a result of a shift in lipophilic content within the adipose tissue, possibly due to the presence of inflammation.[5]

Abdominal aortic aneurysm pathophysiology is incompletely understood. It appears to result from medial wall atrophy and degeneration,[6] which may be triggered by an initial inflammatory response, and neutrophil infiltration at the junction between the media and adventitia.[6, 7] A previous study reported that the presence of an abdominal aortic aneurysm was an independent predictor of higher perivascular adipose tissue attenuation around the aneurysm sac and correlated with aortic volume.[8]

The objective of the present study was to assess periaortic adipose tissue attenuation on CT imaging in different abdominal aortic aneurysm disease states.

2. Materials and Methods

2.1 Study Design and Patients

This was a single center, retrospective, observational study conducted between January 2018 and December 2022. Three groups comprised the study sample: (i) asymptomatic patients with an unruptured abdominal aortic aneurysm, (ii) symptomatic patients with CT evidence of a ruptured abdominal aortic aneurysm, or unruptured abdominal aortic aneurysm proceeding to repair, and (iii) controls with a normal calibre abdominal aorta.

Asymptomatic patients and control subjects were consecutive study participants recruited in the Sodium [^{18}F]Fluoride Imaging of Abdominal Aortic Aneurysms (SoFIA^{3}) study (NCT02229006). This was a prospective case-control observational cohort study of patients with asymptomatic abdominal aortic aneurysms under US surveillance. Controls were recruited through the National Health Service Lothian National Abdominal Aortic Aneurysm Screening Programme and had documented normal caliber aortas (<30-mm anteroposterior diameter).[(9)] The national programme for abdominal aortic aneurysm screening in Scotland invites all men aged 65 years who are sent an invitation to attend for an abdominal US screening test. Men who have a normal result are discharged from the screening programme after one visit. This study was performed with Research Ethics Committee approval (14/SS/0080) with informed patient consent and in accordance with the declaration of Helsinki.

Symptomatic patients were those who had presented to their local Emergency Department with abdominal pain and a clinical suspicion of ruptured abdominal aortic

aneurysm. Patients were included only if there was CT evidence of a ruptured abdominal aortic aneurysm or if they proceeded to undergo emergency repair of their abdominal aortic aneurysm. This use of retrospective imaging was approved by the Research Ethics Committee (21/ES/0044), and the requirement for obtaining informed consent was waived.

2.2 Study assessments

2.2.1 CT imaging

Asymptomatic patients and control subjects underwent a contrast-enhanced CT angiography (120 kV, 145 mAs, 3/3 mm, field of view 400; and 1/1 mm, field of view 300; triggered at 181 HU) at their study visit. Symptomatic patients underwent CT imaging according to local Emergency Department and Radiology protocols. Aortic diameter was measured in the antero-posterior plane using the Picture Archiving and Communications System (PACS, Carestream Health).

2.2.2 Adipose tissue assessment

Data were exported in Digital Imaging and Communications in Medicine (DICOM) format. Assessment of visceral and subcutaneous adipose tissue attenuation was performed using OsiriX (version 13.0.0; OsiriX Imaging Software, Geneva, Switzerland). For each scan, four circular regions of interest were drawn in both the visceral fat and the subcutaneous fat at the mid-level of the third lumbar vertebra.[10, 11] The mean attenuation values were then calculated for each patient.

Periaortic adipose tissue attenuation assessment was performed using semi-automated software (Autoplaque, version 2.5, Cedars-Sinai Medical Center, Los

Angeles).[12] Analysis for each individual was performed in two aortic regions: (i) the abdominal aorta (from just below the lowermost renal artery until the aortic bifurcation), and (ii) the normal aorta (a 20-mm straight segment of non-aneurysmal aorta, usually in the suprarenal abdominal aorta). In patients with rupture, the normal segment of the aorta excluded areas with CT-evidence of hemorrhage. The mean tissue attenuation at a 2-mm distance from the vessel wall was considered. Conversion factors were used on the attenuation values to adjust for different scans having been performed at different tube potentials.[13, 14]

2.3 Statistical analysis

Continuous variables with normal distribution are presented as mean ± standard deviation, and skewed continuous variables are presented as median [interquartile range]. Categorical variables are presented as number (percentage). Periaortic adipose tissue attenuation values between the three groups were first assessed using Students t-test and Wilcoxon rank sum test. Following this a multi-regression model was used, using periaortic adipose tissue attenuation as the independent variable and age, hypertension and hypercholesterolemia as the independent variables. Subcutaneous and visceral adipose tissue attenuation was assessed using Wilcoxon rank sum test. Further statistical significance between the groups was assessed using Pearson's Chi-squared test, Fisher's exact test and Kruskal-Wallis rank sum test as appropriate. All statistical analysis was performed in RStudio (V2022.02.3+492, RStudio, PBC). A two-sided $P<0.05$ was considered statistically significant.

3. Results

The study sample comprised 88 individuals (median age, 70 [IQR, 65-78] years; 78 male, 10 female): 40 had an asymptomatic unruptured abdominal aortic aneurysm, 30 had a symptomatic abdominal aortic aneurysm (6 of which were unruptured), and 18 were controls with a normal aortic diameter. All patients without aortic rupture presented with abdominal pain that was deemed to be due to an unruptured abdominal aortic aneurysm found on CT imaging. Symptomatic patients had a median C-reactive protein of 21 [6 to 54] mg/L. Control subjects were younger and had fewer medical co-morbidities than the other two groups (Table 1).

3.1 Periaortic adipose tissue attenuation

There was no evidence of a difference in periaortic adipose tissue attenuation in the aneurysmal aorta between asymptomatic patients (-81.44±7 HU) and controls (-83.27±9 HU; P=0.43) (Figure 1). Similarly, in the non-aneurysmal abdominal aorta, the periaortic adipose tissue attenuation of asymptomatic patients (-75.43±8 HU) did not differ from comparable segments in control subjects (-78.81±6 HU; P=0.08).

In symptomatic patients, both the aneurysmal abdominal aorta (-57.85±7 HU) and the non-aneurysmal abdominal aortic segment (-58.16±8 HU) demonstrated higher periaortic adipose tissue attenuation values when compared with the other two groups (all P<0.0001; Figure 2). This was also true for separate analyses of symptomatic patients without rupture (aneurysmal segment -58.72±6 HU, both P<0.0001; non-aneurysmal segment -60.84±9 HU, asymptomatic P=0.008 and control P=0.003) and with rupture (aneurysmal segment -57.63±7 HU, both P<0.0001; non-aneurysmal segment -57.47±7 HU, both P<0.0001). In a multivariable regression analysis, acutely

symptomatic aneurysms were associated with high periaortic adipose tissue attenuation values around the aneurysm as an independent variable (univariable Beta-estimate 25.42, P<0.0001) and when corrected for age, the presence of hypertension and the presence of hypercholesterolemia (multivariable Beta-estimate = 25.39, P<0.0001). Similar patterns were seen around segments of normal aorta (univariable Beta-estimate 20.65, P<0.0001; multivariate Beta-estimate = 21.21, P<0.0001) (Figure 1). A weak positive correlation was observed between aortic diameter and the periaortic adipose tissue attenuation in symptomatic patients (r=0.29, P=0.025), but not in the other two groups. No relationship was observed between periaortic adipose tissue attenuation and C-reactive protein.

3.2 Visceral and subcutaneous fat attenuation

Over a median area of 82.5 mm^2, we found no evidence of differences in adipose tissue attenuation values between visceral (-106.1 [-109.5 to -99.9] HU) and subcutaneous fat (-108.2 [-112.2 to -101.3] HU; P=0.06). Visceral fat attenuation was similar across the study groups (controls, -106.5 [-108.7 to -100.6]; asymptomatic patients, -106.2 [-109.9 to -102.1]; symptomatic patients without rupture patients, -102.0 [-107.6 to -93.1]; and symptomatic patients with rupture, -104.3 [-109.5 to -97.0] HU; P>0.05 for all comparisons, Figure 3).

The subcutaneous fat attenuation was slightly higher in the symptomatic patients (-102.1 [-111.6 to -96] HU) when compared with asymptomatic patients (-110.0 [-112.2 to -106.7] HU; P=0.006) but not when compared with controls (-107.8 [-112.2 to -102.7] HU; P=0.095). These differences were most apparent in the subgroup of symptomatic patients with ruptured aneurysms (Figure 3).

4. Discussion

In this case control study, we assessed periaortic adipose tissue CT attenuation in patients with abdominal aortic aneurysm disease. We found that periaortic adipose tissue attenuation was not directly influenced by the presence of abdominal aortic aneurysm disease per se, with similar values observed in individuals with (-81.44±7 HU) and without (-83.27±9 HU) abdominal aortic aneurysms. Indeed, even within the same individual, we observed no differences in periaortic adipose tissue attenuation in areas with and without aneurysm disease (-75.43±8 HU). However, patients with symptomatic aneurysms had increased periaortic adipose tissue attenuation (-57.85±7 HU), which was also present within non-aneurysmal segments of the aorta (-58.16±8 HU). This suggests that increased periaortic adipose tissue attenuation is not a localised feature of active aneurysm disease itself, but a broader generalised aortic response to unstable active disease and acute rupture.

Atherosclerosis is the primary underlying process in the pathogenesis of acute myocardial infarction.[15] The association of all-cause and cardiac mortality with pericoronary adipose tissue attenuation has been most convincingly demonstrated for the right coronary artery where the volume of fat is greatest.[3, 4] With the abdominal aorta being a much larger vessel and associated with even greater volumes of perivascular fat, we wanted to assess whether periaortic adipose tissue attenuation would associate and correlate with the presence and magnitude of abdominal aortic aneurysm disease. Histological analysis of aneurysm tissue has shown the involvement of various inflammatory cell types, such as macrophages and lymphocytes, and a role of inflammation in the pathogenesis of aneurysm disease has been proposed.[16, 17] Furthermore, murine models have shown a link between

vascular inflammation and aneurysm formation and perivascular adipose tissue through the angiotensin II type 1a receptor.[18] It is therefore plausible that periaortic adipose tissue attenuation could be linked to, or be a marker of, abdominal aortic aneurysm disease activity. However, we identified no such association in patients with established stable aneurysm disease, with periaortic adipose tissue attenuation being similar between not only patients and control subjects, but importantly, also between normal regions of aorta and regions affected by aneurysmal disease within the same individual.

In a retrospective study, Dias-Neto and colleagues have previously found that when compared to patients with occlusive aortoiliac disease and without aortic disease, the presence of an abdominal aortic aneurysm was an independent predictor of higher perivascular adipose tissue attenuation around the aneurysm sac and the healthy neck.[8] They did however employ a rather different methodology to the pericoronary adipose tissue methodology that we employed here. First, they generated a density value taking the summation of all the attenuation values from a range of -107 to -45 HU and divided them by the area of the region of interest. Second, they included a large 10-mm distance from the outer wall of the aorta, which will likely include non-adipose tissue structures. We have here used previously validated semi-automated software[12] to obtain mean attenuation values at a 2-mm distance from the aortic wall. We also considered the neck and body of the aorta together and compared a more remote region of healthy aorta away from the aneurysm sac and disease.

In pericoronary adipose tissue, Oikonomou and colleagues used a cut-off of above -70 HU as their at-risk population threshold.[3] Moreover, the overall differences in

pericoronary adipose tissue between those with and without future coronary events were small (~4-6 HU). Here, we have observed very large differences in periaortic adipose tissue attenuation between those with asymptomatic and symptomatic disease, approximately 25 HU. This dramatic difference is striking and importantly was observed in both the region of the aneurysm as well as the more distant non-aneurysmal unruptured aorta. This suggests that this is not a regional effect at the segment of active disease and rupture but a more global aortic phenomenon. We wondered if this was a systemic effect that would affect all adipose tissue throughout the body and therefore explored adipose tissue attenuation in both subcutaneous and visceral fat. Here, we noted only a slight difference (approximately 8 HU) in the subcutaneous fat tissue of symptomatic patients with ruptured aneurysms. This change may perhaps reflect fluid shifts in the extracellular space consequent on systemic shock or intravenous fluid resuscitation leading to non-specific increases in adipose tissue attenuation. However, this effect was not observed in visceral fat tissue.

Whilst extravasation of blood in acute rupture may account for the difference in periaortic adipose tissue attenuation, this difference was also observed in symptomatic patients with unruptured aneurysms. The differences in subcutaneous tissue attenuation between the groups are however modest, and could plausibly account for only approximately a third of the overall difference. We believe that the observed changes are most likely to represent the consequences of acute rupture rather than a true reflection of adventitial disease activity because of the lack of a regional effect with generalised changes observed in non-aneurysmal as well as aneurysmal segments of the aorta. Speculatively, this could be related to the increased fibrotic changes and presence of increased adipocyte clusters in the

adventitia[19], or perhaps to changes in tissue density in response to adventitial neurovascular reflexes as part of the physiological response to aortic rupture and systemic hypotension.[20]

Our study had some limitations. First, data for symptomatic patients were collected in a retrospective fashion from image archives and only minimal clinical data could be collected. Despite this, symptomatic patients were observed to have higher cardiovascular risk profiles than the other two groups, and this may account for some of the observed results. Second, asymptomatic patients and control subjects were imaged on a research scanner with a dedicated imaging protocol, whereas symptomatic patients were scanned on a variety of clinical scanners in different vascular centres around Scotland. Whilst we have corrected the data for tube potential, imaging protocols were not uniform. Despite this, we have demonstrated that visceral fat attenuation between scans did not vary between the three study groups. Third, whilst the reproducibility for periaortic fat analysis has not been established, quantitative adipose tissue attenuation has previously shown excellent repeatability in much smaller structures, such as the pericoronary adipose tissue.[21] Fourth, this study provides a single snapshot assessment of periaortic adipose tissue but does not provide an assessment over time; thus, we cannot comment on its association with aneurysm development or progression.

In conclusion, periaortic adipose tissue CT attenuation was similar between asymptomatic individuals with abdominal aortic aneurysm disease and controls without aneurysms. Symptomatic patients with aneurysms had higher adipose tissue attenuation when compared with the other two groups. However, this difference was not localized to the abdominal aortic aneurysm and is likely to have been an acute

non-specific systemic effect. This suggests that periaortic adipose tissue attenuation may be of limited clinical value as a biomarker or risk stratification tool in abdominal aortic aneurysm disease. Further exploration of the implications and mechanisms underlying the generalized changes in adipose tissue attenuation in patients with symptomatic aneurysm disease are warranted.

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Tables

Table 1.

Patient characteristics

	Patient groups			P-values		
Characteristic	**Controls**	**Asymptomatic (Unruptured) Abdominal Aortic Aneurysm**	**Symptomatic Abdominal Aortic Aneurysm (Non-rupture & Rupture)**	**Control versus Asymptomatic[2]**	**Control versus Symptomatic[2]**	**Asymptomatic versus Symptomatic[2]**
	N = 18[1]	N = 40[1]	N = 30[1]			
Age (years)	66 ± 2	72 ± 6	74 ± 9	<0.001	<0.001	0.30
Sex				0.30	0.14	0.70
Male	18 (100%)	35 (88%)	25 (83%)			
Female	0 (0%)	5 (12%)	5 (17%)			
Aortic diameter (mm)	20 (19, 20)	49 (44, 55)	74 (62, 89)	<0.001	<0.001	<0.001
Medical History						
Hypertension	5 (28%)	23 (57%)	22 (73%)	0.04	0.002	0.20
Cerebrovascular accident	0 (0%)	4 (10%)	4 (13%)	0.30	0.30	0.70
Ischemic heart disease	1 (5.6%)	7 (18%)	11 (37%)	0.40	0.02	0.07
Peripheral arterial disease	0 (0%)	7 (18%)	2 (6.7%)	0.09	0.50	0.30
Diabetes	1 (5.6%)	6 (15%)	2 (6.7%)	0.40	>0.90	0.50

	Patient groups			P-values		
Characteristic	**Controls**	**Asymptomatic (Unruptured) Abdominal Aortic Aneurysm**	**Symptomatic Abdominal Aortic Aneurysm (Non-rupture & Rupture)**	**Control versus Asymptomatic[2]**	**Control versus Symptomatic[2]**	**Asymptomatic versus Symptomatic[2]**
	N = 18[1]	N = 40[1]	N = 30[1]			
Hypercholesterolemia	6 (33%)	32 (80%)	21 (70%)	<0.001	0.01	0.30
Current or ex-smoker	3 (21%)	12 (31%)	21 (81%)	0.70	<0.001	<0.001
Medications						
Antiplatelet agents	2 (11%)	26 (65%)	19 (63%)	<0.001	<0.001	0.90
Anticoagulant	0 (0%)	2 (5.0%)	3 (10%)	>0.90	0.30	0.60
Statins	6 (33%)	33 (82%)	21 (70%)	<0.001	0.01	0.20
Beta-blocker	1 (5.6%)	8 (20%)	8 (27%)	0.20	0.13	0.50
Angiotensin Converting Enzyme Inhibitors or Angiotensin Receptor Blockers	3 (17%)	18 (45%)	14 (47%)	0.04	0.04	0.90

Note.—

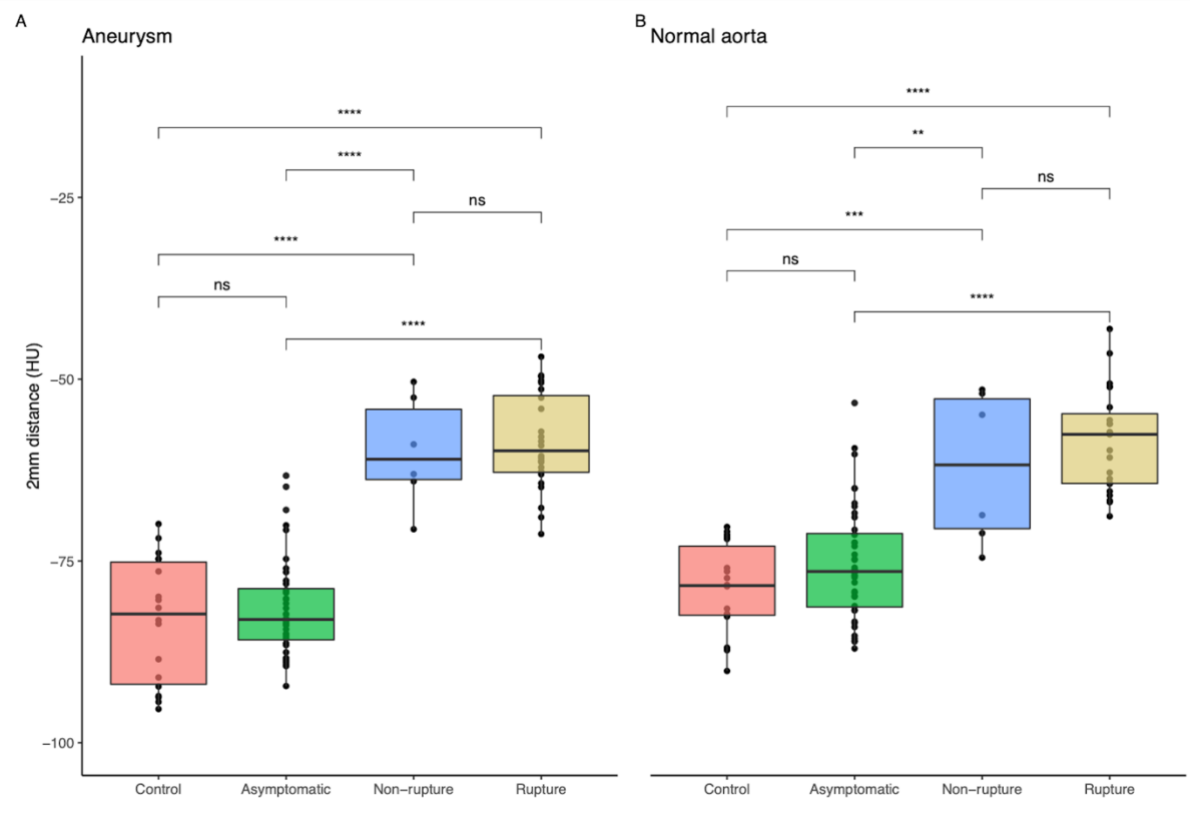
[1]Mean ± SD; n (%); Median (IQR)

[2]Wilcoxon rank sum test; Fisher's exact test; Pearson's Chi-squared test

Figures

Figure 1.

Periaortic adipose tissue attenuation within 2-mm distance from vessel wall.



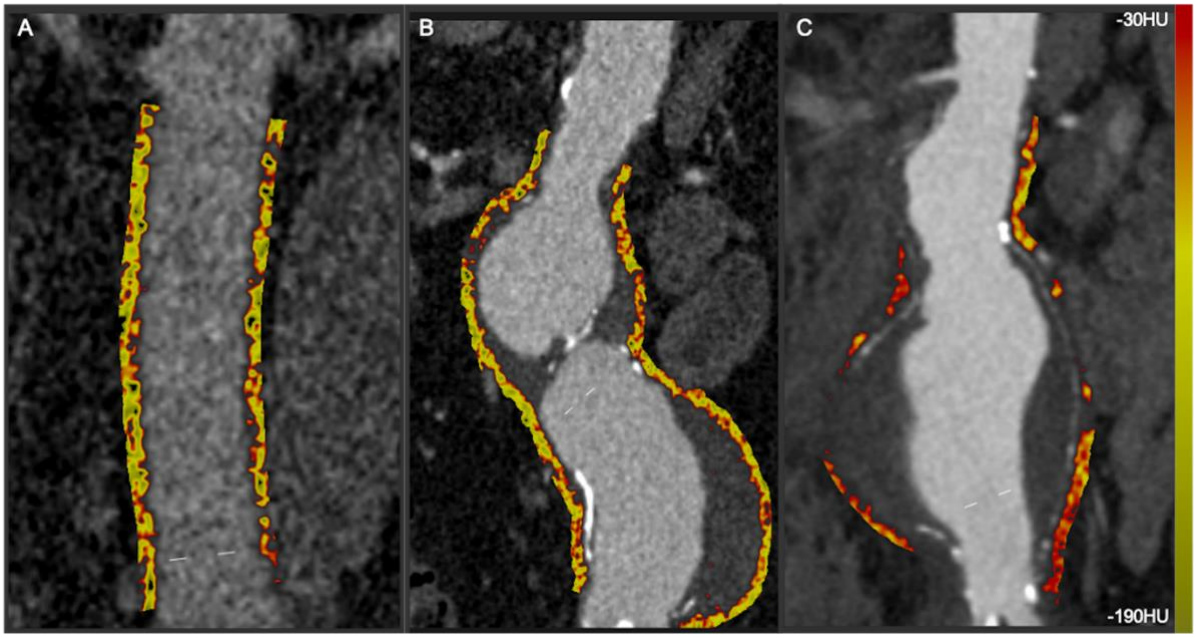
Periaortic adipose tissue attenuation in (A) the aneurysmal and (B) non-aneurysmal segments of the abdominal aorta in controls[†], and patients with asymptomatic and symptomatic aneurysms. Patient groups with symptomatic aneurysms were sub-divided into those with and without rupture.

Each black point represents individual attenuation values. The upper and lower edges of the box represent the interquartile range and the middle horizontal line represents the median value in each group.

HU, Hounsfield units; **** = P<0.0001; *** = P<0.001 ns = not significant. [†]Comparable but non-aneurysmal segments in controls were used.

Figure 2.

Periaortic adipose tissue in representative patients.

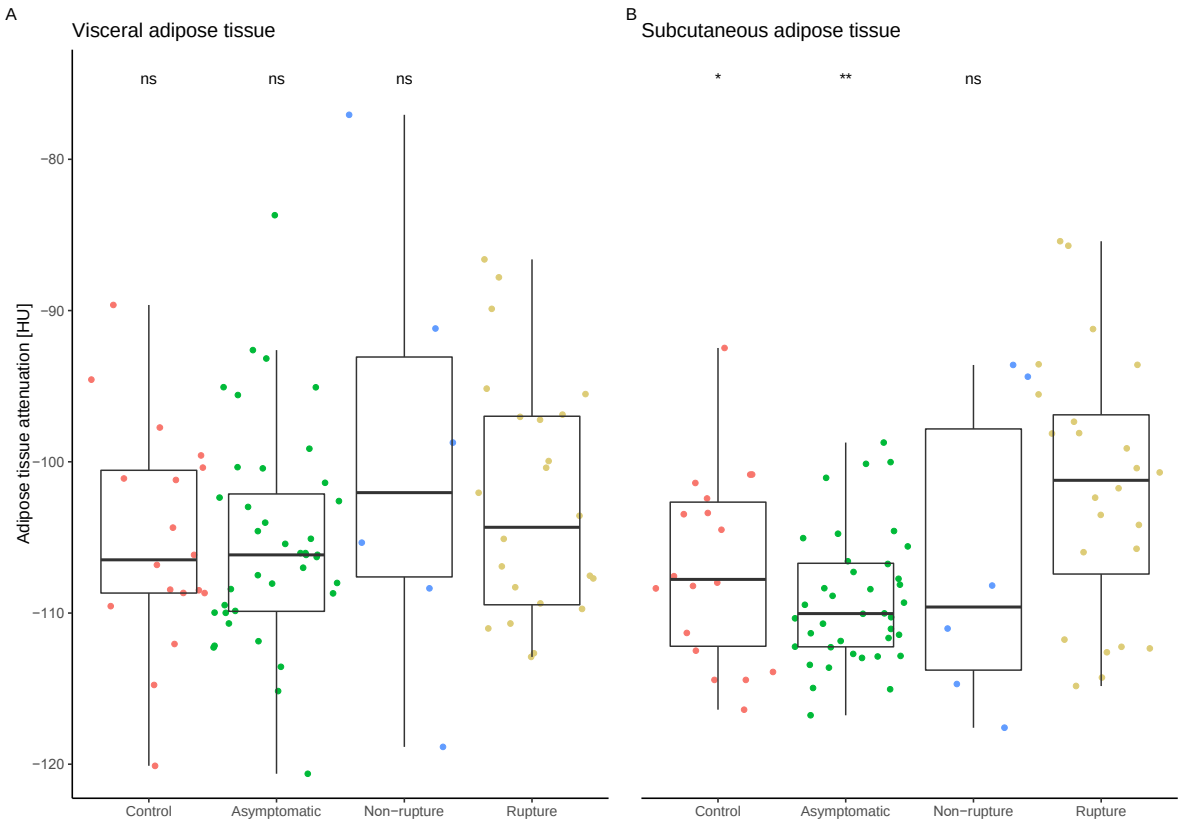


Assessment of periaortic adipose tissue attenuation in sagittal CT images of the aorta in (A) a 66-year-old male individual from the control group (mean -93.57±41 HU), (B) a 79-year-old male patient with an asymptomatic aneurysm (mean -89.45±36 HU) and (C) a 71-year-old female patient with aneurysm rupture (mean -59.12±21 HU).

HU, Hounsfield units.

Figure 3.

Visceral and subcutaneous adipose tissue attenuation.



Sub-group analyses of (A) visceral and (B) subcutaneous adipose tissue attenuation in controls, patients with asymptomatic aneurysms and patients with symptomatic aneurysms sub-divided into those with and without rupture.

Each point represents individual attenuation values, displayed at different locations along the X-axis to prevent over plotting. The upper and lower edges of the box represent the interquartile range and the middle horizontal line represents the median value in each group.

HU, Hounsfield units; * = P < 0.05; ** = P<0.01; ns = not significant.