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RESEARCH ARTICLE

Association between epicardial adipose tissue and cardiac dysfunction in subjects with severe obesity

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Aim Epicardial adipose tissue (EAT) plays a role in obesity-related heart failure with preserved ejection fraction. However, the association of EAT thickness with the development of cardiac dysfunction in subjects with severe obesity without known cardiovascular disease is unclear. The aim of this study was to determine the association between EAT thickness and cardiac dysfunction and describe the potential value of EAT as an early marker of cardiac dysfunction.

Methods and results Subjects with body mass index ≥35 kg/m^2 aged 35 to 65 years, who were referred for bariatric surgery, without suspicion of or known cardiac disease, were enrolled. Conventional transthoracic echocardiography and strain analyses were performed. A total of 186 subjects were divided into tertiles based on EAT thickness, of whom 62 were in EAT-1 (EAT <3.8 mm), 63 in EAT-2 (EAT 3.8–5.4 mm), and 61 in EAT-3 (EAT >5.4 mm). Parameters of systolic and diastolic function were comparable between tertiles. Patients in EAT-3 had the lowest global longitudinal strain (GLS) and left atrial contractile strain (LASct). Linear regression showed that a one-unit increase in EAT thickness (mm) was independently associated with a decrease in GLS (%) (β coefficient −0.404, $p = 0.002$), and a decrease in LASct (%) (β coefficient −0.544, $p = 0.027$). Furthermore, EAT-3 independently predicted cardiac dysfunction as defined by a GLS <18% (odds ratio 2.8, $p = 0.013$) and LASct <14% (odds ratio 2.5, $p = 0.045$).

Conclusions Increased EAT thickness in subjects with obesity without known cardiac disease was independently associated with subclinical cardiac dysfunction. Our findings suggest that EAT might play a role in the early stages of cardiac dysfunction in obesity before this may progress to overt clinical disease.

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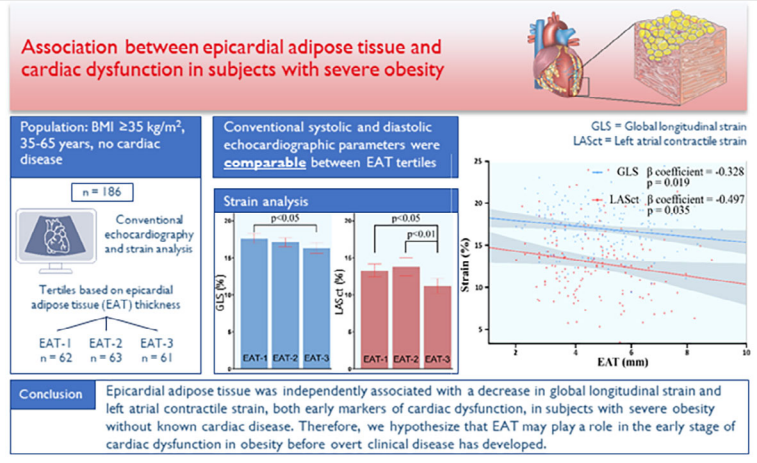
Graphical Abstract



Association between epicardial adipose tissue (EAT) and cardiac dysfunction in severe obesity. BMI, body mass index; GLS, global longitudinal strain; LASct, left atrial contractile strain.

Keywords Cardiac dysfunction • Echocardiography • Epicardial adipose tissue • Obesity • Strain analyses

Introduction

Obesity is a global pandemic. It affects over 650 million adults, and, if the current trend persists, it is anticipated that up to 20% of the world's adult population (1.2 billion adults) will have obesity by 2030.[1–3] Moreover, obesity is a major risk factor for heart failure with preserved ejection fraction (HFpEF).[4,5] A one-unit increase in body mass index (BMI) is associated with a 34% increased risk of future HFpEF, and more than 80% of HFpEF patients are either overweight or obese.[6,7] Recently, there has been a growing interest in the role of epicardial adipose tissue (EAT) in the progression and development of obesity-related HFpEF.[8,9] Obesity facilitates a state of inflammation that can lead to the expansion of EAT, which in turn becomes a source of pro-inflammatory and pro-fibrotic markers. In addition, EAT is unique in its anatomy and unobstructed proximity to the heart, as a fascia does not separate it from the myocardium.[10] Subsequently, secreted factors produced in the EAT can directly infiltrate the myocardium and may cause cardiac remodelling and dysfunction.[8]

Although the role of EAT in patients with obesity and prevalent HFpEF has already been investigated,[11–13] it is unknown whether EAT is related to subclinical cardiac dysfunction in apparently healthy subjects. Our study group has previously shown that global longitudinal strain (GLS) and left atrial (LA) strain are valuable parameters of subclinical cardiac dysfunction in subjects with obesity.[14–16] The aim of the current study was to determine the relationship between EAT and GLS and LA strain in individuals with obesity without known cardiac disease in order to improve the understanding of the relationship between EAT and cardiac dysfunction. Moreover, we sought to address the potential value of EAT as a diagnostic parameter of subclinical cardiac dysfunction.

Methods

Study design

For this study, the CARDIOBESE (The CARdiac Dysfunction In Obesity: Early Signs Evaluation) and AF OBESE (Atrial Fibrillation detection in OBESity using E-health) databases were used.

The CARDIOBESE and AF OBESE studies are prospective studies in which 192 subjects with obesity were included who were referred for bariatric surgery at The Franciscus Gasthuis and Vlietland and Maasstad Hospital, both in Rotterdam, The Netherlands.[17] Subjects aged 35 to 65 years with a BMI ≥35 kg/m^2 were enrolled between October 2016 and January 2022. Patients with a suspicion of or known cardiac disease were excluded, such as coronary artery disease, heart failure, valvular heart disease, cardiomyopathies, congenital heart disease, and arrhythmias. The study was approved by the medical ethics committee, and conducted in accordance with the Declaration of Helsinki. All patients signed informed consent for the study.

Echocardiography

Conventional and speckle tracking echocardiography was performed in all participants. Two-dimensional greyscale harmonic images were obtained in the left lateral decubitus position using a commercially available ultrasound system (EPIQ 7, Philips, The Netherlands) equipped with a broadband (1–5 MHz) X5-1 transducer. All acquisitions and

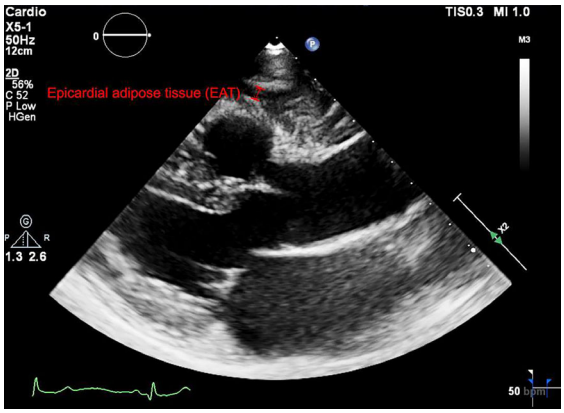


Figure 1 Echocardiographic measurement of epicardial adipose tissue from the parasternal long-axis view.

measurements were performed according to the current guidelines.[18,19] EAT was identified as the relatively echo-free space between the myocardial outer wall and the pericardial visceral layer. EAT thickness was measured perpendicularly to the free wall of the right ventricle using parasternal long-axis and short-axis views in end-systole, according to previous recommendations[20] (*Figure 1*). Moreover, the thickness was measured twice on parasternal long-axis views and twice on short-axis views. The measurements were averaged for analysis. A standard upper-limit reference value for epicardial fat thickness has yet to be established. EAT thickness was measured retrospectively by a single operator (J.F.C.) who was trained and experienced in echocardiography. Intra-observer variability was assessed by re-measuring 20 echocardiograms and calculating the intraclass correlation coefficient. A second operator (Y.S.A.) measured EAT thickness in 20 echocardiograms in order to determine the inter-observer variability. The intra-observer correlation coefficient was 0.91, and the inter-observer correlation coefficient was 0.85.

Global longitudinal strain and LA strain were measured with speckle tracking and analysed offline with dedicated software (2D Cardiac Performance Analysis version 4.5; TomTec Imaging Systems, Unterschleissheim, Germany). GLS was assessed using three consecutive cardiac cycles from all apical views (4-, 2-, and 3-chamber). In end-diastole, the endocardial and epicardial borders were included in the region of interest using automated border tracking and a manual 'point and click' method. If the tracking was not accurate, it was fine-tuned manually.[21] The peak regional longitudinal strain was measured in 16 myocardial regions, and a weighted mean was calculated to obtain GLS.[18] LA strain was preferably assessed using the apical 4-chamber view. However, if the image quality of the 4-chamber view was inadequate, the 2-chamber view was utilized as an alternative.[22] The frame corresponding to end-diastole was chosen by aligning with the opening and closure of the mitral valve, supported by electrocardiographic data utilizing R–R intervals. For automated border detection, reference landmarks were positioned at the mitral annuli and the posterior wall of the LA on the atrial endocardium. Subsequent to the automated border tracking analysis, a thorough visual assessment of the tracking quality was conducted. In instances where the tracking quality did not meet expectations, minor manual adjustments were carried out. Images of insufficient quality to perform GLS and LA strain analyses were excluded. LA function was classified based on the three phases of the LA cycle[23]: LA reservoir strain (LASr), LA conduit strain (LAScd), and LA contractile strain (LASct). Focus was on LASct since LASr is mostly associated with increased left ventricular filling pressures in individuals with decreased left ventricular ejection fraction,[24] and our population consisted of patients with no history of cardiac disease. Strain values were presented as absolute values for better understanding and interpretation.

Statistical analysis

Normally distributed variables are presented as means and standard deviation, non-normally distributed variables as medians and interquartile range, and categorical variables as percentages and frequencies. The normality of the variables was assessed through two approaches: visual examination of histograms and the Shapiro–Wilk test. The study population was divided into three subgroups based on EAT tertiles. Between-group differences were compared using the one-way analysis of variance, Kruskal–Wallis test, or chi-square test, as appropriate. To determine whether EAT was associated with the dependent variable GLS and LASct, independent of potential confounders, univariable and multivariable linear regression analyses were performed. The assumption of linearity was assessed through two approaches: quartiles analysis and the inclusion of a quadratic term. Multivariable analyses were adjusted for age, sex, BMI, hypertension, diabetes mellitus, obstructive sleep apnoea syndrome, oral antidiabetics (metformin/sulfonylureas), insulin, statin, angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, and beta-blocker, because of the potential relationship with the outcome variable. Binary logistic regression was used to calculate univariable and multivariable-adjusted odds ratios (OR) for the association of high EAT thickness values, defined as the highest EAT tertile (>5.4 mm), with abnormal GLS, defined as GLS <18%[15,18,25] and/or abnormal LASct, defined as LASct <14%.[24] A two-tailed *p*-value of <0.05 was considered statistically significant. Statistical analyses were performed with SPSS version 28.0 (SPSS Inc., Chicago, IL, USA).

Results

Clinical and echocardiographic characteristics of the study population

Echocardiographic image quality was insufficient in six subjects to assess EAT reliably, leaving 186 subjects for the current analysis. The mean age of the study population was 52.2 ± 8.1 years, 75.3% were female, and the mean BMI was 42.3 ± 4.5 kg/m^2. The study population was divided into three subgroups based on EAT tertiles: 62 subjects with EAT <3.8 mm (EAT-1), 63 subjects with EAT 3.8–5.4 mm (EAT-2), and 61 subjects with EAT >5.4 mm (EAT-3). There was no significant difference in clinical characteristics, including BMI and diabetes mellitus, between the subjects in these EAT tertiles. Details are presented in *Table 1*. An overview of the echocardiographic characteristics of the EAT groups is shown in *Table 2*. There were no differences in left ventricular ejection fraction and conventional diastolic parameters between groups.

Association between epicardial adipose tissue and strain analysis

Image quality was insufficient in 18 subjects for reliable assessment of GLS and in 39 subjects for LA strain. There was a significant

Table 1 Clinical characteristics of the study population

	Total ($n = 186$)	EAT-1 <3.8 mm ($n = 62$)	EAT-2 3.8–5.4 mm ($n = 63$)	EAT-3 >5.4 mm ($n = 61$)	*p*-value*
Age, years	52.2 ± 8.1	51.3 ± 7.6	51.4 ± 7.0	54.0 ± 9.3	0.115
Female sex, *n* (%)	140 (75.3)	42 (67.7)	51 (81.0)	47 (77.0)	0.214
Weight, kg	122.0 ± 19.0	123.8 ± 18.8	120.6 ± 18.6	121.5 ± 19.9	0.638
Height, m	1.69 ± 0.09	1.71 ± 0.10	1.69 ± 0.10	1.68 ± 0.09	0.177
BMI, kg/m^2	42.3 ± 4.5	42.0 ± 4.1	42.3 ± 4.7	42.7 ± 4.6	0.737
Systolic BP, mmHg	146 ± 22	144 ± 21	147 ± 22	148 ± 22	0.472
Diastolic BP, mmHg	80 ± 11	80 ± 12	80 ± 10	79 ± 11	0.817
Heart rate, bpm	79.5 ± 12.3	77.2 ± 11.7	81.3 ± 12.9	79.9 ± 12.0	0.174
BNP, pmol/L	4.0 [3.0–8.0]	4.0 [1.5–8.3]	4.0 [1.5–7.5]	4.0 [3.0–8.0]	0.651
Diabetes mellitus, *n* (%)	38 (20.4)	12 (19.4)	13 (20.6)	13 (21.3)	0.963
Hypertension, *n* (%)	72 (38.7)	22 (35.5)	26 (41.3)	24 (39.3)	0.796
OSAS, *n* (%)	37 (19.9)	12 (19.4)	14 (22.2)	11 (18.0)	0.836
Beta-blocker, *n* (%)	20 (10.8)	6 (9.7)	8 (12.7)	6 (9.8)	0.828
ACEi/ARB, *n* (%)	50 (26.9)	17 (27.4)	14 (22.2)	19 (31.1)	0.530
Diuretics, *n* (%)	39 (21.0)	12 (19.4)	13 (20.6)	14 (23.0)	0.884
Statin, *n* (%)	34 (18.3)	12 (19.4)	8 (12.7)	14 (23.0)	0.324

Normally distributed data are presented as mean ± standard deviation; non-normally distributed data are presented as median [interquartile range]; categorical data are presented as *n* (%).

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; BNP, B-type natriuretic peptide; BP, blood pressure; EAT-1, low tertile epicardial adipose tissue; EAT-2, middle tertile epicardial adipose tissue; EAT-3, high tertile epicardial adipose tissue; OSAS, obstructive sleep apnoea syndrome.

*p-value represents a comparison between EAT-1, EAT-2, and EAT-3.

Table 2 Echocardiographic parameters in the study population

	Total ($n = 186$)	EAT-1 <3.8 mm ($n = 62$)	EAT-2 3.8–5.4 mm ($n = 63$)	EAT-3 >5.4 mm ($n = 61$)	*p*-value
EAT, mm	4.8 ± 1.6	3.2 ± 0.4	4.7 ± 0.4	6.6 ± 1.0	<0.001
LVM, g	190.5 ± 64.5	188.4 ± 69.5	187.4 ± 47.4	196.0 ± 74.7	0.730
LVMI, g/m^2	80.6 ± 22.9	78.6 ± 25.5	80.5 ± 16.9	82.9 ± 25.6	0.591
LVEF, %	56.9 ± 6.1	57.3 ± 6.4	56.3 ± 5.8	57.2 ± 6.2	0.653
E/A ratio	1.0 ± 0.2	1.0 ± 0.2	1.0 ± 0.2	0.9 ± 0.2	0.118
Septal e' velocity, cm/s	7.5 ± 1.9	7.5 ± 2.2	7.7 ± 1.8	7.2 ± 1.9	0.468
E/e' ratio	9.5 ± 2.8	9.3 ± 2.3	9.7 ± 3.0	9.3 ± 3.1	0.626
TR velocity, cm/s	130.5 ± 58.7	134.5 ± 61.8	128.8 ± 64.6	128.5 ± 49.1	0.850
LV diastolic function, *n* (%)					
Normal	145 (92)	46 (88)	53 (96)	46 (90)	0.605
Indeterminate	11 (7)	5 (10)	2 (4)	4 (8)	
Dysfunction	2 (1)	1 (2)	0 (0)	1 (2)	
GLS, %	17.0 ± 2.7	17.6 ± 2.7	17.1 ± 2.4	16.3 ± 2.9*	0.041
LAV, ml	51.7 ± 16.0	54.4 ± 16.2	53.5 ± 17.4	46.8 ± 13.1	0.546
LAVI, ml/m^2	22.2 ± 6.5	23.1 ± 6.5	23.0 ± 6.8	20.5 ± 5.7	0.320
LASr, %	30.3 ± 8.6	31.3 ± 7.6	31.6 ± 9.6	27.7 ± 7.8	0.048
LAScd, %	17.4 ± 7.1	18.0 ± 7.2	17.8 ± 8.0	16.4 ± 5.7	0.516
LASct, %	12.9 ± 4.3	13.3 ± 3.4	13.8 ± 4.8	11.2 ± 4.0**	0.007

Normally distributed data are presented as mean ± standard deviation and categorical data are presented as *n* (%).

e', peak early diastolic mitral annular displacement velocity; E/A ratio, peak early mitral inflow velocity/peak late mitral inflow velocity ratio; EAT, epicardial adipose tissue; EAT-1, low tertile epicardial adipose tissue; EAT-2, middle tertile epicardial adipose tissue; EAT-3, high tertile epicardial adipose tissue; GLS, global longitudinal strain; LAScd, left atrial conduit strain; LASct, left atrial contractile strain; LASr, left atrial reservoir strain; LAV, left atrial volume; LAVI, left atrial volume index; LV, left ventricular; LVEF, left ventricular ejection fraction; LVM, left ventricular mass; LVMI, left ventricular mass index; TR, tricuspid regurgitation.

*$p < 0.05$ vs. EAT-1.

**$p < 0.05$ vs. EAT-1 and EAT-2.

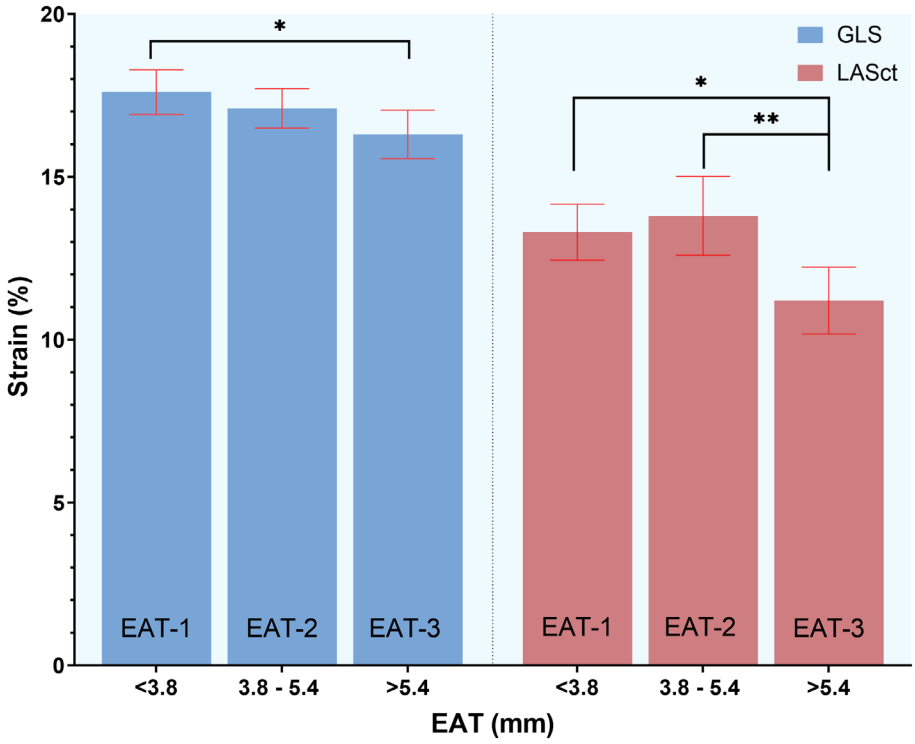


Figure 2 Bar graph comparing (*A*) global longitudinal strain (GLS) and (*B*) left atrial contractile strain (LASct) in patients divided into tertiles based on epicardial adipose tissue (EAT). *$p < 0.05$, and **$p < 0.01$.

difference in GLS between the EAT groups, where patients with the highest EAT had the lowest GLS (*Table 2* and *Figure 2*). A similar result was observed for LA function: patients with the highest EAT had the lowest LASr and LASct. Linear regression analyses showed that a higher EAT thickness value (mm) was significantly associated with a lower GLS value (%) (unstandardized β coefficient −0.328, 95% confidence interval [CI] −0.600, −0.055, $p = 0.019$) and a lower LASct value (%) (unstandardized β coefficient – 0.497 [95% CI −0.959, −0.034], $p = 0.035$) (*Table 3* and *Figure 3*). These associations remained significant in multivariable linear regression (GLS, unstandardized β coefficient −0.404 [95% CI −0.625, −0.156], $p = 0.002$; LASct, unstandardized β coefficient −0.544 [95% CI −1.023, −0.064], $p = 0.027$). Furthermore, binary logistic regression showed that a high EAT thickness value, defined as the highest EAT tertile (>5.4 mm), was significantly associated with abnormal GLS and LASct (*Table 4*). These associations remained statistically significant after multivariable adjustment (GLS: OR 2.8 [95% CI 1.2−6.3], $p = 0.013$, LASct: OR 2.5 [95% CI 1.1−5.9], $p = 0.045$).

Table 3 Association between epicardial adipose tissue thickness (mm) and cardiac function as measured with strain analyses

Linear regression model	*n*	Unstandardized β coefficient [95% CI]	*p*-value
GLS, %			
Univariable	168	−0.328 [−0.600, −0.055]	0.019
Multivariable		−0.404 [−0.625, −0.156]	0.002
LASct, %			
Univariable	147	−0.497 [−0.959, −0.034]	0.035
Multivariable		−0.544 [−1.023, −0.064]	0.027

Multivariable model adjusted for age, gender, body mass index, hypertension, diabetes mellitus, obstructive sleep apnoea syndrome, oral antidiabetics (metformin/sulfonylureas), insulin, statin, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker, and beta-blocker.
CI, confidence interval; GLS, global longitudinal strain; LASct, left atrial contractile strain.

Discussion

In the present study, we examined the association between EAT thickness and cardiac dysfunction and its potential value as an early marker of cardiac dysfunction in subjects with obesity without known cardiac disease. Higher EAT thickness values were independently associated with lower values of both GLS and LASct (*Graphical Abstract*), which are recognized as early markers of cardiac dysfunction.[14–16] Furthermore, a high EAT thickness value (>5.4 mm) independently predicted cardiac dysfunction as defined by a GLS <18% or LASct <14%. These findings suggest that EAT may already play a role in the early stages of cardiac dysfunction in subjects with obesity. Furthermore, considering that measurement of EAT is relatively fast and easy, it may have value as

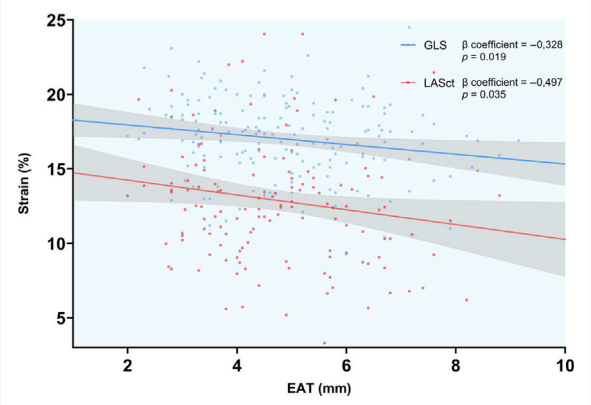


Figure 3 Regression plot of the relationship between epicardial adipose tissue (EAT) thickness and global longitudinal strain (GLS) and left atrial contractile strain (LASct).

an early marker for screening purposes of cardiac dysfunction in the large group of individuals with obesity without known cardiac disease.

The role of epicardial adipose tissue in obesity and cardiac function

In recent years, EAT has gained increasing attention for its potential role in the development of HFpEF, particularly in individuals with obesity. EAT has a unique property as it shares an unobstructed microcirculation with the underlying myocardium[10] and can therefore affect cardiac function via several mechanisms.[8] Obesity promotes chronic inflammation, which may be a key driver of the development and progression of HFpEF and its associated comorbidities.[26] In the context of EAT, chronic inflammation can lead to deranged adipogenesis within this adipose depot. Subsequently, EAT produces pro-inflammatory and pro-fibrotic adipokines that can cause atrial and ventricular fibrosis leading to cardiac dysfunction.[8,9] Furthermore, EAT may also affect cardiac function through the mechanical effects of a large, fibrotic fat pad.[8]

In previous studies, it reported that excess EAT is associated with adverse prognosis in patients with HFpEF and worse cardiopulmonary performance in patients with normal heart function and type 2 diabetes mellitus.[13,27,28] Furthermore, increased EAT in HFpEF patients has been related to worse cardiac function compared to HFpEF patients without increased EAT.[11,12,29,30] Also, in a recent study, Jin *et al.*[29] concluded that increased EAT was associated with decreased GLS and LASct. However, in all these studies, HFpEF populations were investigated. Considering that obesity is related to EAT and that obesity is one of the most important risk factors for HFpEF,[4,5] it may be important to improve the understanding of the role of EAT in the early development of cardiac dysfunction in obesity as well. Our study is the first to investigate the relation between EAT and cardiac dysfunction in individuals with obesity without known cardiac disease and offers insight into the relationship between EAT and cardiac dysfunction. Even though the EAT groups did not significantly differ in clinical characteristics and conventional echocardiographic parameters, higher EAT thickness values were independently related to lower GLS and LASct values.

Clinical relevance of epicardial adipose tissue in individuals with obesity

Considering the very large group of individuals with obesity, an easy, fast, and non-invasive marker of cardiac dysfunction may have value in risk stratification and subsequent decisions on the intensity of follow-up or further analysis in these subjects. In our previous studies, we have shown that both GLS and LASct could be markers of early cardiac dysfunction in individuals with obesity.[14,15] Although speckle-tracking software is broadly available in ultrasound machines from all well-known vendors, strain assessment remains a relatively advanced echocardiographic tool. Moreover, reliable strain analyses require expertise and good image quality. Therefore, in screening large groups of subjects with obesity, a simple measurement with conventional echocardiography would be preferable to identify subjects at high risk of developing cardiac dysfunction. EAT thickness may be such a parameter, using the standard parasternal long- and short-axis views. By using it this way, a finding of a high EAT thickness value may warrant further investigation with more advanced echocardiographic parameters.

It should, however, be noted that although echocardiography has several advantages over other non-invasive cardiac imaging

Table 4 Logistic regression model for the association between high epicardial adipose tissue thickness values and cardiac function as measured with strain analyses

	Abnormal GLS (<18%)		Abnormal LASct (<14%)	
	OR [95% CI]	*p*-value	OR [95% CI]	*p*-value
High EAT (>5.4 mm)				
Univariable	2.169 [1.065–4.420]	0.033	2.078 [0.925–4.668]	0.076
Multivariable	2.813 [1.248–6.344]	0.013	2.452 [1.018–5.903]	0.045

Multivariable model adjusted for age, gender, body mass index, hypertension, diabetes mellitus, obstructive sleep apnoea syndrome, oral antidiabetics (metformin/sulfonylureas), insulin, statin, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker, and beta-blocker.
CI, confidence interval; EAT, epicardial adipose tissue; GLS, global longitudinal strain; LASct, left atrial contractile strain; OR, odds ratio.

techniques, such as the relatively low cost, high accessibility, and good reproducibility, it also has several limitations. As shown in *Figure 3*, there is a relatively large scatter when relating EAT to parameters of cardiac dysfunction, which may at least be partly explained by measurement errors, potentially hampering clinical use of EAT. In contrast to computed tomography (CT) scan and magnetic resonance imaging (MRI), considered the gold standard for EAT quantification, echocardiography only visualizes the part of the epicardial fat located at the free wall of the right ventricle. However, while EAT is unevenly distributed over the heart, approximately 75% of total EAT is estimated to be located over the right ventricle.[31] Furthermore, consistent results for EAT assessment using both MRI and echocardiography were shown in two recent studies,[32,33] although conflicting results have been reported as well.[34] In addition, CT and MRI require relatively high costs, expertise, long acquisition times for MRI, and radiation exposure for CT. Therefore, compared to CT and MRI, echocardiography may be a more useful screening tool to identify individuals with excess EAT.

Study limitations

The study has several limitations. First, due to the cross-sectional design, we could not conclude whether there is a direct causal relationship between EAT and cardiac dysfunction. However, we did perform a multivariable analysis to correct for known potential confounders. Second, our study population consisted of individuals screened for bariatric surgery. Since around 80% of patients who undergo bariatric surgery are female,[35] women were overrepresented in our study, which may have biased the results. Third, it is important to acknowledge that our study had a relatively small sample size, and a larger number of participants may have further strengthened the robustness of our research findings. Nonetheless, to the best of our knowledge, it is noteworthy that our study is the largest one to date to examine the association between EAT and echocardiographic parameters of cardiac dysfunction within the group of individuals with severe obesity without known cardiac disease. While subdividing our study population into more than three groups might have provided increased granularity of the data, the sample size in each group would have been reduced. Therefore, we decided to use tertiles, whereas quartiles or even quintiles would also have been an option. Finally, our study included subjects only with a BMI $\geq$35 kg/m^2. It is undetermined whether our results would also apply to obese individuals with a BMI between 30 and 35 kg/m^2.

Conclusions

Epicardial adipose tissue is negatively associated with both GLS and LASct, which are recognized as early markers of cardiac dysfunction. Therefore, EAT may play a role in the early stage of cardiac dysfunction in obesity before overt clinical disease has developed. The simple measurement of EAT thickness may serve as a marker of subclinical cardiac disease in individuals with obesity.

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Conflict of interest: none declared.

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