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Citation

Hout, M. J. P. van, Dekkers, I. A., Westenberg, J. J. M., Schalijs, M. J., Scholte, A. J. H. A., & Lamb, H. J. (2020). The impact of visceral and general obesity on vascular and left ventricular function and geometry: a cross-sectional magnetic resonance imaging study of the UK Biobank. *European Heart Journal - Cardiovascular Imaging*, 21(3), 273-281.
doi:10.1093/ehjci/jez279

Version: Publisher's Version

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Downloaded from: <https://hdl.handle.net/1887/3184221>

Note: To cite this publication please use the final published version (if applicable).

The impact of visceral and general obesity on vascular and left ventricular function and geometry: a cross-sectional magnetic resonance imaging study of the UK Biobank

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Received 8 March 2019; editorial decision 17 October 2019; accepted 21 October 2019; online publish-ahead-of-print 13 November 2019

Aims

We aimed to evaluate the associations of body fat distribution with cardiovascular function and geometry in the middle-aged general population.

Methods and results

Four thousand five hundred and ninety participants of the UK Biobank (54% female, mean age 61.1 ± 7.2 years) underwent cardiac magnetic resonance for assessment of left ventricular (LV) parameters [end-diastolic volume (EDV), ejection fraction (EF), cardiac output (CO), and index (CI)] and magnetic resonance imaging for body composition analysis [subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT)]. Body fat percentage (BF%) was assessed by bioelectrical impedance. Linear regressions were performed to assess the impact of visceral (VAT) and general (SAT and BF%) obesity on cardiac function and geometry. Visceral obesity was associated with a smaller EDV [VAT: β -1.74 (-1.15 to -2.33)], lower EF [VAT: β -0.24 (-0.12 to -0.35), SAT: β 0.02 (-0.04 to 0.08), and BF%: β 0.02 (-0.02 to 0.06)] and the strongest negative association with CI [VAT: β -0.05 (-0.06 to -0.04), SAT: β -0.02 (-0.03 to -0.01), and BF% β -0.01 (-0.013 to -0.007)]. In contrast, general obesity was associated with a larger EDV [SAT: β 1.01 (0.72–1.30), BF%: β 0.37 (0.23–0.51)] and a higher CO [SAT: β 0.06 (0.05–0.07), BF%: β 0.02 (0.01–0.03)]. In the gender-specific analysis, only men had a significant association between VAT and EF [β -0.35 (-0.19 to -0.51)].

Conclusion

Visceral obesity was associated with a smaller LV EDV and subclinical lower LV systolic function in men, suggesting that visceral obesity might play a more important role compared to general obesity in LV remodelling.

Keywords

Obesity • Body fat distribution • Visceral adipose tissue • Left ventricular function • Left ventricular geometry • Magnetic resonance imaging

Introduction

The disease burden related to obesity increased significantly over the last decades, making excess body weight one of the most challenging public health problems.¹ Most previous studies investigating the relation between obesity and cardiovascular function used indirect

anthropomorphic obesity measures such as body mass index (BMI), rather than measures of body fat distribution such as subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT).² VAT is located around the abdominal organs, and metabolically distinct from SAT as VAT produces more pro-inflammatory adipokines and is related to insulin resistance.³ In addition, visceral obesity has been

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using R (version 3.5.0), *P*-values of < 0.05 were considered statistically significant.

Results

Out of a total of 502 617 participants of the UK Biobank, 5995 participants had available CMR data of whom 4590 participants also had abdominal MRI and BF% measurement data (flowchart shown in *Figure 1*). Baseline characteristics, stratified according to gender, are presented in *Table 1*. Included participants had a mean age of 61.1 ± 7.2 years, 54% was female, 52% of the women, and 68% of the men were overweight or obese.

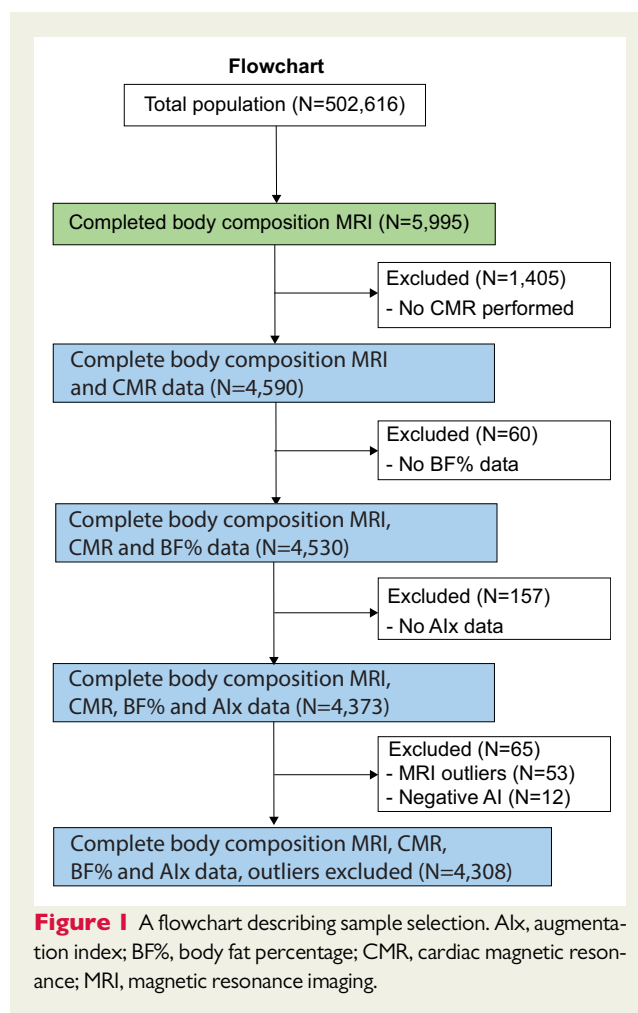
Associations between obesity measures and LV geometry

In the crude association, visceral obesity (VAT) was positively associated with EDV and general obesity measures (SAT and BF%) were negatively associated with EDV. After correction for age and gender (Model 1) this association reversed, showing a negative association of visceral obesity with EDV and a positive association between general obesity and EDV (Table 2). The direction of these associations persisted for Models 2 and 3 [Model 3; VAT: β -1.74 (-1.15 to -2.33), $P < 0.001$; SAT: β 1.01 (0.72–1.30), $P < 0.001$; BF%: β 0.37 (0.23–0.51), $P < 0.001$] (Figure 2A). Per 1 L change in VAT, EDV changed with -1.74 meaning that in an overt obese with 2SD more VAT compared to the average, EDV is on average 7.31 mL lower. Only for BF%, there was a significant difference between men and women (P for interaction < 0.001), where only women showed a positive association with EDV [women: β 0.54 (0.38–0.70), $P < 0.001$; men: β 0.03 (-0.26 to 0.32), $P = 0.84$]. A complete overview of the gender-specific associations is shown in [Supplementary data online, Table S1](#).

Visceral obesity was positively associated with ESV in the crude model, whereas general obesity showed a negative association with ESV. In adjustment Models 1–3, visceral obesity was not significantly associated with ESV (Table 2). General obesity was associated with larger ESV in correction Models 1 and 2 [Model 2; SAT: β 0.45 (0.27–0.63), $P < 0.001$; BF%: β 0.16 (0.06; 0.26), <0.001] (Figure 2B). No significant differences were found between men and women in the associations of obesity measures with ESV. Similar associations were observed between obesity measures and indexed EDV and ESV to height^{2.7} (Supplementary data online, Table S3).

Associations between obesity measures and LV systolic function

In the unadjusted analysis, a negative association was observed between visceral obesity and EF [β -0.49 (-0.41 to -0.57), $P < 0.001$], where general obesity showed a mild positive association [SAT: β 0.17 (0.11–0.23), $P < 0.001$; BF%: β 0.12 (0.10–0.14), $P < 0.001$]. In the adjusted analysis, the negative association of visceral obesity with EF remained significant after adjustment for Model 1, 2, and 3 [Model 3: β -0.24 (-0.12; -0.36), $P < 0.001$] (Table 2 and Figure 2C). In the gender-specific analysis, there was a significant negative association for men between visceral obesity and EF, which remained significant after adjustment [Model 3: β -0.35 (-0.19 to -0.51), $P < 0.001$ for men vs β -0.01 (-0.23 to 0.21), $P = 0.95$ for women, P for interaction = 0.02].



No significant associations were observed between general obesity and EF in the gender-specific analysis.

In crude analysis of general obesity measures, SAT was associated with higher CO and BF% with lower CO. After adjustment for confounders both obesity measures were positively associated with CO (Figure 2D). In the unadjusted analysis, visceral obesity was positively associated with CO, however, after correction for general obesity (Model 3), visceral obesity alone had no significant effect on CO [β 0.01 (-0.01 to 0.03), $P = 0.51$] (Table 3). Visceral obesity showed a stronger negative association with CI compared to general obesity in the crude association, which remained so in the adjusted models [VAT: β -0.05 (-0.04 to 0.06), $P < 0.001$; SAT: β -0.02 (-0.03; -0.01), $P < 0.001$; BF%: β -0.01 (-0.007 to -0.013), $P < 0.001$] (Figure 2E). No significant differences were found between men and women in the associations of obesity measures with CO and CI. Sensitivity analysis, where participants with diabetes and cardiovascular disease were excluded, showed similar results compared to our main findings for the associations between obesity measures and LV function and geometry (Supplementary data online, Table S2).

Associations between obesity measures and vascular stiffness

All obesity measures were significantly associated with higher Alx in the unadjusted analysis. The associations remained significant after

Table 1 Baseline characteristics stratified by gender

	Gender		Total population (n = 4590)
	Men (n = 2126)	Women (n = 2464)	
Age (years)	61.7 ± 7.1	60.6 ± 7.2	61.1 ± 7.2
Ethnicity (% Whites)	2050 (97.1)	2397 (98.0)	4447 (97.5)
Townsend deprivation index	-1.9 (2.8)	-1.9 (2.7)	-1.9 ± 2.7
Smoking (%)			
Never	1201 (57.0)	1576 (64.8)	2777 (61.1)
Former	794 (37.7)	767 (31.5)	1561 (34.4)
Current	112 (5.3)	90 (3.7)	202 (4.4)
Alcohol			
Never	121 (5.7)	170 (7.0)	291 (6.4)
Special occasions	148 (7.0)	360 (14.8)	508 (11.2)
1–3x/month	181 (8.6)	341 (14.0)	522 (11.4)
1–2x/week	539 (25.5)	653 (26.8)	1192 (26.2)
3–4x/week	635 (30.1)	565 (23.2)	1200 (26.4)
Daily	489 (23.1)	349 (14.3)	838 (18.4)
Systolic blood pressure (mmHg)	135.8 ± 16.9	136.2 ± 21.5	136.0 ± 19.5
Diastolic blood pressure (mmHg)	71.4 ± 10.0	66.9 ± 11.2	69.0 ± 10.9
History of			
Hypertension (%)	581 (27.5)	492 (20.2)	1073 (23.6)
Diabetes (%)	117 (5.5)	78 (3.2)	195 (4.3)
Angina (%)	47 (2.2)	32 (1.3)	79 (1.7)
Myocardial infarction (%)	65 (3.1)	16 (0.7)	81 (1.8)
Stroke (%)	37 (1.8)	18 (0.7)	55 (1.2)
Body surface area (m ²)	2.0 ± 0.2	1.7 ± 0.2	1.9 ± 0.2
Body mass index (kg/m ²)	26.8 ± 3.5	26.0 ± 4.4	26.4 ± 4.0
Overweight (%)	1092 (51.4)	864 (35.1)	1956 (42.7)
Obese (%)	358 (16.8)	401 (16.3)	759 (16.6)
Body fat percentage (%)	24.1 ± 5.2	35.4 ± 6.5	30.2 ± 8.2
Visceral adipose tissue (L)	4.7 ± 2.2	2.5 ± 1.4	3.5 ± 2.1
Subcutaneous adipose tissue (L)	5.7 ± 2.3	7.8 ± 3.3	6.9 ± 3.1
LV ejection fraction (%)	54.6 ± 6.2	57.6 ± 5.4	56.2 ± 6.0
LV end-diastolic volume (mL)	157.7 ± 33.6	123.0 ± 24.8	139.1 ± 34.0
LV end-systolic volume (mL)	72.2 ± 21.2	52.4 ± 13.7	61.6 ± 20.1
LV stroke volume (mL)	85.4 ± 18.9	70.5 ± 14.8	77.4 ± 18.4
LV cardiac output (L/min)	5.1 ± 1.1	4.4 ± 1.0	4.7 ± 1.1
LV cardiac index (L/min/m ²)	2.6 ± 0.5	2.5 ± 0.5	2.5 ± 0.5
Augmentation index (%)	19.3 ± 8.0	22.8 ± 8.8	21.2 ± 8.6

Data are shown as n (%) or mean ± SD. Overweight = 25–29.9 kg/m², Obese >30 kg/m². LV, left ventricular.

adjustment for Model 1, 2, and 3 [Model 3: VAT: β 0.71 (0.53–0.89), $P < 0.001$; SAT: β 0.37 (0.29–0.45), $P < 0.001$ and BF%: β 0.15 (0.11–0.19), $P < 0.001$] (Table 3). Higher positive effect estimates were found for visceral obesity and vascular stiffness in women compared to men, as is shown in Figure 2F [β 1.27 (0.94–1.60), $P < 0.001$ vs β 0.46 (0.24–0.68), $P < 0.001$, respectively, P for interaction < 0.001]. Similar positive associations were found for general obesity and vascular stiffness in women and men.

An overview of all results is summarized in Figure 3.

Discussion

In this population-based imaging study, we demonstrated the importance of body fat distribution in the association with cardiovascular function and geometry, where visceral obesity was associated with a

Table 2 Associations of obesity measures with cardiovascular function and geometry

	LV end-diastolic volume (mL)			LV end-systolic volume (mL)			LV ejection fraction (%)		
	β	95% CI	P-value	β	95% CI	P-value	β	95% CI	P-value
VAT (L)									
Crude	3.74	3.29 to 4.19	<0.001	2.46	2.19 to 2.73	<0.001	-0.49	-0.57 to -0.41	<0.001
Model 1	-0.25	-0.72 to 0.22	0.29	0.23	-0.04 to 0.50	0.12	-0.14	-0.24 to -0.04	0.005
Model 2	-0.37	-0.86 to 0.12	0.13	0.10	-0.19 to 0.39	0.52	-0.13	-0.23 to -0.03	0.01
Model 3	-1.74	-2.33 to -1.15	<0.001	-0.33	-0.70 to 0.04	0.08	-0.24	-0.36 to -0.12	<0.001
SAT (L)									
Crude	-0.93	-1.24 to -0.62	<0.001	-0.62	-0.82 to -0.42	<0.001	0.17	0.11 to 0.23	<0.001
Model 1	0.98	0.71 to 1.25	<0.001	0.48	0.30 to 0.66	<0.001	0.001	-0.06 to 0.06	0.98
Model 2	1.01	0.72 to 1.30	<0.001	0.45	0.27 to 0.63	<0.001	0.02	-0.04 to 0.08	0.55
BF% (%)									
Crude	-1.36	-1.48 to -1.24	<0.001	-0.77	-0.83 to -0.71	<0.001	0.12	0.10 to 0.14	<0.001
Model 1	0.33	0.19 to 0.47	<0.001	0.16	0.08 to 0.24	<0.001	0.01	-0.01 to 0.03	0.58
Model 2	0.37	0.23 to 0.51	<0.001	0.16	0.06 to 0.26	<0.001	0.02	-0.02 to 0.06	0.31

Model 1 is adjusted for age and sex. Model 2 is adjusted for Model 1 plus ethnicity, socioeconomic status, alcohol consumption, smoking, hypertension, and diabetes. Model 3 is adjusted for Model 2 covariates plus BF%. BF%, body fat percentage; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue.

smaller EDV, lower systolic cardiac function particularly in men and greater vascular stiffness in women.

Obesity measures and LV geometry

The positive association of general obesity with LV end-diastolic dimensions and CO is consistent with a theory postulated in 1986, which provides a potential pathophysiological process for obesity cardiomyopathy.¹⁶ This process has been described as an increase in perfusable tissue, expanding intravascular volume and by that demanding greater CO. Since heart rate is not influenced by obesity, CO is augmented through stroke volume. The expanded intravascular volume causes an increase in LV pressure, inducing a shift in the Frank–Starling curve and thereby initiating the chamber dilatation necessary for increased stroke volume. This theory recently found support, where through Mendelian Randomization, indications for a causal relation were found between BMI and CO driven by stroke volume.¹⁷ In contrast to general obesity, visceral obesity was associated with a smaller EDV. A possible contributing factor is the increased risk of diabetes and insulin resistance that is associated with visceral obesity, factors linked to smaller LV dimensions.¹⁸ However, in our regression models, we corrected for diabetes and results remained similar after excluding diabetics and patients with cardiovascular disease from the analysis. Additionally, allometric scaling did not change the associations observed between obesity and LV volumes.

Obesity measures and LV systolic function

The association of visceral obesity with EF and CI was consistent across the unadjusted and adjusted models, suggesting that VAT is an independent risk factor for lower systolic LV function. Since men have larger volumes of VAT, the fact that only men had a negative association between VAT and EF supports the suggestion that VAT is an independent risk factor for cardiac dysfunction. Per 1 L change in VAT,

LV EF changed with -0.35% in men, meaning that in an overt obese male with 2SD more VAT compared to the average, LVEF is on average 1.54% lower. While visceral obesity was associated with subclinical lower systolic LV function, general obesity was the predominant factor for the higher CO associated with obesity, where the association between VAT and CO diminished after correction for general obesity. Furthermore, an overall negative association of obesity with CI was found, with the strongest negative association for visceral obesity. We postulate that the mechanisms seen in general obesity leading to a state of high CO might be prevented in visceral obesity by a direct metabolic effect of VAT on LV structure and function resulting in smaller LV dimensions with a lower EF and CI. A process possibly guided by the proatherogenic and prohypertrophic adipokines released from VAT, recently demonstrated in the cardiac aging effect of VAT through myocardial fibrosis by secretion of osteopontin.¹⁹

Obesity measures and vascular stiffness

Overall, all obesity measures were positively associated with vascular stiffness, where visceral obesity in women showed the strongest association with vascular stiffness. The Alx is known to be higher in women compared with men, however, for general obesity, this did not result in higher Alx for women.¹³ It has been suggested that the increased inflammation status and adipokine secretion associated with visceral adiposity leads to endothelial dysfunction and vascular stiffness.²⁰ The exact pathophysiological effect of (visceral) obesity on vascular stiffness and the differences between men and women herein are unclear and need further research. The early reflected pulse wave in vascular stiffness that augments systolic pressure increases afterload and is associated with increased LV concentricity, thereby possibly contributing to the association of visceral obesity with a smaller EDV.²¹

In context of current literature

Until recent, studies using anthropomorphic measures like BMI suggested that obesity was associated with larger LV volumes and

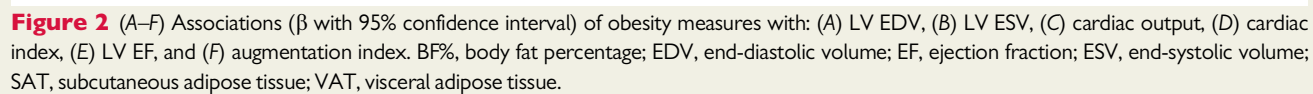


Table 3 Associations of obesity measures with cardiovascular function and geometry

	Cardiac output (L/min)			Cardiac index (L/min/m ²)			Augmentation index (%)		
	β	95% CI	P-value	β	95% CI	P-value	β	95% CI	P-value
VAT (L)									
Crude	0.12	0.11 to 0.13	<0.001	-0.03	-0.04 to -0.02	<0.001	0.13	0.01 to 0.25	0.03
Model 1	0.06	0.04 to 0.08	<0.001	-0.04	-0.05 to -0.03	<0.001	0.64	0.50 to 0.78	<0.001
Model 2	0.05	0.03 to 0.07	<0.001	-0.04	-0.05 to -0.03	<0.001	0.63	0.49 to 0.77	<0.001
Model 3	0.01	-0.01 to 0.03	0.51	-0.05	-0.06 to -0.04	<0.001	0.71	0.53 to 0.89	<0.001
SAT (L)									
Crude	0.02	0.01 to 0.03	0.002	-0.02	-0.03 to -0.01	<0.001	0.47	0.39 to 0.55	<0.001
Model 1	0.06	0.05 to 0.07	<0.001	-0.02	-0.03 to -0.01	<0.001	0.33	0.25 to 0.41	<0.001
Model 2	0.06	0.05 to 0.07	<0.001	-0.02	-0.03 to -0.01	<0.001	0.37	0.29 to 0.45	<0.001
BF% (%)									
Crude	-0.02	-0.024 to -0.016	<0.001	-0.01	-0.012 to -0.008	<0.001	0.26	0.22 to 0.30	<0.001
Model 1	0.02	0.01 to 0.03	<0.001	-0.01	-0.012 to -0.008	<0.001	0.15	0.11 to 0.19	<0.001
Model 2	0.02	0.01 to 0.03	<0.001	-0.01	-0.013 to -0.007	<0.001	0.15	0.11 to 0.19	<0.001

Model 1 is adjusted for age and sex. Model 2 is adjusted for Model 1 plus ethnicity, socioeconomic status, alcohol consumption, smoking, hypertension, and diabetes. Model 3 is adjusted for Model 2 covariates plus BF%. The augmentation index was additionally adjusted for height, heart rate, and mean arterial pressure in Models 2 and 3. BF%, body fat percentage; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue.

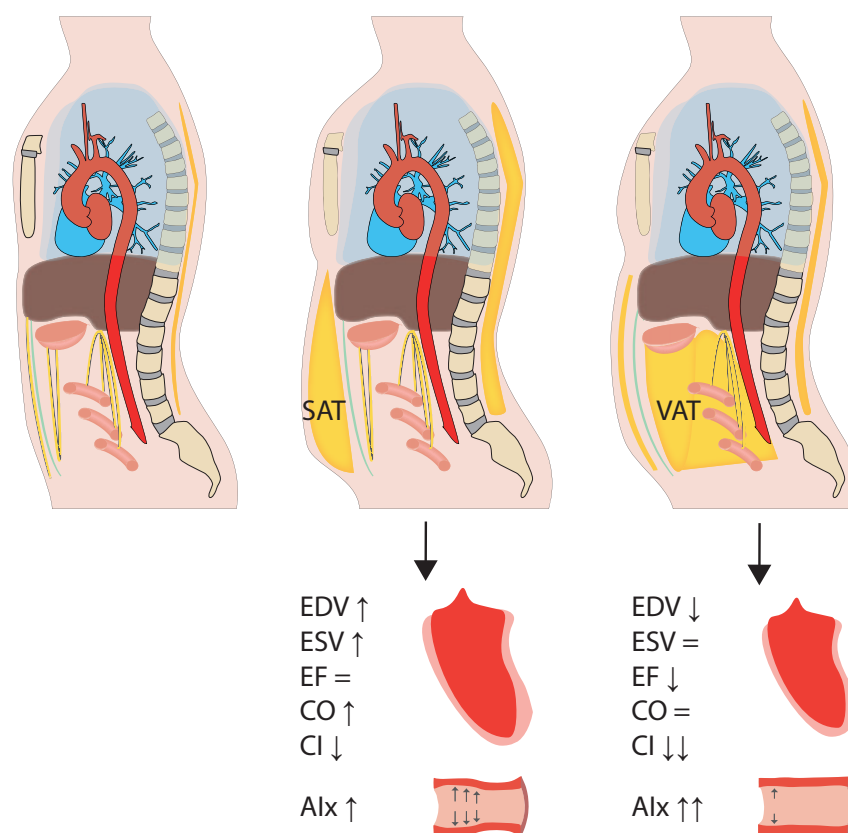


Figure 3 The impact of fat distribution on cardiovascular function and geometry. Visceral obesity (assessed by VAT volume) and general obesity (assessed by SAT volume and BF%) exhibit different associations with structural and functional cardiovascular properties. Where general obesity is associated with larger LV volumes and greater CO, visceral obesity is associated with a smaller end-diastolic volume and lower LV systolic function. All measures of obesity are associated with increased vascular stiffness, with the strongest association for visceral obesity. Alx, augmentation index; CI, cardiac index; CO, cardiac output; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue.

increased CO but not with EF.¹⁷ With the knowledge that obesity increases the risk of heart failure without an apparent negative impact of obesity on systolic function, the effect of obesity on heart failure was contributed to diastolic dysfunction.⁵ More recent studies investigating the impact of body fat distribution on LV geometry, similar to our study, found that visceral adiposity was associated with smaller LV volumes.⁴

The effect of obesity on LV systolic function is complex, where a previous study showed complicated curvilinear associations between obesity measures and the LV.²² Many previous studies, using anthropomorphic obesity measures, observed no change or even an increase in EF with obesity. The MESA study, a population-based cohort, previously stated that LV systolic function was insensitive to myocardial changes associated with anthropomorphic obesity measures.²³ However, a subsequent MESA study investigating fat distribution found that above-median VAT was associated with a significant lower EF.⁶ The Dallas Heart Study did not report EF, however they did show a similar positive association of SAT with CO.⁴ Unlike our results they found a negative association of VAT with CO, possibly due to their notably obese population (43.9% obese versus 16.6% in our population).

For vascular stiffness, in recent years few studies have investigated the influence of fat distribution beyond anthropomorphic measures on Alx. The Framingham Heart Study ($n = 2735$) is the only large cohort to investigate the association of VAT and Alx and found no significant association.⁷ This could be due to the lean population that was studied (mean VAT of 1.76 L compared to 3.5 L in our population). The Framingham study also did not find any gender differences, whereas in our study women showed a significantly stronger association between visceral obesity and vascular stiffness compared to men, an interesting finding which warrants further research.

Limitations

There are several limitations that need to be considered. First, this is a cross-sectional study, therefore, no causal relation between obesity measures and cardiovascular function can be determined. Second, although the adjustment models in our study corrected for many known confounders, we were unable to investigate the influences of medication use, blood serum data or physical activity because the data was unavailable or there were too many missing values. Despite the many LV parameters used in our analysis, data on LV mass and diastolic function were unavailable; consequently, the association of visceral obesity with hypertrophic remodelling and LV relaxation could not be assessed. The MRI scanner that was used has a maximum weight capacity of 250 kg, which could potentially be a source of selection bias. The UK population that was investigated consisted mainly of Caucasians aged 45–73 years, so the results may not be generalizable to other ethnic groups or ages. However, previous large scale studies investigating the impact of VAT on cardiovascular function were performed in the United States, with a notably obese population, and focussed on the USA specific ethnic groups.^{4,6} The current study is more representative for the European population.

Conclusion

In this large population-based imaging study, we showed that visceral and general obesity are associated with different structural and functional cardiovascular properties. Visceral obesity was associated with a smaller LV EDV and subclinical lower LV systolic function in men, suggesting that visceral obesity might play a more important role compared to general obesity in LV remodelling.

Supplementary data

Supplementary data are available at *European Heart Journal - Cardiovascular Imaging* online.

Acknowledgements

This research has been conducted using the UK Biobank Resource under Application Number '20666'.

Conflict of interest: none declared.

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IMAGE FOCUS

doi:10.1093/ehjci/jez251

Online publish-ahead-of-print 4 October 2019

Mystifying mass in the right ventricle

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An asymptomatic 59-year-old man was referred to the outpatient clinic with a right bundle branch block and left anterior hemiblock. Physical examination revealed no abnormalities. The echocardiogram showed a large mobile mass in the right ventricle (Panel A and [Supplementary data](#) online, [Video S1](#)). There were no signs of pericardial effusion, the right ventricular (RV) and left ventricular function were normal.

The differential diagnosis of this cardiac mass included: myxoma, fibroma, thrombus, sarcoma, or other malignant tumours.

Cardiac magnetic resonance imaging (MRI) showed the mobile mass originating from the RV apex extending into the RV outflow tract (Panel B and [Supplementary data](#) online, [Videos S2](#) and [S3](#)).

T1 and T2 imaging also with fat suppression ruled out a lipoma. Early gadolinium enhancement and late gadolinium enhancement imaging showed hyperenhancement in the core of the mass, unusual for cardiac tumours, e.g. sarcoma (Panel C and [Supplementary data](#) online, [multiview 1](#)).

As the right ventricle contracted well, this makes the diagnosis of RV thrombus less likely. Thus, we narrowed down the differential diagnosis to myxoma, fibroma, thrombus, sarcoma, and we referred the patient to the cardiac surgeon.

The patient was operated on, and a solid mass, partly with thrombus was removed from the right ventricle (Panel D).

Pathology reports that our 'mystifying mass' was an old and organized thrombus with calcifications. We have been unable to find similar clinical cases in the literature.

In conclusion, cardiac MRI can be very helpful with for differentiating cardiac masses, but in some cases the aetiology remains unclear until pathology has been obtained.

[Supplementary data](#) are available at *European Heart Journal - Cardiovascular Imaging* online.

