

Adipose Tissue Distribution and Subclinical Myocardial Dysfunction in Young Adults with Class I Obesity

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fasting glucose, triglycerides, triglyceride/high-density lipoprotein-cholesterol ratio, Visceral Adiposity Index (VAI), and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR; all $p < 0.001$), together with greater visceral adipose tissue (VAT) and epicardial adipose tissue (EAT) thickness.

Importantly, myocardial function was already impaired. Left ventricular global longitudinal strain (GLS) was reduced in the obesity group (-19.0% versus -21.8% ; $p < 0.001$) and correlated with VAT thickness ($r = 0.6$), with each additional millimetre associated with a 0.043% worsening of GLS. EAT correlated inversely with left atrial reservoir strain (LASr; $r = -0.4$), with each millimetre associated with a 1.21% lower LASr. Thus, longitudinal deformation of both left heart chambers was impaired despite the absence of diagnosed disease.

SUMMARY OF KEY RESEARCH FINDINGS

The clinical significance of Class I obesity (BMI: 30.0 – 34.9 kg/m²) in young adults without diagnosed disease remains uncertain. Although their BMI exceeds the threshold associated with increased cardiovascular risk, these individuals often receive only general lifestyle advice. The author assessed whether myocardial function is already impaired at this stage.

In this review, 131 adults aged 21–59 years undergoing a preventive health examination with no diagnosed non-communicable disease and no drug therapy were studied. Of these, 70 had a BMI < 30 kg/m² and 61 had Class I obesity. All underwent anthropometry, echocardiography (including speckle-tracking strain analysis), ultrasound assessment of adipose tissue distribution, and measurement of fasting glucose, insulin, and lipids.

Class I obesity was associated with an adverse metabolic profile, including higher

WHAT CHALLENGE DOES THIS ADDRESS?

VAT and EAT may affect the heart through distinct mechanisms. VAT acts systemically through insulin resistance, lipotoxicity, and adipokine imbalance, and was associated with ventricular deformation. EAT lies directly on the myocardium and may exert local paracrine effects; in this study, it was associated with atrial function. BMI captures neither depot.

Young adults with Class I obesity may therefore be poorly characterised by conventional risk assessment. They often fall outside the age ranges in which risk equations are most informative, and these tools do not account for adipose tissue distribution. Yet obesity is an important precursor of heart failure with preserved ejection fraction, and cardiac remodelling develops before symptoms become apparent. These findings suggest that this preclinical stage can be detected through changes in myocardial deformation.

RELEVANCE TO EUROPEAN PRACTICE

Obesity affects a substantial and increasing proportion of the European adult population. European prevention guidance emphasises weight management but provides limited tools for identifying which individuals with mild obesity already have subclinical cardiac involvement.

These measurements are already available in routine echocardiographic practice. EAT and VAT thickness, GLS, and LASr can be assessed without CT or MRI, ionising radiation, or substantial additional cost. The practical implication is that a structurally normal heart in a young person with mild obesity should not necessarily be considered reassuring. Evidence of increased adiposity together with impaired longitudinal function may identify individuals who could benefit from more intensive early intervention. As anti-obesity pharmacotherapy expands across Europe, cardiac phenotyping of this kind may also help to determine which patients should be prioritised for treatment.

WHAT ARE THE NEXT STEPS FOR THE RESEARCH?

The key unanswered question is reversibility. EAT is metabolically active and responds to weight loss, but whether its regression restores myocardial deformation and ultimately reduces cardiovascular events is unknown. The expanding use of glucagon-like peptide-1 receptor agonists makes this question both testable and clinically relevant.

Prospective studies are also needed to determine whether increased EAT/VAT and reduced strain precede incident hypertension, diabetes, and heart failure with preserved ejection fraction, and whether they provide incremental prognostic value beyond established risk factors. Before clinical implementation, clinically applicable cut-off values should be derived and externally validated, while ultrasound protocols require standardisation and comparison with CT or MRI. These studies will help determine whether epicardial and visceral adiposity are simply markers of early myocardial involvement or potentially modifiable targets in their own right.

References

1. Alieva R et al. Epicardial and visceral adiposity are associated with subclinical myocardial dysfunction in young individuals with class I obesity. Abstract. ESC Congress, 28-31 August, 2026.