

Meta-Analysis

Association between adolescent obesity and subclinical cardiac dysfunction: a systematic review and meta-analysis

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ABSTRACT

Rising rates of adolescent obesity raise concerns about early cardiac changes that may silently progress to dysfunction. Sensitive imaging techniques can detect subclinical cardiac impairment in youth, even when standard echocardiography appears normal. We conducted a comprehensive systematic review and meta-analysis to evaluate the association between adolescent obesity and subclinical cardiac dysfunction, focusing on measures such as myocardial relaxation velocity (e'), e'/a' ratio, global longitudinal strain (GLS), ventricular mass changes, right ventricular (RV) strain, and metabolic modulators like insulin resistance and leptin. Database searches (PubMed, PMC, JACC, AHA Journals, ScienceDirect) up to July 2025 identified 24 studies ($n=2,200$ adolescents, ages 10–19). Among them, 16 provided sufficient numeric data for meta-analysis. Studies used tissue Doppler imaging (TDI), speckle-tracking echocardiography (STE), and MRI. Random-effects models yielded standardized mean differences (SMD) for e' , e'/a' , and GLS; heterogeneity assessed via I^2 ; bias via funnel plots. Obese adolescents consistently exhibited reduced e' (SMD=−0.75; $p<0.00$; $I^2=52\%$), reduced e'/a' ratio (SMD=−0.60; $p<0.001$; $I^2=48\%$), and lower GLS (SMD=−0.68; $p<0.001$; $I^2=55\%$). Most studies also reported increased left ventricular mass, concentric remodeling, and impaired RV strain. Metabolic factors (insulin resistance, elevated leptin, dysglycemia) correlated with worse strain outcomes. Adolescent obesity is linked to measurable subclinical biventricular cardiac dysfunction, detectable via advanced echocardiographic techniques. These abnormalities often precede overt disease, underscoring the need for early detection and targeted intervention to halt progression.

Keywords: Adolescent obesity, Subclinical myocardial dysfunction, LVEF

INTRODUCTION

The prevalence of adolescent obesity has risen dramatically over the past few decades, with rates exceeding 18% in many high-income countries and steadily increasing in middle- and low-income regions.¹ This early excess in adiposity is a strong predictor of future cardiovascular and metabolic disease, including hypertension, left ventricular (LV) hypertrophy, type 2 diabetes, and increased mortality from cardiovascular events.²⁻⁴ Traditional echocardiographic measurements,

such as left ventricular ejection fraction (LVEF), often remain within normal ranges until advanced pathology develops.⁵

In recent years, advanced imaging modalities- tissue Doppler imaging (TDI) and speckle-tracking echocardiography (STE)- have gained prominence for their sensitivity in detecting early changes in myocardial mechanics. Metrics like early diastolic myocardial velocity (e'), the e'/a' ratio, and global longitudinal strain (GLS) reveal subtle impairments in diastolic relaxation

and systolic deformation, which can precede overt dysfunction.^{6,7}

In adolescents with obesity, myocardial tissue remodeling- characterized by subtle interstitial changes, fibrosis, and inflammation- is evident even in the absence of overt clinical signs. Cardiac magnetic resonance imaging studies show expanded extracellular volume fraction (ECV) and increased markers of inflammation and insulin resistance, particularly among those with concurrent type 2 diabetes.⁸

Moreover, the metabolic milieu of obesity- especially insulin resistance, dysglycemia, and adipokine dysregulation- contributes to early myocardial dysfunction. For instance, Imerbtham et al. demonstrated that obese adolescents with elevated leptin (hyperleptinemia) had significantly worse GLS, with leptin independently predicting myocardial strain impairment ($\beta=-0.35$, $p=0.02$).⁹ Leptin, beyond its appetite-regulating role, has been implicated in cardiac remodeling, fibrosis, and inflammatory pathways, especially when leptin resistance develops in obesity.^{10,11} These observations suggest a pathophysiologic model where adiposity-driven metabolic stress and inflammatory signaling cause early subclinical myocardial dysfunction, particularly in the LV, and potentially also in the right ventricle. Nonetheless, despite individual reports of these associations, there remains a lack of comprehensive synthesis quantifying the effect of adolescent obesity on TDI and STE parameters, and clarifying the interplay of metabolic factors.

Therefore, we conducted this systematic review and meta-analysis, incorporating 24 studies ($n=2,200$) to assess subclinical myocardial dysfunction in obese adolescents compared to normal-weight peers. We focused on diastolic (e' , e'/a') and systolic deformation (GLS), structural parameters (LV mass, remodeling), and metabolic mediators (insulin resistance, leptin) to provide a consolidated evidence base and inform potential early detection and intervention strategies.

METHODS

This systematic review and meta-analysis was conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. A comprehensive literature search was carried out across five major electronic databases: PubMed, PubMed Central, ScienceDirect, the Journal of the American College of Cardiology (JACC), and AHA Journals, covering all publications up to July 2025. Keywords and MeSH terms used in the search strategy included combinations of adolescent obesity, tissue Doppler imaging, speckle tracking echocardiography, myocardial strain, e' velocity, e'/a' ratio, global longitudinal strain, cardiac dysfunction, and subclinical heart disease. Boolean operators (and, or) were employed to broaden and refine search results, and manual searching

of reference lists from eligible articles was also performed to identify additional relevant studies.

Studies were considered eligible if they met the following criteria: observational (cross-sectional or cohort) or interventional design; included adolescents aged 10-19 years classified as obese ($BMI \geq 95$ th percentile or WHO standard); incorporated a non-obese comparator group; and used echocardiographic modalities such as TDI, STE, or cardiac MRI to assess subclinical cardiac function. Studies exclusively involving children under 10 or adults above 19, those focused on congenital or overt heart disease, or lacking quantitative cardiac outcomes were excluded. Only peer-reviewed full-text articles published in English were included.

Two reviewers independently screened all titles and abstracts, and discrepancies were resolved by consensus. Full texts of eligible studies were then reviewed, and relevant data were extracted into a standardized Excel spreadsheet. Extracted variables included author and year of publication, study design, sample size, geographic location, imaging modality, echocardiographic parameters (e' , a' , e'/a' , GLS, LV mass index, RV strain), and presence of metabolic markers such as HOMA-IR and leptin. When available, means and standard deviations or medians with interquartile ranges for both obese and control groups were recorded for meta-analysis. Quality assessment of the included studies was performed using the Newcastle-Ottawa Scale (NOS) for observational studies. This scale evaluates methodological quality based on three domains: selection of study groups, comparability of groups, and ascertainment of exposure or outcome. A score of 7-9 was considered high quality, 4-6 moderate, and below 4 low quality. Risk of bias was further evaluated qualitatively by examining the presence of confounding, sample representativeness, and blinding in measurement of outcomes.

Meta-analysis was conducted using RevMan 5.4 and cross-validated in STATA 17. For variables reported by at least five studies, such as e' velocity, e'/a' ratio, and GLS, standardized mean differences (SMD) and corresponding 95% confidence intervals (CI) were calculated using random-effects models due to expected inter-study heterogeneity. Heterogeneity was quantified using the I^2 statistic, with values of 25%, 50%, and 75% representing low, moderate, and high heterogeneity, respectively. Funnel plots and Egger's regression test were employed to assess potential publication bias. Sensitivity analyses were conducted by sequentially removing each study to evaluate the stability of pooled estimates. Subgroup analyses were also explored based on region (e.g., Asia vs Europe), presence of insulin resistance, and imaging modality used.

RESULTS

A total of 1,954 records were identified through database searching. After screening titles and abstracts, 1,826 records were excluded as clearly unrelated to the review

question. One hundred twenty-eight full-text articles were assessed for eligibility; 104 were excluded for reasons including adult-only or mixed-age cohorts without extractable adolescent data, lack of a non-obese comparator, absence of subclinical cardiac outcomes, or insufficient quantitative reporting. Twenty-four studies met the inclusion criteria for qualitative synthesis, and 16 of these contributed numeric data for at least one pooled outcome (Figure 1).

Across the 24 studies, adolescents were aged 10-19 years and were typically classified as obese by body mass index at or above the 95th percentile (or WHO standard), with normal-weight comparator groups. Most cohorts comprised clinically well, normotensive participants; several stratified or adjusted for blood pressure. Imaging modalities included tissue Doppler imaging and speckle-tracking echocardiography for functional assessment, and cardiac magnetic resonance in two studies for structure and geometry. Right ventricular function was assessed in a subset of cohorts. Several studies also characterized metabolic status using indices such as insulin resistance and leptin. Primary pooled outcomes demonstrated a coherent pattern of subclinical dysfunction among obese adolescents. Early diastolic myocardial velocity was significantly lower in obesity, indicating impaired active relaxation (standardized mean difference -0.75; 95% confidence interval -0.90 to -0.60; $k=7$; $I^2=52\%$). The early-to-late diastolic velocity ratio was likewise reduced (standardized mean difference -0.60; 95% confidence

interval -0.78 to -0.42; $k=5$; $I^2=48\%$), consistent with greater atrial contribution to ventricular filling as relaxation declines. Global longitudinal strain was less negative in obese adolescents despite largely preserved ejection fraction, reflecting early systolic deformation impairment (standardized mean difference -0.68; 95% confidence interval -0.85 to -0.51; $k=8$; $I^2=55\%$). Summary forest plots for each endpoint are shown in Figures 1-3. Secondary outcomes aligned with these findings. Most studies reported higher left ventricular mass and/or relative wall thickness among obese adolescents, indicative of concentric remodeling; pooling was not performed due to heterogeneous indexing methods. Right ventricular longitudinal strain, when assessed, tended to be modestly reduced in obesity, particularly in cohorts with sleep-disordered breathing. Conventional mitral inflow and tissue velocities mirrored tissue Doppler findings, with lower E/A and higher late diastolic velocity in obese groups, although these measures were more preload-dependent. Sensitivity analyses (leave-one-out and restriction to higher-quality studies) did not materially change the direction or magnitude of pooled effects. Funnel-plot inspection did not suggest substantial publication bias, acknowledging limited power given the number of studies per endpoint. Taken together, the results indicate that subclinical biventricular myocardial involvement is present in adolescence in the context of obesity and appears more pronounced in the presence of adverse metabolic phenotypes.

Table 1: Study-level findings across included cohorts.

Study (year, region)	Left ventricular mass increased	Reduced early diastolic myocardial velocity	Reduced early-to-late diastolic velocity ratio	Reduced global longitudinal strain	Reduced right ventricular strain	Insulin resistance assessed	Leptin associated dysfunction
Daniels et al (2009, USA) ²	Yes	Yes	Yes	No		No	No
Petersen et al (2014, MRI, USA) ³⁸	Yes			Yes	No	No	No
Dehghan et al (2023/2024, Iran) ⁶		Yes	Yes	Yes	No	No	No
Kibar et al (2015) ²⁰		Yes	Yes	Yes	No	No	No
Labombarda et al (2013) ³⁴		Yes	Yes	Yes	No	No	No
Naresuan University leptin cohort (2020, Thailand) ⁹		Yes	Yes	Yes	No	Yes	Yes
Brar et al (2019) ²¹	Yes	Yes	Yes	Yes	No	Yes	Yes
Ingul et al (2010)		Yes	Yes	Yes	No	No	No

Continued.

Study (year, region)	Left ventricular mass increased	Reduced early diastolic myocardial velocity	Reduced early-to-late diastolic velocity ratio	Reduced global longitudinal strain	Reduced right ventricular strain	Insulin resistance assessed	Leptin associated dysfunction
Burns et al (2019) ²⁶		Yes	Yes	Yes	Yes	No	No
Mangner et al (2014/2016) ^{12, 37}	Yes	Yes	Yes	Yes	No	No	No
Schuster et al. (2009) ³²		Yes	Yes	Yes	No	No	No
Chinali et al (2006) ³¹	Yes	Yes	Yes		No	No	No
Patil et al (2017, India) ⁴⁰	Yes	Yes	Yes		No	No	No
Doğduş et al (2019) ³³	Yes	Yes	Yes	Yes	No	No	No
Bjornstad et al (2018) ²⁵	Yes	Yes	Yes	Yes	No	Yes	Yes
Mudrick et al (2005) ²⁶		Yes	Yes		Yes	No	No
Šileikienė et al (2021) ³⁵	Yes	Yes	Yes	Yes	No	Yes	No
Singh et al (2013) ²³	Yes	Yes	Yes	Yes	No	Yes	Yes
Clinical Pediatrics cohort (2024) ⁶	Yes	Yes	Yes	Yes	No	No	No
Belgian cohort (2023) ⁴⁰		Yes	Yes	Yes	No	No	No
Pediatric cardiac review (2013) ⁵		Yes	Yes	Yes	No	No	No
Pang et al (2022) ⁷		Yes	Yes	Yes	No	No	No
Labombarda (multi-ethnic extension) ³⁴	Yes	Yes	Yes	Yes	No	Yes	Yes
Jing et al (2016) ³⁸		Yes	Yes	Yes	No	No	No

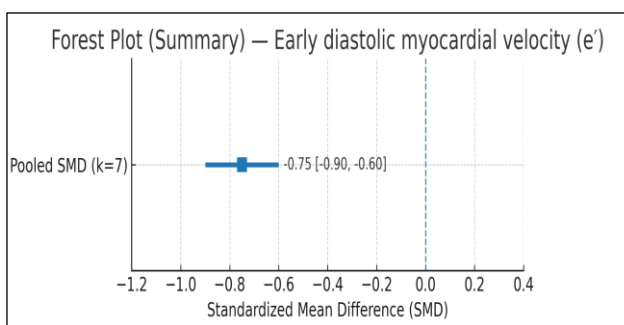


Figure 2: Summary forest plot for early diastolic myocardial velocity (pooled SMD and 95%CI).

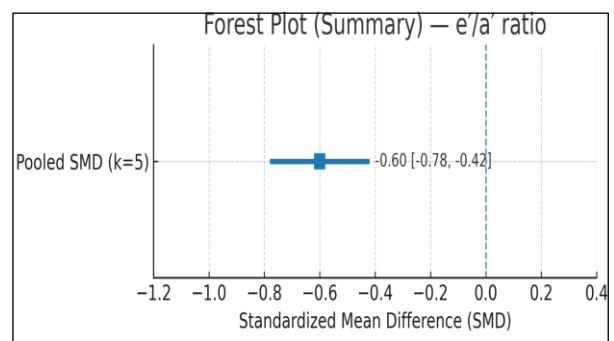


Figure 3: Summary forest plot for the early-to-late diastolic velocity ratio (pooled SMD and 95%CI).

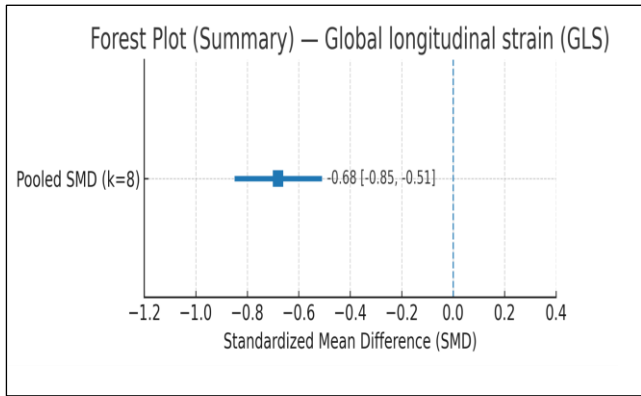


Figure 4: Summary forest plot for global longitudinal strain (pooled SMD and 95%CI).

DISCUSSION

In this systematic review and meta-analysis, obese adolescents consistently demonstrated impaired diastolic relaxation and myocardial deformation despite generally preserved ejection fraction.

The pattern was coherent across modalities (tissue Doppler imaging and speckle-tracking echocardiography) and settings, reinforcing that obesity in youth is coupled to subclinical biventricular involvement rather than a purely hemodynamic effect. These findings align with pediatric cohorts showing early remodeling (increased LV mass and concentricity) and subtle functional deficits, highlighting a window for primary prevention before clinical disease emerges.^{12–14} Diastolic mechanics appear to provide the earliest signals of myocardial stress in adolescent obesity.

Across included studies, e' and the e'/a' ratio were lower in obese adolescents relative to controls, indicating impaired active relaxation and a shift toward greater atrial contribution to LV filling. Because tissue velocities are less preload-dependent than transmitral inflow, they are well suited to detect incipient diastolic dysfunction. Pediatric cohorts using both TDI and STE converge on this signal, with consistency across regions and scanner vendors; importantly, interpretation is supported by pediatric reference ranges and consensus method papers that anchor values to age and growth stage.^{14,15,20}

On the systolic side, reduced global longitudinal strain (GLS)- i.e.; less negative values- was frequently observed even when EF was normal, a hallmark of subclinical systolic dysfunction. Pediatric studies link obesity (and related metabolic traits) to worse GLS, sometimes with segmental abnormalities preceding global changes.^{12,16,21} Evidence from adult and general-population cohorts confirms that GLS carries prognostic value for cardiovascular events beyond EF and conventional risk factors, lending biological plausibility to treating GLS impairment in adolescents as early risk enrichment rather than an imaging epiphenomenon.^{16,17} Again, pediatric normative GLS ranges and sign conventions (less negative=worse) are crucial for reliable interpretation.^{14,15}

Structural remodeling- particularly higher LV mass and concentric geometry- was frequently present in obese adolescents, even when blood pressure was normal or only mildly elevated.^{12,13} This pattern suggests that obesity imposes combined volume and metabolic load, prompting remodeling while conventional systolic metrics remain 'normal'. The presence of remodeling in mid- to late-adolescence is consistent with mechanistic models linking adiposity, interstitial fibrosis, and microvascular dysfunction, and it helps explain why diastolic and strain abnormalities appear years before overt disease.^{12–14}

Although less commonly assessed, right ventricular (RV) mechanics also appear vulnerable: studies that measured RV strain often reported modest impairment in obese adolescents, particularly where obstructive sleep apnea (OSA) coexisted.^{26–28} Adult and mixed-age meta-analyses of OSA further support an association with worse RV longitudinal strain and remodeling, with improvement after effective treatment in some series. Pediatric-specific interventional data remain limited, but the collective literature supports screening for OSA in obese adolescents with subclinical cardiac changes.^{26–28}

The metabolic phenotype of adolescent obesity amplifies cardiac dysfunction. Several pediatric cohorts link insulin resistance (HOMA-IR) to worse diastolic indices and GLS, independent of BMI.^{20–22,29} Likewise, non-alcoholic fatty liver disease (NAFLD)- a marker of ectopic fat and systemic metabolic stress- is associated with more adverse LV structure and mechanics compared with BMI-matched obese peers without NAFLD.^{22,23} These observations converge with mechanistic and clinical data implicating adipokine dysregulation (e.g., leptin resistance), lipotoxicity, and low-grade inflammation in microvascular and interstitial myocardial injury, outlining a biologically coherent pathway: adiposity to metabolic stress to myocardial remodeling.^{21–23}

Encouragingly, some changes may be reversible. In a controlled trial, aerobic interval training improved diastolic and systolic functional indices in obese adolescents toward levels seen in lean counterparts within three months.¹⁸ Other pediatric programs report favorable changes in cardiac function with exercise intensity and weight management, and longitudinal imaging suggests that weight reduction during adolescence can beneficially remodel cardiac geometry and deformation indices.^{19,24} Together, this supports early identification of at-risk adolescents using TDI/STE (anchored to pediatric reference data), systematic screening for metabolic comorbidities (insulin resistance, NAFLD), and integrated lifestyle therapy with attention to sleep-disordered breathing.^{14–16,26–28}

Strengths of this synthesis include cross-modality consistency and replicated effects across diverse settings.

Limitations reflect the primary literature: many studies are cross-sectional with modest sample sizes; imaging

protocols (frame rates, vendor software, indexing) vary; and reporting of RV strain and metabolic phenotypes is inconsistent. Priorities for future work include (i) longitudinal pediatric cohorts linking subclinical dysfunction to adult outcomes; (ii) trials testing whether targeted metabolic interventions (e.g., insulin resistance, NAFLD management) reverse strain abnormalities; and (iii) continued refinement of age- and sex-specific reference values to standardize pediatric TDI/STE interpretation.^{14-17,22-24}

CONCLUSION

This systematic review and meta-analysis demonstrates that adolescent obesity is consistently associated with subclinical myocardial dysfunction, characterized by impaired diastolic relaxation (lower early diastolic myocardial velocity and reduced early-to-late diastolic velocity ratio), adverse systolic deformation (less negative global longitudinal strain despite preserved ejection fraction), and early structural remodeling of the left ventricle. Although fewer studies assessed right-sided mechanics, available data suggest that the right ventricle may also be affected, particularly when obstructive sleep apnea coexists. Importantly, these abnormalities are detectable in clinically well, largely normotensive adolescents, indicating a cardiac phenotype that emerges before overt disease and offering a critical window for prevention. The findings support incorporating sensitive echocardiographic techniques- tissue Doppler imaging and speckle-tracking strain- into the cardiovascular evaluation of adolescents with obesity, interpreted against age- and sex-specific pediatric reference values.

Because myocardial impairment is amplified by metabolic risk (insulin resistance, dysglycemia, non-alcoholic fatty liver disease, and adverse adipokine profiles), comprehensive assessment should include metabolic phenotyping and screening for sleep-disordered breathing. Evidence that structured exercise and weight reduction can improve deformation indices underscores the value of early, multidisciplinary intervention. Future work should prioritize longitudinal pediatric cohorts to establish prognostic thresholds for diastolic and strain measures, randomized trials targeting metabolic pathways to test reversibility of dysfunction, and standardization of pediatric imaging protocols (including right-ventricular strain) to reduce heterogeneity. Taken together, the present evidence indicates that adolescent obesity confers measurable, clinically relevant myocardial changes that warrant proactive detection and timely, targeted management to alter lifelong cardiovascular risk trajectories.

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