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EARLY CARDIAC REMODELING IN OVERWEIGHT INDIVIDUALS: AN EVIDENCE-BASED SYSTEMATIC REVIEW

REMODELAÇÃO CARDÍACA PRECOCE EM INDIVÍDUOS COM SOBREPESO: UMA REVISÃO SISTEMÁTICA BASEADA EM EVIDÊNCIAS

REMODELACIÓN CARDÍACA TEMPRANA EN INDIVIDUOS CON SOBREPESO: UNA REVISIÓN SISTEMÁTICA BASADA EN LA EVIDENCIA

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ABSTRACT

Introduction: Overweight has traditionally been considered an intermediate, relatively benign stage between normal weight and obesity; however, recent evidence suggests that it may already be associated with structural and functional cardiac alterations preceding overt cardiovascular disease. Early cardiac remodeling, defined as subclinical changes in left ventricular geometry, mass, and diastolic function, may represent the earliest manifestation of myocardial maladaptation in this population.

Objective: The primary objective of this systematic review was to synthesize current evidence on early cardiac remodeling among overweight individuals (body mass index 25–29.9 kg/m²) compared with normal-weight controls. Secondary objectives included identifying the most sensitive imaging parameters for early detection, analyzing the modifying role of metabolic health and demographic variables, and evaluating the overall certainty of evidence.

Methods: A comprehensive search was performed in PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov, and ICTRP for studies published between January 2015 and October 2025. Observational, cross-sectional, and interventional human studies assessing structural or functional cardiac changes in overweight individuals without pre-existing cardiovascular disease were eligible**.** Two reviewers independently screened titles, abstracts, and full texts, extracted data in duplicate, and assessed risk of bias using RoB 2, ROBINS-I, or QUADAS-2, with overall certainty rated by GRADE.

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Results and Discussion: 18 studies met inclusion criteria, encompassing 27,800 participants. Most studies reported that overweight individuals exhibited increased left ventricular mass index, concentric remodeling, higher left atrial volume index, and impaired myocardial strain or diastolic function compared with normal-weight subjects. These alterations persisted even after adjusting for blood pressure and metabolic factors, suggesting an independent effect of adiposity. Despite consistent findings across imaging modalities, heterogeneity in study design, populations, and definitions of overweight limited pooled synthesis. Certainty of evidence was graded as moderate for structural outcomes and low for functional outcomes**.**

Conclusion: Overweight status is not metabolically or structurally innocuous. Evidence indicates that early cardiac remodeling can occur before the threshold of obesity, underscoring the importance of early risk assessment and lifestyle interventions. Standardized imaging criteria and longitudinal studies are needed to clarify reversibility and prognostic significance.

Keywords: Overweight. Cardiac Remodeling. Left Ventricular Hypertrophy. Echocardiography.

RESUMO

Introdução: Tradicionalmente, o sobrepeso tem sido considerado um estágio intermediário, relativamente benigno, entre o peso normal e a obesidade; no entanto, evidências recentes sugerem que ele pode estar associado a alterações cardíacas estruturais e funcionais que precedem doenças cardiovasculares manifestas. A remodelação cardíaca precoce, definida como alterações subclínicas na geometria, massa e função diastólica do ventrículo esquerdo, pode representar a manifestação mais precoce de má adaptação miocárdica nessa população.

Objetivo: O objetivo principal desta revisão sistemática foi sintetizar as evidências atuais sobre a remodelação cardíaca precoce em indivíduos com sobrepeso (índice de massa corporal de 25 a 29,9 kg/m²) em comparação com controles de peso normal. Os objetivos secundários incluíram a identificação dos parâmetros de imagem mais sensíveis para a detecção precoce, a análise do papel modificador da saúde metabólica e das variáveis demográficas e a avaliação da certeza geral das evidências.

Métodos: Foi realizada uma busca abrangente nas bases de dados PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov e ICTRP por estudos publicados entre janeiro de 2015 e outubro de 2025. Foram elegíveis estudos observacionais, transversais e intervencionais em humanos que avaliaram alterações cardíacas estruturais ou funcionais em indivíduos com sobrepeso sem doença cardiovascular preexistente. Dois revisores independentes analisaram os títulos, resumos e textos completos, extraíram os dados em duplicata e avaliaram o risco de viés utilizando as ferramentas RoB 2, ROBINS-I ou QUADAS-2, com a certeza geral classificada pelo GRADE.

Resultados e Discussão: Dezoito estudos atenderam aos critérios de inclusão, abrangendo 27.800 participantes. A maioria dos estudos relatou que indivíduos com sobrepeso apresentaram aumento do índice de massa ventricular esquerda, remodelamento concêntrico, maior índice de volume atrial esquerdo e comprometimento da deformação miocárdica ou da função diastólica em comparação com indivíduos com peso normal. Essas alterações persistiram mesmo após o ajuste para pressão arterial e fatores metabólicos, sugerindo um efeito independente da adiposidade. Apesar dos resultados consistentes em diferentes modalidades de imagem, a heterogeneidade no desenho dos estudos, nas populações estudadas e nas definições de sobrepeso limitou a síntese conjunta dos dados.

A certeza da evidência foi classificada como moderada para desfechos estruturais e baixa para desfechos funcionais.

Conclusão: O sobrepeso não é metabolicamente ou estruturalmente inócuo. As evidências indicam que a remodelação cardíaca precoce pode ocorrer antes do limiar da obesidade, ressaltando a importância da avaliação precoce de risco e de intervenções no estilo de vida. Critérios de imagem padronizados e estudos longitudinais são necessários para esclarecer a reversibilidade e o significado prognóstico.

Palavras-chave: Sobrepeso. Remodelação Cardíaca. Hipertrofia Ventricular Esquerda. Ecocardiografia.

RESUMEN

Introducción: Tradicionalmente, el sobrepeso se ha considerado una etapa intermedia y relativamente benigna entre el peso normal y la obesidad; sin embargo, evidencia reciente sugiere que podría estar asociado con alteraciones cardíacas estructurales y funcionales que preceden a la enfermedad cardiovascular manifiesta. La remodelación cardíaca temprana, definida como cambios subclínicos en la geometría, la masa y la función diastólica del ventrículo izquierdo, podría representar la manifestación más temprana de la mala adaptación miocárdica en esta población.

Objetivo: El objetivo principal de esta revisión sistemática fue sintetizar la evidencia actual sobre la remodelación cardíaca temprana en personas con sobrepeso (índice de masa corporal de 25 a 29,9 kg/m²) en comparación con controles de peso normal. Los objetivos secundarios incluyeron identificar los parámetros de imagen más sensibles para la detección temprana, analizar el papel modificador de la salud metabólica y las variables demográficas, y evaluar la certeza general de la evidencia.

Métodos: Se realizó una búsqueda exhaustiva en PubMed, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov e ICTRP para identificar estudios publicados entre enero de 2015 y octubre de 2025. Se incluyeron estudios observacionales, transversales e intervencionales en humanos que evaluaban cambios cardíacos estructurales o funcionales en personas con sobrepeso sin enfermedad cardiovascular preexistente. Dos revisores examinaron de forma independiente los títulos, resúmenes y textos completos, extrajeron los datos por duplicado y evaluaron el riesgo de sesgo mediante RoB 2, ROBINS-I o QUADAS-2, calificando la certeza general mediante GRADE.

Resultados y Discusión: Dieciocho estudios cumplieron los criterios de inclusión, con un total de 27 800 participantes. La mayoría de los estudios reportaron que las personas con sobrepeso presentaban un mayor índice de masa ventricular izquierda, remodelación concéntrica, un mayor índice de volumen auricular izquierdo y una menor deformación miocárdica o función diastólica en comparación con los sujetos con peso normal. Estas alteraciones persistieron incluso tras ajustar por presión arterial y factores metabólicos, lo que sugiere un efecto independiente de la adiposidad. A pesar de la consistencia de los hallazgos en las distintas modalidades de imagen, la heterogeneidad en el diseño de los estudios, las poblaciones y las definiciones de sobrepeso limitó la síntesis conjunta. La certeza de la evidencia se clasificó como moderada para los resultados estructurales y baja para los funcionales.

Conclusión: El sobrepeso no es inocuo desde el punto de vista metabólico ni estructural. La evidencia indica que la remodelación cardíaca temprana puede ocurrir antes de alcanzar el umbral de la obesidad, lo que subraya la importancia de la evaluación precoz del riesgo y

las intervenciones en el estilo de vida. Se necesitan criterios de imagen estandarizados y estudios longitudinales para esclarecer la reversibilidad y el significado pronóstico.

Palabras clave: Sobrepeso. Remodelación Cardíaca. Hipertrofia Ventricular Izquierda. Ecocardiografía.

1 INTRODUCTION

Overweight, defined by a body mass index (BMI) between 25 and 29.9 kg/m², has become a major epidemiological concern, affecting more than 1.9 billion adults globally.[1] The cardiovascular impact of this condition extends beyond traditional risk factors such as hypertension and dyslipidemia, encompassing structural and functional changes in the myocardium that may occur even before overt obesity develops.[1] These early changes, collectively termed cardiac remodeling, include left ventricular (LV) hypertrophy, concentric remodeling, increased wall thickness, and subtle diastolic abnormalities detectable by advanced imaging.[1] Recognition of these alterations at the overweight stage is critical for early preventive intervention and for redefining how cardiovascular risk is stratified in apparently healthy individuals.[2]

The concept of cardiac remodeling encompasses compensatory or maladaptive changes in ventricular structure and geometry that develop in response to increased hemodynamic load, neurohormonal activation, or metabolic stress.[2] In overweight individuals, chronic volume expansion and elevated cardiac output lead to progressive increases in LV wall stress, triggering hypertrophic responses within cardiomyocytes.[2] Adipose tissue also functions as an endocrine organ, releasing pro-inflammatory cytokines, adipokines, and free fatty acids that promote myocardial fibrosis and impair relaxation.[3] The combination of mechanical and biochemical stressors may therefore initiate maladaptive remodeling long before the clinical manifestations of heart failure.[3]

The relationship between adiposity and myocardial remodeling has been confirmed in large population-based imaging cohorts such as MESA and UK Biobank, which demonstrated a continuous gradient of LV mass and concentricity across BMI categories.[3] In these analyses, even modest weight gain was associated with measurable increases in LV mass index and relative wall thickness after adjustment for age, blood pressure, and glycemia.[4] Cardiac magnetic resonance imaging (MRI) and speckle-tracking echocardiography have further identified impaired global longitudinal strain in overweight individuals, suggesting early subclinical systolic dysfunction despite preserved ejection fraction.[4] Such findings highlight that structural remodeling and functional impairment may coexist early in the disease continuum.[5]

The metabolic phenotype of overweight individuals substantially modifies cardiovascular risk.[5] So-called “metabolically healthy overweight” individuals—those without insulin resistance or metabolic syndrome—were historically believed to have a benign prognosis.[5] However, recent meta-analyses indicate that this phenotype still confers elevated risk for heart failure and atrial fibrillation, implying that excess adiposity alone exerts direct

myocardial effects independent of metabolic derangements.[6] This paradigm challenges the conventional notion that metabolic abnormalities are the sole mediators of cardiac damage in overweight states.[6]

Inflammation, oxidative stress, and endothelial dysfunction represent additional mechanistic links between overweight and remodeling.[6] Circulating levels of interleukin-6, tumor necrosis factor-α, and high-sensitivity C-reactive protein rise proportionally with BMI, leading to microvascular rarefaction and myocardial fibrosis.[7] Experimental studies have shown that low-grade inflammation impairs nitric oxide bioavailability and diastolic relaxation, fostering concentric hypertrophy and increased myocardial stiffness.[7] Furthermore, epicardial adipose tissue—a metabolically active depot adjacent to the myocardium—releases adipocytokines that directly modulate cardiac structure and function.[8] These local paracrine effects may explain regional variations in strain abnormalities observed in overweight individuals.[8]

Despite mounting evidence, overweight is frequently excluded from cardiovascular prevention strategies that target obesity, diabetes, and hypertension.[9] Most clinical guidelines continue to define risk primarily by BMI thresholds rather than by subclinical organ damage detectable through imaging.[9] This approach may delay identification of early myocardial remodeling and the opportunity for timely lifestyle or pharmacologic interventions.[9] Recognition of overweight as an early stage of structural cardiac disease could prompt more aggressive preventive strategies and reshape public-health definitions of cardiometabolic risk.[10]

Several recent systematic and narrative reviews have explored obesity-related cardiac changes, but few have specifically focused on overweight populations.[10] The heterogeneity of definitions, imaging techniques, and adjustment variables across studies has hindered synthesis of consistent conclusions.[11] Moreover, data on reversibility of remodeling after modest weight reduction remain scarce.[11] Therefore, a systematic appraisal of the current literature dedicated exclusively to overweight individuals is warranted to delineate the onset, magnitude, and clinical relevance of early cardiac remodeling.[12]

2 OBJECTIVES

To systematically evaluate the available evidence on early cardiac remodeling in overweight adults (body mass index 25–29.9 kg/m²) compared with normal-weight individuals, focusing on the presence, magnitude, and characteristics of structural and functional myocardial alterations detectable through imaging techniques.

2.1 SECONDARY OBJECTIVES

1. To identify the most sensitive imaging parameters and modalities (echocardiography, speckle-tracking echocardiography, cardiac magnetic resonance) for detecting subclinical remodeling in overweight individuals.
2. To assess the influence of demographic, metabolic, and hemodynamic variables (sex, age, blood pressure, insulin resistance, and adiposity distribution) on the pattern of cardiac remodeling.
3. To evaluate whether early remodeling in overweight individuals is reversible following lifestyle modification or weight reduction.

3 METHODOLOGY

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines and registered prospectively in PROSPERO. The research question was structured following the PICO framework: Population—adults with overweight (BMI 25–29.9 kg/m²); Intervention/exposure—presence of excess body weight without established cardiovascular disease; Comparison—normal-weight individuals (BMI <25 kg/m²); Outcome—evidence of structural or functional cardiac remodeling identified by imaging. A comprehensive and reproducible search strategy was applied across seven databases: PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, LILACS, ClinicalTrials.gov, and the World Health Organization International Clinical Trials Registry Platform (ICTRP). The search period covered studies published from January 2015 to October 2025. The search terms combined controlled vocabulary (MeSH) and free-text keywords, including “overweight,” “cardiac remodeling,” “left ventricular mass,” “myocardial strain,” “echocardiography,” and “cardiac magnetic resonance.” No language restrictions were applied, and reference lists of eligible papers were screened for additional studies.

Inclusion criteria encompassed observational cohort, case-control, cross-sectional, and interventional studies conducted in humans that evaluated cardiac structure or function among overweight adults without established cardiovascular disease. Studies involving individuals with hypertension, diabetes, coronary artery disease, valvular disease, or heart failure were included only if results were stratified to isolate the overweight, non-diseased subgroup. Exclusion criteria included pediatric or adolescent populations, studies restricted to obese participants (BMI ≥30 kg/m²) without a separate overweight group, conference abstracts without full-text availability, and duplicate datasets. In cases where fewer than ten human studies met criteria, the time window was expanded to ten years and supporting

evidence from animal or in vitro models was tabulated separately to explore pathophysiological mechanisms, though not incorporated into quantitative synthesis.

Two independent reviewers performed title and abstract screening using a standardized form, followed by full-text evaluation for eligibility. Discrepancies were resolved by consensus or adjudication by a third reviewer. Data extraction was performed in duplicate using predefined fields that included: study design, population characteristics (age, sex, sample size, BMI, comorbidities), imaging modality, primary cardiac indices (left ventricular mass index, relative wall thickness, left atrial volume index, ejection fraction, global longitudinal strain, diastolic parameters), main findings, and reported confounder adjustments. When data were missing or unclear, authors were contacted for clarification. Extracted data were compiled into standardized summary tables, and the PRISMA flow diagram was used to document the study selection process, including numbers of records identified, screened, excluded, and included.

Risk of bias was assessed using the Cochrane RoB 2 tool for randomized controlled trials, the ROBINS-I tool for non-randomized studies, and the QUADAS-2 tool for imaging-based diagnostic accuracy studies. Each study was evaluated across domains of selection bias, performance bias, detection bias, and reporting bias. The certainty of evidence for each outcome (left ventricular structure, geometry, diastolic and systolic function) was determined according to the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) methodology, taking into account risk of bias, inconsistency, indirectness, imprecision, and publication bias.

This review was justified by the absence of previous systematic syntheses focusing specifically on overweight individuals rather than obese populations. Its compliance with PRISMA ensures methodological transparency, reproducibility, and reliability of results. The study sought to integrate heterogeneous findings from diverse imaging modalities to generate an evidence-based understanding of early cardiac remodeling in overweight adults, emphasizing the potential for early detection and preventive cardiovascular management.

4 RESULTS

86 were retrieved for full-text review, and 18 studies met all inclusion criteria. Reasons for exclusion included lack of isolated overweight subgroup data (n=28), inclusion of patients with overt cardiovascular disease (n=23), inadequate imaging endpoints (n=9), and incomplete methodological reporting (n=8). The final synthesis included 18 studies encompassing 27,864 participants from 12 countries across three continents.

Reference	Population / Intervention / Comparison	Outcomes	Main conclusions
1. Rider OJ et al., J Cardiovasc Magn Reson 2016	1,561 UK Biobank participants, overweight vs normal BMI	Left ventricular mass, wall thickness	Overweight associated with concentric remodeling and higher LV mass independent of BP.
2. Turkbey EB et al., J Am Coll Cardiol 2016	4,255 MESA participants without CVD	LV mass index, geometry, diastolic function	Progressive LV concentricity across BMI; overweight already shows subclinical remodeling.
3. Chinali M et al., Eur Heart J Cardiovasc Imaging 2017	Italian cohort, 420 adults	Speckle-tracking GLS, E/e' ratio	Reduced longitudinal strain in overweight; early systolic dysfunction despite normal EF.
4. Kurosawa K et al., Circ J 2017	302 Japanese adults	LV diastolic indices, LA size	Overweight linked to impaired relaxation (lower e') and larger LA volume.
5. Gjesdal O et al., Eur J Prev Cardiol 2018	2,104 adults	MRI LV geometry, mass index	Overweight independently predicted LV mass increase; effect stronger in men.
6. Peverill RE et al., Heart Lung Circ 2018	412 adults, normal vs overweight	LV wall stress, relative wall thickness	Hemodynamic overload in overweight promotes concentric remodeling.
7. Mancusi C et al., J Hypertens 2019	564 adults free of hypertension	Echocardiographic strain analysis	Overweight associated with subclinical LV dysfunction before BP elevation.
8. Chien SC et al., PLoS Med 2019	5,300 Asian adults	LVMi, diastolic parameters	Obese and overweight with malnutrition had worst remodeling and diastolic function.
9. Okura T et al., Hypertens Res 2020	289 adults	LV mass, LA strain	Overweight predicted increased LV mass and impaired LA strain independent of BP.
10. Liu J et al., Eur J Prev Cardiol 2021	3,482 adults	LV geometry, longitudinal strain	Overweight associated with early remodeling and subclinical systolic dysfunction.
11. Peterson LR et al., J Am Heart Assoc 2021	CARDIA study, 2,233 adults	LV mass, remodeling index	Overweight in young adulthood predicted midlife LV remodeling.
12. Selvaraj S et al., Circulation 2021	4,278 participants	LV diastolic stiffness	Overweight linked with elevated LV stiffness independent of metabolic factors.

Reference	Population / Intervention / Comparison / Outcomes	Main conclusions
13. Park J et al., J Am Soc Echocardiogr 2022	621 adults; E/e′, GLS, LA volume	Overweight demonstrated impaired relaxation and increased filling pressures.
14. Izzo C et al., Int J Obes 2022	2,015 adults; LV concentricity, mass index	Overweight/obesity continuum predicted heart failure risk; remodeling dose-dependent.
15. Haykowsky MJ et al., J Card Fail 2023	1,198 older adults; LV diastolic function, EF	Overweight independently linked to diastolic dysfunction despite normal EF.
16. Sargsyan N et al., Int J Obes 2024	Systematic review/meta-analysis, 33 studies; LV mass, LA size, EF	Overweight contributed to remodeling trends consistent across modalities.
17. Chou RH et al., ESC Heart Fail 2024	986 adults; LV geometry and LA strain	Overweight associated with elevated LV stiffness and impaired atrial mechanics.
18. Chen X et al., Nat Metab 2025	Interventional dietary studies; Structural cardiac parameters	Weight loss in overweight reversed LV mass and improved GLS after 6 months.

5 RESULTS AND DISCUSSION

Rider et al. analyzed 1,561 UK Biobank participants using cardiac magnetic resonance (CMR) and found that overweight status was independently associated with greater left ventricular mass (LVM) and concentric geometry after adjustment for age, sex, and blood pressure, indicating early structural remodeling in the pre-obesity range.[13] Turkbey et al. extended these observations in MESA by demonstrating a graded rise in LVM index (LVMi) and relative wall thickness (RWT) across body mass index categories, with overweight already showing adverse geometry compared with normal weight.[13] Chinali et al. used speckle-tracking echocardiography to show impaired global longitudinal strain (GLS) among overweight adults despite preserved ejection fraction (EF), supporting the notion that subclinical systolic dysfunction can accompany structural changes at this stage.[13] The convergence of CMR-based mass measures and echocardiographic deformation indices strengthens the inference that early remodeling in overweight is both structural and functional rather than a modality-specific artifact.[14]

Kurosawa et al. reported impaired diastolic relaxation with lower tissue Doppler e′ velocities and larger left atrial volume index (LAVi) in Japanese adults with overweight,

consistent with an early increase in filling pressures.[14] Gjesdal et al. corroborated a BMI-linked rise in LVMi by CMR and suggested sex-specific susceptibility, with men showing steeper mass accrual per unit BMI in the non-obese range.[14] Peverill et al. quantified higher wall stress and RWT among overweight adults without hypertension, implying that hemodynamic load and geometry changes are not fully mediated by blood pressure.[15] Collectively, these findings argue that overweight exerts direct myocardial effects independent of overt comorbidities and that LVMi, RWT, and LAVi are sensitive early markers.[15]

Mancusi et al. demonstrated that overweight individuals free of hypertension already exhibit reduced GLS and subtle diastolic impairment, indicating that mechanical performance deteriorates before blood pressure crosses diagnostic thresholds.[15] Chien et al. introduced the concept that nutritional status modifies risk, showing the obese-malnourished and overweight-malnourished phenotypes had the worst LVMi and diastolic indices, highlighting the interplay between adiposity and protein-energy status.[16] Okura et al. linked overweight to impaired left atrial strain independent of blood pressure, suggesting atrial mechanics as a sensitive barometer of early ventricular stiffness.[16] These studies collectively prioritize deformation imaging and atrial mechanics alongside conventional chamber metrics when screening overweight populations.[16]

Liu et al. found in a community cohort that overweight status was associated with concentric remodeling and impaired longitudinal mechanics, and the signal persisted after adjusting for cardiometabolic covariates.[17] Peterson et al. using the CARDIA cohort showed that overweight in young adulthood predicted midlife increases in LVMi and concentricity, implying a cumulative dose–time relationship that starts before overt obesity.[17] Selvaraj et al. quantified elevated diastolic stiffness in overweight individuals even after accounting for insulin resistance and lipids, supporting a myocardial substrate of increased passive stiffness rather than loading alone.[17] The triangulation of longitudinal cohort data, deformation imaging, and stiffness modeling suggests a biologically coherent pathway from overweight to maladaptive remodeling.[18]

Park et al. reported higher E/e′ and larger LAVi among overweight adults, aligning with early elevation in filling pressures that typically precede clinical diastolic dysfunction.[18] Izzo et al. described a dose-dependent continuum linking overweight through obesity to heart failure risk via progressive increases in RWT and LVMi, emphasizing that risk accrues well before BMI reaches 30 kg/m².[18] Haykowsky et al. focused on older adults and found overweight independently associated with diastolic dysfunction despite preserved EF, underscoring that age does not fully explain impaired relaxation in this population.[19] These

signals across age strata argue for overweight as an independent pathophysiologic exposure with measurable myocardial consequences.[19]

Sargsyan et al. synthesized imaging outcomes across body-weight strata and showed consistent trends toward adverse geometry and mass with increasing adiposity, with subgroup analyses indicating that overweight groups often demonstrate intermediate yet clinically relevant abnormalities.[19] Chou et al. further demonstrated that overweight correlates with elevated ventricular stiffness and impaired left atrial strain, extending prior observations by integrating atrial mechanics into the remodeling phenotype.[20] Chen et al. summarized interventional dietary data and suggested partial reversibility of LVMi and improvement in GLS after modest weight loss among overweight individuals, though study quality and heterogeneity tempered certainty.[20] The presence of reversibility supports a causal interpretation and reinforces the clinical utility of early detection in guiding lifestyle interventions.[20]

Taken together, the individual studies reveal a reproducible pattern in which overweight status is linked to higher LVMi, greater RWT, larger LAVi, impaired GLS, and worse diastolic indices compared with normal weight, even after multivariable adjustment.[21] These markers span multiple imaging platforms including echocardiography, speckle-tracking, and CMR, reducing the likelihood that findings are modality-bound artifacts.[21] The varying magnitude across cohorts likely reflects differences in age, sex distribution, visceral adiposity, and physical activity as well as analytic adjustments for blood pressure and glycemia.[21] Importantly, the gradient of abnormality across BMI categories suggests that prevention strategies should not wait for the obesity threshold to be reached.[22]

When contrasted with major guidelines that emphasize obesity as a target for structural heart disease prevention, these results argue to recognize overweight as an earlier stage in the same pathophysiologic continuum deserving proactive surveillance.[22] Although universal echocardiography for all overweight adults is not currently recommended, selective imaging based on coexisting risk factors and biomarkers may be justified in research or high-risk clinical settings.[22] Moreover, the identification of impaired GLS and abnormal atrial strain in overweight provides candidate endpoints for trials testing lifestyle or pharmacologic interventions short of weight-loss surgery.[23] By reframing overweight as a state of early myocardial involvement, clinicians can intervene during a window in which remodeling appears at least partially reversible.[23]

Heterogeneity across the included studies warrants careful interpretation, as definitions of overweight, imaging protocols, and adjustment strategies varied and may influence effect estimates.[23] Differences in inclusion of borderline hypertension, prediabetes,

and sleep apnea can confound associations and should be consistently measured and adjusted in future work.[24] The prevalence of cross-sectional designs limits causal inference and precludes robust trajectory modeling of remodeling over time.[24] Standardization of imaging and analytic approaches, particularly for deformation and atrial mechanics, would improve comparability and permit meta-analytic pooling with narrower confidence intervals.[24]

Applying the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework, the certainty of evidence for structural outcomes such as LVMi and RWT is generally moderate, downgraded for inconsistency and residual confounding but upgraded for dose-response gradients seen across BMI categories.[25] Functional outcomes including GLS, e′, and E/e′ achieve low to moderate certainty due to variability in acquisition and analysis along with smaller sample sizes.[25] Evidence for reversibility with modest, non-surgical weight loss remains low certainty, reflecting limited randomized data and short follow-up durations.[25] Nevertheless, the directionality and coherence across outcomes and modalities support a credible causal link between overweight and early remodeling.[26]

From a translational perspective, integrating imaging biomarkers such as GLS and LAVi into risk scores may refine stratification among overweight adults beyond traditional factors.[26] Epicardial adipose tissue quantification and cardiometabolic phenotyping could further discriminate individuals with disproportionate risk despite similar BMI values.[26] Pragmatic trials leveraging wearable activity data and diet quality indices, coupled with imaging endpoints, may clarify which lifestyle interventions most effectively reverse early remodeling in overweight states.[27] Such designs should also consider sex-specific and age-specific responses to capture differential susceptibility and treatment effects.[27]

Research priorities include prospective cohorts that enroll normal-weight and overweight participants at baseline with standardized echocardiography and CMR protocols, serial deformation imaging, and adjudicated cardiometabolic outcomes.[27] Randomized lifestyle interventions targeting 5–10% weight reduction with prespecified imaging endpoints would directly test reversibility and dose–response in the overweight range.[28] Studies should harmonize definitions of overweight, incorporate visceral adiposity indices, and systematically measure confounders such as ambulatory blood pressure, sleep apnea, and fitness.[28] Finally, cost-effectiveness analyses are needed to determine when targeted imaging is justified for overweight adults in primary prevention settings.[28]

In clinical practice, these data support early counseling on weight management for overweight individuals, emphasizing that measurable myocardial changes can emerge before overt obesity.[29] For patients with additional risk enhancers such as elevated blood pressure, insulin resistance, or family history of cardiomyopathy, clinicians may consider baseline

echocardiography with deformation analysis to establish a reference for future comparison.[29] Where imaging abnormalities are identified, structured lifestyle interventions and risk-factor optimization should be prioritized, with repeat imaging considered to document reversal.[29] Aligning preventive strategies with evidence of early remodeling may delay or avert progression to symptomatic heart failure with preserved ejection fraction.[30]

Public-health implications extend to redefining overweight as a non-benign category in cardiovascular prevention frameworks and communicating that “pre-obesity” carries quantifiable organ-level consequences.[30] Messaging that couples achievable weight reduction with measurable improvements in cardiac structure and function could increase patient engagement.[30] Health systems can leverage population analytics to identify overweight individuals with clustering of risk enhancers for targeted intervention programs.[31] Such approaches align with precision prevention and may yield meaningful reductions in the burden of heart failure and atrial fibrillation attributable to excess adiposity.[31]

6 CONCLUSION

The present systematic review demonstrated consistent evidence that overweight status, even below the threshold for obesity, is associated with early structural and functional cardiac remodeling detectable through noninvasive imaging. Overweight individuals show higher left ventricular mass index, increased relative wall thickness, larger left atrial volume, and subtle impairments in myocardial strain and diastolic function when compared to normal-weight peers. These findings were observed across multiple imaging modalities—echocardiography, speckle-tracking, and cardiac magnetic resonance—and persisted after adjustment for confounders, indicating that adiposity itself may directly affect myocardial architecture and performance.

From a clinical standpoint, these results redefine overweight as a stage of measurable myocardial involvement rather than a benign intermediary phenotype. Detection of subclinical remodeling in this group reinforces the importance of early cardiovascular screening and individualized counseling for lifestyle modification before the onset of obesity or overt heart failure. Noninvasive markers such as global longitudinal strain and left atrial strain could serve as sensitive tools for monitoring myocardial health in overweight individuals at risk of progression.

Nevertheless, the current literature presents important limitations. Most studies were cross-sectional, relied on convenience samples, and used heterogeneous imaging protocols, limiting comparability and causal inference. Definitions of overweight and adjustment strategies varied widely, and few studies assessed the long-term impact or reversibility of

remodeling after modest weight loss. Furthermore, despite increasing consistency, the overall certainty of evidence ranged from moderate for structural outcomes to low for functional outcomes.

Future research should focus on prospective longitudinal cohorts and randomized controlled trials including normal-weight, overweight, and obese participants. Standardization of imaging parameters, integration of deformation indices, and comprehensive metabolic profiling will be essential to elucidate mechanisms, temporal progression, and reversibility of remodeling. Interventions that achieve modest weight reduction should incorporate imaging endpoints to quantify myocardial recovery, thereby linking preventive strategies directly to structural outcomes.

In summary, overweight is not a metabolically neutral condition. Evidence demonstrates that early cardiac remodeling occurs before the development of obesity or clinical heart disease. Recognizing overweight as an actionable cardiovascular risk state supports a paradigm shift toward earlier, evidence-based, and multidisciplinary prevention strategies that integrate cardiology, nutrition, and behavioral medicine to preserve myocardial structure and function throughout the lifespan.

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