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Review Article

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Waist Circumference as a Cardiometabolic Vital Sign: A Translational Framework for Early Detection and Prevention of Heart Failure with Preserved Ejection Fraction in Primary Care

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Abstract

Background: Heart failure with preserved ejection fraction (HFpEF) is a common obesity-related complication that often develops before symptoms. Body mass index (BMI) poorly captures visceral adiposity, the metabolically active fat linked to cardiometabolic dysfunction, myocardial remodeling, and diastolic dysfunction. Waist circumference (WC) is a simple, inexpensive surrogate but remains underused.

Objectives: This review summarizes evidence linking central adiposity to left ventricular diastolic dysfunction (LVDD) and HFpEF and proposes a practical framework for integrating WC into routine vitals to enable earlier risk stratification and prevention.

Evidence summary: The Baltimore Longitudinal Study of Aging (BLSA) showed that WC predicts diastolic dysfunction independent of BMI and traditional risk factors. Epidemiologic, imaging, and mechanistic studies support visceral and ectopic adiposity as drivers of inflammation, fibrosis, endothelial dysfunction, impaired relaxation, and cardiac remodeling. Trials of glucagon-like peptide-1 (GLP-1) receptor agonists and dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonists suggest that reducing visceral adiposity improves cardiometabolic and functional outcomes in obesity-related HFpEF.

Translational framework: We propose WC as a cardiometabolic vital sign and introduce the waist-to-heart translational framework, integrating routine WC measurement with cardiometabolic assessment, symptom review, HFpEF risk scores, biomarkers, and targeted echocardiography to identify HFpEF in primary care clinics.

Conclusion: Central adiposity is a stronger determinant of obesity-related cardiac dysfunction than BMI alone and is an actionable prevention target. Routine WC measurement offers a scalable, low-cost strategy to improve risk stratification. Prospective validation is needed to assess clinical impact and implementation.

Keywords

Obesity medicine, Waist circumference, Central adiposity, Visceral adiposity, Heart failure with preserved ejection fraction, Diastolic dysfunction, Cardiovascular risk stratification

Introduction

Obesity, central adiposity, and HFpEF risk

Obesity medicine is shifting from weight reduction alone toward prevention of cardiometabolic organ dysfunction [1]. HFpEF is a major obesity-related

complication that often develops after years of subclinical myocardial remodeling, creating an opportunity for earlier recognition and intervention [2-7]. BMI remains useful for classification but poorly reflects body composition, fat distribution, and individual cardiometabolic risk [8-11]. Patients with similar BMI may have markedly different visceral fat burdens, while patients with normal BMI but increased central adiposity may still have unrecognized cardiovascular risk [8-12].

WC as a cardiometabolic vital sign

WC is a simple, inexpensive, and reproducible surrogate for visceral adiposity [10, 12]. Visceral and ectopic fat act as biologically active endocrine depots, releasing inflammatory mediators, adipokines, and free fatty acids that promote insulin resistance, endothelial dysfunction, oxidative stress, neurohormonal activation, myocardial fibrosis, microvascular dysfunction, impaired relaxation, and ventricular stiffening [6, 8-11, 13-16]. These mechanisms support WC as a clinically meaningful marker of cardiometabolic risk, not merely an anthropometric measure [8-11].

Evidence and Clinical Gap

LVDD is an early manifestation of obesity-related myocardial injury and often precedes symptomatic HFpEF by years [3-7]. The BLSA showed that WC independently predicted LVDD after adjustment for BMI and traditional cardiovascular risk factors, supporting central adiposity as a stronger marker of early myocardial dysfunction than body weight alone [25]. Subsequent cohorts, imaging studies, and consensus statements have reinforced visceral and ectopic fat as contributors to myocardial remodeling and HFpEF progression [6, 8-11, 14, 15].

Purpose of This Review

Despite strong evidence, WC remains inconsistently measured and is often absent from cardiovascular screening pathways. Many patients are identified only after HFpEF symptoms and structural myocardial changes are established, whereas primary care and obesity medicine clinics often see patients earlier. We propose WC as a cardiometabolic vital sign and introduce the waist-to-heart translational framework, which integrates routine measurement with structured cardiovascular risk assessment to identify patients at risk for stage A and stage B HFpEF before symptoms develop.

Pathophysiology Linking Visceral Adiposity to HFpEF

HFpEF is increasingly viewed as a systemic cardiometabolic syndrome rather than an isolated cardiac disorder [5-7, 13]. Obesity, especially central adiposity, promotes metabolic, inflammatory, neurohormonal, and hemodynamic pathways that impair myocardial relaxation and ventricular compliance. Unlike heart failure with reduced ejection fraction, HFpEF often develops gradually through chronic inflammation, endothelial dysfunction, fibrosis, and ventricular stiffening, with visceral adipose tissue serving as a biologically active driver [6, 8-11, 13].

Compared with subcutaneous fat, visceral adipose tissue secretes pro-inflammatory cytokines, adipokines, free fatty acids, and neurohormonal mediators while reducing protective adiponectin. This imbalance promotes inflammation, oxidative stress, insulin resistance, endothelial dysfunction, renin-angiotensin-aldosterone system (RAAS) and sympathetic activation, and myocardial injury beyond the effect of body weight alone [6, 8-11, 13].

Endothelial dysfunction is a central downstream pathway. Inflammatory signaling reduces nitric oxide bioavailability and impairs cGMP–protein kinase G (PKG) signaling, leading to titin hypophosphorylation, cardiomyocyte stiffness, impaired relaxation, fibroblast activation, collagen deposition, and interstitial fibrosis. These changes reduce ventricular compliance and establish LVDD before systolic function declines [3-6, 13].

Visceral adiposity also disrupts myocardial energy metabolism through excess free fatty acid flux, myocardial lipid accumulation, mitochondrial dysfunction, oxidative stress, impaired calcium handling, and reduced contractile reserve, contributing to exertional intolerance despite preserved resting ejection fraction [5, 6, 16].

Epicardial adipose tissue further amplifies HFpEF risk through local inflammation, coronary microvascular dysfunction, atrial remodeling, ventricular fibrosis, impaired filling, and mechanical constraint. Imaging studies show that epicardial fat correlates with diastolic dysfunction and HFpEF severity independent of BMI, underscoring the importance of fat distribution [14, 15].

Hemodynamic stress and comorbidities intensify these mechanisms. Visceral adiposity increases blood volume, cardiac output, RAAS activation, sympathetic tone, arterial stiffness, and LV afterload, while hypertension, diabetes, chronic kidney disease, sleep apnea, and atrial fibrillation accelerate progression from asymptomatic diastolic dysfunction to overt HFpEF [5, 6, 13, 17, 18].

Sex-specific biology may further modify risk, particularly after menopause, when visceral fat redistribution is associated with inflammation, endothelial dysfunction, arterial stiffness, and fibrosis. These changes may help explain stronger associations between WC and LVDD in women.

Importantly, these pathways may be partially reversible. Lifestyle intervention, bariatric surgery, GLP-1 receptor agonists, and dual GIP/GLP-1 receptor agonists reduce visceral adiposity and improve inflammation, endothelial function, exercise capacity, and quality of life, suggesting that earlier intervention before irreversible fibrosis may alter HFpEF trajectory [1, 19-24].

Overall, visceral adiposity is a biologically active driver of myocardial remodeling and HFpEF development, not merely a marker of obesity. Because WC is a practical surrogate for visceral fat, routine measurement may help identify high-risk patients earlier and support the proposed waist-to-heart translational framework (Figure 1).

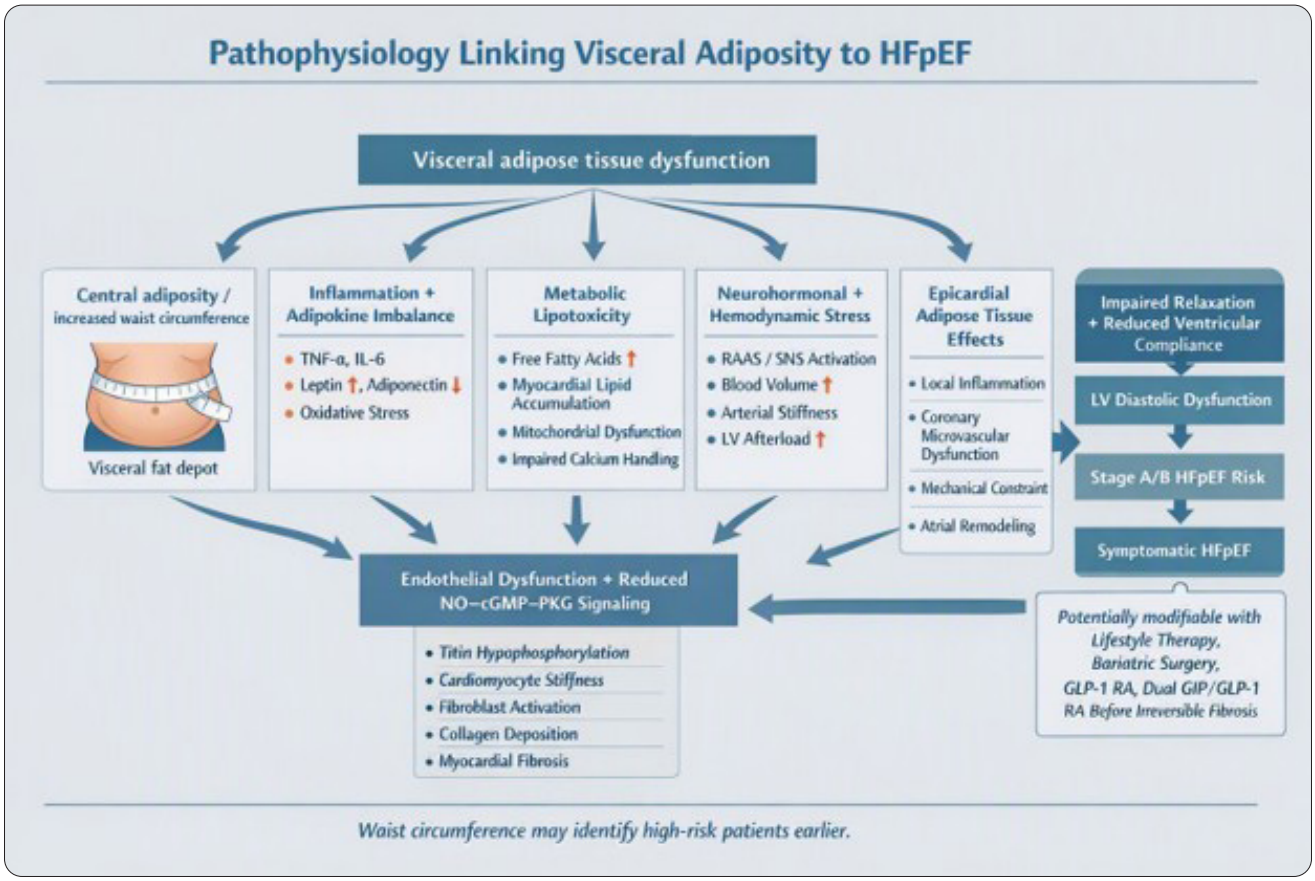


Figure 1: Pathophysiology linking visceral adiposity to HFpEF.

Evidence Linking Central Adiposity to Diastolic Dysfunction and HFpEF: Beyond the BLSA

Evidence linking central adiposity to diastolic dysfunction and HFpEF has expanded well beyond the BLSA. In the BLSA, WC independently predicted LVDD after adjustment for BMI and traditional cardiovascular risk factors, supporting fat distribution as a stronger marker of subclinical myocardial dysfunction than body weight alone (Table 1). Subsequent cohorts, including Multi-ethnic Study of Atherosclerosis (MESA) and the Framingham Heart Study, confirmed that visceral adiposity is associated with adverse cardiac remodeling, metabolic dysfunction, inflammation, insulin resistance, and cardiovascular events [8, 9, 11]. Advanced imaging studies further show that visceral and ectopic fat depots, particularly epicardial adipose tissue, are linked to myocardial inflammation, coronary microvascular dysfunction, ventricular stiffening, elevated filling pressures, and HFpEF severity [6, 14, 15]. Mechanistic data suggest that visceral adiposity drives inflammatory signaling, reduced nitric oxide–cGMP–PKG activity, titin hypophosphorylation, cardiomyocyte stiffness, and interstitial fibrosis [6, 13]. Contemporary trials of GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists, including semaglutide treatment effect on people with obesity (STEP)-HFpEF and study of tirzepatide in participants with HEpEF and obesity (SUMMIT), demonstrate that targeting obesity and visceral adiposity can improve symptoms, function, inflammatory markers, and clinical outcomes in obesity-related HFpEF [19-24]. Although WC-guided screening requires prospective validation, current epidemiologic, imaging, mechanistic, guideline, and interventional evidence supports WC as a practical surrogate for visceral adiposity and a clinically useful tool for early cardiometabolic risk stratification within the proposed waist-to-heart translational framework [1, 10, 12, 17, 18].

The Waist-to-Heart Workflow: A Clinical Framework for Early HFpEF Detection

Obesity medicine and primary care often encounter patients before symptomatic cardiovascular disease develops, creating an opportunity to identify subclinical myocardial dysfunction while it may still be modifiable [1, 5-7]. Yet despite evidence linking visceral adiposity with LVDD, obesity-specific measures such as WC are not routinely integrated into cardiovascular screening [10, 12, 25]. Current HFpEF algorithms are usually applied after symptoms appear, thereby missing a window for prevention in patients with central adiposity [5, 7, 17, 18].

The waist-to-heart workflow is a pragmatic, evidence-informed framework that treats WC as a cardiometabolic vital sign. It complements existing guidelines by combining anthropometric assessment, cardiometabolic risk profiling, symptom review, HFpEF risk scores, selective biomarkers, echocardiography, and multidisciplinary management to identify high-risk patients earlier in obesity medicine and primary care settings (Figure 2).

Table 1: Evidence linking central adiposity to diastolic dysfunction and HFpEF.

Study/Source	Population/Design	Adiposity measure	Key finding	Clinical implication
BLSA	Community-dwelling adults	WC; BMI	WC predicted LVDD independent of BMI and traditional risk factors	Supports WC as a stronger marker of early myocardial dysfunction than BMI alone
MESA	Multi-ethnic observational cohort	Computed tomography (CT)-derived visceral fat	Visceral adiposity was associated with adverse cardiac remodeling and cardiovascular risk	Highlights visceral fat as a marker of subclinical cardiovascular disease
Framingham heart study	Community-based cohort	CT-derived visceral fat	Visceral fat correlated with inflammation, metabolic dysfunction, and cardiovascular events	Reinforces fat distribution as more informative than body weight alone
STEP-HFpEF	Patients with obesity-related HFpEF	Body weight and visceral adiposity reduction	Semaglutide improved symptoms, exercise capacity, and quality of life	Supports obesity treatment as disease-modifying in obesity-related HFpEF
SUMMIT	Patients with obesity-associated HFpEF	Weight and metabolic improvement	Tirzepatide improved functional status and reduced HF-related events	Supports early, aggressive obesity-directed therapy in HFpEF care

Discussion

The rising burden of obesity has made HFpEF a major cardiometabolic complication, yet early myocardial dysfunction often remains undetected until symptoms develop [2-7]. Because LVDD may precede overt heart failure by years, central adiposity-not BMI alone-represents an important target for earlier risk stratification and prevention [3, 4, 10, 25]. Evidence from the BLSA and subsequent epidemiologic, imaging, mechanistic, and interventional studies links WC and visceral adiposity to LVDD, inflammation, endothelial dysfunction, myocardial fibrosis, coronary microvascular dysfunction, and ventricular stiffening [6, 13-16, 25]. These findings support obesity as a biologically active driver of myocardial remodeling and identify visceral adiposity as a modifiable therapeutic target [8-11, 19-24].

Obesity medicine and primary care are well positioned for upstream cardiovascular prevention because patients often present before cardiology referral and before myocardial changes become irreversible. Routine WC measurement is inexpensive, reproducible, and clinically actionable, functioning as a cardiometabolic vital sign that can prompt structured cardiovascular evaluation. The proposed waist-to-heart translational framework bridges evidence and practice by combining WC with cardiometabolic phenotyping, HFpEF risk tools, selective biomarkers, and targeted echocardiography to identify stage A and stage B HFpEF risk earlier. Rather than replacing guidelines, it moves risk assessment upstream into settings where prevention can begin sooner.

Earlier identification is increasingly actionable because GLP-1 receptor agonists, dual GIP/GLP-1 receptor agonists, lifestyle therapy, and metabolic bariatric surgery can reduce visceral adiposity and improve inflammation, metabolic health, exercise capacity, and quality of life; STEP-HFpEF and SUMMIT suggest that treating obesity can improve outcomes in obesity-related HFpEF [1, 19-24]. The framework is also practical, requiring minimal equipment and fitting into routine workflows with electronic health record support [10, 12]. Important limitations remain: most evidence is observational, WC is technique-dependent and does not distinguish visceral from subcutaneous fat, and prospective studies are needed to determine whether WC-guided screening improves detection, management, outcomes, and cost-effectiveness across diverse populations [7, 10, 12].

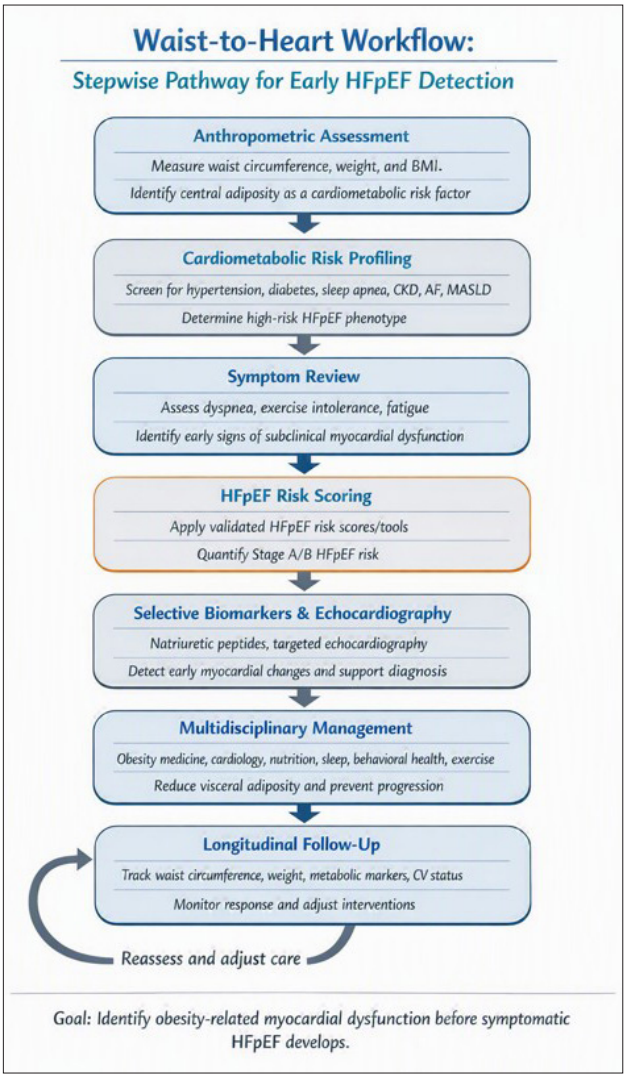


Figure 2: Waist-to-heart workflow.

Overall, WC should be viewed as a practical marker of

cardiometabolic organ risk. Integrating it into structured risk assessment may help obesity medicine and primary care identify HFpEF risk earlier and shift care from treatment of established disease toward prevention of obesity-related cardiac dysfunction.

Clinical Pearls: WC and Early HFpEF Prevention

Move beyond BMI: BMI remains useful for classification, but WC better reflects central adiposity and adds clinically meaningful cardiometabolic risk information.

Treat WC as a cardiometabolic vital sign: Measure WC routinely in patients with overweight or obesity and track it longitudinally alongside weight, BMI, blood pressure, and metabolic markers.

Recognize visceral adiposity as biologically active: Visceral and ectopic fat promote inflammation, endothelial dysfunction, insulin resistance, myocardial fibrosis, and impaired ventricular relaxation.

Screen earlier in high-risk phenotypes: Patients with elevated WC plus dyspnea, exercise intolerance, hypertension, diabetes, sleep apnea, chronic kidney disease, atrial fibrillation, or metabolic dysfunction-associated steatotic liver disease may warrant structured HFpEF risk assessment.

Use risk tools selectively: HFpEF scores, natriuretic peptides, and targeted echocardiography can help identify stage A or stage B HFpEF before symptomatic disease develops.

Intervene upstream: Lifestyle therapy, exercise, sleep optimization, cardiometabolic risk-factor control, GLP-1 receptor agonists, dual GIP/GLP-1 receptor agonists, and bariatric surgery when appropriate may reduce visceral adiposity and improve HFpEF-related outcomes.

Build multidisciplinary pathways: Obesity medicine, primary care, cardiology, nutrition, sleep medicine, behavioral health, and exercise specialists can coordinate care before irreversible myocardial remodeling occurs.

Validate implementation: Prospective studies are needed to determine whether WC-guided screening improves detection, treatment decisions, outcomes, and cost-effectiveness.

Limitations

Several limitations should be considered. The waist-to-heart translational framework is an evidence-informed clinical model, not a validated diagnostic or management guideline. Most evidence linking central adiposity with LVDD and HFpEF is observational, so causality cannot be established and residual confounding may persist [3, 4, 8, 9, 25]. WC is an imperfect surrogate for visceral adiposity because it cannot distinguish visceral from subcutaneous fat, depends on standardized technique, and requires interpretation by sex, ethnicity, age, and clinical context [10, 12]. Biomarkers also require caution, as natriuretic peptide levels may be lower in patients with obesity despite clinically meaningful myocardial dysfunction [5, 7]. Prospective studies are needed to determine whether WC-guided screening improves detection, management, outcomes, feasibility, and cost-effectiveness. Despite these limitations, converging epidemiologic, mechanistic, imaging, and interventional evidence supports WC as a practical marker of cardiometabolic risk and a hypothesis-generating tool for earlier identification of obesity-related myocardial dysfunction before symptomatic HFpEF develops [13-15, 19-25].

Conclusion

The growing burden of obesity-related cardiovascular disease requires a shift from treating established heart failure to identifying risk earlier. Converging epidemiologic, mechanistic, imaging, and clinical trial evidence shows that central adiposity is a stronger marker of myocardial dysfunction and HFpEF risk than BMI alone. As a simple, low-cost, and reproducible surrogate for visceral adiposity, WC adds meaningful information to routine cardiometabolic assessment and may function as a cardiometabolic vital sign. The proposed waist-to-heart translational framework integrates WC with cardiometabolic phenotyping, HFpEF risk assessment, selective biomarkers, targeted echocardiography, and multidisciplinary obesity care to identify stage A and stage B HFpEF risk before irreversible myocardial remodeling occurs. Prospective validation is needed to determine whether WC-guided screening improves detection, outcomes, healthcare utilization, and cost-effectiveness, but this approach offers a practical pathway for linking obesity care with cardiovascular prevention before symptomatic HFpEF develops.

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None.

Conflict of Interest

None.

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