Association Between Cardiometabolic Index and Congestive Heart Failure: A Cross-Sectional Observational Study

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Abstract: ***Background and Objective:*** ***Congestive heart failure (CHF) is a major cause of morbidity and mortality. The Cardiometabolic Index (CMI), derived from waist-to-height ratio and TG/HDL-C ratio, integrates adiposity and dyslipidemia. We evaluated the association between CMI and CHF severity.*** ***Methods:*** ***A hospital-based cross-sectional study enrolled 132 confirmed CHF patients. CMI = WHtR × (TG/HDL-C). Correlation and multivariate regression analyses assessed associations with heart failure severity.*** ***Results:*** ***Mean age 58.6±12.4 years; 62.1% male. CMI was highest in patients with EF <40% (2.74±0.98) vs. EF ≥50% (1.67±0.58; p<0.001). CMI inversely correlated with LVEF (r=−0.42, p<0.001) and positively with NYHA class (r=0.39, p<0.001). CMI independently predicted severe CHF (OR:2.31; 95%CI:1.42–3.76; p=0.001).*** ***Conclusion:*** ***Elevated CMI is significantly associated with CHF severity and reduced LVEF. It is a simple, cost-effective marker for risk stratification in CHF patients.***

Keywords: Cardiometabolic Index, Congestive Heart Failure, Ejection Fraction, Metabolic Syndrome, Cardiovascular Risk

1. Introduction

Congestive heart failure (CHF) is a complex and progressive clinical syndrome resulting from structural or functional impairment of ventricular filling or ejection of blood. It represents one of the most significant challenges in contemporary medicine, contributing substantially to hospitalizations, reduced quality of life, and premature mortality across all age groups. Globally, the prevalence of heart failure is estimated to exceed 64 million individuals, and despite remarkable advances in pharmacological and device-based therapies, the five-year mortality remains unacceptably high, approaching 50% in many cohorts. [1,2]

The pathogenesis of CHF is multifactorial, with cardiometabolic risk factors playing an increasingly recognized role. Obesity, dyslipidemia, insulin resistance, hypertension, and central adiposity are not merely comorbidities but active contributors to myocardial injury, ventricular remodeling, and neurohormonal dysregulation. Traditional anthropometric measures such as body mass index (BMI) provide limited information on fat distribution and fail to capture visceral adiposity, which is more strongly associated with cardiovascular risk than total body fat. [3,5]

The Cardiometabolic Index (CMI) is a recently proposed composite marker that integrates waist-to-height ratio (WHtR)- a validated measure of central adiposity- with the triglyceride-to-HDL cholesterol ratio (TG/HDL-C), which reflects atherogenic dyslipidemia and serves as a surrogate for insulin resistance. By combining these two dimensions of cardiometabolic risk, CMI offers a more comprehensive metabolic profile than either component alone. Prior investigations have demonstrated significant associations between elevated CMI and hypertension, type 2 diabetes mellitus, metabolic syndrome, non-alcoholic fatty liver disease, and atherosclerotic cardiovascular disease. [4,8] [9] Mechanistically, excess visceral adiposity and atherogenic dyslipidemia promote chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and activation of the renin-angiotensin-aldosterone system- all pathways implicated in cardiac remodeling and heart failure progression. [7,10] Despite this growing body of evidence, data specifically examining CMI in relation to CHF severity and ejection fraction subtypes remain sparse, particularly from South Asian hospital-based populations. Identifying a practical, inexpensive marker that integrates adiposity and dyslipidemia could substantially improve early risk stratification and guide allocation of intensive therapeutic strategies in CHF management. The present study was therefore undertaken to evaluate the association between Cardiometabolic Index and congestive heart failure, with specific focus on left ventricular ejection fraction, NYHA functional class, and the independent predictive value of CMI after adjustment for conventional risk factors.

2. Materials and Methods

Study Design and Population

This was a hospital-based cross-sectional observational study conducted in the Department of General Medicine at a tertiary care teaching hospital over a duration of 12 months. A total of 132 patients with a confirmed diagnosis of congestive heart failure were enrolled after obtaining written informed consent. The diagnosis of CHF was established based on clinical criteria (symptoms and signs of heart failure) corroborated by echocardiographic evidence of structural or functional cardiac abnormality, in accordance with the 2023 ESC Guidelines for heart failure. [1]

Inclusion and Exclusion Criteria

Patients aged 18 years and above with confirmed CHF who consented to participate were included. Patients with acute myocardial infarction within the preceding three months, chronic liver disease, end-stage renal disease (eGFR <15 mL/min/1.73m²), active malignancy, or pregnancy were excluded to minimize confounding on metabolic parameters.

Data Collection and Measurements

Detailed demographic information including age and sex was recorded. Anthropometric measurements- height, weight, and waist circumference—were obtained using standardized techniques. Body mass index was calculated as weight (kg) divided by height squared (m²). Waist circumference was measured at the midpoint between the lower costal margin and iliac crest. Blood pressure was recorded using a calibrated sphygmomanometer after five minutes of rest. Fasting venous blood samples were drawn for lipid profile including triglycerides and HDL cholesterol. Echocardiographic assessment of left ventricular ejection fraction (LVEF) was performed by a trained cardiologist using the modified Simpson's biplane method. Patients were classified by LVEF into three categories: preserved (EF ≥50%), mildly reduced (EF 40–49%), and reduced (EF <40%). NYHA functional class was assigned based on clinical evaluation at the time of enrollment.

Calculation of CMI: CMI was calculated using the following validated formula:

$$\text{WHtR} = \text{Waist Circumference (cm)} / \text{Height (cm)}$$

$$\mathbf{CMI = WHtR \times (Triglycerides / HDL\text{-}C)}$$

Statistical Analysis

All statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequency and percentage. Intergroup comparisons were performed using Student's independent t-test for two groups and one-way ANOVA for three or more groups. Pearson correlation coefficient was used to examine associations between CMI and continuous clinical parameters. Multivariate binary logistic regression was employed to identify independent predictors of severe heart failure (defined as LVEF <40% or NYHA class III–IV), after adjusting for age, sex, and BMI as potential confounders. A two-tailed p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

The study was approved by the Institutional Ethics Committee (IEC) in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants. Patient confidentiality was strictly maintained throughout the study period. No investigations were ordered solely for research purposes; all data were collected from routine clinical assessments performed as part of standard patient care.

3. Results

The study enrolled 132 patients with confirmed CHF. The mean age of participants was 58.6±12.4 years, reflecting a predominantly middle-aged to older adult population consistent with the epidemiology of heart failure in India. Males constituted 62.1% (n=82) and females 37.9% (n=50) of the cohort, with a male preponderance mirroring prior Indian heart failure registries. The mean BMI was 27.3±4.8 kg/m², placing the average participant in the overweight category. Mean waist circumference was elevated at 96.2±10.5 cm, exceeding the South Asian-specific threshold of 90 cm for men and 80 cm for women, indicative of widespread central adiposity in this cohort. Mean triglycerides were 182.5±62.4 mg/dL and mean HDL-C was low at 39.6±9.1 mg/dL, together reflecting a highly atherogenic lipid profile. The overall mean CMI of the study population was 2.18±0.91, which is substantially higher than CMI values reported in healthy control populations. Complete baseline characteristics are summarized in Table-I.

Table I: Baseline Characteristics (N=132)

Variable	Value
Sample size	132
Age (years)	58.6±12.4
Male, n (%)	82 (62.1)
BMI (kg/m²)	27.3±4.8
Waist Circumference (cm)	96.2±10.5
Triglycerides (mg/dL)	182.5±62.4
HDL-C (mg/dL)	39.6±9.1
Mean CMI	2.18±0.91

CMI values showed a stepwise and statistically significant increase across worsening ejection fraction categories (p<0.001). Patients with EF ≥50% (preserved, n=46, 34.8%) had the lowest mean CMI of 1.67±0.58, followed by those with EF 40–49% (mildly reduced, n=33, 25.0%) at 2.11±0.74, and patients with EF <40% (reduced, n=53, 40.2%) recorded the highest CMI of 2.74±0.98. The magnitude of difference in CMI between preserved and reduced EF groups was clinically meaningful (Δ=1.07 CMI units), and the graded relationship across all three categories underscores a progressive cardiometabolic burden paralleling worsening systolic function. This pattern was consistent across both sexes on subgroup analysis (Table-II).

Table II: CMI by Ejection Fraction Category

EF Category	n (%)	Mean CMI	p-value
EF ≥50%	46 (34.8)	1.67±0.58	—
EF 40–49%	33 (25.0)	2.11±0.74	—
EF <40%	53 (40.2)	2.74±0.98	<0.001

Pearson correlation analysis revealed a significant inverse correlation between CMI and left ventricular ejection fraction ($r=-0.42$, $p<0.001$), indicating that higher CMI values are associated with lower systolic function. The moderate strength of this correlation is noteworthy given the multifactorial determinants of LVEF. CMI also demonstrated a significant positive correlation with NYHA functional class ($r=0.39$, $p<0.001$), confirming that greater cardiometabolic burden corresponds to worse functional limitation and symptom severity. Additionally, CMI correlated positively with BMI ($r=0.44$, $p<0.001$) and most strongly with waist circumference ($r=0.57$, $p<0.001$), highlighting that the abdominal obesity component of CMI drives much of its cardiometabolic signal. All correlations were statistically highly significant (Table-III).

Table-III: Correlation of CMI with Clinical Parameters

Parameter	r	p-value
Ejection Fraction	−0.42	<0.001
NYHA Class	0.39	<0.001
BMI	0.44	<0.001
Waist Circumference	0.57	<0.001

Multivariate binary logistic regression analysis was performed with severe heart failure as the outcome variable, defined as LVEF <40% or NYHA class III–IV. After adjusting for age, sex, and BMI as potential confounders, CMI remained a statistically significant and independent predictor of severe heart failure with an odds ratio of 2.31 (95% CI: 1.42–3.76; p=0.001). For every one-unit increase in CMI, the odds of severe heart failure increased by 2.31-fold, a clinically meaningful effect size. Age was also an independent predictor (OR:1.04; 95%CI:1.01–1.08; p=0.02), consistent with the age-related increase in heart failure risk. In contrast, sex (OR:1.22; p=0.54) and BMI (OR:1.08; p=0.11) did not reach statistical significance, powerfully illustrating that CMI provides cardiometabolic risk information beyond what BMI alone captures (Table-IV).

Table IV: Multivariate Logistic Regression Analysis

Variable	OR	95% CI	p
CMI	2.31	1.42–3.76	0.001
Age	1.04	1.01–1.08	0.02
Male Sex	1.22	0.64–2.34	0.54
BMI	1.08	0.98–1.19	0.11

4. Discussion

The present study examined the relationship between Cardiometabolic Index and congestive heart failure severity in a cohort of 132 hospitalized patients. The central finding was a robust and statistically significant association between elevated CMI and both reduced left ventricular ejection fraction and higher NYHA functional class. This association persisted after adjustment for traditional cardiovascular risk factors on multivariate analysis, underscoring the independent prognostic contribution of CMI in this population. The stepwise gradient in CMI values across ejection fraction categories—from 1.67±0.58 in preserved EF to 2.74±0.98 in reduced EF- is particularly compelling and suggests a dose-response relationship between cardiometabolic burden and cardiac dysfunction.

The pathophysiological basis for this relationship is multifaceted. Central adiposity, as reflected by WHtR, is associated with increased pericardial fat deposition, which mechanically constrains ventricular filling, promotes diastolic dysfunction, and elevates intracardiac pressures. [5,7] Excess visceral adipose tissue further functions as an active endocrine organ, secreting pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and leptin while suppressing cardioprotective adiponectin. These cytokines activate nuclear factor-kappa B (NF-κB) signaling pathways, driving myocardial fibrosis, cardiomyocyte apoptosis, and accelerating adverse cardiac remodeling. The resulting interstitial fibrosis impairs both systolic contractility and diastolic relaxation, explaining the spectrum of CMI elevation observed across both HFrEF and HFpEF phenotypes in this study. [10]

The TG/HDL-C ratio, the lipid component of CMI, serves as a reliable surrogate marker for insulin resistance and atherogenic small dense LDL particles. Elevated TG/HDL-C reflects impaired lipoprotein metabolism and increased hepatic VLDL secretion, both of which contribute to coronary atherosclerosis, endothelial dysfunction, myocardial ischemia, and ultimately impaired cardiac contractility. Insulin resistance, of which TG/HDL-C is a validated proxy, independently promotes cardiac steatosis, mitochondrial dysfunction, and impaired glucose utilization by cardiomyocytes- a metabolic substrate shift that reduces myocardial energetic efficiency and accelerates heart failure progression. In our study, the strong correlation between CMI and waist circumference ($r=0.57$) compared with BMI ($r=0.44$) highlights the superior contribution of visceral fat distribution over total body adiposity in capturing cardiometabolic risk. [4,8]

Our findings are consistent with and extend those of Liu et al. (2024), who, using data from the National Health and Nutrition Examination Survey (NHANES), demonstrated that CMI was independently and significantly associated with prevalent heart failure after comprehensive multivariable adjustment. [9] The external validity of that large population-based study lends credibility to our hospital-based observations. Similarly, Thorp and Filipp (2024) provided mechanistic underpinning by demonstrating that inflammatory pathways driven by visceral adiposity are central to the development and progression of heart failure with preserved ejection fraction- a subtype increasingly recognized as a cardiometabolic disorder rather than a purely hemodynamic one. [10]

Borlaug et al. (2023) further emphasized in a landmark JACC state-of-the-art review that HFpEF is fundamentally a systemic metabolic disease, where adipose tissue inflammation, skeletal muscle dysfunction, renal sodium retention, and microvascular rarefaction collectively impair cardiac reserve capacity and exercise tolerance. [11] CMI, by simultaneously capturing visceral adiposity and atherogenic dyslipidemia, may serve as an integrated index of this multisystem cardiometabolic phenotype. The independent predictive value of CMI over BMI in our regression analysis (OR:2.31 for CMI vs. OR:1.08 for BMI, the latter non-significant at p=0.11) is particularly noteworthy and directly supports this framework—suggesting that the combination of abdominal fat distribution and lipid dysregulation captured by CMI provides clinically meaningful information beyond what BMI alone can offer.

Comparisons with prior literature on related markers are also instructive. Studies examining waist-to-height ratio alone have reported significant associations with incident cardiovascular disease and heart failure hospitalization; similarly, elevated TG/HDL-C ratio has been linked to coronary artery disease severity and left ventricular hypertrophy. CMI, as a product of these two measures, theoretically captures synergistic cardiometabolic risk that neither index fully conveys independently. Our correlation data support this, with CMI demonstrating stronger associations with both LVEF and NYHA class than would be expected from either anthropometric or lipid measures alone. This synergistic value positions CMI as a candidate screening tool for cardiometabolic risk assessment in outpatient cardiac clinics and general medicine wards. [4,6]

From a clinical standpoint, the 2024 ESC focused update on heart failure management increasingly emphasizes addressing modifiable cardiometabolic risk factors- including obesity, dyslipidemia, and diabetes- as integral components of heart failure prevention and treatment strategies. [12] Emerging pharmacological therapies such as sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists, which demonstrably reduce cardiovascular mortality in heart failure, act partly through favorable modification of the cardiometabolic milieu. Routine CMI assessment could identify patients most likely to benefit from such cardiometabolic therapies and help monitor their therapeutic response. CMI, requiring only a waist measurement and a standard lipid profile, is ideally suited for routine clinical use, particularly in resource-constrained settings where natriuretic peptide assays or advanced cardiac imaging may be unavailable.

Several limitations of this study warrant acknowledgment. The cross-sectional design precludes establishment of causality or temporal relationships between CMI and heart failure severity. As a single-center study conducted at a tertiary care facility, selection bias may limit generalizability to community-dwelling populations. Dietary intake, physical activity levels, medication use (particularly statins, fibrates, and antidiabetic agents that influence lipid profiles), and the duration of heart failure were not systematically captured and may represent residual confounders. Furthermore, CMI was calculated at a single time point; longitudinal changes in CMI and their relationship to heart failure outcomes remain unexplored. Future prospective, multicenter studies incorporating serial CMI measurements alongside hard clinical endpoints—including heart failure hospitalization rates, need for device therapy, and all-cause mortality—are needed to fully establish the prognostic utility and optimal cut-off values of CMI in CHF.

5. Conclusion

Cardiometabolic Index is significantly and independently associated with congestive heart failure severity and reduced left ventricular ejection fraction. A stepwise increase in CMI across worsening ejection fraction categories, combined with its inverse correlation with LVEF and positive correlation with NYHA functional class, collectively supports the role of integrated cardiometabolic burden in the pathogenesis and progression of CHF. On multivariate regression, CMI independently predicted severe heart failure after adjustment for age, sex, and BMI, surpassing the predictive value of BMI alone and affirming its incremental clinical utility. Given that CMI is simple, inexpensive, and calculable at the bedside from routinely available clinical data, it represents a practical and valuable tool for cardiometabolic risk stratification in CHF patients. Routine incorporation of CMI into clinical assessment protocols- particularly in resource-limited settings- could meaningfully enhance early identification of high-risk individuals, guide therapeutic decision-making, and potentially improve outcomes in this vulnerable population.

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