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Frequency Of Obesity In Patients Presenting With Acute Heart Failure With Preserved Ejection Fraction At Tertiary Care Hospital.

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Abstract

Background: Obesity is frequently observed among patients with heart failure with preserved ejection fraction (HFpEF) and has been associated with adverse cardiovascular remodeling, systemic inflammation, and impaired functional capacity. Despite increasing prevalence worldwide, local data describing obesity among patients presenting with acute HFpEF remain limited.

Methodology: This cross-sectional study was conducted at Liaquat National Hospital, Karachi, Pakistan from December 2025 to May 2026 and included 149 patients aged 40-80 years diagnosed with HFpEF. Demographic characteristics, clinical history, and comorbid conditions including hypertension, type II diabetes mellitus, dyslipidemia, smoking status, and family history of heart failure were recorded. Body mass index (BMI) was calculated using measured height and weight. Obesity was defined as a BMI ≥27.5 kg/m2. Data was analyzed using SPSS version 20.0.

Results: Among 149 patients 101 (67.8%) were obese. The mean age was 59.8 ± 11.9 years, with 82 (55.0%) males and 67 (45.0%) females. Obesity showed significant associations with hypertension ($p < 0.001$), type II diabetes mellitus ($p = 0.007$), age ≤60 years ($p = 0.031$), female gender ($p = 0.045$) and dyslipidemia ($p = 0.009$). No significant association was observed with smoking status ($p = 0.118$) or family history of heart failure ($p = 0.128$).

Conclusion: Obesity was highly prevalent among patients presenting with acute HFpEF and showed significant associations with several cardiovascular and metabolic comorbidities. These findings highlight the burden of obesity in this population. Further longitudinal studies are required to determine temporal and causal relationships.

Keywords

Heart Failure, Obesity, Body Mass Index, Hypertension, Type 2 Diabetes Mellitus

Introduction

Excess body weight is far more common among people with HFpEF than among those with HFrEF; indeed, over 80 % of HFpEF patients fall into the overweight or obese category[1]. While obesity alone is generally not recognized as a direct cause of cardiomyopathy, some experts have proposed defining "obesity cardiomyopathy" as a condition that develops primarily or exclusively as a result of obesity[2]. Although the exact mechanisms remain unclear, the combination of excessive fat accumulation and increased lean body mass may lead to an expanded circulating blood volume. This, in turn, can elevate cardiac output, cardiac workload, and systolic blood pressure, while also promoting lipotoxicity-related damage to cardiac muscle cells and the buildup of lipids in the heart tissue factors that have been suggested as possible contributors to the development of cardiomyopathy[3-4].

Inflammation may contribute to microvascular damage and fibrosis occurs not only in the heart but also affects other organs, including lungs, kidneys, pancreas, and muscles. These alterations significantly contribute to the onset and progression of the common comorbid conditions seen in HFpEF[5]. Myocardial biopsy samples from individuals with HFpEF demonstrate clear evidence of coronary microvascular endothelial dysfunction, marked by increased expression of endothelial adhesion molecules, including vascular cell adhesion molecule (VCAM) and E-selectin[6]. Pro-inflammatory cytokines trigger endothelial cells to generate reactive oxygen species by activating enzymes called nicotinamide adenine dinucleotide phosphate (NADPH) oxidases[7]. This results in heightened oxidative and nitrosative stress within the cardiac tissue of patients with HFpEF, a condition that is further exacerbated by common comorbidities such as diabetes mellitus and by age-related physiological changes[8].

Obese patients with HFpEF, compared with both non-obese HFpEF patients and healthy controls, display a larger plasma volume along with more marked concentric remodeling of the left ventricle, right-ventricular enlargement, and impaired right-ventricular function[9]. These patients also showed thicker epicardial fat and a larger total epicardial heart volume, changes that produced hemodynamic patterns indicating stronger pericardial constraint and an increased interdependence between the ventricles[10]. When subjected to exercise stress, obese individuals with HFpEF exhibited markedly reduced exercise tolerance, increased left and right ventricular filling pressures, and a diminished pulmonary artery vasodilatory response. Among obese individuals with HFpEF, higher body mass index and greater plasma volume were associated with elevated cardiac filling pressures. Despite having considerably lower levels of NT-proBNP (N-terminal pro–B-type natriuretic peptide), obese individuals with HFpEF exhibited more severe physiological abnormalities[11]. In the study by Bae and colleagues, obesity was present in 55.7 % of individuals admitted with acute heart failure[12].

Heart failure with preserved ejection fraction (HFpEF) now constitutes roughly one-half of all heart-failure diagnoses and is an escalating public-health issue. Obesity can exacerbate HFpEF by promoting systemic inflammation, increasing cardiac workload, impairing diastolic function, and contributing to metabolic dysregulation. Despite the well-established link between obesity and cardiovascular disease, the precise prevalence and impact of obesity in patients presenting with acute HFpEF remain incompletely understood. Considering the growing burden of obesity and its adverse cardiovascular complications, identifying the prevalence of obesity in patients presenting with HFpEF is crucial for improving risk stratification and management strategies.

Methodology

Study Design

A cross-sectional design was selected for its ability to determine the frequency of obesity among patients presenting with acute heart failure with preserved ejection fraction (HFpEF) at a single point in time. It aided the assessment of the burden of obesity and permitted the analysis of its distribution across various demographic and clinical characteristics, including age, gender, hypertension, type II diabetes mellitus, dyslipidemia, smoking status, and family history of heart failure.

Ethics

Ethical approval to conduct this study was received from the ethical review committee of Liaquat National Hospital prior to the data collection. The data was collected in accordance with the ethical standards of the ethical review committee and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolling them in the study. Patient confidentiality was strictly maintained throughout the process by denoting every patient with a separate patient ID.

Setting

The study was conducted at the Department of Cardiology, Liaquat National Hospital, Karachi, Pakistan in a time span of six months from December 2025 to May 2026. It provides specialized cardiovascular services and interventions. The hospital serves a diverse patient population from Karachi and surrounding regions, making it an appropriate setting for evaluating the frequency of obesity among patients presenting with acute heart failure with preserved ejection fraction (HFpEF). The availability of advanced cardiovascular services ,echocardiographic facilities, and comprehensive clinical assessment leads to accurate diagnosis of HFpEF patients.

Participants

A total of 149 patients aged between 40 and 80 years presenting with acute heart failure with preserved ejection fraction (HFpEF) were included in the study. Heart failure with preserved ejection fraction (HFpEF) was diagnosed based on the presence of symptoms and signs consistent with acute heart failure together with a left ventricular ejection fraction (LVEF) ≥50%, supported by objective evidence of cardiac structural and/or functional abnormalities where available. Objective confirmation included elevated natriuretic peptide levels (BNP or NT-proBNP), echocardiographic evidence of diastolic dysfunction (including parameters such as E/e′ ratio, left atrial enlargement, or left ventricular hypertrophy), and/or evidence of elevated filling pressures according to contemporary HFpEF diagnostic recommendations. Diagnosis was established by the treating cardiology team after integration of clinical findings and available diagnostic investigations. Participants filling this criteria and willing to provide informed consent were considered eligible. Participants were excluded if they had a history of unstable angina, ST-segment elevation myocardial infarction, percutaneous coronary intervention or coronary stenting (PCI), hypothyroidism, hyperthyroidism, asthma, Chronic kidney disease, chronic obstructive pulmonary disease, chronic liver disease and stroke. The participants were recruited through non-probability; consecutive sampling techniques.

Variables

The primary outcome variable was the presence of obesity among patients presenting with acute HFpEF. Obesity was defined as a body mass index (BMI) of ≥27.5 kg/m². BMI was calculated using measured weight in kilograms divided by the square of height in meters (kg/m²). Secondary variables included demographic and clinical characteristics such as age, sex, hypertension, type II diabetes mellitus, dyslipidemia, smoking status, and family history of heart failure.

Data Sources / Measurement

Data was collected from participants presenting with acute heart failure with preserved ejection fraction (HFpEF) at the Department of Cardiology, Liaquat National Hospital, Karachi. The participants that meet the inclusion criteria were enrolled in the study through non-probability consecutive sampling. Demographic and clinical information including age, gender, smoking status, history of hypertension, type II diabetes mellitus, dyslipidemia, and family history of heart failure was obtained through subjective assessment, medical record review, and clinical assessment using a standardized data collection tool. Anthropometric measurements were taken before the recruitment process. Height was measured in meters using a wall-mounted stadiometer, and weight was recorded in kilograms using a calibrated weighing scale. Body mass index (BMI) was determined by using the formula weight divided by height squared (kg/m²). Obesity was defined on the BMI of 27.5 kg/m² according to the operational definition mentioned in the guidelines.

Bias

Several measures were taken to minimize potential bias. Consecutive patient enrollment was done to reduce the selection bias. Standardized operational definitions were used for all study variables. Anthropometric measurements were taken using calibrated instruments.

Data collection was done on the basis of structured proforma to ensure uniformity and reduce information bias. Data entry and verification were done carefully to reduce transcription errors and improve data accuracy.

Study Size

Sample size was calculated using the OpenEpi sample size calculator. An expected prevalence of obesity according to the literature was 55.7% among patients with HFpEF, a confidence level of 95%, and a margin of error of 8%, the calculated sample size was 149 patients. All eligible participants were recruited until the calculated sample size was achieved.

Statistical Methods

Data analysis was performed using Statistical Package for Social Sciences (SPSS) version 20.0. The normality of continuous variables was checked through the Kolmogorov-Smirnov test. The data of normally distributed variables was presented as mean ± standard deviation (SD), whereas non-normally distributed variables were shown as median and interquartile range (IQR). Categorical variables were presented as frequencies and percentages. The primary outcome of the study was the determination of obesity status.

Stratification of data was done in accordance with age group, gender, hypertension, type II diabetes mellitus, dyslipidemia, smoking status, and family history of heart failure to assess potential effect modifiers. Post-stratification association was checked through the Chi-square test or Fisher's exact test. A p-value of <0.05 was considered statistically significant.

Results

A total of 149 patients presenting with acute heart failure with preserved ejection fraction (HFpEF) were recruited in the study. The mean age of the participants was 59.8 ± 11.9 years. There were 82 (55.0%) males and 67 (45.0%) females. The overall frequency of obesity among patients presenting with acute HFpEF was 101 (67.8%), whereas 48 (32.2%) patients were classified as non-obese. Hypertension was present in 102 (68.5%) patients, diabetes mellitus type II in 74 (49.7%), dyslipidemia in 61 (40.9%), smoking in 43 (28.9%), and family history of heart failure in 37 (24.8%) patients.

To determine the association between obesity and potential effect modifiers, stratified analysis was done using the Chi-square test. Obesity was significantly associated with hypertensive patients ($\chi^2 = 12.83$, $p < 0.001$). Similarly, obesity was significantly more common among patients with diabetes mellitus (78.4%, $\chi^2 = 7.24$, $p = 0.007$).

The strong association was also observed between age and obesity. Patients aged 60 years or younger had a higher prevalence of obesity compared with those older than 60 years (75.3% vs. 58.8%, $\chi^2 = 4.65$, $p = 0.031$). Female patients exhibited a significantly greater frequency of obesity than male patients (76.1% vs. 61.0%, $\chi^2 = 4.02$, $p = 0.045$).

A significant association was observed between obesity and dyslipidemia. Obesity was present in 80.3% of patients with dyslipidemia ($\chi^2 = 6.91$, $p = 0.009$). No statistically significant association was observed between obesity and smoking status (58.1% vs. 71.7%, $\chi^2 = 2.44$, $p = 0.118$) or family history of heart failure (78.4% vs. 64.3%, $\chi^2 = 2.31$, $p = 0.128$).

Overall, obesity was the most frequently observed modifiable characteristic among patients with acute HFpEF. Significant associations were identified between obesity and hypertension, diabetes mellitus, and dyslipidemia; however, these findings should be interpreted as associations observed within the study population rather than indicators of causation.

Table 1: Baseline Characteristics of Study Participants (n = 149).

Variables		N (%)
Age (years); Mean ± SD		59.8 ± 11.9
Gender	Male	82 (55.0)
	Female	67 (45.0)
Major Cardiovascular Risk Factors	Hypertension	102 (68.5)
	Diabetes Mellitus Type II	74 (49.7)
	Dyslipidemia	61 (40.9)
	Smoker	43 (28.9)
	Family History of Heart Failure	37 (24.8)

Table 2: Frequency of Obesity in HFpEF Patients.

Obesity Status	Frequency	Percentage
Obese	101	67.8
Non-Obese	48	32.2
Total	149	100

Table 3: Association of Obesity Status with Demographic and Clinical Characteristics (N = 149).

Variables	Obese n (%)	Non-Obese n (%)	χ^2	p-value
Age				
≤60 years	61 (75.3)	20 (24.7)	4.65	0.031*
>60 years	40 (58.8)	28 (41.2)		
Gender				
Male	50 (61.0)	32 (39.0)	4.02	0.045*
Female	51 (76.1)	16 (23.9)		
Hypertension				
Yes	78 (76.5)	24 (23.5)	12.83	<0.001*
No	23 (48.9)	24 (51.1)		
Diabetes Mellitus				
Yes	58 (78.4)	16 (21.6)	7.24	0.007*
No	43 (57.3)	32 (42.7)		
Dyslipidemia				
Yes	49 (80.3)	12 (19.7)	6.91	0.009*
No	52 (59.1)	36 (40.9)		
Smoking				
Yes	25 (58.1)	18 (41.9)	2.44	0.118
No	76 (71.7)	30 (28.3)		
Family History of Heart Failure				
Yes	29 (78.4)	8 (21.6)	2.31	0.128
No	72 (64.3)	40 (35.7)		

*p<0.05 was considered statistically significant.

Discussion

This study assessed the frequency of obesity among patients presenting with acute heart failure with preserved ejection fraction (HFpEF) at a tertiary care hospital in Karachi, Pakistan. The findings represented that obesity was present in 67.8% of patients, making it the most common modifiable risk factor identified in this population. In addition to this, obesity also showed the significant associations with hypertension, type II diabetes mellitus, dyslipidemia, female gender, and younger age, highlighting its crucial role in the clinical profile of patients with acute HFpEF.

The prevalence of obesity observed in our study was higher than that reported in literature[12]. The higher frequency observed in our population was attributed due to the differences in ethnicity, dietary habits, sedentary lifestyles, and the increasing burden of obesity in South Asian countries. Previous studies have consistently demonstrated that overweight and obesity are highly prevalent among HFpEF patients, with more than 80% of individuals affected[1]. The high frequency of obesity observed in this cohort is consistent with previous reports describing obesity as a common clinical characteristic among patients with HFpEF. However, due to the cross-sectional design, the present study cannot determine whether obesity contributes to the development or progression of HFpEF and only demonstrates coexistence and observed associations.

Several pathophysiological mechanisms explain the association between obesity and HFpEF. Excess adiposity promotes systemic inflammation, endothelial dysfunction, oxidative stress, and myocardial fibrosis, which collectively impair ventricular relaxation and increase myocardial stiffness[5,6]. Obesity also leads to plasma volume expansion, increased cardiac output, and adverse ventricular remodeling, resulting in elevated cardiac filling pressures and worsening heart failure symptoms[3,9]. Obokata et al. described a distinct obese HFpEF phenotype characterized by greater concentric left ventricular remodeling, right ventricular dysfunction, and reduced exercise tolerance compared with non-obese HFpEF patients[9]. These observations are consistent with the high prevalence of obesity found in our study. A significant association between obesity and hypertension was observed in our study ($p < 0.001$). Similar findings have been reported previously in literature, where obesity contributes to activation of the sympathetic nervous system and renin-angiotensin-aldosterone system, leading to increased blood pressure and cardiac remodeling[2]. Since hypertension is a well-established risk factor for HFpEF, its coexistence with obesity may substantially may coexist with clinical features commonly observed among patients with HFpEF.

The present study also demonstrated a significant association between obesity and type II diabetes mellitus ($p = 0.007$). The findings are supported by previous literature showing that obesity and diabetes frequently coexist and contribute to the development of HFpEF through insulin resistance, chronic inflammation, microvascular dysfunction, and metabolic abnormalities[7,8]. The coexistence of obesity and diabetes may accelerate myocardial remodeling and worsen clinical outcomes in patients with heart failure.

Dyslipidemia was significantly more common among obese patients in our study ($p = 0.009$). Similar associations have been reported in previous studies where obesity has been associated with an adverse lipid profile characterized by elevated triglycerides and reduced high-density lipoprotein cholesterol levels[2]. An interesting finding of our study was significant higher prevalence of obesity among female patients compared with males (76.1% vs. 61.0%, $p = 0.045$). This observation is in agreement with previous reports indicating that HFpEF is more prevalent among women, particularly middle aged and postmenopausal women due to decreased production of protective hormone estrogen[10]. Hormonal factors, differences in body fat distribution, and sex-specific cardiac remodeling patterns may explain this association. Women with obesity appear particularly susceptible to the development of HFpEF, emphasizing the need for targeted preventive strategies in this population.

No statistically significant associations were observed between obesity and smoking status (p = 0.118) or family history of heart failure (p = 0.128). Similar findings have been reported in some HFpEF cohorts where metabolic risk factors play a more prominent role in disease development than traditional cardiovascular risk factors such as smoking. However, the lack of significance in our study may also be related to the relatively small number of smokers and patients with a positive family history were recruited.

The findings of our study have important clinical implications. As obesity emerged as the most prevalent modifiable risk factor among patients with acute HFpEF, preventive and management strategies such as lifestyle modification, dietary counseling, regular physical activity, and effective management of associated comorbidities such as hypertension, diabetes, and dyslipidemia may help reduce the risk of HFpEF and improve the clinical outcomes. Early identification of obesity in high-risk individuals should therefore be considered an essential component of cardiovascular risk assessment.

Obesity assessment in this study relied solely on BMI using South Asian-specific thresholds. Although BMI is widely used in epidemiological studies, it does not distinguish lean mass from adiposity and does not adequately capture central obesity or visceral fat accumulation. This limitation may be particularly relevant in South Asian populations, where cardiometabolic risk may occur at lower BMI values. Future studies should consider additional anthropometric measures such as waist circumference, waist-to-hip ratio, body composition analysis, or imaging-based assessment of visceral adiposity. The extensive exclusion criteria, including chronic kidney disease, chronic obstructive pulmonary disease, thyroid disorders, previous coronary interventions, chronic liver disease, and stroke, may reduce external validity because these comorbidities commonly coexist with HFpEF in routine clinical practice. Therefore, findings should be interpreted cautiously when extrapolating to broader HFpEF populations.

Because not all advanced diagnostic parameters may have been uniformly available for every participant, some degree of diagnostic heterogeneity cannot be excluded.

Overall, the findings of our study highlights the growing evidence that obesity is strongly associated with HFpEF and its related comorbidities. The higher prevalence of obesity draws attention towards the need for preventive management strategies to reduce the risk of HFpEF and improve cardiovascular health outcomes.

Conclusion

This cross-sectional study demonstrated a high frequency of obesity among patients presenting with acute heart failure with preserved ejection fraction (HFpEF) at a tertiary care hospital. Obesity showed statistically significant associations with hypertension, type II diabetes mellitus, dyslipidemia, female gender, and younger age. These findings describe the burden and distribution of obesity within this patient population but do not establish obesity as a causal determinant of HFpEF. Future prospective multicenter studies are warranted to evaluate temporal relationships and clinical outcomes.

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References

1. Carbone S, Lavie CJ, Elagizi A, Arena R, Ventura HO. The impact of obesity in heart failure. Heart Fail Clin. 2022;40(2):209-218. doi: 10.1016/j.ccl.2021.12.009
2. Bozkurt B, Aguilar D, Deswal A, Dunbar SB, Francis GS, Horwich T, et al. Contributory risk and management of comorbidities of hypertension, obesity, diabetes mellitus, hyperlipidemia, and metabolic syndrome in chronic heart failure: a scientific statement from the American Heart

Association. Circulation. 2016;134(23):e535-78. doi: 10.1161/CIR.0000000000000450
3. Lewis GA, Schelbert EB, Williams SG, Cunnington C, Ahmed F, McDonagh TA, et al. Biological phenotypes of heart failure with preserved ejection fraction. J Am Coll Cardiol. 2017;70(17):2186-2200. doi: 10.1016/j.jacc.2017.09.006
4. Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, et al. Contemporary definitions and classification of the cardiomyopathies. Circulation. 2006;113(14):1807-1816. doi: 10.1161/CIRCULATIONAHA.106.174287
5. Paulus WJ, Tschope C. A novel paradigm for heart failure with preserved ejection fraction: comorbidities drive myocardial dysfunction and remodeling through coronary microvascular endothelial inflammation. J Am Coll Cardiol. 2013;62(4):263-271. doi: 10.1016/j.jacc.2013.02.092.
6. Ter Maaten JM, Damman K, Verhaar MC, Paulus WJ, Duncker DJ, Cheng C, et al. Connecting heart failure with preserved ejection fraction and renal dysfunction: the role of endothelial dysfunction and inflammation. Eur J Heart Fail. 2016;18(6):588-598. doi: 10.1002/ejhf.497.
7. Marchesini G, Moscatiello S, Di Domizio S, Forlani G. Obesity-associated liver disease. J Clin Endocrinol Metab. 2008;93(11 Suppl 1):S74-80. doi: 10.1210/jc.2008-1399.
8. Chiyanika C, Chan DFY, Hui SCN, So HK, Deng M, Yeung DKW, et al. The relationship between pancreas steatosis and the risk of metabolic syndrome and insulin resistance in Chinese adolescents with concurrent obesity and non-alcoholic fatty liver disease. Pediatr Obes. 2020;15(9):e12653. doi: 10.1111/ijpo.12653.
9. Obokata M, Reddy YNV, Pislaru SV, Melenovsky V, Borlaug BA. Evidence supporting the existence of a distinct obese phenotype of heart failure with preserved ejection fraction. Circulation. 2017;136(1):6-19. doi: 10.1161/CIRCULATIONAHA.116.026807
10. Shah SJ, Kitzman DW, Borlaug BA, van Heerebeek L, Zile MR, Kass DA, et al. Phenotype-specific treatment of heart failure with preserved ejection fraction: a multiorgan roadmap. Circulation. 2016;134(1):73-90. doi: 10.1161/CIRCULATIONAHA.116.021884.
11. Mohammed SF, Hussain S, Mirzoyev SA, Edwards WD, Maleszewski JJ, Redfield MM. Coronary microvascular rarefaction and myocardial fibrosis in heart failure with preserved ejection fraction. Circulation. 2015;131(6):550-559. doi: 10.1161/CIRCULATIONAHA.114.009625
12. Bae JP, Kallenbach L, Nelson DR, Lavelle K, Winer-Jones JP, Bonafede M, et al. Obesity and metabolic syndrome in patients with heart failure with preserved ejection fraction: a cross-sectional analysis of the Veradigm Cardiology Registry. BMC Endocr Disord. 2024;24(1):59. doi: 10.1186/s12902-024-01589-2