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Original Article

Sonographic Findings of Myocardial Infarction in Obese Hypertensive and Obese Non-Hypertensive Patients on Echocardiography

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ABSTRACT

Background: Obesity and hypertension are major cardiovascular risk factors that may worsen myocardial remodeling and functional impairment after myocardial infarction. Echocardiography provides a non-invasive method for assessing structural and functional cardiac abnormalities in high-risk patients. Objective: To compare echocardiographic findings between obese hypertensive and obese non-hypertensive patients with myocardial infarction. Methods: This cross-sectional observational study included 103 obese patients with myocardial infarction who underwent echocardiographic assessment at Combined Military Hospital, Lahore. Participants were categorized as hypertensive or non-hypertensive, and echocardiographic abnormalities were analyzed across hypertension status, age group, and gender using frequency distributions, cross-tabulation, chi-square tests, and effect-size estimates. Results: Of 103 participants, 54 (52.4%) were hypertensive and 49 (47.6%) were non-hypertensive; 69 (67.0%) were male and 34 (33.0%) were female. The most frequent echocardiographic findings were left ventricular hypertrophy in 17 participants (16.5%), mitral regurgitation in 16 (15.5%), and increased left ventricular wall thickness in 12 (11.7%). The overall distribution of echocardiographic findings did not differ significantly by hypertension status, $\chi^2(11) = 16.550$, $p = 0.122$, or gender, $p = 0.662$. Age group was significantly associated with gender distribution, $p = 0.018$. Conclusion: Obese patients with myocardial infarction demonstrated a notable burden of echocardiographic abnormalities, particularly left ventricular hypertrophy, mitral regurgitation, and increased left ventricular wall thickness. Hypertensive patients showed selected numerical differences, but overall echocardiographic patterns were not statistically different from non-hypertensive patients. Keywords: obesity, hypertension, myocardial infarction, echocardiography, left ventricular hypertrophy, cardiac remodeling.

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INTRODUCTION

Myocardial infarction remains one of the leading clinical manifestations of ischemic heart disease and occurs when an interruption in coronary blood flow produces myocardial ischemia, cellular injury, and, if prolonged, irreversible myocardial necrosis. The clinical presentation may include chest discomfort, dyspnea, diaphoresis, nausea, vomiting, palpitations, anxiety, or radiation of pain to the arm, shoulder, jaw, or neck; however, a considerable proportion of patients may present atypically or with silent myocardial injury, particularly in the presence of metabolic and cardiovascular risk factors. Conventional diagnosis relies on clinical assessment, electrocardiographic changes, and cardiac

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biomarkers such as troponin and creatine kinase-MB, whereas imaging modalities are important for evaluating the structural and functional consequences of ischemic myocardial damage (1).

Obesity is a major cardiometabolic risk factor that contributes to coronary artery disease, myocardial infarction, ventricular remodeling, and adverse cardiovascular outcomes. Excess adiposity promotes hemodynamic overload, systemic inflammation, insulin resistance, dyslipidemia, endothelial dysfunction, and atherosclerotic progression, all of which increase myocardial oxygen demand while impairing coronary perfusion. A meta-analysis evaluating the incidence of acute myocardial infarction in relation to excess body weight reported that overweight and obesity are associated with increased risk of acute myocardial infarction, emphasizing the clinical importance of obesity as a modifiable cardiovascular risk factor (2). In obese individuals, myocardial injury may therefore reflect both macrovascular mechanisms, such as coronary atherosclerosis and infarction, and microvascular or metabolic mechanisms that contribute to ventricular dysfunction and remodeling.

Hypertension further amplifies cardiovascular risk in obese patients by increasing afterload, promoting left ventricular hypertrophy, accelerating atherosclerotic plaque development, and worsening myocardial oxygen supply-demand imbalance. In patients with obesity and hypertension, persistent pressure overload may lead to increased left ventricular wall thickness, elevated left ventricular mass, impaired diastolic relaxation, atrial and ventricular remodeling, and valvular functional abnormalities. Echocardiographic assessment has therefore become central to identifying cardiac damage in hypertensive obese patients because it allows non-invasive evaluation of chamber size, wall thickness, systolic and diastolic function, ventricular remodeling, and associated valvular regurgitation (3). These changes are clinically important because structural remodeling may precede overt heart failure and may worsen prognosis after myocardial infarction.

The relationship between obesity, hypertension, and post-infarction cardiac remodeling is complex. Obesity-related comorbidities, including hypertension, diabetes, sleep apnea, and lipid abnormalities, may contribute to myocardial dysfunction through overlapping pathways involving volume overload, pressure overload, coronary macrovascular disease, and microvascular dysfunction. These mechanisms may result in systolic impairment after myocardial infarction, diastolic dysfunction, atrial remodeling, and eventual heart failure development (4). Because obesity and hypertension frequently coexist, it is clinically relevant to determine whether obese hypertensive patients with myocardial infarction demonstrate a different echocardiographic abnormality profile compared with obese non-hypertensive patients.

Echocardiography provides a safe, accessible, and non-invasive method for assessing myocardial structure and function in patients with cardiovascular disease. Standard two-dimensional echocardiography, Doppler imaging, color Doppler, and advanced echocardiographic techniques can identify abnormalities such as left ventricular hypertrophy, increased wall thickness, reduced chamber size, altered left ventricular mass, right ventricular dilation, septal thickening, valvular regurgitation, reduced ejection fraction, and diastolic dysfunction. These findings do not diagnose myocardial infarction independently but provide clinically meaningful information regarding myocardial damage, ventricular remodeling, and functional impairment following infarction (5). In hypertensive patients, myocardial injury may also occur due to increased myocardial oxygen demand and vascular dysfunction, even in the absence of acute plaque rupture, further supporting the need for careful cardiac structural assessment in patients with elevated blood pressure (6).

Despite the established relationship between obesity, hypertension, and cardiovascular morbidity, limited local data are available regarding the comparative echocardiographic profile of obese hypertensive and obese non-hypertensive patients with myocardial infarction. Most available literature describes the independent cardiovascular effects of obesity or hypertension, whereas fewer studies directly compare sonographic cardiac findings between hypertensive and non-hypertensive obese patients after myocardial infarction. This gap is important because identifying the most frequent echocardiographic

abnormalities in these groups may support early risk stratification, guide clinical monitoring, and improve understanding of cardiac remodeling patterns in high-risk local populations.

Therefore, the present study aimed to compare echocardiographic findings among obese hypertensive and obese non-hypertensive patients with myocardial infarction. The primary objective was to determine the frequency of major echocardiographic abnormalities, including left ventricular hypertrophy, increased left ventricular wall thickness, altered chamber size, left ventricular mass changes, right ventricular dilation, septal thickening, valvular regurgitation, left ventricular ejection fraction abnormality, diastolic dysfunction, and apical wall thickening, and to assess their association with hypertension status, age group, and gender. The study was guided by the research question: among obese patients with myocardial infarction, are echocardiographic structural and functional abnormalities more frequent in hypertensive patients than in non-hypertensive patients?

MATERIALS AND METHODS

This cross-sectional observational study was conducted among obese patients with myocardial infarction who underwent echocardiographic assessment at Combined Military Hospital, Lahore. The study was designed to compare the frequency and distribution of echocardiographic abnormalities between hypertensive and non-hypertensive obese patients with myocardial infarction. Data were collected after formal permission from the relevant hospital department and in accordance with institutional protocols and ethical requirements. The study population consisted of 103 participants who fulfilled the eligibility criteria and had complete information available for demographic characteristics, hypertension status, and echocardiographic findings.

Patients were eligible for inclusion if they were diagnosed cases of myocardial infarction, aged more than 20 years, had a body mass index greater than 30 kg/m², and were classified as either hypertensive or non-hypertensive according to their documented clinical status. Both male and female patients were included. Patients younger than 20 years, those with a history of coronary stent placement, known congenital heart disease, additional major comorbid conditions other than hypertension, or absence of myocardial infarction were excluded. To resolve inconsistency in the original eligibility description, the revised criteria define the study age range according to the actual enrolled sample, which included adults aged 21 to 70 years.

Participants were selected from eligible patients who underwent echocardiography during the study period. After eligibility screening, demographic data including age and gender were recorded, and patients were categorized into age groups of 21–30, 31–40, 41–50, 51–60, and 61–70 years. Hypertension status was recorded as a binary clinical variable and participants were grouped as hypertensive or non-hypertensive. Obesity was operationally defined as body mass index greater than 30 kg/m². The primary outcome variable was the echocardiographic finding documented for each participant, including left ventricular hypertrophy, increased left ventricular wall thickness, reduced chamber size, altered left ventricular mass, right ventricular dilation, septal thickness at R wave, tricuspid valve regurgitation, mitral regurgitation, left ventricular ejection fraction abnormality, Grade I left ventricular diastolic dysfunction, arterial regurgitation, and apical wall thickening.

Echocardiographic data were obtained using standard cardiac ultrasound assessment techniques. Two-dimensional echocardiography was used as the principal imaging approach for structural cardiac assessment, while Doppler and color Doppler techniques were used for functional and valvular evaluation where clinically indicated. Additional echocardiographic modalities, including three-dimensional echocardiography, strain imaging, and contrast imaging, were considered part of the available assessment framework when required for cardiac evaluation. The findings were documented according to the echocardiographic abnormality identified for each participant and were subsequently grouped for descriptive and inferential analysis.

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25. Quantitative and categorical variables were summarized using descriptive statistics. Age group, gender, hypertension status, and echocardiographic findings were presented as frequencies and percentages. Cross-tabulation was performed to compare hypertension status across age groups, gender distribution across age groups, echocardiographic findings across age groups, echocardiographic findings between hypertensive and non-hypertensive participants, and echocardiographic findings between male and female participants. The chi-square test of independence was used to assess associations between categorical variables. A p-value of less than 0.05 was considered statistically significant. Results with p-values greater than 0.05 were interpreted as statistically non-significant to avoid overstatement of group differences. All analyses were based on the available complete dataset of 103 participants.

Potential sources of bias were addressed by applying predefined inclusion and exclusion criteria, using a uniform echocardiographic assessment framework, and analyzing all eligible participants according to the same categorical definitions. Confounding was considered clinically relevant for age and gender because both variables may influence the prevalence of hypertension and echocardiographic abnormalities; therefore, cross-tabulated analyses by age group and gender were included to support interpretation of the main comparison between hypertensive and non-hypertensive groups. Data integrity was supported through structured data entry, verification of frequency totals against the full sample size, and consistency checking of categorical denominators before statistical analysis.

Ethical approval was obtained from the ethical committee of Combined Military Hospital Lahore on March 10, 2024. Written informed consent was obtained from all participants before data collection. Participation was voluntary, and patient privacy and confidentiality were maintained throughout the study. Data were anonymized before analysis, and no personally identifiable information was disclosed in the research outputs.

RESULTS

A total of 103 obese patients with myocardial infarction were included in the analysis. The largest age group was 41–50 years, comprising 34 participants (33.0%), followed by 31–40 years with 23 participants (22.3%), 61–70 years with 22 participants (21.4%), 51–60 years with 19 participants (18.4%), and 21–30 years with 5 participants (4.9%). Overall, 54 participants (52.4%) were hypertensive and 49 (47.6%) were non-hypertensive. Male participants predominated, representing 69 cases (67.0%), while 34 participants (33.0%) were female. The distribution of hypertension across age groups was not statistically significant, $\chi^2(4) = 6.345$, $p = 0.175$, Cramer's V = 0.248, indicating a small-to-moderate but statistically non-significant association. In contrast, gender distribution differed significantly across age groups, $\chi^2(4) = 11.876$, $p = 0.018$, Cramer's V = 0.340, showing that older age categories had a greater proportion of male participants.

Table 1. Demographic and Clinical Characteristics of the Study Participants

Variable	Category	Frequency (n)	Percentage (%)	Inferential Statistic
Age group	21–30 years	5	4.9	—
	31–40 years	23	22.3	—
	41–50 years	34	33.0	—
	51–60 years	19	18.4	—
	61–70 years	22	21.4	—
Hypertension status	Hypertensive	54	52.4	—
	Non-hypertensive	49	47.6	—
Gender	Female	34	33.0	—
	Male	69	67.0	—
Age group × hypertension status	—	—	—	$\chi^2(4) = 6.345$, $p = 0.175$, Cramer's V = 0.248
Age group × gender	—	—	—	$\chi^2(4) = 11.876$, $p = 0.018$, Cramer's V = 0.340

Left ventricular hypertrophy was the most frequent echocardiographic abnormality, observed in 17 participants (16.5%), followed by mitral regurgitation in 16 participants (15.5%) and increased left ventricular wall thickness in 12 participants (11.7%). Septal thickness at R wave, left ventricular ejection fraction abnormality, and Grade I left ventricular diastolic dysfunction were each observed in 9 participants (8.7%). Less frequent findings included reduced chamber size in 7 participants (6.8%), left ventricular mass abnormality and tricuspid valve regurgitation in 6 participants each (5.8%), apical wall thickening in 5 participants (4.9%), right ventricular dilation in 4 participants (3.9%), and arterial regurgitation in 3 participants (2.9%).

Table 2. Frequency Distribution of Echocardiographic Findings

Echocardiographic Finding	Frequency (n)	Percentage (%)
Left ventricular hypertrophy	17	16.5
Left ventricular wall thickness	12	11.7
Reduced chamber size	7	6.8
Left ventricular mass abnormality	6	5.8
Right ventricular dilation	4	3.9
Septal thickness at R wave	9	8.7
Tricuspid valve regurgitation	6	5.8
Mitral regurgitation	16	15.5
Left ventricular ejection fraction abnormality	9	8.7
Grade I left ventricular diastolic dysfunction	9	8.7
Arterial regurgitation	3	2.9
Apical wall thickening	5	4.9
Total	103	100.0

When echocardiographic findings were compared across age groups, the overall association was statistically non-significant, $\chi^2(44) = 49.323$, $p = 0.269$, Cramer's V = 0.346. This indicates that although specific findings varied numerically across age categories, the distribution did not differ significantly. The most frequent finding in the 41–50-year age group was left ventricular hypertrophy and mitral regurgitation, each affecting 7 participants. Grade I left ventricular diastolic dysfunction appeared most frequently in the 51–60-year group, with 5 cases, while left ventricular ejection fraction abnormality was most common in the 61–70-year group, with 4 cases.

Table 3. Distribution of Echocardiographic Findings Across Age Groups

Echocardiographic Finding	21–30 n	31–40 n	41–50 n	51–60 n	61–70 n	Total n	p-value	Effect Size
Left ventricular hypertrophy	1	4	7	2	3	17		
Left ventricular wall thickness	1	4	5	1	1	12		
Reduced chamber size	0	1	4	1	1	7		
Left ventricular mass abnormality	1	2	0	2	1	6		
Right ventricular dilation	1	2	1	0	0	4		
Septal thickness at R wave	0	4	1	3	1	9		
Tricuspid valve regurgitation	0	2	1	0	3	6		
Mitral regurgitation	0	2	7	2	5	16		
Left ventricular ejection fraction abnormality	1	0	2	2	4	9		
Grade I left ventricular diastolic dysfunction	0	0	2	5	2	9		
Arterial regurgitation	0	0	2	0	1	3		
Apical wall thickening	0	2	2	1	0	5		
Total	5	23	34	19	22	103	0.269	Cramer's V = 0.346

The distribution of echocardiographic findings between hypertensive and non-hypertensive participants was not statistically significant on omnibus chi-square testing, $\chi^2(11) = 16.550$, $p = 0.122$, Cramer's V = 0.401. Numerically, mitral regurgitation was observed in 9 hypertensive participants (16.7%) and 7 non-hypertensive participants (14.3%), while left ventricular hypertrophy was observed in 8 hypertensive participants (14.8%) and 9 non-hypertensive participants (18.4%). Increased left ventricular wall thickness was more frequent in hypertensive participants than non-hypertensive participants, 7 cases (13.0%) versus 5 cases (10.2%). Septal thickness at R wave and Grade I left ventricular diastolic dysfunction were also more frequent among hypertensive participants, each occurring in 6 hypertensive participants (11.1%) compared with 3 non-hypertensive participants (6.1%). Reduced chamber size was

observed only in non-hypertensive participants, while apical wall thickening was observed only in hypertensive participants. Because several cells had low counts, exploratory finding-specific odds ratios should be interpreted cautiously.

Table 4. Echocardiographic Findings by Hypertension Status with Exploratory Odds Ratios

Echocardiographic Finding	Hypertensive n (%)	Non-Hypertensive n (%)	Total n	OR	95% CI	Exact p-value
Left ventricular hypertrophy	8 (14.8)	9 (18.4)	17	0.77	0.27–2.19	0.791
Left ventricular wall thickness	7 (13.0)	5 (10.2)	12	1.31	0.39–4.44	0.764
Reduced chamber size	0 (0.0)	7 (14.3)	7	0.05	0.003–0.94	0.004
Left ventricular mass abnormality	3 (5.6)	3 (6.1)	6	0.90	0.17–4.69	1.000
Right ventricular dilation	1 (1.9)	3 (6.1)	4	0.29	0.03–2.88	0.344
Septal thickness at R wave	6 (11.1)	3 (6.1)	9	1.92	0.45–8.12	0.493
Tricuspid valve regurgitation	4 (7.4)	2 (4.1)	6	1.88	0.32–10.75	0.680
Mitral regurgitation	9 (16.7)	7 (14.3)	16	1.20	0.41–3.51	0.791
Left ventricular ejection fraction abnormality	4 (7.4)	5 (10.2)	9	0.70	0.18–2.79	0.733
Grade I left ventricular diastolic dysfunction	6 (11.1)	3 (6.1)	9	1.92	0.45–8.12	0.493
Arterial regurgitation	1 (1.9)	2 (4.1)	3	0.44	0.04–5.05	0.604
Apical wall thickening	5 (9.3)	0 (0.0)	5	11.00	0.59–204.32	0.058
Overall distribution	54 (100.0)	49 (100.0)	103	—	—	$\chi^2(11) = 16.550$, $p = 0.122$; Cramer's V = 0.401

Note: ORs compare the odds of hypertension among participants with each echocardiographic finding versus all remaining findings. Haldane–Anscombe correction was applied for cells containing zero. Exact p-values are exploratory and should not override the non-significant omnibus chi-square test.

When findings were compared by gender, the overall distribution was statistically non-significant, $\chi^2(11) = 8.559$, $p = 0.662$, Cramer's V = 0.288. Left ventricular hypertrophy occurred in 6 females and 11 males, while mitral regurgitation was evenly distributed, affecting 8 females and 8 males. Left ventricular ejection fraction abnormality was more frequent among males, with 8 male cases compared with 1 female case, but the overall gender-based pattern remained statistically non-significant. Therefore, although male participants numerically accounted for more echocardiographic abnormalities because they represented a larger proportion of the sample, the findings do not support a statistically significant difference in echocardiographic abnormality pattern by gender.

Table 5. Echocardiographic Findings by Gender

Echocardiographic Finding	Female n	Male n	Total n	p-value	Effect Size
Left ventricular hypertrophy	6	11	17		
Left ventricular wall thickness	5	7	12		
Reduced chamber size	1	6	7		
Left ventricular mass abnormality	2	4	6		
Right ventricular dilation	1	3	4		
Septal thickness at R wave	3	6	9		
Tricuspid valve regurgitation	1	5	6		
Mitral regurgitation	8	8	16		
Left ventricular ejection fraction abnormality	1	8	9		
Grade I left ventricular diastolic dysfunction	2	7	9		
Arterial regurgitation	2	1	3		
Apical wall thickening	2	3	5		
Total	34	69	103	0.662	Cramer's V = 0.288

Overall, the results indicate that left ventricular hypertrophy, mitral regurgitation, and increased left ventricular wall thickness were the most frequent echocardiographic abnormalities among obese patients with myocardial infarction. Hypertensive participants showed numerically higher proportions of left ventricular wall thickness, septal thickening, tricuspid regurgitation, mitral regurgitation, Grade I diastolic dysfunction, and apical wall thickening; however, the overall distribution of echocardiographic findings did not differ significantly between hypertensive and non-hypertensive patients. The only statistically significant demographic association was between age group and gender, suggesting that the age composition of male and female participants differed within the sample.

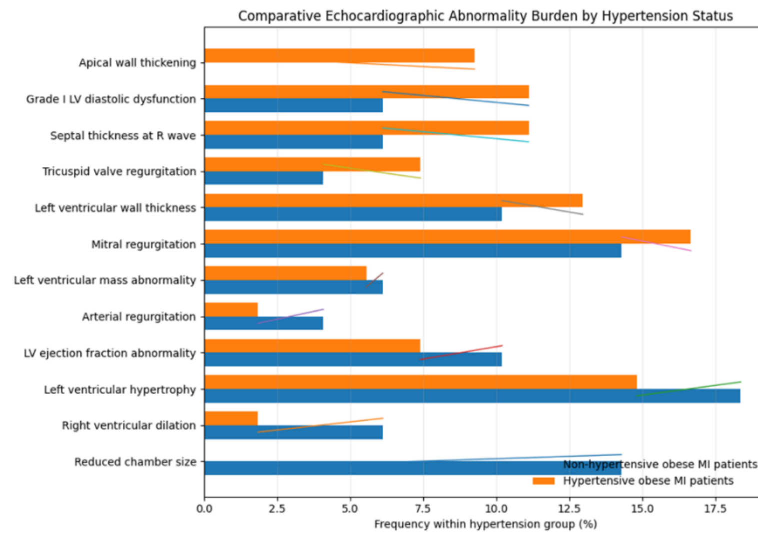

Figure 1. Comparative Echocardiographic Abnormality Burden by Hypertension Status

In Figure 1, the largest hypertensive-minus-non-hypertensive percentage-point gradients were observed for apical wall thickening (+9.3 percentage points), Grade I left ventricular diastolic dysfunction (+5.0 percentage points), and septal thickness at R wave (+5.0 percentage points), suggesting a higher numerical burden of pressure-overload-related remodeling features among hypertensive obese patients with myocardial infarction. In contrast, reduced chamber size showed a negative gradient of −14.3 percentage points and was observed only among non-hypertensive participants, while left ventricular hypertrophy was also slightly more frequent in non-hypertensive patients by −3.6 percentage points. Mitral regurgitation showed a minimal between-group difference of +2.4 percentage points, indicating similar distribution across hypertension status. These gradients support a cautious interpretation: hypertensive patients demonstrated selected structural and diastolic abnormalities more frequently, but the overall echocardiographic distribution remained statistically non-significant.

DISCUSSION

The present study evaluated echocardiographic abnormalities among obese patients with myocardial infarction and compared the distribution of these findings between hypertensive and non-hypertensive participants. The study population consisted of 103 obese patients with myocardial infarction, of whom 54 were hypertensive and 49 were non-hypertensive. The most frequent echocardiographic abnormalities were left ventricular hypertrophy, mitral regurgitation, and increased left ventricular wall thickness. These findings are clinically relevant because myocardial infarction in obese patients often occurs in the presence of overlapping structural, hemodynamic, and metabolic stressors that can influence ventricular remodeling, myocardial performance, and valvular function. Echocardiography remains an important non-invasive modality for identifying these post-infarction structural and functional abnormalities, particularly in high-risk patients with obesity and hypertension (5).

The predominance of left ventricular hypertrophy and increased left ventricular wall thickness is biologically plausible in this population. Obesity increases circulating blood volume, cardiac output, and ventricular workload, while hypertension further increases afterload and promotes pressure-overload remodeling. These mechanisms may contribute to increased myocardial mass, wall thickening, impaired relaxation, and eventual diastolic dysfunction. Previous literature has emphasized that hypertensive obese patients are vulnerable to left ventricular remodeling, diastolic impairment, and early cardiac damage detectable by echocardiography (3). In the present study, left ventricular hypertrophy was observed in 17 participants, while increased left ventricular wall thickness was observed in 12 participants. Although these findings were numerically common, their distribution did not differ significantly between hypertensive and non-hypertensive groups, indicating that obesity and myocardial infarction themselves may also contribute substantially to structural cardiac abnormalities.

Mitral regurgitation was the second most frequent echocardiographic abnormality, affecting 16 participants. This finding may reflect ischemic remodeling, altered ventricular geometry, papillary muscle dysfunction, or annular dilation following myocardial injury. The frequency of mitral regurgitation was slightly higher among hypertensive participants than non-hypertensive participants, but the difference was not statistically significant. This suggests that although hypertension may contribute to adverse ventricular loading conditions, the observed regurgitation pattern cannot be attributed confidently to hypertension alone in this sample. In obese patients with myocardial infarction, valvular abnormalities may emerge from a combined effect of ischemic myocardial injury, ventricular remodeling, and altered intracardiac pressures rather than from a single risk factor.

The comparison of sonographic findings between hypertensive and non-hypertensive patients showed no statistically significant overall association. The omnibus chi-square test produced a p-value of 0.122, indicating that the distribution of echocardiographic abnormalities was not significantly different across hypertension status. However, selected findings showed clinically notable numerical gradients. Septal thickness at R wave, Grade I left ventricular diastolic dysfunction, tricuspid valve regurgitation, mitral regurgitation, and apical wall thickening were proportionally more frequent among hypertensive patients, while reduced chamber size and left ventricular hypertrophy were proportionally more frequent among non-hypertensive participants. These trends should be interpreted cautiously because several categories had small cell counts, and exploratory odds ratios had wide confidence intervals. Therefore, the findings support the possibility of different echocardiographic patterns between groups but do not provide definitive statistical evidence of hypertension-related differences.

The non-significant association between age group and echocardiographic findings also requires careful interpretation. Although older age groups appeared to show higher frequencies of left ventricular ejection fraction abnormality and Grade I diastolic dysfunction, the overall age-based distribution of sonographic findings was not statistically significant. This may reflect limited statistical power due to subgroup fragmentation across five age categories and twelve echocardiographic categories. In contrast, the relationship between age group and gender was statistically significant, showing that male participants were more represented in older age groups. This demographic imbalance may influence the apparent distribution of echocardiographic abnormalities because male participants constituted two-thirds of the total sample. However, the overall distribution of echocardiographic findings by gender was also statistically non-significant, suggesting that gender alone did not explain the observed abnormality pattern.

The findings are consistent with prior evidence that obesity and hypertension contribute to cardiac remodeling through overlapping pathways. Obesity-related cardiovascular disease may arise through macrovascular disease leading to myocardial infarction, as well as microvascular dysfunction, inflammation, metabolic abnormalities, and pressure-volume overload that contribute to ventricular dysfunction and heart failure development (4). Similarly, hypertension is a major driver of myocardial oxygen demand, endothelial dysfunction, vascular inflammation, and myocardial injury, particularly when blood pressure elevation is severe or prolonged (6). The present findings support this pathophysiological framework by showing a high burden of structural and functional abnormalities among obese myocardial infarction patients. However, because this study did not include a non-obese comparison group or longitudinal follow-up, it cannot determine the independent contribution of obesity, hypertension, or infarction severity to each echocardiographic abnormality.

The present study has several strengths. It addresses a clinically important high-risk population and compares hypertensive and non-hypertensive obese patients with myocardial infarction using echocardiographic findings that are directly relevant to cardiac remodeling and functional impairment. The use of cross-tabulated analysis provides a practical overview of how cardiac abnormalities were distributed across hypertension status, age group, and gender. The revised analysis also improves interpretability by reporting effect sizes and avoiding overstatement of non-significant findings.

Several limitations should be acknowledged. The study was cross-sectional, so causal relationships between hypertension and echocardiographic abnormalities cannot be inferred. The sample size was modest, and several echocardiographic categories had low frequencies, which limited statistical power and produced wide confidence intervals in exploratory comparisons. The study did not report infarction type, infarction location, duration since myocardial infarction, medication use, blood pressure severity, diabetes status, lipid profile, smoking status, or detailed echocardiographic measurements such as left ventricular mass index, ejection fraction percentage, chamber dimensions, or diastolic parameters. These factors may confound the relationship between hypertension and echocardiographic abnormalities. In addition, the study relied on categorical echocardiographic findings rather than continuous cardiac measurements, which restricts the ability to perform more advanced regression-based analysis. Future studies should use larger multicenter samples, standardized echocardiographic measurement criteria, and multivariable models adjusting for age, gender, diabetes, smoking, dyslipidemia, infarction characteristics, and medication history.

Overall, the study suggests that obese patients with myocardial infarction demonstrate a substantial burden of echocardiographic abnormalities, particularly left ventricular hypertrophy, mitral regurgitation, and increased left ventricular wall thickness. Although hypertensive patients showed numerically higher frequencies of selected pressure-overload-related findings, the overall distribution of echocardiographic abnormalities did not differ significantly between hypertensive and non-hypertensive groups. These findings highlight the need for careful echocardiographic assessment in obese myocardial infarction patients regardless of hypertension status and support further investigation into the combined effects of obesity, hypertension, and ischemic injury on cardiac remodeling.

CONCLUSION

In this cross-sectional study of 103 obese patients with myocardial infarction, left ventricular hypertrophy, mitral regurgitation, and increased left ventricular wall thickness were the most frequent echocardiographic abnormalities. Although hypertensive participants showed numerically higher proportions of selected abnormalities such as septal thickening, Grade I left ventricular diastolic dysfunction, tricuspid regurgitation, mitral regurgitation, and apical wall thickening, the overall distribution of echocardiographic findings did not differ significantly between hypertensive and non-hypertensive patients. Age group was significantly associated with gender distribution, but neither age group nor gender showed a statistically significant association with the overall pattern of echocardiographic findings. These results suggest that obese patients with myocardial infarction have a clinically important burden of structural and functional cardiac abnormalities, supporting the role of echocardiography in post-infarction evaluation and risk assessment, while larger controlled studies are needed to clarify the independent contribution of hypertension to cardiac remodeling in this population.

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