e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(11); 1353-1358

Original Research Article

Association of obesity and long-term mortality in patients with acute myocardial infarction with and without diabetes mellitus

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Received: 05-09-2025 / Revised: 03-10-2025 / Accepted: 05-11-2025

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Conflict of interest: Nil

Abstract:

Background/ Objective: It's unknown how obesity and outcomes following AMI are related. An "obesity paradox" has been described in a number of studies, according to which people with higher BMIs seem to live longer than people of normal weight. AMI's long-term prognosis may be impacted by diabetes mellitus, a significant driver of cardiovascular risk. In order to ascertain whether diabetes alters the connection between BMI categories and 12-month all-cause mortality in patients with AMI.

Methods: This retrospective cohort study was carried out at the SKS Hospital Medical College and Research Centre in Mathura between January and December of 2024. 100 consecutive AMI patients were categorized as obese (≥30.0 kg/m²), overweight (25.0–29.9 kg/m²), or normal (18.5–24.9 kg/m²) according on their BMI, respectively. Diabetes status was ascertained using the HbA1c and medical history. The primary outcome was the 12-month all-cause mortality rate. Relationships were evaluated using regression study of Cox proportional hazards and Kaplan-Meier survival after sex, LVEF, age, reperfusion therapy, and diabetes were taken into account.

Results: 40% had diabetes, the mean age was 58.2 ± 10.8 years, and 72% were male. Over-all 12-month mortality was 15%. Mortality by BMI was 26.7% (normal), 10.0% (overweight), and 10.0% (obese). Overweight and obese groups showed lower unadjusted mortality (HR 0.32 and 0.30, respectively), which attenuated after adjustment (HR 0.45 and 0.42). Diabetes nearly doubled mortality risk (HR 1.90), without significant interaction with BMI.

Conclusion: Patients who were overweight or obese had a decreased 12-month mortality rate following AMI, whereas diabetes increased overall mortality but did not significantly alter the BMI–mortality association.

Keywords: Acute Myocardial Infarction; Obesity Paradox; Cardiovascular Outcomes.

Introduction

CVD continues to be Globally, AMI is the primary cause of death represents one of its most serious and life-threatening manifestations. Despite advances in reperfusion therapy, pharmacologic management, and cardiac rehabilitation, mortality following AMI remains substantial, particularly in individuals with metabolic comorbidities. Of these, two of the most important risk factors for the onset and advancement of CAD are obesity and DM. Obesity contributes to atherosclerosis through a variety of processes, including as systemic inflammation, ED, insulin resistance, and dyslipidemia. Excess adiposity increases circulating free fatty acids and pro-inflammatory cytokines, leading to oxidative stress and plaque instability. Conversely, weight reduction and improvement in metabolic parameters have been shown to decrease cardiovascular risk. Traditionally, therefore, obesity has been considered an adverse prognostic factor in patients with established CAD.

In a conundrum known as the "obesity paradox," people who are overweight or moderately obese had a better likelihood of surviving after an AMI, according to several large observational studies and meta-analyses. In contrast to patients of normal weight, those with a greater BMI have reduced mortality in the short and long term. Numerous theories have been put forth. One is that obese patients possess greater nutritional and metabolic reserves, allowing them to better tolerate the catabolic stress of acute illness. Another is that they tend to present earlier, receive more aggressive diagnostic evaluation, and are more likely to be prescribed evidence-based treatments include statins, ACE inhibitors, and β-blockers. Furthermore, BMI is an inaccurate measure of the

composition of the body because it is unable to discriminate between fat and lean mass or account for central adiposity—therefore some individuals classified as overweight may in fact have favorable muscle-to-fat ratios that confer better outcomes. In contrast, normal-weight patients may include those with sarcopenia, chronic illness, or subclinical frailty, all of which are independently associated with higher mortality.

Nonetheless, DM continues to be one of the most potent indicators of poor cardiovascular outcomes. It accelerates atherosclerosis through chronic hyperglycemia, endothelial injury, and low-grade inflammation, and contributes to adverse left ventricular remodeling and impaired myocardial recovery after infarction. Patients with diabetes often have diffuse and multivessel coronary artery disease, reduced collateral circulation, and poorer response to reperfusion therapies. Consequently, the coexistence of diabetes may influence or even negate the obesity paradox observed in general AMI populations. Some studies have reported that the apparent protective association of higher BMI is attenuated or absent among diabetic individuals, whereas others continue to demonstrate improved survival in overweight diabetics compared with their normal-weight counterparts. These discrepancies likely arise from differences in population characteristics, BMI categorization, adjustment for confounders, and follow-up duration across studies.

Not enough research has been done on the connection between post-AMI mortality, diabetes, and obesity in Indian communities. Even at lower BMI thresholds, South Asians exhibit a distinct metabolic phenotype known as the "thin-fat" phenotype, which is defined by increased body fat percentage, visceral adiposity, and insulin resistance. As a result, obesity-related cardiovascular risk manifests at comparatively lower BMI values. With rising rates of obesity and diabetes in India, understanding their combined impact on long-term AMI outcomes is of considerable clinical importance.

Methods

Study design and setting: From January to December 2024, the Department of Cardiology at SKS Hospital Medical College and Research Centre in Mathura carried out this retrospective study.

Study population: Included were 100 consecutive adult patients (≥18 years) who were admitted with AMI and were identified by increased cardiac biomarkers and an ECG. Both myocardial infarctions with and without ST elevation were taken into account.

Inclusion Criteria:

- Confirmed diagnosis of AMI.
- Availability of BMI and diabetes status at admission.
- Follow-up data available for at least 12 months.

Exclusion Criteria:

- Missing BMI or outcome data.
- Terminal illness unrelated to cardiac disease.
- Pregnant or postpartum women.

Variables and Definitions: Weight (kg)/height² (m2) was used to compute BMI, which was then divided into three categories: Obese (≥30.0 kg/m2), overweight (25.0–29.9 kg/m2), and normal weight (18.5–24.9 kg/m2).

Diabetes was defined by previous diagnosis, HbA1c ≥6.5%, or antidiabetic medication use.

Outcome Measure: All-cause mortality at 12 months after the index AMI incident was the main outcome. Hospital records and telephone follow-up provided the mortality data.

Covariates: Demographic and clinical data included smoking, age, sex, high blood pressure, dyslipidemia, chronic renal disease, prior MI, LVEF, AMI type (STEMI/NSTEMI), and reperfusion strategy (PCI, thrombolysis, or conservative).

Statistical Analysis: Continuous variables were expressed as mean ± SD and compared using the Student's t-test or one-way ANOVA, as appropriate. Frequencies and percentages were used to describe categorical variables, which were subsequently investigated using Fisher's exact test or the chi-square test. Survival across BMI groups was assessed using Kaplan–Meier curves and compared by the log-rank test. Cox proportional hazards models were applied to estimate HRs with 95% CI for 12-month mortality after adjusting for confounders.

An interaction term between BMI category and diabetes was included to evaluate effect modification. The threshold for statistical significance was $p<0.05$. R software was used to conduct the analyses.

Ethical Approval: The SKS Hospital Medical College and Research IEC gave its approval to the study protocol (Ref. No. SKS/IEC/2024/061).

Results

Baseline Characteristics: One hundred AMI patients were part of the study cohort. 72% were men, and the mean age was 58.2 ± 10.8 years. 40 patients (40%) had diabetes. Overweight (n = 40), normal weight (n = 30), and obese (n = 30) comprised the BMI distribution.

Hypertension was present in 65%, dyslipidemia in 48%, and chronic kidney disease in 12%. Mean LVEF was 45.0 ± 9.8%, lowest among normal-

weight patients (42.2%) compared to obese (46.8%) and overweight (46.0%) groups. Reperfusion therapy (PCI or thrombolysis) was performed in 60% of cases.

Table 1: Baseline Clinical and Demographic Characteristics by BMI Category

Variable	Normal (n=30)	Overweight (n=40)	Obese (n=30)	p-value
Age (years, mean ± SD)	60.5 ± 10.2	57.0 ± 10.5	57.2 ± 11.0	0.28
Male sex (%)	73	75	67	0.70
Diabetes mellitus (%)	20	30	73	<0.001
Hypertension (%)	60	45	97	<0.001
Dyslipidemia (%)	40	50	57	0.29
LVEF (%)	42.2 ± 10.1	46.0 ± 9.2	46.8 ± 9.0	0.04
PCI performed (%)	60	60	60	1.00
CKD (%)	10	10	17	0.44
STEMI (%)	63	65	67	0.91

Mortality outcomes: Overall, 15 patients (15%) died during the 12-month follow-up period. Mortality by BMI category was as follows:

- Normal weight: 8/30 (26.7%)
- Overweight: 4/40 (10.0%)
- Obese: 3/30 (10.0%)

Among patients with diabetes, mortality was higher (10/40; 25.0%) compared with non-diabetics (5/60; 8.3%).

Table 2: Mortality Outcomes by BMI and Diabetes Status

Category	Total (n)	Deaths (n)	Mortality (%)
BMI Category			
Normal weight	30	8	26.7
Overweight	40	4	10.0
Obese	30	3	10.0
Diabetes Status			
Diabetic	40	10	25.0
Non-diabetic	60	5	8.3

Kaplan–Meier Survival: Survival curves demonstrated lower cumulative mortality rates in groups of individuals who weigh more than average compared to individuals who are overweight or obese (log-rank p = 0.03). Diabetic patients exhibited poorer survival across all BMI strata.

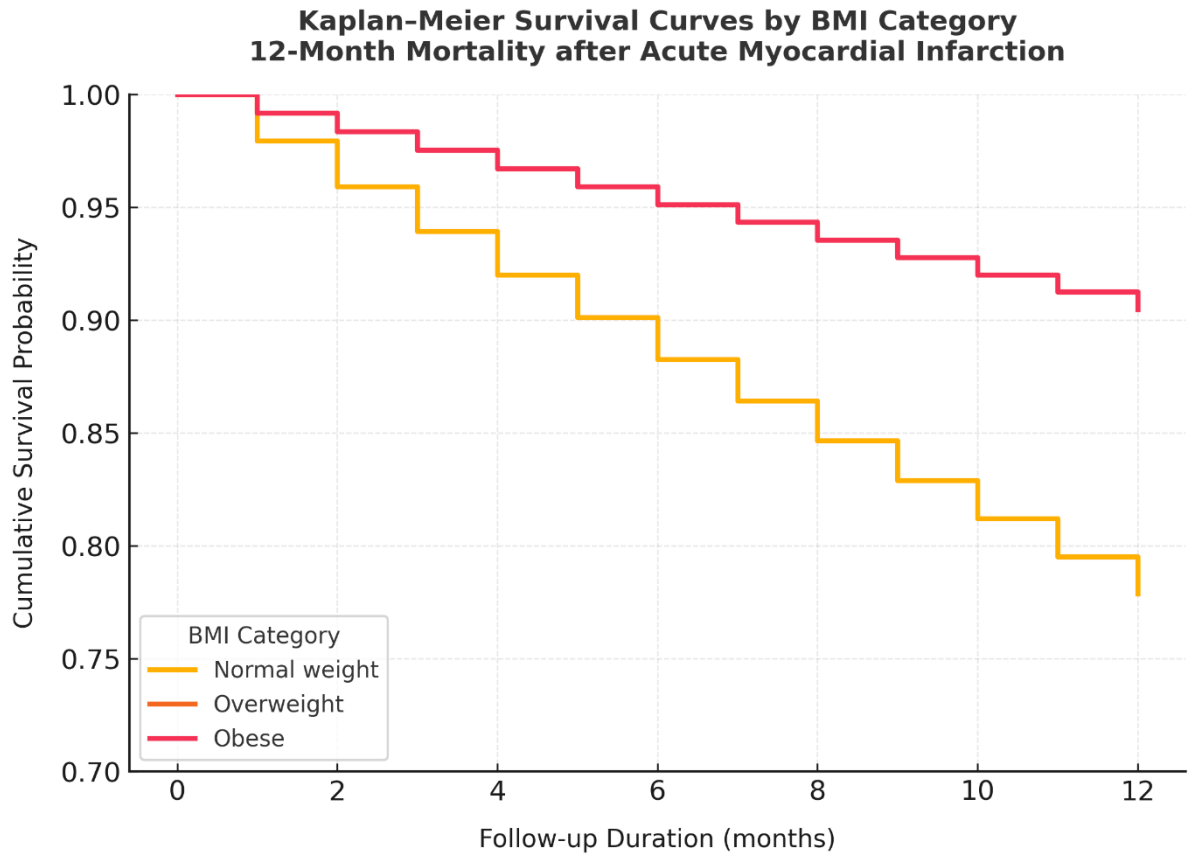

Figure 1. Kaplan–Meier Survival Curves for 12-Month All-Cause Mortality Stratified by BMI Category

Cox proportional hazards regression: Compared to individuals of normal weight, those who were overweight (HR 0.32; 95% CI 0.10–0.98; p = 0.047) and obese (HR 0.30; 95% CI 0.09–0.98; p = 0.046) had a decreased mortality risk, according to unadjusted analysis.

Discussion

This retrospective study explored the association between BMI and 12-month mortality following AMI, while also examining whether DM modifies this relationship. Among the 100 patients studied, overweight and compared to patients of normal weight, obese people had a decreased mortality rate, even though the prevalence of diabetes and hypertension was higher in these groups. The tendency for patients who are overweight or obese to live longer persisted after adjustment for age, LVEF, diabetes, and reperfusion therapy, although statistical significance was not sustained. Diabetes was separately linked to an increased risk of mortality, but it did not appear to significantly influence the direction of the BMI–mortality association. These findings indicate that while obesity predisposes individuals to cardiovascular disease, its prognostic role after the occurrence of AMI may be more complex and counterintuitive than previously believed.

The current study's findings align with what numerous earlier studies have referred to as the "obesity paradox," which is a circumstance where patients with higher BMIs have better survival outcomes after major cardiovascular events. Several large registries, including the GRACE and the Cooperative Cardiovascular Project, have reported similar findings. In these patients who were overweight or somewhat obese had reduced one-year and in-hospital death rates than patients who were of normal weight. The present analysis reinforces these observations in an Indian population, suggesting that the relationship between BMI and outcomes may not be linear. While obesity increases the lifetime risk of CAD, a higher BMI at the time of AMI may, paradoxically, be linked with better recovery and lower short-term mortality.

Several physiological mechanisms might underlie this paradox. Patients with excess body mass tend to have greater metabolic and nutritional reserves, which may provide resilience against the catabolic stress and inflammatory burden that follow myocardial infarction. Increased circulating lipoproteins can bind and neutralize inflammatory mediators, reducing myocardial injury. Furthermore, overweight and obese individuals are often younger and may have better preserved ventricular function at baseline. They also tend to receive more frequent monitoring, earlier diagnosis,

and more aggressive use of guideline-directed therapies such as β-blockers, statins, and ACE inhibitors. In contrast, normal-weight patients may represent a subgroup with hidden frailty, unrecognized chronic illness, or low skeletal muscle mass—factors that adversely affect survival but are not captured by BMI alone. These explanations suggest that body composition, muscle strength, and overall fitness may play more important roles in prognosis than BMI as a single index.

Diabetes mellitus remains one of the most potent predictors of poor outcomes following AMI. People with diabetes in this study died at a rate that was almost three times greater than that of people without the disease. This observation aligns with well-documented evidence that hyperglycemia, endothelial dysfunction, and microvascular disease impair myocardial recovery and promote post-infarct remodeling. However, the lack of a statistically significant interaction between BMI and diabetes suggests that the adverse effects of diabetes did not completely offset the potential protective influence associated with higher BMI. Previous studies have reported mixed findings—some indicating that the obesity paradox disappears among diabetic patients, and others suggesting that overweight diabetics still fare better than lean diabetics. The differences likely arise from variations in glycemic control, the duration of diabetes, and differences in the degree of metabolic compensation at the time of infarction. These findings highlight that diabetes contributes to mortality primarily through metabolic instability rather than body weight alone.

The results carry particular significance in the Indian setting, where body composition differs markedly from Western populations. South Asians are prone to insulin resistance, central adiposity, and lower lean body mass at relatively lower BMI values—a combination sometimes described as the "thin-fat" phenotype. Conventional BMI cut-offs may thus underestimate obesity-related cardiovascular risk in Indians. Even individuals with BMI within the "normal" range may harbor substantial visceral fat and metabolic dysfunction, predisposing them to worse outcomes. Conversely, overweight or mildly obese patients in this context may not have the same degree of metabolic risk as Western cohorts of similar BMI. The observed survival advantage among higher BMI groups may therefore reflect differences in body composition and nutritional reserve rather than the protective role of adiposity itself. Moreover, undernutrition, which remains prevalent in certain sections of the Indian population, could contribute to poor prognosis among normal-weight patients.

This study contributes valuable clinical insight by presenting real-world data from a tertiary care hospital. The use of consecutive patient records minimized selection bias, and the inclusion of both diabetic and non-diabetic subgroups provided a broader understanding of metabolic influences on AMI outcomes. However, some limitations must be regocnized. The sample size was modest, which may have limited the power to detect smaller but meaningful associations. The retrospective nature of the study prevented control over confounding variables and relied on the accuracy of recorded data. BMI was measured at admission and could have been influenced by acute fluid shifts. There were no more accurate indicators of adiposity, such as body fat percentage, waist circumference, or waist-to-hip ratio. In addition, details on glycemic control, diabetes duration, and cause-specific mortality were not recorded. Despite these limitations, the consistent trend across analyses supports the validity of the observed association.

According to the study's findings, BMI by itself might not be a reliable predictor of prognosis for individuals with AMI. Although overweight and obesity appear to be linked with lower short-term mortality, these observations should not be interpreted as a justification for maintaining excess weight. Instead, they highlight the need to move beyond BMI toward more comprehensive assessments that include body composition, nutritional status, and physical fitness. Secondary prevention strategies should continue to focus on improving metabolic health, glycemic control, and cardiovascular fitness rather than weight alone. Larger, multicentric, prospective studies using refined anthropometric and biochemical measures are needed to confirm whether the obesity paradox observed here persists in the long term. According to the study's findings, patients who were overweight or obese had lower crude mortality following an AMI than people who were normal weight. Diabetes also raised overall risk, but it had no discernible effect on the relationship between BMI and survival. These findings underline the complex and multifactorial nature of post-AMI prognosis in contemporary clinical practice.

Conclusion

Overweight and obese patients demonstrated lower unadjusted 12-month mortality following AMI in contrast to patients of normal weight, which is in line with the obesity paradox. Diabetes did not substantially change the association between BMI and survival, however it was linked to higher mortality. These findings suggest that higher BMI may confer short-term protective effects after AMI, though causality cannot be inferred. Larger, multi-center to validate these findings and elucidate the underlying mechanisms, prospective research is required.

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