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Excess weight and life expectancy after acute myocardial infarction: the obesity paradox reexamined

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Abstract

Background—An “obesity paradox” has been described in patients with acute myocardial infarction (AMI), whereby obese and overweight patients have a lower risk of short-term mortality after AMI than normal weight patients. However, the long-term association of obesity with mortality after AMI remains unknown.

Methods—We used data from the Cooperative Cardiovascular Project, a prospective medical record study of Medicare beneficiaries hospitalized with AMI with 17 years of follow-up (N=124,981), to evaluate the association of higher body mass index (BMI) with short and long-term survival after AMI. Cox proportional hazards models were used to estimate life expectancy after AMI and the years of potential life lost or gained attributable to excess weight.

Results—Approximately 41.5% of patients were classified as normal weight, 38.7% as overweight, 14.3% as obese, and 5.5% as morbidly obese. Normal weight patients had the highest crude mortality at all follow-up time-points, whereas obese patients had the lowest. Adjustment for patient and treatment characteristics reduced this difference, but the survival benefit persisted in overweight and obese patients at all time points. Morbidly obese and normal weight patients had a comparable risk of death at 17-years. Life expectancy estimates were generally lowest for morbidly obese patients and highest for overweight patients. Differences in life expectancy

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between BMI groups were most pronounced in younger patients. After adjustment, overweight and obesity were associated with greater life-years at all ages; however, morbid obesity was only associated with better survival in patients ≥ 75 years of age at the time of AMI.

Conclusions—Overweight and obesity are associated with improved short and long-term survival after AMI, which results in moderate gains in life expectancy relative to normal weight patients. These findings suggest that higher BMI confers a protective advantage over the entire remaining lifespan in older patients with AMI.

Keywords

body mass index; obesity; life expectancy; acute myocardial infarction; survival

An “obesity paradox” has been described in patients with acute myocardial infarction (AMI), whereby overweight and obese patients have a lower risk of short-term mortality after AMI than normal weight patients. However, the long-term association of higher BMI with survival after AMI remains unknown. Although studies of short-term mortality have consistently reported lower risk of mortality for overweight and obese patients after AMI, [1-5] studies of long-term mortality have shown mixed results with some finding lower mortality in obese patients[6-9] and others finding no association between BMI and mortality.[10-12] The discrepancies in study results are likely explained by differences in length of follow-up and covariate adjustment. In general, studies reporting an obesity paradox have had shorter follow-up periods (2-3 years)[6-9] than studies finding no effect (8 years)[11,12], suggesting that the relationship between BMI and mortality may attenuate over time. In addition, prior studies have suggested that the obesity paradox may be due to the deleterious effects of cachexia in normal weight patients, rather than the beneficial effects of obesity.[1,13,14] However, few studies have adjusted for conditions or factors associated with cachexia. As a result, the question of whether the obesity paradox exists over the long-term and whether it can be explained by thorough risk adjustment remains unresolved.

Understanding the long-term benefit or burden of obesity after AMI is essential for clinicians when educating overweight and obese patients on prognosis and stratifying patients by risk. However, answering these questions requires the examination of body mass index (BMI) with both short and long-term mortality after AMI while controlling for factors that may be associated with both obesity and cachexia. This in turn requires a large database of AMI patients with detailed clinical information and long-term follow-up. With over 17 years of follow-up and rich clinical information on a nationally representative sample of AMI patients, the Cooperative Cardiovascular Project (CCP) provides this opportunity. We undertook the following study to evaluate the association of BMI with life expectancy after AMI. We chose to use life expectancy to evaluate differences in survival in order to quantify the effects of BMI on mortality after AMI in absolute terms, which may be more meaningful to patients. In addition, we sought to quantify the burden or benefit of obesity after AMI by estimating the years of potential life lost (YPLL) or gained (YPLG) after AMI associated with excess weight. We compared the number of life-years lived after AMI by overweight or obese patients to that of normal weight patients in order to evaluate the effects of BMI over

the entire remaining lifespan. Finally we determined whether these relationships vary by patient age.

Methods

The CCP was a Center for Medicare and Medicaid Services (CMS) project developed to evaluate the quality of AMI care through detailed medical record review. It includes fee-for-service Medicare beneficiaries hospitalized with a principal discharge diagnosis of code of AMI (International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) code 410) from 4,834 acute-care nongovernmental hospitals in the U.S. Individual states were sampled during an 8-month period between January 1994 and February 1996, and all patients with AMI during that period were included in the sample. Readmissions for the same AMI episode (*ICD-9-CM code 410.x2*) were excluded. Trained personnel abstracted information on demographic, medical history, clinical presentation, and treatment from patient medical records using an automated system to ensure standardized data collection.

We excluded patients aged <65 years (n=17,593), patients transferred from post-ambulatory or surgical settings (n=19,379), and patients without confirmed AMI (n=31,186). The diagnosis of AMI was confirmed by 1) elevation of creatine kinase-MB level (>5% of total creatine kinase), or an elevation of lactate dehydrogenase enzyme (LDH) level with isoenzyme reversal ($LDH_1 > LDH_2$), or 2) the presence of at least 2 of the following: chest pain, 2-fold elevation in creatine kinase, and diagnostic electrocardiographic changes (ST-segment elevation or new pathological Q-waves). If patients were admitted more than once for AMI during the study period, we excluded all admissions but the first (N=27,500). Finally we excluded patients with missing height or weight data, for whom BMI could not be calculated (n=27,690, 17.5%). Compared with patients with recorded BMI values, patients with missing BMI were older (mean age 78.6 vs. 76.0, $p < 0.001$) and more often female (56.1% vs. 47.3%, $p < 0.001$). In addition, they were more likely to have dementia (11.9% vs. 4.8%, $p < 0.001$) and to be admitted from nursing homes (14.0% vs. 5.0%, $p < 0.001$) and less likely to be mobile (68.1% vs. 80.7%, $p < 0.001$) or continent (82.0% vs. 92.0%, $p < 0.001$) on admission. Patients with missing BMI had higher in-hospital mortality rates (27.0% vs. 11.9%, $p < 0.001$) and over all of follow-up (17-year mortality 96.1% vs. 92.3%, $p < 0.001$). Among the 130,659 patients with recorded BMI, we excluded an additional 5,678 patients who were underweight ($BMI < 18.5 \text{ kg/m}^2$) because the focus of the study was on excess weight.

Measurement of BMI

BMI values were calculated from patients' admission height and weight data, as abstracted from medical records. Patients were categorized using criteria from the Centers for Disease Control and Prevention (CDC) into normal weight ($18.5\text{-}24.9 \text{ kg/m}^2$), overweight ($25\text{-}29.9 \text{ kg/m}^2$), obese ($30\text{-}34.9 \text{ kg/m}^2$), and morbidly obese ($\geq 35 \text{ kg/m}^2$).[15]

Outcome Variable

We ascertained death over 17 years of follow-up by linking the CCP to the Medicare Denominator Files from 1994-2012. Time to death was defined as days from admission to the date of death and was censored at 17 years of follow-up.

Statistical Analyses

We compared baseline characteristics across BMI classes using chi-squared test for categorical variables and analysis of variance for continuous variables. We assessed differences in crude mortality at 30-days, 1 year, 5 years, and 17 years across the four BMI classes using chi-squared tests and Cox proportional hazards regression with the normal weight group as reference. We then repeated analyses adjusting for demographics (age, sex, race, and ZIP code level median household income), cardiovascular risk factors (diabetes mellitus, hypertension, smoker, and prior coronary events, peripheral vascular disease), clinical presentation (Killip class, systolic blood pressure, heart rate, ST-elevation AMI on ECG, infarct location, cardiac arrest on admission, and renal insufficiency), and therapies received (percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) within 30 days, fibrinolytic therapy, and receipt of aspirin and beta-blockers on admission). In addition, we adjusted for several factors associated with cachexia, including comorbidities known to cause cachexia (congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD), cerebrovascular events, chronic kidney disease (CKD), Human Immunodeficiency Virus (HIV) infection, cancer, dementia, anemia, and hypoalbuminemia) and markers of poor health status or frailty (admission from a nursing home, decreased mobility, and urinary incontinence). Covariates were selected a priori using prior literature and clinical judgment to identify variables that are associated with excess weight or cachexia.

Missing covariate data were minimal, with 9.9% of patients missing any covariate data and 8.7% missing a single covariate value. Patients with missing data on continuous variables (systolic blood or income) were assigned the median value in the cohort and a binary dummy variable to denote missing data. Patients with missing categorical data were included in the model as a separate dummy variable for that covariate.

Life expectancy was defined as mean survival after AMI, which is equivalent to the area under the survival curve. To calculate BMI- and age-specific life expectancy after AMI, we first fit a Cox proportional hazards model that included the BMI categories, age, and their interactions as terms and plotted BMI- and age-specific expected survival curves from this model. Because approximately 7% of patients were still living at the end of follow-up, we extrapolated the expected survival curves to age 100 using exponential models in order to approximate a closed cohort. The constant hazard for each exponential model was defined as the average hazard over the last 2 years of follow-up. Exponential models were selected because they provide a conservative approach for modeling survival when there is limited information on the shape of the survival curves. Age 100 was chosen because the Centers for Disease Control and Prevention uses this age as the upper limit in its life-table analyses for estimating general population life expectancy. Finally, the areas under the Cox and exponential curves were summed to estimate BMI- and age-specific life expectancy. Ninety-

five percent confidence intervals were calculated using the same approach from the upper and lower confidence limits of the expected survival curves. YPLL/YPLG after AMI attributable to excess weight were calculated by subtracting age-specific life expectancy in normal weight patients from that in overweight, obese, and morbidly obese patients.

To determine whether differences in life expectancy between BMI groups were explained by other patient factors, we repeated the life expectancy calculations using multivariable Cox proportional hazards regression. We used age-specific covariate frequencies or mean values to plot the BMI- and age-specific expected survival curves from these models. This approach forces same-aged patients in different BMI classes to have the same values of baseline characteristics, thereby allowing us to directly compare life expectancy across BMI classes at fixed covariate values. Adjusted YPLL/YPLG were again calculated as the difference in life expectancy between normal weight patients and patients in other BMI classes. All analyses were performed using SAS 9.2 (SAS Institute, Cary, NC).

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Results

A total of 124,981 patients met the inclusion criteria. The average age of the study sample was 75.8 ± 7.2 . Forty-six percent of patients were women, and 9.3% were nonwhite. Of these, 41.5% were classified as normal weight, 38.7% as overweight, 14.3% as obese, and 5.5% as morbidly obese using CDC criteria. On average, obese and morbidly obese patients were younger than normal weight patients (**Table 1**).

Compared with normal weight patients, overweight and obese patients had higher rates of diabetes and hypertension but were less likely to smoke. In addition, they had lower rates of most cachexia-related comorbidities including COPD, stroke, CKD, HIV, cancer, dementia, anemia, and hypoalbuminemia than normal weight patients and were less likely to be admitted from nursing homes. Although overweight and obese patients had lower rates of ST-elevation AMI, they were significantly more likely to undergo revascularization in the first 30- days and to receive fibrinolytic therapy and guideline-based medications on admission.

Normal weight patients had the highest crude mortality at all follow-up time-points, followed by morbidly obese then overweight and lastly obese patients (**Table 1**). The greatest differences in mortality were observed at 1-year (**Table 2**).

Adjustment for patient and treatment characteristics reduced this difference, but the survival benefit persisted in overweight and obese patients. At 1-year, overweight and obese patients had 13-14% lower hazards of death than normal weight patients, and by 17-years overweight

was associated with a 9% decrease in mortality and obesity with a 7% reduction. Morbidly obese patients had 12% lower hazards of death at 1- and 5-years than normal weight patients but comparable hazards at 17-years (HR 0.98 (95% CI 0.96-1.01)).

Among younger patients (<80 years-old), life expectancy estimates were shortest for morbidly obese patients and longest for overweight patients (**Figure 1, eTable 1**).

In older patients (>80 years-old), however, normal weight patients had the shortest life expectancy. Differences in life expectancy between BMI groups were most pronounced in younger patients. **Figure 2** shows the unadjusted estimates for the YPLL/YPLG after AMI by overweight, obese, and morbidly obese patients relative to normal weight patients.

Overweight and obesity were associated with gains in life-years relative to normal weight patients at all ages, whereas the relationship between morbid obesity and YPLL varied by age. At younger ages, morbidly obese patients lost between 0.21 (standard error (SE) 0.09) and 1.63 (SE 0.20) years of life after AMI, but at older ages, morbid obesity conferred a survival advantage after AMI (**eTable 2**). After adjustment, the relationship between morbid obesity and YPLLs was attenuated (**Figure 3**).

Morbidly obese patients <75 years of age had similar life expectancies to normal weight patients; however, in older patients (>75 years-old), morbid obesity was associated with life expectancy gains of 0.20 (SE 0.08) to 0.50 (SE 0.10) years. Both overweight and obesity were associated with gains in life expectancy at all ages. On average, overweight was associated with gains in life expectancy of 0.10 (SE 0.03) to 1.12 (SE 0.11) years, and obesity was associated with gains in life expectancy of 0.28 (SE 0.05) to 0.40 (SE 0.07) years relative to normal weight patients (**eTable 3**).

Discussion

We found that overweight and obesity were associated with improved short and long-term survival after AMI, which resulted in moderate gains in life expectancy relative to normal weight patients. On average, overweight and obese patients lived between 0.10-1.12 and 0.28-0.40 years longer than normal weight patients, respectively; however, these estimates varied by age. The longer life expectancies in overweight and obese patients persisted even after accounting for their younger age, lower comorbidity, and higher rates of treatment. In contrast, morbid obesity was associated with small losses in life-years in younger patients but gains in life years in older patients. Excess weight was uniformly protective in patients >75-years-old and associated with increased life expectancy.

Prior studies examining the relationship between excess weight and long-term mortality after AMI have been inconsistent. Although many have reported results similar to ours,[6-9] others have not found evidence of an obesity paradox.[10-12] We hypothesized that the disparate results may be due, in part, to differences in follow-up, sample size, and clinical detail between studies. Interestingly prior studies with nonsignificant results have actually reported similar risk estimates to those in our study but with larger error intervals, suggesting that they may simply have been underpowered to detect an effect.[12] This study is the first to quantify the survival advantage associated with excess weight after AMI over

the entire remaining lifespan. It has several strengths over prior studies including its large sample size of over 120,000 patients with AMI, detailed chart-abstracted data, and the longest follow-up to date.

Unlike prior studies, however, we found that the relationship between excess weight and mortality after AMI varied by age. In younger patients <75 years old, overweight and obese patients had higher life expectancies compared with normal weight and morbidly obese patients, but in older patients >75 years old, all categories of excess weight were associated with gains in life expectancy including morbid obesity. To our knowledge, only one other study has reported an interaction between BMI and age. Hoit et al. found that obesity was associated with increased hospital death in older (≥65-years-old) but not younger patients. [16] These observations conflict with those reported in our study but are likely explained by differences in the age distribution and categorization of BMI between studies. Less than half of the patients in the study by Hoit et al. were ≥65 years old, and very few patients (n=72) were categorized as elderly obese. In addition, obese and morbidly obese patients were evaluated together even though our study demonstrates heterogeneity in the effect of obesity at the highest BMI levels.

Several theories have been proposed to explain the obesity paradox after AMI. Excess weight may confer a survival benefit by providing nutritional reserves to overcome times of acute stress and increased metabolic demands, such as AMI. Consistent with this theory, studies of critically ill patients in intensive care units have found that higher BMI is associated with decreased hospital mortality and improved functional status at discharge across a wide range of conditions.[17-19] In fact, this observation may explain why excess weight was universally protective in older patients with AMI, who have greater frailty and decreased homeostatic reserve to overcome acute insults, but not in younger patients. In addition, prior studies have suggested that clinicians may treat obese patients more aggressively after AMI due to increased awareness of the risks associated with obesity.[20] Indeed, overweight and obese patients in our study had the highest rates of 30-day revascularization and were more likely to receive guideline-based therapies on admission. Because patients with elevated BMI also have higher rates of cardiovascular risk factors, they may also receive more aggressive secondary prevention after discharge.

New evidence also suggests that obesity may play a role in inflammation and endothelial function, which may affect the progression of atherosclerosis and risk of mortality. Although controversial, these studies have shown that classic markers of inflammation like C-reactive protein are poor predictors of atherosclerosis in obese patients and that obese persons have better endothelial function than non-obese persons.[21-23] These findings also support other recent evidence suggesting that obese AMI patients have less severe and less complex coronary artery disease than non-obese AMI patients as assessed by quantitative coronary angiography including SYNTAX scores and number of stenotic lesions.[24]

Interestingly, other studies have hypothesized that the obesity paradox may be more related to confounding by cachexia in normal weight patients than an actual benefit conferred by obesity.[1,13,14] To date, no study has been able to test this theory; however, we evaluated and adjusted for a number of common conditions associated with cachexia. Indeed we found

that normal weight patients had higher rates of several comorbidities including CHF, COPD, CKD, HIV, cancer, and dementia, and they were more likely to be anemic and have low albumin levels, which may reflect poor nutritional status. Moreover, normal weight patients were more likely to be admitted from nursing homes, to require assistance walking, and to be incontinent at baseline, suggesting that they may represent a sicker population altogether. The obesity paradox persisted after adjustment for these variables, suggesting that confounding by cachexia-related conditions does not explain this paradox. Nevertheless, there may still be other unmeasured risk factors in normal weight patients that contribute to the outcomes we observed.

Finally, it is possible and highly likely that obese and non-obese individuals represent two different populations with different cardiovascular and non-cardiovascular risk factors, which may also affect risk of mortality after AMI. Recent studies using directed acyclic graphs have shown that the obesity paradox in patients with heart disease may be explained by collider stratification bias, a form of bias that is introduced when conditioning on a variable that is related to both obesity and mortality. Limiting our sample to patients with cardiovascular disease may introduce a selection bias because obese and non-obese patients likely acquire cardiovascular disease in different ways and thus represent two distinct populations. While such bias can limit causal inferences, it is still informative to evaluate the association of obesity with mortality after AMI in order to better risk stratify patients after AMI and to inform patients about prognosis.

This study has some limitations. First, we used BMI values recorded at the index hospitalization only and thus were unable to evaluate changes in BMI over time. Nevertheless, prior studies have found that most patients neither lose nor gain weight in the year after AMI,[26] which would suggest that weight changes during follow-up are unlikely to affect our results. Second, we lacked information on other anthropometric measures to measure adiposity such as waist circumference or body fat percentage. Although BMI is widely used in clinical practice to evaluate obesity, it is an imprecise measure of total body fat and does not evaluate fat distribution. Third, we excluded 27,690 (17%) patients with missing BMI data from our sample. In general, patients with missing BMI were older and frailer than patients with recorded BMI, and they had significantly higher mortality rates at all follow-up time-points. Thus, our results may be subject to selection bias or may not be generalizable to all patients. Fourth, we adjusted for several clinical and treatment characteristics, some of which may be in the causal pathway between BMI and mortality after AMI. We opted for thorough risk adjustment at baseline in order to determine which variables if any explained the association between current BMI and long-term mortality after AMI. However, it is possible that we adjustment for certain variables such as diabetes or hypertension may have resulted in overadjustment of the models because obesity is correlated with certain comorbidities. Fifth, we were unable to adjust for other factors related to obesity (e.g. ejection fraction,[27] depression,[28] kidney function,[29], dyslipidemia[30], and statin use) and cachexia (e.g. nutritional status, physical disability). As a result, the possibility for residual confounding by these or other unmeasured factors is possible. However, this is the first study to adjust for factors related to both excess weight and cachexia. Finally, the Medicare Denominator files include only all-cause mortality.

Thus, we were unable to differentiate cardiovascular mortality from deaths due to other causes.

In conclusion, we observed an obesity paradox among patients with AMI. Overweight and obese patients had improved short and long-term survival after AMI, which produced moderate gains in life-years relative to normal weight patients. This effect was not explained by other patient or clinical factors associated with obesity or cachexia. In contrast, morbid obesity was associated with improved life expectancy only in older patients. By demonstrating that excess weight is associated with improved life expectancy in older patients, our findings raise the question of whether weight loss recommendations after AMI are beneficial in older adults. However, because obesity is a known risk factor for cardiovascular and other disease processes, our findings do not support weight gain after AMI. Future studies are needed to evaluate weight changes during follow-up in order to inform weight management guidelines in older patients and to understand other potential mechanisms contributing to the obesity paradox.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Life expectancy after acute myocardial infarction by body mass index category and age

*Life expectancy estimates shown are age- and BMI-specific point estimates. A solid line has been used to connect these point estimates for ease of interpretation.

Figure 2.

Unadjusted years of potential life lost (positive) or gained (negative) after AMI in overweight, obese, and morbidly obese patients relative to normal weight patients *Years of life lost associated with excess weight are calculated as the difference in life expectancy between overweight, obese, or morbidly obese patients and normal weight patients. The estimates shown are age- and BMI-specific point estimates. A solid line has been used to connect these point estimates for ease of interpretation

Figure 3.

Adjusted years of potential life lost (positive) or gained (negative) after AMI overweight, obese, and morbidly obese patients relative to normal weight patients *Years of life lost associated with excess weight are calculated as the difference in life expectancy between overweight, obese, or morbidly obese patients and normal weight patients. Analyses have been adjusted for age, gender, race, ZIP code level median household income, cardiovascular risk factors (diabetes, hypertension, smoking, prior coronary events, and peripheral vascular disease), clinical presentation (Killip class, systolic blood pressure on admission, heart rate on admission, ST-elevation AMI, AMI location, and renal insufficiency), therapies received (percutaneous coronary intervention or coronary artery bypass grafting within 30 days of admission, fibrinolytic therapy during the index admission, and among those eligible, aspirin and beta-blockers on admission), cachexia-related factors (congestive heart failure, chronic obstructive pulmonary disease, cerebrovascular disease, chronic kidney disease, Human Immunodeficiency Virus, cancer, dementia, anemia, hypoalbuminemia), and measures of pre-hospital frailty (admission from a nursing home, decreased mobility, and urinary incontinence). The estimates shown are age- and BMI-specific point estimates. A solid line has been used to connect these point estimates for ease of interpretation

Table 1

Sample characteristics by body mass index category (n=124,981)

Characteristics	Normal Weight N=51,896 (41.5%)	Overweight N=48,422 (38.7%)	Obese N=17,822 (14.3%)	Morbidly Obese 6841 (5.5%)
Demographics				
Age, mean (SD)	77.7 (7.4)	75.0 (6.7)	73.8 (6.3)	72.8 (6.0)
Female	25,411 (49.0)	19,283 (39.8)	8910 (50.0)	4317 (63.1)
Nonwhite race	47,293 (91.2)	44,100 (91.1)	15,946 (89.5)	6033 (88.2)
Median household income, mean (SD)	30776 (11684)	30373 (11290)	29982 (10953)	29823 (10740)
Missing	2144 (4.1)	2132 (4.4)	778 (4.4)	272 (4.0)
Risk Factors				
Diabetes mellitus	12,562 (24.2)	15,628 (32.3)	7510 (42.1)	3457 (50.5)
Hypertension	30,051 (57.9)	30,493 (63.0)	12,515 (70.2)	5123 (74.9)
Smoker	8839 (17.0)	6869 (14.2)	2255 (12.7)	813 (11.9)
Prior coronary events (AMI, PCI, CABG)	8794 (17.0)	9544 (19.7)	3243 (18.2)	1012 (14.8)
Peripheral vascular disease	6088 (11.7)	4578 (9.5)	1625 (9.1)	542 (7.9)
Comorbidities				
Congestive heart failure	11,425 (22.0)	8346 (17.2)	3270 (18.4)	1570 (23.0)
Chronic obstructive pulmonary disease	11,353 (21.9)	8575 (17.7)	3213 (18.0)	1357 (19.8)
Cerebrovascular accident/stroke	7502 (14.5)	5955 (12.3)	2166 (12.2)	833 (12.2)
Chronic kidney disease	2643 (5.1)	1915 (4.0)	700 (3.9)	289 (4.2)
HIV/Immunocompromised	932 (1.8)	747 (1.5)	243 (1.4)	91 (1.3)
Cancer	1280 (2.5)	964 (2.0)	260 (1.5)	121 (1.8)
Alzheimer's/Dementia	3297 (6.4)	1587 (3.3)	499 (2.8)	139 (2.0)
Anemia (Hct<30%)	4115 (7.9)	2977 (6.2)	1000 (5.6)	382 (5.6)
Hypoalbuminemia (<3)	2504 (4.8)	1569 (3.2)	596 (3.3)	288 (4.2)
Pre-Admission Status				
Admitted from nursing home	3342 (6.4)	1654 (3.4)	550 (3.1)	220 (3.2)
Mobility at admission				
Walks independently	41,053 (79.1)	40,789 (84.2)	14,691 (82.4)	5197 (76.0)
Walk with assistance	8298 (16.0)	6007 (12.4)	2469 (13.9)	1289 (18.8)

Characteristics	Normal Weight N=51,896 (41.5%)	Overweight N=48,422 (38.7%)	Obese N=17,822 (14.3%)	Morbidly Obese 6841 (5.5%)
Unable to walk	1422 (2.7)	781 (1.6)	338 (1.9)	207 (3.0)
Missing	1123 (2.2)	845 (1.8)	324 (1.8)	148 (2.2)
Urinary continence at admission				
Continent	47,221 (91.0)	45,405 (93.8)	16,591 (93.1)	6271 (91.7)
Totally/Occasionally incontinent	3485 (6.7)	2233 (4.6)	928 (5.2)	441 (6.5)
Anuric	256 (0.5)	123 (0.3)	55 (0.3)	22 (0.3)
Missing	934 (1.8)	661 (1.4)	248 (1.4)	107 (1.6)
Clinical Presentation				
Killip class >2	18,958 (36.5)	16,215 (33.5)	6401 (35.9)	2749 (40.2)
Systolic blood pressure (mmHg), mean(SD)	142.9 (32.4)	147.2 (31.8)	150.1 (32.2)	150.1 (32.9)
Missing	206 (0.4)	161 (0.3)	91 (0.5)	32 (0.5)
Heart rate (bpm), mean (SD)	87.9 (24.9)	86.0 (24.1)	87.5 (23.9)	89.3 (24.0)
STEMI	15,205 (29.3)	14,158 (29.2)	5059 (28.4)	1828 (26.7)
Anterior infarction	24,802 (47.8)	21,915 (45.3)	7953 (44.6)	3094 (45.2)
Cardiac arrest on admission	1572 (3.0)	1335 (2.8)	527 (3.0)	213 (3.1)
Renal insufficiency	6927 (13.4)	4542 (9.4)	1556 (8.7)	670 (9.8)
Treatment				
PCI/CABG in first 30 days	13,984 (27.0)	17,705 (36.6)	6597 (37.0)	2306 (33.7)
Missing	1574 (3.0)	1527 (3.2)	551 (3.1)	209 (3.1)
Fibrinolytic therapy	8533 (16.4)	9916 (20.5)	3517 (19.7)	1285 (18.8)
Aspirin on admission of eligible patients	29,337/38,897 (75.4)	29,690/37669 (78.8)	11,008/14,079 (78.2)	4077/5353 (76.2)
B-blockers on admission of eligible patients	13,797/23,510 (58.7)	15,872/25,429 (62.4)	5848/9163 (63.8)	1956/3192 (61.3)
Crude Mortality Rates				
Hospital	7219 (13.9)	4662 (9.6)	1696 (9.5)	739 (10.8)
30-day	8500 (16.4)	5704 (11.8)	2077 (11.7)	869 (12.7)
1-year	17,523 (33.8)	11,674 (24.1)	4097 (23.0)	1682 (24.6)
5-year	30,841 (59.4)	22,900 (47.3)	8236 (46.2)	3313 (48.4)
17-year	48,770 (94.0)	43,704 (90.3)	16,214 (91.0)	6353 (92.9)

Abbreviations: AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; HIV, Human Immunodeficiency Virus; PCI, percutaneous coronary intervention; SD, standard deviation; STEMI, ST-elevation myocardial infarction.

Table 2

Short and long-term hazards ratios by body mass index category

	30-Day HR (95% CI)	1-Year HR (95% CI)	5-Year HR (95% CI)	17-Year HR (95% CI)
UNADJUSTED				
Normal Weight	1.00	1.00	1.00	1.00
Overweight	0.70 (0.68, 0.73)	0.67 (0.66, 0.69)	0.70 (0.69, 0.72)	0.76 (0.75, 0.77)
Obese	0.69 (0.66, 0.73)	0.64 (0.62, 0.66)	0.68 (0.66, 0.70)	0.77 (0.75, 0.78)
Morbidly Obese	0.76 (0.71, 0.82)	0.69 (0.66, 0.73)	0.72 (0.70, 0.75)	0.83 (0.81, 0.86)
ADJUSTED*				
Normal Weight	1.00	1.00	1.00	1.00
Overweight	0.92 (0.89, 0.95)	0.87 (0.85, 0.89)	0.88 (0.86, 0.89)	0.91 (0.89, 0.92)
Obese	0.96 (0.91, 1.01)	0.86 (0.83, 0.89)	0.88 (0.85, 0.91)	0.93 (0.92, 0.95)
Morbidly Obese	1.02 (0.95, 1.10)	0.88 (0.84, 0.93)	0.88 (0.85, 0.91)	0.98 (0.96, 1.01)

Abbreviations: AMI, acute myocardial infarction; CI, confidence interval; HR, hazard ratio.

* Adjusted for age, gender, race, ZIP code level median household income, cardiovascular risk factors (diabetes, hypertension, smoking, prior coronary events, and peripheral vascular disease), clinical presentation (Killip class, systolic blood pressure on admission, heart rate on admission, ST-elevation AMI, AMI location, and renal insufficiency), therapies received (percutaneous coronary intervention or coronary artery bypass grafting within 30 days of admission, fibrinolytic therapy during the index admission, and among those eligible, aspirin and beta-blockers on admission), cachexia-related factors (congestive heart failure, chronic obstructive pulmonary disease, cerebrovascular disease, chronic kidney disease, Human Immunodeficiency Virus, cancer, dementia, anemia, hypalbuminemia), and measures of pre-hospital frailty (admission from a nursing home, decreased mobility, and urinary incontinence).