Although it is well established that obesity increases the risk for incident cardiovascular (CV) disease, prior work has reported that obese patients experience paradoxically lower in-hospital mortality after non-ST-segment–elevation myocardial infarction than their normal-weight counterparts, yet whether these associations persist long term is unknown.

Methods and Results—We linked detailed clinical data for patients with non-ST-segment–elevation myocardial infarction aged ≥65 years in the Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the American College of Cardiology/American Heart Association Guidelines (CRUSADE) Registry to Medicare claims data to obtain longitudinal outcomes. Using height and weight measured on admission, patients were categorized into 6 body mass index (BMI [kilograms per meter squared]) groups. Multivariable Cox proportional hazards models were used to estimate the association between BMI and (1) all-cause mortality, (2) all-cause readmission, (3) cardiovascular readmission, and (4) noncardiovascular readmission for 3 years after hospital discharge. Among older patients with non-ST-segment–elevation myocardial infarction (n=34,465), 36.3% were overweight and 27.7% were obese. Obese patients were younger and more likely to have hypertension, diabetes mellitus, and dyslipidemia than normal or underweight patients. Relative to normal-weight patients, long-term mortality was lower for patients classified as overweight (BMI, 25.0–29.9), obese class I (BMI, 30.0–34.9), and obese class II (BMI, 35.0–39.9), but not obese class III (BMI ≥40.0). In contrast, 3-year all-cause and cardiovascular readmission were similar across BMI categories. Relative to normal-weight patients, noncardiovascular readmissions were similar for obese class I but higher for obese class II and obese class III.

Conclusions—All-cause long-term mortality was generally lower for overweight and obese older patients after non-ST-segment–elevation myocardial infarction relative to those with normal weight. Longitudinal readmissions were similar or higher with increasing BMI. (Circ Cardiovasc Qual Outcomes. 2014;7:102-109.)

Key Words: acute coronary syndrome • obesity

Although it is well established that obesity increases the risk for incident cardiovascular (CV) disease, prior work has reported that obese patients experience paradoxically better in-hospital outcomes than their normal body mass index (BMI) counterparts after heart failure, chronic kidney disease, coronary artery disease, and diabetes mellitus. This obesity paradox has been well documented for short-term mortality and may be at least partially explained by more aggressive in-hospital management of overweight and obese patients. Nevertheless, whether or not this effect persists long term is unclear. Furthermore, much of the published data represent younger acute coronary syndrome populations, and less information exists on the impact of BMI on outcomes after non-ST-segment–elevation myocardial infarction (NSTEMI) in older adults.

We examined the association between BMI and all-cause mortality, CV rehospitalization, and non-CV rehospitalization during 3 years of follow-up in a population of older patients with NSTEMI.

Methods

Study Population

We used data from the Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the American College of Cardiology/American Heart Association Guidelines (CRUSADE) NSTEMI Registry linked to Medicare claims data to obtain longitudinal outcomes. Details of the CRUSADE study design have been previously published. Briefly, CRUSADE was a national hospital-based initiative designed to track adherence to treatment guidelines and improve the quality of care for patients with NSTEMI.
WHAT IS KNOWN

• Although obesity increases the risk of cardiovascular disease, prior studies have reported lower in-hospital mortality in obese patients with non–ST-segment–elevation myocardial infarction relative to those with normal body mass index.
• This association may be at least partially explained by more aggressive in-hospital management of overweight and obese patients.

WHAT THE STUDY ADDS

• In this large cohort of elderly patients with non–ST-segment–elevation myocardial infarction, we found a U-shaped association between weight and all-cause mortality after non–ST-segment–elevation myocardial infarction, with lower mortality among overweight or obese patients.
• Adjusting for in-hospital treatment did not substantially alter observed associations between body mass index and adverse outcomes, suggesting that the protective effects of increasing body mass index persist despite accounting for more aggressive management.
• Rates of hospital readmissions over 3 years among obese and overweight patients were similar or higher than their normal-weight counterparts.

Trained abstractors collected data on demographics, medical history, signs and symptoms on presentation, laboratory values (including cardiac biomarkers), in-hospital treatments and interventions, discharge therapies, and clinical outcomes for eligible patients. Participating sites were required to obtain approval from local institutional review boards (or equivalent) before data entry.

To capture postdischarge outcomes, we used a previously validated methodology to link clinical data from 442 CRUSADE hospitals to Medicare Part A inpatient claims to identify subsequent all-cause hospitalizations occurring during the 3-year period after discharge. In addition, hospitalizations were classified as either CV related or non-CV related based on a prespecified set of International Classification of Diseases, Ninth Revision discharge codes. Because of the possibility of planned readmission for revascularization procedures after NSTEMI hospitalization, we excluded readmissions for percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery within 60 days of hospital discharge, unless these readmissions were accompanied by a discharge diagnosis that was clearly inconsistent with an elective readmission (ie, heart failure, acute myocardial infarction, unstable angina, arrhythmia, and cardiac arrest). Finally, we created a composite end point of all-cause mortality and CV rehospitalization.

Statistical Analysis

We compared distributions of baseline patient and event characteristics among BMI categories. Differences between BMI groups were assessed using χ² tests for categorical variables and Kruskal–Wallis tests for continuous variables. We used univariable and multivariable Cox proportional hazard models to estimate unadjusted and adjusted associations separately for each end point of all-cause mortality, CV rehospitalization, non-CV rehospitalization, and the composite end point of CV rehospitalization and all-cause mortality. The normal-weight BMI category (BMI, 18.5–24.9 kg/m²) was used as the referent group for all models. All multivariable models were adjusted using a modified set of variables from the CRUSADE long-term mortality model, controlling for age, sex, race, family history of coronary artery disease, current or recent smoking status, prior myocardial infarction, prior PCI, prior coronary artery bypass graft, prior congestive heart failure, prior stroke, heart failure at presentation, heart rate, electrocardiographic findings, initial hematocrit, and initial troponin ratio. Patients were censored at 3 years after hospital discharge in mortality models and on the date of death, on losing fee-for-service eligibility, or at 3 years postdischarge in rehospitalization models. By censoring at the time of mortality, we evaluate associations with the cause-specific hazard for nonfatal end points. This can be interpreted as the hazard ratio among patients who remain at risk (alive and under follow-up). Follow-up information for all-cause mortality at 3 years was complete for all patients. Approximately 5% of patients in the readmission analysis were lost to follow-up because of loss of Medicare Part A eligibility.

The percentage of missing values for all variables used in regression analyses ranged from 0% to 3%. In adjusted models, missing values were imputed to the median of nonmissing values for continuous covariates and the most frequent category for categorical variables. All statistical analyses were performed using SAS software version 9.2 (SAS Institute, Cary, NC).

Results

Characteristics of the patient population at hospital admission are shown in Table 1. The majority of patients in CRUSADE-CMS (Centers for Medicare and Medicaid Services) were overweight or obese (64.0%). The largest proportion of patients were classified as overweight (36.3%), followed by normal weight (32.5%), obese class I (17.7%), obese class II (6.5%), underweight (3.6%), and obese class III (3.6%). We observed an inverse association between BMI and age, with a median age of 72 years (interquartile range, 68–77) for obese class III, compared with a median age of 80 years (interquartile range, 74–86) in the normal-weight group. Obese patients had higher rates of comorbidities, including hypertension, diabetes mellitus, and dyslipidemia than normal-weight patients. Normal-weight and underweight patients were significantly more likely to be smokers than overweight and obese patients. Signs and symptoms at presentation were similar across categories, with the exception of heart rate, which was highest in underweight

Body Mass Index

BMI was calculated using weight and height values recorded on hospital admission. Patients were categorized into the following 6 BMI classes according to World Health Organization standards²: (1) underweight (BMI <18.5 kg/m²); (2) normal weight (BMI, 18.5–24.9 kg/m²); (3) overweight (BMI, 25.0–29.9 kg/m²); (4) obese I (BMI, 30.0–34.9 kg/m²); (5) obese II (BMI, 35.0–39.9 kg/m²); and (6) obese III (BMI ≥40.0 kg/m²). BMI was treated as a categorical variable for all analyses.

Outcomes

We searched Medicare claims records for information on vital status and rehospitalization for 3 years after hospital discharge. All-cause mortality was ascertained from Medicare denominator files. We used Medicare Part A inpatient claims to identify subsequent all-cause hospitalizations occurring during the 3-year period after discharge. In addition, hospitalizations were classified as either CV related or non-CV related based on a prespecified set of International Classification of Diseases, Ninth Revision discharge codes. Because of the possibility of planned readmission for revascularization procedures after NSTEMI hospitalization, we excluded readmissions for percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery within 60 days of hospital discharge, unless these readmissions were accompanied by a discharge diagnosis that was clearly inconsistent with an elective readmission (ie, heart failure, acute myocardial infarction, unstable angina, arrhythmia, and cardiac arrest). Finally, we created a composite end point of all-cause mortality and CV rehospitalization.

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and obese class III patients. Signs of heart failure were least common among overweight and obese class I patients. Table 2 shows the use of in-hospital medications, invasive procedures, discharge medications, and in-hospital events by BMI category. In the first 24 hours of hospitalization, overweight and obese patients were more likely to receive aspirin, glycoprotein IIa/IIIb inhibitors, and clopidogrel than normal-weight patients. Rates of invasive procedures, including cardiac catheterization, PCI, and coronary artery bypass graft, were also higher among overweight and obese patients than among normal-weight patients. At hospital discharge, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers and lipid-lowering medications were more commonly prescribed to overweight and obese patients than to normal-weight patients. The lowest rates of both CRUSADE major bleeding events and in-hospital congestive heart failure were observed among overweight, obese class I, and obese class II patients. No significant differences in rates of cardiogenic shock were found between BMI groups.

Conditional on surviving to hospital discharge, all-cause mortality at 3 years in the total study population was 36.6%. Unadjusted 3-year mortality was highest among underweight patients (62.4%), followed by normal-weight patients (45.6%). Obese class I patients had the lowest 3-year rates of unadjusted mortality of the 6 BMI groups (28.0%; Figure 1).

Unadjusted and adjusted hazard ratios for all-cause mortality, rehospitalization (CV and non-CV), and the composite end point of death and CV rehospitalization are shown for the 6 BMI classes in Figure 2 (unadjusted) and Figure 3 (adjusted). After adjustment, when compared with normal-weight patients, overweight and obese patients class I and II had lower all-cause mortality, obese class III patients had similar outcomes, and underweight patients had significantly higher mortality.

### Table 1. Baseline Patient and Event Characteristics by Body Mass Index Category

<table>
<thead>
<tr>
<th>Variable</th>
<th>Underweight* (n=1236)</th>
<th>Normal Weight (n=11186)</th>
<th>Overweight (n=12506)</th>
<th>Obese I (n=6089)</th>
<th>Obese II (n=2226)</th>
<th>Obese III (n=1222)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>82 (75–86)</td>
<td>80 (74–86)</td>
<td>77 (71–82)</td>
<td>75 (69–80)</td>
<td>73 (69–79)</td>
<td>72 (68–77)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Male sex</td>
<td>30.7</td>
<td>49.3</td>
<td>59.4</td>
<td>54.7</td>
<td>46.1</td>
<td>35.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>45.4 (41.3–50.1)</td>
<td>63.0 (56.2–70.3)</td>
<td>78.1 (70.8–85.7)</td>
<td>90.5 (81.6–99.8)</td>
<td>101.2 (90.9–111.9)</td>
<td>117 (104.6–132.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>White race</td>
<td>83.8</td>
<td>86.7</td>
<td>86.7</td>
<td>86.5</td>
<td>86.3</td>
<td>84.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>71.1</td>
<td>73.5</td>
<td>76.2</td>
<td>81.2</td>
<td>84.6</td>
<td>86.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>16.9</td>
<td>25.4</td>
<td>34.3</td>
<td>44.8</td>
<td>55.7</td>
<td>61.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>33.9</td>
<td>46.5</td>
<td>54.6</td>
<td>59.3</td>
<td>60.7</td>
<td>58.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Current/recent smoker</td>
<td>19.7</td>
<td>14.3</td>
<td>12.5</td>
<td>10.6</td>
<td>10.1</td>
<td>9.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>21.0</td>
<td>24.4</td>
<td>28.0</td>
<td>29.6</td>
<td>31.4</td>
<td>30.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Prior PAD</td>
<td>13.8</td>
<td>15.2</td>
<td>14.6</td>
<td>14.3</td>
<td>13.3</td>
<td>14.2</td>
<td>0.1880</td>
</tr>
<tr>
<td>Prior CHF</td>
<td>29.0</td>
<td>23.6</td>
<td>19.9</td>
<td>20.6</td>
<td>24.1</td>
<td>32.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Prior MI</td>
<td>26.9</td>
<td>30.6</td>
<td>30.3</td>
<td>31.3</td>
<td>30.9</td>
<td>29.5</td>
<td>0.0815</td>
</tr>
<tr>
<td>Prior stroke</td>
<td>15.3</td>
<td>14.8</td>
<td>12.6</td>
<td>12.5</td>
<td>12.5</td>
<td>12.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Prior CABG</td>
<td>15.2</td>
<td>22.6</td>
<td>25.2</td>
<td>25.0</td>
<td>24.8</td>
<td>21.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>13.0</td>
<td>18.7</td>
<td>22.1</td>
<td>24.3</td>
<td>24.8</td>
<td>25.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Signs/symptoms at presentation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Signs of HF</td>
<td>35.4</td>
<td>31.4</td>
<td>27.5</td>
<td>27.6</td>
<td>30.2</td>
<td>36.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SBP</td>
<td>137 (116–159)</td>
<td>142 (121–163)</td>
<td>145 (125–166)</td>
<td>147 (127–168)</td>
<td>147 (126–170)</td>
<td>147 (128–170)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>90 (76–107)</td>
<td>85 (71–102)</td>
<td>82 (70–99)</td>
<td>83 (70–100)</td>
<td>84 (70–100)</td>
<td>87 (74–103)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>37.6 (33.7–41.2)</td>
<td>38.3 (34.4–42.0)</td>
<td>39.5 (35.6–43.0)</td>
<td>39.8 (35.9–43.4)</td>
<td>39.8 (35.7–43.1)</td>
<td>38.8 (35.1–42.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td>1.1 (0.9–1.4)</td>
<td>1.2 (0.9–1.5)</td>
<td>1.2 (1.0–1.5)</td>
<td>1.2 (1.0–1.5)</td>
<td>1.2 (0.9–1.5)</td>
<td>1.2 (1.0–1.6)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Troponin ratio, ×ULN</td>
<td>3.2 (1.0–12.0)</td>
<td>2.4 (0.7–10.3)</td>
<td>2.0 (0.6–9.3)</td>
<td>2.0 (0.5–8.7)</td>
<td>1.8 (0.4–7.8)</td>
<td>1.9 (0.4–7.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ECG findings</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST depression</td>
<td>24.2</td>
<td>28.5</td>
<td>27.3</td>
<td>26.6</td>
<td>26.2</td>
<td>25.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Transient ST elevation</td>
<td>3.6</td>
<td>3.6</td>
<td>3.2</td>
<td>3.2</td>
<td>3.4</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>Both</td>
<td>0.7</td>
<td>0.8</td>
<td>1.1</td>
<td>0.5</td>
<td>0.4</td>
<td>0.6</td>
<td></td>
</tr>
</tbody>
</table>

All data are presented as percentages except age, weight, SBP, heart rate, hematocrit, and creatinine which are presented as median (interquartile range). CABG indicates coronary artery bypass grafting; CAD, coronary artery disease; CHF, congestive heart failure; HF, heart failure; MI, myocardial infarction; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; and ×ULN, times upper limit of normal.

*World Health Organization body mass index categories (kilograms per meter squared): underweight <18.5; normal weight, 18.5–24.9; overweight, 25.0–29.9; obese class I, 30.0–34.9; obese class II, 35.0–39.9; obese class III ≥40.0.

†P values from χ² tests for categorical variables and Kruskal–Wallis tests for continuous variables.
Being overweight was associated with a modest reduction in all-cause readmission, but this effect was attenuated for obese class I patients and reversed for obese class II and III patients (Figure 3). Adjusted risk of CV readmission was similar across BMI categories. We observed a lower unadjusted risk of non-CV rehospitalization among overweight and obese class I patients, but this effect was attenuated after adjustment. Underweight patients had a slightly higher adjusted risk of non-CV readmission compared with normal-weight patients. For the composite end point of all-cause mortality and CV readmission, overweight and obese patients had lower unadjusted rates than normal-weight patients; however, absolute differences in rates of the composite end point were minimal after adjustment. Being underweight was associated with a slightly higher risk of the composite end point after adjustment.

**Discussion**

To our knowledge, this is the first study of long-term outcomes and BMI in an older NSTEMI population. We had several major findings. First, the majority of older patients with NSTEMI were overweight or obese (64.0%). Second, age at time of myocardial infarction varied inversely with BMI; the oldest patients were in the underweight and normal-weight categories, whereas the youngest patients were in the obese class II and III categories. Third, overweight and obese patients generally have similar or lower all-cause mortality for 3 years after NSTEMI. Finally, although adjusted rates of CV rehospitalization were comparable across BMI categories, non-CV–related rehospitalization was similar or higher with increasing BMI.

The findings from our study are largely consistent with those who have previously reported short-term survival benefits associated with obesity during and directly after acute coronary syndrome hospitalization. In 1 study of BMI and CV outcomes among 4762 patients undergoing PCI in Australia, increasing BMI was associated with linear reductions in major adverse CV events both in hospital and at 12 months postdischarge. In the long-term follow-up of 6560 non–ST-segment acute coronary syndrome participants in the Metabolic Efficiency With Ranolazine for Less Ischemia in Non–ST-Elevation Acute Coronary Syndromes–Thrombolysis in Myocardial Infarction (MERLIN-TIMI) 36 trial suggest the short-term protective effect of obesity. Finally, in a follow-up study of 7427 patients undergoing PCI with drug-eluting stents, lower rates of mortality at 5 years were reported for overweight and obese patients compared with normal-weight patients.

Several physiological and methodological explanations for the obesity paradox have been presented, with varying levels of empirical support. For example, the obesity paradox has been attributed to increased metabolic reserve in obese individuals, which may confer a survival advantage during critical times. However, the mechanisms underlying the obesity paradox remain unclear, and further research is needed to fully understand the complex interplay between BMI and long-term outcomes.
protective in older populations, the observed direction of this
effect is often reversed in younger cohorts, suggesting that
these associations are likely age dependent. In addition, it
has been hypothesized that unexpected BMI mortality associa-
tions may be the result of study design. The obesity paradox
has been documented largely in observational, disease-based
registries, which are vulnerable to issues of selection bias.
Because obese patients experience acute coronary events at
younger ages and have shorter life expectancy than normal-
weight patients, it is possible that only the healthiest obese
patients survive long enough to be hospitalized and captured in
these study populations. If unmeasured extraneous factors
are present among the older obese patients that both confer a sur-
vival advantage and are not present in normal-weight partici-
pants, bias may be introduced to mortality estimates. This type
of survival bias is more evident in associations with short-term
outcomes and theoretically diminishes with increasing follow-
up time. Nonetheless, we observed lower mortality among
overweight and obese patients for 3 years after discharge, sug-
gest that the obesity paradox persists beyond the short term.

In addition to population-level hypotheses, some evidence
suggests that obesity’s protective effect operates through bio-
ological mechanisms that differ by BMI subclass. Compared
with patients in normal and low BMI categories, patients
with high BMI are likely to have increased total lean mass
in addition to increased adiposity—both of which may offer
advantages in the context of acute CV disease. Lean mass
has important homeostatic benefits, including better glucose
and oxygen metabolism, which may positively influence sur-
vival. Patients in normal and low BMI categories are more
likely to have low levels of lean mass, a condition associated
with metabolic dysregulation and increased mortality. Fur-
thermore, because low BMI is associated with lower adi-
posity, patients in underweight and normal-weight categories
may be more vulnerable to adverse cardiometabolic effects of
chronic disease. Patients experiencing acute events undergo
catabolic changes, including inflammation and activation of
neurohormonal systems, which require additional energy
reserves. Increased subcutaneous fat may provide resistance
to the catabolic burden associated with acute cardiac events.

Finally, a developing explanation for the obesity paradox
suggests that more aggressive treatment is received by over-
weight and obese patients during the index hospitalization.
Physicians may perceive overweight and obese patients to be
at higher risk for adverse outcomes or be more likely to rec-
ognize comorbidities and thus may be more likely to prescribe
evidence-based therapies and interventions to patients with
higher BMI. In a 2006 analysis of BMI and in-hospital mortal-
ity in CRUSADE, Diercks et al reported increased use of diag-
nostic catheterization, PCI, and coronary artery bypass graft
among overweight and obese patients compared with those
of a normal weight. Nevertheless, adding treatment received
to models of in-hospital mortality did not substantially alter
observed associations between BMI and adverse outcomes,
suggesting that the protective effects of increasing BMI per-
sist despite accounting for more aggressive management.

Our study has several limitations. First, although we were able
to examine mortality and rehospitalization outcomes up to 3 years
postdischarge, it is possible that obesity is not protective beyond
this time period. Because we were limited to a single measure-
ment of BMI on hospital admission, we were unable to assess
the impact of changes in weight status over time. Second, prior

![Mortality incidence by body mass index (BMI) category. Unadjusted cumulative incidence of mortality (%) by BMI category during 3 years of follow-up is displayed. *World Health Organization BMI categories (kilograms per meter squared): underweight <18.5; normal weight, 18.5 to 24.9; overweight, 25.0 to 29.9; obese class I, 30.0 to 34.9; obese class II, 35.0 to 39.9; obese class III ≥40.0.*](http://circoutcomes.ahajournals.org/)}
studies have reported positive mortality associations for other measures of adiposity, such as waist-to-hip ratio or waist circumference. Because these were not captured in CRUSADE, we were unable to examine whether these associations persisted in our study population. Third, because cause-of-death information was not available in our study, we were unable to assess whether BMI associations persisted by death classification. Fourth, our population experienced a high rate of mortality, which represents a competing risk for nonfatal end points. We handled this by studying associations with the cause-specific hazard, which is estimable from the observed data, but does not provide an indication of what could have happened if death did not occur. Finally, because we evaluated the association between obesity and long-term mortality after NSTEMI in an older, mostly white population, our results may not be generalizable to younger populations or those with different racial or ethnic distributions.

In summary, we found that overweight and obese patients with NSTEMI experience lower all-cause mortality than their normal-weight counterparts for 3 years after hospital discharge. Further examination is needed regarding the possible mechanisms for obesity’s protective effect in older acute coronary syndrome populations.

**Acknowledgments**

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Association of Body Mass Index and Long-Term Outcomes in Older Patients With Non–ST-Segment–Elevation Myocardial Infarction: Results From the CRUSADE Registry
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