Relation Between Body Mass Index, Waist Circumference, and Death After Acute Myocardial Infarction

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Methods and Results—We evaluated 2229 consecutive patients with AMI. Patients were classified according to BMI as normal, overweight, obese, and very obese (BMI <25, 25 to 29.9, 30 to 34.5, and >35 kg/m², respectively) and as increased waistline (WC >88/102 cm for women/men) or normal. Half of the patients were overweight (n=1044), and one quarter were obese (n=397) or very obese (n=128). Increased WC was present in half of the patients (n=1110). Increased BMI was associated with a reduced death rate, with a 5% risk reduction for each unit increase in BMI (hazard ratio, 0.95; 95% CI, 0.93 to 0.98; P<0.001). In contrast, WC as a continuous variable had no impact on all-cause death (P=0.20). After adjustment for baseline predictors of death, BMI was not independently predictive of death. The group of patients with high WC but low BMI had increased 1-year death rate.

Conclusions—Neither BMI nor WC independently predicts death after AMI. Much of the inverse relationship between BMI and the rate of death after AMI is due to confounding by characteristics associated with survival. This study emphasizes the need to measure both BMI and WC because patients with a high WC and low BMI are at high risk of death. (Circulation. 2008;118:482-490.)

Key Words: body mass index ▪ death ▪ myocardial infarction ▪ obesity

Obesity is a major risk factor for the development of fatal and nonfatal cardiovascular (CV) events. In patients with established CV disease, its impact on risk is less clear. Increased body mass index (BMI) is associated with a higher risk of acute myocardial infarction (AMI). Several studies, however, have reported an apparent paradoxical effect of BMI on outcomes: In patients undergoing elective percutaneous coronary intervention (PCI), BMI was inversely associated with worse outcomes, with obese and overweight patients having improved survival compared with those with normal BMI. In patients with AMI, high BMI appears to have an unexplained protective effect on survival. The role of increased BMI as an independent CV risk factor is controversial. In patients with coronary artery disease, BMI does not adequately discriminate between body fat and lean body mass and may thus help to explain the controversy known as the obesity paradox. Abdominal obesity, which reflects central body fat distribution, has been suggested as a better marker of the obesity risk.

Wrist circumference (WC) is an anthropometric index usually considered a surrogate marker of abdominal fat mass (subcutaneous and intraabdominal). High WC values are associated with CV complications, with a predictive value superior to that of BMI. Despite the high prevalence of abdominal obesity in patients admitted with AMI, there is a paucity of data on its impact on prognosis after the acute event. Whether increased waistline is involved in the obesity paradox...
remains to be determined. Thus, from a contemporary prospective study of unselected consecutive patients, we compared the prognostic impact of obesity, assessed by both BMI and WC, on short- and intermediate-term mortality after AMI.

Methods

Patients

The design and methods of the Observatoire des Infarctus de Côte d’Or (RICO) Survey have been published.14 Patients presenting with diagnosed AMI15 were included in the present study (see the online Data Supplement). The present study complied with the Declaration of Helsinki and was approved by the ethics committee of University Hospital of Dijon. Each patient gave written consent before participation.

Data Collection

Data on demographics, CV risk factors (history of hypertension, diabetes, treated hyperlipidemia, current smoking), and prior myocardial infarction were collected prospectively, along with admission characteristics and hemodynamic parameters. Blood samples were drawn at admission. Plasma creatinine levels were measured on a Vitros 950 analyzer (Ortho Clinical Diagnostics, Rochester, NY). High-sensitivity C-reactive protein was measured on Dimension Xpand (Dade Behring, Newark, Neb) with an immunonephelometry assay. Plasma N-terminal pro B-type natriuretic peptide (NT-proBNP) was determined by ELISA with an Elecsys NT-proBNP sandwich immunoassay on Elecsys 2010 (Roche Diagnostics, Basel, Switzerland). Overnight fasting blood samples were collected on the morning after admission for blood lipid measurements. High-density lipoprotein cholesterol and triglyceride concentrations were measured on a Dimension analyzer (Dade Behring). The level of low-density lipoprotein cholesterol was calculated from the Friedewald formula.

Echocardiography was performed at day 3±1 by a local investigator according to the Simpson method using the apical views to calculate left ventricular ejection fraction (LVEF). Data on acute (<24 hour) reperfusion procedures (thrombolysis or PCI) were collected. Heart failure, defined as rales over more than half of the lung field (Killip class II), pulmonary edema (Killip class III), or cardiogenic shock (Killip class IV), was evaluated on admission. Duration of hospital stay in the coronary care unit also was collected. After hospital discharge, 30-day or 1-year information was acquired by contacting either each patient individually, their relatives, or treating physician and by reviewing the hospital records if the patient had been rehospitalized. No patient was lost to follow-up (see the Data Supplement).

Group Definition

Anthropometric parameters were measured within 48 hours of admission. BMI was categorized according to the World Health Organization16 standards as normal (≤25 kg/m²), overweight (25 to 29.9 kg/m²), obese (30 to 34.9 kg/m²), and very obese (≥35 kg/m²). WC was measured with a nonelastic tape at the mid distance between the top of the iliac crest and the bottom of the rib cage and as the average of 1 measurement taken after inspiration and 1 taken after expiration. Increased WC was defined using the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP) cutoff of WC >102 cm in men and >88 cm in women.17,18 Data for both BMI and WC were split into sex-specific tertiles for analysis.

Statistical Analysis

Data are presented as median (25th to 75th percentile) or mean±SD as appropriate or as proportion. For continuous variables, a Kolmogorov-Smirnov analysis was performed to test for normality. For the tests across tertiles, we performed the Kruskal-Wallis 1-way ANOVA by rank for nonnormally distributed values or 1-way ANOVA for normally distributed values. Qualitative variables, expressed as numbers and percentiles, were compared by the χ² test for trends. Spearman’s rank correlation was applied to test for associations between continuous variables. The cumulative incidence of all-cause death was estimated according to the Kaplan–Meier method, and the log-rank test was used to evaluate differences between groups. Cox regression analysis was performed to determine the effects of WC or BMI as continuous variables on the rate of death in unadjusted models (models 1 and 2, respectively). BMI was then tested by multivariate analysis. Age, sex (female as reference), and NT-proBNP were individually tested as covariates given the strong correlation between BMI and age or NT-proBNP and because age and NT-proBNP are major contributors to prognosis after MI. The other variables tested in univariate analysis were those known to potentially affect the outcome after MI and variables showing a variation according to BMI tertiles. Overall, the variables tested were the following: age, female gender, diabetes, hypertension, dyslipidemia, smoking, prior MI, acute therapies (statin, β-blockers, angiotensin-converting enzyme inhibitors), biological parameters (NT-proBNP, C-reactive protein, creatinine, high-density lipoprotein cholesterol, and triglycerides), Killip class ≥1, ST-elevation myocardial infarction (STEMI), and LVEF. Among these, age, sex, NT-proBNP, acute therapy, Killip class ≥1, prior MI, hypertension, diabetes, hyperlipidemia, smoking, C-reactive protein, STEMI, LVEF, and creatinine were predictors of prognosis and therefore were included as covariates in multivariate analysis. Because smoking is a potential confounder of the relationship between BMI and the outcome, its interaction was tested in univariate analysis and introduced as a covariate in multivariate analysis. Linearity of the continuous variables with respect to the response variable was assessed by determining the quartile of their distribution. Subsequently, hazard ratios (HRs) for each quartile were calculated. All variables showed a linear trend in the estimated HRs and thus were introduced into the model as continuous. The proportional-hazards assumption in the Cox models was assessed with graphical methods (log-log plots) and with models including time-by-covariate interactions. No violations of the proportional-hazards assumption were identified. Statistical analyses were performed with SPSS software (SPSS, Inc, Chicago, Ill).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Study Population

A total of 2229 patients were included in the study. Fewer than one third (n=660) had a normal BMI; half of the patients were overweight (46.8%, n=1044); and one quarter were obese (n=397) or very obese (n=128). Half of the patients (49.8%, n=1110) had increased WCs.

The patients’ characteristics are summarized in Table 1 for women and Table 2 for men. Time to admission, MI location, and LVEF were similar across BMI subgroups for both sexes. In elevated WC tertiles, heart failure as assessed by Killip class on admission was more frequent in men. For both sexes, elevated systolic blood pressure was found across the BMI or WC tertiles. In men, acute PCI was used less frequently in the high BMI groups, whereas statins and angiotensin-converting enzyme inhibitors were prescribed more frequently. C-reactive protein, creatinine, and triglyceride levels markedly increased with elevated WC, whereas high-density lipoprotein cholesterol decreased. In women, NT-proBNP levels showed no significant changes across anthropometric categories, whereas in men, the propeptide levels were reduced across BMI tertiles.

BMI as a continuous variable was inversely correlated with age and NT-proBNP (r=-0.13, P<0.001 and r=-0.11, P<0.001). In contrast, WC as a continuous variable was
positively correlated with age ($r=0.07$, $P=0.001$) but not with NT-proBNP ($r=0.03$, $P=0.155$). See the online Data Supplement for sex-specific correlations.

**Outcomes**

At 1 year, there were 253 CV deaths (11.4%) and 301 all-cause deaths (13.5%), including 30-day CV (167 [7.5%]) and all-cause (186 [8.3%]) deaths during a median follow-up of 346 days (25th to 75th percentile, 338 to 354 days). Sex-specific death rates were reported in an additional table in the Data Supplement. The rate of death at 1 year was significantly reduced in men with increasing BMI tertiles ($P=0.016$). No significant variation in the rate of death was found across WC tertiles for both sexes. These findings were confirmed by Kaplan–Meier analysis of cumulative survival curves. Death rates across BMI tertiles were not significant in...
women (Figure 1A) (P=0.158). In men, a lower death rate was observed with increasing BMI tertiles (log-rank P=0.019) (Figure 1B). No increased risk was found across WC tertiles in either women (Figure 2A) (P=0.321) or men (Figure 2B) (P=0.547). When analyzed as a continuous variable, BMI also was significantly associated with death, with a 5% reduction in the risk of death for each unit increase in BMI (HR, 0.95; 95% CI, 0.93 to 0.98; P<0.001) (Figure 3, model 2). In contrast, WC as a continuous variable had no impact on all-cause death in the whole study population (HR, 1.00; 95% CI, 1.00 to 1.01; P=0.20) (Figure 3, model 1), men alone (HR, 1.00; 95% CI, 1.00 to 1.01; P=0.20), or women alone (HR, 1.01; 95% CI, 1.00 to 1.02; P=0.11). Multivariate analysis showed that when age (model 3), sex (model 4), NT-proBNP (model 5), or covariates that had a significant impact on outcomes (model 6) were added to the models, BMI was not an independent predictor of outcome (Figure 3). Moreover, no significant interaction between C-reactive pro-

| Table 2. Characteristics of the Study Population According to BMI and WC on Admission in Men (n=1636) |
|-----------------------------------------------|----------------|----------------|----------------|----------------|----------------|
| BMI Tertiles, kg/m                            | T1             | T2             | T3             | P              |
| WC Tertiles, cm                               | 89 (84–92) [56–95] | 100 (97–102) [95–105] | 111 (108–118) [105–158] | <0.001 |

**Risk factors**
- Age, y* 67 (54–77) 67 (55–76) 61 (51–73) <0.001 62 (51–74) 66 (54–75) 66 (55–75) 0.002
- Current smoking, n (%) 208 (38.2) 153 (28.1) 158 (28.9) <0.001 212 (38.8) 160 (29.4) 147 (26.9) <0.001
- Prior MI, n (%) 88 (16.1) 87 (16.0) 79 (14.5) 0.70 69 (12.7) 96 (17.6) 89 (16.3) 0.06
- Hypertension, n (%) 223 (40.9) 279 (51.2) 323 (59.2) <0.001 202 (37.1) 274 (50.3) 349 (63.9) <0.001
- Diabetes, n (%) 93 (17.1) 100 (18.3) 168 (30.8) <0.001 77 (14.1) 108 (19.8) 176 (32.2) <0.001
- Dyslipidemia, n (%) 222 (40.7) 263 (48.3) 285 (52.2) 0.001 229 (42.0) 266 (48.8) 275 (50.4) 0.013

**Clinical data**
- Time from symptom onset to admission, min* 180 (90–420) 180 (90–462) 180 (101–481) 0.46 180 (90–420) 180 (95–500) 195 (100–487) 0.31
- Killip class I>1 on admission 101 (18.5) 103 (18.9) 103 (18.9) 0.98 76 (13.9) 107 (19.6) 124 (22.7) 0.001
- Heart rate, bpm* 75 (62–89) 75 (64–89) 77 (67–89) 0.09 73 (61–87) 75 (84–90) 78 (87–90) <0.001
- Systolic blood pressure, mm Hg* 130 (110–152) 139 (121–160) 140 (125–165) <0.001 131 (116–150) 138 (120–160) 141 (126–167) <0.001
- Diastolic blood pressure, mm Hg* 80 (70–90) 80 (70–92) 80 (70–95) <0.001 80 (70–90) 80 (70–93) 80 (70–93) 0.015
- STEMI, (%) 331 (60.7) 334 (61.3) 296 (54.2) 0.031 338 (62.0) 328 (60.2) 295 (54.0) 0.019
- Anterior wall location, % 201 (36.9) 198 (36.3) 191 (35.0) 0.80 208 (38.2) 189 (34.7) 193 (35.3) 0.44
- LVEF, %* 55 (45–64) 55 (43–64) 55 (45–63) 0.48 55 (45–65) 54 (43–63) 55 (44–63) 0.09

**Biological data**
- hs-CRP, mg/L* 3.4 (1.6–10.1) 3.8 (1.6–7.2) 4.5 (2.2–10.0) 0.015 3.2 (1.5–8.0) 3.8 (1.7–8.2) 4.7 (2.3–10.2) <0.001
- Creatinine, µmol/L* 90 (80–111) 94 (80–115) 96 (80–108) 0.30 89 (80–106) 94 (80–115) 98 (80–115) <0.001
- NT-proBNP, pg/mL* 826 (212–2962) 766 (220–2589) 624 (169–2042) 0.008 629 (166–2134) 735 (192–2683) 832 (243–2651) 0.05
- LDL-C, mg/dL* 122 ± 38 121 ± 38 123 ± 36 0.84 127 ± 37 120 ± 37 119 ± 38 0.006
- HDL-C, mg/dL* 41 (34–51) 40 (31–48) 35 (27–44) <0.001 41 (33–51) 38 (31–48) 36 (28–45) <0.001
- Triglycerides, mg/dL* 100 (74–135) 109 (83–159) 145 (100–222) <0.001 99 (74–145) 116 (82–162) 136 (84–206) <0.001

**Acute treatments**
- <48 h, %
  - Primary PCI 118 (21.7) 84 (15.4) 91 (16.7) 0.018 111 (20.4) 95 (17.4) 87 (15.9) 0.15
  - Thrombolysis 132 (24.0) 134 (24.6) 132 (24.2) 0.98 141 (25.8) 141 (25.8) 116 (21.2) 0.12
  - β-Blocker 431 (79.1) 436 (80.0) 454 (83.2) 0.20 451 (82.8) 438 (80.4) 432 (78.1) 0.30
  - ACE inhibitor 327 (60.0) 319 (58.5) 384 (70.3) <0.001 335 (61.5) 330 (60.6) 365 (66.8) 0.07
  - Statin 417 (76.5) 445 (81.7) 466 (85.3) 0.001 428 (78.5) 444 (81.5) 456 (83.5) 0.11
- Duration of stay in CCU, d* 4 (3–5) 4 (3–6) 4 (3–5) 0.13 4 (3–5) 4 (3–5) 4 (3–5) 0.73

**Abbreviations as Table 1.**
*Median (25th to 75th percentile).
tein and either BMI \( (P=0.53) \) or WC \( (P=0.61) \) was found for outcome.

Stratification of death rates according to both BMI and WC tertiles showed that for both sexes, a high WC is associated with increased rate of death in the 2 lowest BMI tertiles, which identified a subgroup of high-risk patients (Figure 4A and 4B). In male subjects, a high WC but low BMI is observed in almost one quarter of patients (2 of 9) and is associated with a high death rate (almost 1 in 5) (Figure 4B). To further investigate the capacity of BMI to predict death independently of WC, we performed a subgroup analysis on 832 waist-matched patients comparing the outcome of low BMI \( (n=416) \) and high BMI \( (n=416) \). The classification of BMI was based on a median BMI value (26 kg/m²) defining low (inframedian) and high (supramedian) BMI groups. The median WC was 100 cm (25th to 75th percentile, 96 to 103 cm) for both groups. The median BMI was 24 kg/m² (25th to 75th percentile, 23 to 25 kg/m²) for the low BMI and 27 kg/m² (25th to 75th percentile, 27 to 28 kg/m²) for the high BMI group. In this new data set, age and NT-proBNP were significantly different in the low versus high BMI group (75 years [25th to 75th percentile, 65 to 81 years] versus 67 years [25th to 75th percentile, 54 to 76 years], \( P<0.001 \); and 1781 pg/mL [25th to 75th percentile, 431 to 6308 pg/mL] versus 872 [25th to 75th percentile, 190 to 2666 pg/mL], \( P<0.001 \), respectively). In addition, as expected, the rate of death remained markedly increased in the low versus high BMI
group (91 [22%] versus 55 [13%), respectively; \( P=0.001 \)).

Analysis of this subgroup of patients matched for WC confirmed that for the same WC, BMI was still relevant to discriminate patients at high risk.

**Discussion**

In the present study, based on a large, unselected cohort of AMI patients, neither BMI nor WC independently predicted death after acute MI. Much of the apparent obesity paradox

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**Figure 3.** HRs for each unit increase in BMI (per kg/m²) or WC (per cm) for 1-year death rates in unadjusted and adjusted models.

**Table:**

<table>
<thead>
<tr>
<th>Models</th>
<th>Hazard ratios (95% CI) for 1 year mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Per cm increase</td>
</tr>
<tr>
<td>Model 1: WC (unadjusted)</td>
<td>1.00 (1.00-1.01) 0.20</td>
</tr>
<tr>
<td>Model 2: BMI (unadjusted)</td>
<td>0.95 (0.93-0.98) &lt;0.001</td>
</tr>
<tr>
<td>Model 3: model 2+age</td>
<td>0.98 (0.96-1.01) 0.21</td>
</tr>
<tr>
<td>Model 4: model 3+female</td>
<td>0.98 (0.95-1.01) 0.18</td>
</tr>
<tr>
<td>Model 5: model 4+NT-proBNP</td>
<td>1.02 (0.99-1.05) 0.18</td>
</tr>
<tr>
<td>Model 6: model 5+covariates*</td>
<td>1.01 (0.97-1.06) 0.60</td>
</tr>
</tbody>
</table>

* Covariates: acute therapy, Killip\textgreater I, prior MI, hypertension, diabetes, hyperlipidemia, smoking, CRP, STEMI, LVEF, creatinine, BMI*smoking.

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**Figure 4.** Stratification of 1-year death rates according to both BMI and WC tertiles in women (A) and men (B).
(the inverse relationship between BMI and death after MI) is related to confounding by differences in baseline characteristics that predict survival after MI. Moreover, we identified a group of high-risk patients, ie, those with a high WC and a low BMI, compared with patients with both high WC and BMI values.

**BMI-Based Obesity**

In this contemporary, population-based French registry of patients with AMI, the prevalence of overweight and obesity was very high (70%), reflecting the epidemic nature of obesity in Western Europe. This finding underlines the importance of obesity as a CV risk factor and underscores the need to improve our understanding of the relationship between excess weight and CV outcomes. We found a marked association between increasing BMI and younger age in men that was even stronger in women. This association, which illustrates the premature occurrence of AMI, has been reported in previous studies. In accordance with our results, an inverse independent relationship between NT-proBNP and lean mass, rather than fat mass, has been demonstrated in the Framingham Heart Study and the Dallas Heart Study. The mechanisms linking low natriuretic peptide levels and obesity remain to be elucidated but probably involve decreased release of natriuretic peptides from the heart rather than increased clearance. Excess weight was associated with an elevated incidence of the obesity-associated CV risk factors of hypertension, diabetes, and dyslipidemia, also consistent with a high-risk profile. Rates of primary PCI were decreased in higher BMI categories, a finding that differs from the results of a randomized trial showing a more aggressive use of PCI in patients with higher BMI. The use of acute medications was similar across the BMI subgroups, except for statins and angiotensin-converting enzyme inhibitors, which were prescribed more often in patients with a high BMI, a group with a higher prevalence of dyslipidemia at baseline.

**Obesity Paradox**

An apparent protective effect of high BMI on mortality has been found in randomized trials of patients with unstable angina and non-STEMI, or both. In contrast, our data, from a registry study of unselected patients, showed that after adjustment, BMI was not a prognostic factor after myocardial infarction, a finding that is consistent with registry-based studies in AMI. A recent study in patients with non-STEMI found that obesity was significantly, albeit weakly, correlated with improved outcomes after adjustment for confounding prognostic factors. The major impact of younger age in the apparent protection conferred by obesity also has been reported in most recent studies. Moreover, young age may drive an increased use of medications and procedures, a factor that may have a favorable impact on outcomes. Interesting data from the Mayo Clinic registry on MI patients showed that obesity paradox, which is present in the short term, disappears in the long term. In our study, no data were available on in-hospital death rates because only a 30-day follow-up was performed. However, the data at 30 days, although not significant, showed the same trend as the 1-year follow-up, ie, a decrease in the rate of death with increasing BMI. Our findings are confirmed by major contemporary studies in acute coronary syndromes that address the differential impact of BMI-based obesity at the acute phase (ie, in hospital) versus midterm (ie, 6 months) or 1 year. These studies found the same trend of obesity whatever the time delay of follow-up. Overall, these data strongly suggest that the apparent obesity paradox is already present at the early phase of an acute MI.

In acute MI, the level of NT-proBNP, an indicator of the hemodynamic severity of MI, as well as systolic and/or diastolic dysfunction, peaks at the time of admission and is considered one of the most powerful predictors of death. Low proBNP levels in patients with high BMI, with a pattern similar to that of heart failure, may therefore participate in the more favorable outcome reported in the present study. Surprisingly, low propeptide levels, similar time to admission, and LVEF were not associated with lower hemodynamic severity in patients with high BMI. These findings suggest that hemodynamic severity and left ventricular dysfunction may not be the only major contributors of admission propeptide levels in patients with a high BMI.

**WC-Based Obesity**

WC is a surrogate measure of abdominal fat, with which it has been strongly correlated by computed tomography or magnetic resonance imaging. High WC, as a component of metabolic syndrome and insulin resistance–related disorders, is associated with CV death. In healthy subjects, WC is a better predictor of acute coronary events than BMI. Paradoxically, few studies have analyzed abdominal obesity in the setting of AMI. We found that an elevated WC (as defined by the NCEP ATP III threshold) was very frequent in patients with AMI because it is present in half of the patients. In patients with CV disease, a high prevalence of abdominal obesity also was reported. One of the major findings of our study is that, in contrast to the strong negative relationship observed between BMI and age, WC was poorly but positively correlated with age. These findings suggest a weaker impact of WC as a risk factor for AMI than BMI. CV risk has been shown to derive more from abdominal obesity–associated risk factors (high blood pressure, diabetes, and dyslipidemia) than from abdominal obesity per se. Moreover, recent findings from the Dallas Heart Study suggested that WC was not independently associated with prevalent atherosclerosis after adjustment for standard risk factors.

No significant increased risk in intermediate-term death was associated with WC. These findings extend our previous results that showed that NCEP ATP III–defined elevated WC was not an independent predictor of in-hospital outcomes. They also extend to acute MI recent findings showing no increased WC-associated risk for coronary artery disease in men and women with prevalent disease. However, further larger prospective studies in acute MI are needed to confirm these trends. In men, a lower BMI at a given waist girth has been suggested to be associated with higher levels of visceral adipose tissue. In the present study, a strikingly worse
outcome was observed in patients with high WC but normal BMI, which presumably reflects the presence of visceral obesity with low muscle mass and a lack of functional subcutaneous adipose tissue. The identification of such high-risk subgroups has potential major clinical implications and may warrant more aggressive lifestyle and therapeutic interventions for secondary prevention after AMI.

Study Limitations
In the present study, the same kinds of tape measure and scales were used to assess WC and BMI at all of the centers—either public or privately-funded hospitals—participating in the RICO Survey and throughout the inclusion period. Anthropometric measurements were carried out by nurses trained in measuring WC using a standardized procedure for all the centers. However, we cannot exclude the possibility that the impact of WC and BMI might have been blurred by differences in measurement techniques at the different sites. No data were available on the angiographic features of the study population, a variable known to influence outcome after MI. However, in patients with AMI, the angiographic extent of coronary artery disease was found to be similar across obesity categories, suggesting that it may similarly affect prognosis in all of the groups. Moreover, although BMI- and WC-based obesity classifications were unable to independently predict 1-year death rates, we cannot exclude the possibility that obesity may affect the very long-term follow-up because obesity may have a delayed influence on the progression of coronary artery disease. After elective PCI, a U-shaped relation between BMI and the post-PCI risk of death also has been reported, with the lowest risk observed in class I and II obesity and the highest risk in extreme groups, ie, very lean (BMI <18.5 kg/m²) and very severely obese (BMI >40 kg/m²) individuals. However, in the present work, the analysis of the extreme groups was limited by the small number of subjects. Our study enrolled almost exclusively white patients, and it has been suggested that thresholds for defining obesity correlated to CV risk in Asian patients may differ from those in white patients (French law prohibits collecting race or ethnicity-related information). Finally, our study is limited to patients arriving at the hospital alive and able to provide consent and undergo anthropometric measurements. Patients who die in the pre-hospital setting and those with an extremely severe clinical status on arrival may differ from the “average survivor of the early phase” studied here.

Conclusions
The present study underscores the high prevalence of increased BMI and WC in AMI patients, present in one quarter and one half of the patients, respectively. Until the epidemic progression of obesity is confronted, cardiologists will be faced with a growing prevalence of obesity in patients with AMI. Given this high prevalence, the characterization of CV risk associated with obesity after the index event is of importance. We found that much of the apparent obesity paradox can be explained by confounding. Our findings also emphasize the need to measure both BMI and WC for risk stratification after MI and suggest the importance of managing high WC-related modifiable risk factors in secondary prevention, particularly in patients with high WC but without overall obesity. Our results strongly suggest that after MI, neither WC nor BMI has an independent impact on death, taking into account traditional risk factors and other confounding variables. These results appear robust regardless of the type of adjustment. However, as in any observational study, residual confounding cannot be excluded, and these results should be confirmed by large, independent, prospective studies.

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References

**CLINICAL PERSPECTIVE**

The present study underscores the high prevalence of increased body mass index and waist circumference in patients with acute myocardial infarction, present in one quarter and one half of the patients, respectively. Until the epidemic progression of obesity is confronted, cardiologists will be faced with a growing prevalence of obesity in patients with acute myocardial infarction. Given this high prevalence, the characterization of cardiovascular risk associated with obesity after the index event is important. Most studies have reported a lower rate of death after myocardial infarction for patients with increased body mass index (the obesity paradox). We found that much of this apparent obesity paradox is related to confounding by baseline characteristics associated with survival. Neither body mass index nor waist circumference was an independent predictor of survival. However, in both men and women, a high waist circumference with low body mass index (presumably reflecting visceral obesity with low muscle mass and lack of functional subcutaneous adipose tissue) was predictive of increased 1-year death rate. This emphasizes the need to measure both body mass index and waistline in patients with myocardial infarction, particularly to identify this sizable fraction of the patient population at high risk of death.
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