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Body mass index, treatment practices, and mortality in patients with acute heart failure

Timothy P. Fitzgibbons, **Olga T. Hardy**, **Darleen Lessard**, **Joel M. Gore**, **Jorge Yarzebski**, and **Robert J. Goldberg**
Department of Medicine, Division of Cardiovascular Medicine, University of Massachusetts Medical School, Worcester, Massachusetts, USA

Abstract

Objectives—Obesity is associated with an increased risk of heart failure (HF). Among patients presenting with acute HF, however, differences in clinical characteristics, treatment regimens, and short-term prognosis of varying weights are largely unknown, particularly from a broader population-based perspective.

Methods—A total of 3722 patients admitted with acute HF to 11 greater Worcester (Massachusetts, USA) hospitals during 1995 and 2000 were categorized as being lean (*n* = 216), normal weight (*n* = 1465), overweight (*n* = 1007), or obese (*n* = 1034) at the time of hospitalization.

Results—Obese patients with decompensated HF were significantly younger (mean age = 71 years) compared with patients of normal weight (mean age = 79 years). Obese patients were more likely to have a history of diabetes and have previously undergone a percutaneous coronary intervention than patients of normal body weight. Lean patients (body mass index <18.5kg/m^2) were less likely to be treated with effective cardiac therapies than normal weight patients, whereas obese patients were more likely to be treated with diuretics. Obese patients experienced a significantly lower in-hospital (4.3 vs. 7.2%) and 30-day (7.3 vs. 14.5%) death rate than normal weight patients, whereas lean patients experienced the highest in-hospital (10.2%) and 30-day (19.9%) death rates.

Conclusion—The results of this study in residents of a large central New England metropolitan area suggest that obesity is associated with increased survival in patients with acute HF. Further assessment of the ‘obesity paradox’, and careful attention to patients with a low body mass index, in patients with decompensated HF is warranted.

Keywords

body mask index; heart failure; prognosis

Introduction

Approximately, 64 million American adults are obese [1]. Individuals who are obese are nearly twice as likely to die from all causes than their normal weight counterparts, with much of this excess mortality attributed to cardiovascular disease [2]. Furthermore, obesity is associated with a variety of important risk factors for cardiovascular disease, including

Correspondence to Dr Robert J. Goldberg, PhD, Department of Medicine, Division of Cardiovascular Medicine, University of Massachusetts Medical School, 55 Lake Avenue North, Worcester, MA 01655, USA, Tel: +1 508 856 3991; fax: +1 508 856 4596; Robert.Goldberg@umassmed.edu.

insulin resistance, hypertension, and dyslipidemia. Progressive increases in body mass index (BMI), even within the overweight category, have been associated with an increased risk of developing congestive heart failure (HF) [3].

In contrast to the increased risk of newly diagnosed HF in obese patients, an increased BMI may confer a survival advantage in patients with prevalent HF resulting in an 'obesity paradox' [4,5]. Proposed explanations for this observation have included resistance to the catabolic effects of proinflammatory cytokines, lower levels of plasma catecholamines and natriuretic peptides, or to possible study design and methodologic concerns [3,6]. Although some investigators have suggested that the 'obesity paradox' is only operative in patients with HF with a reduced ejection fraction, others have found that it holds true across the spectrum of patients with this clinical syndrome [3,7]. However, the reproducibility and impact of these findings in the broader community-based setting are unclear.

We have recently described differences in the clinical profile, short-term prognosis, and hospital management practices utilized in obese men and women compared with patients of normal body weight in a community sample of patients hospitalized with acute myocardial infarction [8]. Obese patients were significantly younger, were more likely to be prescribed effective cardiac treatment regimens, and were at greater risk of developing adverse outcomes during hospitalization than normal weight patients. Whether or not these differences in prognosis and treatment practices exist in patients with acute HF is unknown.

The objectives of this population-based study were to compare differences in the clinical characteristics, treatment practices, and short-term prognosis of lean, normal weight, overweight, and obese residents of the Worcester metropolitan area admitted for decompensated HF to all greater Worcester medical centers during 1995 and 2000.

Methods

Adult male and female residents of all ages from the Worcester metropolitan area (2000 census estimate = 478000) hospitalized for possible HF at all 11 greater Worcester medical centers during the two study years of 1995 and 2000 comprised the study population [9,10].

The medical records of patients with primary and/or secondary International Classification of Disease (ICD)-9 discharge diagnoses consistent with the possible presence of HF were reviewed in a standardized manner as has been described earlier [9,10]. Patients with a discharge diagnosis of HF (ICD-9 code 428) comprised the primary diagnostic rubric reviewed for the identification of cases of possible HE In addition, the medical records of patients with discharge diagnoses of rheumatic HF, hypertensive heart and renal disease, acute cor pulmonale, cardiomyopathy, pulmonary heart disease and congestion, acute lung edema, edema, dyspnea, and respiratory abnormalities were reviewed by trained study physicians and nurses to identify patients who also may have had new onset HF [9,10]. Confirmation of the diagnosis of HF, based on use of the Framingham criteria, included the presence of two major criteria or presence of one major and two minor criteria [11]. An incident (first) event of HF was defined as the absence of a prior hospitalization for HF, a prior physician diagnosis of HF, or treatment for HF in the past based on the review of data contained in hospital medical records. Patients who developed HF secondary to admission for another acute illness (e.g. acute myocardial infarction) or after an interventional procedure or surgery were not included.

Data collection

Information was collected about patient's demographic, medical history, clinical characteristics, and laboratory test results through the review of information contained in

hospital medical records. This included the information about patient's age, sex, race, prior comorbidities, BMI at the time of hospitalization, presenting symptoms, physical examination findings, laboratory findings (e.g. serum levels of creatinine, blood urea nitrogen), clinical characteristics (e.g. presenting heart rate, blood pressure), and hospital survival status. Ejection fraction findings during the index hospitalization were available for only 37% of the study sample; we did not, therefore, further classify the study population into those with systolic as compared with those with diastolic HF based on the results of this testing modality.

We reviewed physicians' progress notes and daily medication logs for the prescribing of selected cardiac medications and recommendations for changes in various lifestyle practices [10,12]. We examined the use of cardiac medications that have been shown to be of benefit in improving the prognosis of patients with decompensated HF [angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and β-blockers], medications shown to be effective in improving the symptomatic status of patients with acute HF (digoxin and diuretics), and other effective cardiac medications.

Data analysis

Differences in the characteristics, treatment practices, and short-term case-fatality rates of patients who were considered to be lean/cachectic (BMI < 18.5 kg/m^2), of normal body weight (BMI 18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), obese (30–34.9 kg/m^2), and markedly obese (≥ 35 kg/m^2) were examined through the use of χ^2 tests and analysis of variance for categorical and continuous variables, respectively. A logistic regression approach was utilized to examine the association between patients' baseline BMI with the use of various treatment regimens and hospital outcomes while controlling for a variety of potentially confounding demographic and clinical factors. Multivariable adjusted odds ratios and accompanying 95% confidence intervals were calculated in a standard manner. As the length of hospital stay had declined over time, we also examined 30-day death rates after hospital admission in our respective comparison groups.

Results

Baseline characteristics

The study sample consisted of 3722 residents of the Worcester metropolitan area hospitalized at all greater Worcester medical centers with independently confirmed acute HF in whom information about BMI was available through the review of information contained in hospital charts. The average age of the study sample was 76 years, 43% were men, and the majority (94%) was Caucasian. In this sample, 45% of patients had what was considered to be a normal BMI (n = 1681), 27% were overweight (n = 1007), and 28% (n = 1034) were considered to be obese. Among patients with a BMI < 25kg/m^2, the majority (87%) had a BMI between 18.5 and 24.9 kg/m^2, whereas the remainder (n = 216) had a BMI < 18.5 kg/m^2 and were considered to be lean/cachectic. Among obese individuals, 538 had a BMI between 30 and 34.9 kg/m^2, whereas 496 patients were considered to be markedly obese.

Obese patients were significantly younger, more likely to be non-Caucasian, and were less likely to have a do not resuscitate order in their hospital charts (Table 1). Patients with an elevated BMI were significantly less likely to have a history of atrial fibrillation than those who were less overweight (Table 1). In contrast, patients who were obese were more likely to have a history of chronic pulmonary disease, diabetes, and hypertension, and to have received a percutaneous coronary intervention in the past than patients of normal body weight (Table 1). Obese patients were more likely to present with varying signs and symptoms of acute HF, including chest pain, dyspnea, edema, orthopnea, and weight gain.

Obese patients were also more likely to have a worse physiologic profile than patients of lower body weight, including serum glucose levels, blood pressure, and renal function (Table 1).

Hospital treatment practices

Patients who were obese were significantly less likely to have been treated with digoxin during their index hospitalization than patients with a lower BMI (Table 2). Obese and overweight patients were more likely to have been prescribed ACE inhibitors/ARBs, calcium channel blockers, and lipid-lowering medications than patients with a BMI < 25 kg/m^2. Lean/cachectic patients with acute HF were the least likely of all the groups examined to have been prescribed effective cardiac therapies.

In examining the utilization of these cardiac medications during hospitalization for acute HF in patients with obesity of increasing severity (Table 2), markedly obese patients were less likely to have been treated with ACE inhibitors/ARBs, β-blockers, digoxin, lipid-lowering medications, and nitrates than patients with a BMI between 30 and 34.9 kg/m^2.

After controlling for age and a variety of potentially confounding factors, lean/cachectic patients were less likely to have been treated with ACE inhibitors/ARBs, β-blockers, diuretics, calcium channel blockers, and lipid-lowering agents than patients with a BMI between 18.5 and 24.9 kg/m^2 (Table 3). Patients of normal body weight were significantly less likely to have been treated with digoxin, whereas patients with a BMI of 30–34.9 kg/m^2 were significantly less likely to have received digoxin, but more likely to have been prescribed ACE inhibitors/ARBs, than patients of normal body weight; markedly obese patients were significantly less likely to have been prescribed digoxin and nitrates than patients considered to be of normal body weight.

Nonpharmacologic treatment practices

Obese patients were more likely to have been prescribed a low-fat diet and restrict their intake of fluids and salt than patients with a BMI < 25 kg/m^2 (Table 4). After controlling for several potentially confounding factors that might affect these treatment recommendations, differences in these dietary practice recommendations remained (Table 4).

Among patients with a BMI < 25 kg/m^2, leaner patients were less likely to be told by a healthcare professional to modify the vast majority of lifestyle practices recommended (Table 4). With the exception of receiving recommendations to restrict their fluid intake to a greater extent, there were no marked differences in the receipt of recommendations to modify various lifestyle practices in patients with varying degrees of obesity (Table 4).

Hospital case-fatality rates

The in-hospital case-fatality rate was significantly higher in patients with a normal BMI (BMI < 25 kg/m^2) compared with obese patients (7.2 vs. 4.2%) ($P < 0.05$) (Table 5). After controlling for age only, obese patients remained at decreased risk of dying during the acute hospitalization compared with patients with a BMI < 25 kg/m^2, although these differences were no longer significant and become attenuated after controlling for additional important prognostic factors. Similar trends were observed when we examined differences in 30-day death rates after hospital admission in patients of varying BMIs (Table 5).

In patients with a BMI < 18.5 kg/m^2, their hospital case-fatality rates were more than one-third higher than in those with a BMI between 18.5 and 24.9 kg/m^2 (10.2 vs. 6.8%). The 30-day post-admission death rates were also considerably higher in patients with a BMI < 18.5kg/m^2 (19.9% dying) as compared with those with a BMI 18.5–24.9 kg/m^2 (13.7%

dying). Among obese patients, patients who were the most obese were the least likely to die during the acute hospitalization (1.9%) as compared with patients with a BMI between 30 and 34.9 kg/m^2 (4.5%) and those with a BMI between 35 and 39.9 kg/m^2 (6.2%).

Discussion

Data from the Worcester Heart Failure study provide insights into the characteristics of a community-wide population of patients hospitalized with decompensated HF at all medical centers in central New England. Our results show that overweight and obese patients are younger than their leaner counterparts when hospitalized for acute HF. Increasing BMI was associated with relatively small differences in hospital treatment practices and trends toward a lower in-hospital mortality compared with normal weight patients. Patients with a particularly low BMI (< 18.5 kg/m^2) were at the greatest risk for not being treated with effective cardiac medications and for dying over the short-term. These results suggest that the assessment of BMI at the time of hospital admission can identify groups of patients that differ with respect to clinical characteristics, prognosis, and perhaps underlying pathophysiology requiring a more tailored therapeutic approach to their management.

Demographic and clinical characteristics according to body mass index

Our population-based study of patients hospitalized with acute HF showed slightly lower prevalence rates of overweight and obesity than have been observed in recent national surveys of both apparently healthy adults and those with decompensated HF Using data from the 1999–2002 National Health and Nutrition Examination Survey, an estimated two-thirds of adults from the United States were found to be either overweight or obese [13]. In the Acute Decompensated Heart Failure National Registry (ADHERE), approximately 30% of patients with HF were considered to be of normal body weight, 29% as overweight, and the remainder as obese [14].

Several notable differences were observed in patient characteristics across BMI categories in this study. These included a larger proportion of women, a greater prevalence of various comorbidities and adverse physiologic factors, and symptom patterns in patients who were obese. Perhaps most striking, however, was the younger age of patients with decompensated HF who were obese, which was also a major finding in the ADHERE study [14]. Several earlier studies have confirmed that obesity is a risk factor for incident episodes of acute HF, independent of associated comorbidities [3,15,16]. The markedly earlier age of onset of acute HF in obese patients may suggest a more accelerated course of underlying disease in these patients.

In contrast, more recent data suggest that the measurement of BMI alone is not a specific enough measure of adiposity in assessing the risk of cardiovascular disease. In particular, abdominal obesity, as measured by waist circumference, is a better determinant of the risk of coronary heart disease and hypertension [17,18]. A recent analysis from the Health ABC Study examined the role of abdominal obesity as an independent risk factor for HF in elderly patients [19]; waist circumference was shown in multivariate analyses to be a stronger predictor of HF than BMI. In the MESA study [20], a community-based multicenter cohort study, obesity was shown to be an independent risk factor for HF. However, this association was found to no longer be significant after controlling for level of baseline systolic function or markers of inflammation. These findings suggest that the association between obesity and the development of HF may be mediated by left ventricular systolic dysfunction and/or systemic inflammation. One might speculate that the preponderance of obesity in the youngest patients with acute HF in our study is mediated by these same mechanisms. Systemic inflammation originating in visceral fat depots may contribute to insulin

resistance, partially explaining the greater proportion of patients with diabetes mellitus observed among obese patients in this study.

There have been divergent findings with regard to the risk of HF in patients with insulin resistance [15,20]. In addition to the important influences of diabetes mellitus and the metabolic syndrome, the increased sympathetic tone associated with visceral fat depots, increased oxidative stress, and derangements in the renin–angiotensin and natriuretic peptide systems are also possible further mechanistic explanations for the association between an elevated BMI and risk of acute HF [21,22].

The greater prevalence of atrial fibrillation in patients with a normal BMI was not surprising, as this group was significantly older than heavier patients. Atrial fibrillation has been thought to be predominantly a disease of the elderly; hypertension, underlying cardiovascular disease, and diabetes mellitus have also been consistently shown to be associated with the development of atrial fibrillation [23], However, obesity has also been associated with the occurrence of this atrial arrhythmia in a case–control study [24].

Treatment practices according to body mass index

Digoxin was significantly less often prescribed to obese patients than to those of lower body weights, whereas several lifestyle recommendations were more commonly prescribed to heavier patients. Moreover, patients with the lowest BMIs were least likely to be treated with effective cardiac therapies. The safety and efficacy of digoxin has been shown in the Digitalis Investigation Group trial [25]. Data from large national registries of patients with HF and recent trials involving patients with HF have shown a decline in the use of digoxin during recent years likely because of a variety of reasons [26].

Receipt of lifestyle practice recommendations according to body mass Index

Patients who were obese in this study were more likely to be prescribed and counseled on the benefits of a low-fat diet and restrict their intake of fluids and salt compared with leaner patients.

A low-fat diet (e.g. 25–30% of calories from fat) is considered the first-line therapy for treating obesity. Data obtained from dietary intervention trials designed to decrease the risk of cardiovascular disease indicate that decreasing dietary fat intake results in decreased total energy intake and weight loss [27]. Patients who were obese in this study also had a greater prevalence of hypertension at the time of hospital presentation than patients with a lower BMI. Restricting dietary sodium and fluid intake is of paramount importance in controlling blood pressure and preventing exacerbations of HF in these high-risk patients.

Hospital death rates according to body mass index

Patients who were obese were less likely to die during the acute hospitalization compared with normal weight and overweight patients. Results from the ADHERE study showed that in-hospital case-fatality rates decreased across successively higher BMI quartiles, even after adjustment for known prognostic factors [14]. Our findings provide further confirmation of the existence of an obesity paradox among patients with acute HF. In addition, patients with a BMI < 18.5 kg/m^2 were at particularly increased risk of dying over the next 30 days after being hospitalized for an episode of decompensated HF, emphasizing the adverse effects associated with being cachectic [28].

The mechanisms responsible for the counter-intuitive association of higher BMI and lower mortality among patients hospitalized with acute HF are not clear. The younger age of obese patients may partially contribute to the survival advantage of these patients and older

patients are likely to have many more comorbid conditions present. Patients with a higher BMI may also present with less severe HF and/or have diastolic HF and be at lower risk of dying during an acute episode of HF [4,5].

Obesity may exert a protective effect by providing sufficient metabolic reserve to overcome the increased catabolic stress resulting from an acute exacerbation of HF [29]. Interestingly, endocrine markers of catabolism have been associated with the development of incident HF and increased total mortality in elderly cohorts [30]. Investigators from the Framingham Heart Study first described an inverse relationship between serum insulin-like growth factor-1 (IGF-1) and risk of incident HF in elderly individuals [31]. A recent report from the Cardiovascular Health Study showed that high baseline insulin-like growth factor-binding protein-1 (IGFBP-1), the endogenous inhibitor of IGF-1 activity, was a significant predictor of incident HF in elderly individuals [30]. IGF-1, apart from its anabolic actions, also has been shown to have beneficial cardiovascular effects, including vasodilation [32], inhibition of myocardial apoptosis [33], and promotion of myocyte hypertrophy [34]. Thus, one might speculate that pathological senescence, characterized by systemic inflammation and protein catabolism, is a poor substrate for the additional neuroendocrine stress of acute decompensated HF.

Several other physiologic parameters that are unique to obese individuals may contribute to the ‘obesity paradox’. Elevations in plasma levels of adiponectin have been shown to be an independent predictor of mortality in patients with HF [35]; increases in circulating adiponectin levels may be a marker of the wasting process in patients with HF. The neurohormonal response to stress seems to be attenuated in obese hypertensives as compared with lean hypertensives, and this heightened catecholamine response may increase the risk of dying in lean individuals with HF [36].

Study strengths and limitations

The strengths of our study include the multihospital population-based sample of patients hospitalized with acute HF in a large Central New England metropolitan area whose socio-demographic characteristics reflect those of the nation as a whole. However, the present results should be evaluated in the context of several limitations. Information about a number of important clinical and risk factor data, such as smoking status and alcohol use, was not collected. These and additional prognostic factors may have adversely affected the hospital outcomes under study and/or receipt of selected therapies and may have differed according to patients’ weight. Patients’ weights were obtained during hospitalization for acute decompensated HF and may not represent the patients’ true dry weight. We also did not collect information on recent changes in patients’ weight before their hospitalization for AMI, which may have affected the classification of several of our weight groups and some of the associations observed. The measurement of BMI has inherent limitations because it is not a direct measure of body composition and may not accurately reflect the degree of body fat content, especially in patients who have lost muscle mass. We did not have data available on body fat composition or waist circumference to further refine our classification of obesity. The lack of information about inpatient ejection fraction findings and assessment of left ventricular function in a large proportion of our study sample was an additional important study limitation, as we were unable to systematically classify patients into those with either systolic or diastolic HF, which may have mediated a number of the associations observed with regard to BMI.

Conclusion

In this community-based study of patients hospitalized for decompensated HF, there was an inverse relationship between BMI and patient’s age. Despite their higher risk of mortality,

patients with normal or low body weight are less likely to receive important cardiac medications indicated for the secondary prevention of HF. Refinement of current methods to measure body fat content should provide further insights into the pathophysiology behind the apparent ‘obesity paradox’ in patients with acute HF and other cardiovascular diseases. Particular attention also needs to be paid to patients who present with a low BMI at the time of hospitalization for acute HF so that these high-risk patients might be more optimally treated and their prognosis enhanced.

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Table 1

Differences in selected characteristics according to body mass index at the time of hospitalization for acute heart failure

	Body mass index (kg/m^2)					
Characteristic	**<18.5 (*n* = 216)**	**18.5–24.9 (*n* = 1465)**	**25–29.9 (*n* = 1007)**	**30–34.9 (*n* = 538)**	**≥ 35 (*n* = 496)**	***P* value**
Age (years) (%)						
<65	8.8	8.1	13.6	17.7	39.1	<0.001
65–74	17.7	17.7	27.1	29.6	26.2	
75–84	32.6	44.4	38.3	37.5	27.4	
≥ 85	40.9	29.8	21.0	15.2	7.3	
Age (mean, years)	79.6	78.9	75.7	73.7	67.3	<0.001
White race (%)	95.4	95.6	95.2	93.9	88.5	<0.001
Male sex (%)	25.0	42.9	50.7	43.7	32.9	0.58
Do not resuscitate order (%)	45.4	34.1	22.2	19.9	18.4	<0.001
Current smoker (%)	9.7	9.0	9.6	10.6	11.5	0.11
Medical history (%)						
Atrial fibrillation	41.7	39.6	33.9	33.1	24.6	<0.001
Chronic obstructive pulmonary disease	43.5	32.4	33.1	39.4	42.7	<0.01
Coronary artery bypass grafting	8.8	20.6	22.7	21.8	12.5	0.34
Diabetes	16.7	32.2	43.6	53.4	56.5	<0.001
Heart failure	74.5	76.2	70.6	74.9	76.8	0.99
Hypertension	56.5	63.8	67.9	70.1	67.5	<0.01
Liver disease	1.9	2.7	4.2	2.4	2.4	1.00
Percutaneous coronary intervention	3.2	5.9	9.0	12.1	8.3	<0.001
Peripheral vascular disease	12.5	18.1	21.0	18.0	15.5	0.96
Renal disease	15.7	24.3	25.9	22.3	19.8	0.45
Presenting symptoms (%)						
Chest pain	23.6	31.2	33.0	35.7	32.3	<0.05
Dyspnea	95.4	93.9	95.1	96.5	95.8	0.05
Edema	44.0	59.3	69.4	79.0	89.9	<0.001
Nausea	20.4	15.0	14.3	14.3	15.5	0.39

Characteristic	Body mass index (kg/m^2)					
	<18.5 (*n* = 216)	**18.5–24.9 (*n* = 1465)**	**25–29.9 (*n* = 1007)**	**30–34.9 (*n* = 538)**	**≥ 35 (*n* = 496)**	***P* value**
Orthopnea	29.6	30.4	36.0	41.5	40.9	<0.001
Weakness	30.6	30.4	26.0	22.5	26.0	<0.01
Weight gain	3.7	5.6	9.7	10.8	15.5	<0.001
Physiologic factors (mean)						
Serum glucose (mg/dl)	144.3	160.6	171.2	171.3	175.8	<0.001
Heart rate (bpm)	95.8	90.3	90.4	89.6	90.4	<0.05
Systolic blood pressure (mmHg)	142.3	143.1	146.4	149.4	146.9	<0.01
Diastolic blood pressure (mmHg)	75.6	75.7	77.0	78.1	78.6	<0.05
Ejection fraction (%)	43.4	45.1	44.1	50.0	44.6	0.32
Estimated glomerular filtration rate (mg/dl)	75.0	61.6	61.1	62.3	67.8	<0.001
Serum sodium (mg/dl)	137.1	137.0	137.1	137.5	137.6	0.13
Length of hospital stay (days)	6.9	6.2	6.4	5.8	6.7	0.16

Table 2

Receipt of selected cardiac medications and nonpharmacologic treatment regimens according to body mass index

	Body mass index (kg/m^2)					
	<18.5 (*n* = 216)	**18.5–24.9 (*n* = 1465)**	**25–29.9 (*n* = 1007)**	**30–34.9 (*n* = 538)**	**≥ 35 (*n* = 496)**	***P* value**
Medication (%)						
ACE inhibitors/ARBs	44.9	54.0	55.6	61.5	54.2	<0.05
Beta-blockers	32.9	40.7	45.1	42.2	39.1	0.43
Calcium channel blockers	26.4	35.4	36.4	41.3	41.1	<0.001
Digoxin	58.3	57.3	51.5	46.1	36.7	<0.001
Diuretics	97.7	98.0	97.9	98.5	99.0	0.10
Lipid-lowering medications	3.2	12.9	18.2	19.3	18.4	<0.001
Nitrates	60.7	66.7	68.2	70.1	59.9	0.45
Lifestyle recommendations (%)						
Fluid restriction	18.5	22.1	20.8	23.8	28.8	<0.01
Increase physical activity	36.6	42.6	43.4	42.1	40.1	0.94
Low-fat diet	41.7	57.6	61.5	67.1	63.3	< 0.001
Reduce alcohol consumption	0.5	0.4	1.0	0.6	1.4	<0.05
Reduce salt intake	71.8	81.5	81.6	86.1	85.5	< 0.001
Rehabilitation	3.7	7.5	9.4	9.7	8.5	<0.05

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker.

Table 3

ling to body mass index status at the time of hospitalization for acute heart failure

	Body mass index (kg/m^2)														
	< 18.5 (*n* = 1681)			18.5–24.9 (*n* = 1007)			25–29.9 (*n* = 1034)			30–34.9			≥ 35		
ude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)
44.9	0.69 (0.52–0.93)	0.78 (0.58–1.06)	54.0	1.0	1.0	55.6	1.02 (0.87–1.20)	0.98 (0.82–1.16)	61.5	1.27 (1.04–1.56)*	1.24 (1.00–1.55)*	54.2	0.86 (0.70–1.07)	0.84 (0.67–1.05)
32.9	0.72 (0.53–0.98)	0.86 (0.62–1.20)	40.7	1.0	1.0	45.1	1.20 (1.02–1.41)	1.11 (0.93–1.33)	42.2	1.06 (0.87–1.30)	0.93 (0.74–1.16)	38.9	0.92 (0.74–1.15)	0.93 (0.74–1.18)
58.3	1.04 (0.76–1.39)	1.06 (0.77–1.47)	57.3	1.0	1.0	51.5	0.77 (0.66–0.91)	0.75 (0.63–0.90)	46.1	0.62 (0.50–0.75)	0.57 (0.46–0.72)	36.7	0.40 (0.32–0.50)	0.38 (0.30–0.49)
97.7	0.88 (0.34–2.29)	0.56 (0.21–1.51)	98.0	1.0	1.0	97.9	1.08 (0.61–1.90)	1.05 (0.55–2.00)	98.5	1.62 (0.73–3.59)	1.19 (0.52–2.74)	99.0	2.92 (1.09–7.80)	2.54(0.84–7.70)
26.4	0.66 (0.48–0.91)	0.72 (0.51–1.01)	35.4	1.0	1.0	36.4	1.03 (0.87–1.22)	0.90 (0.75–1.07)	41.3	1.24 (1.01–1.52)	1.08 (0.87–1.34)	41.1	1.19 (0.95–1.48)	1.05 (0.83–1.33)
3.2	0.23 (0.10–0.49)	0.30 (0.14–0.67)	12.9	1.0	1.0	18.2	1.39 (1.11–1.73)	1.17 (0.92–1.48)	19.3	1.41 (1.08–1.85)	1.08 (0.81–1.43)	18.4	1.12 (0.84–1.50)	1.01 (0.74–1.37)
60.7	0.76 (0.57–1.02)	0.90 (0.65–1.27)	66.7	1.0	1.0	68.2	1.11 (0.93–1.31)	0.99 (0.81–1.20)	70.1	1.22 (0.98–1.52)	0.99 (0.77–1.26)	59.9	0.82 (0.66–1.02)	0.72 (0.56–0.93)

e; ARB, angiotensin receptor blocker; CI confidence interval; OR, odds ratio.

suscitate status, history of prior chronic obstructive pulmonary disease, angina, atrial fibrillation, hypertension, coronary heart disease, diabetes, cardiac catheterization, ntion, serum glucose and sodium findings, blood pressure, and estimated glomerular filtration rate.

Table 4

endations according to body mass index status at the time of hospitalization for acute heart failure

Body mass index (kg/m^2)														
< 18.5 (*n* = 1681)			18.5–24.9 (*n* = 1007)			25–29.9 (*n* = 1034)			30–34.9			≥ 35		
rude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted OR[a] (95% CI)	Crude (%)	Age-adjusted OR (95%CI)	Multivariable-adjusted OR[a] (95% CI)
41.7	0.53 (0.40–0.71)	0.58 (0.43–0.80)	57.6	1.0	1.0	61.5	1.16 (0.98–1.37)	1.07 (0.90–1.28)	67.1	1.47 (1.19–1.81)	1.39 (1.07–1.67)	63.3	1.20 (0.97–1.50)	1.23 (0.97–1.55)
18.5	0.81 (0.56–1.17)	0.85 (0.58–1.24)	22.1	1.0	1.0	20.8	0.89 (0.73–1.08)	0.92 (0.75–1.13)	23.8	1.04 (0.82–1.31)	1.10 (0.86–1.41)	28.8	1.25 (0.98–1.60)	1.35 (1.05–1.75)
36.6	0.79 (0.59–1.06)	0.79 (0.58–1.07)	42.6	1.0	1.0	43.4	1.00 (0.85–1.18)	0.96 (0.81–1.14)	42.2	0.94 (0.77–1.15)	0.92 (0.74–1.14)	40.1	0.82 (0.66–1.02)	0.82 (0.65–1.03)
0.5	0.86 (0.10–7.55)	1.39 (0.14–14.1)	0.4	1.0	1.0	1.0	2.01 (0.71–5.65)	2.04 (0.64–6.55)	0.6	0.84 (0.20–3.45)	1.02 (0.22–4.78)	1.4	1.07 (0.34–3.37)	0.90 (0.24–3.44)

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uscitate status, history of prior chronic obstructive pulmonary disease, angina, atrial fibrillation, hypertension, coronary heart disease, diabetes, cardiac catheterization, tion, serum glucose and sodium findings, blood pressure, and estimated glomerular filtration rate.

Table 5

Short-term CFR according to BMI status at the time of hospitalization for acute heart failure

		Hospital			30-day CFRs		
BMI	***n***	**CFR (%)**	**Age-adjusted OR (95% CI)**	**Multivariable-adjusted OR[a] (95% CI)**	**CFR (%)**	**Age-adjusted OR (95% CI)**	**Multivariable-adjusted OR[a] (95% CI)**
< 18.5	216	10.2	1.54 (0.95–2.51)	1.44 (0.84–2.48)	19.9	1.54 (1.07–2.23)	1.33 (0.87–2.03)
18.5–24.9	1465	6.8	1.0	1.0	13.7	1.0	1.0
25–29.9	4,007	6.0	0.92 (0.66–1.28)	1.37 (0.94–2.00)	10.7	0.75 (0.63–1.04)	1.06 (0.80–1.42)
30–34.9	538	4.5	0.72 (0.45–1.14)	1.24 (0.75–2.07)	7.1	0.54 (0.38–0.78)	0.78 (0.53–1.17)
35	496	3.8	0.69 (0.41–1.16)	0.78 (0.42–1.40)	7.5	0.66 (0.45–0.97)	0.71 (0.46–1.10)

BMI, body mass index; CFR, case-fatality rate; CI, confidence interval.

[a]Controlling for age, race, do not resuscitate status, history of chronic obstructive pulmonary disease, angina atrial fibrillation, hypertension, coronary heart disease, diabetes, cardiac catheterization, percutaneous coronary intervention, serum glucose and sodium findings, blood pressure, and estimated glomerular filtration rate.

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