Influence of admission blood pressure on mortality in patients with acute decompensated heart failure

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Summary

Objectives: To determine the relationship between admission blood pressure (BP) and prognosis in patients hospitalized for acute decompensated heart failure (HF).

Background: The relationship between BP admission blood pressure and outcomes in decompensated HF is controversial. It has been suggested that this presentation may be a specific disorder, but their mechanisms and clinical relationships are poorly defined.

Methods: We evaluated the association between initial BP (systolic, diastolic and mean BP) with readmission and mortality, as well as potential interactions with age, clinical characteristics, renal function, left ventricular dysfunction, comorbidities and treatment. By using Cox regression models the association between each outcome and BP was tested.

Results: A total of 581 patients (77.5-years-old, range 51–100) were included. At admission, mean BP in quartiles was 77.09 mm Hg (53.3–85.0) (Q1); 91.46 mm Hg (85.0–96.7) (Q2); 103.41 mm Hg (96.7–109.9) (Q3) and 124.79 mm Hg (109.9–209.0) (Q4). Median duration of follow-up was 8 months [95% confidence interval (CI) 5.2–11.1]. Mortality was 15.5% (Q1), 9.2% (Q2), 12.6% (Q3) and 7.3% (Q4). Interquartile hazard ratio (95% CIs) for mortality was 0.40 (0.19–0.85) \( P=0.017 \). Body mass index (BMI) was higher in Q4 29.59 k/m\(^2\) than in Q1 28.25 k/m\(^2\) \( P=0.018 \). There were no differences in age, clinical antecedents, renal function, comorbidities or severity of HF between groups.

Conclusions: Higher mean BP at admission is associated with significantly lower mortality during follow-up, in patients hospitalized for HF. With the exception of BMI, positively correlated with blood pressure, this relationship is independent of other clinical factors and medications.
Introduction

Heart failure (HF) accounts for more hospitalizations than acute myocardial infarction (AMI) and contributes significantly to morbidity, mortality and health-care expenditures. Patients hospitalized due to HF in Internal Medicine departments are older, with more comorbidities and predominantly with preserved ejection fraction. The identification of significant risk factors for adverse outcomes in such a population is a critical first step in designing strategies to improve prognosis and to provide more effective management for such a complex syndrome.

The major risk factors for the development of HF include coronary artery disease, smoking, hypertension, diabetes, obesity and valvular disease.

The Acute Decompensated Heart Failure National Registry (ADHERE), in which more than 100,000 patients with acute decompensated HF were enrolled, provides evidence that a history of hypertension is present in >70% of the patients hospitalized with heart failure, and that the mean blood pressure (MBP) for this patient group is hypertensive with a systolic BP (SBP) >140 mmHg in >50% of cases.

The treatment of hypertension is a cornerstone in HF prevention. In 1991, the Systolic Hypertension in the Elderly Program (SHEP) study provided evidence that treatment of isolated systolic hypertension could reduce cardiovascular events and prevent HF development. Although treatment of hypertension has proven its efficacy in preventing and reducing target organ damage and cardiovascular diseases, it is not so clear that lowering BP could have a beneficial effect on outcomes in patients who already have a cardiovascular disease. This phenomenon, also known as J Curve, is based on the data of some trials and post hoc analyses of the results of other trials. In high-risk patients, antihypertensive treatment regimens that reduce SBP to values close to or below 120–125 mmHg, and diastolic BP (DBP) to <70–75 mmHg, may be accompanied by an increase—rather than a further reduction—in the incidence of coronary events. In the OPTIMIZE-HF and other studies, patients with HF and left ventricular systolic dysfunction, low SBP and DBP were associated with greater mortality. Other studies, however, have shown a shortened survival in those with either reduced or increased values of discharge SBP.

Counterintuitively, it has also been reported that in patients admitted with acute decompensated HF, higher admission BP was associated with a reduction in mortality risk during follow-up. The data concerning this association are still contradictory. It has been suggested that it may be a specific disorder linked to HF with preserved ejection fraction. Nevertheless, associations with systolic HF, irrespective of the type and severity, have also been found.

Patients with HF hospitalized in Internal Medicine are different from those hospitalized in Cardiology departments. In a previous study, we found that the former were older, mainly women, with more comorbidities and predominantly with preserved ejection fraction. As stated earlier, the influence of BP in outcomes of patients hospitalized with HF is still controversial, and the data from patients admitted to Internal Medicine units, with their own peculiarities, are scarce.

The aim of the present study was to determine the relationship between systemic BP (systolic, diastolic and MBP), clinical characteristics and outcomes in patients admitted to Internal Medicine units due to an acute decompensation of HF. We obtained initial BP readings in a consecutive cohort of patients, and evaluated the association between elevated BP on admission with baseline characteristics and clinical outcomes.

Methods

Patients and methods

Patient data were collected between 9 March 2008 and 30 September 2009 and belong to the Spanish National Registry on HF—Registro Nacional de Insuficiencia Cardiaca (RICA), supported by the Spanish Working Group of Heart Failure of the Spanish Society of Internal Medicine (Grupo de Trabajo de Insuficiencia Cardiaca de la Sociedad Española de Medicina Interna (SEMI-IC))—which is a multicenter, prospective, cohort study. This registry includes data from 52 public and private hospitals in Spain. The study protocol was approved by the Ethics Committee of the Hospital University ‘Reina Sofía’, Córdoba, Spain, and informed consent was obtained from all patients prior to inclusion in the study. All patients, consecutively admitted to the Internal Medicine units with acute decompensation of HF, and cared for by physicians enrolled in the registry, were included in the study. In addition to giving their informed consent, patients were required to fulfill the following criteria.

Inclusion criteria

Patients aged >50 years, admitted due to HF according to the criteria of the European Society of Cardiology, presenting first episode of HF (incident HF), or decompensation of an already known, chronic HF (prevalent HF), or acute pulmonary...
edema or cardiogenic shock. In addition, patients were required to have completed a follow-up period ≥3 months.

**Exclusion criteria**

HF due to pulmonary hypertension and unwillingness to participate in the study.

Patients were included in RICA when first admitted (admission index) for acute HF. Follow-up consisted of two compulsory visits scheduled at 3 months and 1 year. Additional visits, whenever necessary and according to clinical judgement, are allowed. New admissions or death after admission index are considered end-points. However, readmission during follow-up does not imply exclusion from the registry. Patients and relatives were expected to give reasons for failure to attend scheduled visits.

Data were collected through a web site—https://www.registrorica.org/—which contains the database, accessed with a personal password. Confidentiality is preserved since no personal data are stored, except date of birth and name initials to avoid data duplication.

**Study variables**

The registry includes sociodemographic data, clinical antecedents, comorbidities (Charlson Index), basal functional status for basic activities of daily living (Barthel Index), clinical data (BP, heart rate, weight and height), complications during admission and prescriptions at discharge. With regard to HF severity, we have included functional class [New York Heart Association (NYHA) scale], left ventricle ejection fraction (LVEF) by means of 2D echocardiography, cardiac index through chest X-ray and EKG. Usual biochemical variables included kidney function, lipid and glucose profile, uric acid, troponin or natriuretic peptides (when available).

Primary end-points were first readmission, due to acute decompensation of HF, and death, by any cause. Secondary end-points were time to first event (days, from the first admission to either first readmission or death) and survival time was the number of days between inclusion date and death.

**Statistical analysis**

We reported data as mean ± standard deviation and interval for continuous variables and number (percentage) for categorical variables. Patient characteristics, disease severity measurements and 3-month outcomes are presented for the admission MBP quartile that was estimated as (admission SBP + 2 × DBP)/3. The rank correlation between MBP and each baseline characteristic is presented. For descriptive purposes, patients were divided into four quartiles, Q1–Q4, according to baseline MBP. Outcomes were defined as the time to the event or at least 3 months of follow-up if no event occurred. Cumulative event curves were estimated by Kaplan–Meier method and compared by log-rank test. The association between each outcome and MBP was tested using Cox regression models. Probability values of <0.05 were statistically significant. These analyses were completed with the Statistical Package for Social Sciences (SPSS) program (version 18.0, SPSS Inc. Chicago, IL, USA).

In the size sample calculation, for a relative precision of 26%, a relative risk of 1.6, an exposed and non-exposed ratio of 1, and a confidence interval of 95% (CI 95%), we estimated a sample of 564 patients.

**Results**

The sample included 581 patients (77.5-years-old, range 51–100); 317 women (54.6%), and mean LVEF 50.2% (13–82).

MBP on initial presentation was 98.6 ± 18.9 mm Hg and SBP/DBP was 140.5 ± 29.4/77.6 ± 16.7 mm Hg. High SBP (first measured SBP >140 mm Hg) was present in 47.5% of patients.

General characteristics of the population included are shown in Table 1.

We divided patients into quartiles according to blood pressure. Systolic blood pressureSBP/DBP in the highest quartile was 177.90 ± 24/98.24 ± 14 mm Hg. The baseline characteristics, disease severity measurements and outcomes for baseline MBP quartiles are presented in Table 2. More than 80% of the patients in our series had antecedents of hypertension. We did not find significant differences between quartiles in main clinical antecedents, LVEF, atrial fibrillation (AF), renal function and sodium concentration. Patients in the fourth MBP quartile had a significantly higher body mass index (BMI), heart rate and Barthel index and higher hemoglobin concentration. Functional class was, as a whole, better among patients in Q4 as compared with those in Q1 (Table 2). Length of in-hospital stay was significantly shorter among patients in the highest MBP quartile (Table 2).

In a subgroup of 177 patients (30.4%) N-terminal pro Brain Natriuretic Peptide (NT-proBNP) was available (Table 3). Mean concentration of NT-proBNP in each quartile was above cut-in values for diagnosis of HF.18 In addition, there was a significant and inverse correlation between NT-proBNP and MBP.

The percentage of patients taking either Angiotensin converting enzyme inhibitors (ACEi) or
Angiotensin receptor blockers (ARBs) was between 75 and 86% respectively, and 51.7% of the patients were receiving beta-blockers (BBs). There was no difference in the treatment of patients from one MBP quartile to another, except for ARBs and statins, which were more frequently prescribed among patients in the highest quartile of MBP (Table 4).

Median follow-up duration was 8 months (95% CI, 5.2–11.1). Rates of death, readmission and combined events, among the entire cohort, were 11.2, 21.2 and 28.6% respectively. Data on mortality and readmissions during follow-up are presented in Table 5 and Figure 1. There was no difference in the number of readmissions during follow-up or in-hospital medical complications according to the level of BP at admission. In contrast, follow-up mortality was significantly lower in patients with highest baseline mean and DBPs. The difference in SBP between Q4 and Q1 had a weaker prognostic value. Only the rate of combined events, death and readmission, was significantly lower among the patients in Q4, according to SBP (Table 5).

**Discussion**

Our study coincides with other studies in reporting elevated BP at admission as a common phenomenon in patients with acute decompensated HF.14,15 Our series included patients commonly seen in daily Internal Medicine practice. They were elderly, mostly women, with preserved ejection fraction, had multiple comorbidities and AF was present in ~50%. A hypertensive background was present in three-quarters, and one-quarter had antecedents of MI. A mild renal impairment, with an estimated glomerular filtration rate (eGFR) <60 ml/min, was common across the entire population. ADHERE showed significant differences between the usual case-type of patients hospitalized for HF, and patients who have participated in randomized clinical trials, whether hospitalized or as outpatients.15 Studies of patients hospitalized for HF have involved mostly patients with systolic dysfunction who are less hypertensive than those enrolled in the ADHERE. The sort of patient depicted in the ADHERE, and in our study, seems to be more common than expected but not, as yet, completely understood, due to the scarcity of studies on such a population.

Treatment in our patients was similar to the treatment reported in other series, and it closely adhered to established guidelines; ~80% of them received loop diuretics and an ACEi or ARBs; 25% received digoxin or nitrates and ~25% were treated with aldosterone blockers (ABs). Prescription of BBs in this series was ~50%. This figure is closer to other published series, and is clearly higher than the level of prescription recently reported in patients followed in primary care19,20 or by specialists21 in our field. These data probably reflect a selection bias, since participants in RICA are specialists from an in-hospital setting, directly involved in, and more sensitive to, the problem of HF. Patients in the fourth MBP quartile received a prescription of ACEi and statins more frequently than those in the first quartile, probably reflecting a higher degree of obesity and dyslipemia among them.

When our patients were stratified according to MBP at admission, several interesting differences...
between groups arose. Patients with the highest MBP were more obese, had higher heart rate and a slightly, although significant, increase in hemoglobin concentration. None of the differences can be attributed to ageing since mean age was similar among the four quartiles of MBP. Unlike other similar studies, the percentage of patients in our series with AF, antecedents of MI, hypertension, renal dysfunction and LVEF was similar across the MBP quartiles. As regards LVEF, we found a tendency, although not significant, for it to be lower among patients with the lowest MBP.

A second, and more intriguing, fact emerges from our study. The group of patients with 'a priori' worse prognosis, namely, the more hypertensive, more obese and with a higher heart rate, had a better prognosis. When patients in the first and fourth quartiles of MBP at admission were compared, the latter

### Table 2 Baseline characteristics of study population according to MBP quartiles

<table>
<thead>
<tr>
<th>Quartile</th>
<th>Age (years)</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
<th>MBP (mm Hg)</th>
<th>LVEF</th>
<th>NYHA class (%)</th>
<th>Heart rate (bpm)</th>
<th>BMI (kg/m²)</th>
<th>Creatinine (mg/dl)</th>
<th>eGFR (ml/min)</th>
<th>Sodium (mEq/l)</th>
<th>Hemoglobin (g%)</th>
<th>Charlson index</th>
<th>Barthel index</th>
<th>Length of stay (days)</th>
<th>AMI</th>
<th>HBP</th>
<th>AF</th>
<th>DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q1 (53.3–85.0) (n=148)</td>
<td>77.62 (8.06)</td>
<td>110.11 (12.42)</td>
<td>60.57 (6.56)</td>
<td>77.09 (6.09)</td>
<td>48.09 (15.63)</td>
<td>I 4.7</td>
<td>6.8</td>
<td>28.25 (5.72)</td>
<td>51.41 (25.50)</td>
<td>138.03 (5.10)</td>
<td>11.77 (1.97)</td>
<td>6.93 (2.29)</td>
<td>62.4 (10.65)</td>
<td>23.6%</td>
<td>81.8%</td>
<td>60.1%</td>
<td>42.6%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q2 (85.0–96.7) (n=153)</td>
<td>77.62 (8.94)</td>
<td>131.60 (13.69)</td>
<td>71.39 (7.24)</td>
<td>91.46 (3.39)</td>
<td>50.77 (16.44)</td>
<td>II 45.3</td>
<td>4.6</td>
<td>28.43 (5.35)</td>
<td>52.14 (24.87)</td>
<td>69.3 (2.33)</td>
<td>11.94 (2.08)</td>
<td>85.92 (22.93)</td>
<td>62.4 (9.35)</td>
<td>22.2%</td>
<td>82.4%</td>
<td>45.1%</td>
<td>42.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3 (96.7–109.9) (n=143)</td>
<td>77.89 (8.45)</td>
<td>145.80 (14.42)</td>
<td>82.22 (8.36)</td>
<td>103.41 (4.02)</td>
<td>50.22 (15.15)</td>
<td>III 43.2</td>
<td>9.0</td>
<td>29.15 (5.46)</td>
<td>54.21 (26.56)</td>
<td>67.3 (2.48)</td>
<td>12.61 (2.08)</td>
<td>84.54 (20.15)</td>
<td>62.4 (9.35)</td>
<td>25.2%</td>
<td>82.5%</td>
<td>52.4%</td>
<td>39.9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4 (109.9–209.0) (n=137)</td>
<td>76.98 (9.46)</td>
<td>177.90 (24.14)</td>
<td>98.24 (14.22)</td>
<td>124.79 (13.48)</td>
<td>51.87 (15.34)</td>
<td>IV 6.8</td>
<td>6.3</td>
<td>29.59 (5.97)</td>
<td>55.33 (25.99)</td>
<td>139.11 (4.34)</td>
<td>12.62 (2.11)</td>
<td>87.04 (18.61)</td>
<td>62.4 (9.35)</td>
<td>10.2%</td>
<td>90.5%</td>
<td>46.7%</td>
<td>44.5%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD
Significant value P ≤ 0.05
bpm: beats per minute; DM: diabetes mellitus; eGFR (Cockroft-Gault formula).

### Table 3 Mean NT-proBNP concentration according to MBP quartiles

<table>
<thead>
<tr>
<th>Quartile</th>
<th>Spearman correlation</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q1 (53.3–85.0) (n=43)</td>
<td>7684.7 pg/ml (9603.4)</td>
<td>0.147 0.05</td>
</tr>
<tr>
<td>Q2 (85.0–96.7) (n=48)</td>
<td>5888.0 pg/ml (6563.5)</td>
<td>0.147 0.05</td>
</tr>
<tr>
<td>Q3 (96.7–109.9) (n=45)</td>
<td>6015.3 pg/ml (5622.8)</td>
<td>0.043 0.05</td>
</tr>
<tr>
<td>Q4 (109.9–209.0) (n=41)</td>
<td>4300.4 pg/ml</td>
<td>0.043 0.05</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD
Significant value P ≤ 0.05

Admission BP and acute decompensated HF
showed a shorter length of stay, a non-significant reduction of 22% in readmission and a dramatic and significant reduction of 60% in mortality during follow-up. These differences cannot be attributed to age, medical antecedents, renal dysfunction, clinical profile or current treatment, since the four groups were similar in all these parameters. Although high BP (HBP) is a classical risk factor for development of HF, once it is established, the relationship between the two becomes more complex. A low level of SBP is a sign of poor prognosis among patients with HF and reduced ejection fraction. The opposite situation, prognosis of patients with a high MBP at admission, is still controversial. In the Reappraisal of Guidelines on hypertension management, published in 2009, it

Table 5 Main outcomes at follow-up according to BP

<table>
<thead>
<tr>
<th>MBP</th>
<th>Q1 (53.3–85.0)</th>
<th>Q2 (85.0–96.7)</th>
<th>Q3 (96.7–109.9)</th>
<th>Q4 (109.9–209.0)</th>
<th>RRb 95% CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death, n (%)</td>
<td>23 (15.5)</td>
<td>14 (9.2)</td>
<td>18 (12.6)</td>
<td>10 (7.3)</td>
<td>0.40 (0.19–0.85)</td>
<td>0.017</td>
</tr>
<tr>
<td>Readmission, n (%)</td>
<td>30 (20.3)</td>
<td>37 (24.2)</td>
<td>30 (21.0)</td>
<td>26 (19.0)</td>
<td>0.78 (0.46–1.33)</td>
<td>0.372</td>
</tr>
<tr>
<td>Combined event, c n (%)</td>
<td>46 (31.1)</td>
<td>49 (32.0)</td>
<td>41 (28.7)</td>
<td>30 (21.9)</td>
<td>0.58 (0.37–0.93)</td>
<td>0.024</td>
</tr>
</tbody>
</table>

Table 4 Treatment according to quartiles of MBP at admission

<table>
<thead>
<tr>
<th>MBP</th>
<th>Q1 (53.3–85.0)</th>
<th>Q2 (85.0–96.7)</th>
<th>Q3 (96.7–109.9)</th>
<th>Q4 (109.9–209.0)</th>
<th>Spearman correlation</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loop diuretics</td>
<td>81.1</td>
<td>77.1</td>
<td>86.7</td>
<td>86.9</td>
<td>0.079</td>
<td>0.057</td>
</tr>
<tr>
<td>ACEi</td>
<td>54.1</td>
<td>48.4</td>
<td>53.8</td>
<td>51.8</td>
<td>-0.003</td>
<td>0.948</td>
</tr>
<tr>
<td>ARB</td>
<td>27.7</td>
<td>32.7</td>
<td>37.1</td>
<td>40.9</td>
<td>0.103</td>
<td>0.013</td>
</tr>
<tr>
<td>BB</td>
<td>46.6</td>
<td>51.0</td>
<td>55.9</td>
<td>53.3</td>
<td>0.057</td>
<td>0.171</td>
</tr>
<tr>
<td>AB</td>
<td>29.7</td>
<td>30.7</td>
<td>23.8</td>
<td>23.4</td>
<td>-0.065</td>
<td>0.116</td>
</tr>
<tr>
<td>Digitalis</td>
<td>26.4</td>
<td>26.1</td>
<td>25.9</td>
<td>27.0</td>
<td>0.004</td>
<td>0.924</td>
</tr>
<tr>
<td>Statins</td>
<td>29.7</td>
<td>33.3</td>
<td>40.6</td>
<td>39.4</td>
<td>0.085</td>
<td>0.040</td>
</tr>
<tr>
<td>Nitrates</td>
<td>26.4</td>
<td>23.5</td>
<td>25.2</td>
<td>26.3</td>
<td>0.003</td>
<td>0.942</td>
</tr>
</tbody>
</table>

Significant value p ≤ 0.05.
Data are expressed as percentages of patients receiving each specific group of drug at discharge.
ACEi: angiotensin converting enzyme inhibitors; ARB: angiotensin receptor blockers; BB: beta blockers; AB: aldosterone blockers.

Significant value P ≤ 0.05.
\(^a\) Q4 vs. Q1.
\(^b\) Relative risk by Cox risk proportional regression analysis.
\(^c\) Death and readmission during follow-up.
has been pointed out that when low BP values are achieved, there is an associated increase, rather than an associated decrease, in the risk of cardiovascular outcomes. In HF, some studies have shown a better prognosis when BP is increased but other authors have found that the relationship follows a U-shaped curve with a worse prognosis when MBP is too low or too high. Whether such a hypothetical protective effect of HBP is restricted to patients with HF with preserved ejection fraction, or whether it also involves patients with HF and reduced ejection fraction is still a matter of debate, but it probably represents a common clinical association.

There are several possible explanations for this counterintuitive relationship between HBP and HF survival. Firstly, patients with higher admission BP may constitute a group whose heart structure (pump function) and function (tissue perfusion) is less deteriorated than those with lower BP. This hypothetical situation would enable the heart to work successfully under higher load conditions. The differential behavior between SBP and DBP in our cohort, might point in this direction. Patients with higher DBP—i.e. a higher rise in peripheral resistance—would perform better on the sole condition that their hearts were better preserved structurally and functionally. Though it sounds attractive, this picture seems unrealistic, since the relation between BP and HF persists irrespective of the type and severity of HF. Secondly, it is possible that our cohort is contaminated with patients misdiagnosed with HF, and in whom dyspnea can be explained for other reasons. In an interesting article, Ingle et al. revealed that patients being treated for a clinical diagnosis of HF with preserved ejection fraction had the same self-reported symptoms and exercise tolerance as patients with HF and reduced ejection fraction, yet had normal NT-proBNP levels and better prognosis. The perception of their symptoms was disproportionate to the evidence of their cardiac pathology. In the present study, however, mean concentration of NT-proBNP was above the cut-off point for diagnosis in the four quartiles of patients. This reinforces the role of an underlying ventricular dysfunction across the entire sample, excluding misdiagnosis of HF as the reason for the better prognosis of patients with higher MBP. Another possible explanation is that the decrease in mortality among hypertensive patients is due not to BP itself, but to obesity. Obesity is a well-recognized independent cardiovascular risk factor. In the general population, a higher BMI is associated with an increased risk for cardiovascular events and new-onset HF. However, in patients with HF, obesity carries a better, in-hospital and long-term, prognosis in terms of mortality. A number of potential mechanisms have been proposed to explain this paradoxical observation, including diagnosis of HF at an earlier stage in obese subjects, adverse effects of weight loss, residual measured and unmeasured confounding variables, a decreased catecholamine response and greater protection against malnutrition–inflammation–cachexia in patients who are overweight and obese. In our study, patients with higher MBP also had higher BMI, although unlike other studies, subgroups do not show any difference in renal function, ejection fraction or clinical antecedents, which might explain differences in prognosis. Range and mean values of BMI in our series (between 28.25 and 29.59 kg/m²) were clearly lower than those reported in the ADHERE series (21.4–38.6 kg/m²), thus attenuating the impact of obesity in prognosis, and allowing, partially at least, BP itself to have a role in ameliorating prognosis. There is still one further possibility. As suggested by Milo-Cotter et al., the association of high MBP with acute decompensated HF may be a specific disorder related to a distinct yet unknown pathophysiological mechanism. It is thus possible that we are facing a specific HF phenotype that involves higher levels of BP, a greater heart rate and overweight or obesity, as key elements, irrespective of age, HF severity, preservation of ejection fraction, renal function or other additional factors.

HF is a complex and poorly understood syndrome in which the traditional and static view can probably be challenged. For instance, is the dichotomy of classification according to the degree of ejection fraction preservation appropriate? Or, how important is the role of volemia in HF with preserved ejection fraction? Or, should classical
cardiovascular risk factors be treated similarly throughout all stages and types of HF?\textsuperscript{15} We believe that there might be factors contributing to the complexity of HF that are not sufficiently understood. In this context, it is possible that specific subgroups of patients, with their specific characteristics, have, not until now, received due attention. A better understanding of the factors influencing prognosis can help to reduce mortality by means of tailoring therapy rather than applying general and less efficient measures.\textsuperscript{33}

Our study has some limitations. All physicians are enrolled in RICA voluntarily; hence, our cohort was obtained from patients admitted under the care of a group of specially committed physicians, particularly interested in HF, which could represent a sample bias and might explain the low mortality rate of our cohort.

We could also have committed a selection bias. For ethical reasons, patients must give informed consent for their data to be included in the registry. Probably, more willing patients are included and those with greater disability or a higher degree of dependence may have been excluded.

Finally, since our patients were in-patients, BP records are based on clinically isolated measurements, obtained at the time of admission. In all likelihood, a more complete measurement based on ambulatory, or home BP monitoring, could give us a more precise view about the role BP has on the outcomes of HF.

In conclusion, HBP in patients with acute decompen-sated HF seems to be a frequent condition, which carries a better prognosis. Whether this presentation is a specific phenotype, what the mechanisms underlying its relationships are, and if it would require specific treatment, are areas which all require further study.

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**Appendix**


**References**

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