Heart Failure Is a Risk Factor for Orthopedic Fracture
A Population-Based Analysis of 16 294 Patients

Sean van Diepen, MD; Sumit R. Majumdar, MD, MPH; Jeffrey A. Bakal, PhD; Finlay A. McAlister, MD, MSc; Justin A. Ezekowitz, MBBCh, MSc

Background—Heart failure (HF) is associated with factors that may contribute to accelerated bone loss and subsequent fractures. Whether it leads to an increased fracture risk is unknown.

Methods and Results—A population-based cohort of consecutive patients ≥65 years of age with cardiovascular disease presenting to all emergency rooms between 1998 and 2001 in Alberta, Canada (n=16 294 patients), was used. The 2041 patients with a new diagnosis of HF were compared with a control group of 14 253 patients with non-HF cardiovascular diagnoses. The primary outcome was any orthopedic fracture requiring hospital admission in the year after the emergency room visit. Patients with HF had a median age of 78 years (interquartile range, 72 to 84 years), and 51.9% were female; control subjects had a median age of 73 years (interquartile range, 68 to 79 years), and 53.2% were female. In the first year after the emergency room visit, 4.6% of the HF cohort (n=93) and 1.0% of patients without HF (n=147) sustained an orthopedic fracture (P<0.001). Hip fractures occurred in 26 HF patients (1.3%) and 18 patients (0.1%) without HF (P<0.001). After multivariable adjustment, HF was independently associated with a greater risk of any orthopedic fracture (adjusted odds ratio, 4.0; 95% CI, 2.9 to 5.3) or hip fracture (adjusted odds ratio, 6.3; 95% CI, 3.4 to 11.8).

Conclusions—HF is associated with an increased risk of subsequent orthopedic fracture, particularly hip fracture. This suggests that screening for and treatment of osteoporosis to reduce fracture risk need to be considered in those with HF.

Key Words: epidemiology | heart failure | population | osteoporosis

C hronic heart failure (HF) and osteoporosis are 2 common and important public health conditions in North America. HF has a prevalence of 2.2% in the general population and 8.4% in those ≥75 years of age, and it is a leading cause of acute-care hospitalizations and mortality.1,2 Osteoporosis affects ~10 million Americans (25% of women and 12% of men >50 years of age), putting them at risk for nontraumatic fractures, particularly of the wrist, spine, and hip.3 The most devastating complication of osteoporosis is hip fracture; it is associated with a 30% 1-year mortality and, among those who survive, leads to a loss of function and independence, often necessitating admission to long-term care.4 It is particularly noteworthy that HF and osteoporosis share a number of common risk factors such as older age, female sex, smoking, and type 2 diabetes,5,6 yet the degree to which they may coexist and concurrently contribute to incident fractures is uncertain.

Clinical Perspective p 1952
Recent evidence suggests that HF is associated with factors that may contribute to accelerated bone loss and subsequent fractures. Although the prevalence of osteoporosis in unscreened patients with HF is unknown, a case series of 101 patients with HF evaluated for cardiac transplant reported that 26% had low vitamin D levels and 30% had elevated parathyroid hormone levels. Furthermore, low bone mass according to bone density testing was identified in 43% of patients at the lumbar spine and 42% at the femoral neck.7 Additionally, a series of 18 patients with HF demonstrated significantly elevated levels of parathyroid hormone–related peptide and angiotensin II levels compared with non-HF control subjects.8 In patients with HF, aldosterone levels also are elevated,4 and animal models have suggested that this elevation may stimulate increased calcium excretion and elevated parathyroid hormone levels.8,9 In addition, commonly used cardiac medications may influence osteoporosis risk. In a meta-analysis of 54 nonrandomized studies conducted in patients without HF, β-blockers and thiazide diuretics were associated with reduced fracture frequency, and loop diuretics were associated with an increased fracture frequency.10 In a large case-control study, angiotensin-
converting enzyme inhibitors were associated with a significant reduction in fracture risk, whereas angiotensin receptor blockers and potassium-sparing diuretics showed no effect on fractures.13 To the best of our knowledge, no study has directly examined the link between HF and subsequent orthopedic fracture. Therefore, we used a population-based cohort to explore the risk of fracture in elderly men and women with HF and compared them with a control group of patients with non-HF cardiovascular diagnoses. We chose such a control group (rather than healthy members of the general population) to ensure that even our control subjects were likely to receive cardiac medications and share some cardiovascular risk that may influence osteoporosis risk.

Methods

Study Design
This population-based cohort study examined the incidence of hospitalization for fracture after an initial emergency department (ED) presentation with a cardiovascular diagnosis in individuals ≥65 years of age living in the province of Alberta, Canada.

Data Sources
A database was created by linking 4 databases maintained by Alberta Health and Wellness: (1) the Ambulatory Care Database, which tracks all ED visits and outpatient hospital-based clinic visits; (2) the Alberta Inpatient Discharge Abstract Database, which records information (including dates, diagnoses, and procedures) on all admissions to acute-care facilities; (3) the Alberta Health Care Insurance Registry, which tracks vital status; and (4) the Blue Cross Medication Database, which includes medication data on all patients >65 years of age.

Subjects
The HF cohort was created by using the database to identify all residents of Alberta who presented to an ED between April 1, 1998, and March 31, 2001, with a primary diagnosis of HF (International Classification of Diseases, 9th revision, clinical modification [ICD-9-CM] code 428.x). The accuracy of hospital and ED data for case identification for patients with HF has previously been validated.8,14 The control cohort was created by using the database to identify individuals who presented to an ED between April 1, 1998, and March 31, 2001, with any of the following cardiovascular diagnoses: acute myocardial infarction (ICD-9-CM 410.x), cardiac dysrhythmia (ICD-9-CM 427.x), chest pain (ICD-9-CM 786.5, 786.51, and 786.59), stable angina (ICD-9-CM 413.x), and unstable angina (ICD-9-CM 411.1).15 A control cohort consisting of non-HF cardiovascular problems was selected because these patients would share some cardiovascular risk factors and potentially be eligible for the same cardiovascular medications used to treat HF and that might be associated with reduced or increased frequency of orthopedic fracture (eg, β-blockers and thiazide diuretics). Patients were tracked from their index ED visit for a maximum of 1 year, until they emigrated from Alberta, or until they died; follow-up was 99.5% (n = 77 patients were lost to follow-up).

Outcomes and Measurements
The primary outcome was any fracture (ICD-9-CM 733.0 and 810.x through 829.x, excluding traumatic or pathological fractures) requiring hospital admission in patients ≥65 years of age in the first year after the index ED visit for HF. The main secondary outcome was hip fracture (ICD-9-CM 820.x, 821.0, and 821.1). The accuracy and validity of diagnostic coding for fracture in these databases have previously been described.17,18 Adjustments were made for patient age, sex, medications, and comorbidities. Patient comorbidities were identified by the use of ICD-9-CM codes from the index ED visit, which allows coding for 5 other complicating or comorbid conditions. These comorbidities included hypertension (ICD-9-CM 401 to 405), diabetes (ICD-9-CM 250), atrial fibrillation (ICD-9-CM 427.31), previous myocardial infarction (ICD-9-CM 412), cerebrovascular disease (ICD-9-CM 439), peripheral vascular disease (ICD-9-CM 443), renal disease (ICD-9-CM 586), cataracts (ICD-9-CM 366), and chronic obstructive pulmonary disease (ICD-9-CM 490 to 492, 494, 496). Information on comorbidities for each patient was supplemented by records from the Alberta Inpatient Discharge Abstract Database up to 2 years before and 30 days after the index ED visit for each patient. The Inpatient Discharge Abstract Database allows coding for up to 16 other medical problems at each hospitalization. Previous studies have shown that the clinical diagnoses extracted from administrative discharge databases using ICD-9-CM codes are highly specific compared with chart review.4,19

To create a cohort of patients with an index presentation of HF and a non-HF control cohort, patients with a hospitalization for HF in the 2 years before their index ED were excluded (n = 16 653). All patients in the control group with an HF admission in the year after their index cardiovascular ED visit also were excluded (n = 13 499). In addition, 5285 patients were excluded because they had conditions known to substantially modify fracture or fall risk (see the Appendix in the online Data Supplement).

Prescription medication use was ascertained by identifying Blue Cross Medication Database claims in the 60 days before and 60 days after the index ED visit. A priori medication classes of interest included angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, β-blockers, calcium channel blockers, statins, antiplatelet agents (excluding aspirin because it is available as an over-the-counter preparation), long-acting nitrates, diuretics, spironolactone, thiazide diuretics, vitamin K antagonists, acid suppressants (proton pump inhibitors and H2-receptor blockers), bisphosphonates, other osteoporosis medications (calcitonin, estrogen preparations, hormone replacement therapy, selective estrogen receptor modulators, and recombinant parathyroid hormone), glucocorticoids, thyroid replacement hormones, anticonvulsants, sedatives (benzodiazepines, chloralhydrate, buspirone, and zopiclone), and psychiatric medications (antidepressants and antipsychotics).

Statistical Analysis
Categorical data were summarized as percentages, and differences were tested with the χ2 test; continuous variables were summarized as medians and interquartile ranges, and differences were tested with the Wilcoxon rank-sum test.

To adjust for differences in baseline characteristics in Table 1, a multivariable logistic regression was performed with 1-year hospitalized fracture as an outcome. Age, female sex, and HF were forced into the model. All other variables with values of P<0.20 in univariate analysis were candidate variables for entry into the model using a stepwise selection procedure; we used a value of P=0.15 as an exit criteria. All 2-way and 3-way interactions between medications were tested; because none were significant at a value of P<0.1, none were included in the final models. We then determined the adjusted odds ratios (ORs) for 1-year risk of hospitalization for fracture after controlling for other potentially confounding factors.

Sensitivity analyses were conducted to check for 3 additional situations: indications (ie, osteoporosis treatments prescribed to those with clinically evident disease), presentation of HF, and the difference in the distributions for age, sex, and diuretic use. To control for possible confounding by indication, the regression models were reevaluated with patients on bisphosphonates excluded. We examined presentation of HF by including only those who were admitted to hospital for their cardiovascular presentation. To control the imbalances in baseline characteristics between the distributions of age, sex, and diuretic use between the HF and non-HF group, a bootstrap model validation was conducted. This validation was done by dividing and matching the cohort into 16 groups based on age, sex, diuretic use, and HF status. A sample of 100 patients (corresponding to the approximate size of the smallest group) was drawn from each, and the regression model was estimated. This sampling...
### Results

#### Participants
Between April 1, 1998, and March 31, 2001, 39,806 elderly patients with a cardiovascular diagnosis presented to an Alberta ED. The final study cohort consisted of 2,041 HF patients and 14,253 non-HF cardiovascular control patients (1,233 with acute myocardial infarction, 4,443 with cardiac dysrhythmia, 4,966 with chest pain, 2,067 with stable angina, and 1,554 with unstable angina). Compared with control subjects, patients with HF tended to be older and were more likely to have diabetes, hypertension, atrial fibrillation, prior cerebrovascular disease, peripheral vascular disease, renal disease, and chronic obstructive pulmonary disease (Table 1). Patients with HF also were more likely to be prescribed angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, diuretics, spironolactone, vitamin K antagonists, and thyroid replacement hormones. Conversely, the use of bisphosphonates and other osteoporosis medications was higher in the non-HF control group. The 1-year mortality was 18.1% (n = 370) in the HF cohort and 8.9% (n = 1261) in the control group.

#### HF and Fracture Risk
In the first year after their index ED visit, 4.6% of patients (n = 93) in the HF cohort and 1.0% of patients (n = 147) in the control cohort sustained a fracture requiring a hospital admission (unadjusted OR, 4.6; 95% CI, 3.5 to 6.0). Similarly, the 1-year rate of hospital admission for hip fracture was 1.3% (n = 26) in the HF cohort and 0.1% (n = 18) in the non-HF control subjects (unadjusted OR for HF, 10.2; 95% CI, 5.6 to 18.6). Variables associated with 1-year risk of fracture are listed in Table 2.

#### Multivariable Analysis
After adjustment for age, sex, comorbidities, and medications, HF patients still had a 4-fold risk of sustaining a fracture requiring hospitalization in the first year after their cardiovascular ED visit (adjusted OR, 4.0; 95% CI, 3.0 to 5.2; c statistic, 0.74; the Figure). In a sensitivity analysis that excluded patients who used bisphosphonates at the time of entry into the cohort, the adjusted risk of hospitalized fracture was of similar magnitude and was statistically significant (adjusted OR, 4.0; 95% CI, 2.9 to 5.3). In adjusted analyses restricted to hip fracture as the outcome, the adjusted OR for HF was 6.3 (95% CI, 3.4 to 11.8; c statistic, 0.82; Figure). Other independent predictors of fractures requiring hospitalization included increasing age and the use of bisphosphonates. In a further sensitivity analysis restricted to those patient admitted to hospital for their index cardiovascular presentation, HF remained a significant predictor of all fractures (adjusted OR, 9.9; 95% CI, 7.2 to 13.7) and hip fractures (adjusted OR, 13.7; 95% CI, 7.3 to 26.1). In the matched sensitivity analysis (matched for age, female sex, and diuretic use), HF remained a significant predictor for all

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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.
fractures (adjusted OR, 4.6; 95% CI, 4.2 to 5.0) and hip fractures (adjusted OR, 5.4; 95% CI, 4.7 to 10.2).

**Discussion**

In our population-based cohort of >16,000 elderly patients followed up for 1 year, we found that HF was associated with a 4-fold higher risk of sustaining any fracture requiring hospitalization compared with other cardiovascular diagnoses. This novel finding remained significant after adjustment for age, sex, concurrent medications, and other conditions associated with osteoporosis-related fracture.

Is there a plausible pathophysiological link between HF and osteoporosis? Beyond shared risk factors, recent animal models suggest a more direct mechanistic link between HF and osteoporosis: Elevated aldosterone levels present in HF may stimulate increased calcium excretion, leading to elevated parathyroid hormone levels (ie, secondary hyperparathyroidism). In separate animal models, elevated aldosterone levels have been associated with increased urinary calcium and magnesium excretion, hyperparathyroidism, and diminished bone mineral density.10,11 These changes were enhanced by furosemide and attenuated with spironolactone.11 A case-control study of male veterans also demonstrated that fewer fractures were seen in those patients on spironolactone.21 Vitamin D levels are lower in HF patients than in age-matched control subjects, 22 and deficiency of 25-hydroxy-vitamin D was a risk factor for future cardiac events in the Framingham cohort.23 However, a small randomized trial of vitamin D supplementation in patients with HF did not alter survival or echocardiographic parameters but did improve markers of inflammation and other biochemical markers of secondary hyperparathyroidism.24 Further elucidation

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**Table 2. Unadjusted OR for 1-Year Fracture Risk in the Overall Cohort**

<table>
<thead>
<tr>
<th>Factor</th>
<th>No Fracture (n=16,054)</th>
<th>Fracture (n=240)</th>
<th>OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HF as presenting ED diagnosis, n (%)</td>
<td>1948 (12.1)</td>
<td>93 (38.8)</td>
<td>4.5 (3.5–6.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Age, median (IQR), y</td>
<td>74.1 (69.2–80.0)</td>
<td>79.4 (72.8–85.2)</td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>8460 (52.7)</td>
<td>178 (74.2)</td>
<td>2.6 (1.9–3.4)</td>
<td></td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>2861 (17.8)</td>
<td>42 (17.5)</td>
<td>1.0 (0.7–1.4)</td>
<td>0.9</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>1072 (6.7)</td>
<td>11 (4.6)</td>
<td>0.7 (0.4–1.2)</td>
<td>0.2</td>
</tr>
<tr>
<td>Atrial fibrillation, n (%)</td>
<td>1379 (8.6)</td>
<td>28 (11.7)</td>
<td>1.4 (0.9–2.1)</td>
<td>0.1</td>
</tr>
<tr>
<td>Previous MI, n (%)</td>
<td>780 (4.9)</td>
<td>10 (4.2)</td>
<td>0.9 (0.5–1.6)</td>
<td>0.6</td>
</tr>
<tr>
<td>Cerebrovascular disease, n (%)</td>
<td>444 (2.8)</td>
<td>7 (2.9)</td>
<td>1.1 (0.5–2.3)</td>
<td>0.9</td>
</tr>
<tr>
<td>PVD, n (%)</td>
<td>175 (1.1)</td>
<td>1 (0.4)</td>
<td>0.4 (0.1–2.7)</td>
<td>0.3</td>
</tr>
<tr>
<td>COPD, n (%)</td>
<td>1126 (7.0)</td>
<td>22 (9.2)</td>
<td>1.3 (0.9–2.1)</td>
<td>0.2</td>
</tr>
<tr>
<td>Renal disease, n (%)</td>
<td>230 (1.4)</td>
<td>7 (2.9)</td>
<td>2.1 (1.0–4.4)</td>
<td>0.06</td>
</tr>
<tr>
<td>Cardiac medications, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE or ARB</td>
<td>5382 (33.5)</td>
<td>86 (35.8)</td>
<td>1.1 (0.8–1.4)</td>
<td>0.5</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>6220 (38.7)</td>
<td>88 (36.7)</td>
<td>0.9 (0.7–1.2)</td>
<td>0.5</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>1812 (11.3)</td>
<td>32 (13.3)</td>
<td>1.2 (0.8–1.8)</td>
<td>0.3</td>
</tr>
<tr>
<td>Nitrates</td>
<td>4531 (28.2)</td>
<td>68 (28.3)</td>
<td>1.0 (0.8–1.3)</td>
<td>0.9</td>
</tr>
<tr>
<td>Thiazide diuretics</td>
<td>2328 (14.5)</td>
<td>38 (15.8)</td>
<td>1.1 (0.8–1.6)</td>
<td>0.6</td>
</tr>
<tr>
<td>Other diuretics</td>
<td>2556 (15.9)</td>
<td>76 (31.7)</td>
<td>2.4 (1.9–3.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>231 (1.4)</td>
<td>7 (2.9)</td>
<td>2.0 (1.0–4.4)</td>
<td>0.06</td>
</tr>
<tr>
<td>Statins</td>
<td>2487 (15.5)</td>
<td>30 (12.5)</td>
<td>0.8 (0.5–1.1)</td>
<td>0.2</td>
</tr>
<tr>
<td>Antiplatelets</td>
<td>427 (2.7)</td>
<td>6 (2.5)</td>
<td>0.9 (0.4–2.1)</td>
<td>0.9</td>
</tr>
<tr>
<td>Vitamin K antagonists</td>
<td>1887 (11.8)</td>
<td>30 (12.5)</td>
<td>1.1 (0.7–1.6)</td>
<td>0.7</td>
</tr>
<tr>
<td>Noncardiac medications, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acid suppressants</td>
<td>3911 (24.4)</td>
<td>58 (24.2)</td>
<td>1.0 (0.7–1.3)</td>
<td>0.9</td>
</tr>
<tr>
<td>Bisphosphonates</td>
<td>863 (5.4)</td>
<td>35 (14.5)</td>
<td>3.0 (2.1–4.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Other osteoporosis medications</td>
<td>1499 (9.3)</td>
<td>21 (8.8)</td>
<td>0.9 (0.6–1.5)</td>
<td>0.8</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>850 (5.3)</td>
<td>11 (4.6)</td>
<td>0.9 (0.5–1.6)</td>
<td>0.6</td>
</tr>
<tr>
<td>Hormone replacement therapy</td>
<td>1439 (9.0)</td>
<td>19 (7.9)</td>
<td>0.9 (0.5–1.4)</td>
<td>0.6</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>2966 (1.8)</td>
<td>7 (2.9)</td>
<td>1.6 (0.76–3.4)</td>
<td>0.2</td>
</tr>
<tr>
<td>Psychiatric drugs</td>
<td>1799 (11.2)</td>
<td>38 (15.8)</td>
<td>1.5 (1.1–2.1)</td>
<td>0.02</td>
</tr>
<tr>
<td>Sedatives</td>
<td>3561 (22.2)</td>
<td>62 (25.4)</td>
<td>1.2 (0.9–1.6)</td>
<td>0.2</td>
</tr>
<tr>
<td>Thyroid replacement</td>
<td>2017 (12.6)</td>
<td>30 (12.5)</td>
<td>1.0 (0.7–1.5)</td>
<td>0.9</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1. Psychiatric medications include antidepressants and antipsychotics. *P<0.001.
of the potential mechanism for increased orthopedic fracture risk in HF patients requires further pathophysiological studies.

What are the implications of our study? Regardless of the increased risk associated with HF, all of these patients would have already met criteria for screening with bone mineral density testing because they are >65 years of age. Only a handful of patients in our cohort were on therapeutic interventions recommended by major guidelines, although calcium and vitamin D intake was not recorded because both are available without prescription. Nevertheless, the low rate of primary or secondary measures taken is consistent with prior literature; multifaceted process-of-care interventions are clearly needed. Two of the 4 lifestyle changes recommended in osteoporosis guidelines also are endorsed as recommendations for HF patients: smoking cessation and increased physical activity. Although our data do not allow us to determine whether other comorbidities that coexist with or are part of the underlying pathophysiology of HF (eg, anemia and malnutrition) are the cause of fracture, patients with HF are readily identifiable and need to have better attention paid to bone mass and amelioration of fracture risk. Specialized HF clinics are ideally suited to ensuring that HF patients are screened for osteoporosis and treated as necessary; methods to improve primary prevention strategies should incorporate patients and their primary providers.

The increased fracture risk associated with bisphosphonates is inconsistent with the results of previous studies in non-HF populations. A previous meta-analysis has reported that alendronate and risedronate significantly improve bone mineral density and reduce both vertebral and nonvertebral fractures. We hypothesize that the true prevalence of osteoporosis in our study population was underreported on discharge abstracts and consequently these patients were not excluded from the original cohorts. Osteoporotic patients on bisphosphonates are at a higher risk of fracture than individuals with normal bone density. A sensitivity analysis excluding all individuals on bisphosphonates showed that the risk of hospitalized fracture after the diagnosis of HF remained highly significant.

Study Limitations
Our observational study has a number of inherent limitations, although most of these limitations would lead to an underes-
timate of the risk of association between HF and fracture. First, our reliance on administrative data may have resulted in an underestimation of HF. The coding of HF on ED and hospital discharge abstracts has been well validated; HF has been shown to be highly specific (95% to 98%) but under-coded (sensitivities, 59% to 77%).14–16,19,20 Second, it is possible that individuals with preexisting HF may have been inappropriately entered into the non-HF control group. However, by excluding all patients with HF listed as a primary diagnosis or as a comorbidity on ED visits and hospitalizations 2 years before the initial ED visit with a primary diagnosis of HF, we likely created a cohort of primarily index HF patients. Again, this would tend to bias to the null. Third, the administrative discharge data may underrepresent the true fracture risk because we did not capture fractures treated on an outpatient basis. Previous studies have reported that outpatient fractures tend to be undercoded in administrative databases (sensitivity, 72.9%); however, when used for ED or inpatient hip fractures, the sensitivity is up to 98%.17 Fourth, the risk for mechanical falls goes up with age, comorbid conditions, and environmental hazards (eg, stairs and lighting); this is likely underrepresented in our analysis because falls account for 5% to 10% of fractures in the elderly.30,31 However, we mitigated this risk by excluding those at highest risk of a fall. In addition, because calcium and vitamin D are available without prescription, we were unable to capture this key information. Finally, even with statistically robust techniques, residual bias and confounding by indication may still exist, as is evident with our findings with bisphosphonates.

**Interpretation**

We found a statistically significant association between the diagnosis of HF and risk of osteoporotic fracture in a large population-based cohort of elderly patients with cardiovascular diagnoses. However, osteoporosis interventions were prescribed infrequently in these patients. This suggests that increased attention needs to be paid to the screening for and treatment of osteoporosis to reduce fracture risk in those with HF.

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**Disclosures**

None.

**References**


The present study evaluates the association between heart failure and orthopedic fracture in a large community cohort of patients. Although many patients may have risk factors for osteoporosis, heart failure patients also have elevated aldosterone, elevated parathyroid hormone levels, low vitamin D levels, and low bone density; take medications that modify fracture risk; and generally are less physically active than age-matched control subjects, putting them at additional risk for osteoporosis and subsequent fracture. In this study, patients presenting with cardiovascular disease, specifically heart failure, were followed up for a year to evaluate subsequent fracture requiring hospitalization. In the next year, 4.6% of the heart failure patients (and 1% of patients with other cardiovascular disease) sustained an orthopedic fracture. This included many who sustained hip fractures. Even after adjustment for traditional risk factors for fractures, heart failure conferred a 4-fold independent risk for fracture. This suggests that screening for and treatment of osteoporosis to reduce fracture risk need to be considered in patients with heart failure. Further understanding of the mechanisms underlying this risk requires exploration.
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