Original Article

Bone Mineral Density in Chronic Heart Failure: Association with Heart Failure Phenotype and Severity

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Abstract

Background: Chronic heart failure (HF) is increasingly recognized as a systemic disorder associated with multiple extracardiac complications, including impaired bone health. Osteopenia and osteoporosis are frequently observed in HF patients and may contribute to increased frailty, fracture risk, morbidity, and mortality. However, limited data are available regarding bone mineral density (BMD) across different HF phenotypes and its relationship with disease severity. **Material and Methods:** This hospital-based cross-sectional analytical study was conducted at Government Medical College, Srinagar, between 2023 and 2025. Fifty adult male patients with chronic heart failure were enrolled. Clinical assessment included demographic characteristics, duration of HF, New York Heart Association (NYHA) functional class, history of acute decompensated heart failure (ADHF), echocardiographic evaluation, and NT-proBNP measurement. Patients were categorized into heart failure with preserved ejection fraction (HFpEF), mildly reduced ejection fraction (HFmrEF), and reduced ejection fraction (HFrEF). **Results:** The mean age of participants was 71.6 ± 9.2 years. The mean DEXA T-score was −2.12 ± 0.77. Osteopenia was present in 64% of patients, osteoporosis in 32%, and only 4% had normal BMD. Fragility fractures were documented in 16% of participants. A significant association was observed between HF phenotype and DEXA diagnosis ($p = 0.001$), with osteoporosis predominating in HFrEF patients (60%). T-score demonstrated strong negative correlations with NYHA functional class ($\rho = -0.739$, $p < 0.001$) and duration of heart failure ($\rho = -0.795$, $p < 0.001$). **Conclusion:** Reduced bone mineral density is highly prevalent among patients with chronic heart failure. Bone loss is significantly associated with heart failure phenotype and disease severity, with patients having HFrEF exhibiting the greatest skeletal impairment. Routine assessment of bone health should be considered in patients with chronic heart failure, particularly those with reduced ejection fraction and advanced disease severity, to facilitate early identification and management of osteoporosis.

Keywords: Chronic heart failure; Bone mineral density; Osteoporosis; Osteopenia; DEXA; Heart failure with reduced ejection fraction; NYHA class; Fragility fracture.

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Introduction

Heart failure (HF) is a syndrome or clinical state that is caused by a structural or functional disorder of the heart causing decreased cardiac filling and/or ejection. It continues to be a serious health condition in the world, impacting over 64 million people globally, and is now responsible for significant morbidity, mortality, repeat hospitalizations and cost of healthcare.[1] Although there has been tremendous progress in pharmacological and device management of HF, the long-term prognosis remains poor, especially in elderly patients and those with multiple comorbidities.[2] HF is currently divided into three broad categories according to its left ventricular ejection fraction (LVEF): heart failure with reduced ejection fraction (HFrEF) (LVEF ≤40%), heart failure with mildly reduced ejection fraction (HFmrEF) (LVEF 41–49%), and heart failure with preserved ejection fraction (HFpEF) (LVEF ≥50%).[3] These phenotypes have different pathophysiological and clinical features, therapeutic response and prognosis.

Heart failure is regarded as a disease of the whole organism and is gaining recognition as a systemic condition outside the cardiovascular system. The multisystem involvement includes metabolic derangements, skeletal muscle, kidneys, lungs, etc., caused by chronic neurohormonal activation, persistent inflammation, oxidative stress and endothelial dysfunction.[4,5] Of these noncardiac complications, bone disease is a significant complication that is often overlooked. Osteoporosis and osteopenia are extremely common disease states associated with decreased bone mass and loss of bone microarchitecture which can occur and increase the risk of fragility fractures.[6] The coexistence of both conditions occurs more frequently and can have a significant impact on functional disability, frailty and mortality in older adults, as both

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conditions are predominantly seen in older adults.[7] Several mechanisms have been proposed to explain the association between HF and reduced bone mineral density (BMD). Chronic activation of the renin–angiotensin–aldosterone system and sympathetic nervous system promotes increased bone resorption through stimulation of osteoclast activity.[8] Inflammatory cytokines such as tumor necrosis factor-α and interleukin-6, which are elevated in HF, further contribute to skeletal deterioration by disrupting the balance between bone formation and bone resorption.[9] Additionally, vitamin D deficiency, secondary hyperparathyroidism, reduced physical activity, cachexia, sarcopenia, and prolonged use of loop diuretics may accelerate bone loss in patients with chronic HF.[10,11] Dysregulation of the receptor activator of nuclear factor-kappa B ligand (RANKL)/osteoprotegerin pathway has also been implicated in increased osteoclast-mediated bone resorption observed in advanced HF.[12]

Evidence from observational studies has consistently demonstrated lower BMD among patients with HF compared with age- and sex-matched controls. Kenny et al. reported significantly reduced femoral BMD and increased frailty among elderly HF patients, accompanied by elevated parathyroid hormone levels and vitamin D deficiency.[13] Jankowska et al. However, observed significant decreases in total body and regional BMD in men with chronic systolic HF and showed that progressive bone loss was directly affected by disease severity and by androgen deficiency independent of each other.[14] In another study, Terrovitis et al. also concluded that lower BMD and secondary hyperparathyroidism was related to more severe HF and poor clinical outcomes.[15] More recently, Martens et al. showed a direct pathophysiological link between cardiac dysfunction and metabolic bone disease, as HF itself has been found to be an independent determinant of metabolic bone disease even after the adjustment for traditional osteoporosis risk factors.[16]

In addition to BMD decreases, patients with HF are also at risk of osteoporotic fractures. In a meta-analysis of over 53,000 patients with HF, Ge et al. found an elevated risk of overall fracture and specifically hip fracture, which is linked to high morbidity, mortality and loss of independence.[17] Vertebral fractures are also prevalent, but often overlooked, in HF populations. Verheyen et al. showed that vertebral fracture was an independent predictor of cardiovascular death and admission to hospital for an HF exacerbation, confirming the prognostic value of vertebral fracture in this population.[18]

The association between HF and osteoporosis has garnered more and more interest, but there are still some areas of important knowledge that have yet to be fully addressed. Prior studies have been mainly conducted in patients with HFrEF, and only sparse data are available regarding alterations of BMD in all disease severity stages of HF (HFmrEF and HFpEF). Additionally, in many populations, the relationship between skeletal health and markers of HF severity (NYHA functional class, duration of disease, natriuretic peptide levels) has not been well defined. Knowledge of these relationships could help identify patients who are at higher risk for osteoporosis or fragility fracture sooner, and could offer clues to the systemic effects of HF.

For this reason, the present study was set up to assess the bone mineral density in patients with chronic heart failure by dual-energy X-ray absorptiometry (DEXA) and to determine the correlation between BMD and the heart failure phenotype and the severity of heart failure. The purpose of this study is to add to the expanding literature around the importance of the comprehensive assessment of the skeleton as a key part of the care of patients with heart failure.

Materials and Methods

Study Design and setting: This was a cross-sectional analytical study conducted in Postgraduate Department of General Medicine, Government Medical College (GMC), Srinagar, Jammu and Kashmir, India for two years from 2023 to 2025 in the hospital. Patients with chronic heart failure were eligible for the study to assess bone mineral density (BMD) in this population and to examine relationships between it and phenotype of heart failure and the severity of the disease.

The Institutional Ethics Committee, Government Medical College, Srinagar, reviewed and approved the study protocol. The ethical rules set forth in the Declaration of Helsinki were followed in all procedures. All participants gave written informed consent prior to enrolment.

Study Population

Chronic heart failure (CHF) diagnosed adult males coming to the outpatient department or emergency and in-patient ward of SMHS hospital, Srinagar were screened consecutively for eligibility.

Heart failure was diagnosed by clinical evaluation, echocardiography, and biochemical markers according to current heart failure guidelines.

Inclusion Criteria

- Male patients aged ≥18 years.
- Established diagnosis of chronic heart failure irrespective of etiology.
- Ability and willingness to provide informed consent.

Exclusion Criteria

Patients were excluded if they had:

- Known hypogonadism.
- Chronic kidney disease.
- Chronic liver disease.
- Parathyroid disorders.
- Malabsorption syndromes or inflammatory bowel disease.
- Active malignancy.
- Long-term corticosteroid therapy.
- Use of medications known to affect bone metabolism and induce osteoporosis.
- Female sex.

Sample Size and Sampling Technique

A total of 50 patients with chronic heart failure were enrolled using non-probability convenience sampling. The sample size was determined based on feasibility considerations, availability of eligible participants during the study period, and accessibility of dual-energy X ray absorptiometry (DEXA) scanning facilities. The selected sample size was considered adequate to explore associations between bone mineral density, heart failure

phenotype, and markers of disease severity.

Clinical Evaluation: Thorough history and physical examination with a pre-designed proforma were performed in all enrolled participants. Demographic data, cardiovascular risk factors, comorbidities, and medication history, duration of heart failure, and a history of acute decompensated heart failure (ADHF)-related hospitalization was documented.

Anthropometric measurements, such as height and weight, were taken using standard procedures; body mass index (BMI) was computed as weight in kg. divided by height in m^2.

Assessment of Heart Failure Phenotype

A detailed transthoracic echocardiogram was performed by expert cardiologists on all patients using the standard parasternal long-axis, short-axis and apical views.

The left ventricular ejection fraction (LVEF) was determined and patients were classified based on current guideline definitions:

- Heart failure with preserved ejection fraction (HFpEF): LVEF ≥50%
- Heart failure with mildly reduced ejection fraction (HFmrEF): LVEF 41–49%
- Heart failure with reduced ejection fraction (HFrEF): LVEF ≤40%

Heart failure phenotype was analyzed both as a categorical variable and through continuous assessment of ejection fraction values.

Assessment of Heart Failure Severity

Heart failure severity was assessed using multiple clinical and biochemical indicators.

New York Heart Association Functional Class

Functional capacity was assessed by evaluating the New York Heart Association (NYHA) classification which was divided into 4 classes based on symptom severity and exercise limitation.

NT-proBNP Measurement

Standard blood collection techniques were used in the laboratory. The plasma levels of N-terminal pro-B-type natriuretic peptide (NT-proBNP) were determined by standard immunoassay methods. The biochemical marker of cardiac wall stress and severity of heart failure was recorded as NT-proBNP levels with pg/mL being used.

The length of time that an individual has been experiencing heart failure and how many times they have previously had a decompensation. The time elapsed from first documented diagnosis of heart failure to date of enrolment was the duration of HF, which was measured in years. Hospitalization for acute decompensated heart failure was observed as a marker of instability and severity of heart failure.

Bone Mineral Density Assessment

Dual-energy X-ray absorptiometry (DEXA) was used as the main intervention of the study, to measure bone mineral density. DEXA scans were obtained at standard operating conditions at clinically established skeletal sites, such as the lumbar spine and the femoral neck.

The results of comparison of the bone mineral density were expressed as T-scores, which are the number of standard deviations above or below the peak bone mass of healthy young adult reference population.

Based on World Health Organization criteria, participants were classified as:

- Normal bone mineral density: T-score ≥ −1.0
- Osteopenia: T-score between −1.0 and −2.5
- Osteoporosis: T-score ≤ −2.5

The lowest T-score obtained from the measured skeletal sites was considered for diagnostic classification.

Assessment of Fragility Fractures

A history of fragility fracture was recorded through patient interviews and review of available medical records. Fragility fracture was defined as a fracture occurring after minimal trauma, such as a fall from standing height or less.

Study Outcomes

Primary Outcome

- Bone mineral density measured by DEXA-derived T-score.

Secondary Outcomes

- Prevalence of osteopenia and osteoporosis among patients with chronic heart failure.
- Comparison of BMD across HFpEF, HFmrEF, and HFrEF phenotypes.
- Association between BMD and heart failure severity indicators including NYHA class, NT-proBNP levels, duration of heart failure, and previous ADHF hospitalization.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Python-based statistical software. Continuous variables were assessed for normality and presented as mean ± standard deviation (SD) or median with interquartile range (IQR), as appropriate. Categorical variables were summarized as frequencies and percentages.

Associations between categorical variables, including heart failure phenotype and DEXA diagnostic categories, were assessed using the Chi-square test or Fisher's exact test when applicable. Differences in DEXA T-scores across heart failure severity categories were evaluated using the Kruskal–Wallis test. Correlations between T-scores and continuous indicators of heart failure severity were assessed using Spearman rank correlation analysis. To identify independent predictors of bone mineral density, multivariable linear regression analysis was performed with DEXA T-score as the dependent variable. Covariates entered into the model included heart failure phenotype, NYHA functional class, duration of heart failure, and history of acute decompensated heart failure. Regression coefficients (β) with 95% confidence intervals (CI) were reported.

All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant.

Results

A total of 50 male patients with chronic heart failure were included in the study. The mean age of the study population was 71.6 ± 9.2 years, with a mean body mass index of 23.3 ± 2.8 kg/m^2. The mean left ventricular ejection fraction was 42.3 ± 13.0%, while the mean duration of heart failure was 4.2 ± 2.2 years. HFrEF constituted the largest subgroup (40%), followed by HFpEF (36%) and HFmrEF (24%).

[Table 1] summarizes the baseline demographic and clinical characteristics of the study population. The mean age of the participants was 71.6 ± 9.2 years, indicating that chronic heart failure predominantly affected elderly individuals. The mean body mass index was 23.3 ± 2.8 kg/m², suggesting that most patients had a normal to mildly reduced nutritional status. The mean left ventricular ejection fraction was 42.3 ± 13.0%, reflecting the inclusion of patients across the spectrum of heart failure phenotypes. The median NT-proBNP level was 1515 pg/mL, indicating a substantial degree of neurohormonal activation and cardiac dysfunction. The mean duration of heart failure was 4.2 ± 2.2 years, demonstrating a chronic disease course in most participants. Additionally, 42% of patients had a prior history of hospitalization for acute decompensated heart failure, reflecting significant disease burden and clinical instability.

Table 1: Baseline Clinical and Heart Failure Characteristics of the Study Population (N = 50)

Variable	Value
Age (years), mean ± SD	71.6 ± 9.2
BMI (kg/m²), mean ± SD	23.3 ± 2.8
Ejection Fraction (%), mean ± SD	42.3 ± 13.0
Duration of HF (years), mean ± SD	4.2 ± 2.2
NT-proBNP (pg/mL), median	1515
History of ADHF, n (%)	21 (42.0)

Table 2: Distribution of Heart Failure Phenotypes and NYHA Functional Class

Variable	n (%)
Heart Failure Phenotype	
HFpEF	18 (36.0)
HFmrEF	12 (24.0)
HFrEF	20 (40.0)
NYHA Functional Class	
Class I	8 (16.0)
Class II	17 (34.0)
Class III	13 (26.0)
Class IV	12 (24.0)

[Table 2] presents the distribution of heart failure phenotypes and functional status among study participants. Heart failure with reduced ejection fraction (HFrEF) was the most common phenotype, accounting for 40% of cases, followed by heart failure with preserved ejection fraction (HFpEF) in 36% and heart failure with mildly reduced ejection fraction (HFmrEF) in 24%. Assessment of symptom severity using the NYHA classification revealed that the majority of patients belonged to Class II (34%) and Class III (26%), while 24% were classified as Class IV and 16% as Class I. These findings indicate that most patients had moderate to severe functional limitation, providing a suitable population for evaluating the impact of heart failure severity on bone mineral density.

Table 3: Bone Mineral Density Characteristics of the Study Population

Variable	Value
T-score, mean ± SD	-2.12 ± 0.77
Osteopenia, n (%)	32 (64.0)
Osteoporosis, n (%)	16 (32.0)
Normal BMD, n (%)	2 (4.0)
Fragility fracture history, n (%)	8 (16.0)

[Table 3] demonstrates the burden of skeletal involvement among patients with chronic heart failure. The mean DEXA-derived T-score was −2.12 ± 0.77, indicating overall reduced bone mineral density within the study population. Osteopenia was identified in 64% of patients, while osteoporosis was present in 32%. Only 4% of participants demonstrated normal bone mineral density. Thus, 96% of patients exhibited evidence of reduced bone mass. Furthermore, a history of fragility fracture was documented in 16% of participants, indicating clinically significant skeletal fragility. These findings highlight the high prevalence of metabolic bone disease among patients with chronic heart failure.

Table 4: Association Between Heart Failure Phenotype and DEXA Diagnosis

Heart Failure Type	Normal	Osteopenia	Osteoporosis
HFpEF (n=18)	2	12	4
HFmrEF (n=12)	0	12	0
HFrEF (n=20)	0	8	12

[Table 4] evaluates the relationship between heart failure phenotype and bone mineral density status. A statistically significant association was observed between heart failure type and DEXA diagnosis (Chi-square test, p = 0.001), with a strong effect size (Cramer's V = 0.615). Among patients with HFmrEF, all participants were classified as osteopenic, whereas no cases of osteoporosis were identified. In the HFpEF group, osteopenia remained the predominant

diagnosis, although osteoporosis was observed in a smaller proportion of patients. In contrast, osteoporosis was the most common DEXA diagnosis among patients with HFrEF, affecting 60% of this subgroup. No patient with HFrEF had normal bone mineral density. These findings suggest that worsening systolic dysfunction is associated with progressively greater skeletal deterioration and a higher prevalence of osteoporosis.

Table 5: Correlation Between T-score and Heart Failure Severity Indicators

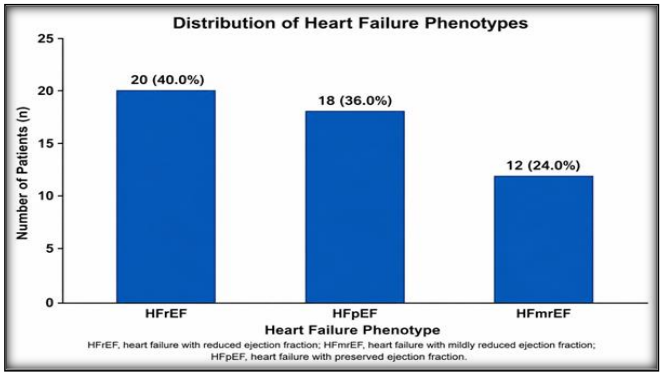
Variable	Correlation Coefficient (ρ)	p-value
NYHA Functional Class	-0.739	<0.001
Duration of HF (years)	-0.795	<0.001

[Table 5] illustrates the relationship between bone mineral density and markers of heart failure severity. A strong negative correlation was observed between NYHA functional class and DEXA T-score ($\rho = -0.739$, $p < 0.001$), indicating that patients with more severe symptoms and functional impairment had lower bone mineral density. Similarly, duration of heart failure demonstrated a strong inverse correlation with T-score ($\rho = -0.795$, $p < 0.001$), suggesting progressive skeletal deterioration with increasing chronicity of disease. These findings indicate that both symptom burden and long-standing heart failure contribute significantly to bone loss.

Table 6: Multivariable Linear Regression Analysis Predicting T-score

Predictor	β Coefficient	95% CI	p-value
HFpEF	0.41	0.09 to 0.73	0.014
HFrEF	-0.86	-1.25 to -0.47	0.0001
History of ADHF	0.19	-0.03 to 0.41	0.095
NYHA Class	0.00	-0.17 to 0.17	0.969
Duration of HF (years)	-0.08	-0.16 to 0.00	0.061

[Table 6] presents the results of multivariable linear regression analysis performed to identify independent predictors of bone mineral density. The overall model demonstrated excellent explanatory power ($R^2 = 0.809$; adjusted $R^2 = 0.788$) and was highly significant ($p < 0.001$). Heart failure phenotype emerged as the strongest independent predictor of T-score. Compared with the reference category, HFpEF was associated with significantly higher T-scores ($\beta = 0.41$, $p = 0.014$), indicating relatively preserved bone mineral density. Conversely, HFrEF was independently associated with significantly lower T-scores ($\beta = -0.86$, $p = 0.0001$), reflecting more severe bone loss. Although duration of heart failure and history of acute decompensated heart failure showed trends toward association, they did not retain statistical significance after adjustment for other variables. These findings suggest that heart failure phenotype is the principal determinant of bone mineral density in this cohort.



[Figure 1] demonstrates the distribution of heart failure phenotypes among study participants. HFrEF constituted the largest subgroup, followed by HFpEF and HFmrEF, indicating adequate representation of all major heart failure phenotypes.

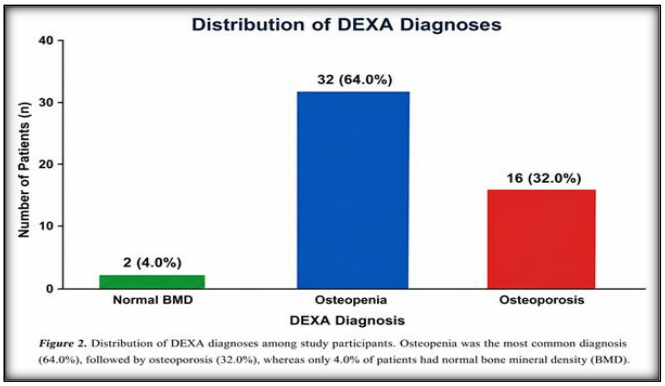
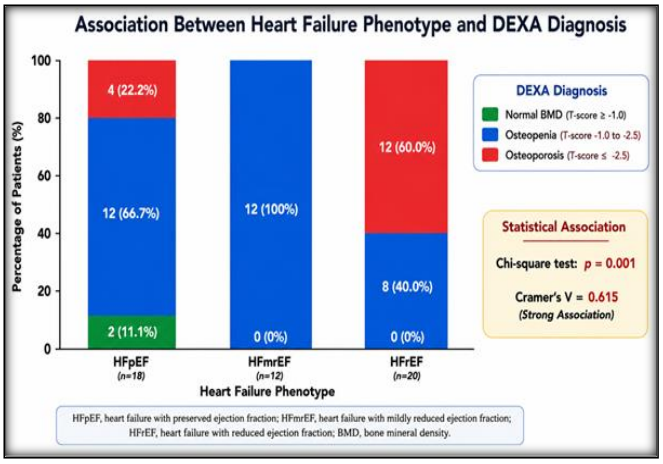


Figure 2. Distribution of DEXA diagnoses among study participants. Osteopenia was the most common diagnosis (64.0%), followed by osteoporosis (32.0%), whereas only 4.0% of patients had normal bone mineral density (BMD).

[Figure 2] illustrates the prevalence of normal bone mineral density, osteopenia, and osteoporosis in the study population. Osteopenia was the most common diagnosis, followed by osteoporosis, whereas only a small proportion of patients demonstrated normal bone mineral density.



[Figure 3] shows the distribution of DEXA diagnostic categories across heart failure phenotypes. Osteoporosis was markedly more prevalent among patients with HFrEF, whereas osteopenia predominated in HFpEF and HFmrEF.

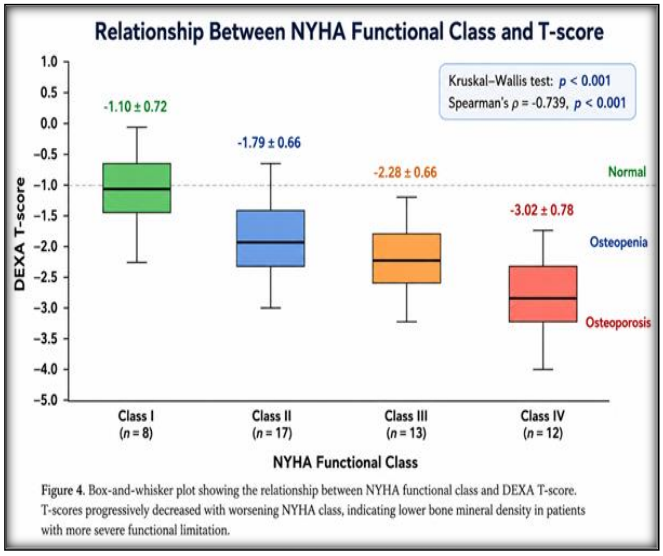


Figure 4. Box-and-whisker plot showing the relationship between NYHA functional class and DEXA T-score. T-scores progressively decreased with worsening NYHA class, indicating lower bone mineral density in patients with more severe functional limitation.

[Figure 4] depicts the association between NYHA functional class and DEXA T-score. Progressive reductions in T-score were observed with increasing NYHA class, indicating worsening bone mineral density among patients with more severe functional impairment.

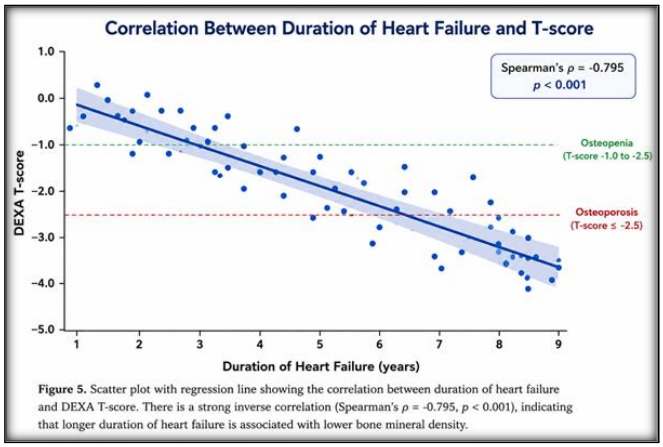


Figure 5. Scatter plot with regression line showing the correlation between duration of heart failure and DEXA T-score. There is a strong inverse correlation (Spearman's $\rho = -0.795$, $p < 0.001$), indicating that longer duration of heart failure is associated with lower bone mineral density.

[Figure 5] demonstrates a significant inverse relationship between duration of heart failure and bone mineral density. Patients with longer disease duration exhibited progressively lower T-scores, suggesting cumulative adverse effects of chronic heart failure on skeletal health.

Discussion

The current research aimed to assess the BMD in chronic heart failure (HF) patients and to address the correlation between heart failure phenotype and the severity of the disease. The main findings were that: (1) prevalence of those with reduced BMD was extremely high with 96% of patients having either osteopenia or osteoporosis; (2) there was a strong association between the HF phenotype and skeletal health with patients with HFrEF having the highest prevalence of reduced BMD; (3) there was a strong inverse relationship between the markers of HF severity, NYHA functional class and duration of HF, to the prevalence of reduced BMD; (4) the HF phenotype was the strongest independent predictor of BMD in multivariable analysis.

The rate of reduced BMD as seen in our study is much greater than what has been reported in the general elderly population and this is important as it shows both the role for skeletal involvement in chronic HF disease and the possibility that there may be a targeted role for bone-targeted therapy in this disease. The rate of osteopenia and osteoporosis was 64% and 32% respectively. This study is in line with previous studies showing that there is a high prevalence of metabolic bone disease among HF patients. In the men, Jankowska et al. have found the bone loss to be significant during follow-up and reduction in total body and regional bone mineral density in chronic systolic heart failure.[19] Likewise, Terrovitis et al. showed that osteoporosis and secondary hyperparathyroidism were very common in patients with advanced HF and were correlated with poor clinical outcomes.[20] The high prevalence of skeletal abnormalities in our cohort further corroborates the notion that HF is a disorder of more than one system, as it is a multisystem disorder and not just one affecting cardiovascular health.

The association between HF and low BMD may be a result of several pathophysiological mechanisms. Several mechanisms, including chronic activation of the renin–angiotensin–aldosterone system (RAAS), sympathetic nervous system (SNS) stimulation, systemic inflammation, oxidative stress, vitamin D deficiency, secondary hyperparathyroidism, reduced mobility and cachexia, have been associated with bone loss resulting from HF.[21,22] Bone resorption is seen because of increased levels of inflammatory cytokines (tumor necrosis factor-α and interleukin-6) and decreased physical activity, which leads to a decrease in bone formation. All of these mechanisms are linked and lead over time to the deterioration of bone health in chronic HF patients.

An intriguing result of this present study was that there was a strong relationship between heart failure phenotype and bone mineral density. HFrEF had a higher prevalence of osteoporosis and HFpEF and HFmrEF had a higher prevalence of osteopenia. Furthermore, HFrEF remained independently associated with lower T-scores after multivariable adjustment. These findings are biologically plausible because patients with HFrEF generally exhibit more pronounced neurohormonal activation, systemic inflammation, and catabolic metabolism than those with preserved ejection fraction. Our results are consistent with those reported by Verheyen et al., who demonstrated a high prevalence of osteoporosis and vertebral fractures in patients with HFrEF and showed that skeletal abnormalities were associated with poorer outcomes.[24] Similarly, Loncar et al. observed significantly lower BMD among patients with severe systolic dysfunction compared with individuals without HF.[25]

The present study also demonstrated a strong inverse relationship between NYHA functional class and BMD. Patients with more advanced symptoms exhibited significantly lower DEXA T-scores, and correlation analysis revealed a strong negative association between functional limitation and

bone density. This is a similar result to previous studies that indicated that functional status would decline as skeletal deterioration would increase. In advanced HF there is reduced exercise capacity and physical inactivity which leads to muscle wasting, diminished mechanical load of bone, and increased bone resorption.[26] Moreover, advanced NYHA class is often associated with greater neurohormonal and inflammatory activation, further exacerbating bone loss.[27]

The length of heart failure also showed a significant inverse correlation with bone mineral density. T-scores decreased with increasing duration of the disease, indicating possible cumulative effects of chronic HF on skeletal health. This was also observed by Jankowska et al. who showed gradual bone loss with aging in patients with chronic systolic HF.[19] This temporal relationship may be due to the bone tissue's long-term exposure to neurohormonal dysregulations, inflammatory mediators, nutritional inadequacies, and diminished physical activity. These findings highlight the need for early osteoporosis screening and preventive measures within patients with chronic HF.

Interestingly, NYHA class and duration of HF were independently associated with T-score in univariate analysis, but were not independent in the multivariable regression model. Heart failure phenotype was the most influential factor for bone mineral density. The results indicate that the phenotypically distinct forms of HF may explain a significant amount of the skeletal health variability. Cardiac dysfunction in patients with HFrEF is likely associated with worse systemic effects of cardiac dysfunction and consequently may result in more profound bone loss than in patients with HFpEF or HFmrEF.

These findings have a number of clinical implications. In patients with cardiovascular disease, mobility impairment, decreased quality of life, hospitalization and mortality associated with osteoporosis and fragility fractures are all significant.[28] Bone health should be monitored in selected patients with HF, especially men, with a longer disease course, more advanced NYHA class and/or HFrEF, given the high prevalence of osteopenia and osteoporosis seen in our cohort. Early detection of skeletal abnormalities can help with early intervention such as lifestyle modifications, optimizing nutrition and vitamin D replacement, fall prevention and appropriate osteoporosis treatment.

This study overall helps the field of research by providing more evidence of the relationship between cardiovascular and skeletal health. The results underscore the notion that it is important to consider chronic heart failure as a systemic disease with musculoskeletal implications. Larger and more diverse studies in the future are required to elucidate the mechanisms involved in bone loss associated with HF and should assess the effectiveness of skeletally targeted intervention for both the skeletal and cardiovascular effects.

Conclusion

This present study shows that the reduction in bone mineral density was found to be highly prevalent among CHF patients with majority being either osteopenic or osteoporotic. There was a significant association between BMD and heart failure phenotype, functional status, and duration of disease. Patients with HF with reduced EF (HFrEF) had the most impaired skeletal status and highest prevalence of osteoporosis compared with other patients with either HF with mid-range EF (HFmrEF) or HF with preserved EF (HFpEF).

As markers of the severity of heart failure, such as NYHA functional class and duration of HF, increased, a strong inverse correlation was seen between them and the DEXA T-score, suggesting progressive bone loss with increased HF severity and duration of disease. Heart failure phenotype was the strongest independent risk factor for bone mineral density after multivariable analysis, underscoring the relationship between cardiac dysfunction and bone mineral density.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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