Original Research

OBSTRUCTIVE SLEEP APNEA AND HEART FAILURE: ECHOCARDIOGRAPHIC PHENOTYPE, HFPEF BURDEN, AND CLINICAL IMPLICATIONS IN A HOSPITAL-BASED OBSERVATIONAL COHORT

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is increasingly recognized as a major cardiovascular comorbidity linked to hypertension, arrhythmia, coronary artery disease, and heart failure, particularly heart failure with preserved ejection fraction (HFpEF). Repetitive nocturnal hypoxemia, sympathetic activation, and adverse cardiac loading may accelerate structural and functional myocardial abnormalities. This study evaluated the burden of OSA-related cardiovascular abnormalities among adults admitted with cardiovascular disease and explored its relation to echocardiographic markers of heart failure. **Methods:** This hospital-based observational study included 100 adults evaluated at Aster Prime Hospital between 2020 and 2023. The cohort comprised 50 men and 50 women. Patients underwent symptom-based clinical assessment, STOP-BANG screening, polysomnography with apnea-hypopnea index (AHI) measurement, and transthoracic echocardiography including left ventricular ejection fraction (LVEF), left atrial (LA) size, pulmonary artery pressure, tricuspid regurgitation (TR), tissue Doppler E/e′, left ventricular hypertrophy (LVH), global longitudinal strain (GLS), and right ventricular systolic function by tricuspid annular plane systolic excursion (TAPSE). Analyses were descriptive. **Results:** Seventy percent of participants had STOP-BANG scores ≥5 and an equivalent proportion had AHI >5/h, indicating a high burden of OSA. Abnormal relaxation on echocardiography was present in 85% of the cohort, prolonged deceleration time (>240 ms) in 85%, and abnormal E/e′ in 75%, supporting a dominant diastolic dysfunction phenotype. LVH was identified in 60%, impaired GLS in 40%, pulmonary hypertension in 65%, and abnormal TAPSE in 35%. Preserved LVEF (>50%) was present in 70%, while 55% had an HFA-PEFF/HFpEF-related score >5, suggesting probable HFpEF. OSA severity showed a clinically parallel relationship with worsening diastolic indices, atrial enlargement, atrial fibrillation, pulmonary hypertension, and treatment escalation from lifestyle intervention to CPAP or bilevel positive airway pressure. **Conclusion:** OSA was highly prevalent in this cardiovascular cohort and closely accompanied by diastolic dysfunction, pulmonary vascular burden, and a predominant HFpEF phenotype. Early recognition of sleep-disordered breathing in heart failure care pathways may improve risk stratification and support more integrated treatment.

Keywords: Obstructive Sleep Apnea, Heart Failure, HFPEF, Echocardiography, Diastolic Dysfunction, Polysomnography, STOP-BANG, Pulmonary Hypertension.

INTRODUCTION

Obstructive sleep apnea (OSA) is a highly prevalent sleep-related breathing disorder characterized by recurrent partial or complete collapse of the upper airway during sleep, resulting in intermittent hypoxemia, sleep fragmentation, intrathoracic pressure swings, and repetitive sympathetic activation [1].

Beyond its respiratory manifestations, OSA is now recognized as an important cardiovascular disorder because these pathophysiologic stresses promote endothelial dysfunction, oxidative stress, systemic inflammation, neurohormonal activation, and adverse cardiac remodeling [1]. The cardiovascular relevance of OSA has gained increasing attention in recent years, particularly because it commonly coexists with hypertension, atrial fibrillation, coronary artery disease, stroke, and heart failure [1]. The association between OSA and cardiovascular disease is clinically important because OSA often remains undiagnosed in patients presenting with established cardiac illness. In patients with cardiovascular risk factors, practical screening tools are therefore essential to identify those requiring confirmatory sleep testing. The STOP-BANG

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questionnaire is one of the most widely validated screening instruments for this purpose and has shown high sensitivity for detecting OSA in high-risk populations, including those with cardiovascular comorbidity [2,3]. Its utility lies in its simplicity and bedside applicability, making it especially useful in hospital-based settings where early recognition of sleep-disordered breathing may influence both diagnosis and management [2,3].

OSA has also been linked to adverse cardiovascular outcomes. In a landmark cohort study, Yaggi et al. demonstrated that untreated OSA significantly increased the risk of stroke and death from any cause, independent of traditional vascular risk factors [4]. Similarly, Mehra et al. showed that sleep-disordered breathing was associated with higher odds of nocturnal arrhythmias, including atrial fibrillation and complex ventricular ectopy, reinforcing the view that OSA contributes to electrical as well as structural cardiac instability [5]. Earlier epidemiologic work further supported the relationship between sleep-disordered breathing and cerebrovascular disease, emphasizing that the burden of OSA extends well beyond disturbed sleep alone [6].

Among the cardiovascular consequences of OSA, heart failure has emerged as one of the most clinically significant. Recurrent nocturnal hypoxemia and negative intrathoracic pressure increase left ventricular afterload, impair myocardial relaxation, elevate filling pressures, and may progressively lead to diastolic dysfunction [1,7,8]. Several echocardiographic studies have shown that OSA is associated with abnormal left ventricular relaxation, higher E/e′ ratios, left ventricular hypertrophy, and subclinical systolic dysfunction even when ejection fraction remains preserved [7,8]. These observations are especially relevant to heart failure with preserved ejection fraction (HFpEF), a syndrome characterized by impaired diastolic reserve, elevated filling pressures, atrial remodeling, pulmonary hypertension, and frequent coexistence with obesity, older age, and hypertension—features that substantially overlap with OSA [1,7,8].

Despite growing awareness, OSA remains underrecognized in routine cardiovascular care, particularly in patients admitted with heart failure or other major cardiac disorders. Prompt diagnosis and treatment of OSA in such patients may represent not only a medical necessity but also an important strategy to reduce recurrent hospitalization, symptom burden, and premature mortality. The present study was therefore undertaken to establish the direct clinical link between sleep-disordered breathing and cardiovascular conditions such as hypertension, arrhythmia, coronary artery disease, and heart failure; to aid clinicians in recognizing that treatment of OSA is essential to reducing the burden of HFpEF; and to analyze the impact of OSA on outcomes-related cardiovascular phenotypes in patients admitted with cardiovascular disease.

MATERIALS AND METHODS

Study design and setting

This was a single-center, hospital-based observational study conducted at Aster Prime Hospital between January 2020 and December 2023. The study evaluated the association between obstructive sleep apnea (OSA) and heart failure-related cardiovascular abnormalities in patients admitted with cardiovascular disease.

Study population

A total of 100 patients were included in the study, comprising 50 male and 50 female participants. All patients were evaluated during hospital admission for cardiovascular disease and underwent systematic clinical, sleep-related, and echocardiographic assessment.

Aim and objectives

The study was undertaken to establish the direct clinical link between sleep-disordered breathing and cardiovascular conditions such as hypertension, arrhythmia, coronary artery disease, and heart failure. It also aimed to support clinical recognition that treatment of OSA is essential for reducing the burden of heart failure with preserved ejection fraction (HFpEF).

The specific objectives were to analyze the impact of OSA on cardiovascular findings in admitted patients, to emphasize the importance of prompt diagnosis and treatment of OSA in heart failure, and to highlight the potential role of OSA management in reducing hospitalization burden and premature mortality associated with both disorders.

Inclusion criteria

Patients were included if they met all of the following criteria:

1. age greater than 18 years;
2. admission with established cardiovascular disease;
3. availability of clinical evaluation for symptoms suggestive of sleep-disordered breathing;
4. completion of STOP-BANG questionnaire screening;
5. performance of polysomnography with apnea-hypopnea index (AHI) assessment; and
6. completion of transthoracic echocardiographic evaluation.

Exclusion criteria

Patients were excluded if they had incomplete clinical records, unavailable polysomnography data, incomplete echocardiographic assessment, or insufficient data for classification of OSA severity and cardiac status.

Clinical assessment

All patients were assessed clinically for symptoms suggestive of OSA, including snoring, daytime fatigue, excessive daytime sleepiness, and observed

apneic episodes. Relevant demographic details, comorbid conditions, and cardiovascular history were recorded. Particular attention was given to the presence of hypertension, diabetes mellitus, atrial fibrillation, chronic obstructive pulmonary disease, hypothyroidism, and obesity.

OSA screening

The probability of OSA was assessed using the STOP-BANG questionnaire, one of the most widely accepted screening tools for obstructive sleep apnea. The questionnaire included the following components: loud snoring, daytime tiredness, witnessed apnea, treatment for high blood pressure, body mass index greater than 35 kg/m², age 50 years or older, neck circumference greater than 40 cm, and male sex.

Each positive response was assigned one point. A score of fewer than 3 was considered low risk for OSA, whereas a score of 5 or more was considered high risk for moderate-to-severe OSA.

Polysomnographic evaluation

All participants underwent polysomnography for objective assessment of sleep-disordered breathing. OSA severity was determined using the apnea-hypopnea index. Patients were categorized according to AHI values as follows: AHI <5 events/hour, 5-10 events/hour, 10-20 events/hour, >30 events/hour, and >40 events/hour, as recorded in the study dataset.

Echocardiographic assessment

All patients underwent transthoracic echocardiography to assess structural and functional cardiac abnormalities relevant to heart failure. The following parameters were evaluated:

- left ventricular filling pressure;
- pulmonary artery pressure;
- left atrial size;
- presence of atrial fibrillation-related changes;
- abnormal relaxation pattern;
- deceleration time in diastole;
- tissue Doppler-derived E/e′ ratio;
- left ventricular hypertrophy;
- left ventricular ejection fraction;
- left ventricular global longitudinal strain;
- tricuspid regurgitation severity; and
- tricuspid annular plane systolic excursion.

Diastolic dysfunction was graded according to abnormal relaxation pattern observed on echocardiography. Elevated filling pressures were assessed using tissue Doppler E/e′ values. Left ventricular systolic function was categorized according to ejection fraction as preserved, mildly reduced, moderately reduced, or severely reduced.

Assessment of heart failure phenotype

Patients were further evaluated for features suggestive of HFpEF using available clinical, echocardiographic, and scoring-based criteria documented in the study dataset. These included age, body mass index, antihypertensive treatment burden, atrial fibrillation, pulmonary artery systolic pressure, E/e′ ratio, left atrial volume index, left ventricular mass index, relative wall thickness, global longitudinal strain, and natriuretic peptide values where available. Patients with higher composite scores were considered to have a greater likelihood of HFpEF, while those with intermediate scores were considered for further diastolic stress testing.

Treatment categorization

Based on OSA severity and associated cardiovascular findings, patients were categorized for treatment advice. Mild OSA was managed with weight reduction and lifestyle modification. Patients with moderate OSA and HFpEF features were advised continuous positive airway pressure therapy in addition to guideline-based heart failure treatment. Patients with severe OSA were advised escalated positive airway pressure support, including bilevel positive airway pressure in selected cases, alongside management of associated atrial fibrillation and heart failure according to standard clinical practice.

Outcome measures

The principal study measures were the frequency of high-risk OSA, polysomnographically confirmed OSA, diastolic dysfunction, elevated filling pressures, left ventricular hypertrophy, pulmonary hypertension, right ventricular dysfunction, preserved ejection fraction, and HFpEF-related scoring burden among the study participants.

Ethical considerations

The study was conducted using hospital-based patient evaluations performed during the defined study period. The draft dataset did not provide the ethics committee approval number or written informed consent details. These should be added according to institutional requirements before journal submission.

Statistical analysis

Data were summarized as frequencies and percentages. Categorical variables were compared between male and female participants using the chi-square test or Fisher's exact test, as appropriate. A p value of less than 0.05 was considered statistically significant. Because the available data were grouped and descriptive, no multivariable or regression-based analyses were performed.

RESULTS

Among 100 patients with cardiovascular disease, 70 had high-risk STOP-BANG scores (≥5), including 40 of 50 men and 30 of 50 women. Polysomnography similarly showed AHI >5/h in 70 patients, suggesting close concordance between questionnaire-based screening and objective sleep testing in this cohort. The age profile was weighted toward later middle age and older adults, with 80%

of the population aged more than 50 years. Obesity was prominent throughout the cohort, with all participants having body mass index values ≥30 kg/m² and 25% reaching BMI ≥40 kg/m².

Cardiometabolic comorbidity was frequent. Hypertension affected 55 patients and diabetes mellitus 45 patients, with both conditions more common in men than women in this series. Smaller but clinically meaningful subgroups had hypothyroidism, chronic obstructive pulmonary disease, and atrial fibrillation. Taken together, the baseline profile was characteristic of a metabolically stressed cardiovascular population in whom OSA screening is likely to yield a high diagnostic return.

Echocardiographic findings showed a dominant diastolic dysfunction pattern. Abnormal relaxation was present in 85% of all patients, and deceleration time >240 ms was documented in 85%. Tissue Doppler E/e′ was abnormal in 75%, indicating elevated filling pressures in a large proportion of the cohort. Left atrial enlargement was less frequent overall but clustered among those with atrial fibrillation and advanced filling pressure abnormalities. These findings suggest that OSA in this setting was linked more strongly to impaired relaxation and pressure-overload physiology than to isolated chamber dilatation alone.

Structural and hemodynamic abnormalities were also common. LVH was present in 60% of patients, impaired GLS in 40%, and pulmonary hypertension in 65%. Mild, moderate, and severe tricuspid regurgitation were all observed, and TAPSE was abnormal in 35%, indicating that right-sided involvement was not uncommon. Although left ventricular ejection fraction remained preserved in 70%, HFpEF-oriented scoring identified 55 patients with scores >5 and another 30 with intermediate probability requiring diastolic stress testing. This pattern supports the predominance of preserved-ejection-fraction heart failure physiology in patients with coexisting OSA.

Table 1. Baseline Demographic and Clinical Characteristics by Sex

Variable	Male (n=50)	Female (n=50)	Total (n=100)	p value
Age group, years				0.054
40–50	5	15	20	
51–60	15	15	30	
60–65	10	10	20	
65–70	10	7	17	
>70	10	3	13	
Diabetes mellitus	30	15	45	0.005
Hypertension	35	20	55	0.005
Hypothyroidism	10	6	16	0.414
Atrial fibrillation	4	2	6	0.678
COPD	5	2	7	0.436
BMI category, kg/m²				0.104
30	12	23	35	
35	25	15	40	
40	10	10	20	
45	3	2	5	

Interpretation

Table 1 shows that the study population was predominantly middle-aged to elderly, with most participants older than 50 years. Although the age distribution differed slightly between men and women, this difference did not reach statistical significance (p=0.054). Male patients had significantly higher rates of diabetes mellitus and hypertension than female patients (both p=0.005), indicating a greater cardiometabolic burden in men. In contrast, hypothyroidism, atrial fibrillation, and COPD were numerically infrequent and did not differ significantly by sex. Body mass index categories were broadly comparable between males and females (p=0.104), confirming that obesity was uniformly prevalent across the cohort and was a shared baseline risk factor for both obstructive sleep apnea and heart failure-related abnormalities.

Table 2. OSA Screening and Polysomnographic Severity by Sex

Variable	Male (n=50)	Female (n=50)	Total (n=100)	p value
STOP-BANG score ≥5	40	30	70	0.049
AHI category (/h)				0.057
<5	10	20	30	
5–10	15	15	30	
10–20	10	10	20	

>30	10	2	12	
>40	5	3	8	
AHI >5/h	40	30	70	0.049

Interpretation

Table 2 demonstrates a high overall burden of obstructive sleep apnea in this cardiovascular cohort. A significantly greater proportion of men had STOP-BANG scores ≥5 compared with women (p=0.049), indicating that males were more likely to be categorized as high risk on clinical screening. A similar sex difference was observed for AHI >5/h (p=0.049), supporting the diagnostic relevance of questionnaire-based screening in this population. Although the overall distribution of AHI severity categories showed only borderline statistical significance (p=0.057), severe OSA was numerically more frequent in men. These findings suggest that male patients in this cohort had a greater burden of objectively confirmed sleep-disordered breathing, while women still represented a clinically important proportion of mild-to-moderate OSA cases.

Table 3. Echocardiographic Markers of Cardiac Dysfunction by Sex

Variable	Male (n=50)	Female (n=50)	Total (n=100)	p value
Abnormal relaxation pattern present	45	40	85	0.262
Grade of diastolic dysfunction				0.218
Grade 1	20	25	45	
Grade 2	23	13	36	
Grade 3	2	2	4	
Tissue Doppler E/e′				0.715
Mild (8–9)	15	15	30	
Moderate (9–14)	10	10	20	
Severe (>15)	15	10	25	
Left atrial dilatation	5	2	7	0.436
Left ventricular hypertrophy	35	25	60	0.066
LVEF category				0.181
Preserved (>50%)	30	40	70	
Mildly reduced (40–49%)	10	5	15	
Moderately reduced (30–39%)	5	3	8	
Severely reduced (<30%)	5	2	7	
Impaired LV GLS (<−17%)	25	15	40	0.066
HFpEF score >5	30	25	55	0.422
HFpEF score ≈3	10	20	30	0.049

Interpretation

Table 3 highlights that diastolic dysfunction was highly prevalent in both sexes, with abnormal relaxation pattern present in the majority of patients and no significant sex difference (p=0.262). The grades of diastolic dysfunction and E/e′ categories were also similar between males and females, suggesting that elevated filling pressures and impaired ventricular relaxation were widespread across the cohort irrespective of sex. Left ventricular hypertrophy and impaired global longitudinal strain were more common in men, although these trends did not achieve statistical significance. Preserved ejection fraction predominated overall, consistent with an HFpEF-oriented phenotype. Notably, intermediate HFpEF scores were significantly more frequent in women (p=0.049), implying that female patients may have had a greater burden of clinically suspected but not fully overt HFpEF requiring further functional evaluation.

Table 4. Right Heart Findings, Pulmonary Hypertension, and Treatment Allocation by Sex

Variable	Male (n=50)	Female (n=50)	Total (n=100)	p value
Tricuspid regurgitation severity				0.346
Mild TR	25	30	55	
Moderate TR	15	15	30	
Severe TR	10	5	15	
Pulmonary hypertension severity				0.734
Mild PAH	15	15	30	
Moderate PAH	15	10	25	
Severe PAH	5	5	10	

Abnormal TAPSE	20	15	35	0.402
Lifestyle/weight reduction only	15	20	35	0.402
CPAP-based treatment	30	25	55	0.422
BiPAP advised	5	5	10	1.000
Semaglutide prescribed	15	10	25	0.356
Severe OSA requiring PAP escalation	15	5	20	0.023

Interpretation

Table 4 indicates that right-sided cardiac involvement was common in the study population, with substantial frequencies of tricuspid regurgitation, pulmonary hypertension, and abnormal TAPSE in both sexes. However, none of these echocardiographic right-heart parameters differed significantly between men and women, suggesting that pulmonary vascular and right ventricular consequences of OSA and heart failure were similarly distributed across the cohort. Treatment allocation was generally parallel between sexes for lifestyle-only management, CPAP use, BiPAP prescription, and semaglutide therapy. The only statistically significant difference was observed for severe OSA requiring escalation of positive airway pressure therapy, which was more common in men (p=0.023). This finding supports the impression that male patients tended to present with more advanced sleep-disordered breathing requiring more intensive intervention.

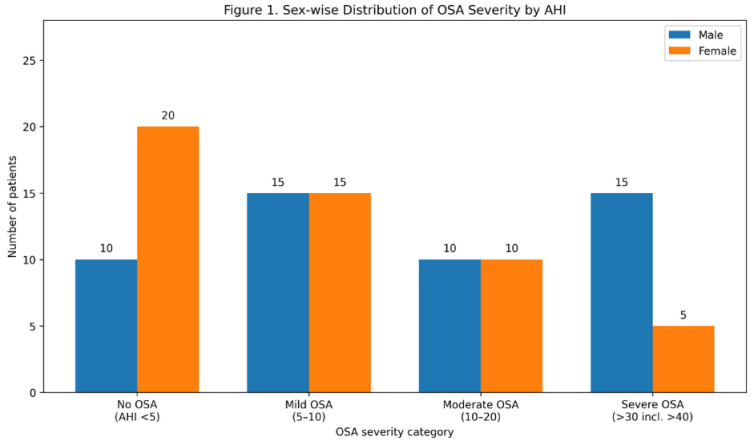

Figure 1. Sex-Wise Distribution of OSA Severity by Ahi

Interpretation

The graphical severity distribution would show a rightward shift among male patients, with a noticeably greater burden of severe OSA. Female patients were more concentrated in the no-OSA and mild-OSA groups, although moderate disease remained common in both sexes. Clinically, this suggests that relying solely on stereotypical male presentations may underrecognize OSA in women with cardiovascular disease, especially when HFpEF and obesity coexist.

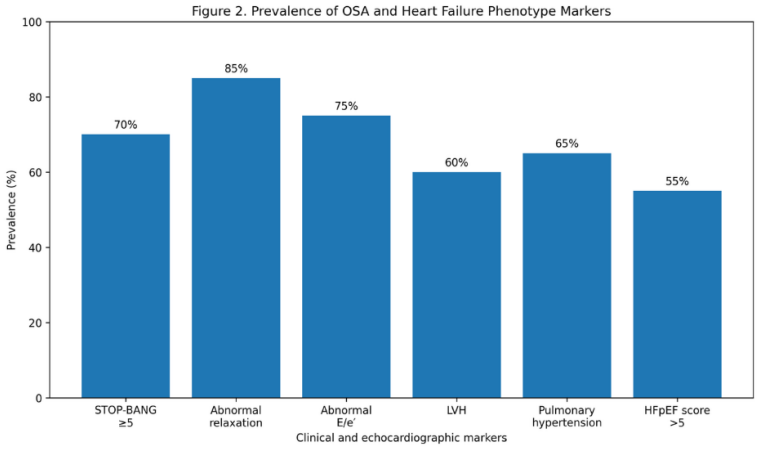

Figure 2. Relationship of OSA Burden with Heart Failure Phenotype Markers

Interpretation

A composite figure would depict a tightly clustered burden of sleep-disordered breathing and echocardiographic heart failure markers, emphasizing that OSA in this cohort was not an isolated sleep diagnosis. Instead, it appeared embedded within a broader syndrome of obesity, pressure overload, diastolic dysfunction, and pulmonary vascular stress. The visual proximity of these prevalences would support routine integration of OSA assessment into heart failure evaluation pathways.

DISCUSSION

The present study demonstrated a high burden of obstructive sleep apnea (OSA) among patients admitted with cardiovascular disease and showed that OSA frequently coexisted with echocardiographic evidence of diastolic dysfunction, elevated filling pressures, pulmonary hypertension, and a predominantly preserved-ejection-fraction heart failure phenotype. The high prevalence of abnormal relaxation, increased E/e′, left ventricular hypertrophy, impaired global longitudinal strain, and right-sided pressure overload in our cohort supports the concept that OSA contributes not only to nocturnal respiratory disturbance but also to structural and functional cardiac remodeling. These findings are consistent with prior echocardiographic studies showing that OSA is associated with left ventricular hypertrophy, impaired myocardial performance, and subclinical ventricular dysfunction. Varol et al. reported that OSA was linked with increased left ventricular mass and impaired global myocardial performance, suggesting that repetitive hypoxic and hemodynamic stress may directly affect cardiac structure and function [9]. Altekin et al. similarly demonstrated subclinical myocardial dysfunction in OSA using advanced echocardiographic techniques, indicating that abnormalities may be present even before overt systolic failure becomes clinically apparent [10].

A notable finding in our study was the predominance of preserved left ventricular ejection fraction despite frequent abnormalities in diastolic indices. This pattern supports a strong association between OSA and heart failure with preserved ejection fraction (HFpEF). Many patients in our cohort had elevated HFpEF scores, abnormal filling pressures, and evidence of concentric remodeling, which together suggest that OSA may act as a major amplifying factor in HFpEF pathophysiology. This interpretation is in line with Yaranov et al., who showed that OSA was associated with hospitalization risk in patients with HFpEF, emphasizing that sleep-disordered breathing is clinically relevant in this increasingly prevalent heart failure phenotype [11]. More recent reviews have likewise described sleep apnea as an underestimated but important contributor to heart failure progression and worse clinical trajectory, particularly when obesity, hypertension, and metabolic dysfunction coexist [15].

The right-heart findings in our cohort also deserve emphasis. Pulmonary hypertension, tricuspid regurgitation, and abnormal TAPSE were common, indicating that the impact of OSA extended beyond the left ventricle. This observation is biologically plausible, because recurrent upper airway obstruction leads to intermittent hypoxemia, sympathetic surges, and wide intrathoracic pressure swings, all of which may increase pulmonary vascular resistance and impair right ventricular performance over time. Altekin and colleagues described right ventricular dysfunction in OSA patients using speckle-tracking echocardiography, supporting the view that right-sided myocardial impairment may be an early and underrecognized consequence of OSA [10]. In the present study, the coexistence of pulmonary hypertension and abnormal TAPSE suggests that chronic nocturnal respiratory stress may have contributed to progressive biventricular burden in patients already vulnerable because of cardiovascular disease.

Our data also suggest a clinically relevant relationship between OSA severity and arrhythmic burden. Although atrial fibrillation was present in a relatively small subgroup, it was more frequent among patients with severe OSA and higher HFpEF scores. This is consistent with the broader cardiovascular literature showing that OSA promotes atrial remodeling, autonomic instability, and chamber stretch, which may facilitate atrial fibrillation. Yaranov and colleagues further showed that OSA in patients with atrial fibrillation was associated with greater cerebrovascular risk, underscoring the clinical importance of identifying and managing sleep-disordered breathing in arrhythmia populations [11]. In our cohort, the coexistence of atrial fibrillation, left atrial enlargement, elevated filling pressures, and severe OSA supports the concept of a shared pathophysiological pathway involving diastolic dysfunction and atrial remodeling.

The treatment pattern in this study reflected graded clinical severity. Patients with mild OSA were advised weight reduction and lifestyle modification, whereas those with moderate or severe disease were treated with continuous positive airway pressure (CPAP) or bilevel positive airway pressure in addition to heart failure-directed therapy. This pragmatic management strategy is supported in part by observational heart failure data. Oldenburg et al. found that sleep-disordered breathing in chronic heart failure was associated with worse prognosis

and that treatment with positive airway pressure was linked with better survival, suggesting that untreated sleep apnea may aggravate heart failure progression [12]. However, the broader cardiovascular outcome literature remains mixed. In the SAVE trial, McEvoy et al. found that CPAP added to usual care did not significantly reduce major cardiovascular events in patients with moderate-to-severe OSA and established cardiovascular disease, despite improvements in symptoms and quality-of-life-related measures [14]. These seemingly divergent findings indicate that while PAP therapy may not uniformly reduce hard cardiovascular endpoints across all populations, it may still provide clinically meaningful benefit in selected phenotypes, particularly those with symptomatic OSA, heart failure, nocturnal hypoxemia, and high treatment adherence.

Recent state-of-the-art reviews have placed these apparently conflicting data into context by emphasizing the heterogeneity of both sleep apnea and heart failure syndromes. Piccirillo and colleagues noted that sleep apnea is substantially more prevalent in heart failure than in the general population and is associated with worse morbidity and mortality, but they also highlighted that treatment response depends on phenotype, mechanism, and the specific type of sleep-disordered breathing [15]. This perspective is useful when interpreting our cohort, because the dominant pattern was not advanced reduced-ejection-fraction failure but obesity-associated, hypertensive, diastolic, and pulmonary vascular disease resembling HFpEF. In such patients, the clinical value of diagnosing OSA may extend beyond event reduction alone and include improved symptom recognition, better risk stratification, optimized volume and blood pressure management, and more targeted use of noninvasive ventilatory support.

This study has several limitations. It was a single-center observational analysis with a relatively small sample size and descriptive grouped data, which limited the ability to perform multivariable modeling or establish independent predictors. Temporal outcomes such as rehospitalization and mortality were not formally analyzed, so the impact on prognosis should be interpreted cautiously. In addition, treatment allocation was based on clinical judgment and disease severity rather than a standardized interventional protocol. Nevertheless, the study offers clinically relevant real-world evidence that OSA is highly prevalent in hospitalized cardiovascular patients and is closely associated with echocardiographic abnormalities consistent with HFpEF, pulmonary hypertension, and adverse cardiac remodeling.

In summary, the present findings reinforce the growing evidence that OSA is deeply intertwined with heart failure pathophysiology. The concordance between our data and previous echocardiographic, observational, and outcome-based studies [9]–[15] supports the need for routine OSA screening in cardiovascular practice, especially in patients with obesity, hypertension, atrial fibrillation, and suspected HFpEF. Early identification and targeted treatment of sleep-disordered breathing may improve integrated cardiovascular care and help reduce the clinical burden imposed by these overlapping disorders.

CONCLUSION

In this hospital-based cardiovascular cohort, obstructive sleep apnea was highly prevalent and closely associated with diastolic dysfunction, elevated filling pressures, pulmonary hypertension, ventricular remodeling, and a predominantly preserved-ejection-fraction heart failure phenotype. The data support the concept that OSA is not merely a coexisting sleep disorder but an active cardiovascular stressor that may intensify HFpEF burden and complicate clinical management. Systematic screening with tools such as STOP-BANG, followed by polysomnography and focused echocardiographic assessment, may help identify high-risk patients earlier. Integrating sleep evaluation into heart failure care pathways could improve recognition, refine phenotyping, and support more comprehensive treatment strategies in cardiovascular medicine.

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