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Sleep Disordered Breathing in Heart Failure

Prof. Dr. Md. Harisul Hoque[1*], Dr. Mohammad Al Mamun[2], Dr. Shampriti Islam[3], Dr. Nilufar Fatema[4]

[1]Professor, Department of Cardiology, Bangladesh Medical University (BMU), Dhaka, Bangladesh
[2]Associate Professor, Department of Cardiology, Bangladesh Medical University (BMU), Dhaka, Bangladesh
[3]Assistant Professor, Department of Respiratory Medicine, Bangladesh Medical University (BMU), Dhaka, Bangladesh
[4]Assistant Professor, Department of Cardiology, Bangladesh Medical University (BMU), Dhaka, Bangladesh

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***Corresponding author:** Prof. Dr. Md. Harisul Hoque
Professor, Department of Cardiology, Bangladesh Medical University (BMU), Dhaka, Bangladesh

Abstract **Original Research Article**

Background: Sleep-disordered breathing (SDB) is a common but underdiagnosed comorbidity in patients with chronic heart failure (CHF), potentially affecting up to 70–80% of this population. The aim of this study was to assess the frequency and characteristics of SDB among CHF patients and to compare sleep parameters between sleepier and less sleepy individuals based on the Epworth Sleepiness Scale (ESS). ***Methods:*** This cross-sectional observational study was conducted in the Departments of Cardiology and Respiratory Medicine, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from July 2022 to June 2023. Ninety-six stable CHF patients aged 18 to 75 years with elevated BNP levels and confirmed left ventricular systolic dysfunction (EF ≤ 40% or preserved) by echocardiography were enrolled. ***Results:*** The mean age of the participants was 56 ± 4.5 years; 63% were male. The mean EF was 38.5%, with 65.7% having reduced EF. The average total sleep time was 348 ± 74 minutes in Group A and 270 ± 79 minutes in Group B. The apnea-hypopnea index (AHI) was significantly higher in Group B (31 ± 14) compared to Group A (18 ± 13), suggesting more severe SDB in less sleepy patients. Arousal index and REM sleep percentages were comparable between groups. Although the frequency of OSA and CSA did not differ significantly between groups, less sleepy patients had higher AHI values, which may be linked to increased mortality risk. ***Conclusion:*** SDB is highly prevalent among CHF patients, with both OSA and CSA commonly observed. Interestingly, less sleepy CHF patients demonstrated more severe SDB, highlighting the importance of screening even in the absence of excessive daytime sleepiness.

Keywords: Chronic heart failure, Sleep-disordered breathing, Obstructive sleep apnea, Central sleep apnea, Epworth Sleepiness Scale, Apnea-Hypopnea Index, Polysomnography.

INTRODUCTION

Heart failure (HF) is a clinical syndrome which results from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.[1] Heart failure patients suffer from sleep apnea which is mostly remaining under diagnosed. For instance, individuals with heart failure may experience obstructive sleep apnea, central sleep apnea, or a combination of both. Epidemiological studies indicate that 70% to 80% of heart failure patients are affected by sleep apnea, which also serves as an independent risk factor for the onset of heart failure. Moreover, untreated sleep apnea may be associated with an increased risk for death in patients with heart failure, and treatment can reduce the risk for death and hospitalization. Timely identification of sleep apnea is needed, as treatment may improve heart failure–related outcomes. There has been an increased recognition of one factor in recent time is the contribution of sleep disordered breathing to the excess morbidity and mortality in HF. This condition is marked by recurring episodes of prolonged breathing pauses and partial neurological awakenings, which significantly affect sleep quality and overall well-being. Sleep-disordered breathing (SDB) is generally categorized into two types: obstructive sleep apnea (OSA) and central sleep apnea (CSA). OSA is common and occurs in both the general and HF populations, whereas the CSA is particularly associated with HF.2,3,4 The gold standard test for diagnosing CSA is polysomnography, or overnight sleep study, which is performed in a sleep laboratory. Optimization of HF therapy is of paramount importance, as a number of studies have shown that once HF is clinically improved, CSA may improve as well.5,6,7 Despite being one of the most populous areas of the world, no adequately powered study has been conducted in Bangladesh, evaluating the effect on sleep in heart failure population.

Thus, we had conducted a cross sectional study involving adult (18 year and above) heart failure individuals attended in outdoor or admitted indoor the department of Cardiology, BSMMU. The aim of this study was to observe the sleep pattern of heart failure patients and to the frequency of HF patient develop OSA and CSA. Then we could optimize the medical therapy of HF in this group to reduce future morbidity and mortality. Sleep disordered breathing (SDB) is common symptoms in chronic heart failure (CHF) patients.8,9,10 The absence of classic symptoms such as daytime sleepiness makes diagnosis and treatment difficult in this group.[11,12] An apnea-hypopnea index (AHI) ≥ 15 events per hour is typically used as the cutoff for diagnosis and treatment of SDB in CHF but this index is dependent on the criteria used to identify respiratory events.13,14,15,16 Respiratory scoring rules are known to influence diagnosis of obstructive sleep apnea (OSA) 17,18,19 and an important source of variation is the definition of hypopnea.18,20,21,22 . Hypopneas were scored using the American Academy of Sleep Medicine (AASM) "alternative" rule requiring an associated ≥ 3% oxygen desaturation or electroencephalographic (EEG) arousal,23 compared with a more conservative hypopnea definition requiring a corroborative ≥ 4% oxygen desaturation.24 The classification of SDB as OSA or central sleep apnea (CSA) is important when determining treatment options in CHF.[25] The scoring criteria can also impact the classification of SDB, especially if they lead to a bias towards scoring either central or obstructive respiratory events. Central apneas are typically associated with less severe oxygen desaturation compared to obstructive and mixed apneas.26,27,28 The goal of this study is to investigate the two types of Sleep disordered breathing in CHF patients and symptom analysis in during categorization of central or obstructive apneas.

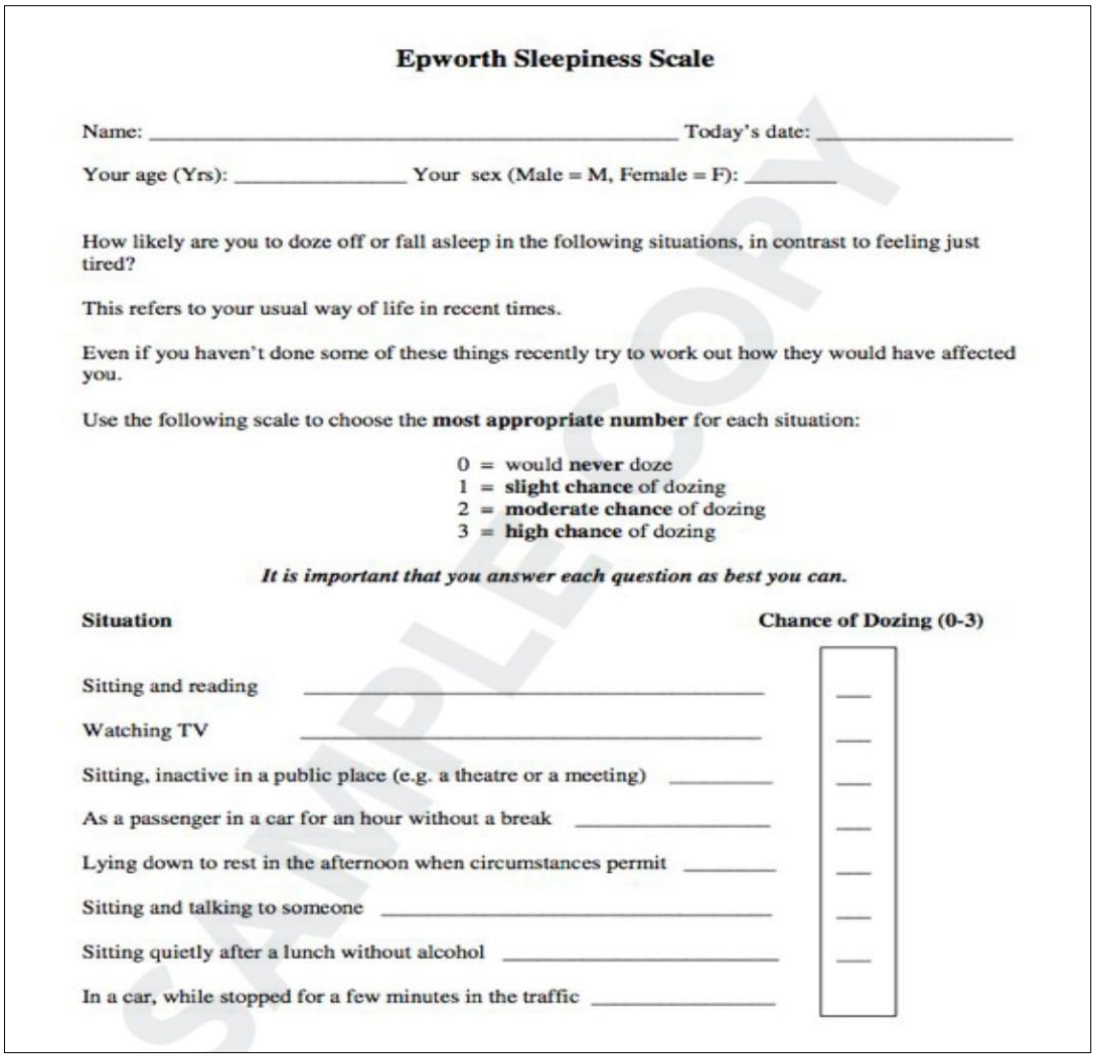
MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Departments of Cardiology and Respiratory Medicine at Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbagh, Dhaka, Bangladesh, over a one-year period from July 2022 to June 2023. A total of 96 patients aged 18 to 75 years with stable chronic heart failure (CHF) were enrolled from both inpatient and outpatient units of the Cardiology department. Patients presented with exertional or resting dyspnea consistent with NYHA class III or IV. After obtaining detailed history and performing thorough cardiovascular examination, all patients underwent electrocardiography (ECG), chest X-ray (posteroanterior view), and B-type natriuretic peptide (BNP) testing. Heart failure diagnosis was confirmed in patients with elevated BNP levels. Subsequently, color Doppler echocardiography was performed using a GE Vivid 7 echocardiography machine to detect left ventricular systolic dysfunction, with ejection fraction (EF) calculated by the Simpson method. Echocardiography was conducted by a single operator who was blinded to the clinical grouping of the patients.

All patients were then referred to the Sleep Laboratory for polysomnographic evaluation to assess the presence and frequency of sleep-disordered breathing (SDB), specifically obstructive sleep apnea (OSA) and central sleep apnea (CSA). The respiratory events were scored according to the Apnea-Hypopnea Index (AHI), where apnea was defined as complete cessation of airflow for at least 10 seconds and hypopnea as reduced airflow accompanied by ≥4% oxygen desaturation. Based on subjective sleepiness, as measured by the Epworth Sleepiness Scale (ESS), patients were categorized into two groups: Group A (sleepier, ESS ≥6) and Group B (less sleepy, ESS <6). Comparative analysis was performed between the two groups. Patients unwilling to participate were excluded from the study. Written informed consent was obtained from all participants, and data were collected using a structured data collection sheet. Statistical analysis was performed using SPSS version 25. A p-value of <0.05 was considered statistically significant.

RESULTS

Total 96 subjects were eligible for current study analysis. Mean age of study population was (56 + 4.50) years. Figure 2 shows characteristics of study subjects. Proportion of males was 57%. Sixty-three percent of the patients were male. The mean ejection fraction was 38.5%. 65.7% that is majority of the patients had a reduced ejection fraction (≤ 40%) whereas the remaining 34.3% had a preserved ejection fraction. The average total sleep time from polysomnography was 348 minutes (5.8 hr ; SD: 2.0) and the average recording time was 8 hr (SD: 1.4). Table 1 shows Group A is Sleepier ESS ≥ 6 (n = 63) and group B is Less sleepy ESS < 6 (n = 33) by using Epworth Sleepiness Scale; Table 4 described Total sleep time (min) 348 ±74 in Group A and 270 ± 79 in group B. Slow-wave sleep (% of total sleep time) 13 ± 8 and 10± 7 in Group A and B respectively. REM sleep (% of total sleep time) 15 ± 10 and 17 ± 8(P-value: 0.37), Sleep latency (min) 28 ± 23 and 27 ± 31 (P-value: 0.59). Table 5 revealed AHI (no/h of sleep) 18 ± 13 and 31 ± 14 (P-value: 0.06), Obstructive events (%) 53 ± 28 and 51 ± 29 (P-value: 0.76), Obstructive sleep apnoea, n (%) 28 and 15 (P-value: 0.98), Central sleep apnoea, n (%) 22 and 18 (P-value: 0.67), Arousals (no/h of sleep) 30 ± 12 and 31 ± 13 (P-value: 0.05), Mean SaO2 (%) 96 ± 2 and 95± 1 (P-value: 1.86), Periodic leg movements (no/h of sleep) 20 ±28 and 26 ± 27 (P-value: 0.08) in Group A and Group B respectively. (Values are expressed as mean ± standard deviation).



Epworth Sleepiness Scale

Name: _____ Today's date: _____

Your age (Yrs): _____ Your sex (Male = M, Female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the **most appropriate number** for each situation:

0 = would **never** doze
1 = **slight chance** of dozing
2 = **moderate chance** of dozing
3 = **high chance** of dozing

It is important that you answer each question as best you can.

Situation	Chance of Dozing (0-3)
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g. a theatre or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

Figure 1: Epworth Sleepiness Scale (Sleepier ESS ≥ 6, less sleepy ESS< 6)

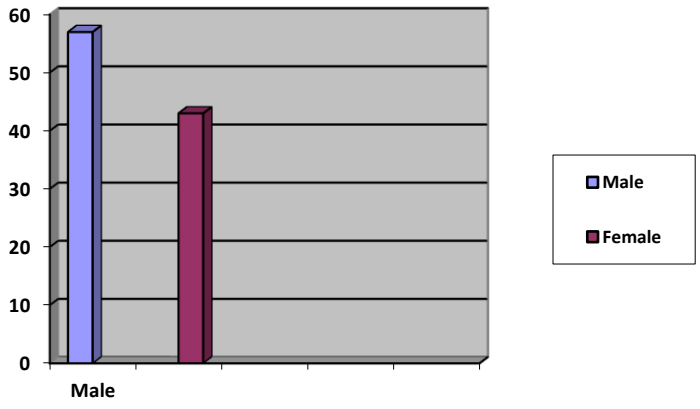


Figure 2: Sex distribution of study group; Male 57% and Female 43% (N=96)

Table 1: Groups of study subjects by Epworth Sleepiness Scale (Sleepier ESS ≥ 6, less sleepy ESS< 6)

Study population	(N=96)	Percentage
Sleepier	63	65.62%
Less sleepy	33	34.37%

Table 2: Characteristic factors of patients (N=96)

Variable	Sleepier ESS>6 (n=63)	less sleepy ESS< 6(n=33)	P-value
Age	56±4.5	63±3.8	0.19
BMI	30±5	27±4	0.23
NYHA-III	37	20	0.91
NYHA- IV	27	12	0.97
Atrial fibrillation	6	5	0.43
LVEF (%)	25±7	24±6	0.83
Ischemic cardiomyopathy	21	17	0.49
History of Diabetes Mellitus	43	24	0.91
History of hypertension	46	37	0.51
History of dyslipidaemia	48	28	0.17
On diuretic:frusemide	50	23	0.29
On ACEi/ ARB	43	25	0.07
On Betablocker	39	18	0.82
On Spiranolactone	21	7	0.14

Table 3: Vital parameters in polysomnography (N=96)

Variable	Sleepier ESS>6 (n=63)	less sleepy ESS< 6(n=33)	P-value
Heart rate	76±06	84±08	0.95
Systemic blood pressure(SBP)	125±08	132±06	0.92
Diastolic blood pressure(DBP)	76±04	85±05	0.79
respiratory rate	16±04	18±03	1.65
Mean SpO2	96±02	95±01	1.86

Table 4: Sleep duration in polysomnography (REM=rapid eye movement)

Variable	Sleepier ESS>6 (n=63)	less sleepy ESS< 6(n=33)	P-value
Total sleep time (min)	348± 74	270±79	0.31
Slow wave sleep(% of total sleep time)	13±8	10±7	0.35
REM sleep(% of total sleep time)	15±10	17±8	0.37
Sleep latency(min)	28±23	27±31	0.59

Table 5: Polysomnography data of study patients. (N=96), AHI, apnoea–hypopnoea index; ESS, Epworth Sleepiness Scale;

Variable	Sleepier ESS>6 (n=63)	less sleepy ESS< 6(n=33)	P-value
AHI (no/hr of sleep)	18±13	31±14	0.06
Obstructive events(%)	53±28	51±29	0.76
Obstructive sleep apnoea; n(%)	28	15	0.98
Central sleep apnea; n(%)	22	18	0.67
Arousal (no/hr of sleep)	30±12	31±13	0.05
Periodic leg movements (no/hr of sleep)	20±28	26±27	0.08

DISCUSSION

The present study provides insight into the relationship between SA and patients with HF. First, this study made the observation that in patients with HF and SA, those who were less sleepy, defined as an ESS score <6, had increased mortality risk than those who were sleepier, defined as an ESS >=6, independently of other risk factors. These findings suggested that in HF patients with SA, lower ESS values or lack of subjective sleepiness would have poor prognosis. The ascending reticular activating system that involves the brainstem, posterior hypothalamus, thalamus, and forebrain plays an important role in promoting wakefulness. And a specific group of nuclei in the brainstem appears to be essential for the control of sleep and wake states.[29] Neurons in the brainstem reticular formation receive inputs from the sensory systems and project upwards to the thalamus, hypothalamus, and basal forebrain. Firing of neurons projecting from those areas to the cortex produces cortical activation and increases the level of alertness.[29,30] The solitary tract nucleus in the brainstem is a point of convergence of many autonomic neural afferents, including inputs from both the arterial baroreceptors in the carotid sinus and peripheral chemoreceptors in the carotid body.[31] The recurrent cycles of hypoxia and re-oxygenation characteristic of SA sensitize carotid body chemoreceptors.[32] Enhanced neural input to solitary tract nuclei with projections to cortical autonomic regions augments efferent sympathetic discharge[33] and elicits arousal via the Locus

Coeruleus in the ascending reticular activating system. When HF and SA co-exist, these independently and additively reset upwards resting efferent sympathetic discharge during wakefulness, suggesting the concurrent induction of hyper-arousal.[34] Chronically, such hyper-arousal could manifest as lack of objective and subjective excessive daytime sleepiness, in spite of less time spent asleep.[35] Therefore, although sleep fragmentation by repetitive apnoeas and arousals in SA patients without HF could promote daytime sleepiness, such an effect might be counteracted centrally, in patients with both conditions, by markedly increased sympathetic nerve activity (SNA). Indeed, a study reported that in HF patients with OSA, the degree of daytime sleepincrease assessed by the ESS scores was inversely related to muscle SNA during wakefulness and the degree of very low frequency heart rate variability, an index of SNA, during sleep such that the greater the SNA, the less sleepy the patient.[36] Elevated SNA is associated with increased mortality in patients with HF.[34] Overall, among individuals with both heart failure (HF) and sleep apnea (SA), those who report less daytime sleepiness may face a higher risk of mortality compared to those who feel sleepier—potentially due to heightened sympathetic nerve activity (SNA). In contrast, for patients without HF, SA tends to induce sleepiness and elevate SNA, but in most cases, the sedative impact of SA surpasses the arousing influence of increased SNA. Therefore, in non-HF individuals, the presence of daytime sleepiness may simply reflect the intensity of SA and the extent of sleep fragmentation caused by repeated apneas and arousals. In such situations, because subjective sleepiness correlates with SA severity, sleepier patients may actually have a worse prognosis than those who feel less sleepy. This has been supported by findings showing that, in a non-HF population, individuals with SA and excessive daytime sleepiness had a significantly higher mortality risk compared to those without such sleepiness.[37] Furthermore, prior research in HF patients with reduced left ventricular ejection fraction (LVEF) has demonstrated that both obstructive sleep apnea (OSA) and central sleep apnea (CSA) with an apnea-hypopnea index (AHI) above 15 were linked to increased mortality compared to those with an AHI below 15. In addition, CSA patients who received CPAP treatment because results from the CANPAP trial,[38] CPAP for CSA, revealed no effect on mortality. That is why, an AHI cut-off point of 15 was used in the present study. In group A, sleepier HF patients show less AHI that less sleepy group B HF patients. High AHI would have increase mortality which is in group B patient in this study and has poor prognosis.

CONCLUSION

This study demonstrates that HF patients became less sleepy due to increase frequency of obstructive sleep apnea at night. Among heart failure patients, those who reported less daytime sleepiness exhibited a higher apnea-hypopnea index (AHI) compared to their sleepier counterparts. This elevated AHI in the less sleepy group is associated with an increased risk of mortality, indicating a poorer prognosis. CPAP can be used to improve prognosis in patient who have OSA with optimum medication for heart failure.

Limitations: This was a single centered study.

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Conflicts of interest: There are no conflicts of interest.

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