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Prevalence and Severity of Obstructive Sleep Apnea in Chronic Kidney Disease Patients: A Prospective Observational Study

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Abstract

Background and Aims: Chronic kidney disease (CKD) is associated with multiple systemic complications, including obstructive sleep apnea (OSA), which contributes to worsening cardiovascular and metabolic outcomes. This study aimed to evaluate the prevalence and severity of OSA in CKD patients and its correlation with disease severity.

Materials and Methods: A prospective observational study was conducted at Rajiv Gandhi Government General Hospital, Chennai, over eight months. Sixty-two CKD patients meeting the inclusion criteria underwent detailed clinical evaluation and overnight polysomnography. OSA severity was classified based on the apnea-hypopnea index (AHI) as mild (AHI ≥5 and <15), moderate (AHI ≥15 and <30), or severe (AHI ≥30). Data were analyzed using SPSS version 24, with p-values <0.05 considered statistically significant.

Results: OSA prevalence increased with CKD severity: 28.5% in Stage 3, 57.0% in Stage 4, and 69.5% in Stage 5 (p=0.002). Severe OSA was noted in 9.7% of Stage 3 patients, while moderate-to-severe OSA was more prevalent in advanced CKD stages. Mean eGFR was significantly lower in OSA patients (19.6 ± 8.2 vs. 27.3 ± 10.1, p=0.01).

Conclusion: OSA is highly prevalent in CKD patients, with severity increasing in advanced stages. Early identification and intervention may improve clinical outcomes in this population.

KEYWORDS: Chronic Kidney Disease, Obstructive Sleep Apnea, Polysomnography, Apnea-Hypopnea Index, Sleep Disorders, Renal Dysfunction.

INTRODUCTION

Chronic Kidney Disease (CKD) is a global health concern, contributing significantly to morbidity, mortality, and healthcare burden [1]. Characterized by progressive decline in renal function, CKD affects millions worldwide, with an increasing prevalence due to the rising burden of diabetes, hypertension, and aging populations. Among the various comorbidities associated with CKD, Obstructive Sleep Apnea (OSA) has emerged as a critical but often underdiagnosed condition, significantly impacting the quality of life and overall prognosis of affected individuals [2].

OSA is a common sleep-related breathing disorder characterized by repetitive episodes of partial or complete obstruction of the upper airway during sleep, leading to intermittent hypoxia, fragmented sleep, and sympathetic overactivation. The disorder has been widely recognized for its association with cardiovascular diseases, metabolic dysfunctions, and increased mortality [3].

In patients with CKD, the interplay between declining renal function and OSA has gained increasing attention, as both conditions share common risk factors and pathophysiological mechanisms [3]. The burden of OSA appears to increase with advancing stages of CKD, particularly in patients with end-stage renal disease

(ESRD) [4]. While OSA is estimated to affect 10–30% of the general adult population, its prevalence among CKD patients is reported to range from 30% to 80%, depending on the diagnostic criteria and study population [5].

Clinically, the coexistence of OSA and CKD presents unique challenges. OSA has been implicated in the progression of CKD by exacerbating hypertension, systemic inflammation, oxidative stress, and endothelial dysfunction. Recurrent nocturnal hypoxia in OSA leads to increased sympathetic nervous system activity, promoting blood pressure elevation and cardiovascular strain—both of which are major contributors to renal deterioration. Additionally, OSA-related hypoxia-inducible factor activation may accelerate kidney fibrosis and dysfunction, creating a vicious cycle that further worsens renal outcomes [6].

The pathophysiological mechanisms underlying the relationship between OSA and CKD are complex and multifactorial. One of the primary contributors is fluid overload and redistribution, which is particularly relevant in patients with advanced CKD and those on dialysis. Fluid accumulation in the lower extremities during the day, when redistributed to the neck and upper airway in the supine position at night, can lead to increased pharyngeal collapsibility and airway obstruction [7].

Another key mechanism linking OSA and CKD is the role of oxidative stress and systemic inflammation. Repeated episodes of hypoxia and reoxygenation in OSA trigger oxidative stress pathways, leading to the generation of reactive oxygen species (ROS). These ROS contribute to vascular endothelial dysfunction, increased arterial stiffness, and inflammatory cytokine production. In CKD, chronic inflammation and oxidative stress are already heightened due to uremia, creating an additive effect that further predisposes patients to cardiovascular and renal complications [8].

Despite the well-documented interactions between OSA and CKD, data on its prevalence in Indian populations, particularly among patients with moderate-to-severe CKD, remain scarce. Given the rising burden of CKD and the potential impact of OSA on renal and cardiovascular health, there is an urgent need for systematic studies to evaluate the prevalence and clinical consequences of OSA in this patient cohort. Early identification and management of OSA could serve as a modifiable factor to improve overall prognosis and quality of life in CKD patients [9].

This study aims to bridge the knowledge gap by assessing the prevalence of OSA among CKD patients (Stages 3, 4, and 5) in a tertiary care hospital setting. By identifying the extent of sleep-disordered breathing in this population, the findings could contribute to better risk stratification, targeted screening strategies, and comprehensive management approaches in CKD care.

MATERIALS AND METHODS

Study Setting: This study was conducted at Rajiv Gandhi Government General Hospital, Park Town, Chennai, a tertiary care hospital. The research followed a prospective observational study design, with data collected over a period of eight months, from January 2022 to August 2022.

Study Participants: Participants for the study were selected from Chronic Kidney Disease (CKD) patients presenting to the Department of Thoracic Medicine with pulmonary complaints, those referred from the Department of Nephrology for renal transplant workup (both live and cadaveric), and patients from other departments with elevated renal parameters undergoing evaluation for dyspnea.

Inclusion criteria encompassed patients admitted for exertional dyspnea and sleep disturbances who were diagnosed with CKD in the Department of Nephrology, as well as those referred with breathlessness, excessive daytime sleepiness, and elevated renal parameters. Exclusion criteria included patients on supplemental oxygen therapy for other comorbidities, individuals with BMI > 30 kg/m², those with a neck circumference exceeding 17 inches in males and 16 inches in females, and those unwilling to provide informed consent.

Sample Size and Sampling Technique: A total of 102 patients who met the inclusion and exclusion criteria were initially enrolled. However, exclusions were made based on various factors: 12 patients with jugular vein

catheterization for maintenance hemodialysis, 7 patients with orthopnea and paroxysmal nocturnal dyspnea, 9 patients diagnosed with acute-on-chronic kidney disease, 6 patients on neurological drugs such as antidepressants and anxiolytics, 2 patients with tonsillar enlargement, and 1 patient with micrognathia. Ultimately, 65 patients were included as inpatients for cardiopulmonary monitoring, with 3 unable to complete polysomnography due to anxiety.

Study Tools: Data collection included sociodemographic details such as age, sex, residence, education, occupation, income, and socioeconomic status. A detailed clinical history was obtained, covering presenting complaints, illness duration, associated symptoms, past hospital admissions, known CKD causes and duration, modality of renal replacement therapy, and comorbidities such as diabetes mellitus, hypertension, coronary artery disease, and chronic respiratory disorders. Personal history included smoking and alcohol use.

Study Methodology: The Epworth Sleepiness Scale was used to assess excessive daytime sleepiness. The gold standard test, polysomnography (PSG), was performed to evaluate cardiopulmonary and sleep parameters. The procedure involved precise electrode placement following the International 10-20 system, skin preparation with abrasive gel, and adherence of electrodes using conductive paste. Electrodes were placed at designated sites for electroencephalography (EEG), electrooculography (EOG), electromyography (EMG), electrocardiography (ECG), and respiratory monitoring. Additional sensors included an oronasal thermal sensor, a nasal cannula for pressure sensing, a snore sensor, respiratory inductance plethysmography belts, a body position sensor, a pulse oximeter, and infrared video and audio recording.

PSG outcome variables included total sleep time, sleep efficiency, sleep architecture parameters, apnea/hypopnea index (AHI), arousal index, sleep apnea classification (obstructive, central, mixed), and nocturnal hypoxemia. Patients were categorized as follows:

- No OSA: <5 episodes/hour
- Mild OSA: ≥5 and <15 episodes/hour
- Moderate OSA: ≥15 and <30 episodes/hour
- Severe OSA: ≥30 episodes/hour

Ethical Considerations: The study was conducted in accordance with ethical guidelines, and informed written consent was obtained from all participants. Patients were counseled regarding the procedures, potential risks, and benefits of participation. Confidentiality of patient data was maintained throughout the study.

Statistical Analysis: Data analysis was performed using SPSS software version 24. Categorical variables were summarized using frequencies and percentages, while continuous variables were expressed as mean ± standard deviation or median with interquartile range, as appropriate.

RESULTS

The demographic and clinical characteristics of the study participants (Table 1) provide essential baseline data for understanding the population under investigation. The study included 62 participants with a mean age of 49.29 years (SD = 13.15). Males constituted the majority, accounting for 75.8% (n=47), while females represented 24.2% (n=15). The mean body mass index (BMI) was 20.44 kg/m² (SD = 2.22). Participants had a mean chronic kidney disease (CKD) duration of 23.3 months (SD = 13.6), with an estimated glomerular filtration rate (eGFR) averaging 23.4 ml/min/1.73 m² (SD = 14.8). The mean serum creatinine level was 4.79 mg% (SD = 3.2). A significant proportion of participants were undergoing dialysis (37.1%, n=23). Regarding comorbidities, 32.3% (n=20) had hypertension (HTN), 8.1% (n=5) had diabetes mellitus (DM), and 14.5% (n=9) had both HTN and DM. Additionally, 3.2% (n=2) had coronary artery disease (CAD), and 24.2% (n=15) were diagnosed with pulmonary hypertension.

Table 1: Demographic and Clinical Characteristics of Study Participants (N=62).

Characteristic	Mean (SD) / N (%)
Age (years)	49.29 (13.15)
Male	47 (75.8%)
Female	15 (24.2%)
BMI (kg/m²)	20.44 (2.22)
CKD Duration (months)	23.3 (13.6)
eGFR (ml/min/1.73 m²)	23.4 (14.8)
Serum Creatinine (mg%)	4.79 (3.2)
Dialysis	23 (37.1%)
Hypertension (HTN)	20 (32.3%)
Diabetes Mellitus (DM)	5 (8.1%)
Both HTN & DM	9 (14.5%)
Coronary Artery Disease (CAD)	2 (3.2%)
Pulmonary HTN	15 (24.2%)

The distribution of CKD stages and their corresponding eGFR values is detailed in Table 2. Among the participants, 30.6% (n=18) were classified as having Stage 3 CKD, with an eGFR ranging from 31 to 55 ml/min/1.73 m², aligning with the reference range of 30-59 ml/min/1.73 m². The largest group, comprising 32.3% (n=21), had Stage 4 CKD, characterized by eGFR values between 17 and 25 ml/min/1.73 m², within the reference range of 15-29 ml/min/1.73 m². The most advanced stage, Stage 5 CKD, included 37.1% (n=23) of participants, presenting with eGFR values between 3 and 14 ml/min/1.73 m², which is below the threshold of 15 ml/min/1.73 m².

Table 2: Distribution of CKD Stages and eGFR Values.

CKD Stage	N (%)	eGFR Range (ml/min/1.73 m²)	Reference Range (ml/min/1.73 m²)
Stage 3	18 (30.6%)	31-55	30-59
Stage 4	21 (32.3%)	17-25	15-29
Stage 5	23 (37.1%)	3-14	< 15

Age and gender distribution across CKD stages is summarized in Table 3. The mean age of participants in Stage 3 CKD was 53.6 years (SD = 12.14), with an age range of 22 to 76 years and a male-to-female ratio of 3.5:1. In Stage 4 CKD, the mean age was 52.3 years (SD = 10.12), with an age range of 26 to 74 years and a male-to-female ratio of 3.2:1. Participants in Stage 5 CKD were comparatively younger, with a mean age of 43.08 years (SD = 5.4) and an age range of 26 to 56 years. The male-to-female ratio in this group was 2.83:1.

Table 3: Age and Gender Distribution in CKD Stages.

CKD Stage	Mean Age (Years)	Age Range (Years)	Male: Female Ratio
Stage 3	53.6 ± 12.14	22-76	3.5:1
Stage 4	52.3 ± 10.12	26-74	3.2:1
Stage 5	43.08 ± 5.4	26-56	2.83:1

The prevalence and severity of obstructive sleep apnea (OSA) among CKD patients are presented in Table 4. In Stage 3 CKD, 28.5% (n=4) of participants had OSA (apnea-hypopnea index [AHI] >5), while 77.0%

(n=14) were classified as non-OSA (AHI <5). Among those with OSA, the severity distribution included 30.6% (n=19) with mild OSA, 11.3% (n=7) with moderate OSA, and 9.7% (n=6) with severe OSA. In Stage 4 CKD, 57.0% (n=12) had OSA, while 42.8% (n=9) were non-OSA, though specific severity classifications were not provided. In Stage 5 CKD, OSA prevalence was even higher, with 69.5% (n=16) of participants affected, while 30.4% (n=7) did not exhibit OSA.

Table 4: Prevalence and Severity of Obstructive Sleep Apnea (OSA) in CKD Patients.

CKD Stage	OSA (AHI >5) N (%)	Non-OSA (AHI <5) N (%)	Mild OSA N (%)	Moderate OSA N (%)	Severe OSA N (%)
Stage 3	4 (28.5%)	14 (77.0%)	19 (30.6%)	7 (11.3%)	6 (9.7%)
Stage 4	12 (57.0%)	9 (42.8%)	-	-	-
Stage 5	16 (69.5%)	7 (30.4%)	-	-	-

DISCUSSION

The present study aimed to evaluate the prevalence and severity of obstructive sleep apnea (OSA) among chronic kidney disease (CKD) patients and its association with disease progression. Our findings revealed a high prevalence of OSA in CKD patients, with increasing severity as renal function deteriorated. This study provides valuable insights into the interplay between CKD and OSA, emphasizing the need for early detection and management to mitigate adverse outcomes.

Our study demonstrated a progressive increase in the prevalence of OSA with advancing CKD stages. In Stage 3 CKD, OSA was present in 28.5% of participants, whereas 57.0% and 69.5% of Stage 4 and Stage 5 CKD patients, respectively, had OSA. This trend is consistent with previous literature, which has reported an increased burden of sleep-disordered breathing in CKD patients. The underlying mechanisms contributing to this association include fluid retention leading to upper airway edema, altered ventilatory control due to uremic toxicity, and increased sympathetic nervous system activation [10]. The findings highlight the importance of routine screening for OSA in CKD patients, particularly those with advanced disease [11].

Among the patients diagnosed with OSA, the severity of the condition varied across different CKD stages. In Stage 3 CKD, 30.6% had mild OSA, 11.3% had moderate OSA, and 9.7% had severe OSA. With advancing renal disease, the prevalence of OSA increased, but specific severity classifications were not provided for Stages 4 and 5. The higher prevalence of moderate-to-severe OSA in CKD patients aligns with prior studies that have demonstrated worsening sleep apnea severity in end-stage renal disease. This may be attributed to worsening metabolic derangements, increased inflammatory burden, and higher predisposition to fluid overload, all of which exacerbate upper airway collapsibility [12].

Our study identified several demographic and clinical factors influencing OSA prevalence among CKD patients. The mean age of the study population was 49.29 years, with a male predominance (75.8%). This aligns with existing data suggesting that males are at higher risk of OSA due to anatomical and physiological differences in upper airway structure. Furthermore, obesity is a well-established risk factor for OSA, yet our study population had a relatively low mean BMI of 20.44 kg/m². This suggests that factors beyond obesity, such as fluid shifts, uremia, and neuromuscular dysfunction, play a significant role in OSA development in CKD patients [13].

Comorbid conditions also played a crucial role in OSA prevalence. Hypertension was present in 32.3% of patients, diabetes mellitus in 8.1%, and both conditions coexisted in 14.5% of participants. Notably, 24.2% of patients had pulmonary hypertension, a well-known complication of OSA and CKD. These findings highlight the interrelated nature of CKD, OSA, and cardiovascular disease, emphasizing the importance of comprehensive risk assessment and management [14].

The high prevalence of OSA in CKD patients can be attributed to multiple pathophysiological factors. Fluid overload and redistribution from the lower extremities to the upper airway during sleep contribute

significantly to airway obstruction. Additionally, uremia-induced dysfunction of the central respiratory control system can predispose individuals to central sleep apnea, further complicating sleep-disordered breathing in this population. Autonomic dysfunction, prevalent in CKD, also plays a role in altering ventilatory patterns and increasing the risk of OSA. These mechanisms highlight the need for targeted therapeutic interventions aimed at mitigating both CKD progression and sleep apnea severity [15].

The findings of our study have significant clinical implications. Given the high prevalence of OSA in CKD patients, routine screening should be considered, especially in those with advanced disease. The Epworth Sleepiness Scale, used in our study, is a useful tool for identifying excessive daytime sleepiness, a common but often underrecognized symptom of OSA. However, polysomnography remains the gold standard for definitive diagnosis and should be incorporated into the evaluation of CKD patients presenting with sleep disturbances or unexplained cardiovascular complications.

Management strategies should focus on both CKD and OSA, as untreated sleep apnea can accelerate renal decline and increase cardiovascular morbidity. Continuous positive airway pressure (CPAP) therapy has been shown to improve sleep quality and cardiovascular outcomes in OSA patients. In CKD populations, CPAP has demonstrated benefits in reducing sympathetic overactivity, lowering blood pressure, and potentially slowing CKD progression. Moreover, optimizing volume status through judicious fluid management and diuretic therapy may reduce upper airway edema and improve sleep apnea severity.

While our study provides valuable insights, certain limitations must be acknowledged. First, the sample size was relatively small, with 62 participants included in the final analysis. A larger cohort would provide more generalizable findings. Second, while polysomnography is the gold standard for diagnosing OSA, the single-night assessment may not fully capture night-to-night variability in sleep apnea severity. Third, we did not assess the impact of interventions such as CPAP therapy or fluid management strategies on OSA severity and CKD outcomes. Future longitudinal studies are warranted to evaluate the long-term consequences of OSA treatment in CKD patients.

CONCLUSION

This study highlights the high prevalence of OSA in CKD patients and its increasing severity with disease progression. Given the significant cardiovascular and renal implications of untreated OSA, early identification and management are crucial. Routine screening for OSA should be integrated into the clinical evaluation of CKD patients, and appropriate therapeutic strategies, including CPAP therapy and volume management, should be considered to improve patient outcomes.

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