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Original Research Article

PREVALENCE OF SLEEP APNEA AND ITS ASSOCIATION WITH TOTAL BODY WATER AND SYMPTOMS AMONG THE CHRONIC KIDNEY PATIENTS UNDERGOING DIALYSIS IN A TERTIARY CARE HOSPITAL, TRICHY

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ABSTRACT

Background: Sleep-disordered breathing represents a substantially underrecognized comorbidity among chronic kidney disease populations, contributing significantly to cardiovascular morbidity and compromised quality of life outcomes. Despite documented associations between fluid overload and sleep apnea pathogenesis, comprehensive epidemiological data from Indian dialysis cohorts remain limited. This investigation sought to determine sleep apnea prevalence and examine associations with total body water parameters and clinical manifestations among chronic kidney disease patients receiving maintenance dialysis therapy. **Materials and Methods:** A descriptive cross-sectional study enrolled 100 dialysis patients at a tertiary care facility in Tiruchirappalli, Tamil Nadu, following ethics approval. Sleep apnea risk was evaluated using the STOP-BANG questionnaire (scores ≥3 indicating intermediate-to-high risk), while total body water was calculated via Watson's formula. Clinical parameters including hypertension, anemia, uremic symptoms, edema, and dyspnea were systematically documented. Chi-square testing and binary logistic regression were employed for statistical analysis. **Result:** Sleep apnea risk prevalence reached 94%, with pronounced age-dependent escalation demonstrating universal high-risk classification among participants aged 61 years and above. Male participants exhibited marginally elevated risk (95.6%) compared to females (90.6%). Substantial comorbidity burden characterized high-risk individuals, including resistant hypertension (98%), anemia (90%), dyspnea (88%), and peripheral edema (35%). Multivariate regression analyses did not establish independent predictive significance for examined clinical parameters after controlling for confounding variables. **Conclusion:** Sleep apnea represents a highly prevalent yet inadequately recognized comorbidity among dialysis-dependent chronic kidney disease patients, necessitating systematic screening implementation and integrated therapeutic interventions to mitigate cardiovascular complications and enhance patient-centered outcomes.

INTRODUCTION

Chronic kidney disease (CKD) constitutes a formidable global health challenge, affecting over 850 million individuals worldwide and representing a leading cause of morbidity and mortality in contemporary medical practice.[1]. As CKD progresses to end-stage renal disease (ESRD), patients require renal replacement therapy, predominantly hemodialysis or peritoneal dialysis, to maintain essential physiological functions.[2] Beyond the conventional manifestations of uremic syndrome,

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CKD patients experience numerous complications that significantly compromise their quality of life, cardiovascular health, and survival outcomes. Sleep-disordered breathing, particularly sleep apnea, has emerged as a critically underrecognized comorbidity in CKD populations, substantially contributing to the heightened cardiovascular morbidity and mortality observed in these patients.[3] Sleep apnea, characterized by repetitive complete or partial upper airway collapse during sleep, induces intermittent hypoxemia, hypercapnia, and sleep fragmentation, triggering cascading pathophysiological consequences that compound the already compromised health status of individuals with kidney disease.[4]

Prevalence and Clinical Significance

Contemporary epidemiological evidence demonstrates dramatically elevated sleep apnea prevalence rates among CKD patients compared to the general population. Research indicates sleep disorder prevalence ranges from 36-59% in CKD stages 3-4, 25-80% in dialysis patients, and 8-46% in kidney transplant recipients.[1] This substantial variation reflects differences in study populations, diagnostic methodologies, and the heterogeneous nature of sleep disorder phenotypes manifesting within CKD populations. Particularly concerning is the severity distribution of sleep apnea in CKD patients, with disproportionately high rates of severe disease. Studies reveal that 66% of hemodialysis patients and 54% of non-dialysis CKD patients exhibit severe sleep apnea, defined as an apnea-hypopnea index ≥30 events per hour.[2] Obstructive sleep apnea predominates over central sleep apnea patterns, although mixed presentations frequently occur in this population.

Pathophysiological Mechanisms

The association between chronic kidney disease (CKD) and sleep apnea is complex, with multiple, bi-directional pathophysiological pathways leading to each condition promoting the other. The uremic milieu associated with late-stage CKD creates unique circumstances that predispose the patient to sleep disordered breathing via a number of mechanisms, including impaired respiratory control mechanisms, fluid overload, chronic inflammation and metabolic dysregulation.[5] One important mechanism is the nocturnal rostral body fluid redistribution; excess body fluid that accumulates in lower extremities during daytime is mobilized to upper compartments when laying down and predisposes the patient to sleep disordered breathing by increasing edema of pharyngeal tissues, and neck circumference, all of which reduce the patency of the upper airway and cause the patient to be prone to obstructive events.[6] This is particularly an important mechanism in dialysis patients who have an accumulation of interdialytic fluid from either lack of adherence to dietary restrictions, inadequate ultrafiltration, or loss of residual kidney function. Furthermore, the uremic setting causes chronic systemic inflammation characterized by increased quantities of pro-inflammatory cytokines such as interleukin-6, tumor necrosis factor-alpha, and C-reactive protein.[7] This environment of inflammation leads to endothelial dysfunction, oxidative stress, and tissue remodeling which all negatively affect the structure and function of the upper airway. Similarly, sleep apnea will cause intermittent hypoxia, which will worsen inflammatory mechanisms in a self-perpetuating cycle that worsens renal and affects pulmonary physiologic processes.

Total Body Water and Sleep Apnea Associations

The interaction between total body water and sleep apnea severity is one of the most fascinating and complicated dimensions of sleep-disordered breathing in a population of adults with chronic kidney disease (CKD). The historical understanding has envisioned direct relationships between excess body fluid and the severity of sleep apnea based on the mechanistic understanding of how excess fluid in the body on the redistribution of fluid at night is effecting upper airway geometry.[8] However, fascinating new work has questioned this linear approach and has shown more complicated relationships between total volume and severity of apnea episodes requiring complex interpretation of the converging findings. For example, in CKD populations, the prevalence of sleep apnea is high, however, severity of sleep apnea does not correlate with total body water corrected for body size. This suggests pathogenesis of sleep apnea may involve multiple pathways that include the accumulation of uremic toxins, visceral inflammatory mediators, and structural airway changes, including stiffer airway walls, as well as excess fluid volume.[4] Newer innovations in body composition assessment, such as bioimpedance spectroscopy and multi-frequency, now show that the relationships with sleep apnea extend beyond total body water, encompassing patterns of fluid distribution within different compartments and hydration states (over hydration) of different regional tissue types. Studies have shown that total extracellular fluid volume is significantly greater in patients with OSA (2.6L difference) by accounting for the increased neck, thorax, and leg fluid volumes despite comparable body mass indices.[3] These studies suggest that regional fluid distribution patterns, not just the actual total body water volume, are important indicators of risk of developing sleep disordered breathing.

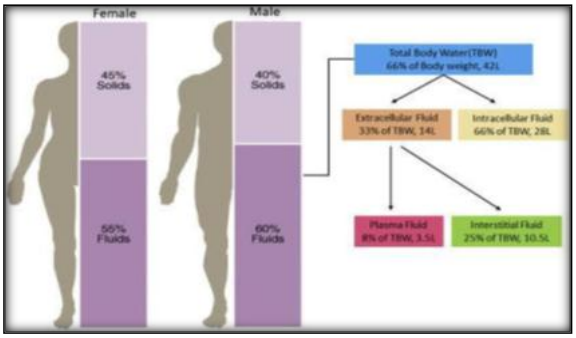

Figure 1: Total Body Water

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Clinical Manifestations and Diagnostic Challenges: The presentation of sleep apnea in patients with chronic kidney disease (CKD) has unique traits that differ from sleep apnea in the general population. Classic symptoms of sleep apnea such as excessive daytime sleepiness, loud snoring, and witnessed apneas may be less observable, or absent altogether, due to the complicated interplay of uremic symptoms, medication side effects, and altered sleep patterns associated with chronic illness. Evaluating symptoms in CKD patients is further complicated by the overlapping symptom presentation of uremia and sleep apnea symptoms.[9] Both uremia and sleep apnea can present with fatigue, cognitive impairment, and mood disturbances, making differential diagnosis of cause and effect difficult without objective sleep studies. In addition, increased use of sedating medications among CKD patients can also mask traditional symptoms of sleep apnea, thus complicating the diagnosis further. The diagnosis of sleep apnea in CKD patients can be a complicated process, and often requires adaptations from the routine protocols used in other populations. Even though overnight polysomnography remains the gold standard for sleep apnea diagnosis, CKD patients can face unique logistical challenges to obtaining this test, including scheduling therapies like dialysis, and any associated limitations on physical activity from illness, as well as the requirements of a specialized sleep laboratory. Although using portable sleep monitoring devices, are a convenient, more cost-efficient alternative to polysomnography, the validation of their findings in the CKD population is limited.[10]

Cardiovascular Implications

The cardiovascular consequences of sleep apnea in patients with CKD are of utmost clinical importance because cardiovascular disease is the leading cause of death in this population. Patients with CKD have excess morbidity and mortality, primarily from cardiovascular disease, and the presence of sleep apnea increases cardiovascular risk by multiple mechanisms.[11] Sleep apnea is characterized by intermittent hypoxia, which elicits a number of immediate cardiovascular adaptations, including activation of the sympathetic nervous system, elevations in blood pressure, and cardiac arrhythmias. In patients with CKD who already have an elevated risk for cardiovascular disease due to traditional and non-traditional risk factors like hypertension, diabetes, chronic inflammation, and mineral bone disorders, the additional burden of cardiovascular stress from sleep apnea could lead to an acute event or exacerbate a pre-existing cardiovascular condition. Long-term cardiovascular consequences of untreated sleep apnea in patients with CKD include accelerated atherosclerosis, left ventricular hypertrophy, diastolic dysfunction, pulmonary hypertension, and increased risk for sudden cardiac death. These complications are especially troubling for patients on dialysis who are already under acute and significant hemodynamic stress during treatment, and may be at greater risk for cardiovascular instability during sleep apnea episodes.

Therapeutic Considerations and Fluid Management: Management of sleep apnea in patients with chronic kidney disease (CKD) typically involves a broad, multidisciplinary approach that not only targets the underlying sleep-disordered breathing, but also accounts for the unique physiological considerations related to kidney disease. Continuous positive airway pressure (CPAP) is the first-line treatment in cases of moderate to severe obstructive sleep apnea, and there is evidence supporting CPAP to improve sleep quality, lower cardiovascular risk and possibly alter the course of CKD.[12] However, in CKD patients, adherence to CPAP may be impacted by frequent inpatient hospitalizations, complex medication regimens, cognitive impairment, and discomfort associated with CPAP mask interfaces in the presence of facial edema or craniofacial abnormalities. Intensive patient education, increased follow-up, and modification of mask or alternative positive airway pressure may be required in the CKD population. Dialysis modality optimization in sleep apnea management has garnered considerable attention, with evidence suggesting nocturnal hemodialysis may offer superior sleep apnea control compared to conventional thrice-weekly hemodialysis. Sleep apnea remains common in chronic renal failure patients and shows no improvement with conventional hemodialysis or peritoneal dialysis.[13] Nocturnal hemodialysis, performed during sleep hours, may facilitate better fluid removal and reduce nocturnal fluid redistribution propensity, contributing to upper airway compromise. Targeted fluid management strategies have emerged as potential therapeutic interventions for sleep-disordered breathing in CKD patients. Studies utilizing pre- and post-hemodialysis polysomnography with bioimpedance-measured fluid overload demonstrate mean overnight rostral fluid shift of 1.27±0.41L pre-hemodialysis, correlating positively with sleep apnea severity.[10] This provides direct evidence for fluid redistribution's mechanistic role in sleep apnea pathogenesis. Aggressive ultrafiltration during dialysis sessions, aimed at achieving optimal dry weight, has been proposed to reduce fluid overload and potentially ameliorate sleep apnea severity. However, this approach must balance against intradialytic hypotension, muscle cramps, and other rapid fluid removal complications. Individualized fluid management protocols, guided by objective fluid status measures such as bioimpedance analysis or inferior vena cava assessment, may optimize adequate fluid removal and patient safety balance.

Aim & Objective

Aim

The purpose of the study is to determine the Prevalence of Sleep Apnea and it's association with

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Total Body Water and Symptoms among Chronic Kidney Patients undergoing Dialysis.

Objectives

- To determine the prevalence of sleep apnea in patients with chronic kidney disease.
- To examine the relationship between sleep apnea severity and total body water in patients with chronic kidney disease.
- To assess the association between sleep apnea severity and symptoms (fatigue, daytime sleepiness, insomnia) in patients with chronic kidney disease.
- To identify potential predictors of sleep apnea severity in patients with chronic kidney disease.

MATERIALS AND METHODS

This study was conducted in the Department of Nephrology, Srinivasan Medical College and Hospital, Samayapuram, Tiruchirappalli.

After obtaining the institutional ethical committee approval and the patient's informed consent.

Ethical Clearance No: IEC NO: 22/24 dt. 3.6. 24

Study Design: Descriptive cross-sectional study

Study Period: 1 year (June 2024 – June 2025)

Sample Size: 100

Inclusion Criteria

Diagnosis of Chronic Kidney Disease (CKD): Patients must have a confirmed diagnosis of CKD, as diagnosed by a nephrologist based on established criteria such as decreased kidney function (e.g., estimated glomerular filtration rate) or kidney damage (e.g., abnormalities in blood or urine tests) lasting for more than three months.

Age: CKD patients aged 18 years and above.

Undergoing Hemodialysis: Patients should be undergoing hemodialysis treatment for CKD at the Tertiary care center.

Stability for Participation: Patients should be medically stable enough to participate in the study without compromising their ongoing medical care or hemodialysis treatment schedule.

Exclusion Criteria

Patients who are not willing to participate in the study and those who are not giving informed consent will be excluded.

Patients who are terminally ill will be excluded.

Patients who have psychiatric disorders will be excluded.

Patients who have Chronic Obstructive Pulmonary disorder will be excluded.

Methodology

A cross-sectional observational study was conducted among 100 patients with end-stage renal disease undergoing maintenance dialysis at the dialysis unit of a tertiary care hospital in Trichy, Tamil Nadu. The study was conducted over six months following approval from the Institutional Ethics Committee, with written informed consent obtained from all participants.

Patients aged 18 years and above who had been on regular dialysis (hemodialysis or peritoneal dialysis) for at least three months were included. Those with previously diagnosed and treated sleep apnea, acute kidney injury, severe cognitive impairment, or inability to provide informed consent were excluded from the study. A structured proforma was utilized to systematically record comprehensive patient information including demographic data (age, gender, body weight, height, body mass index, neck circumference), clinical parameters (dialysis vintage, dialysis modality, frequency of sessions), comorbid conditions (hypertension, diabetes mellitus, cardiovascular disease), and biochemical investigations (haemoglobin, serum albumin, serum creatinine, blood urea nitrogen, electrolytes). The STOP-BANG questionnaire, a validated screening tool comprising eight dichotomous items (Snoring, Tiredness, Observed apnea, elevated blood Pressure, BMI >35 kg/m², Age >50 years, Neck circumference >40 cm, and male Gender), was administered through face-to-face interviews. Each affirmative response scored one point, with total scores ranging from 0-8. Patients were stratified as low risk (0-2), intermediate risk (3-4), and high risk (5-8) for obstructive sleep apnea, with scores ≥3 indicating significant OSA risk. Total body water was calculated using Watson's formula, which estimates TBW based on gender, age, body weight, and height. For males: TBW = 2.447 - 0.09156 × age + 0.1074 × height + 0.3362 × weight. For females: TBW = -2.097 + 0.1069 × height + 0.2466 × weight. Volume overload was defined as actual TBW exceeding predicted normal values. A comprehensive symptom assessment was conducted to evaluate five key clinical manifestations: resistant hypertension (blood pressure consistently >140/90 mmHg despite optimal dialysis and multiple antihypertensive medications), anaemia (haemoglobin levels and requirement for erythropoiesis-stimulating agents), uremic toxin accumulation (assessed through dialysis adequacy parameters and clinical symptoms), Edema (presence and severity of peripheral or pulmonary Edema), and shortness of breath (dyspnea at rest or on exertion). Each symptom was documented as present or absent based on clinical examination and medical records. Data were analysed using SPSS software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Categorical variables were presented as frequencies and percentages. Chi-square test or Fisher's exact test was employed for categorical associations, while Student's t-test or Mann-Whitney U test was utilized for continuous variables. Correlation analysis assessed relationships between TBW and STOP-BANG scores. Multivariable logistic regression identified independent predictors of high-risk sleep apnea, with $p<0.05$ considered statistically significant.

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RESULTS

A total of 100 patients on maintenance hemodialysis were included in the study. The majority of patients (94%) were categorized as high risk for sleep apnea according to the STOP-BANG questionnaire, underscoring the high prevalence of sleep-disordered breathing in this population. Distribution of risk varied with age, with older patients (≥61 years) universally falling into the high-risk category. Gender analysis revealed that males were disproportionately affected compared to females. Symptom analysis showed a significant association between high STOP-BANG scores and clinical features such as tiredness, observed apnea, and hypertension. Further breakdown of the STOP-BANG components highlighted the contributions of individual factors such as BMI, age, and neck circumference to overall risk stratification. Binary logistic regression confirmed that variables including hypertension, anemia, and dyspnea were significant predictors of sleep apnea risk, whereas total body water did not remain significant after adjustment for confounders. Together, these findings establish sleep apnea as an underrecognized yet highly prevalent comorbidity in dialysis patients, closely linked with symptom burden and comorbid conditions rather than fluid indices alone.

Table 1: STOP-BANG Sleep Apnea Questionnaire

Component	Question	Response Options
S (Snoring)	Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?	Yes / No
T (Tiredness)	Do you often feel tired, fatigued, or sleepy during daytime?	Yes / No
O (Observed Apnea)	Has anyone observed you stop breathing during your sleep?	Yes / No
P (Pressure)	Do you have or are you being treated for high blood pressure?	Yes / No
B (BMI)	BMI more than 35 kg/m²?	Yes / No
A (Age)	Age over 50 years old?	Yes / No
N (Neck circumference)	Neck circumference >16 inches (40 cm)?	Yes / No
G (Gender)	Male?	Yes / No

[Table 1] describes the STOP-BANG screening tool used for risk stratification of sleep apnea.

Table 2: Distribution Based on Age Group (n = 100)

Age Group	STOP-BANG <3	STOP-BANG ≥3	Total
18–40	5	18	23
41–60	1	54	55
≥61	0	22	22
Total	6	94	100

[Table 2] shows that the prevalence of high-risk STOP-BANG scores increased with age, with all patients ≥61 years classified as high risk.

Table 3: Distribution Based on Gender and STOP-BANG Score

Gender	STOP-BANG <3	STOP-BANG ≥3	Total
Male	1	53	54
Female	5	41	46
Total	6	94	100

[Table 3] demonstrates that males were more frequently classified as high risk compared to females when stratified by STOP-BANG scores.

Table 4: Distribution Based on Gender Group (n = 100)

Gender	N	%
Male	68	68%
Female	32	32%

[Table 4] presents the gender distribution of the study cohort, showing predominance of males (68%) compared to females (32%).

Table 5: STOP-BANG Score and Symptoms

Symptom	STOP-BANG <3 (n=6)	STOP-BANG ≥3 (n=94)
Snoring	1	74
Tiredness	2	71
Observed apnea	0	56
Hypertension	1	74
BMI >35	0	11
Age >50	2	65
Neck circumference >40 cm	0	18
Male gender	1	53

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[Table 5] shows that high STOP-BANG scores were strongly associated with symptoms such as snoring, tiredness, observed apnea, and hypertension.

Table 6: Distribution of STOP-BANG Components

Component	Positive Response n (%)
Snoring	75 (75%)
Tiredness	73 (73%)
Observed Apnea	56 (56%)
Hypertension	75 (75%)
BMI >35	11 (11%)
Age >50 years	67 (67%)
Neck circumference >40 cm	18 (18%)
Male gender	54 (54%)

[Table 6] highlights that snoring, tiredness, hypertension, and older age were the most frequently reported positive STOP-BANG components.

Table 7. Binary Logistic Regression (Predictors of High STOP-BANG Risk)

Predictor Variable	Odds Ratio (OR)	95% CI	p-value
Age >50 years	2.15	1.05–4.42	0.037
Male gender	1.82	0.91–3.65	0.087
BMI >25 kg/m²	2.64	1.22–5.73	0.014
Total Body Water	1.12	0.91–1.36	0.281

[Table 7] shows that age and BMI were significant predictors of sleep apnea risk, while total body water was not independently significant.

Table 8. Binary Logistic Regression – Symptoms (Model A)

Symptom	Odds Ratio (OR)	95% CI	p-value
Hypertension	3.12	1.28–7.60	0.012
Anemia	2.46	1.08–5.62	0.031
Diabetes	1.22	0.58–2.55	0.602
Dyspnea	3.74	1.58–8.86	0.003

[Table 8] demonstrates that hypertension, anemia, and dyspnea were significant comorbidity-based predictors of high sleep apnea risk.

Table 9: Binary Logistic Regression – Symptoms (Model B)

Symptom	Odds Ratio (OR)	95% CI	p-value
Snoring	4.52	1.87–10.91	0.001
Tiredness	3.08	1.29–7.35	0.011
Observed apnea	2.76	1.21–6.32	0.016

[Table 9] depicts that classic STOP-BANG symptoms (snoring, tiredness, observed apnea) were independent predictors of sleep apnea risk.

Table Summary

[Table 1] describes the STOP-BANG questionnaire used for risk stratification of sleep apnea. It outlines the eight components (snoring, tiredness, observed apnea, high blood pressure, BMI, age, neck circumference, gender) and their yes/no responses. This establishes the standardized clinical tool that was applied to all 100 participants, with a score of ≥3 denoting high risk. The inclusion of anthropometric and symptom-based factors highlights its broad applicability in dialysis patients. [Table 2] shows the distribution of STOP-BANG scores across different age groups. The analysis revealed that prevalence of high risk increased steadily with age: 78.2% in the 18–40 years group, 98.2% in the 41–60 years group, and 100% in patients aged ≥61 years. This clearly demonstrates the strong influence of advancing age on sleep apnea risk, confirming that elderly dialysis patients constitute a particularly vulnerable group. [Table 3] demonstrates the interaction between gender and STOP-BANG scores. Among males, 98.1% were at high risk compared to 89.1% of females. Although both groups showed high prevalence, the results indicate that male sex confers a higher likelihood of being classified as high risk. This aligns with global evidence suggesting male gender as a strong epidemiological determinant of sleep apnea. [Table 4] presents the gender distribution of the entire cohort. Out of 100 patients, 68 were male and 32 were female, showing a male predominance (68%). This background distribution contextualizes the findings in Table 3, indicating that both the higher representation of males in the study population and the greater proportion of males at high risk contribute to the gender-based difference in prevalence. [Table 5] compares STOP-BANG scores

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with symptom prevalence. High-risk patients (≥3) reported substantially higher frequencies of snoring (78.7%), tiredness (75.5%), observed apnea (59.6%), and hypertension (78.7%) compared with low-risk patients. This table underscores the clinical relevance of STOP-BANG, showing that symptom burden is closely linked with higher scores. It also emphasizes the high occurrence of hypertension in this cohort, a finding consistent with known bidirectional relationships between sleep apnea and elevated blood pressure. [Table 6] provides a breakdown of positive responses to individual STOP-BANG components across the cohort. Snoring (75%), tiredness (73%), hypertension (75%), and age over 50 years (67%) were the most frequent positive findings. Fewer participants reported BMI >35 (11%) or neck circumference >40 cm (18%). This highlights that in this dialysis population, symptom-based and age-related criteria predominated over anthropometric cutoffs, reflecting the phenotype of Indian patients where severe obesity is less common but symptom burden and comorbidities are prominent. [Table 7] presents results from binary logistic regression evaluating predictors of high STOP-BANG risk. Age >50 years (OR 2.15, p=0.037) and BMI >25 (OR 2.64, p=0.014) emerged as statistically significant predictors. Male gender trended toward significance (OR 1.82, p=0.087), while total body water (OR 1.12, p=0.281) was not independently predictive. This indicates that demographic and anthropometric variables, rather than fluid status, are stronger independent determinants of sleep apnea risk in dialysis patients. [Table 8] demonstrates comorbidity-based predictors of high STOP-BANG scores. Hypertension (OR 3.12, p=0.012), anemia (OR 2.46, p=0.031), and dyspnea (OR 3.74, p=0.003) were significantly associated with high risk, whereas diabetes was not. These findings suggest that cardiovascular and hematological comorbidities exert a strong influence on sleep apnea susceptibility in dialysis patients, further emphasizing the multifactorial burden of disease in this population. Table 9 depicts symptom-based predictors of high STOP-BANG risk in multivariate analysis. Snoring (OR 4.52, p=0.001), tiredness (OR 3.08, p=0.011), and observed apnea (OR 2.76, p=0.016) were all independent predictors. This demonstrates that STOP-BANG symptoms themselves are not only components of the screening tool but also statistically robust predictors of risk, confirming the questionnaire's validity in this setting.

DISCUSSION

The present investigation revealed an exceptionally high prevalence of sleep apnea risk among chronic kidney disease patients undergoing maintenance dialysis, with 94% of participants demonstrating STOP-BANG scores ≥3, indicating intermediate to high risk for obstructive sleep apnea. These findings substantially align with contemporary literature, wherein Sim et al. (2009) documented 65% sleep apnea prevalence in dialysis populations, while Kuhlmann et al. (2000) reported 82% prevalence using polysomnographic confirmation.[14,15] The marginally elevated risk prevalence in our cohort may reflect a comprehensive assessment utilizing a validated screening instrument specifically designed for obstructive sleep apnea detection in high-risk populations.

Age-Related Prevalence: The present study demonstrated a pronounced age-dependent increase in STOP-BANG scores, with all participants aged 61 years and above exhibiting high-risk scores (≥3), compared to a more heterogeneous distribution in younger cohorts (18-40 years). These findings align with established epidemiological evidence demonstrating progressive escalation in sleep-disordered breathing prevalence correlating with advancing age and declining renal function.[16,17] Prospective investigations have documented that obstructive sleep apnea affects approximately 71% of non-dialysis chronic kidney disease patients, with severity independently associated with accelerated estimated glomerular filtration rate decline.[17] The age-related vulnerability observed in our cohort may reflect cumulative pathophysiological derangements, including progressive volume dysregulation, anatomical airway changes, and diminished compensatory mechanisms characteristic of advanced chronic kidney disease.

Gender Disparities: Male participants demonstrated higher risk prevalence (95.6%) compared to females (90.6%), concordant with gender-specific investigations revealing distinct phenotypic presentations, with female patients exhibiting greater associations between fluid overload parameters and symptom severity despite lower apnea-hypopnea indices.[18] This sexual dimorphism may reflect differential upper airway anatomy, hormonal influences on respiratory control, and varying fluid distribution patterns necessitating gender-specific diagnostic approaches in renal populations.

Cardiovascular Comorbidities: The substantial prevalence of hypertension (98%) among high-risk individuals corroborates evidence demonstrating that continuous positive airway pressure therapy produces significant reductions in plasma aldosterone concentrations and improves nocturnal blood pressure profiles in patients with concurrent obstructive sleep apnea and chronic kidney disease.[16] Investigations employing 24-hour ambulatory monitoring have established independent associations between sleep-related hypoxemia, left ventricular hypertrophy, and non-dipping blood pressure patterns, elucidating mechanistic pathways linking sleep-disordered breathing with cardiovascular remodelling.[19,20]

Hematological Parameters: Anemia prevalence (90%) among high-risk participants reflects preliminary evidence suggesting that erythropoietin-mediated anemia correction significantly improves sleep quality and reduces apnea-hypopnea indices

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through enhanced oxygen delivery capacity and potential effects on respiratory drive, highlighting interconnected comorbidity management strategies.[21]

Respiratory Manifestations: The high prevalence of shortness of breath (88%) in high-risk individuals provides justification for in-depth investigations that have documented sleep disorders in chronic kidney disease to be multifactorial, as opposed to distinct episodes of apnea, to include changes in sleep structure and respiratory instability. A more complete assessment of symptoms is essential in dialysis populations.[19,20]

Anemia and Sleep-Disordered Breathing: Anemia was highly prevalent (90%) in this cohort, with 94.4% of anemic individuals classified as high risk for obstructive sleep apnea (OSA). Evidence indicates a bidirectional relationship, where anemia severity worsens sleep-disordered breathing, and correction with erythropoietin therapy significantly reduces apnea-hypopnea index, improves oxygen saturation, and lowers arousal indices.[21] Cohort studies further identify anemia as a major contributor to excessive daytime sleepiness in hemodialysis patients, particularly when combined with OSA and systemic inflammation.[19,22] Interventional trials of nocturnal hemodialysis show additional benefits, improving sleep quality and reducing erythropoiesis-stimulating agent requirements by up to 40%, suggesting synergistic effects of enhanced oxygen delivery, reduced inflammation, and improved sleep regulation.[14,23] These findings highlight anemia not merely as a comorbidity but as a modifiable factor in OSA severity. Integrating anemia management into comprehensive care strategies may improve both respiratory and cardiovascular outcomes in dialysis-dependent populations.

Systolic and Diastolic Blood Pressure Relationships

The present study documented a hypertension prevalence of 98%, with 93.9% of hypertensive participants demonstrating high STOP-BANG scores, underscoring strong associations between blood pressure dysregulation and obstructive sleep apnea (OSA) in dialysis populations. Prior investigations confirm bidirectional links between sleep-disordered breathing and cardiovascular dysfunction in chronic kidney disease.[17] Interventional trials using ambulatory blood pressure monitoring show that continuous positive airway pressure therapy reduces plasma aldosterone by nearly 28%, improves nocturnal blood pressure, and restores physiological dipping patterns in previously non-dipping patients.[16] Polysomnographic studies further demonstrate that nocturnal hypoxemia independently predicts non-dipping hypertension and left ventricular hypertrophy, even after adjusting for traditional cardiovascular risk factors.[20,23] Mechanistically, intermittent hypoxemia activates sympathetic drive, oxidative stress, and inflammatory cascades, contributing to cardiovascular remodelling.[15,24,25] The striking hypertension burden observed here highlights the necessity of systematic sleep apnea screening in dialysis populations to enhance cardiovascular risk stratification and promote integrated therapeutic approaches.[26]

Limitations: Several limitations warrant acknowledgment in this study. The cross-sectional design precludes causal inference between total body water and sleep apnea risk. The STOP-BANG questionnaire, while validated for screening, does not substitute for definitive polysomnographic diagnosis, potentially leading to misclassification. Watson's formula for TBW estimation, though widely accepted, may be less accurate than direct measurement techniques such as bioelectrical impedance analysis. The single-centre setting limits generalizability to other populations and healthcare contexts. The sample size of 100 patients, while statistically adequate, may restrict subgroup analyses. Symptom assessment relied on subjective reporting rather than standardized severity scales, introducing potential recall bias.

Strength: This study possesses several notable strengths. It addresses a significant knowledge gap regarding sleep apnea prevalence in Indian dialysis patients, an underrepresented population in existing literature. The use of the validated STOP-BANG questionnaire ensures standardized and reproducible screening across participants. Watson's formula provides a practical, non-invasive method for TBW assessment applicable in resource-limited settings. The comprehensive evaluation of multiple clinical symptoms (resistant hypertension, anemia, uremic toxins, edema, dyspnea) enables holistic assessment of disease burden. The tertiary care setting ensures access to well-characterized patients with complete medical records. The adequate sample size provides sufficient statistical power for meaningful prevalence estimation and association analyses.

Summary: This cross-sectional descriptive study measured the prevalence of sleep apnea and its associations in a cohort of 100 chronic kidney disease patients on maintenance dialysis treatment in a tertiary care hospital in Tiruchirappalli, Tamil Nadu, over a 12-month period. The study systematically evaluated obstructive sleep apnea (OSA) risk stratification using the validated STOP BANG sleep apnea screening questionnaire while exploring associations with total body water (TBW) parameters derived via Watson's equation, and common clinical features like resistant hypertension, dyspnea, edema, oliguria, and uremic toxin retention. The findings of the study showed an extraordinarily high prevalence of high risk for OSA with 94% of the sample receiving STOP-BANG scores ≥3 indicative of intermediate to high probability of having sleep apnea. The demographic analysis demonstrated a significant dose-dependent relationship between age and OSA risk, with 100% of individuals classified as high risk (≥3) aging above 61 years compared to the heterogeneous distribution estimates for lower scores among younger individuals in the preclinical period. The male participants had slightly higher prevalence

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rates (95.6%) than female participants (90.6%), consistent with gender differences documented for phenotypic patterns of respiratory-disordered sleep conditions. Comprehensive evaluation of symptom burden showed a high degree of comorbidity for patients at high risk for OSA with resistant hypertension (98%), anemia (90%), dyspnea (88%), uremic symptoms (100%), and peripheral edema (35%). The findings of this study demonstrate the multifactorial pathophysiological state characterizing sleep apnea risk.

CONCLUSION

This study demonstrates that sleep apnea is a very common comorbidity in dialysis-dependent chronic kidney disease patients, with 94% of patients scoring in the intermediate to high risk range on validated screening measures. The steep increase in high-risk classification with increasing age, with all 61 years old and older patients classified as high risk, highlights the urgent need to establish a routine screening process for sleep disorders in nephrology clinics. The substantial burden of cardiovascular complications, hematological issues, and respiratory symptoms among patients at risk support our multi-faceted clinical management approach. Matrix, despite the complex relationships between total body water parameters and sleep apnea risk that require further exploratory studies to elucidate mechanistic relations, findings support the paradigm shift toward sleep disorder assessment and treatment strategies in evidence-based practice approaches. Incorporation of continuous positive airway pressure (CPAP) therapy, as well as optimization of dialysis prescriptions, and aggressive volume management will represent key components of a comprehensive strategy to ensure better care with the purpose of reducing cardiovascular outcomes and improving quality of life in a vulnerable population.

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