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Original Research Article

Assessment of the Prevalence of Sleep Apnea in Patients with Chronic Kidney Disease

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Abstract: Sleep apnea is a common but underdiagnosed comorbidity in patients with chronic kidney disease (CKD), and its severity with declining renal function. To determine the prevalence of sleep apnea in patients with CKD using the Apnea-Hypopnea Index (AHI) on polysomnography, and to examine its association with CKD stage and estimated glomerular filtration rate (eGFR). An observational, cross-sectional study was conducted over six months in the renal department of a tertiary care hospital and enrolled 100 patients with CKD (stages 1–5, age ≥18 years, both sexes). Sleep apnea risk was screened using the stop-bang questionnaire; patients classified as moderate or high risk underwent polysomnography for AHI-based diagnosis and severity grading. Associations between CKD stage and AHI were analysed with one-way ANOVA, and the eGFR–AHI relationship was assessed using Pearson correlation. Of 100 patients, STOP-BANG classified 60% as high risk and 11% as moderate risk. Among the 71 patients who underwent polysomnography, AHI-confirmed sleep apnea was severe in 42%, moderate in 19%, and mild in 10%. Mean AHI rose progressively with CKD stage, from 20.67 in stage 2 to 48.90 in stage 5 (one-way ANOVA, $p < 0.0000$). eGFR correlated strongly and negatively with AHI ($r = -0.7891$, $p < 0.001$). Only male gender was significantly associated with sleep apnea among candidate risk factors ($p = 0.026$). Sleep apnea is highly prevalent among patients with CKD and becomes progressively more severe as renal function declines. STOP-BANG-based screening followed by confirmatory polysomnography identifies a substantial burden of unrecognised sleep apnea and supports routine screening, particularly in advanced CKD.

Keywords: Sleep Apnea, Obstructive; Renal Insufficiency, Chronic; Polysomnography; Glomerular Filtration Rate.

1. Introduction

Chronic kidney disease (CKD) is a progressive condition affecting renal excretory and regulatory function and is frequently accompanied by sleep-disordered breathing Sleep apnea, most commonly obstructive, involves recurrent partial or complete upper-airway obstruction during sleep, causing intermittent hypoxia, sleep fragmentation, and sympathetic activation. Central and mixed forms also occur, particularly with cardiac or neurological comorbidity.

Evidence links sleep apnea to CKD through bidirectional mechanisms. Fluid overload and rostral fluid shift in advanced CKD promote pharyngeal oedema and airway narrowing; uraemic toxin accumulation, metabolic acidosis, and disturbed potassium/carbon-dioxide handling can blunt central respiratory drive; and autonomic and cardiovascular derangement further predispose to unstable breathing. Conversely, intermittent hypoxia, oxidative stress, and sympathetic surges from untreated sleep apnea may accelerate glomerular injury, creating a self-reinforcing cycle.

Reported prevalence of sleep apnea in CKD ranges roughly 30–70%, rising further in end-stage disease. Sleep evaluation is nonetheless not routine in nephrology, partly because polysomnography is resource-intensive. Validated tools such as STOP-BANG offer practical risk stratification, reserving polysomnography for moderate/high-risk patients.

Several interlocking mechanisms have been proposed to explain the high burden of sleep apnea in CKD. Fluid overload, especially in advanced disease, produces oedema of upper-airway soft tissue that narrows the pharyngeal lumen.Obesity and abnormal fat distribution around the neck and abdomen add mechanical load on the airway and increase pharyngeal collapsibility. Neurohormonal disturbance in uraemia, together with hyperkalaemia and hypercapnia, can blunt brainstem respiratory responsiveness and predispose to central apnoeic events, while CKD-associated hypertension and autonomic dysfunction further destabilize breathing control during sleep. In patients on haemodialysis, intradialytic fluid shifts and associated hypoxaemia may compound these effects.

Symptomatic overlap (fatigue, poor concentration, disturbed sleep) further obscures recognition, while both conditions independently worsen cardiovascular outcomes and quality of life. As sleep apnea may be a modifiable contributor to CKD progression, systematic assessment of its prevalence and relation to renal function is clinically relevant.

2. Methods

Observational, cross-sectional study (described as prospective observational in the original abstract) conducted in the renal department of a tertiary care hospital over six months.

Sample size

Estimated using $n = Z^2 \cdot p \cdot (1-p)/d^2$ ($Z = 1.96$, $p = 0.4$, $d = 0.1$) → ≈93; with 10% dropout buffer, final target n = 100.

Eligibility

Inclusion: Chronic kidney stages 1–5, age ≥18 years, both sexes.

Exclusion: primary sleep disorders other than sleep apnea; dialysis patients ,pregnant women; significant cognitive impairment.

Ethics

Informed consent obtained from each participant; confidentiality maintained.

Tools

Questinnaire STOP-BANG (8 items) scored 0–2 low, 3–4 moderate, 5–8 high risk; moderate/high-risk patients underwent polysomnography (PSG) for AHI (gold standard): <5 normal, 5–15 mild, 15–30 moderate, >30 severe.

Process

Patients were enrolled with consent; baseline data, STOP-BANG, and (where indicated) PSG/AHI, CKD stage and eGFR were recorded. Data were analysed in Excel with descriptive statistics, one-way ANOVA (CKD stage vs AHI), and Pearson correlation (eGFR vs AHI); $p<0.05$ considered significant.

Participant Flow

Figure 1: summarises participant flow through screening, risk stratification, PSG confirmation, and analysis Exact numbers excluded at initial eligibility screening were not reported in the original study; the diagram begins at consent/enrolment (N=100).

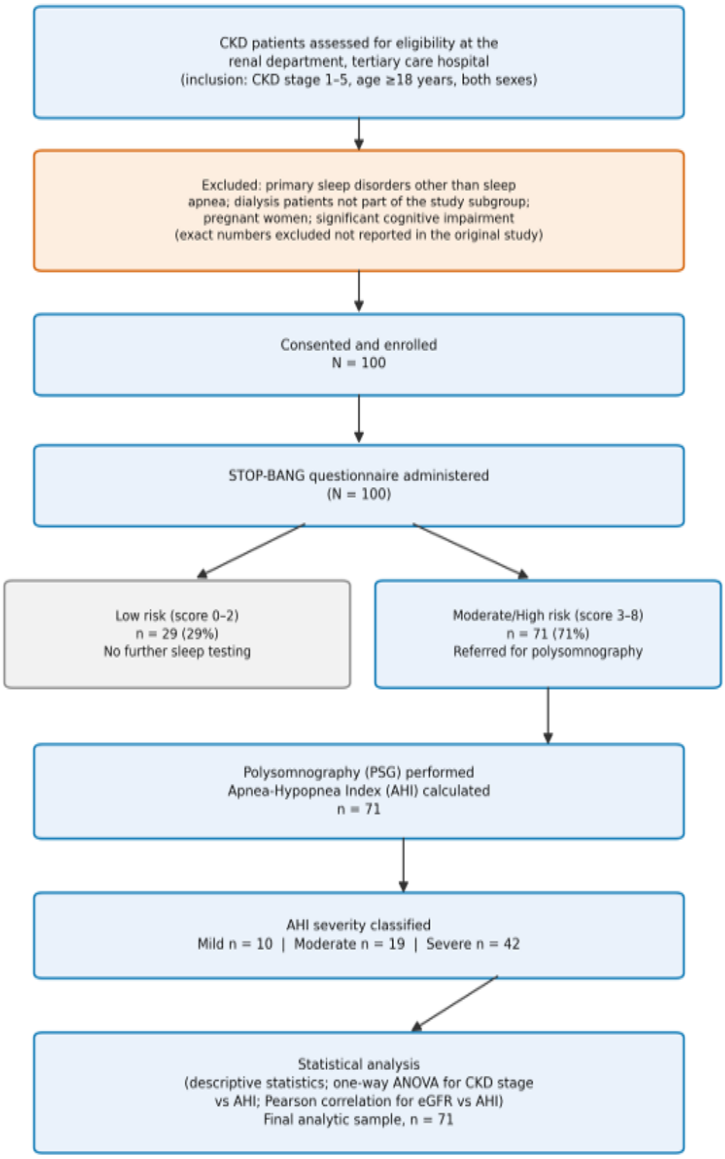


Figure 1. STROBE-style participant flow diagram. PSG, polysomnography; AHI, Apnea Hypopnea Index.

Table 1. Baseline Demographic and Clinical Characteristics (N = 100)

Variable	Category	n (%)
Gender	Male	57 (57)
	Female	43 (43)
Age (yrs)	21–30 / 31–40 / 41–50	1 / 11 / 22
	51–60 / 61–70 / >70	24 / 30 / 12
Mean age	56 years (as reported)	—
Residence	Urban / Rural	60 (60) / 40 (40)
Employment	Employed/Retired/Unemployed	52 / 22 / 26
BMI	Normal / Overweight / Obese	21 / 32 / 47
Habits	Smoking / Alcohol use	46 (46) / 65 (65)
Comorbid.	HTN / DM / Heart dis. / GERD	76 / 58 / 60 / 52
	COPD / Liver problems	45 (45) / 38 (38)
Family Hx	CKD / Sleep apnea	57 (57) / 51 (51)
CKD stage	I / II / III / IV / V	20 / 19 / 23 / 16 / 22

Duration of CKD ranged 0.5–10 years, with 2-year and 3-year duration each reported in 13% of patients (fullest distribution in original Table 14, not reproduced here for brevity). HTN, hypertension; DM, diabetes mellitus; GERD, gastro-oesophageal reflux disease; COPD, chronic obstructive pulmonary disease.

Table 2. STOP-BANG Risk Categories and AHI-Based Severity

Category	n	%
STOP-BANG low (0–2)	29	29 of N=100
STOP-BANG moderate (3–4)	11	11 of N=100
STOP-BANG high (5–8)	60	60 of N=100
Underwent polysomnography	71	71 of N=100
AHI – mild	10	10 (as reported)
AHI – moderate	19	19 (as reported)
AHI – severe	42	42 (as reported)

The original study reports AHI severity percentages alongside a stated 71% overall positivity within the PSG cohort; denominators were not reconciled in the source and are reproduced exactly as reported. AHI, Apnea-Hypopnea Index.

Table 3. CKD Stage, eGFR and Sleep Apnea Severity

CKD stage	Mean AHI	SD	ANOVA p
Stage 1	Not reported	Not reported	-
Stage 2	20.67	12.3	-
Stage 3	27.05	7.25	<0.0000
Stage 4	41.43	3.77	-
Stage 5	48.9	2.34	-

Correlation: eGFR vs AHI — Pearson r = −0.7891, p < 0.001 (strong negative correlation).

ANOVA (as reported): Between groups SS=10240.4666, df=3; Within SS=4207.7761, df=76; Total SS=14448.2427, df=79; F=61.6537, p=0.0000. AHI, Apnea-Hypopnea Index; eGFR, estimated glomerular filtration rate; SD, standard deviation.

3. Figures

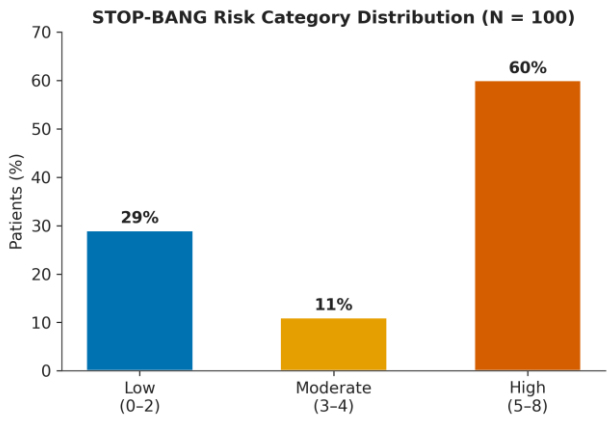


Figure 1. STOP-BANG risk distribution (N=100).

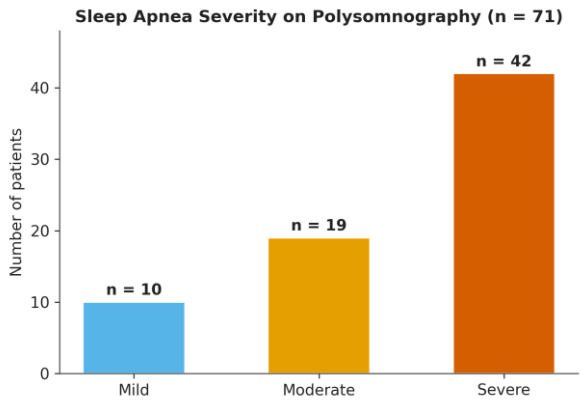


Figure 2. AHI-based sleep apnea severity (n=71).

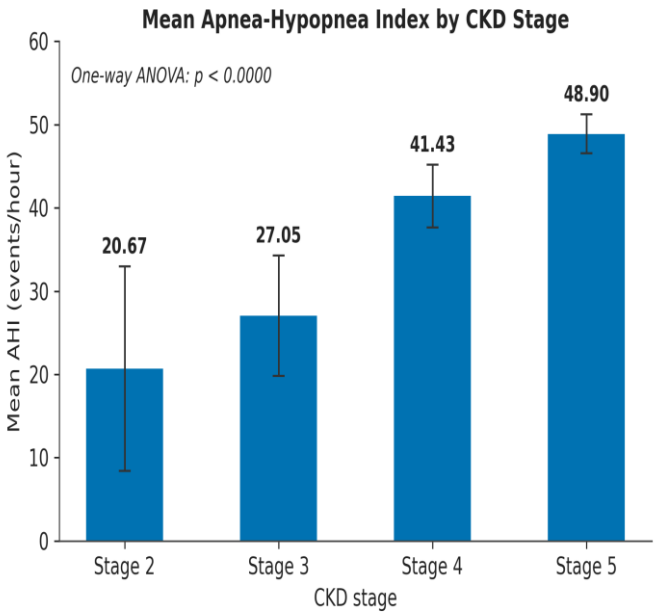


Figure 3. Mean AHI by CKD stage (ANOVA p<0.0000). Stage 1 not reported.

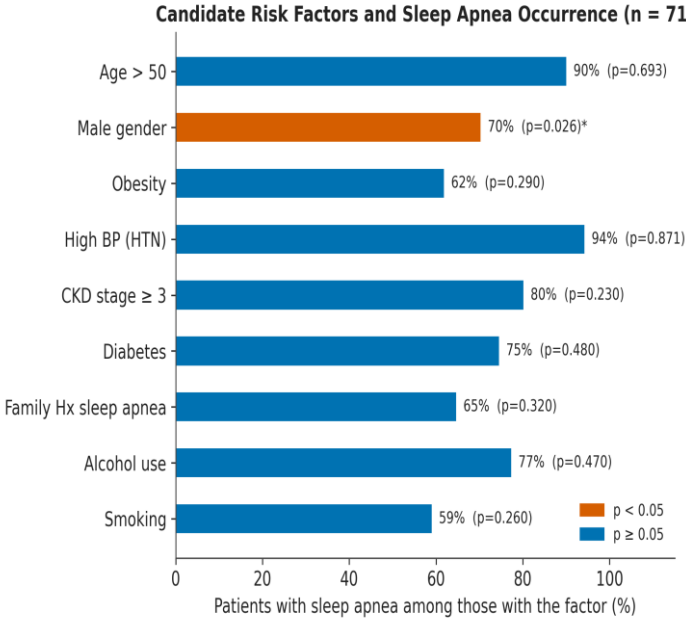


Figure 5. Risk factors and sleep apnea occurrence (n=71); OR/CI not reported, so proportions with p-values are shown, not aforest plot.

Figure 4 (eGFR vs AHI scatter with regression line and confidence band): individual patient-level eGFR/AHI pairs and the 95% confidence band were not reported in the original study, so the scatter plot is not reproduced; the summary correlation (r=−0.7891, p<0.001) .

This study explored the possibility of using machine learning techniques to identify mental health risk based on Instagram usage behaviour. The primary objective of this study was to determine whether behavioural usage patterns on Instagram could be used to identify potential mental health risks through machine learning techniques. Five behavioural features were used as input: age, daily Instagram usage hours, posting frequency, average likes per post, and follower count. Since the dataset had no pre-existing mental health labels, K-Means clustering was applied first to group users into Low Risk and High-Risk categories. Four classification models were then trained and tested on these labels.

4. Results and Conclusion

100 patients with CKD enrolled, 57% were male and 43% female (Table 1), consistent with prior hospital-based series.[16] Most patients were older than 50 years, with the largest band 61–70 years (30%; mean age 56 years); 60% resided in urban areas — consistent with age-related decline in upper-airway muscle tone and possibly reflecting referral patterns at tertiary urban centres.

BMI was elevated in most patients (47% obese, 32% overweight; only 21% normal). As pharyngeal/neck adiposity mechanically promotes airway collapse, this distribution indicates elevated background OSA risk independent of renal status. Smoking (46%) and alcohol use (65%) were common lifestyle factors that can further promote airway inflammation or pharyngeal relaxation.

Comorbidity burden was high (hypertension 76%, diabetes 58%, heart disease 60%, GERD 52%, COPD 45%, liver problems 38%), mirroring the multimorbid nature of CKD and its shared pathways of fluid retention and sympathetic activation with sleep-disordered breathing.[19,21] Family history of CKD (57%) and sleep apnea (51%) raise the possibility of familial predisposition, though causality cannot be inferred from this cross-sectional design.

STOP-BANG classified 60% of the cohort as high risk and 11% as moderate risk (Table 2, Fig.1); only 29% were low risk. The most frequent positive components were high blood pressure (76%), age>50 (69%), and snoring (63%), followed by

neck circumference>40cm (60%), male gender (57%), tiredness (62%), observed apnea (48%), and BMI>35 (47%) — supporting STOP-BANG as a practical first-line nephrology screening tool.

Among the 71 patients who underwent polysomnography, AHI-based severity was severe in 42%, moderate in 19%, and mild in 10% (Table 2) the source study describes an overall 71% positivity in this subgroup. This predominance of moderate-to-severe disease among referred patients echoes systematic reviews reporting sleep apnea across all CKD stages, exceeding 50% even in end-stage disease.[6] Symptom-based recognition alone is therefore likely to miss substantial disease, reinforcing systematic screening.

Severity differed markedly by gender: 33 of 50 tested males (66%) had severe disease versus 9 of 21 tested females (43%). Consistently, male gender was the only significant risk factor for sleep apnea ($p=0.026$; Table 3) aligning with the well-established male predominance in OSA attributed to airway anatomy, fat distribution, and hormonal effects on pharyngeal dilator function.[16]

Mean AHI rose progressively with CKD stage — 20.67 (stage 2), 27.05 (stage 3), 41.43 (stage 4), 48.90 (stage 5) — by one-way ANOVA ($p<0.0000$; Table 3, Fig.3), consistent with prior work linking advancing CKD to more severe AHI via fluid overload, uraemic respiratory-drive derangement, and autonomic dysfunction.[16,18] This is not universal, however: at least one prior study found no significant CKD-stage/OSA-severity relationship, indicating population- and sample-size-dependent variability.[9]

eGFR correlated strongly and negatively with AHI ($r=-0.7891$, $p<0.001$; Table 3), stronger than some previously reported cohorts, supporting a close mechanistic link between declining renal function and worsening sleep-disordered breathing. Given the cross-sectional design, causal direction cannot be established; renal impairment worsening sleep apnea and sleep apnea accelerating renal decline are both compatible and plausibly mutually reinforcing.

Male gender, none of age>50, obesity, hypertension, CKD stage≥3, diabetes, family history, alcohol use, or smoking reached significance (Table 3-linked risk-factor data, Fig.5), which the original authors attributed to limited power (n=71).

Collectively, a high STOP-BANG-positive rate, a high burden of moderate-to-severe disease on confirmatory testing, and a clear AHI gradient with declining renal function support incorporating validated screening into routine nephrology assessment, prioritising PSG referral for advanced CKD stage, low eGFR, or high STOP-BANG score.

These findings should be interpreted within the constraints of a single-centre, cross-sectional design with a moderate sample size, discussed further under Limitations, the consistency of the STOP-BANG risk gradient, the AHI severity distribution on confirmatory PSG, the CKD-stage/AHI relationship.

Conclusion

Sleep apnea was highly prevalent among patients with CKD, with most patients screened as moderate/high risk by STOP-BANG and most of those undergoing polysomnography confirmed to have moderate-to-severe disease. AHI increased significantly with advancing CKD stage and correlated strongly and negatively with eGFR, indicating that sleep apnea burden intensifies as renal function deteriorates; male gender was the only significantly associated risk factor. These findings support STOP-BANG as a practical first-line screening tool in nephrology practice, with confirmatory polysomnography prioritised for moderate-to-high-risk patients, particularly those with advanced CKD. Early recognition and management may help mitigate cardiovascular risk and support renal and quality-of-life outcomes; larger, adequately powered studies are needed to clarify the independent contribution of established risk factors in this population.

Strengths

- Validated screening (STOP-BANG) combined with confirmatory gold-standard polysomnography rather than screening alone.
- A structured, purpose-built data collection form capturing a comprehensive set of demographic, clinical, and lifestyle variables.
- Formal, pre-specified sample size estimation using a standard prevalence-based formula.

5. Conflict of Interest: The authors declare no conflict of interest.

6. References

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