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Study of Renal Impairment in Patients with Obstructive Sleep Apnea Syndrome and Its Relation to the Disease Severity

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Abstract:

Background: Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder associated with systemic complications, including renal impairment resulting from intermittent hypoxia and sympathetic activity. This study aimed to evaluate renal dysfunction in cases with OSA, investigate its correlation with disease severity, and assess the impact of continuous positive airway pressure (CPAP) therapy on kidney function. **Methods:** In this prospective cohort study, 100 participants (80 OSA cases and 20 healthy controls) were enrolled from Benha University Hospital (December 2022 and December 2024). All participants underwent comprehensive clinical evaluation, overnight polysomnography (PSG), renal function testing [serum creatinine, blood urea, estimated glomerular filtration rate (eGFR), urinary albumin-to-creatinine ratio (UACR)], and renal ultrasonography. OSA cases with moderate-to-severe disease received CPAP therapy for three months, followed by repeat renal evaluation. **Results:** The apnea–hypopnea index correlated positively with creatinine (r = 0.483), urea (r = 0.339), and UACR (r = 0.615), and negatively with eGFR (r = –0.387) (P < 0.001). After 3 months of CPAP, mean creatinine decreased from 2.01 ± 0.89 to 1.45 ± 0.64 mg/dL, and eGFR increased from 40 ± 22.88 to 56.51 ± 32.53 mL/min/1.73 m² (P < 0.001). Compared with controls, OSA patients had greater neck circumference, higher STOP-BANG and Epworth Sleepiness Scale scores, higher AHI and oxygen desaturation index (ODI), lower mean oxygen saturation, and worse renal indices. **Conclusion:** OSA severity is strongly associated with renal dysfunction. CPAP therapy leads to significant improvement in kidney function, highlighting its potential renoprotective role in OSA management.

Keywords: Obstructive Sleep Apnea, Chronic Kidney Disease, Apnea–Hypopnea Index, CPAP Therapy, Renal Function.

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Introduction

Obstructive sleep apnea (OSA) stands as the most common presentation within the spectrum of sleep-disordered breathing (SDB), typified by recurrent episodes of upper airway occlusion during sleep. These obstructive events precipitate cycles of intermittent oxygen desaturation, fragmented sleep architecture, and dynamic alterations in intrathoracic pressure [1, 2]. From a clinical perspective, OSA often manifests through persistent loud snoring, unrefreshing sleep with excessive daytime somnolence, chronic fatigue, and, in some cases, coexisting insomnia. Its likelihood increases notably in males, individuals with obesity, shortened cervical length, or anatomical variations narrowing the upper airway [3].

OSA is now recognized not merely as a disturbance of nocturnal ventilation but as a multisystem disorder with substantial cardiovascular, metabolic, and neurocognitive repercussions. The repetitive hypoxic–hypercapnic burden incurred during sleep provokes sustained stimulation of the sympathetic nervous system, augments oxidative stress, disrupts endothelial homeostasis, and fosters chronic low-grade inflammation[4]. These mechanisms contribute to a markedly higher incidence of hypertension, coronary artery disease (CAD), diabetes mellitus (DM), cerebrovascular insults, and sudden nocturnal cardiac mortality[5].

Globally, population-based data indicate that OSA affects around 4–7.5% of men and around 2% of women, with region-specific adult prevalence rates often reported between 3% and 5% [6]. As per the American Academy of Sleep Medicine (AASM), overnight polysomnography (PSG) remains the definitive diagnostic modality. The apnea–hypopnea index (AHI), representing the hourly frequency of obstructive or hypopneic events, serves as the principal grading tool: mild (<15), moderate (15–29), and severe (≥30) events per hour [7].

Chronic kidney disease (CKD), defined by either persistent structural or functional renal impairment or a sustained drop in glomerular filtration rate (GFR) for over three months, constitutes a growing global health crisis [8]. In 2016, it ranked as the 11th leading cause of mortality worldwide [9]. The disease often evolves insidiously, escaping early detection, particularly in those without conventional risk factors but with possible contributors like sleep-related respiratory disturbances.

An expanding body of literature exhibits that OSA has a direct role in renal injury. Due to their high metabolic demand and constant requirement for adequate oxygen delivery, the kidneys are particularly vulnerable to intermittent hypoxic stress. In OSA, mechanisms including sustained hypoxemia, oxidative damage, and sympathetic hyperstimulation can induce glomerular hyperfiltration, compromise endothelial integrity, and promote albuminuria. These alterations may hasten the decline in renal function even in cases lacking the more traditional risk profiles for CKD, like hypertension or DM[10].

Therapeutically, continuous positive airway pressure (CPAP) remains the cornerstone of management for moderate-to-severe OSA, with robust evidence supporting cardiovascular benefit. However, its specific impact on renal outcomes, especially in individuals without established comorbidities, has yet to be fully elucidated [11].

On this basis, the current investigation sought to examine renal function among cases with OSA, analyze the correlation between renal impairment and OSA severity, and determine whether CPAP use confers measurable renoprotective effects.

Patients and methods:

Patients

This prospective cohort study was implemented over a continuous two-year

period, from December 2022 to December 2024, within the Sleep Study Unit of the Chest Department at Benha University Hospital.

All research activities complied with the ethical principles delineated in the Declaration of Helsinki and adhered to Good Clinical Practice (GCP) guidelines. Before participation, all individuals received detailed oral and written explanations about the study's aims, methodology, potential risks, and benefits. Only those who provided voluntary written informed consent were enrolled. To protect participant privacy, a unique, anonymized alphanumeric identifier was assigned to each subject, ensuring that all collected data remained confidential and traceable only by the primary investigators.

The study protocol underwent rigorous ethical scrutiny and obtained formal approval from the Research Ethics Committee, Faculty of Medicine, Benha University (Approval No. MD-9-11-2022).

Sample Size Estimation

Sample size estimation was done via the STATCALC module of the Epi Info software, drawing on statistical parameters derived from the work of Chung and co-authors [(12)]. The computation assumed a two-sided 95% confidence titer, a statistical power of 80%, and a margin of error set at 5%. An odds ratio of 1.115 was used in the calculation. Based on these parameters, the statistical model determined that a minimum of 100 participants was necessary to achieve sufficient study power

Eligibility Criteria

Participants were included if they were adults aged between 30 and 70 years, had a confirmed diagnosis of OSA by polysomnography, and could provide informed consent. Exclusion criteria were designed to eliminate potential confounding conditions and included psychiatric illness, active malignancy, autoimmune disease, chronic pulmonary disorders like COPD or interstitial lung disease, hematologic abnormalities, thyroid dysfunction, use of nephrotoxic drugs, and the presence of treated diabetes mellitus or hypertension.

Methods:

Clinical and Sleep Assessments

All participants underwent a structured clinical assessment encompassing a comprehensive medical history and physical examination. The history covered prior illnesses, surgical interventions, current medications, lifestyle factors including smoking and alcohol consumption, and sleep-related complaints like snoring, witnessed apneas, and unrefreshing sleep. Special attention was paid to symptoms suggestive of renal involvement, including nocturia, flank pain, and peripheral or periorbital edema. OSA risk was screened via the STOP-BANG questionnaire[(13)], while the degree of daytime somnolence and functional impairment was quantified via the Epworth Sleepiness Scale (ESS) [(14)]. Anthropometric data were collected for body weight, height, and neck circumference, with body mass index (BMI) estimated and categorized according to World Health Organization (WHO) guidelines [(15)].

Physical Examination

Vital signs were recorded at rest, including blood pressure, heart rate, respiratory rate, body temperature, and peripheral oxygen saturation (SpO_2). Local examination focused on identifying structural abnormalities of the upper airway, like enlarged tonsils, nasal septal deviation, or retrognathia, which could contribute to OSA pathophysiology. Signs suggestive of renal dysfunction were also sought, including loin tenderness, percussion dullness, periorbital swelling, peripheral edema, and dermatological changes consistent with uremia.

Laboratory Investigations

Blood and urine samples were collected from all participants following standard

aseptic procedures. Renal function was assessed via serum creatinine, blood urea nitrogen (BUN), and urinary albumin-to-creatinine ratio (UACR). Fasting blood glucose was measured to exclude undiagnosed diabetes mellitus, and a complete blood count (CBC) was obtained to identify hematologic disorders. The estimated glomerular filtration rate (eGFR) was estimated via the Modification of Diet in Renal Disease (MDRD) equation [16].

$$GFR = 186 \times (Scr)^{-1.154} \times (Age)^{-0.203} \times (0.742 \text{ if female}) \times (1.212 \text{ if African American})$$

CKD staging was determined in strict accordance with the **Kidney Disease: Improving Global Outcomes (KDIGO)**[17], which offers a unified, internationally recognized system for grading renal impairment. Under this schema, Stage 1 denotes an eGFR of ≥90 mL/min/ 1.73 m² in the presence of structural or functional evidence of kidney injury, like microalbuminuria or abnormal imaging findings. Stage 2 reflects a mildly reduced eGFR of 60–89 mL/min/1.73 m², again requiring corroborative evidence of renal damage. Stages 3a and 3b represent moderate functional decline, with eGFR ranges of 45–59 mL/min/1.73 m² and 30–44 mL/min/ 1.73 m², respectively. Stage 4 encompasses severe drop in renal filtration capacity (15–29 mL/min/1.73 m²), while Stage 5 marks end-stage renal disease (ESRD), characterized by eGFR values below 15 mL/min/1.73 m², often necessitating dialysis or kidney transplantation. Applying the KDIGO classification ensured that renal status was assessed via a robust, clinically relevant framework, allowing for precise stratification and direct comparability with another CKD-related research in the context of OSA.

Radiological Investigations

Renal ultrasonography was done by an experienced radiologist blinded to the participants' group allocation. Assessments included kidney size, cortical thickness, cortical echogenicity relative to the liver and spleen, and the integrity of corticomedullary differentiation (CMD). Parenchymal changes were graded from Grade 0 (normal CMD) to Grade IV (severe pathological change with complete CMD loss) [16].

Polysomnographic Evaluation

Overnight attended polysomnography was done for all participants via SOMNOscreen™ plus (SOMNOmedics AG, Germany). Parameters recorded included electroencephalography (EEG) to determine sleep stages, electrooculography (EOG) to detect rapid eye movement (REM) phases, electromyography (EMG) for muscle tone and limb movement assessment, electrocardiography (ECG) for cardiac rhythm monitoring, nasal and oral airflow measurement via pressure transducers, thoracoabdominal respiratory effort monitoring via inductance belts, peripheral oxygen saturation via pulse oximetry, and body position recording [18]. Participants were instructed to avoid caffeine intake and remove nail polish prior to the study, in line with American Academy of Sleep Medicine (AASM) recommendation [7, 19]. OSA severity was categorized according to the apnea–hypopnea index (AHI) as mild (5–14 events per hour), moderate (15–29 events per hour), or severe (≥30 events per hour). The respiratory disturbance index (RDI) and oxygen desaturation index (ODI) were estimated, with ODI defined as the number of ≥3–4% oxygen desaturation events per hour[20]. Minimum and baseline SpO_2 values were documented to assess the severity of nocturnal hypoxia [21].

CPAP Intervention and Follow-up

Cases in Group A with moderate-to-severe OSA commenced on CPAP therapy. Optimal therapeutic pressures were determined during titration studies to eliminate apneas and hypopneas while minimizing residual respiratory events. Participants were advised to use CPAP

nightly for three consecutive months, and adherence was monitored via device-generated compliance data. At the end of the follow-up period, renal function markers, including serum creatinine, BUN, eGFR, and UACR, were reassessed, and renal ultrasonography was repeated. This longitudinal evaluation allowed for the determination of whether effective OSA management with CPAP had a stabilizing or restorative effect on renal function and morphology.

Statistical analysis

Data analysis utilized IBM SPSS Statistics, Version 26.0 (IBM Corp., Armonk, NY, USA). Distribution normality was assessed via the Kolmogorov–Smirnov test. Quantitative variables were expressed as mean ± SD and compared via the student's t-test; categorical variables were presented as frequencies per percentage and compared via the Chi-square test. Correlations between continuous variables were examined with Pearson's correlation coefficient. Logistic regression analyses (univariate and multivariate, backward Wald method) identified independent predictors of renal impairment. A p-value < 0.05 denoted statistical significance.

Results:

Analysis revealed marked differences between the OSA cohort and the control group across both anthropometric and sleep-related variables. Cases with OSA demonstrated greater neck circumference (mean 45.34 cm) compared with controls (39.6 cm) and scored significantly higher on STOP-BANG (5.93 vs. 3.95) and ESS (18.49 vs. 4.6). Their AHI was markedly elevated (35.10 vs. 3.59 events per hour), accompanied by a higher ODI (37.32 vs. 4.63) and RDI (35.16 vs. 3.57; all $P \leq 0.001$). Baseline SpO_2 was lower in OSA cases (93.31% vs. 95.55%), and their minimum SpO_2 was substantially reduced (77.54% vs. 91.05%, $P \leq 0.05$). Renal function measures also differed substantially between the two groups. The OSA cohort showed higher creatinine titers (2.01 mg/dL vs. 0.86 mg/dL), higher urea (102.88 mg/dL vs. 35.6 mg/dL), and elevated UACR (432.85 vs. 29.35), while eGFR was considerably lower (40 vs. 88.2 mL/min/ 1.73 m^2; all $P \leq 0.001$) (**Table 1**).

When stratified by OSA severity, sex distribution varied significantly; females predominated in the mild group, whereas males represented the majority in moderate and severe categories ($P \leq 0.05$). Severe OSA was associated with the largest neck circumference (48.44 cm), highest STOP-BANG (6.70) and ESS (20.77) scores, and the greatest AHI (50.27), ODI (54.3), snoring index (369.62), and RDI (50.32; all $P \leq 0.001$). Conversely, cases with mild disease had the highest minimum SpO_2 (85.31%, $P \leq 0.001$). Notably, 100% of the severe group fell into the category of severe excessive daytime sleepiness according to ESS classification (**Table 2**).

Renal profile analysis across severity groups showed a progressive increase in creatinine, urea, and UACR with disease severity, peaking in the severe group (2.4 mg/ dL, 131.16 mg/dL, and 632.45 mg/mL, respectively; all $P \leq 0.001$). The lowest eGFR was recorded in the severe group (33.7 mL/min/1.73 m^2, $P \leq 0.05$). CKD staging also differed: stage 5 was observed in 9.1% of the severe group, with no cases in other severity categories ($P \leq 0.05$). Renal US revealed Grade IV parenchymal changes in 18.2% of severe cases and 8.7% of moderate cases, while no such changes were detected in the mild or control groups ($P \leq 0.001$) (**Table 3**).

Correlation testing within the OSA group demonstrated that AHI correlated positively with neck circumference, STOP-BANG, ESS, ODI, snoring index, creatinine, urea, and UACR, while showing a negative correlation with eGFR ($P \leq 0.05$ for all). Negative associations were also noted between AHI and both sex (male as reference) and minimum SpO_2 ($P \leq 0.05$) (**Table 4**, **Figure 1).** AHI

and eGFR showed a significant inverse relationship (r = –0.387, *P* = 0.0001) **(Figure 2).**

After three months of CPAP therapy in moderate-to-severe cases, renal parameters improved significantly: creatinine decreased from a mean of 2.01 to 1.45 mg/ dL, while eGFR rose from 40 to 56.51 mL/min/ 1.73 m² (both *P* ≤ 0.001) (**Table 5**).

Regression modeling identified several factors associated with greater post-CPAP improvement in eGFR. In univariate analysis, younger age, smaller neck circumference, lower STOP-BANG and ESS scores, lower AHI and ODI, higher baseline and minimum SpO_2, lower RDI, and reduced creatinine and urea were all significant predictors (*P* ≤ 0.05). Multivariate analysis confirmed younger age and lower baseline creatinine as independent predictors of greater eGFR recovery following CPAP (*P* ≤ 0.001) (**Table 6**).

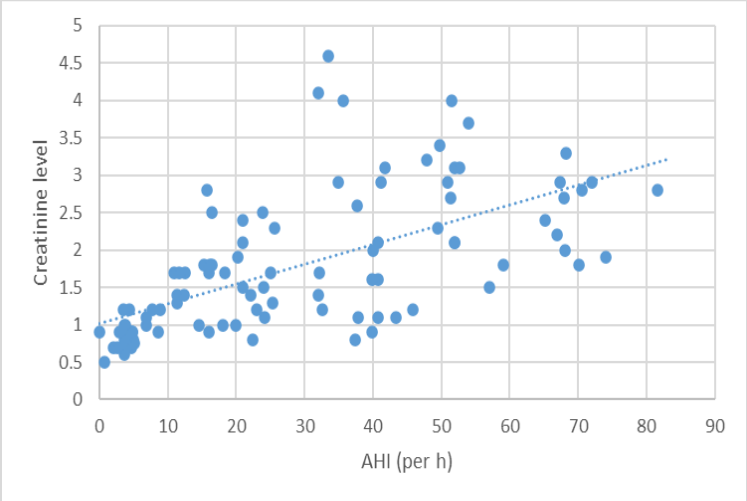

Figure 1: Scatter diagram showing positive correlation between AHI and creatinine titer among OSAS patients**.**

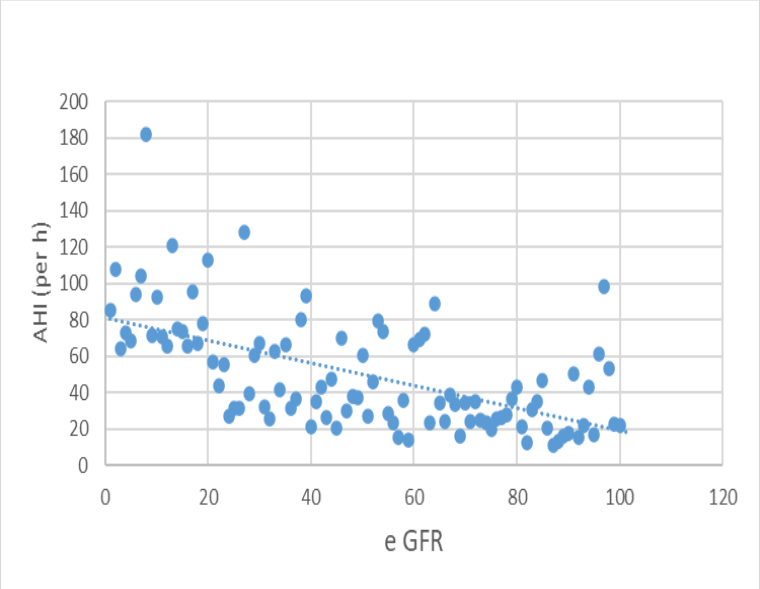

Figure 2: Scatter diagram showing negative correlation between e GFR and AHI.

Table 1: Comparison between control and OSAS groups regarding anthropometric, clinical, polysomnographic, and renal data.

Characteristics		Control (n=20)	OSAS (n=80)	Test of significance	P value
Age (year)		50.75 ± 10.77	54.40 ± 10.61	t= 0.252	.095
Sex	Male	9 (45%)	48 (60%)	X^2 = 1.469	0.226
	Female	11 (55%)	32 (40%)		
BMI (Kg/ m^2)		36.05 ± 9.15	40.59 ± 9.34	t= 1.954	0.054
Neck Circumference (cm)		39.60 ± 6.13	45.34 ± 5.53	t= 4.060	**.000 ****
STOP BANG		3.95 ± 0.69	5.93 ± 1.22	t= 6.953	**.000 ****
STOP BANG classification	Intermediate risk	16 (80%)	11 (13.8%)	X^2 = 35.629	**.000****
	High risk	4 (20%)	69 (86.2%)		
Epworth score		4.6 ± 0.99	18.49 ± 3.71	t=29.479	**.000****
Epworth score classification	Lower Normal	16 (80%)	0 (0%)	X^2 = 87.500	**.000****
	Higher Normal	4 (20%)	4 (5%)		
	Mild Excessive	0 (0%)	3 (3.8%)		
	Moderate Excessive	0 (0%)	9 (11.2%)		
	Severe Excessive	0 (0%)	64 (80%)		
AHI (per h)		3.59 ± 1.35	35.10 ± 20.08	t= 6.988	**.000 ****
Severity	Normal	20 (100%)	0 (0%)	X^2 = 100	**.000****
	Mild	0 (0%)	13 (16.3%)		
	Moderate	0 (0%)	23 (28.7%)		
	Severe	0 (0%)	44 (55%)		
ODI		4.63 ± 4.06	37.32 ± 27.62	t= 3.558	**.001****
ODI classification	Normal	14 (70%)	12 (15%)	X^2 = 30.955	**.000****
	Mild	6 (30%)	6 (7.5%)		
	Moderate	0 (0%)	17 (21.25%)		
	Severe	0 (0%)	45 (56.25%)		
Snoring index		218.67 ± 217.60	306.67 ± 185.20	t= 1.834	.070
Sleep efficiency (%)		88.16 ± 7.60	85.87 ± 10.03	t= 0.954	0.342
Baseline O_2 (%)		95.55 ± 2.06	93.31 ± 3.35	t= 2.848	**.005***
Minimal O_2 (%)		91.05 ± 4.35	77.54 ± 10.24	t= 8.998	**.000****
RDI		3.57 ± 1.33	35.16 ± 20.05	t= 13.975	**.000****
Creatinine (mg/ dl)		0.86 ± 0.18	2.01 ± 0.89	t= 5.735	**.000****
Urea (mg/dl)		35.60 ± 14.18	102.88 ± 73.53	t= 4.058	**.000****
e GFR		88.21 ± 28	40 ± 22.88	t= 8.048	**.000****
UCAR (mg/ g)		29.35 ± 10.93	432.85 ± 306.37	t= 5.867	**.000****
CKD staging	1	9 (45%)	2 (2.5%)	X^2= 51.273	**.000****
	2	11 (55%)	14 (17.5%)		
	3a	0 (0%)	9 (11.2%)		
	3b	0 (0%)	25 (31.3%)		
	4	0 (0%)	26 (32.5%)		
	5	0 (0%)	4 (5%)		
US	Normal	14 (70%)	10 (12.5%)	X^2= 41.042	**.000****
	I	6 (30%)	9 (11.2%)		
	II	0 (0%)	27 (33.8%)		
	III	0 (0%)	24 (30%)		
	IV	0 (0%)	10 (12.5%)		

t= independent t test, X2 = chi squared test, * significant, ** highly significant

Table 2: Comparison between different groups of the OSAS patients regarding anthropometric, clinical, and Polysomnographic data (n=80).

characteristics		Mild group (n=13)	Moderate group (n=23)	Severe group (n= 44)	Test of significance	P value
Age (year)		51.62 ± 9.16	57.09 ±10.41	53.82 ± 11.01	F= 1.260	0.289
Sex	Male	3 (23.1%)	13 (56.5%)	32 (72.7%)	X^2 = 10.470	**.005***
	Female	10 (76.9%)	10 (43.5%)	12 (27.3%)		
BMI (Kg/m^2)		42.09 ± 12.3	41.06 ± 9.63	39.9 ± 8.32	F= 0.314	0.732
Neck Circumference (cm)		39.11 ± 2.49	42.91 ± 2.97	48.44 ± 5.02	F= 30.324	**.000****
STOP BANG		4.46 ± 0.52	5.26 ± 0.81	6.70 ± 0.9	F=47.174	**.000****
STOP BANG classification	Intermediate risk	7 (53.8%)	4 (17.4%)	0 (0%)	X^2 =24.895	**.000****
	High risk	6 (46.2%)	19 (82.6%)	44 (100%)		
Epworth score		12.23 ± 2.49	17.65 ± 2.35	20.77 ± 1.80	F=86.673	**.000****
Epworth score classification	Lower Normal	0 (0%)	0 (0%)	0 (0%)	FET = 150.953	**.000****
	Higher Normal	4 (30.8%)	0 (0%)	0 (0%)		
	Mild Excessive	3 (23.1%)	0 (0%)	0 (0%)		
	Moderate Excessive	5 (38.4%)	4 (17.4%)	0 (0%)		
	Severe Excessive	1 (7.7%)	19 (82.6%)	44 (100%)		
AHI (per h)		9.92 ± 2.75	20.3 ± 3.52	50.27 ± 13.7	F=106.947	**.000****
ODI		12.7 ± 27.9	23.31 ± 17.47	54.3 ± 22.75	F= 41.037	**.000****
ODI classification	Normal	9 (61.5%)	3 (17.4%)	0 (0%)	X^2 = 64.711	**.000****
	Mild	4 (38.5%)	2 (17.4%)	0 (0%)		
	Moderate	0 (0%)	10 (39.1%)	7 (15.9%)		
	Severe	0 (0%)	8 (26.1%)	37 (84.1%)		
Snoring index		278.41 ± 211.06	202.23 ± 150.34	369.62 ± 169.96	F= 7.375	**.001****
Sleep efficiency (%)		86.70 ± 8.07	84.51 ± 10.51	86.34 ± 10.43	F= 0.298	0.744
Baseline O_2 (%)		93 ±3.92	92.91 ±4.47	93.61 ±2.43	F= 0.391	0.677
Minimal O_2 (%)		85.31 ±6.28	79.57 ±10.60	74.18 ±9.61	F= 7.655	**.001****
RDI		10.07 ±2.73	20.35 ±3.52	50.32 ±13.68	F= 106.862	**.000****

F= ANOVA test, X^2 = chi squared test, FET = Fisher's Exact Test, * Significant, ** highly significant.

Table 3: Comparison between different groups of the OSAS patients regarding renal characteristics (n=80).

Characteristics		Mild group (n=13)	Moderate group (n=23)	Severe group (n= 44)	Test of significance	P value
Creatinine (mg/dL)		1.26 ± 0.31	1.68 ± 0.56	2.4 ± 0.94	F= 13.740	**.000****
Urea (mg/dL)		49.46 ± 12.27	78.96 ± 32.1	131.16 ± 85.89	F= 9.627	**.000****
e GFR		50.88 ± 27.42	45.9 ± 21.56	33.7 ± 20.47	F= 4.224	**.018***
UCAR (mg/g)		136 ± 84.06	218.78 ± 80.11	632.45 ± 274.36	F= 43.947	**.000****
CKD staging	1	1 (7.7%)	0 (0%)	1 (2.3%)	X^2 = 20.721	**.023***
	2	3 (23%)	6 (26.1%)	5 (11.4%)		
	3a	4 (30.8%)	2 (8.7%)	3 (6.8%)		
	3b	5 (38.5%)	9 (39.1%)	11 (25%)		
	4	0 (0%)	6 (26.1%)	20 (45.5%)		
	5	0 (0%)	0 (0%)	4 (9.1%)		
US	Normal	3 (23.1%)	5 (21.7%)	2 (4.5%)	X^2 = 29.837	**.000****
	I	6 (46.2%)	1 (4.3%)	2 (4.5%)		
	II	4 (30.8%)	8 (34.8%)	15 (34.1%)		
	III	0 (0%)	7 (30.4%)	17 (38.6%)		
	IV	0 (0%)	2 (8.7%)	8 (18.2%)		

F= ANOVA test, X2 = chi squared test, * significant, ** highly significant

Table 4: Correlation analyses of the relationships between AHI (per h) and different covariates among OSAS patients (n=80).

characteristics	AHI All (n=100)		Control (n=20)		OSAS (n=80)	
	r	P value	R	P value	r	P value
Age (year)	0.158	0.116	0.130	0.585	-.034	0.766
Sex (male is reference)	-0.246	.014*	-.056	0.814	-0.242	**.030***
BMI (Kg/ m^2)	0.102	0.314	0.398	.082	-.020	0.861
Neck Circumference (cm)	0.598	.000**	0.239	0.310	0.567	**.000****
STOP BANG	0.769	.000**	0.363	0.116	0.677	**.000****
Epworth score	0.838	.000**	0.203	0.390	0.824	**.000****
ODI	0.791	.000**	0.100	0.674	0.865	**.000****
Snoring index	0.282	.004*	0.346	0.135	0.248	**.027***
Sleep efficiency (%)	-0.026	0.799	0.487	.029*	.033	0.772
Baseline O_2 (%)	-0.129	0.199	-0.303	0.194	.043	0.705
Minimal O_2 (%)	-0.636	.000**	0.120	0.613	-0.502	**.000****
RDI	0.999	.000**	0.992	.000**	0.987	**.000****
Creatinine (mg/ dl)	0.629	.000**	0.315	0.176	0.483	**.000****
Urea (mg/ dl)	0.474	.000**	0.226	0.338	0.339	**.002***
UACR	0.726	.000**	0.487	.030*	0.615	**.000****
e-GFR	-0.578	.000**	-0.379	.099	-.049	0.664

r= Pearson correlation, * significant, ** highly significant

Table 5: Effect of CPAP on creatinine titer and e GFR among OSAS patients (n=80).

	Before CPAP	After CPAP	Test of significance	P value
Creatinine (mg/ dl)	2.01 ± 0.89	1.45 ± 0.64	t= 12.477	**.000****
e GFR	40 ± 22.88	56.51 ± 32.53	t= 6.54	**.000****

t= paired t test, ** highly significant

Table 6: Simple and multiple linear regression analyses of e GFR after CPAP and different covariates among OSAS patients (n=80).

Characteristics	Simple Linear Regression e GFR after CPAP			Multiple Linear Regression e GFR after CPAP		
	B	P value	CI 95%	B	P value	CI 95%
Age (year)	-0.775	**.024***	-1.444 - -0.106	-0.295	**.002***	-1.466 - -0.340
Sex (male is reference)	-6.159	0.410	-20.968 – 8.649			
BMI (Kg/ m^2)	-0.398	0.313	-1.178 -0.382			
Neck Circumference (cm)	-1.839	**.005***	-3.098 - -0.579	-.008	0.945	-1.434 – 1.337
STOP BANG	-6.517	**.029***	-12.346 - -0.688	0.224	.102	-1.208 – 13.160
Epworth score	-0.228	**0.042***	-3.917 - -0.073	0.113	0.436	-1.524 – 3.495
AHI (/ h)	-0.497	**.006***	-0.844 - -0.149	-0.130	0.680	-0.758 – 0.497
ODI	-0.369	**.005***	-0.621 - -0.117	-0.023	0.900	-0.446 – 0.393
Snoring index	-.036	.066	-.075 - .003			
Sleep efficiency (%)	0.321	0.382	-0.406 – 1.048			
Baseline O_2 (%)	1.913	**.080***	-0.232 – 4.058	-0.025	0.804	-2.224 – 1.730
Minimal O_2 (%)	0.986	**.005***	0.305 – 1.666	0.116	0.297	-0.332 – 1.071
RDI	-0.497	**.006***	-0.845 - -0.149	-0.119	0.581	-0.887 – 0.502
Creatinine (mg/ dl)	-24.305	**.000****	-30.434 - -18.176	-0.908	**.000****	-44.204 - -22.02
Urea (mg/dl)	-0.199	**.000****	-0.288 - -0.110	.236	0.108	-.024 - 0.223

B= B coefficient, CI= Confidence Interval, * significant, ** highly significant.

Discussion:

OSA is a highly prevalent disorder of sleep-related breathing in which repeated episodes of upper airway collapse occur during sleep, leading to intermittent reductions in blood oxygen saturation and generating a cascade of systemic physiological disturbances. Over recent years, accumulating clinical and epidemiological evidence has suggested a substantial association between OSA and CKD, given that the kidney's complex microvascular structure and high metabolic demand render it particularly vulnerable to hypoxic injury. While this relationship has been increasingly recognized, the precise extent to which OSA severity accelerates renal decline remains uncertain. Furthermore, although CPAP therapy is the gold-standard intervention for OSA, its potential renoprotective benefits, particularly in improving kidney function or slowing CKD progression, have yet to be fully elucidated [(22)]. In light of these uncertainties, the present study was designed to evaluate renal function in OSA cases, investigate its relationship with disease severity, and assess the magnitude of renal improvement following CPAP therapy.

In the present analysis, AHI, considered a key polysomnographic marker of OSA severity, demonstrated statistically significant positive correlations with neck circumference, STOP-BANG score, ESS, ODI, snoring index, serum creatinine, urea, UACR, and eGFR. The concurrent rise in AHI alongside these parameters reflects a pattern in which more severe OSA is linked not only to greater sleep-related breathing abnormalities but also to more pronounced biochemical evidence of renal injury. Conversely, negative correlations between AHI and both minimal SpO_2 and sex were identified, implying that higher AHI values coincide with deeper nocturnal desaturation and that males in the OSAS+ subgroup, on average, exhibited slightly lower AHI scores than their female counterparts. Interestingly, BMI, often considered a major contributor to OSA pathophysiology, was not significantly correlated with AHI in this cohort, suggesting that other anatomical or physiological factors may have exerted a

stronger influence on OSA severity in this sample.

These observations are in agreement with the findings of Chou and co-authors [23],who examined 40 middle-aged cases with OSA and documented a mean AHI of 51.6 ± 39.2 eventsper h, mean eGFR of 85.4 ± 18.3 mLper minper 1.73 m², and mean UACR of 13.4 ± 23.4 mgper g. In their cohort, the prevalence of CKD among individuals with severe OSA reached 18%. Through multivariate regression analysis, AHI emerged as an independent predictor of UACR ($\beta = 0.26$, $P = 0.01$, $R^2 = 0.17$), while ODI independently predicted eGFR ($\beta = 0.32$, $P < 0.01$, $R^2 = 0.32$). In contrast, findings from the Wisconsin Sleep Cohort substudy by Canales and co-authors [24], which followed 855 participants over a mean of 13.9 years, failed to identify a statistically significant relationship between OSA severity and the rate of GFR decline. In that study, annual GFR drop was nearly identical between those with and without OSA (−0.7 vs. −0.9 mLper minper 1.73 m²per year, $P = 0.134$). This divergence from other studies may be explained by methodological differences, including the lower prevalence of OSA (11%), younger mean participant age (50.4 years), and relatively preserved baseline GFR (89.3 ± 13.8 mLper minper 1.73 m²).

In the current investigation, the introduction of CPAP therapy for 3 months in cases with moderate-to-severe OSA led to statistically significant improvements in renal function, as indicated by reductions in serum creatinine and increases in eGFR. These observations suggest that CPAP use may help protect kidney function by improving nocturnal oxygenation, attenuating sympathetic overstimulation, and reducing cyclical intrathoracic pressure changes, all of which can contribute to renal stress. The renoprotective potential of CPAP is supported by prior work.

Kinebuchi and co-authors [25] demonstrated that short-term CPAP reduced glomerular hyperfiltration, likely by improving renal plasma flow and decreasing the filtration fraction. Similarly, Koga and co-authors [26] showed that 3 months of CPAP in 27 male OSA cases resulted in a decrease in creatinine from 0.87 to 0.82 mgper dL and an increase in eGFR from 72.9 to 79.3 mLper minper 1.73 m². While both sets of findings achieved statistical significance, their broader clinical applicability remains uncertain due to small sample sizes, absence of control groups, and limited adjustment for confounding variables.

Regression analyses in the present study revealed that greater post-CPAP improvements in eGFR were predicted by younger age, smaller neck circumference, lower STOP-BANG and ESS scores, lower AHI and ODI, higher baseline and minimum SpO_2, lower RDI, and lower baseline creatinine and urea ($P \leq 0.05$). Notably, in multivariate modeling, only younger age and lower baseline creatinine retained statistical significance as independent predictors ($P \leq 0.001$). This suggests that both physiological reserve and the absence of advanced baseline renal injury may enhance the potential for renal recovery with CPAP therapy.

The interplay between OSA, systemic inflammation, and renal health is further supported by Yokoe and co-authors [27], who found that 1 month of CPAP therapy in 30 cases with OSA significantly improved AHI (57.7 ± 4.3 to 1.7 ± 0.5, $P < 0.0001$), increased nadir SpO_2 (66.9 ± 2.6 to 90.5 ± 1.3, $P < 0.0001$), extended total sleep time, and reduced systemic inflammatory markers like CRP (0.29 ± 0.02 to 0.11 ± 0.03, $P < 0.0001$) and IL-6 (1.20 ± 0.15 to 0.45 ± 0.08, $P < 0.001$). Importantly, the magnitude of AHI drop positively correlated with reductions in IL-6 ($r = 0.59$, $P < 0.05$), CRP ($r = 0.57$, $P < 0.05$), and ESS ($r = 0.60$, $P < 0.05$), suggesting that a decrease in systemic inflammation may be a plausible

mechanism linking CPAP therapy to both cardiovascular and renal benefits.

The **strengths** of this study include its prospective cohort design, which allows temporal relationships to be observed; the inclusion of a healthy control group, which improves the validity of between-group comparisons; the broad and detailed collection of clinical and laboratory measures; and the inclusion of a pre–post CPAP intervention analysis, which directly assesses treatment-related changes in renal function. Nonetheless, **limitations** must be acknowledged, including the single-center setting, the relatively small sample size, and the short follow-up duration of 3 months, which may not reflect the durability of the observed renal improvements.

Conclusion:

OSA is linked to clinically relevant renal impairment, as evidenced by elevated creatinine and urea alongside reduced eGFR. The observed renal improvements following CPAP therapy suggest a renoprotective role for this intervention, potentially extending its benefits beyond the correction of sleep-disordered breathing.

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Author Contributions:

All authors made equal and substantial contributions to the study. Their involvement covered the development of the research concept and design, participant recruitment and data collection, statistical analysis, interpretation of findings, and drafting and critical revision of the manuscript. All authors reviewed and approved the final version and took full responsibility for the accuracy and integrity of the work.

Conflicts of Interest:

The authors confirm that they have no personal, professional, or financial conflicts of interest that could have influenced the conduct, results, or reporting of this research.

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