Sarcouncil Journal of Internal Medicine and Public Health

ISSN(Online): 2945-3674

Volume- 05| Issue- 02| 2026

Research Article

Received: 10-03-2026 | **Accepted:** 05-04-2026 | **Published:** 21-04-2026

Obstructive Sleep Apnea Syndrome: Cardiovascular Comorbidities and the Role of Collaborative ENT-Internal Medicine Care

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Abstract: Background: Obstructive Sleep Apnea Syndrome (OSAS) is slowly being identified as a risk factor on its own in cardiovascular conditions. Nonetheless, treatment of cardiovascular comorbidities related to OSAS usually remains disjointed between internal medicine and otorhinolaryngology. The present study compares the rates of cardiovascular comorbidities among the groups of OSAS severity. The results of a structured collaborative ENT-Internal Medicine care model, as well as patients, were subjected to full ENT examination (including Drug-Induced Sleep Endoscopy), cardiovascular examination (echocardiography, ambulatory blood pressure monitoring, cardiac biomarkers), and treated in a weekly multidisciplinary team conference. The evaluation was done at 6 months. There were moderate to strong correlations between all cardiovascular parameters and AHI (r = 0.28-0.52). The collaborative strategy resulted in a 62 percent mean AHI decrease, 9.8 mmHg systolic BP decrease, almost doubling BP control rates (39.5 percent to 68.3 percent), and CPAP observance of 72.4 percent at 6 months. Conclusions: Multisystem nature OSAS can be successfully treated in a collaborative ENT-Internal Medicine care model, and treatment adherence and cardiovascular outcomes can be improved in comparison with the historical single-specialty standards. The findings can be used to justify the adoption of integrated care paths when working with OSAS patients with cardiovascular comorbidities.

Keywords: Obstructive Sleep Apnea, cardiovascular parameters, BP, AHI, OSAS, ENT.

INTRODUCTION

Obstructive Sleep Apnea Syndrome (OSAS) is a pervasive multifactorial condition of partial or total obstruction of the upper airway during sleep, resulting in intermittent hypoxia, fragmentation of sleep, and excessive daytime drowsiness. Although OSAS is a disease spectrum, its consequences are much more profound than nocturnal symptoms, including high levels of cardiovascular morbidity, dysmetabolism, neurocognitive impairment, and decreased quality of life where OSAS epidemiologically impacts a relatively large proportion of the adult population whose prevalence rates rise with age, body mass index, and male gender, although postpartum, postmenopausal and some ethnic groups exhibit some unique rates [McDonagh, T. A. *et al*., 2023; Williams, B. *et al*., 2018]. The clinical picture of OSAS is diverse: some patients have loud snoring and observed apneas, and some have non-specific symptoms: in the morning, they may have headaches, fatigue, or mood swings. Notably, a lot of people are not diagnosed or not treated adequately, even though the negative health outcomes are well-documented. Cardiovascular disease (CVD) risk matrices are similar to classical risk factors, which creates a two-way relationship of OSAS enhancing cardiovascular risk and cardiovascular disease increasing sleep-disordered breathing via fluid movements, obesity, and autonomic dysregulation [Zhang, W., & Si, L. Y. 2012; Parati, G. *et al*., 2014]. The repetitive hypoxia and reoxygenation cycles' chronicity stimulates sympathetic response, inflammation, oxidative stress, and endothelial dysfunction, which, collectively, support blood pressure increase and vascular remodeling. Other than hypertension, OSAS is associated with arrhythmias, such as atrial fibrillation, ventricular ectopy, and sudden cardiac death, especially during co-morbid cardiovascular disease and heart failure. Coronary artery disease and cerebrovascular events are also more common in individuals with OSAS, which is probably due to common risk factors, including obesity, metabolic syndrome, and dysregulated autonomic tone [Meng, F. *et al*., 2016; Baguet, J. P. *et al*., 2009; Noda, A. *et al*., 1995]. Also, metabolic imbalances that are typical of OSAS, including insulin resistance, dyslipidemia, and systemic inflammation, only intensify the cardiovascular risk, which is why a vicious cycle of cardiovascular risk increases the importance of early diagnosis and holistic treatment [Goudis, C. A., & Ketikoglou, D. G. 2017; Iwasaki, Y. K. *et*

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DOI- https://doi.org/10.5281/zenodo.19677748

al., 2012; Thareja, S. *et al*., 2024]. From a pathophysiological perspective, a number of interconnected pathways can be used to clarify how OSAS leads to cardiovascular disease. Oxidative stress and systemic inflammation caused by recurrent hypoxic episodes, in its turn, encourage endothelial dysfunction and a prothrombotic state. Fragmentation of sleep and decreased slow-wave sleep disrupted autonomic balance, increased nocturnal and daytime sympathetic activity, changes in heart rate variability, and blood pressure variability [Gami, A. S. *et al*., 2007; Mehra, R. *et al*., 2006]. The renin-angiotensin-aldosterone system is also activated by intermittent hypoxia, which also helps to retain fluid, raise blood pressure, and remodel the heart [Linz, D. *et al*., 2018]. This model of collaboration acknowledges that the use of continuous positive airway pressure (CPAP) therapy, mandibular advancement devices, weight management, and lifestyle modification are some of the basic treatments to OSAS, but their efficacy and patient adherence are improved when incorporated into a multidisciplinary model that also covers cardiovascular risk modulation, metabolic control, and psychosocial support. [Barbé, F. *et al*., 2012; McEvoy, R. D. *et al*., 2016]

Observational studies and randomized trials provide evidence of the influence of a successful treatment of OSAS on cardiovascular outcomes, although there is a wide range of variability between populations and adherence rates [Marin, J. M. *et al*., 2005]. The primary intervention in the treatment of OSAS, CPAP therapy, has been shown to lower blood pressure and enhance left ventricular functioning in some patients and reduce the burden of the arrhythmia, especially when applied regularly [Chen, H. *et al*., 2024]. The effectiveness of treatment, however, depends on the adherence of the patient, proper diagnosis, and the right choice of the device. In this respect, care models that can be used in collaboration will provide the opportunity to make shared decisions, provide thorough consideration of comorbidities, and customize interventions to the risk profile of an individual. Indicatively, assessments of airway patency that are initiated by the ENT can determine candidates of surgical or less invasive airway procedures that could improve CPAP tolerance or can be substitutes in some cases [Battisha, A. *et al*., 2025; Peker, Y. *et al*., 2016]. Antihypertensive regimens, lipid-lowering therapies, glycemic control and screening of cardiac arrhythmias can be used to optimize cardiovascular risk management in internists and cardiologists, and sleep specialists ensure the efficacy and adherence to CPAP, reassess treatment regimens, and change the therapy as cardiac comorbidities change in status [Iftikhar, I. H. *et al*., 2013] so This background establishes the groundwork of a scientific discussion of the interaction of OSAS and cardiovascular disease and how a structured, interdisciplinary ENT–internal medicine care model may enhance patient outcomes

METHODOLOGY

Design and Ethics of the study

This prospective observational cohort study was undertaken in the Department of Otorhinolaryngology and the Department of Internal Medicine (Division of Cardiology) of a tertiary different hospitals in Iraq between January 2024 and December 2025, where eighty-six sequential adult patients (18 years and above) diagnosed with Obstructive Sleep Apnea Syndrome (OSAS) were recruited. Diagnosis was made based on the results of an overnight Level I polysomnography (PSG) according to the 2012 criteria of the American Academy of Sleep Medicine (AASM), an Apnea-Hypopnea Index (AHI) of 5 or more events/ hour with symptoms of excessive daytime sleepiness, observed apneas, or choking/gasping during sleep.

The inclusion criteria included: (1) age 18 years old and older, (2) diagnosed with OSAS using PSG, (3) willingness to use the collaborative ENT-Internal Medicine care program, and (4) the possibility of following up regularly. Inclusion criteria were: (1) central sleep apnea (central apnea index > 5 events/hour), (2) previous surgical intervention on OSAS, (3) malignancy, (4) severe psychiatric illness that would not allow informed consent, (5) pregnancy, and (6) life expectancy < 12 months other than cardiovascular causes.

AHI was used to stratify patients into three categories according to AHI severity levels: Mild OSAS (AHI 5-14.9 events/hour, n = 24), Moderate OSAS (AHI 15-29.9 events/hour, n = 31), and Severe OSAS (AHI 30 events/hour and above, n = 31). A multidisciplinary evaluation protocol was followed on all patients. Polysomnographic Assessment: Overnight attended Level I PSG was done on a 32-channel digital system (Compu medics Grael, Australia). Parameters that were recorded were electroencephalography (EEG: C3-A2, C4-A1, O1-A2, O2-A1), electrooculography (EOG), submental and bilateral tibial

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electromyography (EMG), nasal pressure transducer, oronasal thermistor, and thoracic and abdominal respiratory inductance plethysm. Certified polysomnographic technologists scored sleep stages and respiratory events via the AASM criteria of 2012.

ENT Examination: A complete upper airway examination was done by a senior otorhinolaryngologist, including: (1) flexible nasopharyngolaryngoscopy with Muller maneuver, (2) Modified Mallampati classification, (3) Friedman tongue position and tonsil grading, (4) nasal septal deviation, turbinate hypertrophy and nasal polyposis, (5) tongue

Cardiovascular Evaluation: The internal medicine staff conducted: (1) office and 24-hour ambulatory blood pressure (ABPM), (2) 12-lead electrocardiography, (3) transthoracic echocardiography (GE Vivid E95) with measurement of left ventricular ejection where The collaborative ENT-Internal Medicine model of care was designed according to the weekly multidisciplinary team (MDT) conference during which the case of each patient was discussed. Collaborative decision-making was used to make treatment decisions using the following algorithm:

Step 1 - Severity Assessment: PSG results, ENT assessment, and cardiovascular assessment are conducted together to determine the severity of the disease and the main locations of upper airway obstruction.

Step 2 -Risk Stratification: Risk stratification of cardiovascular risk based on existing scoring systems (Framingham Risk Score, ESC/ERS guidelines) combined with OSAS severity to establish the urgency of treatment.

Step 3 — Treatment Planning: Personalized treatment plans that include: (a) CPAP initiation and titration with nasal airways optimization by an ENT, (b) surgical procedures at anatomically favorable sites of obstruction based on DISE, (c) cardiovascular risk factor management, including antihypertensive optimization, statin therapy, glycemic control, and lifestyle change, and (d) weight loss

Step 4 -Follow-up Protocol: The patients were assessed after 1, 3, and 6 months in terms of CPAP download information, symptom measurement (ESS), blood pressure analysis, and repeat echocardiography and laboratory tests performed at 6 months.

Statistical Analysis

The SPSS version 28.0 were used to perform statistical analyses. Means (SD) were used in the normally distributed variables, and a median (IQR) in the skewed variables as the continuous variables. The Shapiro-Wilk test was used to test normality. Categorical variables were in the form of frequencies and percentages.

One-way ANOVA with post-hoc Bonferroni correction of normally distributed data, or Kruskal-Wallis test with Dunn post-hoc test of non-parametric data were used to compare between-groups in continuous variables. The Chi-square or Fisher's exact test, whichever was appropriate, was used to compare categorical variables. Paired t-tests or Wilcoxon signed-rank tests were done when paired comparisons (baseline vs. 6 months) were done.

The Spearman rank correlation coefficient was used to test correlations between AHI and cardiovascular parameters. The independent predictors of cardiovascular comorbidity were identified using multivariate logistic regression, by age, sex, BMI, smoking status, and diabetes. The p-value of 0.05 was taken to be statistically significant.

Calculation of the sample size: Assuming a correlation coefficient of r = 0.30 between AHI and cardiovascular parameters, 0.05 was the alpha, and 0.80 was the power, a minimum of 82 patients was needed. We recruited 86 patients in case of dropouts.

RESULTS

Table 1. Demographic and Baseline Characteristics of 86 OSAS Patients.

Characteristic	Total (n=86)	Mild OSAS (n=24)	Moderate OSAS (n=31)	Severe OSAS (n=31)
Age (years), mean ± SD	52.4 ± 11.7	48.2 ± 10.3	52.8 ± 11.9	55.6 ± 11.8
Male, n (%)	58 (67.4)	14 (58.3)	21 (67.7)	23 (74.2)
Female, n (%)	28 (32.6)	10 (41.7)	10 (32.3)	8 (25.8)
BMI (kg/m²), mean ± SD	31.8 ± 5.4	28.3 ± 3.9	31.6 ± 4.8	34.9 ± 5.6

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Neck circumference (cm), mean ± SD	41.2 ± 4.1	38.4 ± 3.2	41.0 ± 3.6	43.8 ± 3.9
Waist circumference (cm), mean ± SD	104.6 ± 13.2	96.8 ± 10.4	104.2 ± 12.1	111.8 ± 13.0
Current smoker, n (%)	29 (33.7)	6 (25.0)	10 (32.3)	13 (41.9)
Alcohol use (≥3 drinks/week), n (%)	22 (25.6)	4 (16.7)	8 (25.8)	10 (32.3)
Epworth Sleepiness Scale, mean ± SD	13.4 ± 4.8	9.2 ± 3.1	13.1 ± 3.9	17.2 ± 4.1
Duration of symptoms (years), mean ± SD	4.8 ± 3.2	2.9 ± 2.1	4.6 ± 2.8	6.7 ± 3.5
Family history of OSAS, n (%)	19 (22.1)	4 (16.7)	7 (22.6)	8 (25.8)
Diabetes mellitus, n (%)	21 (24.4)	3 (12.5)	7 (22.6)	11 (35.5)
Dyslipidemi, n (%)	38 (44.2)	8 (33.3)	14 (45.2)	16 (51.6)

Table 2. Polysomnographic Parameters by OSAS Severity.

Parameter	**Total (n=86)**	**Mild (n=24)**	**Moderate (n=31)**	**Severe (n=31)**	**p-value**
AHI (events/h), mean ± SD	32.6 ± 21.4	9.8 ± 2.9	22.1 ± 4.3	58.7 ± 16.2	**<0.001***
Obstructive apneas (events/h)	18.4 ± 16.8	3.2 ± 2.1	11.6 ± 5.4	36.8 ± 14.3	**<0.001***
Hypopneas (events/h)	14.2 ± 8.6	6.6 ± 2.4	10.5 ± 3.8	21.9 ± 8.1	**<0.001***
ODI (events/h), mean ± SD	29.8 ± 20.1	8.4 ± 3.1	19.6 ± 5.8	54.2 ± 15.8	**<0.001***
Mean SpO_2 (%), mean ± SD	91.4 ± 4.2	94.6 ± 1.8	92.1 ± 2.4	87.8 ± 4.1	**<0.001***
Minimum SpO_2 (%), mean ± SD	76.8 ± 10.4	86.2 ± 4.3	78.4 ± 6.2	67.1 ± 9.8	**<0.001***
Time SpO_2 < 90% (min), mean ± SD	48.6 ± 42.3	8.2 ± 6.4	32.4 ± 18.6	96.8 ± 38.4	**<0.001***
Total sleep time (min), mean ± SD	342.8 ± 58.4	361.2 ± 48.6	348.6 ± 54.2	321.4 ± 64.8	**0.038***
Sleep efficiency (%), mean ± SD	78.4 ± 12.6	84.2 ± 8.4	79.6 ± 11.2	72.4 ± 14.8	**0.002***
REM sleep (%), mean ± SD	14.8 ± 6.2	18.4 ± 4.8	15.2 ± 5.6	11.6 ± 6.4	**<0.001***
N3 sleep (%), mean ± SD	12.4 ± 7.8	16.8 ± 6.2	12.6 ± 7.4	8.6 ± 7.2	**<0.001***
Arousal index (events/h), mean ± SD	38.2 ± 18.6	18.4 ± 6.8	34.6 ± 10.4	56.8 ± 14.2	**<0.001***
Supine AHI (events/h), mean ± SD	42.8 ± 26.4	14.6 ± 5.8	32.4 ± 10.2	72.4 ± 22.6	**<0.001***

Table 3. Prevalence of Cardiovascular Comorbidities Among OSAS Patients.

Cardiovascular Comorbidity	**Total (n=86) n (%)**	**Mild (n=24) n (%)**	**Moderate (n=31) n (%)**	**Severe (n=31) n (%)**
Systemic hypertension	52 (60.5)	9 (37.5)	19 (61.3)	24 (77.4)
- Resistant hypertension	14 (16.3)	1 (4.2)	4 (12.9)	9 (29.0)
- Nocturnal non-dipping pattern	34 (39.5)	5 (20.8)	12 (38.7)	17 (54.8)
Atrial fibrillation	12 (14.0)	1 (4.2)	3 (9.7)	8 (25.8)
Coronary artery disease	15 (17.4)	2 (8.3)	5 (16.1)	8 (25.8)
Heart failure (EF < 50%)	8 (9.3)	0 (0.0)	2 (6.5)	6 (19.4)
Pulmonary hypertension	11 (12.8)	1 (4.2)	3 (9.7)	7 (22.6)
Left ventricular hypertrophy	22 (25.6)	3 (12.5)	7 (22.6)	12 (38.7)
Diastolic dysfunction	31 (36.0)	4 (16.7)	11 (35.5)	16 (51.6)
History of stroke/TIA	7 (8.1)	1 (4.2)	2 (6.5)	4 (12.9)

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Peripheral arterial disease	6 (7.0)	0 (0.0)	2 (6.5)	4 (12.9)
Metabolic syndrome	41 (47.7)	6 (25.0)	15 (48.4)	20 (64.5)
≥2 CV comorbidities, n (%)	38 (44.2)	4 (16.7)	13 (41.9)	21 (67.7)

Table 4. ENT Findings and Upper Airway Assessment.

ENT Finding	Total (n=86) n (%)	Mild (n=24) n (%)	Moderate (n=31) n (%)	Severe (n=31) n (%)
Modified Mallampati Score				
Class I	8 (9.3)	5 (20.8)	2 (6.5)	1 (3.2)
Class II	22 (25.6)	10 (41.7)	8 (25.8)	4 (12.9)
Class III	34 (39.5)	7 (29.2)	14 (45.2)	13 (41.9)
Class IV	22 (25.6)	2 (8.3)	7 (22.6)	13 (41.9)
Tonsil Grade				
Grade 0–1	28 (32.6)	12 (50.0)	10 (32.3)	6 (19.4)
Grade 2	30 (34.9)	8 (33.3)	12 (38.7)	10 (32.3)
Grade 3	18 (20.9)	3 (12.5)	6 (19.4)	9 (29.0)
Grade 4	10 (11.6)	1 (4.2)	3 (9.7)	6 (19.4)
Nasal septal deviation	38 (44.2)	9 (37.5)	14 (45.2)	15 (48.4)
Inferior turbinate hypertrophy	46 (53.5)	10 (41.7)	17 (54.8)	19 (61.3)
Nasal polyposis	8 (9.3)	2 (8.3)	3 (9.7)	3 (9.7)
Tongue base hypertrophy	32 (37.2)	5 (20.8)	11 (35.5)	16 (51.6)
Soft palate elongation	42 (48.8)	8 (33.3)	15 (48.4)	19 (61.3)
Retrognathia	14 (16.3)	3 (12.5)	4 (12.9)	7 (22.6)
Friedman Stage				
Stage I	12 (14.0)	7 (29.2)	4 (12.9)	1 (3.2)
Stage II	34 (39.5)	11 (45.8)	14 (45.2)	9 (29.0)
Stage III	40 (46.5)	6 (25.0)	13 (41.9)	21 (67.7)
DISE: Complete concentric collapse	18 (20.9)	2 (8.3)	5 (16.1)	11 (35.5)

Table 5. Correlation Between OSAS Severity and Cardiovascular Parameters.

Cardiovascular Parameter	Total (n=86)	Mild (n=24)	Moderate (n=31)	Severe (n=31)	r (correlation)
Systolic BP (mmHg), mean ± SD	142.6 ± 18.4	132.4 ± 12.8	141.8 ± 16.2	151.8 ± 19.6	0.42
Diastolic BP (mmHg), mean ± SD	88.4 ± 11.2	82.6 ± 8.4	87.8 ± 10.6	93.8 ± 11.8	0.38
Mean arterial pressure (mmHg)	106.5 ± 12.8	99.2 ± 9.2	105.8 ± 11.6	113.1 ± 13.4	0.41
Heart rate (bpm), mean ± SD	76.8 ± 12.4	72.4 ± 9.6	76.2 ± 11.8	81.2 ± 13.8	0.28
LV ejection fraction (%), mean ± SD	56.8 ± 8.6	61.4 ± 5.2	57.8 ± 7.4	52.1 ± 9.8	-0.39
E/A ratio, mean ± SD	0.92 ± 0.28	1.12 ± 0.22	0.94 ± 0.24	0.74 ± 0.26	-0.48
E/e' ratio, mean ± SD	11.4 ± 4.2	8.6 ± 2.4	11.2 ± 3.6	13.8 ± 4.6	0.44
LVMI (g/m²), mean ± SD	108.4 ± 28.6	92.4 ± 18.2	106.8 ± 24.6	123.4 ± 30.8	0.41
LA diameter (mm), mean ± SD	39.8 ± 5.6	36.4 ± 4.2	39.6 ± 5.0	42.8 ± 5.8	0.40
RVSP (mmHg), mean ± SD	32.4 ± 10.8	26.2 ± 6.4	31.4 ± 8.6	38.4 ± 12.4	0.42

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NT-proBNP (pg/mL), median (IQR)	186 (82–412)	68 (34–142)	164 (86–328)	386 (168–642)	0.52
hs-CRP (mg/L), median (IQR)	3.8 (1.6–7.2)	1.4 (0.8–2.6)	3.4 (1.8–6.2)	6.8 (3.4–11.4)	0.48
Homocysteine (µmol/L), mean ± SD	14.8 ± 6.2	11.2 ± 3.8	14.4 ± 5.4	18.2 ± 6.8	0.38

Table 6. Treatment Outcomes After 6 Months of Collaborative ENT-Internal Medicine Management.

Outcome Measure	Baseline (n=86)	6 Months (n=82)	Change	p-value
AHI (events/h), mean ± SD	32.6 ± 21.4	12.4 ± 10.8	-20.2 ± 14.6	<0.001*
AHI reduction ≥ 50%, n (%)	—	62 (75.6)	—	—
ESS score, mean ± SD	13.4 ± 4.8	7.2 ± 3.4	-6.2 ± 3.8	<0.001*
BMI (kg/m²), mean ± SD	31.8 ± 5.4	30.4 ± 5.1	-1.4 ± 1.2	<0.001*
Systolic BP (mmHg), mean ± SD	142.6 ± 18.4	132.8 ± 14.2	-9.8 ± 10.6	<0.001*
Diastolic BP (mmHg), mean ± SD	88.4 ± 11.2	82.4 ± 9.6	-6.0 ± 7.4	<0.001*
BP control achieved, n (%)	34 (39.5)	56 (68.3)	+22 (28.8%)	<0.001*
LV ejection fraction (%), mean ± SD	56.8 ± 8.6	59.2 ± 7.4	+2.4 ± 4.2	0.004*
E/A ratio, mean ± SD	0.92 ± 0.28	1.02 ± 0.24	+0.10 ± 0.18	0.002*
NT-proBNP (pg/mL), median (IQR)	186 (82–412)	124 (56–286)	-62 (−184 to −18)	<0.001*
hs-CRP (mg/L), median (IQR)	3.8 (1.6–7.2)	2.4 (1.0–4.8)	-1.4 (−3.2 to −0.4)	<0.001*
CPAP prescribed, n (%)	—	58 (70.7)	—	—
CPAP adherence ≥ 4h/night, n (%)	—	42 (72.4)	—	—
Surgical intervention, n (%)	—	28 (34.1)	—	—
- UPPP/tonsillectomy	—	16 (19.5)	—	—
- Nasal surgery (septoplasty/turbinoplasty)	—	18 (22.0)	—	—
- Tongue base reduction	—	6 (7.3)	—	—
SF-36 Physical Component, mean ± SD	38.4 ± 8.6	46.8 ± 7.2	+8.4 ± 6.4	<0.001*
SF-36 Mental Component, mean ± SD	42.2 ± 10.4	52.4 ± 8.6	+10.2 ± 7.8	<0.001*
New AF episodes, n (%)	—	2 (2.4)	—	—
Cardiovascular events, n (%)	—	3 (3.7)	—	—

DISCUSSION

Our 86-patient cohort has a demographic distribution showing mainly males (67.4%), which is in line with the established male preponderance in OSAS. The average age of 52.4 years with 11.7 standard deviation is consistent with the highest prevalence range found in epidemiological studies by Peppard *et al*. (2013), whose highest-risk range of sleep-disordered breathing prevalence was found to be 50-70 years old.

It is intriguing to note that BMI has been on the rise in all severity categories, 28.3 -3.9 kg/m2 in mild OSAS, and 34.9 -5.6 kg/m2 in severe OSAS ($p < 0.001$). Such a dose-response interrelationship between obesity and the severity of OSAS highlights the importance of adipose tissue deposition in the pharyngeal area being one of the most important pathogenic factors. The results of the Epworth Sleepiness Scale were dramatically increased in mild cases (9.2 ± 3.1) and severe cases (17.2 ± 4.1), with a p value of less than 0.001, meaning that the subjective daytime sleepiness is significantly associated with the objective severity of the disease. The average duration of the symptoms at 4.8 ± 3.2 years, with the highest range at 6.7 ± 3.5 years, indicating a worrying lag in diagnosis that could be involved in the development of cardiovascular morbidity.

Metabolic comorbidities were also significantly higher than the general population estimates, with diabetes mellitus (24.4) and dyslipidemia (44.2) being of particular interest, which substantiates the idea that OSAS can be considered to be a part of the metabolic syndrome. The tendency towards increased prevalence of diabetes among severe OSAS (35.5% vs. 12.5% in mild) was not statistically significant in our sample, but this is probably due to the small sample size, and not necessarily the lack of association. The polysomnographic data showed that the anticipated

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gradient of respiratory disturbance by level of severity was present, with the severe OSAS group exhibiting an average AHI of 58.7 /16.2 events/hour- almost six times greater than the mild group (9.8 /12 events/hour). Oxygen desaturation index (ODI) was more similar to AHI values, which indicated that airway obstruction events were tightly coupled with hemoglobin desaturation in our cohort.

Of cardiovascular interest is the particularly alarming oxygenation parameters. The severe OSAS group had a mean minimum SpO 2 of 67.1 + 9.8 with an average of 96.8 + 38.4 minutes spent below the 90% level of saturation. These are the deep sporadic hypoxic events that are the major contributors to sympathetic nervous system stimulation, oxidative stress, and endothelial dysfunction. This pathophysiological triad connects OSAS to cardiovascular disease, as outlined by Lévy *et al*. (2015).

There was significant disruption of sleep architecture in relation to the severity of the disease. The percentage of REM sleep dropped by 18.4 percent, from mild to 11.6 percent severe OSAS ($p < 0.001$), and deep sleep (N3) dropped by 16.8 percent to 8.6 percent. This disruption in restorative sleep phases adds to the metabolic imbalance, loss of glucose tolerance, and increased inflammatory condition in severe OSAS patients. The arousal index of 56.8 ± 14.2 events/hour in the severe group implies that sleep interruption is almost continuous, with an average of less than one minute of continuous sleep in the patients.

The supine AHI was significantly greater than the overall AHI in all groups, with the biggest effects of positioning being observed in the mild group (supine AHI 14.6 vs. overall AHI 9.8), indicating that positional therapy could be a feasible adjunctive intervention measure, especially in milder cases. The burden of cardiovascular comorbidity of our OSAS cohort is significant and shows a well-defined severity-based gradient. The most common cardiovascular condition was systemic hypertension, with 60.5% of all patients having it, with an amazing rise of 37.5 to 77.4 in mild and severe OSAS, respectively ($p = 0.008$). This is higher than the estimates in the general population by about two-fold and much higher than the results of the Wisconsin Sleep Cohort Study by Peppard *et al*. (2000) that showed a dose-response relation between AHI and incident hypertension.

High levels of resistance hypertension (16.3% overall, 29.0% in severe OSAS) and nocturnal non-dipping blood pressure pattern (39.5% overall, 54.8% in severe OSAS) are of particular clinical importance. The non-dipping effect is directly explained by repeated sympathetic bursts at night due to apnea and is known to be an independent predictor of cardiovascular outcomes. These results are in line with existing guidelines to screen all patients with resistant hypertension with OSAS.

The prevalence of atrial fibrillation (AF) of 14.0% in our cohort, and of 25.8% in severe OSAS, is similar to that of Mehra *et al*. (2006), who found a 2.5-fold increased risk of AF in severe OSAS. The suggested mechanisms are atrial remodeling due to chronic intermittent hypoxia, swings in intrathoracic pressure that lead to atrial stretch, and the imbalance of the autonomic nervous system in favor of vagal prevalence during apnea.

Reduced ejection fraction heart failure was found in 9.3% of the patients, but only in the moderate and severe categories. The prevalence of diastolic dysfunction was higher (36.0%), where more than half of severe OSAS patients were affected. This trend indicates that cardiac injury in OSAS occurs as a consequence of diastolic dysfunction, which transitions to systolic failure, mediated by sustained pressure overload, intermittent chronic episodes of myocardial injury caused by hypoxia, and neurohormonal stimulation.

The observation that 67.7% of the severe OSAS patients had two or more cardiovascular comorbidities ($p < 0.001$) highlights the idea of OSAS as a cardiovascular risk multiplier and not a single upper airway disease. This comorbidity clustering is the best reason to support the collaborative ENT-Internal Medicine care model that is tested in this research. The ENT examination demonstrates a complicated anatomy of the upper airway, which adds to the obstruction at several levels. The distribution of Modified Mallampati scores exhibited a significant change towards higher classes as the severity of OSAS increased ($p = 0.018$), where 41.9% of the severe OSAS patient group were Mallampati IV, against 8.3% of the mild cases. The severity of Obstructive Sleep Apnea Syndrome (OSAS) in relation to cardiovascular parameters is correlated, which demonstrates that there is a considerable structural and functional cardiac impairment associated with sleep-disordered breathing. OSAS is associated with high blood pressure, and systolic blood pressure is very high even in the presence of

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antihypertensives, which is supported by a significant correlation with the Apnea-Hypopnea Index (AHI). The conventional findings of the echocardiography show gradual diastolic dysfunction and concentric hypertrophy, which are defined by some of the key metrics such as the E/A ratio and the left ventricular mass index. Right heart involvement and pulmonary vasoconstriction are signs that indicate possible complications, including pulmonary hypertension. There are signs of cardiovascular stress through biomarkers; the NT-proBNP and high-sensitivity CRP levels are found to be related to AHI. The interdisciplinary approach to treatment led to a considerable AHI decline and a greater adherence to the CPAP treatment, which was supported by simultaneous surgical procedures. Blood pressure management was significantly enhanced, which is consistent with the literature that relates CPAP to significant cardiovascular improvements. Minimal improvements in left ventricular performance with decreases in biomarkers suggest a reduced burden of myocardial stress and inflammation. In general, there was a great increase in quality of life, with low new cardiovascular events in the follow-up period, which indicates the efficiency of the integrated management approach.

CONCLUSION

This is a prospective group of 86 patients with Obstructive Sleep Apnea Syndrome (OSAS), which proves the usefulness of a joint care approach between ENT and Internal Medicine. The main results are that cardiovascular comorbidities are high in more than 60% of patients with OSAS, and it is significantly related to the severity of the disease. The multilevel nature of upper airway obstruction makes comprehensive ENT assessments necessary in order to achieve optimal treatment. The teamwork strategy led to a 62 percent average decrease in Apnea-Hypopnea Index (AHI), and 75.6 percent of the patients reported significant improvement. A 9.8 mmHg decrease in systolic blood pressure and significant improvements in quality of life measures indicated that cardiovascular health had significantly improved after 6 months. The adherence level to CPAP was also higher than historical levels at 72.4%. These results suggest the need to incorporate ENT-Internal Medicine pathways in the management of OSAS, especially in patients with a severe disease and cardiovascular problems, which means that the old approaches to the treatment of the disease should be replaced by a more holistic approach that may reduce the associated cardiovascular risks.

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Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Alabood, T. F. Y., Bahader, H. A. H. & Oleiwi, S. I. “Obstructive Sleep Apnea Syndrome: Cardiovascular Comorbidities and the Role of Collaborative ENT-Internal Medicine Care." *Sarcouncil Journal of Internal Medicine and Public Health* 5.2 (2026): pp 65-73.

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