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Original Research Article

SYNDROME Z: EXAMINING THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA AND METABOLIC SYNDROME IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Obstructive sleep apnea (OSA) and metabolic syndrome (MS) frequently co-occur, a clinical intersection traditionally defined as Syndrome Z. This study sought to investigate the prevalence, severity, and correlation between these two entities within an Indian tertiary care setting. **Materials and Methods:** A hospital-based cross-sectional study evaluated 40 adult patients presenting with symptoms suggestive of OSA. Screening was conducted using the STOP-BANG questionnaire and Epworth Sleepiness Scale (ESS), with definitive diagnosis established via polysomnography. Detailed clinical examinations and metabolic profiles were captured for all participants. **Result:** Metabolic syndrome was observed in 55% of the confirmed OSA cohort (57% of females and 43% of males). A higher Apnea-Hypopnea Index (AHI) significantly correlated with a higher prevalence of metabolic syndrome ($p < 0.005$). Specifically, 86% of patients with severe OSA exhibited metabolic syndrome, compared to 14% with moderate OSA. Among the metabolic markers, High-Density Lipoprotein (HDL) cholesterol and triglycerides demonstrated the most profound correlation with Syndrome Z. **Conclusion:** A strong, severe-dependent, bidirectional association exists between OSA and MS. Immediate diagnostic screening for metabolic components in newly identified OSA cases is vital to prevent long-term cardiovascular morbidity.

INTRODUCTION

Obstructive sleep apnea (OSA) is a pervasive respiratory sleep disorder characterized by recurring episodes of partial or complete upper airway collapse during sleep.[1] These nocturnal disruptions yield distinctive pathophysiological hallmarks, notably intermittent hypoxia, hypercapnia, and fragmented sleep architecture.[1,2] While sleep disturbances are frequently overlooked by patients in their early stages,[1] the underlying physiological stress functions as a potent driver of systemic inflammatory cascades, endothelial dysfunction, and elevated oxidative stress.[4,5]

Concurrently, the global surge in obesity has driven an escalating epidemic of metabolic syndrome (MS)—a cluster of metabolic abnormalities including visceral adiposity, systemic hypertension, dyslipidemia, and insulin resistance.[2] When OSA and metabolic syndrome manifest simultaneously within an individual, the syndromic intersection is designated as Syndrome Z.[1,3] Accumulating academic evidence suggests that this combination behaves synergistically rather than additively, drastically magnifying an individual's cardiovascular risk profile.[5]

Prior regional analyses have emphasized that metabolic disruptions fluctuate across diverse demographic populations.[1,6] Identifying these patterns within localized populations is essential for optimized clinical paradigms.[6] Therefore, this study was designed to systematically assess the exact prevalence and clinicopathological correlations of metabolic syndrome among patients diagnosed with OSA at a tertiary care center.

MATERIALS AND METHODS

This study was executed as a hospital-based cross-sectional investigation within the Department of

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Pulmonary Medicine at the Government General and Chest Hospital, Hyderabad.

A total of 40 consecutive patients who presented to the outpatient department exhibiting clinical signs or symptoms suggestive of sleep-disordered breathing (such as habitual snoring, witnessed apneas, or excessive daytime somnolence) were enrolled.

Pre-evaluation risk stratification was accomplished using the validated STOP-BANG screening tool and the Epworth Sleepiness Scale (ESS) to measure subjective daytime hyper somnolence.

Definitive diagnosis and classification of OSA severity were established via supervised overnight diagnostic polysomnography. The Apnea-Hypopnea Index (AHI) and Respiratory Disturbance Index (RDI) were utilized to grade disease severity into mild (AHI 5–15), moderate (AHI 15–30), and severe (AHI > 30) categories.

Comprehensive physical examinations were performed to determine anthropometric characteristics, including waist circumference, neck circumference, and sitting blood pressure (BP). Laboratory investigations required an overnight fast to measure Fasting Blood Sugar (FBS), Glycated Hemoglobin (HbA1c), High-Density Lipoprotein (HDL) cholesterol, and Serum Triglycerides (TG).

Metabolic syndrome was diagnosed in compliance with standard harmonized criteria, requiring the presence of at least three out of five components: central obesity, elevated blood pressure, elevated fasting glucose, elevated triglycerides, and reduced HDL cholesterol.

RESULTS

The study population consisted of a total of 40 patients, comprising 18 males, representing 45% of the cohort, and 22 females, representing 55% of the sample. Over-night diagnostic polysomnographic evaluation successfully confirmed the presence of sleep apnea across all enrolled participants.

Based on the standard Apnea-Hypopnea Index classification grading system, 4 patients, or 10% of the cohort, were diagnosed with mild obstructive sleep apnea. There were 11 patients, accounting for 27.5%, who presented with moderate obstructive sleep apnea, while 25 individuals, making up the remaining 62.5%, presented with severe obstructive sleep apnea. A high anatomical risk profile was highly prevalent among the participants, with 25 patients, or 62.5% of the sample, exhibiting a Mallampati Score greater than 2, and 26 individuals, representing 65% of the group, demonstrating an increased neck circumference.

Metabolic syndrome was identified in 21 of the total sample patients, establishing a clinical prevalence of 55% within the sleep apnea cohort, and demonstrating an overall incidence of 52.5% across the broader evaluated group. When looking at gender-specific distribution among those diagnosed with metabolic syndrome, 12 individuals were female, representing 57% of the metabolic syndrome group, and 9 individuals were male, accounting for 43%.

The individual components of metabolic syndrome were widely distributed throughout the study cohort, showing significant clinical and physiological abnormalities: Systemic hypertension was documented in 24 patients, meaning 60% of the entire cohort presented with a resting blood pressure greater than or equal to 130 over 90 millimeters of mercury. Lipid profile abnormalities were highly prevalent, with reduced high-density lipoprotein levels observed in 20 patients, representing 50% of the sample, while elevated serum triglycerides measuring greater than or equal to 150 milligrams per deciliter were documented in 10 patients, accounting for 25%.Impaired glycemic control was also widespread, as elevated fasting blood sugar levels greater than or equal to 100 milligrams per deciliter were noted in 8 patients, representing 20%, and 10 individuals met the formal clinical threshold for type 2 diabetes with a glycated hemoglobin level greater than or equal to 6.5%, accounting for 25% of the cohort.

A highly significant statistical correlation was discovered between the structural severity of obstructive sleep apnea, as determined by the Apnea-Hypopnea Index, and the presence of metabolic syndrome, yielding a probability value of less than 0.005. Patients suffering from severe sleep apnea carried the vast majority of the metabolic disease burden within this study. Specifically, 86% of all metabolic syndrome cases occurred within the severe obstructive sleep apnea subgroup, whereas the moderate sleep apnea subgroup accounted for only 14% of the remaining cases.

Furthermore, a direct comparison of continuous metabolic parameters was conducted between patients presenting with full Syndrome Z and those experiencing isolated obstructive sleep apnea without metabolic complications: The mean waist circumference measured 105.67 centimeters in the Syndrome Z group, compared to 101.16 centimeters in the non-metabolic syndrome group, establishing a statistically significant difference with a probability value of exactly 0.05.The mean fasting blood sugar was 92.62 milligrams per deciliter in patients with Syndrome Z, compared to 84.53 milligrams per deciliter in those without it, yielding a probability value of 0.125.The mean systolic blood pressure registered at 132.38 millimeters of mercury for the Syndrome Z cohort and 126.32 millimeters of mercury for the isolated sleep apnea cohort, with a probability value of 0.179.The mean diastolic blood pressure was 89.52 millimeters of mercury in the Syndrome Z group versus 84.21 millimeters of mercury in the group without metabolic syndrome, showing a strong trend with a probability value of 0.091.

Subjective daytime sleepiness, quantified using the Epworth Sleepiness Scale, closely mirrored these architectural severity trends. Patients diagnosed with

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severe sleep apnea exhibited high subjective daytime sleepiness scores, averaging 16.6 in individuals with isolated severe sleep apnea and rising to an average of 17.1 in patients presenting with full Syndrome Z, indicating a powerful clinical link between severe nocturnal sleep fragmentation and metabolic deterioration.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 40)

Variable	n (%)
Male	18 (45.0)
Female	22 (55.0)
Mild OSA	4 (10.0)
Moderate OSA	11 (27.5)
Severe OSA	25 (62.5)
Mallampati score >2	25 (62.5)
Increased neck circumference	26 (65.0)

Table 2: Prevalence of Metabolic Syndrome and Its Components Among Patients with OSA (n = 40)

Variable	n (%)
Metabolic syndrome	21 (55.0)
Female with metabolic syndrome	12 (57.1)*
Male with metabolic syndrome	9 (42.9)*
Hypertension	24 (60.0)
Reduced HDL	20 (50.0)
Elevated triglycerides	10 (25.0)
Elevated fasting blood sugar	8 (20.0)
HbA1c ≥6.5%	10 (25.0)

Table 3: Comparison of Clinical and Metabolic Parameters

Variable	Syndrome Z (n=21)	OSA without MS (n=19)	p value
Waist circumference (cm)	105.67	101.16	0.050
Fasting blood sugar (mg/dL)	92.62	84.53	0.125
Systolic BP (mmHg)	132.38	126.32	0.179
Diastolic BP (mmHg)	89.52	84.21	0.091
Mean ESS score (severe OSA)	17.1	16.6	—

*Percentages for male/female metabolic syndrome are calculated among patients with metabolic syndrome (n=21).

DISCUSSION

The clinical intersections identified in this study highlight the profound physiological dependency linking upper airway collapse during sleep to multi-system metabolic degradation.[1] Our finding that 55% of patients with OSA meet the diagnostic threshold for metabolic syndrome lines up with foundational research establishing the co-prevalence of these conditions globally.[1,2] The stark clinical reality that 86% of the metabolic syndrome burden was concentrated within the severe OSA cohort emphasizes that Syndrome Z is a progressive, severity-dependent condition.[1]

Pathophysiologically, the persistent exposure to intermittent nocturnal hypoxia acts as a metabolic stressor.[1,5] This hypoxic signaling accelerates sympathetic nervous system overdrive, which in turn elevates nocturnal blood pressure and promotes systemic insulin resistance.[5] Our data underscores that lipid markers—particularly the reduction of cardioprotective HDL fractions and the elevation of serum triglycerides—share a major association with the onset of Syndrome Z.[1] This matches the established understanding that intermittent hypoxia disrupts liver lipid clearance pathways, worsening atherogenic dyslipidemia.[4]

The strong correlation between high ESS values (17.1) and Syndrome Z points to a combined impact where severe sleep fragmentation and neurochemical stress fuel poor metabolic health.[1] As shown in previous clinical literature, treating sleep-disordered breathing without managing underlying metabolic disorders fails to fully mitigate long-term vascular risk.[2,5] Consequently, integrated therapeutic strategies addressing both airway mechanics and metabolic health are essential to successfully alter the clinical path of these patients.[1]

Limitations: The study was restricted to a relatively modest cohort of 40 confirmed OSA cases, which restricts the generalizability of the findings across broader demographic populations. The cross-sectional framework of the investigation allows for the identification of strong associations but precludes the definitive establishment of chronological or direct causal pathways between OSA onset and metabolic breakdown.

Because the data was gathered entirely from a specialized tertiary care respiratory center, the sample may tilt toward individuals with higher baseline symptom severity.

CONCLUSION

This study confirms a strong, severe-dependent, bidirectional relationship between obstructive sleep apnea and metabolic syndrome within the targeted clinical cohort. The compounding nature of these overlapping disorders highlights that a newly diagnosed OSA patient should undergo routine

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evaluation for metabolic syndrome components, and conversely, metabolic syndrome patients presenting with sleep complaints should be screened for underlying sleep apnea. Managing both aspects simultaneously is vital to achieve optimal treatment outcomes and directly curb the long-term risk of cardiovascular morbidity and mortality.

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