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SEVERE OBSTRUCTIVE SLEEP APNEA IN A PATIENT WITH OBESITY: A CLINICAL CASE REPORT OF CARDIOMETABOLIC INTERACTIONS

KEY WORDS: obstructive sleep apnea; obesity; cardiometabolic; BiPAP

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ABSTRACT

Obstructive sleep apnea (OSA) is a highly prevalent yet underdiagnosed sleep-related breathing disorder with well-established cardiometabolic consequences. We present a case of a 59-year-old female with obesity, type 2 diabetes mellitus, and hypertension who presented with acute breathlessness. Clinical evaluation revealed a critically elevated neck circumference of 49 cm, an Epworth Sleepiness Scale score of 16/24, and a STOP-BANG score of 7/8, collectively indicating high-risk OSA. Overnight polysomnography confirmed severe OSA with an apnea-hypopnea index (AHI) of 33.7 events per hour and a minimum nocturnal oxygen saturation of 75%. Multi-system assessment demonstrated cardiomegaly, hepatomegaly with portal vein dilatation, dependent ground-glass opacities on HRCT thorax, and grade I hypertensive retinopathy. Bilevel positive airway pressure (BiPAP) therapy was initiated alongside a structured multidisciplinary management plan. This case illustrates the bidirectional relationship between obesity and OSA, the cumulative cardiometabolic burden of untreated sleep-disordered breathing, and the importance of systematic multi-organ assessment. Early identification through validated screening tools and prompt initiation of positive airway pressure therapy are essential in preventing progressive end-organ damage in this high-risk population.

INTRODUCTION

Obstructive sleep apnea is defined as recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in apneas and hypopneas accompanied by oxygen desaturation and sleep fragmentation. It affects an estimated 9 to 38 percent of the adult population, with a markedly elevated prevalence among individuals with obesity, approaching 70 percent in this group. A 10 percent increase in body weight raises OSA risk by approximately 10 percent, underscoring the mechanistic relationship between adiposity and airway collapsibility [1,2].

Despite its high disease burden, OSA remains significantly underdiagnosed in routine clinical practice. The condition independently contributes to systemic hypertension, cardiac arrhythmias, pulmonary hypertension, insulin resistance, and non-alcoholic fatty liver disease — collectively constituting the cardiometabolic syndrome of sleep apnea. Chronic intermittent hypoxia perpetuates oxidative stress, sympathetic nervous system over-activation, and systemic inflammation, mechanisms that share and amplify those underlying metabolic disease [3,6].

The present case illustrates the co-existence of severe OSA with multiple cardiometabolic comorbidities in an obese female patient, highlighting the need for heightened clinical suspicion, structured screening, and a coordinated multidisciplinary approach.

CASE STUDY

A 59-year-old female homemaker with a documented history of type 2 diabetes mellitus and systemic hypertension presented to the Department of Medicine, D.Y. Patil Hospital, Navi Mumbai, with a two-day history of progressive breathlessness. The patient denied chest pain, syncope, orthopnoea, or paroxysmal nocturnal dyspnoea. However, on directed history-taking, she reported longstanding loud snoring, witnessed apnoeic episodes by household members, excessive daytime somnolence, and recurrent morning headaches persisting over several months.

On general examination, the patient was afebrile and hemodynamically stable: pulse rate 96 beats per minute, blood pressure 130/70 mmHg, respiratory rate 18 per minute, and peripheral oxygen saturation 98% on room air. Body mass index was 30 kg/m². Neck circumference measured 49 cm, considerably exceeding the established 43 cm threshold for high OSA risk in women.

Systemic examination demonstrated bilateral basal crepitations on respiratory auscultation, hepatomegaly at 17.6 cm on abdominal examination, and normal heart sounds without murmurs.

Functional assessment using validated clinical instruments yielded an Epworth Sleepiness Scale (ESS) score of 16/24, confirming significant excessive daytime sleepiness, and a STOP-BANG score of 7/8, placing the patient in the high-risk OSA category. Current medications included telmisartan-amlodipine 40/5 mg and metoprolol succinate 25 mg for hypertension; glycaemic management was ongoing but suboptimal as corroborated by laboratory investigations.

INVESTIGATIONS

Random blood glucose was 278 mg/dL, indicating poorly controlled diabetes mellitus. Electrocardiogram demonstrated normal sinus rhythm. Chest radiograph showed cardiomegaly with an increased cardiothoracic ratio. Abdominal ultrasonography confirmed hepatomegaly at 17.6 cm with portal vein dilatation of 15 mm, suggesting metabolic-associated hepatic disease with portal hypertension. HRCT thorax revealed dependent ground-glass opacities and mild cardiomegaly. Ophthalmological review identified grade I hypertensive retinopathy bilaterally. ENT assessment showed Mallampati Class I, mild rightward nasal septal deviation, and no tonsillar hypertrophy.

Overnight polysomnography confirmed severe obstructive sleep apnea. Findings are summarised in Table 1.

Table 1: Polysomnography Parameters and Clinical Scores

Parameter	Result / Interpretation
Apnea-Hypopnea Index (AHI)	33.7 events/hr — Severe OSA (normal<5)
Respiratory Disturbance Index (RDI)	33.2
Minimum Nocturnal SpO_2	75% — Clinically significant desaturation
Epworth Sleepiness Scale (ESS)	16/24 — Excessive daytime sleepiness
STOP-BANG Score	7/8 — High risk for OSA

Source: Polysomnography report, D.Y. Patil Hospital,Navi Mumbai

MANAGEMENT

Following polysomnographic confirmation of severe OSA

with clinically significant nocturnal desaturation, bilevel positive airway pressure therapy was initiated with an inspiratory positive airway pressure of 12 cm H O and an expiratory positive airway pressure of 8 cm H O. Continuous overnight monitoring was carried out to evaluate patient tolerance and treatment efficacy.

A comprehensive multidisciplinary management plan was formulated: continuation of antihypertensive pharmacotherapy; endocrinological consultation for optimisation of glycaemic control; cardiological follow-up for surveillance of cardiomegaly and fluid status; and BiPAP compliance monitoring. Structured weight reduction was prescribed as the cornerstone long-term intervention. Adjunct measures included dietary counselling, sleep hygiene education, and positional therapy. Repeat polysomnography was planned following significant weight loss to assess therapeutic response.

DISCUSSION

This case exemplifies the complex, bidirectional relationship between obesity and OSA. Excess adiposity promotes upper airway fat deposition and reduces functional residual capacity, increasing airway collapsibility during sleep. The resulting intermittent hypoxia exacerbates metabolic dysregulation through oxidative stress, sympathoadrenal activation, and adipokine imbalance, creating a self-perpetuating cardiometabolic cycle [6,7].

The critically elevated neck circumference of 49 cm — substantially above the 43 cm predictive threshold in women — served as an important clinical discriminator. Evidence consistently identifies neck circumference as an independent predictor of OSA severity, distinct from but complementary to overall BMI assessment.

The multi-system involvement reflects the systemic consequences of prolonged untreated sleep-disordered breathing compounded by uncontrolled metabolic comorbidities. Cardiomegaly and ground-glass opacities suggest chronic cardiopulmonary remodelling; hepatomegaly with portal vein dilatation points toward metabolic-associated steatotic liver disease; grade I hypertensive retinopathy confirms established microvascular end-organ damage [3,4,9].

The STOP-BANG score of 7/8 affirms the diagnostic value of this validated screening instrument. Current evidence recommends immediate polysomnography referral for scores of five or above in metabolic syndrome patients — a threshold substantially exceeded here [5]. Weight reduction remains the most impactful long-term intervention, with data demonstrating a 30 to 50 percent reduction in AHI per 10 percent weight loss [8]. BiPAP adherence and structured multidisciplinary surveillance form the complementary therapeutic pillars.

CONCLUSIONS

This case underscores the critical importance of proactive screening for OSA among obese patients with concurrent cardiometabolic disease. Systematic use of validated clinical instruments, timely polysomnography, and early positive airway pressure therapy are pivotal in preventing progressive end-organ damage. A coordinated multidisciplinary strategy integrating respiratory, cardiovascular, endocrinological, and dietary care remains essential for optimising long-term outcomes in this clinically complex population.

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