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Prevalence of Obstructive Sleep Apnea Among Patients with Secondary Polycythemia: A Retrospective Cross-sectional Study

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ABSTRACT

Objectives: Obstructive sleep apnea (OSA) and secondary polycythemia occasionally occur together, but their relationship remains unclear. This study aimed to explore the prevalence of OSA among patients with secondary polycythemia in our local population. ***Methods***: In this retrospective cross-sectional study, we screened 524 adult patients with polycythemia who attended the hematology clinic at a major tertiary hospital in Muscat during 2013–2023. Our goal was to identify patients with OSA and secondary polycythemia who also underwent sleep studies and analyzed their data. ***Results:*** Out of 524 polycythemia patients, we identified N = 44 patients (8.4%) who had both OSA and secondary polycythemia. The vast majority were male (42/44; 95.5%). Participants' mean age was 40.7 years, with a body mass index of 31.7 kg/m^2, hemoglobin level of 16.5 g/dL, and hematocrit level of 0.5 L/L. The mean apnea/hypopnea index was 33.3, and the mean desaturation index was 20.3. More than half of the patients (23; 52.3%) had severe OSA (apnea/hypopnea index > 30). ***Conclusions:*** The majority of patients with secondary polycythemia had severe OSA. It is likely that OSA may contribute to secondary polycythemia, warranting further investigation.

Obstructive Sleep Apnea (OSA) is a sleep disorder characterized by repetitive interruptions of breathing during sleep.[1,2] OSA with daytime sleepiness is estimated to affect about 3–7% of adult men and 2–5% of adult women, with higher prevalence among groups such as the elderly and those with high body mass index (BMI).[2] OSA results from upper airway collapse and is often associated with hypoxia, fragmented sleep, and daytime somnolence.[3] Chronic OSA can lead to substantial systemic complications, ranging from cardiovascular and metabolic diseases to neurocognitive impairments.[4,5]

Secondary polycythemia is a less recognized but clinically significant sequela of OSA.[6] This condition, characterized by an abnormal increase in red blood cell (RBC) mass, often results from chronic hypoxemia—a central feature of OSA. During apneic episodes, intermittent oxygen desaturation triggers a compensatory increase in erythropoietin production, which in turn stimulates erythropoiesis.[7] While this adaptive response is aimed at improving oxygen delivery, the resultant polycythemia can increase blood viscosity, increasing the risk of thrombotic complications.[8]

Red cell distribution width, a measure of variability in the size of RBCs, is often elevated in OSA, potentially reflecting the body's response to chronic intermittent hypoxia.[9] Red cell distribution is a helpful parameter in differentiating between secondary and primary erythrocytosis.[10]

Despite the clear physiological link, the relationship between OSA and secondary polycythemia remains underexplored. Understanding the prevalence, pathophysiology, and clinical implications of this association is essential for early diagnosis and tailored management. Moreover, effective treatment of OSA, particularly with continuous positive airway pressure (CPAP), may have the potential to mitigate polycythemia.

This study aims to delve into the interplay between OSA and secondary polycythemia by assessing the

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prevalence and severity of OSA in patients with secondary polycythemia. Our findings emphasize the need for comprehensive diagnostic and treatment strategies to prevent downstream complications such as thrombosis.

METHODS

A hospital-based retrospective cross-sectional study was conducted among patients attending a hematology clinic in Sultan Qaboos University Hospital, a tertiary teaching hospital in Muscat. The study included all patients who attended the hematology clinic with polycythemia and underwent sleep studies between 2013 and 2023. The study was approved by the ethical committee at the College of Medicine and Health Sciences, Sultan Qaboos University (MREC#3075).

The inclusion criteria were as follows: adult patients (≥ 18 years) diagnosed with secondary polycythemia (elevated RBC count and hemoglobin/hematocrit (Hct) levels) who underwent a sleep study as part of investigations into an underlying condition, such as bone marrow abnormality.[11] The exclusion criteria comprised: primary polycythemia,[12] smoking, chronic obstructive pulmonary diseases, renal failure, blood cancer, and patients living at high altitudes.

Patient data was collected using the hospital's TrackCare system. The retrieved demographic data included sex, age, BMI, and blood pressure. Hematological parameters were determined from complete blood count, hemoglobin, hematocrit, mean cell volume, and mean cell hemoglobin.

We also evaluated sleep study data, including the apnea-hypopnea index, baseline oxygen saturation below 90%, desaturation index (DI), and the patient's subjective report of daytime sleepiness evaluated using the Epworth Sleepiness Scale.

The data was analyzed using the IBM SPSS Statistics (IBM Corp. IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp; 2020). Continuous variables were presented as means ± SD and categorical variables as frequencies and percentages. The Chi-square test was used to examine the significance of associations.

RESULTS

After screening 524 patients with polycythemia, 44 patients with secondary polycythemia who had undergone a sleep study were identified and selected for the study. Figure 1 shows the selection process. The patients were overwhelmingly male (42; 95.5%), with only two women. The mean age of the cohort was 40.7 ± 10.6 years, with a mean BMI of 31.7 ± 6.3 kg/m^2 [Table 1]. All 44 patients met the diagnostic criteria for secondary polycythemia, and had a mean hemoglobin of 16.5 ± 1.3 g/dL and a mean Hct of 0.5% [Table 1].

Most participants (52.3%) had severe OSA (mean apnea-hypopnea index: 33.3 ± 23.2), followed by moderate (18.2%) and mild (29.6%) OSA [Figure 2]. Patients with an oxygen saturation index below 90% had a mean OSA of 47.7 ± 24.5 (p = 0.036) [Table 2]. Kurskal-Wallis test revealed that higher Hct levels were significantly associated with severe OSA (p = 0.036) [Figure 3]. Nevertheless, there was no statistically significant association between the severity of OSA and other hematological parameters.

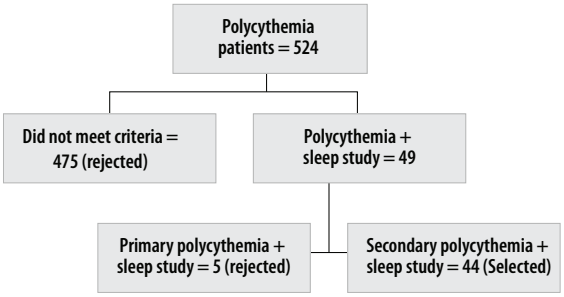


Figure 1: Flow chart of the selection process for secondary polycythemia patients who underwent sleep study.

Table 1: Demographic and hematological characteristics of patients with secondary polycythemia (N = 44).

Variables	Mean ± SD
Age, years	40.7 ± 10.6
Body mass index, kg/m^2	31.7 ± 6.3
Systolic blood pressure, mmHg	137.0 ± 16.0
Diastolic blood pressure, mmHg	84.0 ± 11.0
Red blood cells, ×106 /µL	6.3 ± 0.9
Hemoglobin, g/dL	16.5 ± 1.3
Hematocrit, %	0.5 ± 0.04
Mean cell volume, fL	80.0 ± 7.8
Mean cell hemoglobin, pg	27.7 ± 9.3
White blood cells, ×103 /µL	6.7 ± 1.9
Platelets, ×103 /µL	263.7 ± 109.1

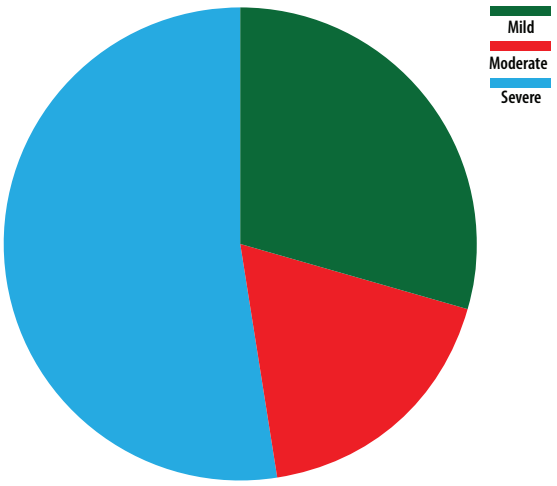
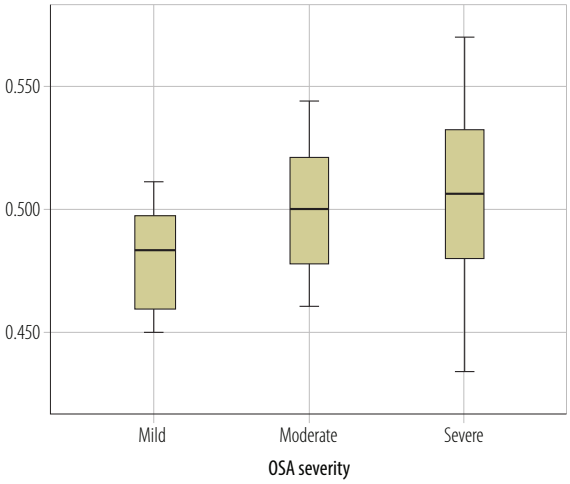


Figure 2: Distribution of severity of obstructive sleep apnea among patients with secondary polycythemia (N = 44).



ANOVA: Analysis of Variance; AHI: Apnea-hypopnea index.

Figure 3: Analysis of Variance comparison of hematocrit levels (g/dL) across three categories of obstructive sleep apnea (OSA). Hematocrit levels are seen significantly higher in patients with severe OSA (Apnea-hypopnea index (AHI > 30) compared to those with mild OSA (AHI = 5–15) and moderate OSA (AHI = 15–30); $p = 0.036$.

Table 2: Characteristics of OSA in patients with secondary polycythemia (N = 44).

Characteristics	Mean ± SD
Epworth sleepiness scale score	11 ± 5
Apnea-hypopnea index, events/hour	33.3 ± 23.2
Baseline SpO_2, %	94.3 ± 3.4
Minimum SpO_2, %	81.1 ± 10.1
Time with SpO_2 below 90%, % of total sleep time	47.7± 24.5
Overall desaturation index	20.3 ± 85.2

OSA: obstructive sleep apnea; SpO_2: peripheral oxygen saturation.

Table 3 presents the analysis of variance assessing the association between Hct levels and the severity of OSA.

DISCUSSION

This study assessed the prevalence and severity of OSA among patients with secondary polycythemia. The majority of patients in the study (70.5%) had moderate to severe OSA, which was accompanied by elevated Hct levels. Our results suggest that greater severity of sleep-disordered breathing may have contributed to increased Hct levels, which may have triggered erythrocytosis. This points to a potential correlation between the severity of sleep-disordered breathing and the degree of erythrocytosis. Previous research shows that characteristic intermittent hypoxia associated with OSA triggers erythropoiesis through increased erythropoietin production, leading to increased RCB mass and Hct level.[7,8,13] Our results support the existing recommendation that patients presenting with unexplained secondary polycythemia should be screened for OSA.

Several studies have indicated a relationship between OSA severity and Hct levels via increased

Table 3: ANOVA of the relationship between hematocrit level and severity of OSA in patients with secondary polycythemia (n = 39).

OSA severity	Patients (n)	Hematocrit (g/dL) mean ± SD (min–max)	Std. error	95% CI for mean lower bound–upper bound	*p*-value (ANOVA)
Mild	12	0.48 ± 0.02 (0.45–0.51)	0.006	0.47–0.49	0.036
Moderate	7	0.50 ± 0.03 (0.46–0.54)	0.010	0.47–0.53	
Severe	20	0.51 ± 0.04 (0.43–0.57)	0.008	0.49–0.53	
Total	39	0.50 ± 0.03 (0.43–0.57)	0.005	0.49–0.51	

ANOVA: analysis of variance; OSA: obstructive sleep apnea.

erythropoietin production.[13,14] Our results also emphasize the diagnostic value of sleep study in patients with secondary polycythemia, particularly those with risk factors such as obesity, male sex,[15] and a history of excessive daytime sleepiness.

Additionally, we observed a trend in this cohort towards higher BMI and greater neck circumference, both known risk factors for OSA.[16] This indicates the multifactorial nature of polycythemia in these patients, where obesity-related hypoventilation may have further exacerbated erythrocytosis.[17] The benefit of CPAP therapy in reversing polycythemia has been well-documented, and our findings further emphasize the need for early diagnosis and treatment of OSA in such cases.

Despite the strengths of our study, including a well-defined patient cohort and objective sleep study assessments, several limitations must be acknowledged. First, this was a single-center study with a relatively small sample size, limiting generalizability. Second, the cross-sectional design precluded assessment of causality over the long term. The retrospective nature of data also had potential limitations. Future research should aim to explore the long-term impact of CPAP therapy on Hct levels in secondary polycythemia patients with moderate and severe OSA, as well as the potential genetic predispositions to OSA-related erythrocytosis.

CONCLUSION

Our study highlights the prevalence of OSA among secondary polycythemia patients diagnosed at a single center in Oman, emphasizing the importance of routine OSA screening in patients presenting with unexplained erythrocytosis. Early identification and treatment of OSA in this population may not only improve their sleep-related quality of life but also reduce hematologic consequences, including the risk of thromboembolic complications associated with chronic hypoxia. It is possible that OSA may contribute to secondary polycythemia in some patients, which warrants further investigation.

Disclosure

The authors declare no conflict of interest. No funding was received for this study.

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