EUR REV MED PHARMACOL SCI
2026; 30: 96-104

FACTORS ASSOCIATED WITH HYPOXIA AND SEVERITY IN PATIENTS WITH BOTH OBSTRUCTIVE SLEEP APNEA SYNDROME AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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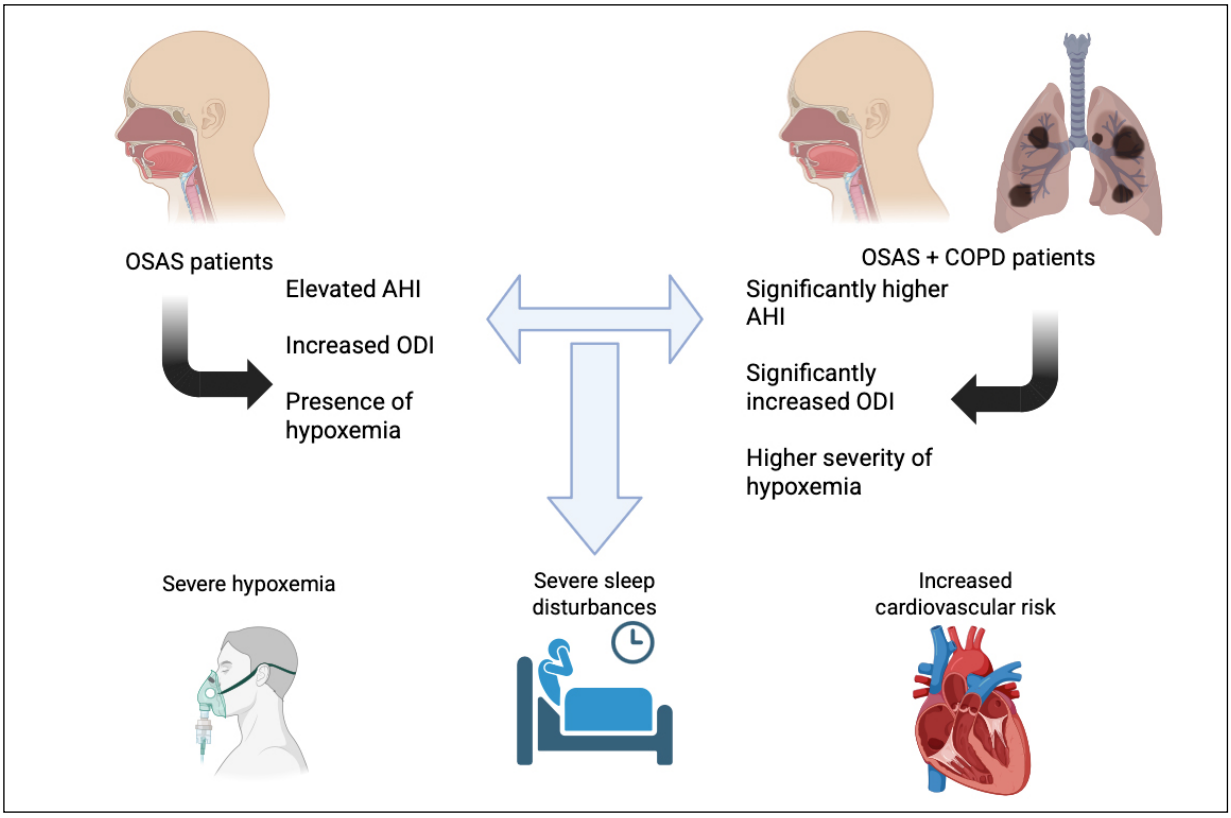
ABSTRACT – *Objective:* The frequent co-occurrence of obstructive sleep apnea syndrome (OSAS) and chronic obstructive pulmonary disease (COPD) gives rise to an overlap syndrome associated with a decline in patients' health. Discerning the nuanced clinical presentations, polygraphy data, and hematological profiles of individuals with isolated OSAS from those with comorbid OSAS and COPD is crucial for tailoring effective therapeutic strategies. The present investigation seeks to delineate the demographic, clinical, polygraphy, and hematological features that differentiate patients with OSAS alone from those with concurrent OSAS and COPD, and further to evaluate the independent predictive value of COPD for OSAS severity.

Materials and Methods: In this cross-sectional analysis, 68 adults diagnosed with OSAS by overnight polygraphy were classified into two groups: OSAS-only (n = 49) and OSAS plus COPD (overlap syndrome, n = 19). Collected data encompassed demographics, comorbidities, sleep indices [including apnea-hypopnea index (AHI), oxygen desaturation index (ODI), and percentage of sleep time with oxygen saturation < 90% (t90%)], and routine hematological examinations. Group comparisons used appropriate statistical tests, and multivariable logistic regression evaluated predictors of severe OSAS.

Results: Compared with patients who had OSAS alone, those with overlap syndrome exhibited more severe sleep-disordered breathing and nocturnal desaturation: median AHI was higher (55.80 *vs.* 38.06; $p = 0.01$), ODI was increased (57.50 *vs.* 41.10; $p = 0.03$), and t90% was longer (32.20% *vs.* 19.00%; $p = 0.03$). Mean corpuscular volume (MCV) was also greater in the COPD group ($p = 0.01$). While COPD showed a trend toward being associated with severe OSAS (odds ratio 2.18), this association did not reach statistical significance ($p = 0.36$). Hypertension was more prevalent among patients with overlap syndrome and can suggest an elevated cardiovascular risk in this subgroup.

Conclusions: The convergence of COPD and OSAS correlates with severe sleep apnea, intensified nocturnal hypoxemia, and an increased prevalence of hypertension, thereby highlighting the imperative for meticulous evaluation and personalized treatment paradigms in this patient population. Limitations inherent in this study, specifically the constrained sample size and cross-sectional methodology, necessitate circumspect interpretation of the findings and suggest the value of prospective, large-scale investigations to corroborate these observations.

KEYWORDS: Sleep apnea, Obstructive disease, Oxygenation, Hematological parameters, Overlap syndrome.



Graphical Abstract. Comparison between patients with obstructive sleep apnea syndrome (OSAS) and those with overlap syndrome (OSAS + COPD). Patients with concomitant COPD show higher apnea-hypopnea index (AHI), increased oxygen desaturation index (ODI), and more severe hypoxemia. These alterations are associated with more severe sleep disturbances and a potentially increased cardiovascular risk.

INTRODUCTION

The increasing recognition of obstructive sleep apnea syndrome (OSAS) as a multifaceted health disorder has stimulated considerable interest in deciphering its interactions with various comorbidities, particularly chronic obstructive pulmonary disease (COPD). Obstructive sleep apnea syndrome is marked by repeated collapse of the upper airway during sleep, causing fragmented sleep and intermittent drops in oxygenation. These disturbances can worsen preexisting pulmonary conditions, such as COPD. Also, studies showed that the combination of OSAS and COPD is frequently associated with greater disease burden and increased mortality, underlining the need for careful comparison of clinical signs, sleep study findings, and blood parameters between patients with both disorders and those with OSAS alone[1-3].

Key aspects of OSAS are the links to various comorbidities, such as cardiovascular diseases and metabolic syndrome, which are commonly observed in patients who have multiple health conditions. Scholars[4] have shown that individuals with OSAS often have notable clinical similarities with COPD, highlighting concerns about the combined impact on respiratory health and overall quality of life. Clinical profiles of individuals diagnosed with OSAS suggest that comorbidity prevalence, including COPD, leads to intensified respiratory and systemic complications[5], positioning this patient group at a heightened risk for cardiovascular

incidents and further health decline[6]. Polygraphic evaluations provide critical insights into the severity of sleep apnea and its implications on respiratory function. Findings[7] indicate that the intensity of OSAS correlates with the severity of nocturnal oxygen desaturation, and the presence of COPD complicates this interaction. The coexistence of both disorders can modify sleep architecture, leading to variable degrees of hypoxemia, differences in apnea-hypopnea indices, and other polygraphic outcomes among patients. Therefore, diagnostic approaches should be individualized and may require assessments beyond standard polygraphy to capture underlying pulmonary pathology[8]. Treatment typically involves a multifaceted strategy, such as behavioral interventions, continuous positive airway pressure (CPAP) therapy, and ongoing evaluation of health metrics. The complete blood count (CBC) is an easy, practical test that yields valuable hematological insights for these patients[9-11]. Certain CBC parameters – such as mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), the hematocrit-to-hemoglobin ratio, and platelet count – may have underappreciated potential in the management of OSAS, especially among obese individuals. Exploring this new hypothesis that incorporates these hematological markers could help clinicians better predict complications, tailor treatments more effectively, and improve overall patient outcomes[7-9]. A thorough analysis is essential regarding the clinical profiles, polygraphic data, and hematological parameters in patients with OSAS, with and without COPD. This approach will help identify the diagnostic and therapeutic challenges faced by healthcare providers managing these complex cases, ultimately leading to improved health results.

MATERIALS AND METHODS

This cross-sectional, retrospective observational study included 68 adult participants recruited from the Pulmonology Clinic at Mureș County Clinical Hospital from 1st of January to 31 December 2024, all of whom had been diagnosed with OSAS based on overnight polygraphy (PG). Participants were divided into two groups: those with OSAS alone (n = 49) and those with comorbid COPD and OSAS (n = 19). COPD diagnosis was confirmed according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD)[10] criteria, based on spirometry data indicating a post-bronchodilator FEV1/FVC ratio less than 0.70. This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent for research and educational purposes was routinely obtained from all patients at the time of admission to the Pulmonology Clinic. This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Mureș County Clinical Hospital, Târgu Mureș, Romania (approval No. 9696, 26 June 2025), which authorized the retrospective analysis of anonymized clinical data collected during routine care.

Inclusion criteria included adults aged 18-75 years with confirmed OSAS [apnea-hypopnea index (AHI)≥ 5 events/hour]. Exclusion criteria included other major pulmonary or cardiac diseases (for example, pulmonary fibrosis or heart failure), neurological conditions causing sleep problems, current use of sedative or hypnotic medications, or being unable to give informed consent. Demographic and lifestyle data – age, sex, height, weight, and smoking history – were collected through structured interviews and medical record reviews. Body mass index (BMI) was calculated as weight in kilograms divided by height squared in meters. Medical history recorded the presence of hypertension, type 2 diabetes mellitus (T2DM), and smoking (pack-years). Hypertension was defined as a prior diagnosis, active antihypertensive therapy, or measured blood pressure ≥ 140/90 mmHg; T2DM was defined as previously diagnosed or currently treated. All participants underwent overnight polygraphy (PG) following a standardized protocol consistent with Romanian recommendations, with monitoring of airflow, respiratory effort, and pulse oximetry (SpO_2). From these recordings, we derived the apnea-hypopnea index (AHI: apneas and hypopneas per hour), the oxygen desaturation index (ODI: number of ≥3% desaturation events per hour), and t90 % SpO_2 (the proportion of total sleep time with SpO_2 < 90%). The STOP-BANG screening tool (score range 0-8) was also completed. Fasting blood specimens were obtained to measure hematologic indices, including hemoglobin (HGB), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), platelet count, and derived ratios such as the hematocrit-to-hemoglobin ratio (HCT/HGB). Laboratory testing was performed on automated analyzers calibrated in accordance with standard procedures.

Statistical analysis

Descriptive statistics were used to summarize demographic, clinical, and laboratory data. Continuous variables were expressed as medians with interquartile ranges (IQR). Categorical variables were presented as counts and percentag-

es. Comparisons between groups employed the Mann-Whitney U test for continuous variables and the Chi-square or Fisher's exact test for categorical variables. Correlations between sleep indices and continuous variables (e.g., BMI, hematological parameters) were assessed using Spearman's rank correlation coefficient. Multivariate logistic regression analysis evaluated whether COPD independently predicted severe OSA (defined as AHI > 30), adjusting for age, BMI, smoking status, hypertension, and T2DM. Odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A p-value < 0.05 was considered statistically significant. Analyses were conducted using SPSS version 26 (IBM Corp., Armonk, NY, USA).

RESULTS

In this study, a total of 68 participants were divided into those with both OSAS and COPD (n = 19) and those with only OSAS (n = 49). Patients had a median age of 57 years overall, with the overlap (OSAS+COPD) group slightly younger at a median of 56 *vs.* 58 years in those with OSAS alone; this was not statistically significant (p = 0.31). Male patients comprised the majority in both groups (68.4% overlap *vs.* 51% in the OSAS-only group), but the difference was not statistically significant (p = 0.19). Both groups exhibited elevated BMIs – 36.33 in the overlap group and 36.80 in the OSAS-only group – without a meaningful difference (p = 0.30). Smoking status showed a significant divergence: 73.7% of the overlap patients were current smokers compared with just 16.3% of the OSAS-only patients (p < 0.001). High blood pressure (HBP) was prevalent, affecting all participants in the OSAS with COPD group (100%) and 65.3% of those without COPD (p < 0.001), indicating an increased cardiovascular load with the presence of both conditions. T2DM did not differ significantly across groups, affecting 25% of the study participants (p = 0.27) (Table I).

The severity of sleep-related breathing disturbances was greater in participants with both OSAS and COPD. Compared with individuals who had OSAS only, participants with both OSAS and COPD demonstrated significantly higher indices of severity: AHI median 55.80 *vs.* 38.06 (p = 0.01), ODI 57.50 *vs.* 41.10 (p = 0.03), and a longer percentage of sleep spent under 90% oxygen saturation (t90% 32.20% *vs.* 19.00; p = 0.03), all pointing to more severe nocturnal hypoxemia in the overlap group. Interestingly, the STOP-BANG scores, used as an initial screening tool, were similar across both groups (p = 0.50), reflecting their limited sensitivity in distinguishing severity influenced by concurrent COPD (Table II).

Table I. Demographic characteristics of patients.

	Total (n=68)	OSAS + COPD (n=19)	OSAS w/o COPD (n=49)	*p*
Age	57 (51-66)	56 (47-63)	58 (51-68)	0.31*
Male gender	38 (55.9%)	13 (68.4%)	25 (51%)	0.19**
BMI	36.53 (31.32-42.12)	36.33 (32.40-38.30)	36.80 (30.76-44.07)	0.30*
Smoker status	22 (32.4%)	14 (73.7%)	8 (16.3%)	**<0.001****
HBP	51 (75%)	19 (100%)	32 (65.3%)	**<0.001*****
T2DM	17 (25%)	3 (15.8%)	14 (28.6%)	0.27**

*Mann-Whitney U test; **Chi-square test; ***Fisher exact test; HBP – high blood pressure; T2DM – type 2 diabetes mellitus; OSAS – obstructive sleep apnea syndrome; w/o – without; COPD – chronic obstructive pulmonary disease.

Table II. Sleep indices.

	Total (n=68)	OSAS + COPD (n=19)	OSAS w/o COPD (n=49)	*p*
AHI	40.90 (25.10-58.37)	55.80 (28.20-78.50)	38.06 (24.75-53.20)	**0.01***
ODI	44.85 (25.00-65.12)	57.50 (44.60-67.70)	41.10 (23.95-62.85)	**0.03***
t90%	24.30 (8.35-53.35)	32.20 (11.00-50.90)	19.00 (6.05-55.90)	**0.03***
STOP-BANG questionnaire	6 (5-7)	6 (5-7)	6 (5-6)	0.50

*Mann-Whitney U test; AHI – apnea/hypopnea index; ODI – oxygen desaturation index; t90% - time spent under 90% saturation; OSAS – obstructive sleep apnea syndrome; w/o – without; COPD – chronic obstructive pulmonary disease.

The study further evaluated hematological parameters and found no significant differences in hemoglobin or hematocrit levels between the groups (p = 0.19 and p = 0.09, respectively). However, the mean corpuscular volume (MCV) was significantly higher in individuals with OSAS and COPD (91.30 compared to 88.30; p = 0.01), suggesting possible macrocytic changes even in the context of chronic lung disease, potentially due to hypoxia-induced mechanisms (Table III).

The correlation analysis highlighted the impact of BMI, which showed weak to moderate positive correlations with indices of sleep apnea severity (AHI: r = 0.279, p = 0.021; ODI: r = 0.441, p < 0.001; t90%: r = 0.549, p < 0.001), indicating that increased BMI exacerbates respiratory issues during sleep. The STOP-BANG questionnaire strongly correlated with AHI (r = 0.716, p < 0.001) and ODI (r = 0.546, p < 0.001), confirming its utility in predicting apnea severity, albeit less for t90%. Finally, the hematocrit-to-hemoglobin (HCT/HGB) ratio showed no significant correlation with sleep indices, suggesting that red blood cell indices may not directly affect or reflect the severity of sleep-related breathing disorders in this cohort (Table IV).

In a multivariable model adjusting for potential confounders, the presence of COPD was associated with approximately twofold higher likelihood of severe OSAS (OR 2.18), but this relationship did not reach statistical significance (p = 0.36), with a wide confidence interval (95% CI 0.405-11.712). Likewise, age, BMI, smoking status, hypertension, and type 2 diabetes mellitus showed no independent, statistically significant associations with OSAS severity in the adjusted analysis. While there was a trend suggesting that COPD might contribute to more severe sleep apnea, the lack of statistical significance indicates that COPD alone may not be an independent predictor in this cohort (Table V).

DISCUSSIONS

The study offers a detailed comparison of demographic, clinical, sleep polygraphy, and hematologic features between patients with combined OSAS and COPD and those with OSAS alone[12]. Overall median age was 57 years, and there was no meaningful age difference between groups (56 *vs.* 58; p = 0.31). Men were more common in both cohorts (68.4% in the overlap group *vs.* 51% in the OSAS-only group), though this difference was not statistically significant (p = 0.19)[13]. Smoking status differed sharply: 73.7% of the OSAS-only patients were current smokers compared with just 16.3% of the overlap group (p < 0.001), a gap that may reflect smoking cessation following a COPD diagnosis or different underlying risk factors[14]. However,

Table III. Bloodwork parameters.

	Total (n=68)	OSAS + COPD (n=19)	OSAS w/o COPD (n=49)	*p*
Hemoglobin	14.05 (13.20-15.07)	14.50 (13.50-15.50)	13.90 (12.55-14.85)	0.19*
MCHC	33.00 (32.42-33.50)	32.90 (32.70-33.40)	33.20 (32.25-33.60)	0.83*
MCV	89.25 (86.50-91.30)	91.30 (88.60-95.00)	88.30 (84.70-90.95)	**0.01***
Hematocrit	43.20 (40.62-45.87)	44.40 (41.70-47.10)	43.10 (38.65-45.35)	0.09*
Platelets	229.50 (196.50-262.50)	231 (210-261)	228 (193-264)	0.68*
HCT/HGB	3.040 (2.988-3.104)	3.048 (3.000-3.086)	3.040 (2.985-3.133)	0.99

*Mann-Whitney U test; MCHC – mean corpuscular hemoglobin concentration; MCV – mean corpuscular volume; HCT/HGB – hematocrit over hemoglobin ratio; OSAS – obstructive sleep apnea syndrome; w/o – without; COPD – chronic obstructive pulmonary disease.

Table IV. Spearman correlations.

	AHI	ODI	t90%
Body mass index	***r*=0.279, *p*=0.021**	***r*=0.441, *p*<0.001**	***r*=0.549, *p*<0.001**
Stop Bang Questionnaire	***r*=0.716, *p*<0.001**	***r*=0.546, *p*<0.001**	***r*=0.226, *p*=0.04**
HCT/HGB	*r*=0.096, *p*=0.438	r=0.095, *p*=0.442	*r*=0.063, *p*=0.610

*Spearman correlation; AHI – apnea/hypopnea index; ODI – oxygen desaturation index; t90% - time spent under 90% saturation; AHI – apnea/hypopnea index; ODI – oxygen desaturation index; t90% - time spent under 90% saturation; HCT/HGB – hematocrit over hemoglobin ratio.

Table V. Multivariate regression model.

	B	S.E.	Wald	*p*	adjOR	CI 95%
Age	0.022	0.027	0.674	0.41	1.022	(0.970-1.078)
Body mass index	0.066	0.039	2.882	0.09	1.068	(0.990-1.152)
Smoker status	-0.005	0.763	0.000	0.99	0.995	(0.223-4.435)
HBP	-0.315	0.716	0.194	0.66	0.730	(0.179-2.971)
T2DM	1.028	0.748	1.888	0.16	2.796	(0.645-12.122)
COPD	0.778	0.858	0.821	0.36	2.18	(0.405-11.712)

HBP – high blood pressure; T2DM – type 2 diabetes mellitus; COPD – chronic obstructive pulmonary disease; adjOR – adjusted odds ratio; CI 95% – 95% confidence interval.

it is critical to address the limitations of our study, particularly the lack of assessment regarding the smoking history of the patients, including the duration, quantity, and intensity of smoking, as well as any cessation attempts. Hypertension was markedly more frequent among those with both conditions (100% *vs.* 65.3%; $p < 0.001$), underscoring a substantial cardiovascular burden in the overlap population and mirroring prior reports of elevated cardiac risk in these patients[15-17]. In contrast, type 2 diabetes mellitus showed no significant between-group difference (affecting 25% overall; $p = 0.27$). Sleep study metrics revealed more severe disordered breathing in the overlap group, with a higher median AHI (55.80 *vs.* 38.06; $p = 0.01$), consistent with greater nocturnal respiratory compromise when COPD coexists with OSAS. This finding is corroborated by other studies[11,12] that have found severe hypoxemia and nocturnal desaturation more prevalent in patients with overlap syndrome. The ODI showed a similar trend, with a value of 57.50 in the OSAS with COPD group compared with 41.10 in the OSAS-only group ($p = 0.03$). Greater time spent with oxygen saturation below 90% (t90%) in the OSAS with COPD group (32.20% *vs.* 19.00%; $p = 0.03$) further underscores the detrimental effects of these coexisting conditions on nocturnal oxygenation, consistent with patterns documented in prior research[13]. In terms of hematological parameters, although no significant differences were observed in hemoglobin or hematocrit levels ($p = 0.19$ and $p = 0.09$, respectively), MCV was significantly elevated in the OSAS with COPD group (91.30 *vs.* 88.30), suggesting macrocytic changes possibly driven by chronic hypoxia[12,14]. This finding aligns with existing literature that suggests chronic intermittent hypoxia associated with OSAS may influence erythropoiesis, differentiating it from the chronic sustained hypoxia typically seen in COPD patients[15]. The clinical significance of this change may vary considerably depending on the underlying hematological conditions of the patients being studied. This nuanced approach acknowledges the complexity of hematological responses in patients with OSAS and COPD, emphasizing the need for individualized clinical assessment. Additionally, the observed positive correlation between BMI and the severity of sleep indices (AHI, ODI, t90%) underscores the importance of considering obesity as a clinically significant factor in this population[16]. Multivariate analysis suggested a potential contribution of COPD to OSAS severity (OR = 2.18; $p = 0.36$), although this did not reach statistical significance, reflecting the complex interplay between these two conditions. This finding is consistent with reports indicating that different clinical phenotypes of COPD can variably influence OSAS severity[17]. The need for further research is warranted, particularly to explore the therapeutic implications of overlap syndrome, especially given evidence that continuous positive airway pressure (CPAP) therapy is known to provide physiological benefits by reducing respiratory complications in patients with both conditions[18]. While this study reveals notable differences in clinical, polygraphic, and hematological parameters between patients with OSAS and COPD *vs.* those with OSAS alone, it also highlights the complexity of managing this overlap syndrome. In addressing the complexities of managing patients with overlap syndrome, a comprehensive, multifaceted clinical approach is imperative.

First, a thorough and comprehensive assessment is paramount. Clinicians should perform polygraphy alongside pulmonary function tests and arterial blood gas evaluations to accurately gauge the severity of both OSAS and COPD. This initial evaluation serves not only to inform the diagnosis but also to facilitate tailored therapeutic interventions[18].

Second, CPAP therapy stands as the cornerstone treatment for OSAS in patients with concurrent COPD. The utilization of CPAP has demonstrated a significant reduction in hypoxemia and an enhancement in sleep quality, thereby translating into improved respiratory outcomes in these patients[18].

Additionally, it is crucial to incorporate bronchodilator therapy into the management regimen. The employment of long-acting beta-agonists (LABAs) along with anticholinergic agents can significantly alleviate respiratory symptoms by improving airflow and reducing the work of breathing, thus enhancing patient comfort. In cases where an inflammatory component is present in COPD, the prescription of inhaled corticosteroids should be considered. Such interventions can potentiate the reduction of airway inflammation and consequently improve lung function, fostering better respiratory health[10].

Long-term oxygen therapy warrants careful consideration for patients exhibiting pronounced hypoxemia. Administering supplemental oxygen can play a vital role in maintaining adequate oxygen saturation levels and improving long-term survival, especially in those with severe disease progression[17].

Weight management represents another critical aspect of effective clinical management. Encouraging weight loss in patients with obesity can lead to a marked reduction in the severity of both OSAS and COPD manifestations. The integration of nutritional counseling and supervised weight loss programs is recommended to effectively guide patients toward healthier body weights[18].

Moreover, patient education should be an integral component of the management strategy. Educating patients about the interrelationship between OSAS and COPD, as well as the importance of adhering to prescribed treatment regimens and implementing beneficial lifestyle modifications, can greatly enhance treatment outcomes[19].

Regular follow-up is also essential to monitor pulmonary status and evaluate the effectiveness of therapeutic interventions. These follow-up assessments provide opportunities for clinicians to adjust treatment plans as necessary, ensuring optimal care.

Finally, a multidisciplinary approach is invaluable in the management of overlap syndrome. Collaboration between pulmonologists, sleep specialists, dietitians, and respiratory therapists can culminate in a comprehensive and cohesive management plan tailored specifically to the unique needs of each patient[20].

By implementing these clinical management strategies, healthcare providers can significantly improve the management of patients with overlap syndrome, ultimately addressing both the immediate respiratory challenges and the underlying conditions that contribute to their overall health deterioration.

To summarize, this study highlights notable differences in clinical, polygraphic, and hematological features between patients with combined OSA and COPD and those with OSA alone. The findings emphasize the increased severity of sleep-related breathing disturbances in overlap syndrome, demonstrated by significantly elevated apnea-hypopnea index, oxygen-desaturation index and longer durations of hypoxemia below 90% saturation. These results are consistent with the existing evidence that individuals with both conditions face greater hypoxemia and an increased risk of cardiovascular complications. Additionally, the higher prevalence of hypertension in the OSAS-COPD group supports the concept that the coexistence of these diseases amplifies cardiovascular burden.

However, several limitations should be acknowledged. The relatively small sample size of 68 participants, especially the 19 individuals with both OSAS and COPD, may restrict the broader applicability of these findings. A larger sample would allow for more robust analysis and stronger conclusions. Given the observational nature of the study, it is not possible to establish causality between COPD and OSAS severity, and potential confounders – such as different COPD phenotypes – may influence the results. The use of the STOP-BANG questionnaire for initial screening, although practical, may have introduced bias due to its limited sensitivity in assessing severity, particularly in patients with comorbid COPD[21]. Furthermore, the absence of detailed longitudinal data on comorbidities, exacerbation history, and COPD-related symptoms limits a comprehensive understanding of this overlap population[22].

CONCLUSIONS

The complexity of treating overlap syndrome necessitates a multifaceted approach that encompasses careful assessment, tailored interventions, and vigilant monitoring. As such, clinicians should prioritize comprehensive evaluations that integrate sleep studies, pulmonary function tests, and hematological assessments to ensure precise diagnosis and management. Treatment strategies should include CPAP therapy, bronchodilator therapy, and long-term oxygen supplementation, alongside lifestyle modifications such as weight management and patient education.

This multifaceted approach, combined with ongoing research into the pathophysiological mechanisms underpinning OSAS and COPD, will be essential in improving patient outcomes. Ultimately, recognizing and addressing the intricate relationship between these two disorders is imperative for enhancing the quality of care provided to affected individuals and for advancing clinical practice in the field of sleep medicine and respiratory health.

Authors' Contributions

Conceptualization, M.A.V. and G.J.; methodology, M.A.V. and G.J.; software, D.H.; A.M.I; validation, M.A.V., L.F., A.D.I. and G.J.; formal analysis, G.J.; investigation, M.A.V.; resources, M.A.V., G.D and F.I; data curation, D.H.; writing – original draft preparation, M.A.V.; writing – review and editing, M.A.V.; visualization, M.A.V.; supervision, G.J.; project administration, I.R.B, M.A.V and G.J. All authors have read and agreed to the published version of the manuscript.

Funding

This research received no external funding.

Ethics Approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Mureș County Clinical Hospital, Târgu Mureș, Romania (approval No. 9696, 26 June 2025), which authorized the retrospective analysis of anonymized clinical data collected during routine care.

Informed Consent

Written informed consent for research and educational purposes is routinely obtained from all patients at the time of admission to the Pulmonology Clinic. The present study was conducted using retrospectively collected, anonymized data from patient medical records.

Data Availability

The dataset is available on request from the corresponding author.

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Conflict of Interest

The authors declare no conflict of interest for this article.

AI Disclosure

The authors confirm that no AI tools were used for the generation of the manuscript's scientific content. Automated software was used only for editorial assistance in revising phrasing and improving textual originality. This did not affect the study design, data analysis, interpretation, or conclusions. The authors take full responsibility for the content of the manuscript.

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