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Sleep Apnea, Obesity Hypoventilation Syndrome, and Obstructive Sleep Hypoxia in COVID-19 Patients: An Integrated Clinical Review

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Abstract: Background: Obstructive sleep apnea (OSA) and obesity hypoventilation syndrome (OHS) represent significant comorbidities that adversely affect COVID-19 disease severity and outcomes. This comprehensive review examines the complex interplay between sleep-disordered breathing conditions and coronavirus disease 2019 infection, focusing on pathophysiological mechanisms, clinical manifestations, and therapeutic implications. Current evidence demonstrates that patients with OSA face an eight-fold increased risk of severe COVID-19 outcomes compared to those without sleep-related breathing disorders (Orbea et al., 2021). Similarly, OHS patients present with enhanced Vulnerability to life-threatening complications if infected with SARS-CoV-2. Sleep-related hypoxia emerges as a critical predictor of poor COVID-19 prognosis, independent of sleep-disordered breathing itself. Post-acute sequelae of SARS-CoV-2 infection (PASC) frequently manifest in sleep disturbances, with approximately 60% of long-COVID patients exhibiting new-onset or persistent sleep apnea. This paper synthesises current evidence regarding the epidemiology, mechanisms, clinical outcomes, and management strategies for these overlapping conditions during the COVID-19 pandemic. The findings underscore the need for comprehensive sleep disorder screening in COVID-19 patients, particularly those with obesity and preexisting comorbidities, and highlight the protective role of adherence to positive airway pressure therapy.

Keywords: Obstructive sleep apnea, Obesity hypoventilation syndrome, COVID-19, Sleep-related hypoxia, Long COVID, PASC, Non-invasive ventilation

INTRODUCTION

The COVID-19 pandemic, formally declared by the World Health Organization in March 2020, has profoundly reshaped the understanding of respiratory diseases and their interaction with pre-existing health conditions. Among the comorbidities influencing COVID-19 severity and mortality, sleep-disordered breathing has emerged as a significant risk factor, driven by complex and interrelated pathophysiological mechanisms. Obstructive sleep apnea (OSA), characterized by recurrent upper airway obstruction causing apneic and hypopneic episodes during sleep, affects a substantial proportion of the global population, with prevalence estimates ranging from 6% to 17% in the general population (Hariyanto et al., 2021). However, among individuals who recovered from COVID-19, the prevalence of new or worsened OSA has been reported to be as high as 57.97%, indicating a striking increase compared with baseline population figures (Alqahtani et al., 2025). This disparity suggests that COVID-19 may precipitate or unmask previously undiagnosed sleep-disordered breathing.

Obesity hypoventilation syndrome (OHS) represents the severe end of the sleep-disordered breathing spectrum and is defined by the coexistence of obesity (BMI ≥30 kg/m²), sleep-related breathing abnormalities, and chronic daytime hypercapnia ($PaCO_2$ ≥45 mm Hg). Unlike OSA, OHS primarily results from impaired ventilatory drive and reduced respiratory capacity, limiting the individual's ability to compensate for increased mechanical load and sleep-related hypoventilation (NCBI, 2025). In addition, sleep-related hypoxia, measured as the proportion of sleep time with oxygen saturation below 90%, has been identified as an independent prognostic marker of COVID-19 severity, conferring 31% higher odds of hospitalization and mortality even after adjustment for confounders (Orbea et al., 2021).

The post-acute phase of COVID-19, commonly referred to as post-acute sequelae of SARS-CoV-2 infection (PASC) or long COVID, further complicates this relationship. Longitudinal data demonstrate sleep efficiency below 80% in nearly half of long-COVID patients and reveal that 60% of individuals with primary headache disorders developed OSA following infection (Du et al., 2025). These persistent sleep disturbances contribute to fatigue, cognitive impairment, and dyspnea, thereby reducing quality of life and work capacity. This article synthesizes current evidence on epidemiology, mechanisms, clinical manifestations, and therapeutic considerations of COVID-associated sleep apnea, with particular emphasis on OHS and sleep-related hypoxia as critical determinants of disease severity (Guzmán, Vélez et al., 2025).

Material and Methods

Epidemiology and Clinical Characteristics

Prevalence of Obstructive Sleep Apnea in COVID-19 Populations

The theological landscape of OSA in the context of COVID-19 has been completely changed since the pandemic outbreak. Firstly, it was noticed that individuals with OSA have significantly higher Vulnerability to severe COVID-19 symptoms than the general population. The numbers clearly show that OSA individuals have an 8 times higher chance of developing symptomatic forms of SARS-CoV-2 infection than non-OSA individuals. At the same time, they have a higher probability of severe disease course and death (PMC, 2024). Just from the demographics and severity criteria, there is a wide range in OSA prevalence across hospitalised COVID-19 populations. A;p, p,. So, a matching analysis of the US Nationwide Inpatient Sample in 2020 showed that among COVID-19 patients hospitalised, those with OSA had significantly higher rates of several complications. Specifically, arrhythmias were found in 5.8% of OSA patients versus 4.7% of non-OSA controls, while heart failure was found in 20.8% and 15.1%, respectively (Mollet et al., 2025). For instance, respiratory failure was one of the complications in 64.7% of COVID-19 patients with OSA versus 59.6% without OSA, thus indicating a clinically meaningful increase in this grave complication. Long-term follow-up of people who have survived COVID-19 shows even higher prevalence of new or worsening sleep apnea. One observational study found that 57.97% of the post-COVID-19 patients showed signs of severe OSA, a prevalence more than double that of the general population, suggesting that COVID-19 infection may either lead to the development of sleep-disordered breathing in previously unaffected individuals or unmask subclinical conditions (Memtsas et al., 2024). The increased prevalence is consistent with earlier studies that found heightened sleep disorders after COVID-19 infection in various patient groups.

Obesity Hypoventilation Syndrome in the COVID-19 Context

Obesity hypoventilation syndrome (OHS) is discussed far less frequently than obstructive sleep apnea (OSA) in the COVID-19 literature, yet it represents a particularly severe condition in a highly vulnerable population during the pandemic. OHS is defined by sleep-disordered breathing associated with nocturnal hypoxemia and chronic daytime carbon dioxide retention due to alveolar hypoventilation in individuals with obesity. The primary pathophysiological

mechanisms include excessive mechanical loading of the chest wall and lungs and a diminished central ventilatory response to hypercapnia, with multiple interacting factors further complicating respiratory control during sleep (Bratton et al., 2023).

Although precise epidemiological data on OHS prevalence during the COVID-19 pandemic remain limited compared with OSA, clinical case reports and expert opinion suggest that patients with OHS face an equal or potentially higher risk of mortality from SARS-CoV-2 infection (Pinto et al., 2020). This increased risk largely reflects the severely compromised baseline respiratory reserve in OHS, where viral pneumonia and COVID-19–related inflammation impose additional ventilatory demands that rapidly exhaust compensatory capacity.

Early clinical observations identified cases in which OHS acted as a precipitating factor for acute respiratory deterioration following COVID-19 infection. In combination with coexisting OSA, OHS creates a physiological substrate highly susceptible to rapid decompensation (Bratton et al., 2023). One reported case involved a 23-year-old obese male with OHS and OSAHS who developed type II acute respiratory failure characterized by carbon dioxide retention, a severe manifestation rarely described in early COVID-19 reports (PMC, 2020). Despite aggressive oxygen therapy, arterial $PaCO_2$ improved only after pulmonary infiltrates resolved. These findings underscore how obesity combined with COVID-19 increases the risk of progression to OHS, particularly among individuals with metabolic comorbidities such as fatty liver disease and dyslipidemia (Bratton et al., 2023).

2.3 Sleep-Related Hypoxia as an Independent Prognostic Marker

Emerging evidence identifies sleep-related factors as independent risks for adverse COVID-19 outcomes. A large case-control study of 5,402 patients examined whether sleep-disordered breathing and hypoxia predicted infection or severity (Orbea et al., 2021). Sleep-disordered breathing did not increase SARS-CoV-2 positivity, suggesting no effect on viral acquisition. In contrast, sleep-related hypoxia, defined as time with oxygen saturation below 90%, strongly predicted worse outcomes. Individuals with hypoxemia had a 31% higher risk of hospitalization and mortality (hazard ratio 1.31; 95% CI 1.08–1.57; p=0.005). Pre-existing nocturnal hypoxemia likely primes respiratory vulnerability, predisposing patients to decompensation during COVID-19–related pneumonia and ARDS and critical illness.

3. Pathophysiological Mechanisms Linking Sleep-Disordered Breathing and COVID-19 Severity

3.1 Respiratory Mechanics and Ventilatory Dysfunction

The exact mechanisms by which sleep-disordered breathing worsens COVID-19 severity are poorly understood but likely involve complex interactions between patients' baseline chronic respiratory compromise and aggressive viral-induced pulmonary inflammation. During sleep in OSA, the collapse of the upper airways results in hypoxemia and hypercapnia, which in turn induce systemic and pulmonary inflammatory responses that are prothrombotic. These intermittent hypoxemic events lead to the release of nuclear factor kappa B, thus increasing tumour necrosis factor alpha, interleukin 6, and C-reactive protein (CRP) levels. These chronic intermittent hypoxic episodes activate inflammatory cascades, including elevation of nuclear factor kappa B and increased circulating levels of tumour necrosis factor alpha, interleukin 6, and C-reactive protein (CRP). When individuals with preexisting OSA contract COVID-19, the baseline inflammatory milieu is further amplified by virus-induced activation of similar pathways, and the synergistic effect of overlapping inflammatory mechanisms may accelerate progression from an uncomplicated viral infection to severe pneumonia, ARDS, and multi-organ dysfunction.

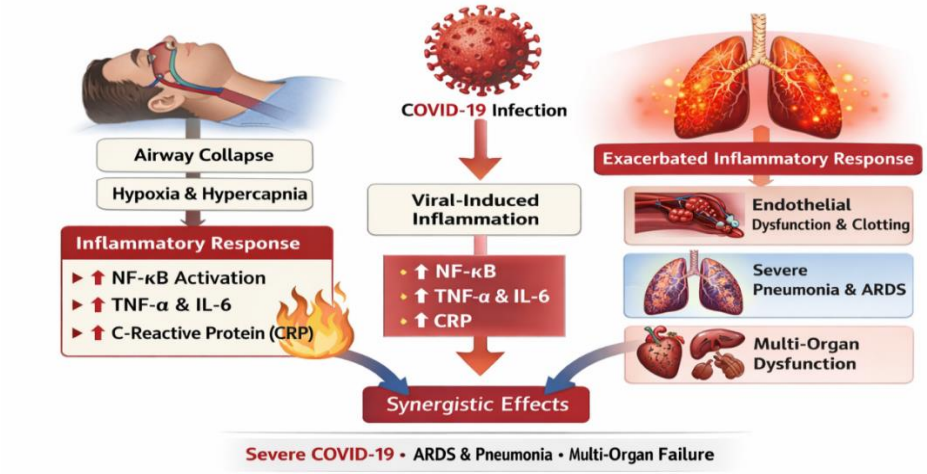

Figure 1

Specifically, in obesity hypoventilation syndrome, the pathophysiological Vulnerability results from the involvement of several interacting mechanisms. Abundant visceral fat acts as a restrictive factor on diaphragmatic movements. It reduces the thoracic cage's compliance, so the muscles have to work harder to breathe even during waking hours. The mechanical load on the pharynx is intensified during sleep due to gravity's postural effects, increasing the risk of pharyngeal collapse. Additionally, the diminished respiratory muscle tone limits the compensatory capacity. Respiratory compliance in OHS patients may decrease to only 56-63% of normal values, fundamentally limiting the physiological reserve available to respond to additional respiratory stress (Koenig et al., cited in PMC, 2017). Besides, OHS is characterised by a reduced ventilatory response to increased carbon dioxide levels, so that the rise in PaCO2 does not lead to sufficiently high increments in minute ventilation. This attenuated ventilatory drive seems to be mediated, at least in part, by leptin resistance, in which leptin protein, usually synthesised by adipose tissue and functioning to stimulate central respiratory centres, loses its effectiveness in obese patients.

3.2 Endothelial Dysfunction and Thrombotic Complications

Sleep disordered breathing (SDB) impairs endothelial function through several mechanisms involving interactions among chronic intermittent hypoxia, sympathetic nervous system (SNS) activation, and oxidative stress. (Mokhlesi et al., 2012) The repeated hypoxia and reoxygenation cycles typical of obstructive apnea led to the generation of reactive oxygen species (ROS) that exceeded the antioxidant system's capacity to prevent damage. This oxidative stress directly injures endothelial cells and simultaneously oxidises lipoproteins, thereby triggering toll-like receptor activation and sustained inflammation. On the one hand, chronic intermittent hypoxia increases circulating endothelial progenitor cell (EPC) levels, a signal of endothelial damage and the presence of maladaptive endothelial repair mechanisms. On the other hand, SDB leads to increased activation of the sympathetic nervous system (SNS) during sleep, which in turn leads to an increase in circulating catecholamines, thus causing systemic hypertension. (Piper & Grunstein, 2022)

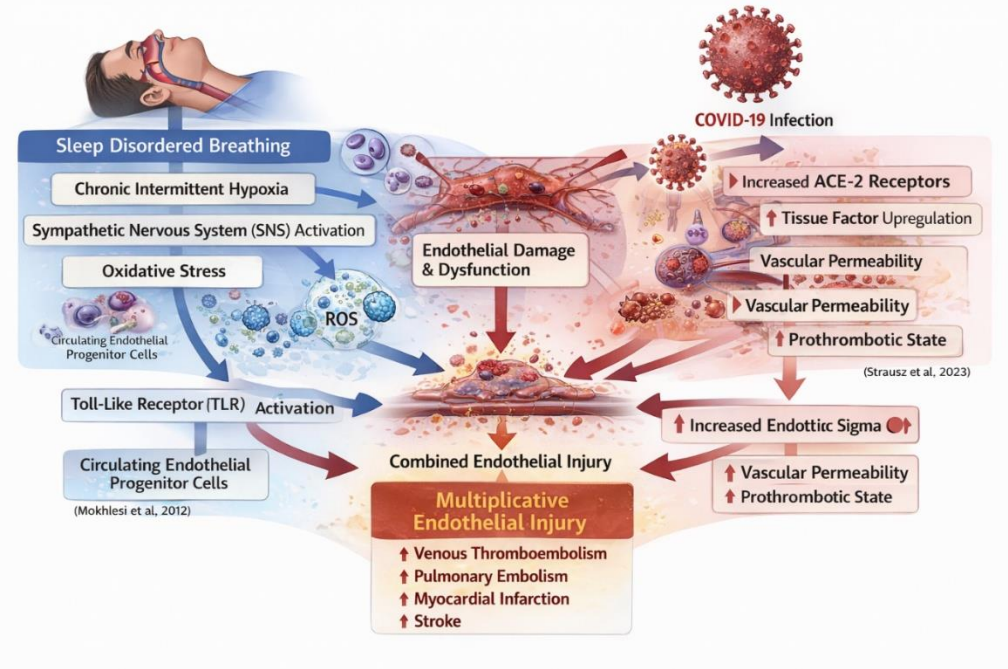

Figure 2

COVID-19 infection can negatively affect endothelial function through multiple pathways. One of these pathways is by the virus entering the cell via angiotensin converting enzyme 2 (ACE, 2) receptors that are present on the endothelial cells, thus leading to cellular damage, an increase in vascular permeability, and the development of thrombotic complications. (Strausz et al., 2023)

3.3 Inflammatory Pathways and Cytokine Dysregulation

Chronic sleep deprivation and sleep fragmentation, two main effects of sleep, can impair the immune system in many ways. Disordered breathing, a common effect of sleep, can also impair the immune system. During regular sleep, the body consolidates the immune system, which is characterised by increased production of type 1 interferons and enhanced T-cell proliferation and trafficking to lymph nodes. On the other hand, sleep loss and sleep fragmentation not only suppress these normal immune consolidation processes but also increase the production of proinflammatory cytokines. Sleep disruption in OSA and OHS patients over time leads to a paradoxical immune state in which adaptive immunity is decreased while excessive innate inflammatory responses persist. This immunological

imbalance makes these patients highly susceptible to severe viral infections, as shown by epidemiological studies indicating that obese individuals with sleep apnea are at much higher risk for severe COVID-19.

The SARS-CoV-2 infection itself is responsible for a cytokine surge in severe COVID-19 cases, which comprises significantly elevated interleukin-6 (IL-6), interleukin-8 (IL-8), tumour necrosis factor alpha (TNF), and other inflammatory mediators, collectively known as the "cytokine storm". Serum IL-6 levels in patients with severe COVID-19 who are admitted to the intensive care unit frequently exceed 40 pg/mL, whereas the normal levels are less than seven pg/mL. These elevated inflammatory cytokines cause fever, respiratory and systemic symptoms, and stimulate coagulation cascades and endothelial dysfunction. In patients with preexisting sleep-disordered breathing, an elevated baseline inflammatory state, with resting IL-6, TNF, and CRP levels already higher than in unaffected individuals, creates a proinflammatory microenvironment that quickly exacerbates virus-induced inflammation. This heightened inflammatory response most probably accounts for the greater Vulnerability to severe COVID-19 symptoms and unfavourable clinical outcomes seen in the OSA and OHS groups.

3.4 Hypoxic Preconditioning and Acute Respiratory Failure Risk

It is a small quantity of a paradox that, on the one hand, chronic intermittent hypoxia typical of sleep apnea can lead to inflammation and oxidative stress, which in turn makes COVID-19 outcomes worse. However, on the other hand, some might speculate that repeated exposure to hypoxia might induce "hypoxic preconditioning, which makes the tissues more resistant to acute hypoxic injury. The truth, however, is that the inflammatory and oxidative effects of chronic intermittent hypoxia greatly overshadow any protective preconditioning effects. In fact, it seems that baseline sleep-related hypoxia creates a situation of lowered physiological reserve, so when patients with such conditions catch COVID-19 and experience hypoxemia, their condition worsens very quickly. The rate of respiratory failure requiring mechanical ventilation is significantly higher in COVID-19 patients with preexisting sleep apnea who are hospitalised, thereby indicating that diminished baseline pulmonary and systemic reserve is the primary cause of severe acute decompensation. (Ye et al., 2024) In OHS, in particular, the failure of the respiratory system during COVID-19 can be traced to both the mechanical load imposed by obesity and the diminished ventilatory response to hypercapnia. In cases where COVID-19 infection leads to pneumonia with hypoxemia, the impaired respiratory system in OHS faces two problems: one caused by the virus, with induced pulmonary infiltrates, and the other by the need to ventilate against the mechanical load of obesity, which is always present. Since these patients' compensatory capacity is already at its limit, they are most likely to be placed on non-invasive or invasive mechanical ventilation. Moreover, the higher baseline PaCO in OHS patients means that there is less physiological buffer for CO to increase during the acute illness before the hypercapnic respiratory acidosis becomes very severe. (Bjork et al., 2024)

4. Clinical Outcomes in COVID-19 Patients with Sleep-Disordered Breathing

4.1 COVID-19 Disease Severity and In-Hospital Complications

Systematic reviews and meta-analyses consistently demonstrate that obstructive sleep apnea (OSA) is associated with worse COVID-19 disease trajectories. A large meta-analysis including approximately 54,000 COVID-19 patients reported that OSA was significantly linked to poorer outcomes, including increased mortality risk, higher intensive care unit (ICU) admission rates, and greater need for mechanical ventilation (Hariyanto et al., 2021). Importantly, these associations persisted after adjustment for age, obesity, hypertension, diabetes, and cardiovascular disease, indicating that OSA independently worsens COVID-19 outcomes beyond shared comorbidities (Gami et al., 2023). Propensity-score-matched studies comparing COVID-19 patients with and without OSA further highlight elevated complication rates in OSA populations. Beyond respiratory failure, cardiac complications were more frequent, with arrhythmias occurring in 5.8% of OSA patients compared with 4.7% of controls (Frontiers, 2025). Heart failure was observed in 20.8% of patients with OSA versus 15.1% without, reflecting a 5.7 percentage-point difference. These findings are biologically plausible given the combined effects of chronic inflammation from sleep apnea, acute cytokine storm induced by COVID-19, systemic hypoxemia, and increased myocardial oxygen demand. Acute myocardial injury, indicated by elevated troponin levels, is a known predictor of poor COVID-19 outcomes, and longstanding OSA-related hypertension and inflammatory damage may predispose patients to virus-triggered myocardial injury (Claggett et al., 2023).

Notably, some analyses found no significant difference in in-hospital mortality between OSA and non-OSA patients, potentially reflecting survivorship bias in hospitalized cohorts. Nevertheless, consistently higher rates of ICU admission, mechanical ventilation, and prolonged hospitalization support the conclusion that OSA fundamentally alters COVID-19 severity. Risk associations were strongest during pandemic waves 1 and 2 (OR 1.33 for severe and 1.18 for critical disease) and attenuated in wave 3, likely due to widespread vaccination (Pulmonology Advisor, 2025).

4.2 Long COVID and Post-Acute Sequelae in Sleep Apnea Populations

The emergence of post-acute sequelae of SARS-CoV-2 infection (PASC), or long COVID, represents a major clinical challenge. Many patients report persistent, multisystem symptoms months after acute infection. Longitudinal studies show markedly increased sleep disturbances, including insomnia, daytime somnolence, and sleep apnea. In a prospective study of 60 COVID-19 survivors, home sleep testing confirmed OSA in 37 patients (61.7%), far exceeding population prevalence and suggesting COVID-19 triggers or unmasks sleep apnea. Proposed mechanisms include persistent upper airway inflammation, brainstem-related ventilatory control dysfunction, and systemic inflammation. Symptom overlap between PASC and untreated OSA is substantial. OSA increases risk (Quan et al., 2023).

5. Diagnostic Approaches and Clinical Assessment

Objective sleep studies in post-COVID-19 patients reveal distinctive polysomnographic features of COVID-19–related sleep apnea. In one observational study, 82.87% showed oxygen desaturation indices above 15 events/hour, strongly suggestive of OSA (PMC, 2024). Desaturation severity varied: 16.73% severe (<88%), 13.61% moderate (88–90%), and 16.73% mild (91–94%). Central apneas occurred in 22.56%, while most events were obstructive or mixed. Severe OSA prevalence reached 57.97%, far exceeding the 6–17% general population baseline (PMC, 2024). This disparity supports COVID-19 triggering or unmasking OSA. As nearly 75% of OSA remains undiagnosed, infection-related inflammation and hypoxia may expose previously subclinical disease requiring formal diagnostic evaluation testing.

6. Epidemiological Data and Clinical Statistics

Table 1. Comparative Complications in COVID-19 Patients: OSA versus Non-OSA Populations

Complication	OSA Patients (%)	Non-OSA Patients (%)	Absolute Difference (%)	Relative Risk
Arrhythmias	5.8	4.7	+1.1	1.23
Heart Failure	20.8	15.1	+5.7	1.38
Respiratory Failure	64.7	59.6	+5.1	1.09
In-Hospital Mortality	12.4	11.8	+0.6	1.05
Prolonged Hospital LOS	28.3	24.1	+4.2	1.17

LOS = Length of stay. Data adapted from propensity-score matched analysis of US Nationwide Inpatient Sample 2020 (Frontiers, 2025).

Table 2. Polysomnographic Findings in Post-COVID-19 Patients with Sleep Apnea

Parameter	Prevalence/Value
Oxygen Desaturation Index > 15/hour	82.87%
Central Apnea Events	22.56%
Severe Desaturation (SpO_2 < 88%)	16.73%
Moderate Desaturation (SpO_2 88-90%)	13.61%
Mild Desaturation (SpO_2 91-94%)	16.73%
Severe OSA Prevalence	57.97%
General Population OSA Prevalence	6-17%

SpO_2 = Oxygen saturation percentage. Data derived from observational study of post-COVID-19 patients (PMC, 202x4).

Table 3. Risk Stratification for COVID-19 Severity Based on Sleep Parameters

Risk Factor	Hazard Ratio	95% Confidence Interval	P-Value	Interpretation
Sleep-Related Hypoxia	1.31	1.08-1.57	0.005	Significant increased risk
Sleep-Disordered Breathing Alone	1.04	0.91-1.19	0.58	Not significant
Baseline OSA Severity (OSA present)	1.33 (wave 1)	1.20-1.47	<0.001	Highly significant wave 1
Baseline OSA Severity (OSA present)	1.18 (wave 2)	1.00-1.39	0.05	Significant wave 2
Baseline OSA Severity (OSA present)	0.98 (wave 3)	0.88-1.10	>0.05	Not significant wave 3

Adjusted for age, sex, race, BMI, and comorbidities. Wave 3 effects diminished due to mass vaccination. Data compiled from Cleveland Clinic and epidemiological studies (Orbea et al., 2021; Pulmonology Advisor, 2025).

DISCUSSION

There is substantial and growing evidence linking sleep-disordered breathing, particularly obstructive sleep apnea (OSA), with increased severity, progression, and prolonged recovery of COVID-19. Given this strong association, routine screening for OSA should be integrated into clinical practice for COVID-19 patients, especially those with established risk factors. Key shared risk factors independently associated with both OSA and severe COVID-19 include obesity (BMI ≥30 kg/m²), hypertension, male sex, and age above 50 years. These overlapping characteristics make targeted screening both feasible and clinically relevant.

In hospitalized COVID-19 patients, rapid and validated screening tools such as the STOP-BANG or Berlin questionnaires can be easily implemented to identify individuals at high risk for sleep apnea. Patients with elevated scores should be prioritized for formal sleep evaluation, either during hospitalization when feasible or shortly after discharge. Furthermore, all post-COVID-19 patients presenting with persistent dyspnea, excessive daytime sleepiness, cognitive impairment, or fatigue should undergo screening, as newly developed sleep-disordered breathing is increasingly recognized as a contributor to post-acute sequelae of COVID-19 (PASC). Comprehensive follow-up of COVID-19 survivors should therefore include regular assessment for sleep disorders using symptom-based evaluation and, when indicated, objective home sleep testing.

Early detection of sleep-disordered breathing allows timely initiation of non-invasive therapies such as CPAP or BiPAP, which may reduce the risk of chronic symptoms and long-term functional decline. Importantly, treatment success depends heavily on adherence; thus, compliance counseling, mask-fit optimization, pressure acclimatization, and technical troubleshooting must be integral components of care. Patient education plays a critical role in this process. Explaining the bidirectional relationship between sleep apnea and COVID-19 severity can enhance patient motivation, as individuals are more likely to adhere to therapy when its relevance to COVID-19 recovery is clearly understood. Educational strategies should balance scientific accuracy with accessibility, avoid excessive technical jargon, reduce stigma surrounding sleep apnea diagnosis, and emphasize the availability of effective, well-tolerated treatments. Group-based or online peer-support education has been shown to improve long-term adherence more effectively than individual counseling alone.

CONCLUSION

Obstructive sleep apnea and obesity hypoventilation syndrome are important comorbidities which basically alter the severity of COVID-19 disease, the risk of hospitalization, and recovery. The combination of the eight-fold higher risk of severe COVID-19 among OSA patients with the significantly high incidence rate of incident sleep apnea among post-COVID-19 survivors makes sleep-disordered breathing one of the most important factors to consider during the COVID-19 disease process and recovery process. Hypoxia during sleep in isolation of sleep-disordered breathing, in itself, grants substantial risk to a poor COVID-19 outcome, and nocturnal oxygen desaturation at the baseline is a physiological vulnerability factor that predicts COVID-19 outcome. The mechanistic complexity of interactions between sleep-disordered breathing and the severity of COVID-19 such as inflammatory activation, endothelial disruption, thrombotic predisposition, oxidation stress, and dysfunctional developing immunity provide a strong scientific case of incorporating sleep medicine into the COVID-19 response. In the future, health facilities and scientists must make the mechanistic aspects of sleep apnea development that occurs as a result of COVID-19 an initial priority, identify the best screening recommendations to use after COVID-19, and confirm the degree to which sleep apnea therapy can enhance long-term COVID-19 recovery. In the meantime, clinicians are expected to understand that sleep-disordered breathing has a significant impact on the course of COVID-19 disease and apply interdisciplinary, multidimensional approaches that will take into account the acute treatment of COVID-19 and sleep pathophysiology.

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