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Review

The Interplay Between Obstructive Sleep Apnea and Respiratory Infections: A Review Article

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ABSTRACT

Obstructive sleep apnea (OSA) is a prevalent sleep-related breathing disorder characterized by recurrent upper airway collapse, intermittent hypoxia, and sleep fragmentation. Increasing evidence suggests a bidirectional association between OSA and lower respiratory tract infections (LRTIs), including pneumonia, bronchitis, and exacerbations of chronic lung disease. Multiple mechanisms may underlie this relationship. Intermittent hypoxia and sleep disruption promote systemic inflammation and immune dysregulation. Impaired mucociliary clearance, microaspiration, alterations in airway microbiota, together with obesity and related comorbidities, further contribute to increased susceptibility to and severity of infections. Observational studies demonstrate that individuals with untreated OSA have higher rates of pneumonia, more severe infections, and delayed recovery from infections compared with non-OSA populations. These risks are particularly evident among older adults and patients with cardiopulmonary comorbidities. Continuous positive airway pressure (CPAP) therapy may mitigate infection risk by maintaining airway patency, reducing hypoxemia, and improving mucociliary clearance. However, concerns remain regarding CPAP device-associated microbial colonization, highlighting the importance of strict hygiene practices and equipment maintenance. OSA appears to be an underrecognized risk factor for LRTIs; this association is driven by overlapping pathophysiological mechanisms and is supported by emerging epidemiological data. Recognizing this interplay may guide infection prevention strategies and improve clinical outcomes in high-risk populations.

KEYWORDS: Obstructive sleep apnea, lower respiratory tract infections, immune dysregulation, CPAP therapy, airway microbiota, COVID-19, community-acquired pneumonia

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INTRODUCTION

Obstructive sleep apnea (OSA) is the most common sleep-breathing disorder, characterized by collapse of the soft tissues of the upper airway during sleep, causing recurrent intermittent hypoxia and hypercapnia, and resulting in frequent nocturnal awakenings and sleep fragmentation.[1] Those alterations induce sympathetic activation, oxidative stress, and metabolic dysregulation,[2] which lead to various cardiovascular diseases and pulmonary, neurological, and cognitive dysfunctions.[3,4] The exact prevalence of OSA is underestimated; available data indicate that about 20% of middle-aged Americans have OSA, and a higher prevalence has been observed among Asian populations.[5,6] The incidence is higher in males aged 50–70 years (17%) than in females diagnosed with moderate-to-severe OSA (9%).[7]

Lower respiratory tract infections (LRTIs) are a major public health problem associated with increased morbidity and mortality. LRTIs include bronchitis, bacterial and viral pneumonia.[8] The host immune response and the virulence of the susceptible organism determine the outcomes of respiratory infections.[9] The proinflammatory state induced by OSA[10] has highlighted the coexistence of OSA and respiratory infections through enhancement of proinflammatory stimuli.[11] The coronavirus disease 2019 (COVID-19) pandemic has heightened awareness of viral LRTIs.[12] In 2019, LRTIs were recognized as one of the leading causes of mortality worldwide. An American observational study in Chicago, using healthcare

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system records, reported that OSA patients had an increased risk of COVID-19 infection, hospitalization, and worse sequelae compared with patients of the same age who received similar treatment.[13] It was assumed that elevated inflammatory markers in OSA patients contributed to unfavorable COVID-19 outcomes. Moreover, COVID-19 and OSA share several risk factors, such as diabetes, hypertension, cardiovascular disease, and obesity.[13,14]

This review explores the potential link between OSA and LRTIs and the burden of both conditions in adults.

Objective

To evaluate, through a literature review, the impact of OSA on susceptibility, pathogenesis, and clinical outcomes of respiratory infections, and to discuss potential management strategies.

METHODS LITERATURE SEARCH

A systematic search of PubMed, Web of Science, and CENTRAL, using specified keywords, was conducted to identify publications up to January 31, 2025. Our search strategy followed a stepwise approach to ensure comprehensive capture of relevant literature. We began with search terms for obstructive sleep apnea (Search #1), combining MeSH terms such as "Obstructive Sleep Apnea" and "Sleep Apnea, Obstructive" with title and abstract keywords, including "Sleep-related breathing disorders" and "OSA". We also included broader MeSH terms like "Sleep-Disordered Breathing" and "Sleep Apnea Syndromes" to ensure comprehensive coverage for respiratory infections, we constructed three separate search sets: one for bacterial pneumonia (Search #2) using terms such as "Pneumonia"[MeSH], "bacterial pneumonia," community-acquired pneumonia, and "Lower Respiratory Tract Infections"[MeSH]; another for influenza (Search #3) using "Influenza, Human"[MeSH] and related terms; and a third for COVID-19 (Search #4) using appropriate MeSH terms and keywords. These respiratory infection searches were combined using the Boolean operator "OR" (Search #5) and were then intersected with the OSA search using "AND" (Search #6). Finally, we applied filters for English-language publications, human studies, an adult population (19+ years), and publication year 2025 (Search #7). The summary of the research strategy is listed in Table 1.

Main Points

- Emerging evidence suggests that obstructive sleep apnea (OSA) may increase lower respiratory tract infections, particularly in individuals with severe disease and higher hypoxic burden.
- Biological mechanisms such as intermittent hypoxia, systemic inflammation, immune dysregulation, impaired mucociliary clearance, and microaspiration may contribute to the observed association between OSA and infection severity.
- Consideration of preventive strategies-including vaccination, comorbidity optimization, and careful continuous positive airway pressure hygiene- may help reduce infectious morbidity in patients with moderate-to-severe OSA.

SEARCH STRATEGY LITERATURE

Both authors (A.M.E., S.K.) searched for, and conducted a full-text review of, the retrieved literature. The two authors discussed the exclusion and inclusion criteria of the retrieved studies and reached a consensus.

Results of Literature Search

A systematic search of the literature was conducted in PubMed, CENTRAL, and Web of Science to evaluate whether OSA in adults is associated with an increased risk of respiratory infections.

The search strategies included free-text terms and controlled vocabulary for OSA ("Sleep Apnea, Obstructive," "OSA," "Sleep-related breathing disorders") and for respiratory infections ("respiratory tract infections," "pneumonia," "bronchitis," "influenza," "COVID-19," and related terms).

The search retrieved a total of 3,975 records (PubMed: 1,440; CENTRAL: 1,294; Web of Science: 1,241). After removal of 1,070 duplicate records, 2,905 unique records remained for title and abstract screening. Full-text articles were subsequently assessed for eligibility, and the reasons for exclusion were recorded. The final numbers of included studies are presented in the PRISMA flow diagram Figure 1.

PATHOPHYSIOLOGICAL MECHANISMS

Hypoxia and Inflammation

Healthy sleep plays a crucial role in regulating the immune system.[15,16] OSA patients experience chronic sleep deprivation and intermittent hypoxia. Immune perturbations secondary to disrupted sleep led to increased oxidative stress and systemic inflammation, which may render those patients susceptible

Table 1. Summary of the research strategy

Items	Specification
Search date	January 31, 2025
Databases searched	PubMed; Web of Science; CENTRAL
Search terms	"Obstructive Sleep Apnea"; "Sleep-related breathing disorders"; OSA; Pneumonia; bacterial pneumonia; community-acquired pneumonia; CAP; respiratory infection; bronchopneumonia
Timeframe	Up to January 31, 2025
Inclusion criteria	English language; non-randomized trials; randomized controlled trials (RCTs); proof-of-concept studies; observational studies (cross-sectional, cohort, case-control); case reports; research protocols
Exclusion criteria	Unavailable full text (abstracts and letters to the editor)
Selection process	Article screening: S.K. and A.M.E.; Literature classification: S.K. and A.M.E.; Review content preparation: S.K. and A.M.E.

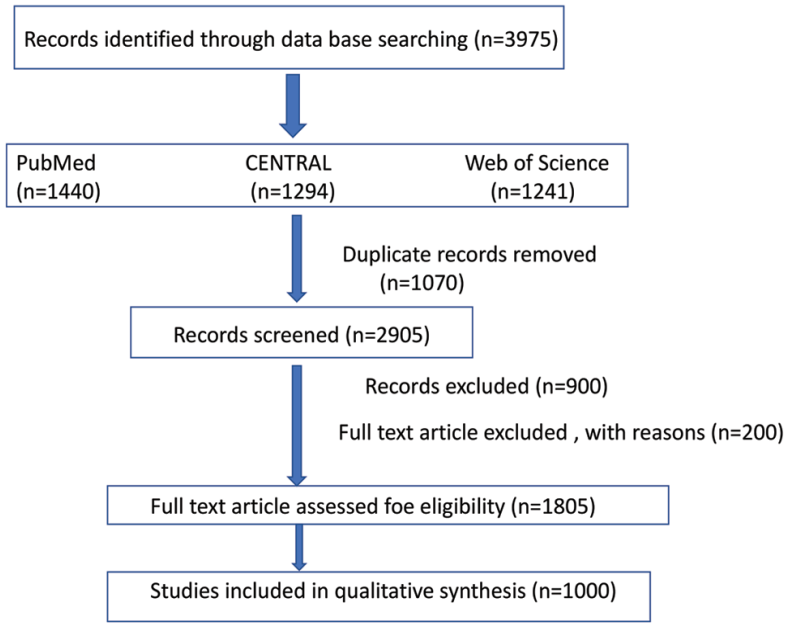

Figure 1. PRISMA flow diagram

to pathogen invasion and LRTIs.[15-18] Impaired lung neutrophil function resulting from hypercapnia that may be associated with OSA could render those patients susceptible to LRTIs.[19]

Impaired Mucociliary Clearance

OSA is associated with alterations in intrathoracic pressure and breathing patterns. Those alterations are more frequently observed during rapid eye movement sleep. They are attributed to inhibition of cortical input to the brainstem cough center. Moreover, at night there is an increase in the peripheral nerves threshold that cause a weak or even an absent cough reflex that leads to an inability to clear pathogens from the airways.[20,21]

Microaspiration Risk

During the night, aspiration of small amounts is normal in healthy individuals.[22] During sleep, patients with OSA have a higher risk of pulmonary aspiration than individuals without OSA. Following episodes of apnea-hypopnea, patients are stimulated to breathe against a closed airway, leading to a greater increase in negative intrathoracic pressure. As a result, the pressure gradient increases, creating a vacuum effect in the upper airway that leads to excessive microaspiration. Excessive microaspirations lead to increases in bacterial pathogens and upper airway and laryngeal inflammation.[23-28] Treatment of gastroesophageal reflux (GER) may lead to a significant decrease in the apnea-hypopnea index (AHI).[25,29]

The Role of Comorbidities and Obesity

There is evidence that obesity and comorbid conditions such as diabetes, hypertension, cardiovascular disorders, cerebrovascular disease, GER, Parkinson's disease, epilepsy, and chronic liver and kidney diseases are associated with greater OSA risk. Those comorbidities increase the risk of infections, including LRTIs. Also, in patients with comorbid chronic obstructive pulmonary disease (COPD) and asthma, those who receive corticosteroids have an increased risk of LRTIs.[30]

Obesity increases the risk of complications in critically ill patients.[31-33] However, the "obesity paradox" confers a protective effect against mortality from LRTIs among obese patients. This effect is the result of an early presentation in those patients, increased hospital admission, and changed immune response characterized by lesser inflammation in those patients (Figure 2).[34-36]

EVIDENCE FROM THE STUDIES

In this retrospective cohort study, adult OSA patients (aged ≥20 years) were enrolled from the Research Database of the Taiwan National Health Insurance. Compared to a cohort control group without OSA, having the same age, sex, and comorbidities. The two cohorts were followed for the occurrence of pneumonia. The study included 34,100 patients (6,816 with OSA and 27,284 controls). During a follow-up period of approximately 4.50 years, 2,757 (8.09%) participants developed pneumonia, including 638 (9.36%) OSA patients and 2,119 (7.77%) in the control cohort. They concluded that pneumonia incidence was higher among OSA patients, based on Kaplan-Meier analysis (log-rank test, $P < 0.001$), and a 1.20-fold increased risk (95% confidence interval 1.10–1.31) after multivariate adjustment. They also stated that the risk was even greater among patients who were treated by continuous positive airway pressure (CPAP).[37] CPAP is the gold-standard therapy for moderate-to-severe OSA. It may decrease sputum expectoration, thereby increasing the risk of aspiration. In addition, humidifiers provide a potential source of bacterial contamination.[38] Therefore, maintenance and regular cleaning of CPAP devices are crucial to avoid the risk of infection.[39]

Chiner et al.[40] conducted a study that included 82 community-acquired pneumonia (CAP) patients and 41 patients with non-respiratory infections. They found that severe OSA patients

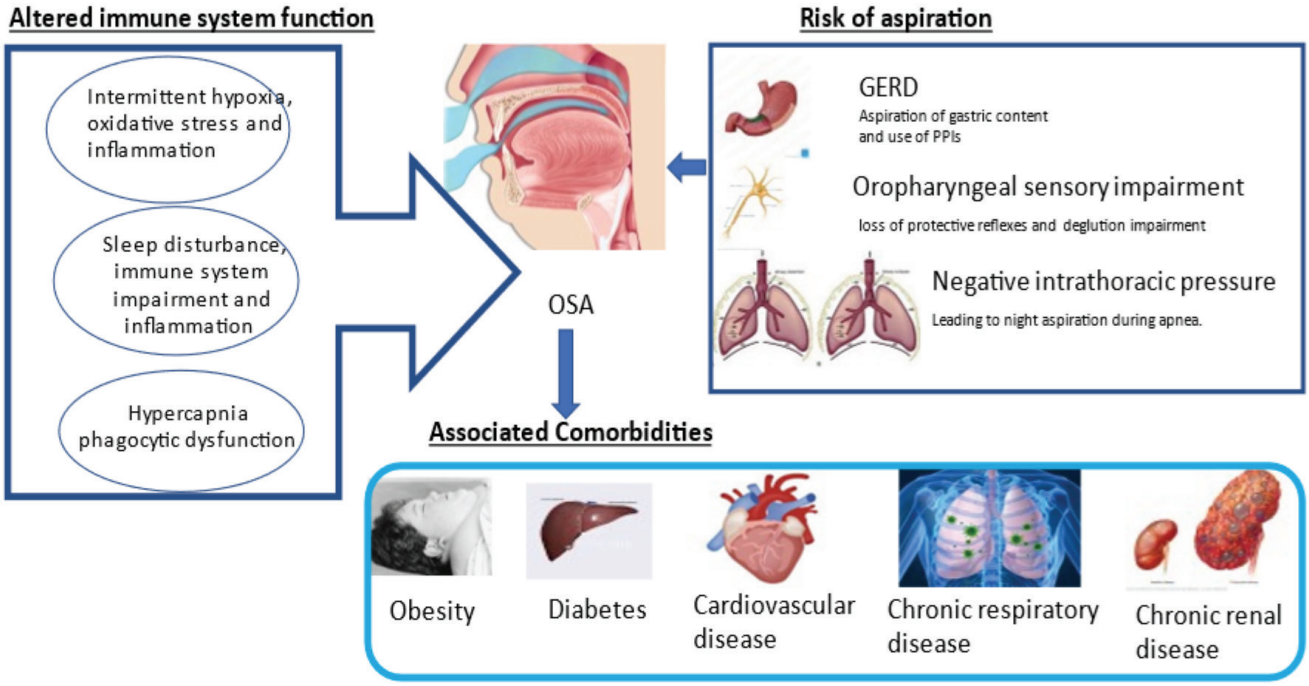

Figure 2. Risk factors and pathophysiological interaction of LRT infections and OSA

LRT: lower respiratory tract infection, OSA: obstructive sleep apnea

had threefold greater odds (odds ratio: 3.18; 1.11–11.56) of developing pneumonia during sleep than normal-breathing patients. They also concluded that, based on AHI results and various indices of oxygen saturation, higher OSA grade was associated with more severe CAP presentation, which highlights the role of OSA as a risk factor that increases susceptibility and influences the clinical outcomes of CAP.[40]

Notably, during a follow-up period of up to 20.4 years, the Atherosclerosis Risk in Communities prospective cohort study reported that severe OSA increased the risk of pneumonia by 87%. Also, they found that a hypoxic burden [which is the time of oxygen saturation below 90% during total time of sleep (T90)] greater than 5%, compared with less than 1%, was associated with a 50% increased risk of pneumonia. This association suggests that the hypoxic burden and its physiological consequences increase the risk of pneumonia, extending beyond the effects of obesity alone.[41]

Moreover, a nationwide Finnish retrospective population-based case-control study, using data from the national primary and secondary health care registers from 2015–2019, investigated the incidence of LRTs in the first year before and after diagnosis of OSA. They included controls matched for sex, age, and multimorbidity. They analyzed the effect of comorbidities and patient characteristics on the risk of LRTIs and on their recurrence. They reported that OSA patients develop LRTIs at a higher incidence than controls. Hazard ratios were 1.35 (95% confidence interval 1.16–1.57) one year before the diagnosis of OSA and 1.39 (95% confidence interval 1.22–1.58) one year after the diagnosis of OSA. They found that in OSA patients, a previous LRTI before OSA diagnosis, having more than one morbidity, asthma, COPD, and age greater than 65 years increased the incidence and recurrence of LRTIs in those patients.

DIAGNOSTIC AND SCREENING CONSIDERATIONS

OSA and LRTIs

CAP is defined as an acute disease characterized by one or more of the following symptoms: cough with or without sputum production, dyspnea, pleuritic chest pain, and fever. On auscultation, adventitious and bronchial breath sounds may be heard. An increased total leucocytic count and the presence of infiltrates on the chest X-ray were observed.[42] Investigations include a complete blood picture, serum C-reactive protein (CRP) levels, and arterial blood gas analysis. Detection of the causative organism is performed by sputum culture or by protected bronchial lavage via fiberoptic bronchoscopy.

After hospitalization, CAP patients suspected of having OSA should be screened with sleep questionnaires to assess OSA symptoms. Daytime sleepiness is screened by the Epworth sleepiness scale.[43] During history taking, smoking status, alcohol consumption, and the presence of associated comorbidities (COPD, diabetes, previous pneumonia, heart failure, asthma, steroid intake during the last 6 months, hospitalization during the previous 3 months, liver and kidney failure, and cerebrovascular disease) should be assessed. Physical examinations include height, weight, body mass index, and the Mallampati scale. The Diagnosis of OSA is confirmed by polysomnography when the AHI is equal to or greater than 5 events per hour of sleep.

Sleep deprivation in OSA patients leads to altered immunity and increases the risk of LRTIs. Hypoxemia and related oxidative stress are linked to activation of the immune system, and inflammatory marker levels increase [proinflammatory cytokines, e.g., interleukin 6 (IL-6), tumor necrosis factor-alpha (TNF-α), and CRP].[44-47] Moreover, sleep deprivation without

hypoxemia induces inflammation and elevates inflammatory markers in untreated sleep apnea patients.[48,49]

OSA and Pneumonia Severity

Chronic inflammation, hypoxia, and sleep fragmentation associated with the pathogenesis of OSA lead to alterations in immune system activity and an increased risk of severe infection.[50-52] Gastro-esophageal reflux disease associated with OSA increases the risk of pulmonary aspiration and the subsequent development of aspiration pneumonia.[53] The airway microbiome in OSA patients is altered compared with that in healthy individuals. It increases predisposition to respiratory infection as its severity increases.[54]

CLINICAL IMPLICATION AND MANAGEMENT

Role of CPAP Therapy

Treatment with a positive airway pressure device, together with lifestyle interventions, remains the gold-standard treatment for moderate-to-severe OSA. CPAP use for at least four hours per night has been shown to decrease sleep fragmentation and improve oxygenation, thereby reducing inflammation and enhancing sleep quality. Special consideration of the use of a suitable interface and its proper hygiene to decrease the risk of infections.[55]

Role of Antibiotic Therapy

Determining the causal agent of CAP is challenging. The American Thoracic Society and Infectious Diseases Society of America (ATS/IDSA) 2019 guidelines recommended empirical antibiotic therapy.[56] Regimen choice is based on the patient characteristics, the pneumonia severity index (PSI), the presence of comorbidities, risk of complications, and the clinical decision to hospitalize.[56-58]

OSA may augment PSI scores and lead to hospitalization because associated comorbid conditions necessitate broader antimicrobial coverage.

The ATS/IDSA guidelines for outpatient management recommend regimens that depend on the risk of complications.[56]

Table 2. The summary of the most relevant literature on the association between OSA and LRTIs

Author and date	Design	Total n (OSA n)	Inclusion and exclusion criteria	Outcomes	Key Findings	Limitations
Maas et al.,[13] 2021	Multicentric, retrospective cohort	5544,884 (~44,877)	I: All patient encounters; January to June 2020	COVID-19, hospitalization, respiratory failure.	OSA: ↑ COVID-19, OR: 8.6, ↑ hospitalization, OR: 1.65, ↑ respiratory failure, OR: 1.98	No PSG data, no data on OSA treatment
Cade et al.,[14] 2020	Multicentric, retrospective cohort	4668 (443)	I: Positive COVID-19 PCR; A minimum of two clinical notes, two encounters, and three ICD diagnoses	Mortality, composite (mortality, MV, ICU), hospitalization.	OSA or CPAP not linked with mortality, MV, ICU, and hospitalization	No PSG data, no data on OSA treatment
Chiner et al.,[40] 2016	Single center case-control	123 (85)	I: Cases: Hospitalized for CAP; Controls: Hospitalized for non-respiratory/non-ENT infection. E: Previous OSA diagnosis and CPAP	Pneumonia, PSI	AHI ≥10: ↑ pneumonia OR: 2.86; AHI ≥30: ↑ pneumonia OR: 3.184; AHI positively correlated with PSI	Small sample size, no data on OSA treatment
Keto et al.,[49] 2023	Case-control from Finland	50,648 (25,324)	I: ICD code for OSA. E: OSA in the two years preceding the index date	LRTI, recurring LRTI	↑ LRTI in the year preceding OSA RR: 1.35, and during the year after OSA RR: 1.39	No PSG data, no data on OSA treatment, no BMI data
Girardin et al.,[59] 2021	Retrospective cohort from NYC and LI	4446 (290)	I: Positive COVID-19 PCR	Hospital mortality	OSA not linked to mortality	No PSG data, no data on OSA treatment, no BMI data
Lutsey et al.,[41] 2023	Post-hoc analysis of the multicentric prospective cohort	1586 (772)	I: Valid PSG data; Self-identify as White. E: CSA; Already had the outcome of interest at the time of visit	Hospitalization: with pneumonia; with respiratory infection; with any infection	OSA not linked to outcomes; T90 >5% ↑ hospitalized pneumonia HR: 1.59, ↑ hospitalized respiratory infection HR: 1.53, ↑ hospitalized any infection HR: 1.25	No data on OSA treatment, mostly White population

AHI: apnea-hypopnea index, BMI: body mass index, CAP: community-acquired pneumonia, CA: Canada, CPAP: continuous positive airway pressure, CSA: central sleep apnea E: exclusion criteria, HR: hazard ratio, I: inclusion criteria, ICD: International Classification of Diseases, ICU: intensive care unit, LRTI: lower respiratory tract infection, n: number of subjects included, NYC: New York City, OSA: sleep apnea, PCR: polymerase chain reaction, PSG: polysomnography, ↑: increased risk, (OSA N): number of study participants with obstructive sleep apnea, ~: approximation based on presented data

Patients with chronic comorbidities should be treated with dual therapy combining a beta-lactam anti-pneumococcal agent (cefuroxime or amoxicillin-clavulanate) and either doxycycline or a macrolide (e.g., azithromycin or clarithromycin). Fluoroquinolones (e.g., levofloxacin or moxifloxacin) can be used as monotherapy.

Patients with OSA are at increased risk of pneumococcal and streptococcal pneumonia.[60] Therefore, those patients require a more aggressive approach. The recommendations of the Specific Risks Guiding Empiric Antibiotic Therapy Guideline for regimens of antibiotics for CAP recommend to cover the most common and typical (*Streptococcus pneumoniae*, *Staphylococcus aureus*, *Moraxella catarrhalis*, *Haemophilus influenzae*) also the atypical pathogens such as (*Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Legionella* species). Furthermore, the CDC recommends specific antiviral therapies for OSA patients with influenza or COVID-19 infection.[56]

Pseudomonas aeruginosa is identified in more than 10% of patients diagnosed with OSA who present with bacterial pneumonia.[40] OSA is associated with several comorbidities[59] that increase the risk of *Pseudomonas* infection. Thus, in severe LRTI cases with one or more associated comorbidities or with OSA, it is advisable to use a fluoroquinolone or an antipseudomonal beta-lactam as early coverage for *Pseudomonas*.[56]

Case reports have documented Legionnaires' disease in OSA patients using CPAP.[61,62] Legionnaires' disease is characterized by neurological disturbances, bradycardia, diarrhea, and hyponatremia.[63] Diagnosis is confirmed by detecting Legionella antigen in urine. Treatment includes respiratory fluoroquinolones.[64]

PREVENTIVE STRATEGIES

Current CDC guidelines recommend pneumococcal vaccination for individuals aged 65 years and older and for patients under 65 years with asthma, COPD, or emphysema.[65] They also recommend annual influenza vaccinations for individuals aged six months or older.[66] COVID-19 vaccinations are advisable in light of new variants.[67,68] We recommend prioritizing patients with OSA for vaccination against these infections.

DISCUSSION

This review highlights the emerging and clinically significant association between OSA and LRTIs. Traditionally, the clinical focus of OSA research has been directed toward cardiovascular, metabolic, and neurocognitive sequelae. However, the accumulating evidence reviewed here suggests that infectious complications —particularly bacterial and viral pneumonias— represent an underrecognized dimension of OSA-related morbidity.

Pathophysiological Insights: The pathophysiological mechanisms outlined in this review support a biologically plausible interplay between OSA and increased LRTIs. Intermittent hypoxia, systemic inflammation, immune dysregulation, impaired mucociliary clearance, microaspiration, and comorbid conditions such as obesity and diabetes converge to create a microenvironment favorable for respiratory pathogens. Importantly, these mechanisms extend beyond simple epidemiological associations, strengthening the argument for causality rather than coincidence. Moreover, the interplay between OSA and gastroesophageal reflux disease (GERD) adds another layer of complexity, as chronic aspiration may act as a critical driver of recurrent infections.

Clinical Evidence: Several cohort and case–control studies reinforce this biological plausibility by demonstrating higher rates of pneumonia and hospitalization among patients with OSA, with evidence of dose–response relationships linked to OSA severity and hypoxic burden. However, heterogeneity across studies should be acknowledged. Some cohort studies suggest an increased risk only in the year before or the year after OSA diagnosis, raising questions about surveillance bias and the role of comorbidities.

Although CPAP therapy improves sleep-related outcomes, observational studies caution that it may increase the risk of aspiration or lead to microbial colonization of equipment, underscoring that therapy can be both preventive and risk-enhancing when hygiene protocols are not strictly followed.

Strengths and Limitations of Current Evidence

The strengths of the available evidence include large-scale population studies, long-term prospective cohorts, and consistent findings across different geographical regions. Nonetheless, several limitations must be considered. First, most studies rely on administrative databases, which may underestimate undiagnosed OSA and introduce misclassification bias. Second, confounding by obesity, smoking, or comorbid cardiopulmonary disease is difficult to eliminate, even with multivariable adjustments. Third, mechanistic studies directly linking OSA-related immune dysfunction to clinical infections remain scarce. Finally, heterogeneity in the definitions of both OSA (AHI thresholds and diagnostic modalities) and LRTIs (clinical versus microbiological confirmation) complicates direct comparisons. A summary of the most relevant studies on the association between OSA and LRTIs is listed in Table 2.

Clinical and Public Health Implications: From a clinical standpoint, recognition of OSA as a risk factor for LRTIs warrants heightened vigilance. Early identification of OSA in patients presenting with recurrent pneumonia, aspiration events, or unexplained severe CAP could guide tailored management strategies. Preventive interventions such as vaccinations (pneumococcal, influenza, and COVID-19), optimization of comorbid conditions, and patient education regarding CPAP hygiene may have additional benefits in reducing infectious morbidity. In the intensive care setting, awareness of OSA may aid in risk stratification for ventilatory support needs, infection risk, and prognosis.

At the public health level, the intersection of two highly prevalent conditions —OSA and LRTIs— magnifies the global burden. In low- and middle-income countries, where OSA remains underdiagnosed and access to diagnostic sleep studies is limited, the potential additive impact of OSA on pneumonia outcomes warrants urgent attention. Screening strategies integrated into primary care and chronic disease management may provide cost-effective opportunities for risk reduction.[69,70]

Future Research Directions: Several important research gaps remain. Future studies should (1) explore biomarkers of OSA-related immune dysfunction (e.g., CRP, IL-6, TNF-α) as predictors of infection risk. (2) Clarify the temporal relationship among OSA onset, diagnosis, and LRTI incidence to disentangle causal pathways from detection bias. (3) Assess the risks and benefits of CPAP therapy in relation to infection outcomes, including prospective studies with microbiome analyses. (4) Investigate whether OSA indices beyond the AHI (e.g., hypoxic burden, sleep fragmentation, nocturnal desaturation) better predict infection susceptibility. (5) Evaluate preventive strategies, including targeted vaccination campaigns and GERD management, among individuals with OSA.

CONCLUSION

The authors conclude that OSA may increase susceptibility to acute LRTIs and the risk of more severe forms of these infections, although there is no clear evidence that it increases pneumonia-related mortality. They emphasize that preventive strategies, such as vaccination and infection control measures, should be considered, particularly for patients with severe OSA.

This review article examines the association between OSA and both acute and chronic LRTIs, including CAP, viral pneumonia (influenza), and COVID-19. It provides a comprehensive synthesis of the existing literature on pathophysiological mechanisms (hypoxia, inflammation, impaired mucociliary clearance, microaspiration, obesity, and comorbidities), epidemiological data, diagnostic approaches, management strategies, and preventive measures.

To our knowledge, there is no indication that a work of this scope and structure has been published previously.

Footnotes

Authorship Contributions

Concept: A.M.E., S.K., Design: A.M.E., S.K., Data Collection or Processing: A.M.E., S.K., Analysis or Interpretation: A.M.E., S.K., Literature Search: S.K., Writing: S.K.

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