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ORIGINAL ARTICLE

Evaluation of Inflammatory Cytokines (IL-6, TNF-α, IL-10) and Upper Airway Obstructive Factors in Patients with Obstructive Sleep Apnea and Their Association with Cardiometabolic Risk

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction during sleep, resulting in intermittent hypoxia, oxidative stress, and systemic inflammation. Increasing evidence suggests that inflammatory cytokines play a crucial role in the pathophysiology of OSA and its associated cardiometabolic complications.

Objective: To evaluate serum levels of inflammatory cytokines (IL-6, TNF-α, and IL-10) and upper airway obstructive factors in patients with obstructive sleep apnea and to determine their association with cardiometabolic risk.

Methods: This cross-sectional study was conducted at Nishtar Hospital, Multan, and Isra University Hospital, Hyderabad, Pakistan, from June 2022 to February 2023. A total of 100 participants were included, comprising patients diagnosed with obstructive sleep apnea and healthy controls. Clinical evaluation included demographic characteristics, body mass index, neck circumference, and upper airway examination to identify structural obstructive factors. Serum inflammatory cytokines including IL-6, TNF-α, and IL-10 were measured using the enzyme-linked immunosorbent assay (ELISA) method. Cardiometabolic risk parameters including fasting blood glucose, lipid profile, and blood pressure were also assessed. Statistical analysis was performed using SPSS version 25, and a p-value <0.05 was considered statistically significant.

Results: Patients with obstructive sleep apnea demonstrated significantly higher levels of IL-6 and TNF-α compared with healthy controls, whereas IL-10 levels were significantly reduced. OSA patients also exhibited higher body mass index, neck circumference, fasting blood glucose levels, and dyslipidemia, indicating increased cardiometabolic risk. Upper airway obstructive factors such as tonsillar hypertrophy, nasal obstruction, elongated uvula, and soft palate enlargement were significantly more prevalent among OSA patients. Furthermore, elevated pro-inflammatory cytokine levels showed positive correlations with obesity, hypertension, and metabolic abnormalities.

Conclusion: The study demonstrates that obstructive sleep apnea is associated with increased systemic inflammation and upper airway structural abnormalities, which contribute to elevated cardiometabolic risk. Measurement of inflammatory cytokines such as IL-6 and TNF-α may serve as useful biomarkers for identifying patients at risk of metabolic and cardiovascular complications associated with OSA.

Keywords: Obstructive sleep apnea, inflammatory cytokines, IL-6, TNF-α, IL-10, cardiometabolic risk, airway obstruction, systemic inflammation.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by repetitive episodes of partial or complete obstruction of the upper airway during sleep. These episodes lead to intermittent hypoxia, hypercapnia, sleep fragmentation, and fluctuations in intrathoracic pressure[1]. As a consequence, patients often experience excessive daytime sleepiness, impaired cognitive function, and reduced quality of life. Globally, OSA is considered a major public health problem due to its high prevalence and its strong association with cardiovascular and metabolic diseases[2].

The pathophysiology of OSA is complex and involves both anatomical and physiological mechanisms. Structural abnormalities of the upper airway, such as enlarged tonsils, elongated soft palate, macroglossia, nasal obstruction, and increased neck circumference, can contribute to airway narrowing and collapse during sleep[3]. Obesity is one of the most important risk factors for OSA because excess adipose tissue around the pharyngeal airway increases airway resistance and reduces muscle tone during sleep. These repeated episodes of airway obstruction cause intermittent hypoxia and reoxygenation cycles that trigger oxidative stress and systemic inflammatory responses[4].

Increasing evidence suggests that chronic low-grade inflammation plays a central role in the development and progression of OSA-related complications. Intermittent hypoxia activates inflammatory pathways and stimulates the production of various cytokines and inflammatory mediators[5]. Among these, interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) are important pro-inflammatory cytokines that are frequently elevated in patients with OSA. These cytokines are involved in endothelial dysfunction, insulin resistance, and the development of atherosclerosis, thereby contributing to increased cardiometabolic risk. In contrast, interleukin-10 (IL-10) functions as an anti-inflammatory cytokine that regulates immune responses and suppresses excessive inflammatory activity. An imbalance between pro-inflammatory and anti-inflammatory cytokines may therefore play a key role in the systemic complications associated with OSA[6].

OSA has been strongly linked with several cardiometabolic conditions, including hypertension, type 2 diabetes mellitus, dyslipidemia, obesity, and metabolic syndrome. The repeated hypoxic events experienced by OSA patients activate the sympathetic nervous system and inflammatory signaling pathways, which promote vascular dysfunction and metabolic disturbances. Consequently, individuals with untreated OSA are at significantly higher risk of developing cardiovascular diseases such as coronary artery disease, stroke, and heart failure[7,8].

Although previous studies have demonstrated elevated inflammatory cytokine levels in patients with OSA, limited clinical research has examined the combined relationship between inflammatory biomarkers, upper airway structural abnormalities, and cardiometabolic risk factors. Understanding these associations may provide valuable insights into the underlying mechanisms linking

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OSA with systemic diseases and may help identify potential biomarkers for early diagnosis and risk stratification[9,10].

Therefore, the present study was conducted to evaluate serum levels of inflammatory cytokines including IL-6, TNF-α, and IL-10 in patients with obstructive sleep apnea. In addition, the study aimed to assess upper airway obstructive factors and determine their association with cardiometabolic risk indicators. By exploring these relationships, the study seeks to improve understanding of the inflammatory and anatomical mechanisms contributing to OSA and its related systemic complications[11,12].

MATERIALS AND METHODS

Study Design and Setting: This cross-sectional clinical study was conducted to evaluate inflammatory cytokines and upper airway obstructive factors in patients with obstructive sleep apnea and their association with cardiometabolic risk. The study was carried out at the Department of Pulmonology and ENT at Nishtar Hospital, Multan, and Isra University Hospital, Hyderabad, Pakistan. These tertiary care hospitals provide specialized diagnostic and clinical services for respiratory and sleep-related disorders, allowing the recruitment of patients presenting with symptoms suggestive of obstructive sleep apnea.

Study Duration and Sample Size: The study was conducted over a period of eight months from June 2022 to February 2023. A total of 100 participants were included in the study using a non-probability consecutive sampling technique. Patients diagnosed with obstructive sleep apnea constituted the study group, while individuals without symptoms of sleep apnea served as controls for comparison. All participants were selected after clinical evaluation and fulfillment of the eligibility criteria.

Inclusion and Exclusion Criteria: Participants aged 30 to 65 years with clinically suspected and confirmed obstructive sleep apnea were included in the study. Diagnosis was based on clinical assessment and sleep study findings consistent with obstructive sleep apnea. Individuals who were willing to participate and provided informed consent were included in the study. Participants were excluded if they had chronic inflammatory diseases, autoimmune disorders, active infections, malignancies, or were receiving anti-inflammatory or immunosuppressive therapy, as these conditions could influence inflammatory cytokine levels. Pregnant women were also excluded from the study.

Clinical Assessment and Upper Airway Examination: All participants underwent a detailed clinical evaluation. Demographic and anthropometric data including age, gender, body mass index (BMI), neck circumference, and blood pressure were recorded. Clinical examination of the upper airway was performed by ENT specialists to identify structural abnormalities that may contribute to airway obstruction during sleep. Upper airway obstructive factors assessed included tonsillar hypertrophy, nasal septal deviation, nasal obstruction, elongated uvula, and soft palate enlargement. These anatomical abnormalities were documented because they are known to increase the risk of pharyngeal airway collapse during sleep and contribute to the pathophysiology of obstructive sleep apnea.

Laboratory Analysis of Inflammatory Cytokines: Venous blood samples were collected from all participants after overnight fasting. Blood samples were centrifuged to separate serum, which was then stored under appropriate laboratory conditions until analysis. Serum levels of inflammatory cytokines including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-10 (IL-10) were measured using the enzyme-linked immunosorbent assay (ELISA) technique according to the manufacturer's instructions. These cytokines were selected because IL-6 and TNF-α are key pro-inflammatory mediators associated with systemic inflammation, while IL-10 acts as an anti-inflammatory cytokine that regulates immune responses.

Assessment of Cardiometabolic Risk Factors: To evaluate cardiometabolic risk among the study participants, several biochemical and clinical parameters were assessed. These included fasting blood glucose levels, serum lipid profile (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides), blood pressure, waist circumference, and body mass index. These parameters were selected because obstructive sleep apnea has been strongly associated with metabolic syndrome, insulin resistance, dyslipidemia, and hypertension.

Ethical Considerations: Ethical approval for the study was obtained from the institutional ethical review committees of Nishtar Hospital Multan and Isra University Hospital Hyderabad. The study was conducted in accordance with ethical principles for human research. Written informed consent was obtained from all participants prior to their inclusion in the study, and confidentiality of patient information was strictly maintained throughout the research process.

Statistical Analysis: All collected data were entered and analyzed using the Statistical Package for Social Sciences (SPSS) version 25. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using the independent sample t-test for continuous variables and the chi-square test for categorical variables. Correlation analysis was conducted to determine the relationship between inflammatory cytokine levels and cardiometabolic risk parameters. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics of the Study Population: A total of 100 participants were included in the study, comprising 60 patients diagnosed with obstructive sleep apnea (OSA) and 40 healthy individuals serving as controls. The mean age of the OSA group was 48.3 ± 8.6 years, while the control group had a mean age of 45.9 ± 7.8 years, showing no statistically significant difference between the groups ($p = 0.09$).

Gender distribution showed that male participants were more prevalent in the OSA group compared with females, which is consistent with the known epidemiology of obstructive sleep apnea. The mean body mass index (BMI) among OSA patients was significantly higher (31.4 ± 4.5 kg/m²) compared with controls (26.2 ± 3.7 kg/m², $p < 0.001$). Additionally, neck circumference and waist circumference were markedly higher among OSA patients, indicating increased obesity-related risk. The prevalence of hypertension and diabetes mellitus was also higher among patients with OSA compared with healthy controls. The findings presented in Table 1 demonstrate that individuals with obstructive sleep apnea have significantly higher body mass index, neck circumference, and waist circumference compared with controls. Furthermore, cardiometabolic conditions such as hypertension and diabetes were more common in the OSA group, indicating increased metabolic risk among these patients.

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter	OSA Patients (n=60)	Controls (n=40)	p-value
Age (years)	48.3 ± 8.6	45.9 ± 7.8	0.09
Male	39 (65%)	22 (55%)	0.29
Female	21 (35%)	18 (45%)	
BMI (kg/m²)	31.4 ± 4.5	26.2 ± 3.7	<0.001
Neck circumference (cm)	41.2 ± 3.4	36.8 ± 2.9	<0.001
Waist circumference (cm)	102.5 ± 9.7	91.4 ± 8.2	<0.001
Hypertension	25 (41%)	6 (15%)	0.01
Diabetes mellitus	18 (30%)	5 (12%)	0.04

Cardiometabolic Risk Parameters: Evaluation of cardiometabolic parameters revealed significant metabolic differences between OSA patients and healthy controls. The mean fasting blood glucose level in the OSA group was 112.6 ± 18.4 mg/dL, whereas the control group demonstrated a lower mean value of 96.3 ± 12.7 mg/dL ($p < 0.001$).

Similarly, lipid profile analysis revealed significantly elevated total cholesterol, LDL cholesterol, and triglyceride levels in OSA patients. In contrast, HDL cholesterol levels were significantly lower

in the OSA group, indicating a higher risk of cardiovascular disease among these individuals. The results shown in Table 2 demonstrate that obstructive sleep apnea is strongly associated with metabolic disturbances including elevated glucose levels, dyslipidemia, and reduced protective HDL cholesterol levels.

Table 2: Cardiometabolic Risk Parameters in Study Participants

Parameter	OSA Patients (n=60)	Controls (n=40)	p-value
Fasting glucose (mg/dL)	112.6 ± 18.4	96.3 ± 12.7	<0.001
Total cholesterol (mg/dL)	208.5 ± 36.7	182.3 ± 29.4	0.002
LDL cholesterol (mg/dL)	132.4 ± 28.6	109.8 ± 24.5	0.001
HDL cholesterol (mg/dL)	38.6 ± 7.9	45.2 ± 8.4	0.003
Triglycerides (mg/dL)	176.8 ± 42.1	134.6 ± 36.5	<0.001

Serum Inflammatory Cytokine Levels: Serum inflammatory cytokine analysis demonstrated significant differences between OSA patients and controls. The mean IL-6 concentration in OSA patients was 7.9 ± 2.3 pg/mL, whereas controls showed significantly lower levels (3.7 ± 1.5 pg/mL, $p < 0.001$).

Similarly, TNF-α levels were significantly elevated in OSA patients (9.4 ± 2.6 pg/mL) compared with controls (4.3 ± 1.7 pg/mL, $p < 0.001$).

In contrast, the anti-inflammatory cytokine IL-10 was significantly lower in OSA patients (2.2 ± 0.9 pg/mL) compared with controls (3.5 ± 1.1 pg/mL, $p = 0.02$). These findings indicate the presence of a systemic inflammatory state in individuals with obstructive sleep apnea. The results in Table 3 demonstrate that patients with obstructive sleep apnea exhibit significantly increased levels of pro-inflammatory cytokines along with reduced levels of anti-inflammatory cytokines.

Table 3: Serum Inflammatory Cytokine Levels

Cytokine	OSA Patients (n=60)	Controls (n=40)	p-value
IL-6 (pg/mL)	7.9 ± 2.3	3.7 ± 1.5	<0.001
TNF-α (pg/mL)	9.4 ± 2.6	4.3 ± 1.7	<0.001
IL-10 (pg/mL)	2.2 ± 0.9	3.5 ± 1.1	0.02

Upper Airway Obstructive Factors: Clinical examination of the upper airway revealed that structural abnormalities were significantly more common among OSA patients. Tonsillar hypertrophy was observed in 22 patients (36%), whereas only 4 individuals (10%) in the control group showed similar findings.

Similarly, nasal obstruction or nasal septal deviation was present in 23 OSA patients (38%), compared with 5 controls (12%). In addition, elongated uvula and soft palate enlargement were detected in approximately 30% of OSA patients, while these anatomical variations were uncommon among healthy controls. The findings presented in Table 4 indicate that structural abnormalities of the upper airway are significantly associated with obstructive sleep apnea and contribute to increased airway resistance and collapse during sleep.

Table 4: Upper Airway Obstructive Factors

Upper Airway Factor	OSA Patients (n=60)	Controls (n=40)
Tonsillar hypertrophy	22 (36%)	4 (10%)
Nasal obstruction / septal deviation	23 (38%)	5 (12%)
Elongated uvula	18 (30%)	3 (8%)
Soft palate enlargement	19 (32%)	3 (7%)

Association Between Cytokines and Cardiometabolic Risk: Further analysis revealed that serum IL-6 and TNF-α levels showed significant positive correlations with BMI, fasting blood glucose, triglyceride levels, and systolic blood pressure among OSA patients ($p < 0.05$). Patients with higher cytokine levels also tended to have more severe airway obstruction and greater cardiometabolic risk.

Conversely, IL-10 levels demonstrated an inverse relationship with inflammatory markers and metabolic risk factors, suggesting that reduced anti-inflammatory activity may contribute to the persistent inflammatory state observed in obstructive sleep apnea.

Overall, the results of this study indicate that patients with obstructive sleep apnea exhibit increased systemic inflammation, upper airway structural abnormalities, and a higher prevalence of cardiometabolic risk factors compared with healthy individuals, highlighting the important role of inflammatory cytokines in the pathophysiology and systemic consequences of the disease.

DISCUSSION

Obstructive sleep apnea (OSA) is increasingly recognized as a systemic disorder characterized not only by repeated episodes of upper airway obstruction during sleep but also by chronic low-grade inflammation and metabolic disturbances[9]. The present study evaluated inflammatory cytokines (IL-6, TNF-α, and IL-10) along with upper airway obstructive factors and cardiometabolic risk indicators among patients with OSA. The findings demonstrated that individuals with obstructive sleep apnea had significantly elevated levels of pro-inflammatory cytokines and a higher prevalence of cardiometabolic abnormalities compared with healthy controls[10].

In this study, OSA patients exhibited significantly higher body mass index, neck circumference, and waist circumference, which are well-established risk factors for the development of obstructive sleep apnea[11]. Obesity plays a central role in the pathogenesis of OSA because increased adipose tissue around the neck and pharyngeal structures leads to narrowing of the upper airway and increased airway collapsibility during sleep. Furthermore, obesity is strongly associated with systemic inflammation and metabolic dysregulation, which may exacerbate the severity of OSA and its complications[12].

The results of the present study also demonstrated a significantly higher prevalence of hypertension, elevated fasting glucose levels, and dyslipidemia among OSA patients compared with the control group[13]. These findings support previous evidence indicating that obstructive sleep apnea is strongly associated with cardiometabolic disorders such as hypertension, metabolic syndrome, and type 2 diabetes mellitus. Repeated episodes of intermittent hypoxia in OSA activate the sympathetic nervous system and stimulate oxidative stress pathways, which contribute to endothelial dysfunction and metabolic abnormalities[14].

One of the most important findings of this study was the significant elevation of pro-inflammatory cytokines IL-6 and TNF-α in patients with obstructive sleep apnea. These cytokines are key mediators of systemic inflammation and are known to play an important role in the pathogenesis of cardiovascular disease and metabolic disorders[15]. Intermittent hypoxia associated with OSA stimulates inflammatory signaling pathways that increase the production of IL-6 and TNF-α, leading to chronic systemic inflammation. Elevated levels of these cytokines may contribute to the development of insulin resistance, vascular inflammation, and atherosclerosis, thereby increasing the risk of cardiovascular complications in patients with OSA[16].

Conversely, the present study demonstrated reduced levels of the anti-inflammatory cytokine IL-10 among OSA patients compared with healthy controls. IL-10 plays an important regulatory role in suppressing excessive inflammatory responses and maintaining immune homeostasis. A reduction in IL-10 levels suggests an imbalance between pro-inflammatory and anti-inflammatory mechanisms in patients with obstructive sleep apnea, which may further promote systemic inflammation and disease progression[17,18].

Another important aspect of the study was the evaluation of upper airway structural abnormalities, which were found to be significantly more prevalent among patients with obstructive sleep apnea. Conditions such as tonsillar hypertrophy, nasal obstruction, elongated uvula, and soft palate enlargement contribute to narrowing of the upper airway and increase the likelihood of airway collapse during sleep. These anatomical factors play a critical role in the development and severity of OSA and may also influence the degree of hypoxia and inflammatory response experienced by patients[19,20].

Correlation analysis further demonstrated that increased levels of IL-6 and TNF-α were positively associated with body mass

index, fasting glucose levels, triglyceride levels, and blood pressure, indicating a strong relationship between systemic inflammation and cardiometabolic risk in OSA patients. These findings suggest that inflammatory cytokines may serve as potential biomarkers for identifying patients at increased risk of metabolic and cardiovascular complications[5-9].

Overall, the findings of this study highlight the complex interaction between upper airway anatomical abnormalities, systemic inflammation, and metabolic disturbances in the pathophysiology of obstructive sleep apnea. Early recognition of these factors may improve risk assessment and guide appropriate management strategies for patients with OSA[15-20].

CONCLUSION

The present study demonstrated that patients with obstructive sleep apnea exhibit significantly elevated levels of pro-inflammatory cytokines (IL-6 and TNF-α) along with reduced levels **of** the anti-inflammatory cytokine IL-10 compared with healthy individuals. These inflammatory changes were strongly associated with upper airway obstructive factors and increased cardiometabolic risk parameters, including obesity, hypertension, dyslipidemia, and impaired glucose metabolism. The findings suggest that systemic inflammation plays a critical role in linking obstructive sleep apnea with cardiovascular and metabolic complications. Structural abnormalities of the upper airway further contribute to disease severity by promoting airway collapse and intermittent hypoxia during sleep. Therefore, assessment of inflammatory cytokines along with evaluation of upper airway anatomy may provide valuable information for early identification, risk stratification, and management of patients with obstructive sleep apnea. Early diagnosis and appropriate therapeutic interventions may help reduce the long-term cardiometabolic consequences associated with this disorder.

Availability of Data and Materials: The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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Authors' Contributions: S.G. contributed to conceptualization, methodology, investigation, and drafting of the manuscript. N.A. participated in data collection, laboratory analysis, and manuscript preparation. H.A. contributed to clinical evaluation of patients, supervision, and data interpretation. W.Q. assisted in statistical analysis, data interpretation, and manuscript revision. R.A. contributed to literature review, data compilation, and manuscript editing. S.J. contributed to study supervision, critical review, and final approval of the manuscript. All authors read and approved the final version of the manuscript.

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