Original Article

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Biochemical Correlates of the Respiratory Disturbance Index among Patients with Obstructive Sleep Apnea in a Tertiary Care Setting

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Abstract

Background: Obstructive Sleep Apnea (OSA) is marked by recurrent upper airway obstruction during sleep, leading to intermittent hypoxia and oxidative stress. The Respiratory Disturbance Index (RDI) a measure of OSA severity, may influence metabolic homeostasis through biochemical alterations. This study aimed to assess the biochemical profile of OSA patients and examine associations between RDI and biochemical parameters.

Materials and methods: A cross-sectional study was conducted at the Department of Respiratory Medicine, Bangladesh Medical University (BMU). Seventy-four OSA patients were included and categorized as severe OSA (RDI ≥30) and non-severe OSA (RDI <30). Ater overnight fasting, venous blood samples were collected for Fasting Blood Glucose (FBG) serum lipid profile [Total cholesterol, triglycerides, Low Density Lipoprotein-Cholesterol (LDL-C) High Density Lipoprotein-Cholesterol (HDL-C)] serum creatinine, serum electrolytes, serum Alanine Aminotransferase (ALT) and calcium.

Results: Patients with RDI ≥30 showed significantly higher FBG (120.2 vs. 110.4 mg/dl, p=0.016), triglycerides (257.1 vs. 204.6 mg/dl, p=0.002) LDL-C (159.8 vs. 130.4 mg/dl, p<0.001) and total cholesterol (276.9 vs. 195.8 mg/dl, p<0.001) along with elevated serum potassium (4.6 vs. 4.4 mmol/L, p=0.008) and calcium (8.8 vs. 8.5 mg/dl, p=0.013). HDL-C was lower in this group (37.9 vs. 39.9 mg/dl, p=0.056). RDI correlated positively with FBG (r=0.325, p=0.005), triglycerides (r=0.351, p=0.002) LDL-C (r=0.430, p<0.001) total cholesterol (r=0.588, p<0.001) potassium (r=0.315, p=0.006) and ALT (r=0.241, p=0.039). Regression analysis identified RDI as an independent predictor of fasting glucose (β=0.250, p=0.011), triglycerides (β=0.368, p=0.001), and LDL-C (β=0.382, p<0.001).

Conclusion: Higher RDI is strongly associated with dyslipidemia, hyperglycemia, and selected biochemical disturbances in OSA patients. RDI may serve as a predictor of metabolic dysfunction, emphasizing the importance of biochemical monitoring alongside respiratory evaluation.

Key words: Biochemical profile; Dyslipidemia; Fasting glucose; Metabolic dysfunction; Obstructive sleep apnea; Respiratory disturbance index.

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Introduction

OSA is a highly prevalent sleep-related breathing disorder characterized by recurrent episodes of upper airway collapse during sleep, resulting in apnea or hypopnea.[1,2] Beyond sleep disturbance, OSA produces pathophysiological consequences that extend to systemic health. Each obstructive event results in hypoventilation, oxyhemoglobin desaturation and, in prolonged cases, hypercapnia. Cycles of hypoxia and reoxygenation activate carotid body chemoreceptors and the sympathetic nervous system, triggering oxidative stress, systemic inflammation and metabolic dysfunction.[3]

These repetitive neurohormonal insults are strongly implicated in glucose dysregulation, insulin resistance, dyslipidemia, hepatic steatosis, and abnormal fat deposition.[4] Over time, such biochemical disturbances contribute to an increased risk of major non-communicable diseases, including diabetes mellitus, Non-Alcoholic Fatty Liver Disease (NAFLD) coronary artery disease, cerebrovascular disease, and metabolic bone disorders. Thus, OSA represents both a sleep disorder and a systemic metabolic condition with profound implications for morbidity and mortality.[4]

OSA affects a substantial proportion of adults worldwide, with prevalence estimates ranging from 15–34% across countries such as India, China, the USA, and South Korea.[5-8] In Bangladesh, population-based data show rates of 11.9% for OSAH and 3.3% for OSAS, with higher prevalence among men.[9] Globally, systematic

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reviews estimate the overall prevalence at nearly 36%, underscoring its significant public health impact.[10]

The RDI, defined as the number of apneas, hypopneas, and respiratory effort-related arousals per hour of sleep, is widely used to classify OSA severity into mild, moderate and severe categories.[11] RDI not only reflects disease burden but also appears to correlate with biochemical disturbances. A standard metabolic profile, including fasting blood glucose, lipid parameters, electrolytes, creatinine, ALT and calciumprovides a window into systemic metabolic health.[12] Alterations in these markers have been consistently associated with OSA. For instance, increasing RDI has been correlated with impaired fasting glucose and greater insulin resistance, derangements in lipid profile with elevated triglycerides, LDL-C and total cholesterol but reduced HDL-C, hepatic dysfunction linked to NAFLD and renal impairment.[13-16]

Despite these observations, most evidence comes from studies in Western or East Asian populations. Data from South Asian countries, including Bangladesh, remain scarce. Given the growing prevalence of OSA in Bangladesh and the associated burden of metabolic disorders, evaluating the biochemical correlates of RDI in this population is both timely and clinically significant. Such data may contribute to improved screening, early detection of metabolic dysfunction, and preventive strategies for reducing non-communicable disease burden in OSA patients. Therefore, present study was conducted to assess the status of the metabolic profile of OSA patients at a tertiary care hospital in Bangladesh and to determine the association of RDI with metabolic profile.

Materials and methods

The cross-sectional analytical study was conducted over a period of 12 months in the Sleep Clinic, Sleep Study Laboratory, and Department of Respiratory Medicine at BMU, Dhaka. Ethical approval had been obtained from the Institutional Review Board of Bangabandhu Sheikh Mujib Medical University (Memo No. BSMMU/2023/ 14063, Date: 16-11-2023). Written informed consent was collected from each participant after the study objectives and procedures had been explained.

Patients aged 18 years and above with a diagnosis of OSA confirmed by in-laboratory PSG were included. Individuals were excluded if they were pregnant, had previously received treatment with continuous positive airway pressure or oral appliances, or suffered from critical illness such as acute coronary syndrome, respiratory failure, or acute stroke. A non-probability consecutive sampling method was applied.

OSA was defined as the presence of five or more predominantly obstructive respiratory events (Apneas, hypopneas, or Respiratory Effort-related Arousals [RERAs]) per hour of sleep recorded on polysomnography.[11] The RDI was calculated as the number of apneas, hypopneas, and RERAs per hour of sleep categorized as severe if the scores were ≥30.[11] The metabolic profile was defined as a panel of biochemical tests that reflected systemic chemical balance and metabolic function.[12] Tests included in this study were FBG, fasting serum lipid profile, serum creatinine, serum electrolytes, ALT and serum calcium. Data were collected using a structured questionnaire capturing demographic, clinical, and laboratory characteristics. After at least eight hours of fasting, venous blood samples were obtained. All biochemical analyses were performed in the Department of Biochemistry, BMU, using an automated spectrophotometric method.

Following data collection, all entries were processed and analyzed using Statistical Package for Social Science (SPSS) version 26 (SPSS Inc., Chicago, IL, USA). Categorical variables were summarized as frequencies and percentages, while continuous variables were expressed as mean and standard deviation. Group comparisons of continuous variables were performed with independent sample t-tests. Associations between categorical variables and RDI were examined using Chi-square tests. Correlation between RDI and biochemical variables was assessed with Pearson's correlation coefficient, and linear regression analyses were applied to evaluate predictive associations. A forward stepwise multiple regression model was employed to identify major determinants of the metabolic profile. A 95% confidence interval was adopted, and statistical significance was set at a p-value of less than 0.05.

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Results

A total of 74 patients diagnosed with OSA were enrolled in the study. The mean age of the participants was 49.1 ± 13.5 years, with an age range of 18 to 80 years. Males accounted for 56.8% of the study population, corresponding to a male-to-female ratio of 1.3:1. Based on RDI values, 38 participants (51.4%) were classified as having RDI <30 events/hour, while 36 participants (48.6%) had RDI ≥30 events/hour.

Comparison of the metabolic profile between patients with RDI <30 and those with RDI ≥30 is presented in Table I. Patients with RDI ≥30 demonstrated significantly higher fasting blood glucose levels (120.2 ± 16.8 vs. 110.4 ± 17.2 mg/dl, p=0.016) triglycerides (257.1 ± 79.0 vs. 204.6 ± 59.0 mg/dl, p=0.002) LDL-C (159.8 ± 36.7 vs. 130.4 ± 25.8 mg/dl, p<0.001) and total cholesterol (276.9 ± 78.3 vs. 195.8 ± 40.0 mg/dl, p<0.001). Serum potassium (4.6 ± 0.4 vs. 4.4 ± 0.5 mmol/L, p=0.008) and serum calcium (8.8 ± 0.5 vs. 8.5 ± 0.4 mg/dl, p=0.013) were also significantly elevated in the higher RDI group. Although mean HDL-C was lower in patients with RDI ≥30 compared to those with RDI <30 (37.9 ± 4.3 vs. 39.9 ± 4.6 mg/dl), the difference did not reach statistical significance (p=0.056). No significant differences were observed in serum creatinine, sodium, chloride, bicarbonate, or ALT levels between the two groups (p>0.05).

Table I Comparison of metabolic profile of OSA patients according to RDI (n=74)

Metabolic profile	RDI		p-value*
	<30(n = 38)	≥30(n = 36)	
Fasting blood glucose (mg/dl)	110.4±17.2	120.2±16.8	0.016
Triglyceride (mg/dl)	204.6±59.0	257.1±79.0	0.002
HDL-C (mg/dl)	39.9±4.6	37.9±4.3	0.056
LDL-C (mg/dl)	130.4±25.8	159.8±36.7	<.0.001
Total Cholesterol (mg/dl)	195.8±40.0	276.9±78.3	<.0.001
Serum Creatinine (mg/dl)	0.9±0.2	1.0±0.2	0.143
Na^+ (mmol/L)	138.6±2.4	138.6±1.5	0.932
K^+ (mmol/L)	4.4±0.5	4.6±0.4	0.008
Cl^- (mmol/L)	101.0±2.8	100.1±2.7	0.162
$HCO3^-$ (mmol/L)	26.6±1.0	26.4±1.4	0.347
ALT (U/L)	52.2±10.5	56.6±12.1	0.101
Serum Calcium (mg/dl)	8.5±0.4	8.8±0.5	0.013

Data were expressed as Mean±SD *Unpaired t-test, p<0.05 was considered as a level of significant.

Correlation analysis demonstrated that RDI was significantly and positively correlated with fasting blood glucose (r=0.325, p=0.005) triglycerides (r=0.351, p=0.002) LDL-C (r=0.430, p<0.001) total cholesterol (r=0.588, p<0.001) serum potassium (r=0.315, p=0.006) and ALT (r=0.241, p=0.039). A negative but non-significant correlation was observed between RDI and HDL-C (r=–0.212, p=0.070). No significant correlations were found between RDI and serum creatinine, sodium, chloride, bicarbonate or calcium (p>0.05) (Table II).

Table II Correlation of RDI with metabolic profile (n = 74)

Variables	Pearson's correlation test	
	r value	p-value
Fasting blood glucose (mg/dl)	+0.325*	0.005
Triglyceride (mg/dl)	+0.351*	0.002
HDL-C (mg/dl)	-0.212	0.070
LDL-C (mg/dl)	+0.430*	<0.001
Total Cholesterol (mg/dl)	+0.588*	<0.001
Serum Creatinine (mg/dl)	+0.202	0.084
Na^+ (mmol/L)	-0.004	0.971
K^+ (mmol/L)	0.315*	0.006
Cl^- (mmol/L)	-0.127	0.282
$HCO3^-$ (mmol/L)	-0.092	0.434
ALT (U/L)	+0.241*	0.039
Serum Calcium (mg/dl)	+0.217*	0.063

Multivariate regression analysis revealed that both diabetes mellitus (β=0.573, p<0.001) and RDI (β=0.250, p=0.011) were significant independent predictors of fasting blood glucose. Age, Body Mass Index (BMI) and hypertension showed no significant associations (p>0.05). The model explained 40.6% of the variance in fasting blood glucose (Adjusted R^2 = 0.406).

Table III Multivariate regression analysis of predictors of fasting blood glucose in OSA patients (n = 74)

Variables	Unstandardized Coefficients (b)	Std. Error Coefficients (β)	Standardized	p-value	95% CI for b
Age (Years)	0.042	0.140	0.032	0.764	–0.237 to 0.321
BMI (kg/m²)	–0.005	0.244	–0.002	0.985	–0.491 to 0.481
Hypertension	0.654	3.914	0.019	0.868	–7.156 to 8.463
Diabetes Mellitus	20.771	3.560	0.573	<0.001	13.667 to 27.874
RDI (Events/h)	0.165	0.063	0.250	0.011	0.039 to 0.291

R^2 = 0.406 (adjusted R^2). Dependent variable: Fasting blood glucose (mg/dl).

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In multivariate regression analysis, RDI (β=0.368, p=0.001) and diabetes mellitus (β=0.274, p=0.015) emerged as significant independent predictors of triglyceride levels. Age, BMI, and hypertension did not show significant associations (p>0.05). The model accounted for 25.2% of the variance in triglyceride levels (Adjusted R^2 = 0.252).

Table IV Multivariate regression analysis of predictors of triglyceride levels in OSA patients (n = 74)

Variables	Unstandardized Coefficients (b)	Std. Error	Standardized Coefficients (β)	p-value	95% CI for b
Age (Years)	–0.070	0.660	–0.013	0.916	–1.386 to 1.247
BMI (kg/m²)	–2.134	1.149	–0.198	0.068	–4.427 to 0.160
Hypertension	27.536	18.467	0.187	0.141	–9.314 to 64.386
Diabetes Mellitus	41.771	16.798	0.274	0.015	8.252 to 75.290
RDI (Events/h)	1.020	0.298	0.368	0.001	0.426 to 1.614

R^2 = 0.252 (Adjusted R^2). Dependent variable: Triglyceride (mg/dl).

Multivariate regression analysis identified diabetes mellitus (β=0.490, p<0.001) and RDI (β=0.382, p<0.001) as significant independent predictors of LDL-C levels. Age, BMI and hypertension were not significantly associated with LDL-C (p>0.05). The model explained 38.4% of the variance in LDL-C concentrations (Adjusted R^2 = 0.384).

Table V Multivariate regression analysis of predictors of LDL-C in OSA patients (n = 74)

Variables	Unstandardized Coefficients (b)	Std. Error	Standardized Coefficients (β)	p-value	95% CI for b
Age (Years)	–0.145	0.281	–0.057	0.607	–0.706 to 0.416
BMI (kg/m²)	–0.323	0.490	–0.064	0.511	–1.301 to 0.654
Hypertension	2.493	7.869	0.036	0.752	–13.210 to 18.195
Diabetes Mellitus	35.087	7.158	0.490	<0.001	20.803 to 49.370
RDI (Events/h)	0.497	0.127	0.382	<0.001	0.244 to 0.751

R^2 = 0.384 (Adjusted R^2). Dependent variable: LDL-C (mg/dl).

Discussion

In this study, patients with higher RDI (≥30) demonstrated significantly elevated fasting blood glucose, triglycerides, LDL-C and total cholesterol compared to those with lower RDI values, while HDL-C showed a non-significant inverse relationship. These findings are consistent with prior studies suggesting that intermittent hypoxia and sympathetic activation in OSA contribute to dyslipidemia and impaired glucose metabolism.[17,18] Multivariate regression confirmed that RDI was an independent predictor of triglyceride and LDL-C levels, reinforcing the impact of OSA severity on lipid dysregulation. The strong correlation between RDI and total cholesterol (r=0.588, p<0.001) observed in this study further underscores the role of OSA in promoting atherogenic profiles, in agreement with Wu et al. and Drager et al.[19,20]

Biochemical markers beyond glucose and lipids also demonstrated significant associations. Serum potassium was modestly but significantly elevated with increasing RDI, an observation that raises clinical concern given the risk of arrhythmias. Similarly, ALT showed a positive correlation with RDI, consistent with studies linking OSA-induced hypoxia to hepatic dysfunction and non-alcoholic fatty liver disease.[21] Serum calcium was significantly higher in the severe OSA group, although correlation analysis did not confirm a direct association, suggesting that calcium homeostasis in OSA is likely influenced by multiple interacting factors including vitamin D, parathyroid hormone and obesity.[22]

Conversely, no significant differences were found for serum creatinine, sodium, chloride or bicarbonate levels across RDI groups. This finding differs from studies such as Zamarron et al. which linked OSA severity to renal impairment in diabetic kidney disease, highlighting the role of population heterogeneity and comorbidities in shaping outcomes.[23] Taken together, the biochemical results of the present study emphasize that OSA severity is strongly linked with metabolic dysregulation, particularly involving glucose and lipid metabolism, with additional signals of hepatic and electrolyte imbalance.

Importantly, while correlation analyses suggested associations between RDI and metabolic variables, regression models revealed that comorbid conditions, particularly diabetes mellitus, exerted stronger influences on fasting blood glucose and LDL-C. This highlighted the multifactorial nature of metabolic abnormalities in OSA, where disease severity, underlying comorbidities, and lifestyle factors all play interrelated roles. The findings align with existing literature that emphasizes OSA as both an independent risk factor and a disease that interacts synergistically with obesity, diabetes, and cardiovascular risk profiles.[24,25]

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Limitations

Several limitations should be considered in interpreting the study findings. First, the study was conducted on a relatively small sample. Second, the cross-sectional design prevents establishment of causal relationships between RDI and biochemical outcomes. Third, lifestyle data, including smoking and diet, were self-reported, introducing the possibility of recall bias. Finally, the single-center design limits the external validity of the results to broader populations.

Conclusion

This study demonstrated that higher RDI, indicative of more severe OSA, was significantly associated with adverse biochemical alterations, including hyperglycemia, dyslipidemia and disturbances in electrolytes and liver function. While regression analyses highlighted the significant contribution of comorbidities such as diabetes, RDI remained an independent predictor of triglycerides and LDL-C. The findings suggest that OSA severity plays a critical role in metabolic dysfunction, reinforcing the need for integrated screening and management strategies that address both respiratory and metabolic health in OSA patients.

Recommendations

Routine biochemical screening should be incorporated into the management of moderate to severe OSA to enable early detection of metabolic abnormalities. Future large-scale longitudinal studies are recommended to clarify causal relationships and strengthen evidence for comprehensive management strategies.

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Contribution of authors

MHK-Conception, acquisition of data, data analysis, drafting & final approval.

MMR-Design, interpretation of data, critical revision & final approval.

IUR-Acquisition of data, critical revision, drafting & final approval.

Disclosure

All the authors declared no conflict of interest.

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