

The mean coagulation profile in different severity groups of obstructive sleep apnea at tertiary care hospital, Karachi

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ABSTRACT

Objective: To determine the mean coagulation profile in different severity groups of obstructive sleep apnea

Material and Methods: This cross-sectional study was conducted at the Department of Pulmonology, Jinnah Postgraduate Medical Centre from December 31, 2020 to June 30, 2021. Each participant underwent detailed history, clinical evaluation, and overnight polysomnography with EEG, EMG, airflow, oxygen saturation, ECG, and video monitoring to assess apnea–hypopnea index. Apnea and hypopnea were defined by standard criteria. After fasting, venous blood samples were collected for platelet count, PT, aPTT, and INR. Anthropometric and demographic data were documented using a structured proforma. Data were analyzed using SPSS Version 20. A p-value of ≤ 0.05 was considered statistically significant.

Results: A total of 23 participants were included in this study. Platelet count was significantly lower in the mild group (135.88 ± 91.88) compared to moderate (221.89 ± 45.93) and severe cases (222.50 ± 25.86) ($p = 0.020$). Prothrombin time increased progressively with severity, from 10.70 ± 0.62 in mild to 12.41 ± 1.09 in severe cases ($p = 0.003$). In contrast, thromboplastin time showed no significant difference among groups ($p = 0.852$). The distribution of demographic and socioeconomic variables showed no significant association with the severity of obstructive sleep apnea.

Conclusions: In conclusion, increasing severity of obstructive sleep apnea is associated with significant hematological alterations, particularly increased platelet counts and prolonged prothrombin time with elevated INR.

Keywords: Obstructive sleep apnea, Platelet count, Prothrombin time, International normalized ratio

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INTRODUCTION

Obstructive sleep apnea (OSA) is a serious disorder linked with metabolic, cardiovascular, and neurocognitive complications [1]. It belongs to the spectrum of sleep-related breathing disorders and is marked by recurrent episodes of partial or complete upper airway blockage, leading to oxygen desaturation and brief arousals [2]. While OSA is recognized as a significant cause of morbidity and mortality in

developed nations, its burden in developing countries has only recently gained attention. Given its considerable impact on health if left undiagnosed, the establishment of dedicated sleep disorder centers remains essential for timely diagnosis and management [3].

Obstructive sleep apnea (OSA) involves episodes of complete airflow cessation (apnea) and partial airflow reduction (hypopnea) [4]. These events occur when the upper airway, particularly the pharynx, collapses for more than 10 seconds due to decreased muscle tone. Such interruptions trigger brief awakenings that restore airway patency [5]. Typically, patients begin snoring shortly after sleep onset, with the sound gradually intensifying until an apnea episode occurs, followed by a loud gasp or snort

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as breathing resumes [6]. Although overall sleep duration is not reduced, OSA patients spend significantly more time in stage 1 non-REM sleep (30–50% compared to the normal 10%) and exhibit diminished slow-wave sleep (stages 3 and 4) [7]. The underlying mechanisms include reduced neuromuscular activity at sleep onset and altered central respiratory regulation [8].

If left untreated, obstructive sleep apnea (OSA) elevates the risk of overall mortality, particularly from cardiovascular causes, and is strongly linked to complications such as stroke, myocardial infarction, hypertension, arrhythmias, and heart failure [9]. A proposed mechanism is the promotion of increased blood clotting tendency, as individuals with OSA often exhibit multiple factors contributing to a hypercoagulable state. Hypoxemia during apneic episodes has been shown to trigger the release of inflammatory mediators that alter vascular and hematologic function, resulting in systemic hypercoagulability. This hypercoagulable state is thought to represent a crucial mechanistic link between OSA, inflammation, and cardiovascular complications [10].

The aim of this study was to determine the mean coagulation profile in different severity groups of obstructive sleep apnea.

MATERIAL AND METHODS

This cross-sectional study took place at the Department of Pulmonology, Jinnah Postgraduate Medical Centre from December 31, 2020 to June 30, 2021, having received approval from the Institutional Review Board (IRB). To establish our study parameters, an extensive review of existing literature was performed. From this analysis, we determined a sample size of 23 participants utilizing the World Health Organization (WHO) sample size calculator. This determination was by taking mean aPTT 31.93 ± 3.38 , margin of error = 1.5% and confidence level 'C.I' = 95% [1]. A non-probability consecutive method of sampling was used for the sampling process.

Patients presented obstructive sleep apnea for more than three months having age between 20

to 60 years including both genders were included in this study.

Patients with a history of metabolic syndrome, diabetes mellitus, bleeding disorders, hematological malignancies, thyroid dysfunction (hypothyroidism or hyperthyroidism), stroke, renal impairment, chronic obstructive pulmonary disease, asthma, or congestive cardiac failure were excluded. Individuals using warfarin, aspirin, or anxiolytic medications, as well as pregnant women confirmed through history and dating scan, were also excluded from the study.

All patients provided written agreement before the enrolment, and their confidentiality was maintained at all levels. The institutional ethics committee's approval was also obtained prior to beginning the study. A detailed demographic and medical history was obtained from each participant prior to the procedure. Following clinical evaluation, electroencephalography (EEG) electrodes and airflow sensors were applied to monitor brain activity and breathing patterns, while electromyography (EMG) electrodes assessed muscle tone. Thoracoabdominal movements were measured using transducer belts, and continuous monitoring of oxygen saturation, blood pressure, and electrocardiography (ECG) was performed. The researcher documented gross body movements, while snoring and body position were recorded with specialized sensors. The system was capable of detecting critical events, including oxygen desaturation and arrhythmias. Overnight polysomnography with video monitoring was conducted from 10:00 PM until patient awakening, and data were manually scored according to internationally accepted standards. Apnea was defined as complete airflow cessation for more than 10 seconds, while hypopnea was defined as a $\geq 50\%$ reduction in airflow for at least 10 seconds with $\geq 3\%$ desaturation or EEG-confirmed arousal. The apnea–hypopnea index (AHI) was calculated as the number of apneic and hypopneic events per hour, with severity classified by an experienced sleep technologist.

After a 12-hour fast, venous blood samples were collected between 9:00–10:00 AM for coagulation studies, including blood coagulation analysis for platelet (PLT); prothrombin time (PT), activated partial thromboplastin time (aPTT) and international normalized ratio (PT/APTT). Anthropometric data (height and weight) were also recorded. Quantitative variables (age, height, weight, coagulation profile, OSA duration) and qualitative variables (gender, socioeconomic status, OSA severity) were documented on a structured proforma.

Data were analyzed using SPSS Version 20. Shapiro Wilk test was performed to check the normality of data. Continuous variables such as age, height, weight, platelet count, prothrombin time, thromboplastin time, and international normalized ratio (INR) were summarized as mean (SD) or median with interquartile range (IQR) depending on data distribution. Categorical variables including gender, age group, monthly income, and severity of obstructive sleep apnea (OSA) were presented as frequencies and percentages. Comparison of hematological parameters across OSA severity groups (mild, moderate, severe) was performed using one-way analysis of variance (ANOVA). For categorical variables, the Chi-square test was applied to assess association with OSA severity. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

A total of 23 participants were included in this study. Out of total, 5 (21.7%) were male and 18 (78.3%) were female. The baseline and demographic characteristics of study participants are shown in table-I. The majority of participants were between 35–50 years of age (13, 56.5%), while 10 (43.5%) were above 50 years. Regarding socioeconomic status, 8 (34.8%) participants had a monthly income of $\leq 25,000$ PKR, whereas 15 (65.2%) reported an income of $>25,000$ PKR. Based on the severity of obstructive sleep apnea (OSA), 9 (39.1%) patients were categorized as mild, 8 (34.8%) as moderate, and 6 (26.1%) as severe. The median

age of the participants was 45.0 years (IQR: 40.0–54.0). The median thromboplastin time was 25.91 seconds (IQR: 24.90–25.56), while the median disease duration was 6.0 months (IQR: 5.0–6.0). The mean height was 1.47 (0.11) meters, mean weight was 101.74 (12.97) kg, and mean platelet count was 188.39 (75.81) $\times 10^9/L$. The mean prothrombin time was 11.36 (1.04) seconds, and the mean normalized ratio was 1.06 (0.10).

Table-II shows the comparison of hematological parameters across different severity levels of obstructive sleep apnea (OSA). The mean platelet count was significantly lower in the mild group 135.88 (91.88) compared to the moderate 221.89 (45.93) and severe groups 222.50 (25.86) ($p = 0.020$), indicating that thrombocytopenia was more common in patients with mild OSA. Prothrombin time increased progressively with severity, from 10.70 (0.62) in mild cases to 12.41 (1.09) in severe cases ($p = 0.003$), showing a statistically significant prolongation with increasing severity of OSA. Similarly, the mean normalized ratio was significantly higher in severe cases 1.16 (0.06) compared to mild 1.02 (0.05) and moderate 1.02 (0.11) groups ($p = 0.007$). In contrast, the mean thromboplastin time did not differ significantly among mild 25.40 (1.80), moderate 25.66 (1.40), and severe 25.15 (1.79) groups ($p = 0.852$).

All p-values were computed using one-way ANOVA.

The distribution of demographic and socioeconomic characteristics across different severity levels of obstructive sleep apnea (OSA) is presented in Table-III. Gender did not show a significant association with OSA severity ($p = 0.645$), as both males and females were nearly equally distributed across mild, moderate, and severe categories. Similarly, age groups were comparable ($p = 0.405$); participants aged 35–50 years had a higher proportion of mild OSA (46.2%), whereas those above 50 years showed a relatively higher proportion of severe OSA (40.0%), though the difference was not statistically significant. Socioeconomic status, measured by monthly income, also

demonstrated no significant relationship with OSA severity ($p = 0.177$). Participants earning $\leq 25,000$ were more likely to have mild OSA (62.5%), while those with income $>25,000$ tended to have moderate OSA (46.7%). Overall,

demographic and socioeconomic characteristics were not significantly associated with the severity of OSA in this study population. All p-values were computed using chi square.

Table-I: Baseline demographic and clinical characteristics of study participants (n=23).

Variables		n (%)
Gender	Male	5 (21.7%)
	Female	18 (78.3%)
Age Group	35 to 50 years	13 (56.5%)
	>50 years	10 (43.5%)
Monthly Income	$\leq 25,000$	8 (34.8%)
	$> 25,000$	15 (65.2%)
Severity of Obstructive Apnea	Mild	9 (39.1%)
	Moderate	8 (34.8%)
	Severe	6 (26.1%)
		Median, IQR
Age (years)		45.00 (40.00–54.00)
Thromboplastin (In Seconds)		25.91 (24.90–25.56)
Duration of Disease (Months)		6.00 (5.00–6.00)
		Mean (SD)
Height (meters)		1.47 (0.11)
Weight (kg)		101.74 (12.97)
Platelets (In 10^9 Per Liter)		188.39 (75.81)
Prothrombin Time (In Seconds)		11.36 (1.04)
Normalized Ratios		1.06 (0.10)

Table-II: Comparison of mean coagulation parameters across severity groups of obstructive sleep apnea (n=23).

Variables	Mild (n=9) Mean (SD)	95% CI	Moderate (n=8) Mean (SD)	95% CI	Severe (n=6) Mean (SD)	95% CI	p-Value
Platelets	135.88 (91.88)	(65.24-206.50)	221.89 (45.93)	(182.49-260.29)	222.50 (25.86)	(195.35- 249.65)	0.020
Prothrombin Time	10.70 (0.62)	(10.22-11.17)	11.31 (0.77)	(10.67-11.96)	12.41 (1.09)	(11.25-13.55)	0.003
Thromboplastin	25.40 (1.80)	(24.01-26.78)	25.66 (1.40)	(24.49-26.83)	25.15 (1.79)	(23.27- 27.03)	0.852
Normalized Ratios	1.02 (0.05)	(0.98- .06)	1.02 (0.11)	(0.93- 1.11)	1.16 (0.06)	(1.10-1.22)	0.007

Values are presented as Mean (SD). CI = Confidence Interval. SD = Standard Deviation. P-values calculated using ANOVA test.

Table-III: Association of demographic and socioeconomic characteristics with severity of obstructive sleep apnea (n=23).

Variables		Severity of Obstructive Apnea			Total	p-Value
		Mild	Moderate	Severe		
Gender	Male	2 (40.0%)	1 (20.0%)	2 (40.0%)	5 (21.7%)	0.645
	Female	7 (38.9%)	7 (38.9%)	4 (22.2%)	18 (78.3%)	
	Total	9 (39.1%)	8 (34.8%)	6 (26.1%)	23 (100.0%)	
Age Group	35 to 50	6 (46.2%)	5 (38.5%)	2 (15.4%)	13 (56.5%)	0.405
	>50	3 (30.0%)	3 (30.0%)	4 (40.0%)	10 (43.5%)	
	Total	9 (39.1%)	8 (34.8%)	6 (26.1%)	23 (100.0%)	
Monthly Income	$\leq 25,000$	5 (62.5%)	1 (12.5%)	2 (25.0%)	8 (34.8%)	0.177
	$> 25,000$	4 (26.7%)	7 (46.7%)	4 (26.7%)	15 (65.2%)	
	Total	9 (39.1%)	8 (34.8%)	6 (26.1%)	23 (100.0%)	

DISCUSSION

In the present study, the median age of patients with sleep apnea was 45 years, with the majority being females, which differs from most international studies where males typically predominate [11,12]. This variation may reflect regional referral patterns, healthcare-seeking behavior, or population-specific risk factors. Although our patients had relatively higher body weights and lower heights compared to other studies, the high body mass index (BMI) underscores obesity as a central risk factor in OSA pathogenesis. Obesity increases pharyngeal fat deposition and reduces airway patency, which may explain the relatively high prevalence of OSA observed in our predominantly female cohort.

In present study, in distribution of severity of obstructive sleep apnea, 9 (39.1%) patients had mild symptoms of obstructive sleep apnea, 08 (34.8%) had moderate while 06 (26.1%) had severe symptoms of obstructive apnea. Hong SN *et al.*, [11] reported to have mild obstructive sleep apnea (OSA) in 21 (21.9%) cases, 30 (20.5%) with moderate OSA, and 43 (29.5%) with severe OSA. The findings suggest that a significant proportion of patients presenting with OSA lie in the moderate-to-severe category, a group particularly vulnerable to cardiovascular and hematological complications. Comparable distributions have been observed in regional studies, reinforcing the burden of severe disease in clinical practice [13].

Hematological parameters in our cohort revealed significantly lower platelet counts in mild OSA compared to moderate and severe cases, suggesting that progressive hypoxia and systemic inflammation may lead to increased platelet production and activation. This is consistent with findings from Varol *et al.*, [14] who demonstrated increased mean platelet volume in OSA patients that normalized following continuous positive airway pressure (CPAP) therapy. Similarly, Yang *et al.*, [15] reported higher platelet distribution width and evidence of platelet activation in severe OSA,

further supporting the role of platelets as potential biomarkers of disease severity.

In our study, prothrombin time increased progressively with severity (10.70 ± 0.62 in mild vs. 12.41 ± 1.09 seconds in severe OSA), while normalized ratios were also significantly higher in severe cases ($p=0.007$). These results contrast with Hong SN *et al.*, [11] who observed shortened prothrombin times in severe OSA, but are in agreement with von Ka R *et al.*, [16] who reported significant alterations in coagulation parameters, including prolonged PT/INR in patients with severe OSA. The discrepancy across studies may be attributable to differences in study populations, comorbidities, and laboratory methodologies. Several reviews have emphasized that intermittent hypoxia, sympathetic surges, and endothelial dysfunction promote a procoagulant state, although the exact direction of PT/INR changes may vary [17-19].

In contrast, thromboplastin time (APTT) in our study showed no significant difference across severity groups, a finding consistent with Hong *et al.*, [11] and Liak C *et al.*, [17] who suggested that the intrinsic pathway may be less sensitive to OSA-related alterations compared to the extrinsic pathway. However, other studies such as Shen *et al.*, [20] reported shortened APTT in pediatric OSA, indicating that age and physiology may influence coagulation responses.

Demographic and socioeconomic characteristics in our study including gender, age group, and monthly income showed no significant association with OSA severity. This contrasts with international literature where male gender, increasing age, and obesity are well-documented risk factors for both prevalence and severity of OSA [12, 13, 21].

LIMITATIONS

This study was limited by its small sample size, single-center design, and lack of control for confounders such as BMI and comorbidities. Advanced coagulation and inflammatory

markers were not assessed, which may have restricted a comprehensive evaluation.

CONCLUSION

This study evaluated demographics, disease severity, and hematological changes in patients with obstructive sleep apnea. A majority of patients were found in the moderate-to-severe category, reflecting a high clinical burden. Hematological alterations, including increased platelet counts and prolonged PT/INR, were associated with increasing severity, suggesting a prothrombotic state. Demographic and socioeconomic variables showed no significant link with OSA severity in this study. These findings emphasize that OSA should be recognized as a systemic disorder with important cardiovascular implications.

CONFLICT OF INTEREST

Authors declare no conflict of interest.

GRANT SUPPORT & FINANCIAL DISCLOSURE

Declared none

AUTHORS CONTRIBUTION

Tuba: Substantial contributions to study design, manuscript drafting, reviewing it critical for important intellectual content, final approval, accountable for all aspects of publication

Sara Mustafa: Substantial contributions to acquisition of data, final approval, accountable for all aspects of publication.

Rahmat Ali: Manuscript drafting, data analysis, critical review, final approval, accountable for all aspects of publication.

Syeda Saba Bukhari: Substantial contributions to study design, data interpretation, critical review, final approval, accountable for all aspects of publication.

Rashid Ali Daudpota: Acquisition of data, critical review, proofreading, final approval, accountable for all aspects of publication.

Sundus: Critical review, proofreading, final approval, accountable for all aspects of publication.

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