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Original Research Article

Association of plasma fibrinogen level with the severity of obstructive sleep apnoea patients in a tertiary care hospital

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ABSTRACT

Background: Obstructive sleep apnoea syndrome (OSAS) is a common sleep-related breathing disorder of multi-risk factorial pathogenesis and is characterized by recurrent, partial or complete upper airway obstruction resulting in intermittent hypoxia during sleep. It has been implicated in both cardiovascular and cerebrovascular diseases. Objective of the study was to determine the association of Plasma Fibrinogen levels with the severity of OSA patients in a tertiary care hospital.

Methods: This cross-sectional observational study with group comparison was conducted among all the patients attending in the Department of Respiratory Medicine, BSMMU with suspicion of OSA within one year after the clearance of institutional review board (IRB) using STOP-BANG questionnaire and Epworth sleepiness scale (ESS) and confirmed by polysomnography. Plasma fibrinogen was measured in all OSA and non-OSA patients. Data were analyzed with the help of statistical package for the social sciences (SPSS) version 26.

Results: Sociodemographic analysis found no significant differences in age, gender, area, occupation, or smoking between OSA and non-OSA groups ($p>0.05$). However, witnessed apnoea ($p=0.002$), breathlessness ($p=0.005$), higher ESS ($p<0.001$), and STOP-Bang scores ($p<0.001$) were associated with OSA. Plasma fibrinogen levels were significantly higher in OSA (319.2±63.7 mg/dl versus 242.5±20.33 mg/dl, $p<0.001$), positively correlating with AHI ($r=+0.876$, $p=0.001$). Positive correlations were also found between fibrinogen levels and daytime sleepiness ($r=+0.393$, $p=0.002$), waist circumference ($r=+0.346$, $p=0.007$), and BMI ($r=+0.297$, $p=0.021$) in OSA patients.

Conclusions: In conclusion, this study establishes a notable connection between plasma fibrinogen levels and the severity of OSA. Elevated fibrinogen levels correlate with increased OSA severity, indicating a link between OSA, inflammation and coagulation.

Keywords: Obstructive sleep apnoea, Arterial hypoxemia, Sleep fragmentation, Cardiovascular diseases, Cerebrovascular diseases

INTRODUCTION

Obstructive sleep apnoea (OSA) is a condition characterized by recurrent partial or complete upper airway closure resulting in recurrent arterial hypoxemia during sleep. Patients with OSA may experience symptoms including loud snoring, frequent arousals, sleep fragmentation, and daytime sleepiness which characterize obstructive sleep apnoea syndrome (OSAS).[1] According to American Journal of Respiratory and Critical Care Medicine 2018, global prevalence of OSA in adult based on objective sleep studies, ranged from 14% to 50% in men and 4% to 31% in women. There is lack of sufficient data from Bangladesh on the epidemiology of OSAH. The prevalence of OSAH among 30-60 years men and women were 17.37% and 6.25% respectively.[2]

OSA has been increasingly implicated in cardiovascular and cerebrovascular diseases.[3-5] Mechanisms such as increased sympathetic activity, endothelial dysfunction, chronic inflammation, and coagulation are potential mediators of increased risk of cardiovascular diseases.[6-9] A recently published systemic review has confirmed the significance of elevated fibrinogen for prediction of future cardiovascular risk even in the healthy, middle aged population.[10] OSA results in repetitive and severe nocturnal hypoxemia and sleep disturbances.[2] Hypoxemia at high altitude causes increased synthesis of both fibrinogen and cytokines.[11,12]

Sleep deprivation also induces an increase in cytokines.[13,14]

Thus, both chronic night time hypoxemia and sleep disturbances in OSA patients may lead to elevated pro inflammatory cytokines, markers of inflammation, and plasma fibrinogen levels. Previous studies in patients with OSA determined that elevated fibrinogen levels were related to and the presence of comorbidities such as hypertension and stroke were improved after nasal continuous positive airway pressure (CPAP) treatment.[15-17] Limited data are available on the effects of severity of OSA on plasma fibrinogen in otherwise healthy OSA patients. Sleep fragmentation and hypoxemia, two important mechanisms of increased fibrinogen in OSA patients. Therefore, we will try to determine whether severity of OSA is associated with elevated levels of plasma fibrinogen in newly diagnosed, untreated, and otherwise healthy OSA patients.

Objectives

General objective of the study was to assess the association between plasma fibrinogen level and severity of OSA patients.

Specific objectives of the study were to categorize the study population into OSA and non-OSA patients, to classify OSA patients according to severity as mild, moderate and severe, to measure plasma fibrinogen in OSA and non-OSA patients, and to compare plasma fibrinogen levels among OSA and non-OSA patients.

METHODS

Study design

It was a cross sectional observational study with group comparison.

Place of study

The study was conducted at the Department of Respiratory Medicine, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka.

Study period

The duration of the study was one year after the approval from IRB (July 2022 to July 2023).

Study population

All patients fulfilling inclusion and exclusion criteria with the clinical features of OSA within this period was taken as the study population.

Sample size determination

The sample size was calculated by using the following formula:

The formula is: $\frac{(u+v)^2(\sigma_1{}^2+\sigma_0{}^2)}{(\mu_0-\mu_1)^2}$

Where, u=1.96 (p value 0.5) and v=0.84 (power 80%), σ_1 and σ_0 are the assumed population standard deviation for groups 1 and 2 μ_0 and μ_1 are the assumed population means for power and sample size calculations μ_0-μ_1 is the difference between population means at which power and sample size calculations are made.

Here, μ_0=398 and μ_1=331 and σ_1=100 and σ_0=100.

So,

$$(1.96+0.84)^2\,(100^2+100^2)/(398-331)^2$$
$$=(2.8)^2(10000+10000)/(67)^2$$
$$=(7.84\times 20000)/4489$$
$$=156800/4489\approx 35$$

Sample size

Group sample sizes of 36 and 36 achieve 80.050% power to reject the null hypothesis of equal means when the population mean difference is μ_0-μ_1) =398.0-331.0=67.0 with a standard deviation of 100.0 for group 1 and 100.0 for group 2 and with a significance level 0.050 using a two-sample unequal-variance t–test.

Inclusion criteria

All OPD, indoor and referred patients of respiratory medicine with clinical sign symptoms of obstructive sleep apnoea with STOP BANG score ≥3 and Epworth sleepiness scale score >9 patients are willing to give informed written consent to participate in the study. Age over 18 years.

Exclusion criteria

Pregnancy complicates chronic pulmonary diseases like chronic obstructive pulmonary disease (COPD) or lung carcinoma, worsening respiratory symptoms and management challenges. Monitoring anticoagulant medication is crucial due to maternal and fetal health risks. Infections and inflammation increase risks, especially with compromised respiratory function. Alcohol or drug abuse worsens concerns for maternal and fetal well-being. Cerebrovascular diseases further complicate care, requiring comprehensive strategies.

Procedure

Patients undergoing in-laboratory polysomnography (PSG) were connected to various monitoring devices, including electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), and electrocardiogram (ECG) electrodes. An infrared camera facilitated observation and communication with the patient. Relevant data such as heart rate, respiratory rate, oxygen saturation, snoring presence/volume, and body position were periodically documented. For CPAP titration, mask changes and pressure adjustments were noted. Physiological variables were digitally recorded during sleep and wakefulness. Technicians manually scored data for sleep stages and abnormalities. Sleep-disordered breathing was diagnosed based on physiological patterns. For thrombin reagent calibration, dilutions of calibration plasma in veronal buffer were prepared. Clotting times were measured after adding thrombin. Patient samples were diluted for testing, and clot formation was observed using optical or mechanical automated equipment. The normal fibrinogen range is approximately 200 to 400 mg/dl. Testing was performed using the SYSMEX Automated Coagulation Analyzer (Model: CS 1600).

Data collection tools

A structured questionnaire containing socio-demographic, anthropometric, clinical characteristics, polysomnography and laboratory investigations including plasma fibrinogen of the patients.

Data analysis

All data were analyzed after collection. Then the data were entered into a computer and statistical analysis of the results was obtained by using Windows-based computer software devised with statistical packages for social science (SPSS) version-26, (SPSS Inc. Chicago, IL, USA). The categorical data were analyzed by Unpaired t-test and Chi-square test. P value of less than 0.05 was considered statistically significant.

Data collection procedure

The study population was the patients attending the Department of Respiratory Medicine, BSMMU with the symptoms of snoring, tiredness or daytime sleepiness. The study population was selected on the basis of a STOP-BANG score ≥3 and Epworth sleepiness scale score >9 and following overnight polysomnography, diagnosed as OSA on the basis of AHI>5 and non-OSA on the basis of AHI <5. Then OSA was classified on the basis of AHI as mild, moderate and severe. Then plasma fibrinogen level was measured in all patients.

Ethical considerations

All patients willing to participate in the study were informed that there would be no extra risk during history taking, physical examination and investigations. Ethical clearance was obtained from the Ethical Review Committee of BSMMU, Dhaka to undertake the present study. According to the Helsinki Declaration for Medical Research Involving Human Subjects 1964, all the patients were informed about the study design, the underlying hypothesis and their rights to withdraw themselves from the projects at any time, for any reason.

RESULTS

Table 1 shows the sociodemographic characteristics of the study subjects. There were no significant differences between the two groups in terms of age, gender, inhabitant area, occupation and smoking status ($p>0.05$). However, there was a slightly higher percentage of rural inhabitants in the non-OSA group compared to the OSA group (36.1% versus 28.3%, $p=0.426$). Regarding educational level, the OSA group had a higher percentage of graduate participants compared to the non-OSA group (36.7% versus 27.8%), while the non-OSA group had a slightly higher percentage of postgraduate participants (16.7% versus 8.3%), but these differences were not statistically significant ($p>0.05$).

Table 2 displays clinical feature associations at presentation between OSA and non-OSA groups. Snoring was universal among participants. Witnessed apnea was significantly more prevalent in the OSA group (66.7% versus 33.3%, $p=0.002$). OSA patients had higher ESS and STOP-Bang scores ($p<0.001$). Shortness of breath was more common in the OSA group (60.0% versus 30.6%, $p=0.005$). OSA patients exhibited higher waist and neck circumferences and BMI ($p<0.001$), with a greater proportion classified as overweight or obese compared to non-OSA individuals.

Table 3 presents an analysis of the association between hypertension and two groups, Among the 60 participants with OSA, 18 (30.0%) had hypertension, while among the 36 participants without OSA, only 4 (11.1%) had hypertension. There was a statistically significant association between hypertension and OSA (p=0.033).

Table 4 shows a comparison of polysomnography findings between the OSA and non-OSA groups. The results indicate that the OSA group had significantly higher apnoea hypopnea index (AHI) and oxygen desaturation index (ODI) compared to the non-OSA group (29.08±22.45 versus 2.49±1.66, p<0.001). The mean SpO_2 was significantly lower in the OSA group compared to the non-OSA group (92.10±4.73 versus 94.14±3.24, p=0.025), and the lowest SpO_2 during sleep was also significantly lower in the OSA group (71.38±17.31% versus 81.75±10.63%, p=0.002). However, there were no significant differences in arousal index events/hours between the two groups (26.57±22.85 versus 18.71±22.26, p=0.103).

Table 5 shows a comparison of plasma fibrinogen levels between the OSA and non-OSA groups. In the OSA group had significantly higher plasma fibrinogen levels compared to the non-OSA group (319.2±63.7 mg/dl versus 242.5±20.33 mg/dl, p<0.001).

Figure 2 shows the distribution of OSA patients by severity. Out of the 60 OSA patients, 15 (25.0%) were classified as having mild OSA, 18 (30.0%) had moderate OSA, and 27 (45.0%) had severe OSA.

Table 6 shows the association of clinical features at presentation with OSA severity. Snoring was present in all patients. The presence of witnessed apnoea and shortness of breath increased with increasing OSA severity (p<0.001 and p=0.003, respectively). The OSA severity groups also had significantly higher scores on the Epworth sleepiness scale (ESS) and STOP-Bang questionnaire compared to the non-OSA group (p<0.001). Anthropometric measurements such as waist and neck circumference, as well as BMI, were also significantly higher in the OSA severity groups compared to the non-OSA group (p<0.001 for BMI).

Table 7 shows a comparison of polysomnographic parameters among the OSA severity and non-OSA groups. The results indicate that the OSA groups had significantly higher AHI, arousal index, lowest SpO_2 during sleep, time at <90% oxygen saturation, and ODI compared to the non-OSA group (p<0.001).

Table 1: Socio-demographic characteristics of the study subjects (n=96).

Variables	OSA (n=60) (%)	Non-OSA (n=36) (%)	P value
Age group (years)			
<50	32 (53.3)	21 (58.3)	0.633
>50	28 (46.7)	15 (41.7)	
Gender			
Male	36 (60.0)	22 (61.1)	0.914
Female	24 (40.0)	14 (38.9)	
Inhabitant			
Rural	17 (28.3)	13 (36.1)	0.426
Urban	43 (71.7)	23 (63.9)	
Educational level			
Can sign or read	0 (0.0)	1 (2.8)	0.425
Class 1-5	3 (5.0)	1 (2.8)	
Class 6-10	11 (18.3)	4 (11.1)	
Class 11-12	19 (31.7)	14 (38.9)	
Graduate	22 (36.7)	10 (27.8)	
Postgraduate	5 (8.3)	6 (16.7)	
Occupation			
Student	1 (1.7)	0 (0.0)	0.734
Housewife	23 (38.3)	13 (36.1)	
Farmer	1 (1.7)	2 (5.6)	
Businessman	28 (46.7)	18 (50.0)	
Other	7 (11.7)	3 (8.3)	
Smoking status			
Smoker	8 (13.3)	4 (11.1)	0.750
Non-smoker	52 (86.7)	32 (88.9)	

Table 2: Association of clinical features at presentation with OSA (n=96).

Variables	OSA (n=60) (%)	Non-OSA (n=36) (%)	P value
Snoring	60 (100.0)	36 (100.0)	0.002
Witness apnoea	40 (66.7)	12 (33.3)	
Epworth sleepiness scale	12.17±2.09	10.61±0.77	<0.001
STOP-Bang questionnaire	4.30±0.94	3.50±0.65	<0.001
Shortness of breath	36 (60.0)	11 (30.6)	0.005
Waist circumference (cm)	103.9±13.8	94.6±11.38	<0.001
Neck circumference (inch)	15.75±0.78	15.52±0.81	<0.001
BMI (kg/m^2)			
Normal (18.5-24.9)	5 (8.3)	10 (27.8)	
Overweight (25.0-29.9)	25 (71.7)	19 (52.8)	
Obesity class I (30-34.9)	16 (26.7)	5 (13.9)	
Obesity class II (35-39.9)	10 (16.7)	2 (5.6)	
Obesity class III (>40)	4 (6.7)	0 (0.0)	
Mean±SD	31.4±6.13	26.93±4.16	<0.001

Table 3: Association of hypertension between two groups (n=96).

Hypertension	OSA (n=60) (%)	Non-OSA (n=36) (%)	P value
Yes	18 (30.0)	4 (11.1)	<0.001
No	42 (70.0)	32 (88.9)	
Total	60 (100.0)	36 (100.0)	0.005

Table 4: Comparison of polysomnography findings between two groups (n=96).

Polysomnography findings	OSA (n=60)	Non-OSA (n=36)	P value
Apnoea hypopnea index	29.08±22.45	2.49±1.66	<0.001
Arousal index events/hours	26.57±22.85	18.71±22.26	0.103
Mean SpO_2	92.10±4.73	94.14±3.24	0.025
Lowest SpO_2 during sleep	71.38±17.31	81.75±10.63	0.002
Oxygen desaturation index	24.06±18.20	3.40±2.48	<0.001

Table 5: Comparison of plasma fibrinogen level between two groups (n=96).

Variable	OSA (n=60)	Non-OSA (n=36)	P value
Plasma fibrinogen (mg/dl)	319.2±63.7	242.5±20.33	<0.001

Table 6: Association of clinical features at presentation with OSA (n=96).

Variables	Non-OSA (n=36)	Mild OSA	Variables	Non-OSA (n=36)	Mild OSA
Snoring	36 (100)	15 (100)	18 (100)	27 (100)	-
Witness apnoea	12 (33.3)	6 (40.0)	10 (55.6)	24 (88.9)	<0.001
Shortness of breath	11 (30.6)	5 (33.3)	11 (61.1)	20 (74.1)	0.003
Epworth sleepiness scale	10.61±0.77	11.53±1.255	11.94±1.477	12.67±2.677	<0.001
Stop Bang questionnaire score	3.50±0.65	3.80±0.86	4.11±0.68	4.70±0.99	<0.001
Waist circumference (inch)	94.6±11.4	101.1±17.3	101.0±9.0	107.5±13.9	0.002
Neck circumference (inch)	15.14±0.78	15.57±0.82	15.83±0.59	15.80±0.78	0.002
BMI (kg/m^2)	26.93±4.16	30.31±6.40	29.74±4.19	33.12±6.80	<0.001

Table 7: Comparison of polysomnographic parameters of OSAS and control group.

Variables	Non-OSA (n=36)	Mild OAS (n=15)	Moderate OAS (n=18)	Severe OAS (n=27)	P value
Apnea hypopnea index (events/hour)	2.49±1.66	8.49±3.15	18.69±2.56	49.56±18.97	<0.001
Arousal index (events/hour)	18.71±22.26	12.20±13.91	31.11±22.81	31.52±24.11	0.012
Mean SpO^2 (%)	94.1±3.24	93.9±3.5	93.6±1.8	90.1±5.8	<0.045
Lowest SpO^2 during sleep (%)	81.75±10.63	78.93±15.96	74.44±13.33	65.15±18.62	<0.001
Time at <90%	39.9±19.5	44.8±12.4	46.89±5.01	114.5±12.7	<0.001
Oxygen saturation (min) ODI	3.40±2.48	7.82±4.51	18.22±10.55	36.99±17.86	<0.001

Table 8: Multivariate regression was performed to see the relationship of plasma fibrinogen with AHI, BMI and blood pressure (n=96).

Variables	Beta	P value	95% CI	
Appnoea hypopnea index (AHI)	2.470	<0.001	2.074	2.641
Body mass index (BMI)	1.216	0.027	0.139	2.293
Systolic blood pressure (SBP)	0.670	0.017	0.124	1.216

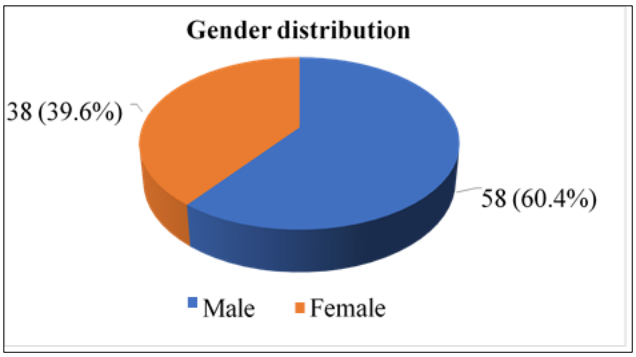

Figure 1: The sex distribution of the study patients.

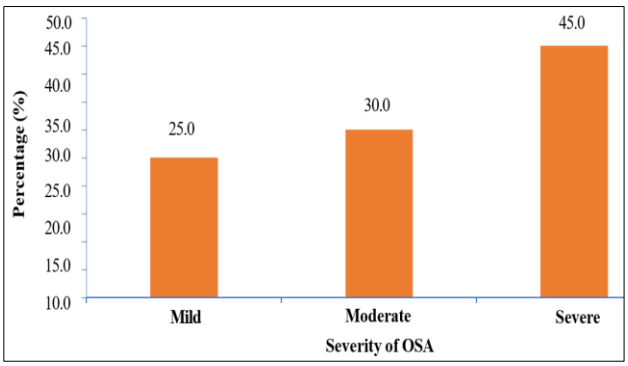

Figure 2: The severity of OSA patients.

The multivariate regression analysis of plasma fibrinogen levels with AHI, BMI and SBP showed that there is a significant positive relationship between each of these variables and plasma fibrinogen levels. Specifically, higher AHI, BMI and SBP were associated with increased levels of plasma fibrinogen. The regression coefficient (beta) for AHI, BMI and SBP were 2.470 (p<0.001), 1.216 (p=0.027), and 0.670 (p=0.017), respectively. The 95%CI for these coefficients were 2.074- 2.641, 0.139-2.293, and 0.124-1.216, respectively (Table 8).

DISCUSSION

OSA is a common sleep disorder characterized by repeated episodes of partial or complete upper airway obstruction during sleep. Fibrinogen is a plasma protein that plays a crucial role in the coagulation cascade and is also involved in the pathogenesis of atherosclerosis. The present study aimed to investigate the association between plasma fibrinogen level and the severity of OSA. In present study showed no significant difference of mean age between the OSA and non-OSA groups, with 53.3% of OSA patients and 58.3% of non-OSA patients being below 50 years old. In gender distribution between the two groups, with 60.0% of OSA patients and 61.1% of non-OSA patients being male. In terms of inhabitant area, the majority of participants in both groups were from urban areas, with 71.7% of OSA patients and 63.9% of non-OSA patients residing in urban areas. Regarding occupation, the most common occupation among OSA patients was businessman (46.7%), followed by housewife (38.3%).

In smoking status between the OSA and non-OSA groups, with 13.3% of OSA patients and 11.1% of non-OSA patients being smokers. These results are consistent with previous research that has identified these clinical features as risk factors for OSA.[18] Moreover, OSA-related hypoxia and fragmented sleep patterns can lead to a dysregulation of blood pressure, particularly during the night. One study conducted a longitudinal analysis and demonstrated that individuals with OSA were at a substantially higher risk of developing hypertension over time.[19] This longitudinal perspective aligns with the present study's cross-sectional findings, indicating a clear link between OSA and hypertension. Another study shows delved into the mechanisms underlying this association.[20] They highlighted how recurrent episodes of hypoxia and arousal from sleep in OSA patients can trigger sympathetic nervous system activation, leading to increased blood pressure. Moreover, addressing OSA through interventions such as continuous positive airway pressure

(CPAP) therapy has been shown to improve blood pressure control and reduce the need for antihypertensive medications, as demonstrated in studies like that of.[21] In present study showed a significantly higher AHI in the OSA group (29.08±22.45) compared to the non-OSA group (2.49±1.66), which is consistent with previous research.[22]

In presents study showed arousal Index events per hour measured sleep disruptions due to arousals, and while the OSA group (26.57±22.85) does show a higher mean compared to the non-OSA group (18.71±22.26), this difference does not reach statistical significance. When compared with severity of OSA, the arousal index (events/hour) was significantly different between the groups (p=0.012). The control group had an arousal index of 18.71±22.26 events/hour, which was lower compared to the mild (12.20±13.91 events/hour), moderate (31.11±22.81 events/hour), and severe (31.52±24.11 events/hour) OSAS groups. In accordance with this the arousal index was significantly higher in the OSAS group (29±2.06 versus 14.6±1.57, p<0.001), indicating more frequent arousals from sleep.[23] In presents study showed that average oxygen saturation during sleep (%) and lowest SpO_2 during sleep (%) both significantly differed among the groups (p=0.014 and <0.001, respectively). Oxygen saturation decreased as OSAS severity increased, with the control group having the highest average oxygen saturation and the severe OSAS group the lowest. These findings are consistent with previous studies that have reported a relationship between OSAS severity and oxygen desaturation during sleep.[22,24]

In present study showed that time spent at <90% oxygen saturation (min) also significantly increased with OSAS severity (p<0.001), ranging from 39.9±109.5 minutes in the control group to 114.5±124.7 minutes in the Severe OSAS group. This parameter is important as it reflects the amount of time an individual spends with low oxygen levels during sleep, which can have negative consequences on their health, which was consisted with findings of.[25] Finally, the oxygen desaturation index (ODI) exhibited a similar trend, with significant differences among groups (p<0.001). ODI increased with OSAS severity, with the control group having the lowest ODI (3.40±2.48) and the Severe OSAS group having the highest (36.99±17.86). The relationship between OSA and fibrinogen is rooted in the complex interplay of inflammatory and coagulation processes within the disorder.[26] It has been proposed that OSA may contribute to elevated plasma fibrinogen levels through mechanisms such as edema and plasma cell infiltration, as well as inflammation of the soft palate, which exacerbates upper airway obstruction during sleep.[27]

In present study strengthens the existing body of evidence linking OSA severity with elevated plasma fibrinogen levels. This relationship is intricately tied to the disruption of inflammatory and coagulation processes in OSA and highlights the clinical significance of monitoring fibrinogen levels in OSA patients. In present study showed plasma fibrinogen exhibits a significant positive association with OSA severity, as evidenced by the apnoeahypopnoea index (AHI) (r= +0.876, p<0.001) and the oxygen desaturation index (r= +0.808, p<0.001). These findings suggest that inflammation, as indicated by fibrinogen levels, may play a crucial role in the pathophysiology of OSA, particularly in individuals with more severe OSA and related comorbidities such as obesity. However, other factors like age, sleep stage duration, neck circumference, and heart rate do not exhibit significant correlations with fibrinogen levels in this study noted fibrinogen levels were reported to be significantly elevated in OSAS patients than that of the control group, and a negative relationship was shown between fibrinogen and average oxygen saturation.[28]

In present study the multivariate regression analysis of plasma fibrinogen levels with AHI, BMI and SBP showed that there is a significant positive relationship between each of these variables and plasma fibrinogen levels. Specifically, higher AHI, BMI and SBP were associated with increased levels of plasma fibrinogen. The regression coefficient (beta) for AHI, BMI and SBP were 2.470 (p<0.001), 1.216 (p=0.027), and 0.670 (p=0.017), respectively. These findings are consistent with the existing literature, which underscores the role of these factors in the development of OSA.[29]

Limitations

The study was conducted at a single tertiary care hospital with different demographics and healthcare access and the sample size might not represent the broader population of OSA patients. The study followed a cross-sectional design, which limits its ability to establish causality. It can show associations but cannot determine whether elevated plasma fibrinogen levels are a cause or consequence of OSA.

CONCLUSION

In conclusion, this study investigated the relationship between plasma fibrinogen levels and the severity of OSA. Most importantly, plasma fibrinogen levels were found to be significantly higher in individuals with OSA compared to those without OSA. Furthermore, fibrinogen levels increased progressively with the severity of OSA. This suggests that fibrinogen may serve as a valuable biomarker for OSA severity and highlights the intricate relationship between OSA, inflammation, and coagulation processes.

Recommendations

Conducting longitudinal research to track changes in plasma fibrinogen levels and OSA severity over time is essential. This would allow for the examination of causal relationships and the impact of interventions, providing insights into the dynamic nature of OSA. Expanding the research to include multiple healthcare centres and a more

diverse patient population can enhance the generalizability of findings. A multicenter approach can also account for regional variations in OSA prevalence and risk factors. Investigate the effect of OSA treatments, such as CPAP therapy, on plasma fibrinogen levels. Assess whether managing OSA leads to reductions in fibrinogen levels and whether this contributes to improved cardiovascular outcomes.

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