Original Article

The Effect of Obstructive Sleep Apnea on Erectile function and Serum Testosterone Level Among Males: An Observational, Analytical Study

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ABSTRACT

Background:
Recurrent episodes of breathing difficulties while asleep are the hallmark of sleep apnea. Sleep-related reductions (hypopneas) or cessations (apneas) in breathing are the hallmarks of this condition.

Objectives:
this research was conducted to evaluate erectile dysfunction and testosterone level in cases diagnosed with obstructive sleep apnea. Clinical Comorbidities, the Epworth Sleepiness Scale (ESS), patients' body mass index (BMI),and their relation to OSAS severity.

Methods: analytical observational cross-sectional research done on individuals diagnosed with obstructive sleep apnea admitted to our tertiary care hospital during the research period.

Result: The current study revealed there was a significant difference across all groups in ESS, AHI, and ODI, with severe OSA patients showing notably higher indices contrasted with control and mild OSA groups. Also, there were significant variations in IIEF-5 and EHS scores across all groups, with severe OSA patients exhibiting notably lower erectile function and erection hardness compared to control (p <0.001). Mild OSA also showed a moderate impact on these indicators, with significant differences from severe OSA in EHS (p = 0.038). Diabetes Mellitus was more prevalent in mild OSA contrasted with control and moderate OSA groups (p<0.001). Cerebrovascular accidents were also significantly higher in severe OSA in contrast to control (p=0.039).

Conclusions: The results of this research pointed to the significant impact of obstructive sleep apnea (OSA) on erectile dysfunction (ED) and hormonal profiles, particularly testosterone levels.

Key Words: Erectile dysfunction, obstructive Sleep Apnea, testosterone level .

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INTRODUCTION

Approximately 900 million people across the world suffer from OSA, a common sleep disease marked by periodic cessations in breathing while sleeping[1]. There is strong evidence that males with OSA have lower serum testosterone levels in contrast to those without the condition. A systematic review and meta-analysis indicated that men with severe OSA exhibited notably lower testosterone levels compared to those without the condition, with findings suggesting that the severity of OSA correlates directly with testosterone deficiency[2].This relationship is attributed to the physiological effects of OSA, which include sleep fragmentation and intermittent hypoxia, leading to increased oxidative stress and insulin resistance, all of which can adversely affect testosterone metabolism[3].

The hormonal interplay between testosterone and sleep is complex. Testosterone is essential for various bodily functions, including sexual health and mood regulation. The hypothalamic-pituitary-gonadal axis governs testosterone production, which is influenced by sleep patterns, particularly REM sleep. In men with OSA, disrupted sleep can lead to decreased luteinizing hormone (LH) secretion, resulting in diminished testosterone levels[4]. Regardless of age or BMI, studies have demonstrated that nocturnal production of LH and testosterone is much decreased in those with OSA contrasted with controls. This suggests that the pathophysiology of OSA directly impacts the pituitary-gonadal axis, leading to secondary hypogonadism in affected individuals[5].

The implications of this relationship are significant for both diagnosis and treatment. Men suffering from OSA often report symptoms of sexual dysfunction, such

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as erectile dysfunction and decreased libido, which are likely exacerbated by low testosterone levels. Therefore, assessing testosterone levels in men diagnosed with OSA especially those with severe manifestations become crucial for comprehensive management[6]. While treatments like Continuous Positive Airway Pressure (CPAP) therapy can improve sleep quality and potentially restore normal testosterone levels, caution is warranted when considering testosterone replacement therapy (TRT), as it may pose risks for worsening respiratory issues during sleep[7,8].

PATIENTS AND METHODS

Study design

The analytical observational cross sectional research was done on 180 participants (130 patients diagnosed with obstructive sleep apnea apnea and 50 participants were control groups had no OSA and normal BMI) admitted to our tertiary care hospital during the research period were selected to be participants in this research according the following inclusion criteria; Age: 18-60 years, males diagnosed with OSA by PSG as well as all individuals included in the trial were required to maintain a stable relationship and engage in frequent sexual activity. Exclusion criteria: Individuals who have a history of other sexual disorders, are currently consuming alcohol at a rate of 2 units per day or abusing substances, are undergoing hormonal therapy, have an IQ below average, have hypercortisolism and / or hypothyroidism, a history of hypogonadism caused by the pituitary gland or testicles, or urological or neurological diseases that may impact erectile function (e.g., spinal cord injury, prostate cancer, multiple sclerosis, etc.), cannot be considered. Also excluded, participants whose prescriptions for vasoactive drugs (such as angiotensin-converting enzyme (ACE) inhibitors, statins, or nitrates) had changed in the past four weeks prior to starting the research.

Data collection

All cases involved in the research were subjected to the following:

Detailed history taking including: age and comorbidities as Diabetes Mellitus, HTN, dyslipidemia, IHD and cerebrovascular accidents .

Clinical examination

BMI is a widely utilized screening tool that assesses body weight relative to height. It was calculated utilizing the formula: BMI=weight kg/ height m^2.

ESS: The Epworth Sleepiness Scale (ESS) is a widely utilized tool for assessing subjective daytime sleepiness, particularly in patients suspected of having OSA. Questions in the ESS range from 0 to 3, with the goal of gauging the probability that a respondent may nod off in different commonplace scenarios. More daytime drowsiness is indicated by higher total scores, which can go from 0 to 24.

Neurophysiological assessment: by polysomnography PSG. PSG includes EEG leads, chest and abdominal belts, oronasal thermistor, nasal air transducer, pulse oximeter, snoring microphone in addition to EOG to assess the eye movements and EMG to assess the muscle tone. AHI and ODI were compared across study groups.

Erectile dysfunction Assessment

The IIEF-5, which is the Arabic version of the International Index of Erectile Function[23], was employed to assess the erectile function. On this survey, a score of 21 or lower indicates ED out of a possible 25. On top of that, ED is classified as mild[17-21], moderate[8-11], mild-to-moderate[12-16], or severe (≤ 7). We will study the Arabic questionnaire that individuals filled out.

In order to calculate the Erection Hardness Score, patients are asked to evaluate the degree of difficulty of their erection and then choose one of the following options: 0. The penis does not grow in size. 1. The penis is big but not particularly firm. 2. Although the penis is hard, it is not hard enough to allow penetration. 3. Although the penis is sufficiently firm for penetration, it is not fully hard-on. 4. The penis is absolutely rigid and composed of a hard material.

Investigations: Testosterone total, Testosterone free in which it was taken as Early morning sample [8:00 - 11:00 AM], FSH, LH, PRL and E2.

Statistical analysis

In accordance with IBM's Statistical Package for the Social Sciences (2017, Released), the data that was obtained was coded and tallied. Armonk, New York, USA: IBM Corp., IBM SPSS Statistics for Windows, Version 25.0. All parameters' data was shown and assessed in accordance with the data kind that was collected. The normality of the data distribution was checked utilizing the Shapiro-Wilk test. If more than two non-parametric variables showed statistically significant differences between the study groups, the Kruskal-Wallis test was employed to find out. When looking for statistical significance across more than two parametric variables in the research groups, a one-way analysis of variance test was employed. To investigate the correlation between the two qualitative factors, a chi-square test was employed. If the *p-value* is below 0.05 on a 95% confidence interval, then it is deemed significant.

RESULTS

The age disparity between those with severe OSA group and control groups was significant (p<0.001) as seen in the demographic data.

Significant variations were noted across all groups (mild, moderate and severe OSA) concerning BMI in relation to control group, as indicated in Table (1), with severe OSA having higher BMI values than other groups.

Table 1: Demographic data of study groups

Parameter	Category	Mild OSA (n=27)	Moderate OSA (n=41)	Severe OSA (n=62)	Control (n=50)	Test Result	Pairwise Comparison
Age (years)	Mean±SD	43.22±9.41	46.37±7.82	50.02±6.40	45.14±9.24	F: 13.065, p<0.001	$p2$=0.154, $p3$<0.001, $p4$=0.491, $p5$=0.025, $p6$=0.595, $p7$=0.011
	Median (IQR)	41.00(34.50-51.50)	47.00(42.00-52.00)	50.00(45.00-56.00)	47.00(37.00-51.75)		
BMI (kg/m²)	Mean±SD	27.06±1.72	34.28±2.46	41.90 ± 6.82	23.12±2.57	H: 147.849, p<0.001	$p2$<0.001, $p3$<0.001, $p4$<0.001, $p5$<0.001, $p6$<0.001, $p7$<0.001
	Median (IQR)	26.31(25.60-29.08)	33.53(32.34-36.16)	41.44(36.17-46.62)	22.68(21.05-25.41)		

F: One way ANOVA test, H: Kruskal-Wallis Test

Regarding comorbidities, Diabetes Mellitus was more prevalent in mild OSA contrasted with control and moderate OSA groups (p<0.001). Cerebrovascular accidents were also significantly higher in severe OSA in contrast to control (p=0.039) as indicated in Table (2).

Table 2: Comorbidities among study groups.

Parameter	Category	Mild OSA (n=27)	Moderate OSA (n=41)	Severe OSA (n=62)	Control (n=50)	Test Result	Pairwise Comparison
DM (Diabetes Mellitus)	No	0 (0.0%)	31 (75.6%)	40 (64.5%)	40 (80.0%)	$\chi2$:184.230, p<0.001	p2<0.001, p3<0.001, p4=0.330, p5=0.804, p6=0.111
	Yes	27 (100.0%)	10 (24.4%)	22 (35.5%)	10 (20.0%)		
HTN (Hypertension)	No	21 (77.8%)	32 (78.0%)	51 (82.3%)	41 (82.0%)	$\chi2$: 0.479, p=0.924	p2=1.000, p3=0.841, p4=0.885, p5=0.784, p6=0.837, p7=1.000
	Yes	6 (22.2%)	9 (22.0%)	11 (17.7%)	9 (18.0%)		
Dyslipidemia	No	22 (81.5%)	34 (82.9%)	42 (67.7%)	41 (82.0%)	$\chi2$: 4.854, p=0.183	p2=1.000, p3=0.285, p4=1.000, p5=0.137, p6=1.000, p7=0.135
	Yes	5 (18.5%)	7 (17.1%)	20 (32.3%)	9 (18.0%)		
IHD (Ischemic Heart Disease)	No	25 (92.6%)	38 (92.7%)	50 (80.6%)	46 (92.0%)	$\chi2$: 5.436, p=0.142	p2=1.000, p3=0.269, p4=1.000, p5=0.159, p6=1.000, p7=0.151
	Yes	2 (7.4%)	3 (7.3%)	12 (19.4%)	4 (8.0%)		
Cerebrovascular Accidents	No	23 (85.2%)	37 (90.2%)	47 (75.8%)	47 (94.0%)	$\chi2$: 8.386, p=0.039	p2=0.803, p3=0.477, p4=0.385, p5=0.112, p6=0.784, p7=0.019
	Yes	4 (14.8%)	4 (9.8%)	15 (24.2%)	3 (6.0%)		

Regarding sleep parameters, there was a significant difference across all groups in AHI, ESS, and ODI, with severe OSA patients showing notably higher indices compared to control and mild OSA groups (p<0.001). as illustrated in Table (3).

Table 3: Sleep assessment parameters in study groups

Parameter	Category	Mild OSA (n=27)	Moderate OSA (n=41)	Severe OSA (n=62)	Control (n=50)	Test Result	Pairwise Comparison
AHI (Apnea-Hypopnea Index)	Mean ± SD	9.52 ± 3.20	22.37 ± 4.11	38.13 ± 4.34	2.52 ± 1.58	H: 165.096, p<0.001	p2 <0.001 p3 <0.001 p4 <0.001 p5 <0.001 p6 <0.001 p7 <0.001
	Median (IQR)	9.00 (7.00-12.00)	22.00 (19.00-26.00)	39.00 (34.25-41.75)	3.00 (1.00-4.00)		
ODI (Oxygen Desaturation Index)	Mean ± SD	9.37 ± 3.05	21.93 ± 4.09	37.94 ± 4.41	1.58 ± 1.34	H: 165.345, p<0.001	p2 <0.001 p3 <0.001 p4 <0.001 p5 <0.001 p6 <0.001 p7 <0.001
	Median (IQR)	10.00 (6.50-12.00)	22.00 (19.00-25.00)	37.50 (34.00-42.00)	1.00 (1.00-3.00)		
ESS							
	Mean ± SD	11.56 ± 0.51	13.83 ± 0.80	20.31 ± 2.36	4.50 ± 3.21	H: 165.691, p<0.001	p2 <0.001 p3 <0.001 p4 <0.001 p5 <0.001 p6 <0.001 p7 <0.001
	Median (IQR)	12.00 (11.00-12.00)	14.00 (13.00-14.00)	21.00 (18.25-22.00)	4.50 (2.00-6.75)		

H: Kruskal-Wallis Test

Regarding erectile function assessment, there were significant differences in IIEF-5 and EHS scores across all groups, with severe OSA patients exhibiting notably lower erectile function and erection hardness compared to control (p<0.001). Mild OSA also showed a moderate impact on these indicators, with significant differences from severe OSA in EHS (p=0.038) as illustrated in Table (4).

Table 4: Erectile function assessment in study groups

Parameter	Category	Mild OSA (n=27)	Moderate OSA (n=41)	Severe OSA (n=62)	Control (n=50)	Test Result	Pairwise Comparison
IIEF-5 (International Index of Erectile Function - 5)	Mean ± SD	19.59 ± 2.66	9.39 ± 1.12	6.95 ± 1.58	23.42 ± 1.13	H: 152.291, p<0.001	$p2$<0.001, $p3$<0.001, $p4$<0.001, $p5$<0.001, $p6$<0.001, $p7$<0.001
	Median (IQR)	19.00 (17.00-21.00)	9.00 (8.00-10.00)	7.00 (5.00-8.00)	23.00 (22.00-24.00)		
EHS (Erection Hardness Score)	Mean ± SD	3.59 ± 0.50	1.46 ± 0.50	1.10 ± 0.84	3.52 ± 0.50	H: 139.234, p<0.001	$p2$<0.001, $p3$<0.001, $p4$=0.548, $p5$=0.038, $p6$<0.001, $p7$<0.001
	Median (IQR)	4.00 (3.00-4.00)	1.00 (1.00-2.00)	1.00 (0.00-2.00)	4.00 (3.00-4.00)		

H: Kruskal-Wallis Test

Regarding hormonal profile, significant differences were observed across all groups for FSH, LH, Testosterone, PRL, and E2 levels, with severe OSA showing lower FSH, LH, and Testosterone levels and higher PRL and E2 levels contrasted with control as indicated in Table (5).

Table 5: Hormonal profile of study groups

Parameter	Category	Mild OSA (n=27)	Moderate OSA (n=41)	Severe OSA (n=62)	Control (n=50)	Test Result	Pairwise Comparison
FSH (Follicle-Stimulating Hormone) (mIU/ml)	Mean ± SD	4.00 ± 1.81	2.22 ± 0.66	2.01 ± 0.62	4.83 ± 1.64	H: 88.597, p<0.001	$p2$<0.001, $p3$<0.001, $p4$=0.059, $p5$=0.102, $p6$<0.001, $p7$<0.001
	Median (IQR)	3.40 (2.66-4.79)	2.32 (1.56-2.68)	1.90 (1.42-2.58)	4.68 (3.64-6.27)		
LH (Luteinizing Hormone) (mIU/ml)	Mean ± SD	4.90 ± 1.74	2.45 ± 0.69	1.74 ± 0.40	4.88 ± 1.72	H: 114.829, p<0.001	$p2$<0.001, $p3$<0.001, $p4$=0.973, $p5$<0.001, $p6$<0.001, $p7$<0.001
	Median (IQR)	5.29 (3.77-6.28)	2.51 (2.04-2.99)	1.64 (1.45-2.09)	4.79 (3.72-6.40)		
Testosterone (Total) (ng/ml)	Mean ± SD	4.68 ± 1.38	2.45 ± 0.58	1.88 ± 0.55	5.50 ± 1.73	H: 127.107, p<0.001	$p2$<0.001, $p3$<0.001, $p4$=0.041, $p5$<0.001, $p6$<0.001, $p7$<0.001
	Median (IQR)	4.35 (3.36-5.90)	2.23 (2.01-3.08)	1.96 (1.47-2.34)	5.32 (4.18-6.94)		
PRL (Prolactin) (ng/ml)	Mean ± SD	10.75 ± 4.69	26.24 ± 7.86	38.54 ± 11.52	9.81 ± 4.74	H: 137.467, p<0.001	$p2$<0.001, $p3$<0.001, $p4$=0.465, $p5$<0.001, $p6$<0.001, $p7$<0.001
	Median (IQR)	10.43 (7.39-15.54)	25.18 (18.87-34.40)	38.73 (27.27-47.95)	9.12 (5.56-3.24)		
E2 (Estradiol) (pg/ml)	Mean ± SD	23.22 ± 6.91	37.30 ± 7.75	48.98 ± 9.79	23.81 ± 8.26	H: 116.873, p<0.001	$p2$<0.001, $p3$<0.001, $p4$=0.741, $p5$<0.001, $p6$<0.001, $p7$<0.001
	Median (IQR)	22.88 (18.12-27.94)	38.44 (30.80-43.69)	49.64 (41.07-56.77)	24.65 (16.47-31.04)		

H: Kruskal-Wallis Test

DISCUSSION

Obstructive apnea is characterized by a full blockage of the upper airways, even when the patient is moving their chest and abdomen continuously, as evidenced by a decrease in oronasal airflow of over ninety percent from the baseline, as measured by the oronasal thermistor or nasal pressure, and lasting at least ten seconds. Respiratory event-related arousal (RERA) is a breathing disorder in which there is reduced airflow without apnea or hypopnea, with increased respiratory effort that resolves with arousal, and the event lasts at least ten seconds. Hypopnea is defined as a decrease of thirty percent from the baseline recorded by an oronasal thermistor or nasal pressure, and it must be accompanied by a decrease in oxygen saturation of no less than four percent from the baseline before the event[2] or three percent desaturation if the reduction in airflow was fifty percent[3].

Investigators have long explored and debated a possible link between OSA and erectile dysfunction and testosterone level. There is considerable evidence linking testosterone levels to the sleep cycle, as multiple studies have demonstrated that levels rise during sleep and fall during wakening in a log-linear pattern[9]. The initiation of REM sleep is impeded by disrupted sleep, which results in a delay in testosterone's peak concentration, as testosterone levels reach their maximum at the time of the first REM sleep[10]. OSA is paradoxically linked to decreased serum testosterone levels. Both hypogonadism and OSA are significantly associated with obesity[11].

Another association between OSA and ED is the amount of nocturnal hypoxia, according to reports[12]. As a key pathophysiological mechanism, elevated oxidative stress with decreased NO bioavailability and impaired vasodilation is thought to play a significant role in this setting[13]. There may be a function for either reduced testosterone or elevated levels of catecholamines and endothelin[14].

According to the demographic data, there was a significant variation in age among the severe OSA and control groups, in addition to among the mild OSA and severe OSA groups (p=0.011). In terms of BMI, significant differences were observed across all groups, with severe OSA having higher BMI values than other groups (p<0.001).

This result was not agreed with Leppänen etal.,[15] who found that; no statistically significant distinction was observed in the distribution among all groups (P =.16). Mild OSA had a median BMI of 28.8 kg/m^2, moderate OSA of 30.1, and severe OSA of 33.3 kg/m^2. There was a median age of 51.2, 51.2 & 50.7 years. The age difference between the OSA severity categories was not statistically significant (P =.63). Regarding BMI, the mild-moderate and moderate-severe groups showed a statistical significant increase BMI (P =.001 and P =.002, correspondingly).

Also, Patial et al.,[16] found that; There was a statistically significant variation in the mean ages of the participants and controls (50.90±8.15 vs. 39.26±8.36; P<0.01). But, the average BMI of the patients and the controls were comparable (32.67±3.27 vs. 31.14±2.01; P<0.061).

Cerebrovascular accidents were also significantly higher in severe OSA in contrast to control (P=0.039). This result was in line with Hui et al.,[17] who estimated that; there was significant association between OSA and the frequency of strokes; more specifically, the severity of OSA is a major risk factor for stroke development. Having a more severe case of OSA raises the risk of stroke. Inadequate CPAP treatment is associated with an increased risk of OSA in individuals with stroke.

Diabetes Mellitus was significantly more prevalent in individuals with mild OSA contrasted with both control and moderate OSA groups. This suggests that even mild forms of OSA are related to an increased risk of developing diabetes, potentially due to the metabolic disturbances linked to sleep-disordered breathing and may have another factors of developing like genetics and life style[18].

Regarding sleep parameters, there was a significant difference across all groups in AHI, ESS, and ODI, with severe OSA patients showing notably higher indices in contrast to control and mild OSA groups.

This result was in similarity with Veugen etal.,[19] who illustrated that a strong concordance of 75.5% between AHI and ODI in classifying OSA severity, with severe OSA patients exhibiting markedly higher indices compared to mild and control groups (p<0.001). This indicates that both AHI and ODI are effective measures for assessing the severity of OSA, with higher values correlating with more severe forms of the disorder. Additionally, while the mean ESS scores did not significantly differ across severity groups,in contrast to the noted trend where higher AHI values were associated with increased daytime sleepiness, suggesting that more severe OSA may lead to greater fatigue during the day.

Also. This study result in agreement with Abdelfattah et al.,[20] who revealed patients with severe OSA had significantly elevated ODI values in contrast to those with mild OSA and controls, supporting its use in clinical settings for evaluating OSA severity. Another investigation highlighted that as OSA severity increases, both AHI and ODI values rised correspondingly, emphasizing their critical role in understanding sleep-disordered breathing.

Regarding erectile function assessment, there were a significant differences in IIEF-5 and EHS scores across all groups, with severe OSA patients exhibiting notably lower erectile function and erection hardness in contrast to control (p<0.001). Mild OSA also showed a moderate impact on these indicators, with significant differences from severe OSA in EHS (P=0.038).

This resultwas in agreement with Abdelfattah et al[20] who found the prevalence of ED among OSA patients was 64.52%, with rates rising to 73.02% in those with severe OSA. The study highlighted that the severity of OSA correlated with increased rates of ED, indicating that higher AHI values are associated with greater erectile dysfunction. This reinforces the notion that OSA significantly impacts erectile function, particularly in more severe cases

Also, in this context Hoyos etal.,[21] revealed that men with severe OSA are particularly susceptible to ED due to decreased testosterone levels and impaired blood flow, both critical for achieving and maintaining erections. Approximately 64.52% of men with OSA experienced erectile dysfunction, emphasizing the strong connection between these two conditions.

Regarding hormonal profile, significant differences were observed across all groups for FSH, LH, Testosterone, PRL, and E2 levels, with severe OSA showing lower FSH, LH, and Testosterone levels and higher PRL and E2 levels compared to control (P<0.001).

This result is in agreement with Ruchała etal.,[22] that found that patients with severe OSA exhibited lower levels of FSH, LH, and Testosterone, while showing higher

levels of PRL and E2 in contrast to control groups, with statistical significance (P<0.001). This suggests that severe OSA may disrupt the hypothalamic-pituitary-gonadal axis, leading to altered hormonal profiles that could contribute to associated health issues, including sexual dysfunction and metabolic disturbances.

Another study indicated that hormonal changes, particularly involving testosterone, may play a crucial role in the pathophysiology of OSA. It was noted that hypogonadal men with low testosterone levels often have more severe OSA symptoms. The administration of testosterone has been shown to influence ventilatory control and may exacerbate OSA severity due to its effects on respiratory stability during sleep[23].

LIMITATION OF STUDY

The sample size is small and the follow up period needs to be longer and needs to be multicentic.

CONCLUSION

The findings of this research underscore the significant impact of OSA on erectile dysfunction (ED) and hormonal profiles, particularly testosterone levels. The study highlighted a clear association among OSA severity and various health parameters, involving the AHI, ESS, and ODI, all of which were significantly higher in severe OSA patients.

LIST OF ABBERVIATIONS

OSA: obstructive sleep apnea

AHI: Apnea hyponea index

ESS: Epworth Sleepiness Scale (ESS)

ODI: oxygen desaturation index

FSH: Follicle-Stimulating Hormone

LH: Luteinizing Hormone

PRL: Prolactin

E2: Estradiol

DM: Diabetes mellitus

HTN: Hypertension

ED: Erectile dysfunction

IIEF-5: International Index of Erectile Function

EHS: Erection hardness score

BMI: Body mass index

PSG: Polysomnography

IHD: Ischemic heart disease

ACE: Angiotensin-converting enzyme (ACE) inhibitors,

ETHICAL CONSIDERATIONS

This work was performed consistent with the Declaration of Helsinki. Confidentiality of data was assured in which Participants' information was replaced with research identification codes (ID Codes), data collection forms were be anonymous. Under the IRB Registration number N283-2023, the study protocol was authorized by the Faculty of Medicine at Cairo University's institutional review board (IRB). Patients could withdraw from the research at any time and still get the full medical service with in the facility. patients could refuse to participate and still get the standard and their right to know the research results were ensured. Administrative approval was obtained from the conduction site.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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