OBSTRUCTIVE SLEEP APNEOEA

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Content

Abstract

The prevalence of obstructive sleep apnea (OSA) continues to rise. So too do the health, safety, and economic consequences. On an individual level, the causes and consequences of OSA can vary substantially between patients. In recent years, four key contributors to Obstructive sleep apnoea pathogenesis. or "phenotypes" have been characterized. These include a narrow, crowded, or collapsible upper airway "anatomical compromise" and "non-anatomical" contributors such as ineffective pharyngeal dilator muscle function during sleep, a low threshold for arousal to airway narrowing during sleep, and unstable control of breathing (high loop gain). Each of these phenotypes is a target for therapy. This review summarizes the latest knowledge on the different contributors to OSA with a focus on measurement techniques including emerging clinical tools designed to facilitate translation of new cause-driven targeted approaches to treat OSA. The potential for some of the specific pathophysiological causes of OSA to drive some of the key symptoms and consequences of OSA is also highlighted.

Keywords: pathophysiology, sleep-disordered breathing, arousal, upper airway physiology, control of breathing, precision medicine

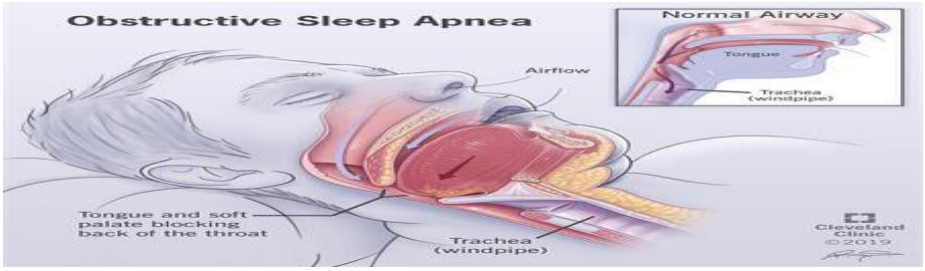
Aim

1.Prevalence of Obstructive sleep apnoea

2. Obstructive sleep apnoea among out patients clinic attendants

3.To familial with this increasing disease

Introduction

. 

About obstructive sleep apnoea

Obstructive sleep apnoea (OSA) is a relatively common condition where the walls of the throat relax and narrow during sleep, interrupting normal breathing.

This may lead to regularly interrupted sleep, which can have a big impact on quality of life and increases the risk of developing certain conditions.

Apnoea and hypopnea

There are two types of breathing interruption characteristic of OSA:

apnoea – where the muscles and soft tissues in the throat relax and collapse sufficiently to cause a total blockage of the airway; it's called an apnoea when the airflow is blocked for 10 seconds or more

hypopnea – a partial blockage of the airway that results in an airflow reduction of greater than 50% for 10 seconds or more

People with OSA may experience repeated episodes of apnoea and hypopnea throughout the night. These events may occur around once every one or two minutes in severe cases.

As many people with OSA experience episodes of both apnoea and hypopnea, doctors sometimes refer to the condition as obstructive sleep apnoea-hypopnea syndrome, or OSAHS.

The term 'obstructive' distinguishes OSA from rarer forms of sleep apnoea, such as central sleep apnoea, which is caused by the brain not sending signals to the breathing muscles during sleep

Symptoms of OSA

The symptoms of OSA are often first spotted by a partner, friend or family member who notices problems while sleep.

Signs of OSA in someone sleeping can include:

1. loud snoring
2. noisy and laboured breathing
3. repeated short periods where breathing is interrupted by gasping or snorting
   Some people with OSA may also experience night sweats and may wake up frequently during the night to urinate.

During an episode, the lack of oxygen triggers the brain to pull out of deep sleep – either to a lighter sleep or to wakefulness – so the airway reopens and can breathe normally.

Causes of OSA

It's normal for the muscles and soft tissues in the throat to relax and collapse to some degree while sleeping. For most people this doesn't cause breathing problems.

In people with OSA the airway has narrowed as the result of a number of factors, including:

- being overweight – excessive body fat increases the bulk of soft tissue in the neck, which can place a strain on the throat muscles; excess stomach fat can also lead to breathing difficulties, which can make OSA worse
- being male – it's not known why OSA is more common in men than in women, but it may be related to different patterns of body fat distribution
- being 40 years of age or more – although OSA can occur at any age, it's more common in people who are over 40
- having a large neck – men with a collar size greater than around 43cm (17 inches) have an increased risk of developing OSA
- taking medicines with a sedative effect – such as sleeping tablets or tranquillisers
- having an unusual inner neck structure – such as a narrow airway, large tonsils, adenoids or tongue, or a small lower jaw
- alcohol – drinking alcohol, particularly before going to sleep, can make snoring and sleep apnoea worse

- smoking – you're more likely to develop sleep apnoea if you smoke
- the menopause (in women) – the changes in hormone levels during the menopause may cause the throat muscles to relax more than usual
- having a family history of OSA – there may be genes inherited from your parents that can make you more susceptible to OSA
- nasal congestion – OSA occurs more often in people with nasal congestion, such as a deviated septum, where the tissue in the nose that divides the two nostrils is bent to one side, or nasal polyps, which may be a result of the airways being narrowed

Treating OSA

OSA is a treatable condition, and there are a variety of treatment options that can reduce the symptoms.

Treatment options for OSA include:

- lifestyle changes – such as losing excess weight, cutting down on alcohol and sleeping on side
- using a continuous positive airway pressure (CPAP) device – these devices prevent airway closing while sleep by delivering a continuous supply of compressed air through a mask
- wearing a mandibular advancement device (MAD) – this gum shield-like device fits around teeth, holding jaw and tongue forward to increase the space at the back of throat while sleep
- Surgery may also be an option if OSA is thought to be the result of a physical problem that can be corrected surgically, such as an unusual inner neck structure.

- However, for most people surgery isn't appropriate and may only be considered as a last resort if other treatments haven't helped.

Method

We reviewed clinical data for 50 patients with obstructive sleep apnea at Al Nasiriyah General Hospital to calculate the data . It was evaluated by a clinical outcome study. Demographic data were obtained through a structured interview and
review of medical records. Self-reported measures included the Epworth Somnolence Scale ,stop bang score,

The eligibility criteria excluded persons already treated for obstructive sleep apnea, coexisting with other sleep disturbances
such as insomnia or restless leg syndrome, regular use of sedative, hypnotic, stimulant, shift or night work, or pregnancy.
They were used to obtain data on sociodemographic factors including age, gender, race, marital status, years of formal education and comorbidities. Weight was measured on a digital scale with the affected person wearing light clothing and without shoes. Height was measured on a wall-mounted stadiameter. BMI was calculated using the following formula
: BMI = weight (kg) / height (m2).

Subjective daytime sleepiness was quantified with the Epworth Somnolence Scale (ESS) , an 8-item questionnaire
asking respondents to rate the likelihood of their sleepiness in eight drug situations. Somnolence is rated on
a 4-point
Likert scale and ranges from 0 "no chance of falling asleep" to 3 "high chance of falling asleep".
The overall score on the ESS was the endpoint for determining subjective daytime sleepiness.
We also assessment The STOP-BANG that include some questions such asSnoring: Do you snore? Loud enough to be heard through closed doors or loud enough to disturb your partner?

Tired: Do you often feel tired, fatigued or sleepy during the daytime?

Observed: Has anyone observed you stop breathing, choking or gasping while you were sleeping? Pressure: Are you being treated for high blood pressure? Body Mass Index (BMI)? Age: Are you older than 50? Neck: Is your neck size larger than 43 cm (if male) or 41 cm (if female)? Gender: Are you male?

OSA - Low Risk: Yes to 0 - 2 questions

OSA - Intermediate Risk: Yes to 3 - 4 questions

OSA - High Risk: Yes to 5 - 8 questions

or Yes to 2 or more of 4 STOP questions + male gender

or Yes to 2 or more of 4 STOP questions + BMI > 35kg/m2

or Yes to 2 or more of 4 STOP questions + neck circumference 17 inches / 43cm in male or 16 inches / 41cm in female

RESULTS

Basic demographic data of the patients: We tested 50 patient whom 44% were male and 56% were female. The median age was 44.1 years (range, 18–68years) and the median

BMI was 24.5 kg/m2 (IQR, 8.7-50.8kg/m2). The entire group had a large neck circumference [median neck circumference 39.25 cm

(IQR 28–53 cm)]. The proportion of patients with OSA was 64.0%.

There was no difference in age, sex, BMI, AHI, and ESS between groups

Demographic information and clinical characteristics of the subjects are summarized in Tables.. Patients with OSA were more likely to be

male and older. A higher prevalence of a history of smoking.

the data were also analyzed according to sex, which was assumed to be

a confounding factor,

Significant differences were observed with regard to typical and atypical complaints, including snoring, witnessed nocturnal apnea,

struggling for breath upon awakening, excessive daytime sleepiness, nocturia, and dry mouth in the morning. With regard to physical examination, all indicators of obesity (BMI, BMI grade, central obesity, and neck circumference) were significantly greater in the OSA group

than in the control group

, there were no statistically significant differences in other comorbidities.

Table 1: Sociodemographic data.

Parameter	Value	Count	Percent
Gender	Male	22	44%
	Female	28	56%

Age	<= 18 years	3	6%
	19-40 years	21	42%
	41-64 years	12	24%
	>= 65 years	14	28%
Job	Student	8	16%
	Housewife	7	14%
	Teacher	7	14%
	Retired	6	12%
	Doctor	3	6%
	Employer	3	6%
	Pharmacist	3	6%
	Engineer	2	4%
	House wife	2	4%
	Soldier	2	4%
	Child	1	2%
	Housewife	1	2%
	Lawyer	1	2%
	Police officer	1	2%
	Shopkeeper	1	2%
	Soldier	1	2%
	Unemployed	1	2%
Address	Nasiriyah	19	36%
	Shatra	11	22%
	Gharraf	6	12%
	Diwaniya	2	4%
	Al-Nasr	2	4%
	Baghdad	2	4%
	Suq-Al-Shuyouk	2	4%

	Saed-Dikhel	2	4%
	Batha	1	2%
	Jibayish	1	2%
	Al-Fajr	1	2%
	Fuhood	1	2%
Total		50	100%

Table 2: Co-morbidities:

Parameter	Value	Count	Percent
Smoking	No	33	66.0%
	Yes	17	34.0%
Snoring	No	23	46.0%
	Yes	27	54.0%
Fatigue	No	15	30.0%
	Yes	35	70.0%
HT	No	30	60.0%
	Yes	20	40.0%
DM	No	38	76.0%
	Yes	12	24.0%
COPD	No	38	76.0%
	Yes	12	24.0%
IHD	No	36	72.0%
	Yes	14	28.0%
CKD	No	47	94.0%
	Yes	3	6.0%
Hypothyroidism	No	46	92.0%

	Yes	4	8.0%
Allergic Rhinitis	No	14	28.0%
	Yes	36	72.0%
Apnea During Sleep	No	18	36.0%
	Yes	32	64.0%
BMI	< 18	3	6.0%
	> 32	8	16.0%
	18-24	19	38.0%
	25-28	12	24.0%
	29-32	8	16.0%
Total		50	100%

Table 3: Obstructive Sleep Apnea scores:

Parameter	Value	Count	Percent
Epwarth Score	0-10	35	70.0%
	11-14	10	20.0%
	15-17	4	8.0%
	18-24	1	2.0%
Stop Bang Score	<= 2	16	32.0%
	>= 5	15	30.0%
	3-4	19	38.0%
Total		50	100%

Discussion

Although limited by small sample size, this study has demonstrated an association between OSA and HT. More specifically it has shown that treated or untreated HTs also BMI is a major confounding variable in this association, but age and sex also contribute. The association between OSA and HT persists when allowance is made for the confounding variables. A detailed examination of the data demonstrated that the BP level itself is quantitatively involved in the relationship.(1)

The important features of this study are, the subjects were not biased in any way towards having sleep-related problems

The results of this study have indicated that the relationship between HT and OSA is partly due to confounding by the common risk factors, BMI, age, and sex and partly due to an association independent of these risk factors. There is insufficient evidence at present to attribute causality to the association, but studies with intra-arterial measurement of BP have shown repetitive acute rises in BP with each apnea in patients with OSA.(2)

This study complements the study of Hla and coworkers(3) which examined the relationship from the reverse direction by assessing the prevalence of HT among non snorers, snorers, and apneics selected from a general population. The relationship persisted when allowance was made for the confounding variables BMI, age, and sex. Grunstein and coworkers(4) also found that in subjects with OSA, AHI was a determinant of BP independent of obesity. Carlson and coworkers(5) found that OSA, age, and obesity were independent risk factors for HT. An increased prevalence of OSA in HTs compared with that in NTs matched for age and percentage of ideal body weight has also been found. Defining OSA using the number of desaturations per hour, Gleadhill and coworkers(6) found that after allowing for BMI, a group of treated HTs had 1.9 times the desaturation index compared with a NT group. Even though an association between OSA and HT has been demonstrated to be largely (although not completely) due to BMI, further conclusions about cause and effect cannot be drawn from this type of study. It is known that obesity can lead to both OSA and HT and this has been suggested as an adequate explanation of the OSA-HT link. Weight loss has also been shown to reduce HT and OSA in obese subjects (7)

Our data do not support this as there is some relationship persisting when allowance is made for BMI. Even when the relationship is mainly due to elevated BMI, it is still possible that there is a direct causal link between OSA and HT in overweight subjects. A number of studies have achieved a reduction in BP by

relieving OSA with nasal continuous positive airway pressure suggesting that there is a direct causal link.(8)

It is also possible that obesity may predispose to HT, secondarily causing OSA, or obesity may predispose to OSA, secondarily causing HT. There is evidence although not conclusive, to support both possibilities It has been postulated that in essential HT there is increased drive from arterial chemoreceptors, which produce ventilatory instability and sleep apnea. Repetitive apneas can cause a sustained rise in BP through the night but it is not clear if this is a cause of HT.(9)

Hoffstein and Mateika(10) found that in nonapneic snorers and in those with mild OSA the evening BP was higher than the morning BP, which is consistent with our data, although in severe OSA the morning BP was higher than the evening BP, and the difference was eliminated when subjects were matched for age and weight.

A relationship between snoring and OSA was not convincingly demonstrated in this study, and this is not unexpected as it is recognized that subjects' self-reports of snoring are not particularly accurate. As an objective measurement of snoring can be difficult to obtain and as this was not a primary aim of the study, the issue was not pursued further. It is possible that there is not a true relationship between simple snoring (without OSA) and HT, thus weakening the association between snoring overall and HT, although it has been shown in a large sample that there is an increased risk of snoring in HTs after adjusting for age and BMI (9)

In summary, this study has shown that there is a relationship between OSA and HT that, although partially explained by the confounding variables BMI, age, and sex, persists when these are controlled for

Conclusions:

Snoring, witnessed nocturnal apnea, tonsillar size,, and central obesity were used to predict the presence and severity of OSA. The results suggest

that it could potentially be useful in clinical applications used to identify patients who are at increased risk for OSA

References

1. *Wilcox, I., R. R. Grunstein, F. Collins, J. Doyle, D. Kelly, and C. Sullivan. 1992. 24-hour blood pressure in patients with obstructive sleep apnoea: "dippers" vs "nondippers." J. Sleep Res. 1(S):254.*
2. *1. Tilkian A. G., Guilleminault C., Schroeder J. S., Lehrman K. L., Simmons F. B., Dement W. C.Hemodynamics in sleep-induced apnea.*
3. *Hla K. M., Young T. B., Bidwell T., Palta M., Skatrud J. B., Dempsey J.Sleep apnea and hypertension: a population-based study.*
4. *Grunstein, R. R., I. Wilcox, T. S. Yang, J. A. Hedner, Y. Gould, and M. J. Dodd. 1992. Sleep apnoea: a confounding factor in the relationship between pattern of obesity and systemic hypertension?*
5. *Carlson J. T., Hedner J. A., Ejnell H., Peterson L.High prevalence of hypertension in sleep apnea patients independent of obesity.*
6. *Rajala R., Partinen M., Sane T., Pelkonen R., Huikuri K., Seppalainen A. M.Obstructive sleep apnoea syndrome in morbidly obese patients*
7. *Naughton M., Pierce R. J.Effects of continuous positive airway pressure on blood pressure and body mass index in obstructive sleep apnoea*
8. *Tilkian A. G., Guilleminault C., Schroeder J. S., Lehrman K. L., Simmons F. B., Dement W. C.Hemodynamics in sleep-induced apnea*
9. *Hla K. M., Young T. B., Bidwell T., Palta M., Skatrud J. B., Dempsey J.Sleep apnea and hypertension: a population-based study*
10. *Hoffstein V., Mateika J.Evening-to-morning blood pressure variations in snoring patients with and without obstructive sleep apnea*