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Short Review

Obstructive Sleep Apnea Hypopnea Syndrome

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Abstract

Obstructive sleep apnea hypopnea syndrome (OSAHS) is an important medical condition which is on the increase in the past 50 years. It causes significant morbidity and mortality in both developed and developing nations of the world. To review the relevant literature on obstructive sleep apnea. Literature review was carried out on the pathophysiology, clinical presentation, complications, diagnostic modalities, and treatment using computerized search. Additional information was obtained by cross referencing and using texts and journals in the medical libraries of Federal Medical Centre Owerri and University of Nigeria, Enugu. Most of the literature was from developed countries, with very few reports from Africa. Comprehensive management plan was lacking in many textbooks. OSAHS is an increasing medical and, more importantly, respiratory condition across the world. Early recognition and prompt appropriate intervention measures will reduce the mortality and improve the quality of life in patients with such conditions.

Keywords: Obstructive sleep apnea hypopnea syndrome, Apnea, Hyponea treatment

Introduction

Obstructive sleep apnea hypopnea syndrome (OSAHS) is an important medical condition brought to limelight in the last five decades.[1] It is a major cause of morbidity and significant cause of mortality worldwide, including developed and developing nations. A survey done in Abuja, Nigeria,[2] showed that OSAHS may be a more common medical problem than previously imagined. It is a common cause of daytime sleepiness. As the prevalence of obesity and overweight increases in both developed and developing nations of the world, the medical and, more importantly, respiratory implications are often underestimated.[3] Patients with this condition present to the clinician and early recognition and effective management of this condition will improve the patients' quality of life.

This review provides information for the clinicians assessing patients with OSAHS. It discusses the definition, pathophysiology, clinical presentation, complications, polysomnography findings, and treatment of OSAHS.

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Apnea refers to a pause in respiration for more than 10 seconds and is seen in both central sleep apnea (CSA) and obstructive sleep apnea (OSA). They are differentiated by a lack of respiratory effort in CSA versus continued but ineffective respiratory effort in OSA. Hypopnea is defined as reduction in ventilation of at least 50% that results in a decrease in arterial saturation of 4% or more due to partial airway obstruction.[1,4]

OSAHS is a syndrome associated with characteristic clinical picture and specific abnormalities on assessment. In OSAHS, there is repetitive collapse of the upper airway, which may be either partial or total resulting in hypopnea or apnea, respectively; during sleep, it occurs more than five times per hour (apnea–hypopnea index (AHI)).[3]

Review Methods

Literature review was carried out on the pathophysiology, clinical presentation, complications, diagnostic modalities, and treatment using internet Google search PubMed and Henari. Additional information was obtained by cross referencing and using texts and journals in the medical libraries of Federal Medical Centre Owerri and University of Nigeria Enugu.

Pathophysiology

Apnea and hypopnea are caused by the airway being sucked close on inspiration during sleep. This occurs as the upper airway dilating muscles, which are also striated muscles,

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normally relax during sleep. In patients with OSAHS, the dilating muscles can no longer successfully oppose negative pressure within the airway during inspiration.[1] The patients have narrow upper airways. The airway is kept patent by the dilating muscles which have higher than normal activity during wakefulness. But during sleep, the muscle tone falls and airway narrows.[1,5,6] Snoring may then occur; followed by airway occlusion and subsequent apnea. The characteristics of this condition include hypoxemia, hypercapnia, large intrathoracic pressure swings (to -120 mm Hg), and surges of systemic blood pressure of up to 250/150 mm Hg which is associated with arousals and sleep fragmentation, up to 100 times per hour.[3,5]

Symptoms include excessive daytime somnolence, non-refreshed sleep, nocturia, loud snoring, apneas and choking during sleep, morning headaches, and sexual dysfunction.

Predisposing factors include all the factors which cause narrowing of pharynx, e.g. obesity (more than 50% of obese patients have body mass index (BMI) greater than 30 kg/m^2), shortening of the mandible or maxilla. Change in jaw shape may be mild and familial. Hypothyroidism and acromegaly predispose to OSAHS by narrowing the upper airway with tissue infiltration. Other factors include male gender, middle age (40–65 years), myotonic dystrophy, Ehlers-Danlos syndrome, and possibly smoking.[1,7] The syndrome also occurs in childhood and is usually associated with tonsil or adenoid enlargement.

Consequences of OSAHS

Neurobehavioral and social

Excessive daytime sleepiness, impaired vigilance, mood disturbances, and cognitive dysfunctions are the features of OSAHS. The sleepiness may result in inability to work efficiently and may damage interpersonal relationship and prevent socializing. The somnolence is dangerous when driving and causes three- to sixfold increase in road accidents or when operating machinery.[7] Partners of patients with OSAHS experience poor sleep, and often it is the partner who prompts the evaluation, seeking relief from loud snoring.

Cardiovascular

The intermittent hypoxia, negative intrathoracic pressure variations, and arousals characteristic of apneas and hypopneas lead to increase in blood pressure at the termination of disordered breathing events evolving into sustained hypertension via chronically heightened sympathetic nervous system activity and arterial baroreceptor dysfunction.[7,8] Hypertension in the setting of OSAHS may be more difficult to treat. Large population-based studies have associated OSAHS with cardiovascular and cerebrovascular diseases.[8] Observational studies suggest an increase in the risk of myocardial infarction and stroke in untreated OSAHS.[9] Cardiac arrhythmias and cor pulmonale are commoner in these patients.[8,9]

Diabetes mellitus

Recent data suggest OSAHS is associated with insulin resistance, independent of obesity.[1] The association of OSAHS with diabetes mellitus (DM) is not just due to obesity being common in both conditions. Obesity is associated with DM, and DM may cause vascular and neuropathic damage to the dilator pharyngeal muscles and reduced upper airway sensation; this needs to be further investigated.[3,10]

Liver

Hepatic dysfunction has also been associated with irregular breathing during sleep. Nonalcoholic subjects with apnea and hypopnea during sleep were found to have raised liver enzymes and fibrosis on liver biopsy, independent of body weight.[1]

Perioperative and postoperative

Patients with OSAHS may have an increased perioperative risks. In such patients, endotracheal intubation may be more difficult and recovery may be more prolonged postoperatively.[1]

Differential diagnosis

The differential diagnoses of OSAHS include the following:

- Insufficient sleep: A good history taking can always reveal this diagnosis.
- Shift work: This is a major cause of sleepiness in workers either on rotating shift or with night work patterns.
- Psychological conditions: Depression is a major cause of sleepiness.
- Drugs: This is common with sedatives and stimulant drugs.
- Narcolepsy: Is much less common than OSAHS and usually commences from childhood.
- Idiopathic hypersomnolence: This is a term used to define long duration of sleep and sleepiness.

Clinical assessment of patients with OSAHS

The history focuses on breathing disturbances during sleep, unsatisfactory sleep quality, and daytime somnolence. History should be obtained from both patients and sleeping partners. History of habitual socially disruptive snoring and witnessed apneas terminated by gasps increases the diagnostic accuracy. OSAHS is 2–3 times greater in men. This sex protective effect is diminished in premenopausal overweight women (BMI $\geq$ 32 kg/m^2), menopausal women not on replacement therapy, and overweight women on hormone replacement therapy.[11] Other factors include smoking, alcohol, drugs, and nasal congestion.[12]

The physical examination may reveal craniofacial and soft tissue enlargement associated with upper airway resistance such as retrognathia, deviated nasal septum, low-lying soft palate, enlarged uvula and base of the tongue. Other clinical pointers include a causal role for obesity (BMI$\geq$28 kg/m^2) and neck

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circumferences of ≥43 cm.[7,12,13] OSAHS patients with other co-morbidities may predispose to severe pulmonary hypertension.

Diagnosis of OSAHS

A patient with OSAHS should be admitted. Sleep study should be performed on such a patient. Ideally, full somnographic studies should be done with recording of multiple respiratory and neurophysiologic signals during sleep. But in most centers, especially outside the USA, only "limited studies" are done which involve recording respiratory and oxygenation patterns overnight without neurophysiologic recording.[7] Episode of apnea/hypopneas occurring more than five times and lasting for ≥10 seconds is regarded as significant. This is called AHI. The OSAHS is arbitrarily defined by greater than five apneas/hypopneas per hour plus symptoms of daytime sleepiness or when AHI is >15. AHI has been used to grade the degree of severity of OSAHS. AHI of 5–14 is regarded as mild, 15–30 as moderate, and greater than 30 as severe OSAHS.[3,7]

The Epworth Sleepiness Scale,[1,14] first published in 1991 and revised in 1997, has been used to assess patients with OSAHS. Patients with scores ≥11 and experiencing sleepiness during work or driving are regarded as having OSAHS. Both the patients and the partner should be assessed and, in this case, the higher of two scores should be accepted.

Pulse Oximetry

Obstructive sleep apnea and hypopnea results in repetitive "saw tooth" oscillations in the oxyhemoglobin saturation on a time-compressed profile.[3,7] It shows marked and progressive hypopneas over the period with evidence of fragmented sleep. For diagnosing OSAHS, pulse oximetry is not considered a single alternative to polysomnography.

The Berlin questionnaire[15] is a good assessment tool for initial screening of patients suspected of having OSAHS. The questionnaire has three parts. The first part focuses on snoring, the second part on breathing pauses and daytime sleepiness, and the third part focuses on the presence of obesity and hypertension.

Treatment of OSAHS

OSAHS is a chronic condition; therefore, patient education, alleviation of airway obstruction, and follow-up are important in the optimal management of patients.

Conservative management

Lifestyle modification is important in the management of patients with OSAHS. Co-morbidities should be identified and also treated. Lifestyle modification includes weight reduction, reduction of alcohol intake, withdrawal of drugs and sedatives which affect airway tone, smoking cessation, avoidance of sleep deprivation, and adjustment of sleeping positions. Longitudinal data from Wisconsin sleep cohort study indicate that a 10% weight reduction predicts a 26% decrease in the AHI.[7]

Continuous positive airway pressure

Once decision has been made to treat OSAHS, continuous positive airway pressure (CPAP) is the preferred treatment of choice. It is a device that pneumatically splints the upper airway during inspiration and expiration while the patient is asleep. CPAP is titrated to a level that eliminates snoring, usually 5–20 cm Hg. A randomized placebo-controlled trial showed that CPAP can improve breathing during sleep, sleep quality, blood pressure, vigilance, cognition, and driving ability as well as mood and quality of life in patients with OSAHS. Problems are encountered during initiation and use of CPAP. The following questions should be asked by the clinician during assessment of patients on CPAP:

- Does anything interfere with your use of CPAP?
- Do you feel sleepy during the day?
- Does your bed partner observe snoring or breathing pauses when you use CPAP?
- Have you observed any change in weight since CPAP therapy was prescribed or last adjusted?
- When was your CPAP equipment assessed last?

Mandibular repositioning splint

Mandibular repositioning splints (MRSs)[1,16] or oral devices work by holding lower jaw and the tongue forward, thereby increasing the pharyngeal airway.

Surgery

Four forms of surgery benefit patients with OSAHS.[16]

Bariatric surgery can be curative in patients with morbid obesity. Tonsillectomy is highly effective in children. Tracheostomy is curative but rarely used because of increased morbidity. Jaw advancement surgery, especially maxilla-mandibular osteotomy, is effective in patients with retrognathia.

Drugs

Modafinil has been shown to offer a marginal improvement in sleepiness in patients with OSAHS who remain sleepy despite CPAP.[1] The clinical value and cost implication are debatable. Modafinil is contraindicated in pregnancy and lactation.

Conclusion

OSAHS is an increasing medical and, more importantly, respiratory condition across the world. Early recognition and prompt appropriate intervention measures will reduce the mortality and improve the quality of life in patients with such conditions.

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