Obstructive sleep apnoea hypopnoea syndrome (OSAHS)

G C Mbata and J C Chukwuka

Introduction

Obstructive sleep apnoea hypopnoea syndrome (OSAHS) is an important medical condition that has received increasing attention in the last five decades.[1] It is a major cause of morbidity and a significant cause of mortality worldwide, in both 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 the developed and developing nations of the world, the medical and more importantly the respiratory implications are often underestimated.[3] Patients with this condition will 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.

Apnoea refers to a pause in respiration for more than 10 seconds and is seen in both central sleep apnoea (CSA) and obstructive sleep apnoea (OSA). They are differentiated by a lack of respiratory effort in CSA versus continued but ineffective respiratory effort in OSA. Hypopnoea 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]

OASHS is a syndrome associated with a 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 hypopnoea or apnoea respectively; during sleep, occurring more than five times per hour of sleep (apnoea–hypopnoea index (AHI).[3]

Pathophysiology

Apnoea and hypopnoea 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 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 the airway narrows.[1,5,6] Snoring may then occur; followed by the airway occlusion and subsequent apnoea. The characteristics of this condition include hypoxaemia, hypercapnia, and large intra-thoracic pressure swings (to −120 mmHg) and surges of systemic blood pressure of up to 250/150 mmHg which is associated with arousal and sleep fragmentation, up to 100 times per hour.[3,5]

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

Predisposing factors include: all factors which cause narrowing of pharynx, e. g. obesity – greater than 50% of obese patients have a body mass index (BMI) greater than 30 kg/m^2; shortening of the mandible or maxilla. Change in jaw shape may be mild and may be familial. Hypothyroidism, and acromegally predispose to OSAHS by narrowing the upper airway with tissue infiltration. Other factors include male gender, middle age (40–65 years), myotonic dystrophy, Erhlers 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

Neurobehavioural and social

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

Cardiovascular

The intermittent hypoxia, negative intra-thoracic pressure variations, and arousal characteristics of apneoas and hypopnoeas lead to increase in blood pressure at the termination of disordered breathing events evolving into

Dr G C Mbata, Department of Internal Medicine, Federal Medical Centre Owerri, Imo State, Nigeria; and Dr J C Chukwuka, Department of Internal Medicine, University of Nigeria Teaching Hospital, Enugu, Nigeria. Correspondence to: Dr G C Mbata, Department of Medicine F.M.C Owerri. PMB 1010, Owerri, Imo State, Nigeria. Email mbatag@yahoo.com.

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 is not just due to obesity being common in both conditions. Obesity is associated with diabetes and this 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. Non-alcoholic subjects with apnoea and hypopnoeas during sleep were found to have raised liver enzymes and fibrosis on liver biopsy, independent of body weight.[1]

Peri-operative and post-operative

Patients with OSAHS may have an increased peri-operative risk. In such patients, endotracheal intubation may be more difficult; and recovery may be more prolonged post-operatively.[1]

Differential diagnosis

The differential diagnoses of OSAHS include:

- 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 night work patterns;
- psychological conditions – depression is a major cause of sleepiness;
- drugs – sleepiness is common in those using 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 habitualy socially disruptive snoring and witnessed apnoeas terminated by gasps increase diagnostic accuracy. OSAHS is 2–3 times more common in men. This sex-protective effect is diminished in premenopausal overweight women (BMI ≥ 32 kg/m^2), menopausal women not on hormone 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 ≥ 28 kg/m$^{2)}$ and neck circumferences of ≥43cm.[7,12,13] OSAHS patients with other comorbidities may be predisposed to severe pulmonary hypertension.

Diagnosis of OSAHS

A patient with OSAHS should be admitted. Sleep studies should be performed on such patients. Ideally, a full somnographic study should be done with recording of multiple respiratory and neurophysiologic signals during sleep. However, in most centres, especially outside the USA, only `limited studies' are done – these involve recording respiratory and oxygenation patterns overnight without neurophysiologic recording.[7] Episodes of apnoea/hypopnoea occurring more than five times and lasting for ≥10 seconds is regarded as significant. This is called the apnoea–hypopnoea index (AHI). The OSAHS is arbitrarily defined by greater than 5 apnoeas/hypopnoeas per hour plus symptoms of daytime sleepiness or when AHI >15. The AHI has been used to grade the degree of severity of OSAHS. An AHI of 5–14 is regarded as mild, 15–30 as moderate, and greater than 30 is 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 oxymetry

Obstructive sleep apnoeas and hypopnoeas result in repetitive 'saw tooth' oscillations in the oxyhaemoglobin saturation on a time compressed profile.[3,7] The profile will show marked and progressive hypopnoeas over the period with evidence of fragmented sleep. For diagnosing OSAHS, pulse oxymetry is not considered a singular 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 focuseson snoring, the second part focuses 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 educa-

tion, alleviation of airway obstruction, and follow-up is 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 that affect airway tone, smoking cessation, avoidance of sleep deprivation, and adjustment of sleeping positions. Longitudinal data from the Wisconsin sleep cohort study indicate that a 10% weight reduction predicts a 26% decrease in the AHI.[7]

Continuous positive airway pressure (CPAP)

Once a 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 cmHg. A randomised 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 last assessed.

Mandibular repositioning splint (MRS)

Mandibular reposition splints[1,16] or oral devices work by holding the lower jaw and 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 means that its benefits are debatable. Modafinil is contraindicated in pregnancy and lactation.

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