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Review Article

OBSTRUCTIVE SLEEP APNOEA HYPOPNEA SYNDROME – AN OVERVIEW

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ABSTRACT
Obstructive sleep apnoea hypopnoea syndrome (OSAHS) is a common cause of breathing-related sleep disorder, causing excessive daytime sleepiness. Common clinical features of OSAHS include snoring, fragmented sleep, daytime somnolence and fatigue. This article aims to provide a comprehensive review of the condition, including its management.
Keywords: Obstructive sleep apnoea, sleep evaluation, positive airway pressure, oral appliances.
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INTRODUCTION

Breathing-related sleep disorder refers to a spectrum of conditions ranging from simple snoring to obesity hypoventilation syndrome. Sleep disorders causing fragmented sleep during the night is known to have a negative impact on physical and psycho-social aspects of life.[1,2] Patients commonly complain of excessive daytime sleepiness (EDS). It is estimated that the prevalence of EDS in the community varies between 0.3-35%.[1,3,4] A recent study reported an EDS prevalence of 15% among the Malaysian population.[5] Although there are many possible conditions which can explain the symptom, a recent survey shows that the most common diagnosis for EDS is a condition called obstructive sleep apnoea hypopnoea syndrome (OSAHS), accounting for approximately 67% of the diagnosis.[6] This article aims to provide a comprehensive review of the clinical features and treatment of OSAHS.

The following case is a typical presentation of OSAHS:

Mr M is a 60-year-old retired engineer, who complains of lethargy and excessive sleepiness during the day. He finds that he can easily fall asleep in various situations such as when watching television, reading and after meals. His daytime sleepiness was worse before he retired as he had problems concentrating in meetings. He has had two near misses whilst driving in the past year but has never had an accident. His wife says that he snores loudly when he sleeps. Often, she notices that he chokes or stop breathing during sleep but is unrousable. However, he does get up frequently at night to pass urine. When asked about his sleeping habits, he feels that he slept well but this does not translate to feeling fresh during the morning. He also complained of a headache upon waking up. He is an ex-smoker of five years with a 40 pack year history of smoking. He denies alcohol intake and is otherwise well and independent. Examination revealed a pleasant middle aged gentleman, who was nodding off intermittently during the consultation. His body mass index (BMI) was 34. There was evidence of micrognathia and his collar size was 20 inches. He had a blood pressure (BP) recording of 153/90 mmHg. Cardiovascular, respiratory and abdominal examination was otherwise normal.

What is OSAHS?

OSAHS is a condition which causes intermittent and repeated upper airway narrowing or collapse, usually at multiple levels. This result in partial or total obstruction of the upper respiratory tract, also referred to hypopnea and apnoea, respectively. Once thought to be uncommon, the prevalence of OSAHS in middle-aged men (30-65 years) is estimated to range from 0.3-4%, with most studies quoting a prevalence of 1-2%.[7-9] It is estimated that the prevalence in women is half of that in men.[8] OSA appears to be more common in the elderly.[11]

What are the risk factors for developing the condition?

It is well recognized that narrowing of the upper airway, at multiple levels, is responsible for the consequences of OSAHS.[11] Increased muscle tone prevents the upper airways from collapsing during wake. The reverse occurs during sleep. When combining collapsibility of the airways, extraluminal pressure exerted from surrounding soft tissue structures and

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negative intraluminal pressure of the upper airway during inspiration, this results in upper airway collapse during sleep. Obesity can further reduce upper airway caliber causing more severe clinical consequences, compared to non-obese subjects.[12]

Studies show a genetic predisposition of OSAHS. The probability of developing OSAHS is greater in those with a positive family history.[13] The risk of OSAHS is higher among relatives with the condition, despite adjustments made for well known risk factors.[14] It is postulated to be related to inherited craniofacial abnormalities such as high and narrow hard palate, shorted mandibles, retroposed mandible and maxillae, and longer soft palates.

Other risk factors outlined in Table 1, should prompt the search for the condition.

Table 1: Risk factors for OSAHS

Cardiac	Atrial fibrillation
	Congestive cardiac failure
	Refractory hypertension
	Nocturnal dysrhythmias
Respiratory	Pulmonary hypertension
Endocrine	Diabetes mellitus type 2
	Obesity
Neurological	Stroke
Others	High risk driving population
	Pre-operative for bariatric surgery

What are the clinical features?

The possibility of OSAHS should be considered in any patients with risk factors and typical clinical features. Collateral history from bed partners, family members or fellow employees are helpful in deriving to a diagnosis, as they will often notice symptoms suggestive of the condition such as falling asleep unintentionally in certain situations, of which the patient may be unaware of. In the case illustration, the history was not only from the patient but the description from his wife was equally important.

Patients with OSAHS or their bed partners frequently complain of a long history of loud habitual snoring, followed by episodes of gasping and breathing pauses during sleep, resulting in wakefulness. It is not uncommon for patients with OSAHS to report undisturbed sleep when the apnoea or hypopneoa episodes occur during rapid eye movement (REM) sleep stage. In this stage of sleep, there is total muscle paralysis and any disturbances rarely results in awakening. Nevertheless, the outcome is the same regardless of presence of arousal. Repeated sleep disturbances causes chronic sleep fragmentation and EDS.

Other common complaints are unrefreshing sleep, nocturia, reduced libido, impotence, reduced concentration and morning headaches. It is not uncommon to have a history of chronic nasal congestion, hypertension and recent weight gain. Often, patients can also present with insomnia due to frequent arousals and inability to obtain a consolidated sleep.[14] A search for symptoms suggestive of the condition should be enquired as part of routine health examination (Table 2).

Table 2: Routine questions to search for OSAHS

1. Do you snore?
2. Do you suffer from daytime sleepiness?
3. Does the patient have hypertension?
4. Is the patient obese?
5. Is the patient retrognathic?

Examination is helpful. It is mainly directed to look for risk factors for OSAHS. Measurement of neck circumference and BMI are good predictors for OSAHS. However, a normal BMI does not exclude the diagnosis. Patients may have crowded oropharynx and micrognathia. Mallampati classification is a rough classification of tongue size in relevant to the oral cavity (the diagram can be obtained from *BMJ* 2004;329:955-959). It is generally used by anaesthetists to predict the ease of intubation. Mallampati class III and IV are good predictors of OSAHS.[15] BP and oxygen saturation are equally important as it may have therapeutic implications. It is important to exclude corpulmonale which can complicate long standing untreated OSAHS and other endocrinology abnormalities such as hypothyroidism and acromegaly.[30]

Are there any screening tools to measure daytime sleepiness?

Epworth sleepiness scale (ESS) is a widely used tool to assess probability of falling asleep in different situation (Table 3). It was first developed by an Australian researcher in 1990. It is currently used widely in clinical practice as a simple rapid assessment of subjective sleepiness. In some centres, it is used to prioritize investigations.

For each question, subjects are required to score between 0 to 3, with scores of 0 equate to 'unlikely to doze' and scores of 3 equate to 'high chance of dozing'. The maximum score is 24. A score of nine or less is considered normal. Patients who score more than nine are excessively sleepy and may have underlying OSAHS. In general, a higher score suggest excessive daytime sleepiness but does not confirm the diagnosis. Therefore, such screening tool should be used in conjunction with other mode of assessment.

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Table 3: Epworth sleepiness scale

How likely are you to doze off or fall asleep in the situations described above, in contrast to just feeling tired?	
Situation	**Score**
Sitting and reading	
Watching television	
Sitting in a public place (e.g. theatre or meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

How do you diagnose the condition?

When the condition is suspected and other causes of EDS have been excluded, referral to appropriate specialty is advised for further investigation. The specialties involved in the management of OSAHS differ between countries. In Malaysia, suspected cases are generally referred to respiratory physicians or ear, nose and throat (ENT) surgeon.

Pulse oximetry is useful as a screening tool for OSAHS. It is universally available and cheap. However, limited data is available to define abnormal frequency of desaturation or oxygen desaturation index (ODI). The sensitivity and specificity of pulse oximetry increase in more severe disease, indicating that it may be an effective tool for screening in certain groups of patients.[17]

The gold standard for diagnosis is polysomnography (PSG). When adequately supervised, it provides comprehensive information on sleep and breathing pattern. PSG is a multichannel recording of sleep and breathing variables. Apart from identifying patients with OSAHS and quantifying severity, it also analyzes abnormal sleep behaviours, thus facilitating other diagnosis such as periodic limb movement disorder and central sleep apnoea. Other indications for PSG are to evaluate effectiveness of treatment and titration of continuous positive airway pressure (CPAP) pressures. It is traditionally performed overnight but can also be done during daytime nap. In the latter, care must be taken in interpretation of results as it may underestimate severity.

PSG is routinely done in a dedicated sleep lab. Various electrodes and wires are attached to the patient non-invasively, at specific body position, to detect and measure different variables simultaneously during sleep. The variables or data is translated into digital signals to allow interpretation of results. Electroencephalogram (EEG), electrooculogram (EOG) and electromyogram (EMG) records brain, eye and muscle activities, respectively during sleep. The recordings help to define wakefulness and stages of sleep. Defining the sleep stage can assist in determining significant respiratory events, as apnoea and hypopnoea commonly occurs in REM sleep. Changes in respiratory effort and airflow are detected using a thoracic and abdominal belts, and nasal pressure cannulae, respectively. Interpretation is done in conjunction with pulse oximetry, measured at earlobes or fingers. This portion of PSG is central to making a diagnosis of OSAHS.

Other variables measured include body position, electrocardiograph, limb movements and snoring.

How do you classify severity of OSAHS?

The severity of OSAHS is determined by the apnoea/hypopnoea index (AHI), which is the number of apnoea and hypopnea per hour of sleep. It can be classified as mild, moderate and severe based on the AHI (Table 4). The minimum criteria for diagnosing OSAHS are excessive daytime sleepiness with AHI of more than 5. The use of an event frequency of more than 5 per hour as a threshold is based on epidemiologic data suggesting that significant adverse health events such as accidents, sleepiness and hypertension occur at or above this threshold.[16,17] The minimal threshold value for diagnostic purpose probably varies with the age of the patient. The AHI of 1 in a child may be sufficient to diagnose OSAHS. Therefore, diagnosis and treatment should be tailored to individual patients, especially in presence of adverse physiologic and neurocognitive events.

Table 4: Classification of severity based on AHI

Apnoea hypopnoea index (AHI)	Severity of OSAHS
5-15	Mild
16-30	Moderate
>30	Severe

What are the treatment options available?

Treatment options for OSAHS are conservative, surgical and positive airway pressure. The choice of treatment should be tailored to individual patients, depending on the severity, patient's preference and presence of comorbidities. Drugs have little role unless a known causative factor such as hypothyroidism is present.

Conservative treatment is usually directed to those with mild OSAHS without significant daytime symptoms or comorbidities. Lifestyle changes such as weight reduction, eliminating precipitating factors such as ethanol and drugs, can improve

outcome. Patients with positional OSAHS are advised to adopt a lateral decubitus position or head elevation using pillows when sleeping. In some patients, the use of oral appliances such as mandibular advancement devices is effective.

Driving is a potential hazard is OSAHS, as patients may not be able to make an emergency stop due to sleepiness and reduced concentration. In some countries, regulations are in place to ensure such patients are advised to refrain from driving and to subsequently inform the relevant driving authorities, until further assessment and treatment are given. Unfortunately, such regulations are not available in Malaysia. Nevertheless, appropriate advice on driving should be given when the condition is suspected.

CPAP is the treatment of choice for patients with moderate to severe OSAHS, and symptomatic mild OSAHS. It has been shown to improve daytime sleepiness, cognitive function, mood and quality of life.[18-20] Recent studies have shown that CPAP therapy in moderate to severe disease, also improves BP control and reduces cardiovascular risk factors.[21] A flow of pressurized air is delivered via a nasal or face mask to reduce the pressure difference between the upper airway and mask. This prevents closure of the upper airway. The pressure required to act as an effective 'pneumatic splint' varies between patients. The effective pressure can be determined by performing a repeat PSG whilst the patient is on CPAP and observe its effects. Alternatively, an auto-CPAP machine, which adjusts pressure depending on needs, can be used. In most centres, a positive clinical outcome is adequate to measure the effectiveness of treatment, without the need to repeat PSG whilst on CPAP.

CPAP therapy is widely available in Malaysia. As mentioned previously, the initiation of such therapy requires a firm diagnosis of OSAHS, based on clinical and physiological evaluation. Upon diagnosis, patients are commonly referred for an overnight trial of CPAP therapy to determine the appropriate pressures and tolerability. In some centres with good support, patients may be able to 'borrow' the machine for a short period, for a test drive in an acquainted home environment. This would allow the patient to familiarize usage of the machine and may improve future compliance. Purchasing a CPAP machine can be costly and ranges between RM3000 to RM8000, depending on the available functions. In some cases, with support from the attending physician, reimbursement from certain government or private agencies, use of personal long term savings, such as Employees Provident Funding (EPF) or application from social service, may assist funding.

Surgery can be considered in certain cases, when CPAP has failed or is not tolerated. Presence of bilateral tonsillar hypertrophy in symptomatic patients should prompt a referral for tonsillectomy. Other surgical approach involves resection of various parts of the upper airway, thus increasing its diameter and airflow. This includes uvulopalatopharyngoplasty (UPPP), laser-assisted uvulopalatoplasty (LAUP) and anterior mandibular osteotomy with genioglossus advancement. The success rate of surgical approach varies from 27-67%, with less well documented long-term outcome.[22,23] In one study, 63% of patients with moderate OSAHS had worsening AHI post-operatively.[24] The gold standard treatment is tracheostomy, whereby air entry bypasses the problematic upper airway. However, it is not without significant physical and psychological implications.

What are the complications of the condition?

Apart from the adverse events related to chronic fragmented sleep mentioned above, various cardiovascular complications of OSAHS are documented. Studies have shown that presence of OSAHS is an independent predictor for raised BP when all confounding variables have been allowed for.[17,19] In normal adults, systemic BP falls during sleep and rises on awakening. Subjects with variability in BP associated with a normal sleep wake cycle are called 'dippers'. In OSAHS, repeated and frequent interruption of sleep due to arousal from apnoea and hypopneoa results in a rise in BP. A significant proportion of OSAHS patients may fail to have a mean fall in BP at night and are called 'non-dippers'.[25] 'Non-dippers' are at increased risk of developing end-target organ damage such as left ventricular hypertrophy.[26] Left ventricular hypertrophy appears to be more common in OSAHS patients and the frequency increases with severity of OSAHS.[27,28] Despite evidence that treatment of OSAHS with CPAP results in a drop of 10 mmHg in both night and day time BP, it does not eliminate the need for pharmacological therapy if the need arise.[29]

Corpulmonale has been reported as a consequence of untreated OSAHS.[30] The exact mechanism is difficult to ascertain due to many confounding factors. However, it is thought to be similar to the pathogenesis of other chronic lung disease. Other cardiovascular complications which have been reported include bradycardia-tachycardia pattern, other dysrhythmias and increased risk of coronary artery disease.

CONCLUSION

OSAHS is a condition which tends to go unrecognized and underdiagnosed. With the rising of obesity prevalence worldwide, the condition is thought to become more common in the future. It has serious effects on overall health and is potentially treatable. Careful consideration of the condition, based on complaints or risk factors, is vital to ensure referrals to the appropriate specialty to confirm the diagnosis. Evidence suggests that the mainstay of treatment for moderate to severe disease is CPAP therapy. Surgical intervention may play a role but the outcome is unpredictable and not well documented.

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