

An Overview on Obstructive Sleep Apnea Diagnosis and Management in Primary Health Care Centre

Malak Abdullah Aldahasi, Johara Abdulrahman Alnafie, Maha Jamil Alhothali, Jaber Ahmed Alamri, Saleh Khalid Alghuwainem, Ozuf Nasser Alzahrani, Khaled S F Alajmi, Abdullah Saleh Alobaisy, Ahmed Khaled Ahmed, Mustafi Jamal Alkhanani, Rana Ali Alowa

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Abstract

Background: Sleep apnea is a common disorder with a range of harmful sequelae. This term includes obstructive sleep apnea and central sleep apnea. The distinction between these two types is important to optimize management for the patient. Most patients are comorbid and they suffer from obesity, heart failure, type 2 DM, or CNS disease. **Objectives:** We aimed to review the literature on sleep apnea, along with the etiology, pathophysiology, clinical presentation, diagnosis, and new management updates. **Methodology:** PubMed database was used for article selection, papers were obtained and reviewed. **Conclusion:** Sleep apnea is an important cause of the reduction in quality of life. Proper diagnosis and management require both optimal care from the treating physician and adherence by the patient. The treatment has been evolving to be tailored to each patient's needs and not following the "one-size-fits-all" method that used to be used.

Keywords: Sleep apnea, Obstructive sleep apnea, OSA, Central sleep apnea, CSA.

Malak Abdullah Aldahasi, Johara Abdulrahman Alnafie, Maha Jamil Alhothali, Ozuf Nasser Alzahrani
Faculty of Medicine, Umm Al Qura University, Makkah, KSA.

Jaber Ahmed Alamri*
Faculty of Medicine, King Khalid University, Abha, KSA.

Saleh Khalid Alghuwainem
Faculty of Medicine, King Faisal University, Al Ahsa, KSA.

Khaled S F Alajmi
Surgical Department, Al Farwaniya Hospital, Kuwait.

Abdullah Saleh Alobaisy
Faculty of Medicine, Alfarabi Medical College, Riyadh, KSA.

Ahmed Khaled Ahmed
Faculty of Medicine, King Hamad University, Bahrain.

Mustafi Jamal Alkhanani
General Practitioner, King Saud Medical City, Riyadh, KSA.

Rana Ali Alowa
Emergency Department, Qaryat Alulia General Hospital, KSA

*Email: jaber.mahssen@gmail.com

Introduction

Sleep apnea is a condition characterized by repetitive episodes of apnea or hypopnea during sleeping. (Javaheri and Dempsey, 2013; Lévy et al., 2015; Alqahtani et al., 2020; Saat et al., 2020; Alqahtani et al., 2018; Hamzekhani et al., 2019) It can be classified depending on the etiology into obstructive sleep apnea and central sleep apnea. Of the two, the obstructive variant is more common than the central one, affecting 15-30% of males and 10-15% of females in North America. However, recent data suggest that postmenopausal women have the same incidence rate as elderly males. (Peppard et al., 2013; Benjafield et al., 2019) On the other hand, central sleep apnea seems to be more prevalent in patients with heart failure, with a general prevalence of about 0.9%. (Donovan and Kapur, 2016) In this review, we will look into the pathophysiology of both central and obstructive sleep apnea, along with the clinical manifestations, diagnosis, and management of this condition.

Methodology

PubMed database was used for articles selection, and the following keys used in the mesh (((Central sleep apnea) OR Obstructive sleep apnea) OR Sleep apnea). In regards to the inclusion criteria, the articles were selected based on the inclusion of one of the following topics; Obstructive sleep apnea, Central sleep apnea, Sleep apnea, Apnea-hypopnea index. Exclusion criteria were all other articles that did not have one of these topics as their primary endpoint.

Review:

Obstructive Sleep Apnea (OSA) is a breathing-related sleep disorder in which breathing decreases or ceases completely due to upper airway obstruction. (Lévy et al., 2015) Apnea is defined as a respiratory arrest for 10 seconds or more, while hypopnea is defined as airflow by 30% or more for 10 seconds or more in combination with a reduction in blood oxygenation by $\geq 3\%$ or EEG arousal. (Rossi et al., 2020; He et al., 2020)

Pathophysiology

Multiple physiological and metabolic changes occur during the sleeping period. Sleeping is associated with decreased metabolic rate, loss of the wakefulness drive to breathe, and reduced motor output in ventilatory muscles. This means that when an individual is sleeping, the main driving force for breathing is dependent on

the level of chemo- and mechanoreceptor stimulation. (Orr et al., 2017) The main changes that affect the airway during sleep are the decreased muscle activity affecting both the neck, pharynx and respiratory muscles; decreased pharyngeal diameter; and increased airway resistance and compliance leading to a narrow airway that is easily deformed. As a result, this will lead to an increased load on the ventilatory muscles. (Lévy et al., 2015; Orr et al., 2017) The increased load during wakefulness does not represent an issue as the muscles will compensate for the increased resistance by increasing the effort. However, during sleeping, these mechanisms are absent. (D'Cruz et al., 2020)

Another factor that may predispose an individual to OSAS is their upper airway patency. The craniofacial structure is an important determinant for airway patency. Retrognathia, the extension of the lower jaw further back than the upper jaw, is one of those craniofacial abnormalities that may lead to crowding of the upper airway during sleep. Thus, resulting in OSA. (Chi et al., 2011) Another important determinant is the soft tissue structure around the neck. If the soft tissue is excessive, whether due to adipose, connective, or lymphatic tissue, it could lead to airway collapse when the muscles of the neck are relaxed during sleeping. (D'Cruz et al., 2020)

Risk factors

Several risk factors have been closely linked to the development of OSA. These include older age, male gender, obesity (BMI ≥ 30 kg/m²), craniofacial or neck soft tissue abnormalities, smoking, and family history of OSAS. (Donovan and Kapur, 2016; Morley et al., 2017) OSA has also been linked to several other conditions such as hypertension, stroke, pregnancy, acromegaly, hypothyroidism, type 2 DM, COPD, and end-stage renal disease. (Reutrakul and Mokhlesi, 2017)

Clinical Manifestation

Most patients with OSA complain of daytime sleepiness, loud snoring, choking or gasping during sleep, morning headaches, repetitive awakenings, and nocturia. (Lévy et al., 2015; D'Cruz et al., 2020) Additionally, patients may present with the conditions that are associated with OSA, as mentioned earlier, and then found to have OSA with thorough history taking. On physical examination, most patients are found to be obese with a BMI ≥ 30 kg/m². Other signs of note include crowded airway secondary to retrognathia, macroglossia, tonsillar hypertrophy, or elongated uvula; large neck circumference; and signs of the associated conditions. (Lévy et al., 2015; Reutrakul and Mokhlesi, 2017) Thus, it is imperative to take a detailed history and do a thorough physical examination to detect those conditions.

Diagnosis

OSA is not a clinical diagnosis. It requires several tests to confirm the diagnosis. However, OSA should be suspected in all patients that present with daytime sleepiness, snoring, or choking during sleep. (Rossi et al., 2020) Most experts suggest that testing should be done in patients who suffer from daytime sleepiness on most days and the presence of at least two of the following: loud snoring, witnessed apnea or choking during sleep, and a diagnosis of

hypertension. (Kapur et al., 2017) While there are questionnaires that may suggest the diagnosis of OSA, such as STOP-Bang, most sleep experts do not routinely use them. (Kapur et al., 2017) The gold standard test for OSA is in-laboratory polysomnography. (Kapur et al., 2017; Rossi et al., 2020) There are two techniques to do this test: Full-night study and Split-night study. In the full-night study, the patient is monitored for the entire night with the polysomnography, then treatment can be titrated the next day. On the other hand, in a split-night study, the patient is monitored only during the first half of the night, then treatment can be titrated during the second half. While full-night studies remain the gold standard for the diagnosis, most patients choose split-night studies as they can be diagnosed and start treatment within the same night. (Morley et al., 2017; Kapur et al., 2017) Home testing is also available, but it is only recommended in patients with high pretest probability and no comorbidities. (Mohammadieh et al., 2017) The choice for whether to test the patient in-laboratory or using a home kit depends largely on the severity of the symptoms, the nature of the patient's job, and in cases where non-respiratory etiology is suspected. (Kapur et al., 2017; Mohammadieh et al., 2017)

The criteria for the diagnosis of OSA depends on the result of the polysomnography. To make the diagnosis, the patient must experience 15 or more obstructive respiratory events per hour of sleep or if the patients experience 5 or more obstructive respiratory events per hour of sleep and one of the following is present: sleepiness and fatigue, gasping or choking, snoring or witnessed apnea, or associated conditions such as hypertension, type 2 DM, stroke, or atrial fibrillation. The obstructive respiratory events include apneas, hypopneas, and respiratory effort-related arousals (RERA). (Kapur et al., 2017; Mohammadieh et al., 2017) The polysomnography can report two values of importance, the apnea-hypopnea index (AHI) and respiratory disturbance index (RDI). AHI can be calculated by adding the episodes of apneas and hypopneas that the patient experienced divided by the total sleeping time in hours (apnea + hypopnea/sleeping time in hours). RDI adds arousals to the equation (apnea + hypopnea + RERA / sleeping time in hours). (He et al., 2020; Kapur et al., 2017; Mohammadieh et al., 2017) We can use AHI/RDI to classify the disease severity in patients to mild, moderate, and severe. In mild disease, the patient's AHI/RDI is between 5 and 14. In moderate disease, the patient's AHI/RDI is between 15 and 30. In severe disease, the AHI/RDI must be over 30. (He et al., 2020; Morley et al., 2017)

Management

The goal of the therapy is to improve sleep quality, resolve the signs and symptoms, and normalize the AHI/RDI. (Kapur et al., 2017; Mohammadieh et al., 2017) Patient education is an important part of the management. Patients must be encouraged to lose weight, change their sleeping position, avoid alcohol, and certain drugs that may exacerbate the condition such as benzodiazepines, antidepressants, and antihistamines. (Mohammadieh et al., 2017) The mainstay therapy for OSA is continuous positive airway pressure (CPAP) therapy. (Morley et al., 2017; Mohammadieh et al., 2017) The idea is that using CPAP the airway should remain patent during the entire period of sleep. Thus, preventing airway collapse and improving sleep quality. This therapy is both effective

and acceptable to patients. However, adherence remains an issue. (Al-Abri et al., 2020; Jonas et al., 2017) While there are other modalities to deliver positive airway pressure, including bilevel positive airway pressure (BPAP) and auto-titrating positive airway pressure (APAP), CPAP remains the therapy of choice as it is the most heavily tested option. (Morley et al., 2017) Follow-up is important after the initiation of therapy especially during the first few weeks to titrate the pressure to the level most suitable to the patient. Once the pressure has been titrated, patients should be followed up annually to monitor the progression of the symptoms and monitor behavioral and weight reduction. (Mohammadih et al., 2017)

Other treatment options include oral appliances, upper airway surgery, and pharmacologic therapy. Oral appliances are indicated in patients with mild disease who fail to adhere to CPAP therapy since adherence is an essential aspect of successful management. (Bratton et al., 2015) Surgical management is reserved for patients who do not improve with CPAP or oral appliances for at least three months, and whose condition can be cured surgically, for instance, patients with tonsillar hypertrophy. (Senchak et al., 2015) Pharmacologic therapy is still being investigated in hope of replacing the burdensome therapies mentioned above. (Mason et al., 2013)

Complications

Patients suffering from OSA are at an increased risk to develop systemic hypertension, coronary artery disease, arrhythmias, heart failure, pulmonary hypertension, type 2 DM, non-alcoholic fatty liver disease, neuropsychiatric dysfunction, and motor vehicle crashes. (Brzecka et al., 2020; Bouzerda, 2018; Sanderson et al., 2020; Shah et al., 2020)

Central Sleep Apnea (CSA)

CSA is a disorder characterized by repetitive cessation or reduction of both airflow and ventilatory effort during sleep. (Javaheri and Dempsey, 2013)

Classification

CSA can be classified into primary, i.e. idiopathic and secondary CSA. Secondary CSA is more common than primary CSA and occurs in association with Cheyne-Stokes breathing, heart failure, drug intake, or high altitude breathing. (Dempsey, 2019) It can also be categorized based on the breathing type into hyperventilation- and hypoventilation- related CSA. Hyperventilation-related CSA is more common and it is usually associated with heart failure, Cheyne-Stokes breathing, and high-altitude periodic breathing. Hypoventilation-related CSA is related to drug intake and CNS disease. (Javaheri and Dempsey, 2013; Dempsey, 2019)

Epidemiology

CSA is not as common as OSA in the general population. However, its prevalence increases in comorbid populations. In the general public, OSA was found in 0.9% of people. However, it was much more common in patients with heart failure with a prevalence of 4.8%. (Donovan and Kapur, 2016)

Pathophysiology

In the hyperventilation variant, the main factor that leads to the development of apnea is the level of chemoreceptor activity. During non-rapid eye movement sleep, breathing is dependent on the level of arterial carbon dioxide tension (PaCO_2). When the patient hyperventilates, the level of PaCO_2 drops below the apneic threshold. Multiple stimuli may induce hyperventilation, one of which is hypoxia which is relatively common during sleep. (Orr et al., 2017) In normal individuals, low levels of PaCO_2 does not lead to apnea due to short-term potentiation (STP). STP delays the effect of the ventilatory decline that follows hypocapnia. The sequence of events in CSA is cyclic, which explains why apneas are recurrent, unlike some forms of OSA. (Orr et al., 2017; D'Cruz et al., 2020)

As stated earlier, the hypoventilation variant is secondary to drug intake or CNS disease. During sleep, there is the loss of the wakefulness stimulus to breathing. As a result, because of loss of the wakefulness stimulus to breathing and loss of ventilatory drive from the disease process, the apnea will develop and won't be resolved until the patient is aroused from sleep to breathe. (Orr et al., 2017; D'Cruz et al., 2020)

Risk factors

The prevalence of CSA appears to be higher among older adult males, those with comorbid conditions such as heart failure, CNS disease such as stroke, and patients who use opioids. (Ratz et al., 2018)

Clinical Features

Patients suffering from CSA present similar to patients suffering from OSA. They typically present with symptoms of disturbed sleep, such as excessive daytime sleepiness, poor sleep quality, and poor concentration. (Dempsey, 2019) It is important in history to assess the patient's attention, concentration, fatigue and decreased libido since most patients do not report daytime sleepiness as a symptom due to the chronicity of the condition. Also, always ask about drug history. (Morley et al., 2017) In the physical exam, one must be mindful that there are no specific signs in CSA. However, it is important to fully examine the nervous and cardiovascular system, since CSA commonly presents with heart failure and CNS disease. (Dempsey, 2019; Mehra, 2019)

Diagnosis

The diagnostic scheme for CSA is similar to OSA. The major difference is that if the patient is suspected to suffer from OSA, full-night in-laboratory polysomnography is indicated. Ambulatory testing, like home-based testing, should not be used. (Mohammadih et al., 2017; Dempsey, 2019; Baillieul et al., 2019) The diagnostic criteria for CSA depend on the etiology. In case of primary CSA, a diagnosis is made if the polysomnography reports 5 or more central apneas per hour of sleep with no evidence of Cheyne-Stokes breathing, the patient reports daytime sleepiness and waking during the night with shortness of breath, no evidence of daytime or nighttime hypoventilation, and if the disorder cannot be explained by other conditions. (Baillieul et al., 2019) In the case of Cheyne-Stokes breathing, it has similar criteria for primary

CSA. However, the polysomnography must report findings that are consistent with Cheyne-Stokes breathing and a positive history of heart failure, atrial fibrillation, or a neurological deficit. (Baillieul et al., 2019; Sanchez et al., 2020)

Management

The goal of the therapy is to restore sleep quality to improve the symptoms and patient's daily function. However, complete management requires good management of the comorbidities of the patient. (Morley et al., 2017) In mild disease, management of the cause, if present, can be all that is needed to improve the condition and possibly eliminate it. In severe cases, therapy directed towards CSA must be started early as attempts are made to control the comorbidities of the patient.

The first-line therapy in the hyperventilation variant is CPAP. The mechanism of effect is by preventing airway narrowing and hypoxemia, we can prevent the patient from hyperventilating during sleep. Thus, we can eliminate the vicious cycle of CSA that we have discussed earlier. (Dempsey, 2019; Baillieul et al., 2019) BPAP can be used as an alternative in cases where CPAP cannot be tolerated or in case of failure of therapy. Positive airway therapy should be titrated to eliminate all episodes of central apneas in all positions of sleep. (Dempsey, 2019) Additionally, some patients may benefit from acetazolamide, a respiratory stimulant. However, due to its severe and harmful side effects, it should be given only to selected patients and those patients should be monitored closely. (Germany, 2017) In the hypoventilation variant, CPAP is not effective and BPAP should be used. Also, since most hypoventilation variant patients either suffer from CNS disease or are using opioids, adding acetazolamide and substituting the opioids could be beneficial. (Baillieul et al., 2019; Germany, 2017)

Conclusion

Sleep apnea is a major cause of disturbed sleep and a reduction in quality of life. Most patients with sleep apnea have multiple comorbidities along with the condition itself, for instance, type 2 DM, obesity, heart failure, or CNS disease. Most patients present with daytime sleepiness, choking or gasping during sleep, loud snoring, and reduced quality of sleep. This puts patients at risk of motor vehicle accidents and may put others at risk depending on the patient's occupation. Diagnosis is made with polysomnography study and assessment of severity is done with the help of the apnea-hypopnea index. Most patients are treated with continuous positive airway pressure therapy and the results are excellent. However, proper management requires proper patient education and treatment of the comorbidities of the patient.

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