Obstructive sleep apnea - risk factors, diagnosis and management

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Abstract

OBSTRUCTIVE SLEEP APNEA

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder that is characterised by obstructive apneas, hypopneas and respiratory effort-related arousals caused by repetitive collapse of the upper airway during sleep. Most pauses in breathing last between 10 and 30 seconds, but some may persist longer [1].

Key words: obstructive sleep apnea; snoring; polysomnography; CPAP

RISK FACTORS

The major risk factors for OSA include:
1) older age
2) obesity
3) male gender
4) craniofacial abnormalities
5) upper airway abnormalities
6) nasal congestion
7) smoking
8) alcohol
9) family history of snoring

Anatomical changes that contribute to oropharyngeal space reduction are among the most important risk factors of OSA. Patients with increased neck circumference and craniofacial alterations—such as increased tongue base, amygdala and uvula—or maxillomandibular deficiencies are at greater risk for apnea, because there is a reduction in the lumen of the upper airways. Sleeping in the supine position also facilitates the occurrence of apneas due to the posterior repositioning of the tongue by gravitational effect. What is more, OSA often coexists with the following comorbidities: obesity hypoventilation syndrome, congestive heart failure, hypertension, end-stage renal disease, type 2 diabetes mellitus, chronic obstructive pulmonary disease, stroke, pregnancy, hypothyroidism, acromegaly, polycystic ovary syndrome, Parkinson’s disease, gastroesophageal reflux disease. Some medications may exacerbate OSA, e.g. benzodiazepines, narcotics, alcohol.

Patients with OSA are at increased risk for: driving and motor vehicle crashes, cardiovascular and cerebrovascular morbidity, neuropsychiatric dysfunction, right heart failure, metabolic syndrome [2,3].
SYMPTOMS
Most patients with OSA complain with:
1) daytime sleepiness
2) loud snoring, choking or gasping during sleep
3) morning headaches
4) sleep maintenance insomnia
5) nocturia

During physical examination the following findings draw attention:
1) obesity
2) anatomical changes in oropharyngeal airway: retrognathia, micrognathia, lateral peritonsillar narrowing, macroglossia, tonsillar hypertrophy, an elongated or enlarged uvula, a high arched or narrow palate, nasal septal deviation, and nasal polyps
3) large neck circumference [4,5].

Screening questionnaires are evaluation tools to search for patients at risk of OSA. However, their validity in practice still remains weak, because they may detect OSA in highly symptomatic patients. The most common questionnaires used by physicians include the following:
1) the Epworth Sleepiness Scale (ESS) - a simple questionnaire measuring the probability of falling asleep in a variety of situations. The conceptual basis of the ESS involves a four-process model of sleep and wakefulness [6].
2) the STOP-Bang – an eight-item survey that incorporates information on snoring, tiredness, observed apneas (obstruction), blood pressure, body mass index, age, neck circumference and gender. A score of three or higher identifies people with OSA [7].
3) the sleep apnea clinical score (SACS) – The SACS is a four-item questionnaire that incorporates information on neck circumference, hypertension, habitual snoring, and nocturnal gasping or choking to generate a score ranging from 0 to 100. It reliably predicts OSA for patients in family medicine practice: scores greater than 15 result in a probability of OSA of 25 to 50 percent [8].
4) the Berlin questionnaire – The Berlin questionnaire consists of 10 items relating to snoring, nonrestorative sleep, sleepiness while driving, apneas during sleep, hypertension, and BMI. The results stratify patients as having a high or low risk for OSA [9].
5) the NoSAS score – The NoSAS score assigns points based upon five parameters (neck circumference, body mass index, snoring, age, and gender). In a derivation and validation analysis, a score ≥8 identified individuals at risk of clinically significant sleep-disordered breathing [10].
DIAGNOSIS
Polisomnography (PSG) remains the gold-standard diagnostic test for OSA.
In patients who undergo PSG, the diagnosis of OSA is confirmed if either of the two criteria below are met:
1) There are 15 or more apneas, hypopneas, or RERAs per hour of sleep (ie, an AHI or respiratory disturbance index [RDI] ≥15 events per hour) in an asymptomatic patient.
2) There are five or more obstructive apneas, obstructive hypopneas, or RERAs per hour of sleep (ie, an AHI or RDI ≥5 events per hour) in a patient with the following: sleepiness, nonrestorative sleep, fatigue, or insomnia symptoms; waking up with breath holding, gasping, or choking; habitual snoring, breathing interruptions, or both noted by a bed partner or other observer; and/or hypertension, mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus [11].

Polysomnographic subtypes, distinguished by respiratory event association with hypoxemia, arousals, or both, exhibit varying risks of cardiovascular disease and potential response to therapy [12].

TREATMENT
Behavior modification, which is indicated for most patients with OSA includes:
1) losing weight (if overweight or obese)
2) exercising
3) changing the sleep position (if OSA is positional)
4) abstaining from alcohol
5) avoiding certain medications eg. benzodiazepines, barbiturates, antiepileptic drugs, sedating antidepressants, antihistamines, opiates.

Positive airway pressure therapy is recommended as initial therapy for adults with OSA. The mechanism of continuous positive airway pressure (CPAP) involves maintenance of a positive pharyngeal transmural pressure so that the intraluminal pressure exceeds the surrounding pressure. CPAP also stabilizes the upper airway through increased end-expiratory lung volume. As a result, respiratory events due to upper airway collapse (eg, apneas, hypopneas) are prevented. [13, 14, 15].

OSA is a heterogeneous disorder. There are 3 basic subtypes replicated in multiple studies:
1) a group where insomnia is the main symptom;
2) an asymptomatic group;
3) a group with marked excessive sleepiness.

The symptomatic benefit from treatment with nasal CPAP varies between these 3 subtypes. Data from the Sleep Heart Health Study reveal that the increased risk of cardiovascular disease from OSA occurs only in the excessively sleepy group [16].

Other treatments include dental devices and surgery. According to Chapman et al. pharmacologic therapy (with agents such as modafinil or armodafinil) may be beneficial as adjunctive therapy for excessive daytime sleepiness that persists despite documentation of adequate and successful conventional therapy (eg, positive airway pressure, oral appliances) [17].
Liu et al. discovered that acetazolamide improves sleep apnea at high altitude by decreasing AHI and percentage of periodic breathing time and increasing nocturnal oxygenation. Acetazolamide is more beneficial in healthy participants than in OSA patients, and a 250 mg daily dose may be as effective as higher daily doses for healthy trekkers [18].

Upper airway stimulation (UAS) has been shown to reduce severity of OSA. In a large multicentre international registry, UAS is an effective treatment option with high patient satisfaction and low adverse events. Increasing age and reduced BMI are predictors of treatment response [19].

REFERENCES