REVIEW ARTICLE

Pediatric Obstructive Sleep Apnea: A Narrative Review

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Abstract

Pediatric obstructive sleep apnea (OSA) is a broad-spectrum disorder that needs timely diagnosis and management. It is mainly caused by anatomical factors. Adenotonsillar hypertrophy is an important correctable cause. We describe a narrative review of this disease in children. Although uncommon in adults, it is an important disorder causing mortality and morbidity in children which warrants timely diagnosis and management.

Keywords: Apnea–hypopnea index, Obstructive sleep apnea, Pediatric.

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Introduction

Pediatric sleep-disordered breathing is a spectrum which includes primary snoring, upper airway resistance syndrome, obstructive hypoventilation, and obstructive sleep apnea syndrome (OSAS). Obstructive sleep apnea syndrome in children was described by Osler in 1892. The first case series was published by Guilleminault et al. in 1976.[1] Habitual snoring is reported in 8–12% of children but only 1–3% show associated OSAS.[2] Pediatric OSAS is most commonly caused by anatomical abnormalities like adenoid and tonsillar hypertrophy, which is correctable by surgery. It is also observed with obesity, syndromes associated with obesity, and orofacial abnormalities in children. Pediatric OSAS can adversely affect the child's physical, mental, and emotional growth. We discuss the definitions, etiology, diagnostic approach, and management.

Definition

The American Association of Pediatrics (AAP) guideline defines OSAS in children as a "disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts normal ventilation during sleep and normal sleep patterns," accompanied by symptoms or signs, as listed in Table 1.[3]

Etiology

The various causative factors for OSAS are enumerated in Table 2. The pathophysiology of childhood OSAS remains poorly understood.[4] However, it is proposed to be caused by a combination of anatomic and neuromotor factors, that is, by the superimposition of structural aberrations upon an integrally more collapsible upper airway. The most important contributory anatomic factor is adenotonsillar hypertrophy, particularly in the preschool age group; the other being craniofacial abnormalities. Obesity leads to changes in upper airway anatomy, upper airway neuromotor tone, and imbalance of the pharyngeal dilator and constrictor muscles, and the inflammatory hypothesis suggests the role of leukotrienes.[5,6] Children are at increased threat of developing pulmonary hypertension, cor pulmonale, and neurocognitive dysfunction.[7,8] Complications of pediatric OSAS are illustrated in Table 3.

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Table 1: Symptoms and signs of OSAS

History	Frequent snoring (≥3 nights/week), labored breathing during sleep, gasps/snorting noises/observed episodes of apnea, sleep enuresis (especially secondary enuresis), sleeping in a seated position or with the neck hyperextended, cyanosis, headaches on awakening, daytime sleepiness, attention-deficit/hyperactivity disorder, and learning problems
Physical examination	Underweight or overweight, tonsillar hypertrophy, adenoidal facies, micrognathia/retrognathia, high-arched palate, failure to thrive, and hypertension

History and Clinical Examination

History and physical examinations are essential to diagnose OSAS in children. The AAP guidelines recommend that clinicians as a part of routine health-maintenance visits should ask for snoring and look for other clinical effects of obstructive sleep

Table 2: Predisposing factors to pediatric OSA

• Adenotonsillar hypertrophy
• Obesity
• Allergic rhinitis
• Craniofacial malformations
• Neuromuscular diseases
• Genetic syndromes
• Metabolic syndromes

Table 3: Obstructive sleep apnea complications in children

1.	Effects on growth	• Obesity • Lethargy • Abnormalities related to syndromic association
2.	Neurocognitive	• Hyperactivity, inattention, aggression • Impaired school performance • Daytime sleepiness • Depression
3.	Cardiovascular consequences	• Pulmonary hypertension • Atherosclerosis, PVD • Corpulmonale • Systemic hypertension • Atrial fibrillation and arrhythmias
4.	Metabolic problems	• Thyroid associated abnormalities • Insulin resistance and DM • Hyperlipidemia

Table 4: Syndromes associated with OSA in children

• Prader Willi syndrome, Teacher–Collins syndrome, Bardet–Biedl syndrome, and Beckwith–Wiedemann syndrome
• Achondroplasia
• Crouzon syndrome and Aperts syndrome
• Duchenne muscular dystrophy and spinal muscular atrophy
• Myelomeningocele
• Pierre Robbin syndrome
• Cerebral palsy
• Down syndrome
• Sickle cell disease
• Choanal stenosis
• Osteopetrosis
• Klippel–Feil syndrome and Hallerman–Streiff syndrome
• Mucopolysaccharidosis

apnea (OSA).[9,10] Audiotapes or videotapes of the sleeping child recorded by the parent can occasionally be used by healthcare teams to hear and watch for noticeable apneic episodes.[11,12] The regular healthcare visits and school clinic visits should screen for sleep history. History of labored breathing during sleep observed apnea, restless sleep, diaphoresis, enuresis, cyanosis, excessive daytime sleepiness, and behavior or learning problems should be asked for. Certain syndromes are associated with OSA in children (Table 4). Findings such as malnutrition (under or overweight), adenoid facies, nasal obstruction, adenotonsillar hypertrophy, micro/retrognathia, and hypertension may be present on clinical examination. Systemic hypertension, an enhanced pulmonic component of the second heart sound representing pulmonary hypertension, and reduced growth may be observed as complications of underlying OSA. Adenotonsillar hypertrophy may be present. It is graded as follows: Grade I, less than 25% space between pillars; grade II, less than 50% space between pillars; grade III, less than 75% space between pillars; and grade IV, tonsils in direct contact.[13,14]

Table 5: Obstructive sleep apnea severity criteria in children and adults[7]

OSA severity	*AHI in children*	*AHI in adults*
None	0	0–5
Mild	1–5	5–15
Moderate	5–10	15–30
Severe	>10	>30

Sleep Questionnaires

Sleep questionnaires were developed to (1) diagnose pediatric OSAS and (2) assess quality of life and response to OSAS therapy. These questionnaires and clinical scoring scales are not completely accurate and standardized. They could be used as per the clinic/institute/sleep physician practice preferences for triage management in children. Some of these questionnaires include Chervin et al.[15] developed the pediatric sleep questionnaire (PSQ); Owens and Dalzell[16] developed "BEARS" sleep screening score, Franco et al.[17] developed OSA 18, de Serres et al.[18] developed OSD 6, Kadmon et al.[19] published questionnaire I'M SLEEPY.

Polysomnography

Overnight polysomnography (PSG) evaluation in a sleep laboratory is the gold standard for diagnosing OSA at all levels of severity. Pediatric apnea–hypopnea index (AHI) severity criteria are as follows (<12 years of age): AHI, from 1 to <5 is mild; from 5 to ≤10 is moderate; and above 10 is severe (Table 5). Polysomnography is costly, time consuming, requires specialized expertise, has limited accessibility, and may entail long waiting periods and costs. There are other challenges in carrying out and interpreting PSG in children compared with a supportive adult. The laboratory and technologist must show a friendly approach and be comfortable with the child while not being too childish to discourage adolescents.

In adults, obstructive apneas of 10 seconds or longer are scored, but in children, apnea is scored if the decrease in oronasal flow of more than 90% is for at least two respiratory cycles in the presence of respiratory effort. The diagnostic algorithm involves triage and integrates the use of screening tools, questionnaires, and alternative tests for OSAS, and selective referral for PSG in resource-limited situations.

The AAP guidelines recommend that clinicians as a part of regular health-maintenance visits should ask about snoring, that is, whether the child or adolescent snores.[3] If a child snores on a regular basis and reveals any of the problems or findings given in Table 1, clinicians should either get a polysomnogram (level A) or refer the child to a sleep specialist or otolaryngologist for a more wide-ranging evaluation (level D). If PSG is not obtainable, then clinicians may direct for other diagnostic tests, such as

nocturnal video recording, nocturnal oximetry, daytime nap PSG, or ambulatory PSG (level C).

If a child is detected as experiencing OSAS, shows a clinical inspection consistent with adenotonsillar hypertrophy, and does not have a contraindication, then adenotonsillectomy can be suggested as the first-line therapy. The clinical decision is mandatory to examine the benefits of adenotonsillectomy compared with other therapies in obese children with different degrees of adenotonsillar hypertrophy (level B).[3]

Management of Obstructive Sleep Apnea in Children

Obstructive sleep apnea in children is a distinct disorder from that occurs in adults with respect to clinical manifestations, PSG diagnostic criteria, and treatment approaches. A simplified multimodality diagnostic algorithm is needed for early diagnosis and treatment. Adenotonsillectomy is the therapy of choice in typical cases. Treatment with montelukast results in a signification decrease in adenoid size and in respiratory-related sleep parameters.[20] Anti-inflammatory therapy of childhood OSA in the form of intra nasal corticosteroid is an encouraging method that might substitute surgical treatment in children with mild OSA.[3] Continuous positive airway pressure device (CPAP) is suggested as therapy if OSAS perseveres postoperatively and weight loss, in addition to other treatments in patients who are overweight or obese. A holistic evaluation and management for endocrinopathies, obesity, and metabolic disorders are necessary, as OSA is often an accompaniment of syndromes common in childhood especially those causing obesity.

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