Pediatric Obstructive Sleep Apnea Syndrome - Mini Review

Abstract
Obstructive sleep apnea syndrome (OSAS) affects about 2% of children in the pre-school phase and can reach 12% in the pre-pubertal phase. The main clinical sign is snoring, and this should not be ignored. Characterized by nighttime respiratory arrest from total or upper airway obstruction, the disease leads to chronic sleep deprivation and consequently a series of metabolite, cognitive and behavioral changes in children. Treatment should occur as early and as effectively as possible, in an attempt to prevent the progression of the disease and its side effects in adult life. Adenotonsillectomy is gold standard treatment, providing an improvement in nasal and pharyngeal permeability. However, it does not correct morphological changes; it can lead to preoperative complications besides being costly. A more affordable, less invasive and less risky alternative treatment for children is needed. Studies addressing the rapid expansion of maxilla have been conducted; the results are promising but are still not well established in the literature.

Keywords: Pediatric obstructive sleep apnea; Primary snoring; Adenoidectomy; Tonsillectomy; Polysomnography; Upper airway resistance syndrome; Rapid expansion of maxilla

Abbreviations: OSAS: Obstructive Sleep Apnea Syndrome; PSG: Polysomnography; AHI: Apnea and Hypopnea Index; CPAP: Continuous Positive Airway Pressure

Introduction
Sleep
Sleep is an activity that occupies one third to one half of our lives and is fundamental to good mental and emotional health, as well as being essential in the healthy maintenance of the body. Electroencephalographic patterns as well as the duration of different stages of sleep change throughout life. Children have different needs from adults, and adults, in turn, with different ages and stages of life, also have individual needs.

Obstructive sleep apnea syndrome
Obstructive Sleep Apnea Syndrome (OSAS) is classified as respiratory sleep disorder, affecting individuals of all ages, from neonates to the elderly. Recurrent episodes of total obstruction (apnea) or partial (hypopnea) of the upper airway simultaneously with respiratory effort during sleep lead to chronic sleep deprivation and the child suffers from all its consequences [1]. The spectrum of the disease begins in snoring, progresses to resistance syndrome, obstructive sleep hypopnea until apnea due to total obstruction of the upper airway [2]. Studies show that even children with only primary snoring already present behavioral changes and cognitive deficits when compared to healthy children [3,4]. According to the International Classification of Sleep Disorders (ICSD-2) [5], OSAS is mentioned in the category of "sleep-related respiratory disorders". This category is one of eight that describe such disorders. It classifies them according to their pathophysiological and clinical characteristics including the disorders of the adult, the child and the adolescent.

Diagnostic method
The International Classification of Sleep Disorders describes that the diagnosis of OSAS involves specific questionnaires, clinical history, physical examination and classical polysomnography (PSG) [5]. The use of PSG in a specialized laboratory with the accompaniment of trained professionals allows the evaluation of electroencephalogram (EEG), electro-oculogram (EOG), noninvasive electromyography (EMG) of the chin and lower limbs, oronasal flow measurements, thoracic-abdominal movement, electrocardiogram (ECG), pulse oximetry, and body position. With these records, we can calculate AHI (sleep apnea and hypopnea index per hour of sleep), oxyhemoglobin desaturation, percentages of sleeps stages; sleep efficiency and fragmentation, which can be attributed to respiratory events. It can be stated that PSG is the main objective method of diagnosis of OSAS [6]. The severity of OSAS is based on its PSG indices; by the intensity of its symptoms; by its impact on social and professional functions, taking into account gender, age and profession; and by the presence of cardiovascular diseases [1]. In children, OSAS is classified as mild when the patient presents AHI between 1 and 5 respiratory events per hour of sleep, moderate apnea for children presenting between 5 and 10 events / h and severe when recorded in PSG, more than 10 events per time of sleep [1].
Discussion

The exact mechanism of this syndrome in children has not yet been fully elucidated, although important anatomical risk factors have been identified and are correlated with adjacent structures affecting the size and shape of the airway. Several techniques such as lateral neck radiography, cephalometry and magnetic resonance imaging have shown that the airway of children with apnea is lower than that of healthy children [7,8]. During childhood, in the majority of cases, hypertrophy of the soft tissues attached to the upper airway, such as adenoid and palatine tonsils, may explain the clinical symptoms of children with OSAS and the surgical removal of these tissues may resolve or minimize this disorder [9]. However, it is estimated that in 10 to 15% of children with apnea, this disorder cannot be treated by simply removing the palatine and adenoid tonsils [10]. It is possible that the three-dimensional orientation of these tissues and how they overlap in the airway are preponderant factors and can significantly affect airway resistance [11]. In 2001, Sanchez-Armengol and colleagues reported that the prevalence of OSAS was estimated at 1.9% for adolescents [12]. It is not known if this disorder appears in adolescence as an extension of the alteration found in children, that is, where the main risk factor is adenotonsillar hypertrophy, or if it represents an early manifestation of the disease found in adults, being obesity and retrognathism mandibular risk factors [11]. Some studies have discussed the relationship between sleep-disordered breathing and the developmental phase (childhood and adolescence). In a retrospective study, it was reported that this disorder was more frequent in adolescents who underwent adenotonsillectomy during childhood [13]. On the other hand, Guilleminault et al. [14] noticed changes in craniofacial morphology in adolescents with apnea and a history of upper airway obstruction during childhood [14]. These authors also reported that the predisposition of these patients to this disorder would be related to genetic factors that would result in alterations in craniofacial growth or secondary alterations of the growth pattern in response to adenotonsillar hypertrophy. It is worth emphasizing the need for randomized studies to evaluate the long-term effects of treatments on the improvement of AHI levels as well as oxyhemoglobin saturation, considering the effect of infant growth, as well as the spontaneous resolution of the disease [15].

References


