Review Article

OBSTRUCTIVE SLEEP APNEA IN CHILDREN: A REVIEW

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Abstract

Disruptive sleep apnea in offspring is highly prevalent disorders caused by a assortment of complex patho-physiological process which may leads upper airway obstacle. Disruptive sleep apnea's experimental significance stems from its link to significant morbidities that alter systemic and metabolic systems. The result shows that a multipart interchange involving adenotonsillar hypertrophy and loss of neuromuscular tendency may be involved in the pathophysiology of OSA in kids. This review will brief highly structured some of the pathogenetic elements leading to disruptive sleep apnea in kids.

Keywords : Obstructive sleep apnea, Obesity, Sleeping disorders , Snoring.

INTRODUCTION

Obstructive sleep apnea (OSA) is a sleep breathing condition marked by periods of prolonged upper airway resistance and frequent episodes of partial and/or total obstruction of the upper respiratory tract, resulting in normal breathing and oxygenation are interfered with, as well as typical nap pattern. On polysomnography, an apnea hypopnea index (AHI) greater than one can aid in the detection of OSA in children. If the occurrence are primarily obstructive and not central in personality.[1]

Obstructive sleep-disordered breathing is frequent in children. Snoring, mouth breathing, and obstructive sleep apnea are common causes for parents to seek medical care for their children (OSA). Snoring affects 3 to 12 % of youth, while OSA affect 1 to 10% of the population.[2–4] The majority of these kids have little symptoms, and many of them outgrow it. Adenotonsillar hypertrophy, neuromuscular dysfunction, and craniofacial anomalies are all common causes of OSA.

Sleep-disordered breathing includes snoring, upper airway resistance syndrome, obstructive hypopnea syndrome, and OSA.[5] Upper airway resistance syndrome is the mildest form of OSA in children. Affected kids contain symptoms of OSA except the complementary polysomnographic outcome. Whereas many kids snore and breathe through their mouths on occasion, severe OSA causes medical complications such as failure to thrive, behavioural simplicity, enuresis, and corpulmonale.

Given the increased incidence of obesity and hyperactivity in this population, children's sleep-disordered breathing is a pressing public health issue. Sleep-disordered breathing was found in a substantial number of children with hyperactivity or inattentive tendencies, according to the study.[6] The use of stimulant medicines in these children would be more effective than correct detection and treatment of sleep-disordered breathing.[7-9]

EPIDEMIOLOGY

OSA affects between 2% and 3% of all children from birth to puberty, according to estimates.[10-14] It affects both boys and girls equally, and is most common between the ages of two and eight. This corresponds to the age when adenotonsillar hypertrophy is generally noticeable. Due to increased obesity prevalence in this time range in

numerous part of the human race, particularly the US, an increase in prevalence has lately been documented in the middle age and teenager age groups. The outstanding difference stuck between OSA in adults and offspring are scheduled in table 1.

TABLE-1. Features of Paediatric and Adult OSA

Characteristic	Paediatric OSA	Adult OSA
Gender	Males = females	Males more than females
Peak period	nursery (2-8 years)	central point age
Etiology	Adenotonsillar	Hypertrophy ,Obesity
Clinical features	Snoring	Snoring
Diagnosis	Polysomnography	Polysomnography
Primary treatment	Surgical Medical (CPAP)	
	(adenotonsillectomy) [1,5]	

PATHOPHYSIOLOGY

In most kids with OSA, physical examination indicates adenotonsillar hypertrophy. Adenotonsillectomy has been shown to ameliorate clinical symptoms. [7-9] [Evidence level B, clinical cohort studies, Strength of Recommendation (SOR)] Many children with adenotonsillar hypertrophy, on the other hand, never develop OSA symptoms. This finding shows that adenotonsillar hypertrophy and loss of neuromuscular tone may play a role in the development of OSA in youngsters. Children with craniofacial disorders have fixed anatomic differences that predispose them to airway blockage, whereas hypotonia causes obstruction in kids with neuromuscular illness. [15]

CLINICAL FEATURES

The characteristic of paediatric OSA is snoring and OSA is extremely doubtful in the nonexistence of snoring on a regular basis [16] not all children who snore, however, have OSA. According to the results of polysomnography, between 6% and 12% of all children snore at night, with only 10% to 30% of these children having obstructive sleep apnea.[16] During their annual health check-up, all children should be tested for snoring.[1] The symptoms of OSA in children can be divided into two categories: night time symptoms and daytime symptoms, as shown in table2.[1,14,16-21]

TABLE- 2. Symptoms of OSA in kids

Nighttime symptoms	• wheeze • Sleeping in unusual positions • Nocturnal sweating • Enuresis
Daytime symptoms	• Learning difficulties • Deterioration in academic performance • Morning headaches • Mouth breathing • History of recurrent infectious illness

kids with OSA may have clinical symptoms indicative of the condition or have a entirely normal physical appraisal. Obesity or a failure to thrive are both potential outcomes.[1,16,17] Although the presence or absence of adenotonsillar hypertrophy does not authenticate or rule out the diagnosis.

It was possible to see retrognathia, midface hypoplasia, and other craniofacial anomalies linked to certain hereditary diseases. A hyponasal voice, nasal polyps, choanal atresia, or septal deviation may be discovered during a nasal cavity examination. Hypotonia is common

in patients with neuromuscular diseases, thus muscle tone should be checked. While laying supine, these persons may adopt unique postures, such as a frog-leg position, reduced joint resistance to passive movement, and enhanced limb range of motion. Even though a thorough medical history and physical examination can help uncover potential cause and effects of obstructive sleep apnea, clinical criteria are neither specific nor sensitive enough to analyse OSA or make a distinct it from primary snoring.[1]

Cardio pulmonary	hypertension • Impaired right ventricular function– rare •Corpulmonale – exceptional
Expansion and metabolism	• Malfunction to succeed • Postponed mental expansion

DIAGNOSIS

As illustrated in Table-3,[1,20-25] untreated OSA in children can lead to serious cardiovascular, metabolic, and neuropsychological problems. A thorough physical exam and a thorough medical history are helpful in detecting OSA, but they are not diagnostic.

TABLE-3. Complications of OSA in Children

Behavioural	•Violence •Hyperactivity • Nervousness •Miserable frame of mind •Psychosocial problems •Night-time enuresis
Cognitive	•Carelessness • Impaired supervisory implementation • Impaired recollection • Impaired scholastic utility
	• Systemic

Polysomnography (PSG) is the gold standard and favoured diagnostic test since it not only confirms the diagnosis of OSA but also classify the cruelty of symptom.[1] This overnight test, which attempts to measure several physiologic parameters at same time, which together with sleep stages, eye arrangements, muscle activity, respiration (PCO2 and SaO2), cardiac activity, and snoring, is best performed in a sleep centre committed to paediatrics over conditions that are as close to the child's sleep environment as possible.

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PSG in the paediatric age group, such as the lack of standardisation of data analysis and interpretation, as well as the lack of paediatric dedicated sleep centres and personnel. Due to their inherent limitations, these tests have been rigorously reviewed and are currently only used for screening reasons. Even negative results must be followed up with an overnight PSG if there is a high index of suspicion for OSA.[1]

TREATMENT

It has been previously reviewed about the physiopathology of the osa[26,27] and even while obesity is now a far more prevalent occurrence, and requires a lesser degree of lymphadenoid enlargement, it is obvious from this research and other sources that OSA in children is most typically related with adenotonsillar hypertrophy.[28-30] Surgical removal of the adenoids and tonsils is the recommended first treatment, even in obese youngsters.[31,32] However, not all children with OSA who have an adenotonsillectomy are cured.[33–35] The success rate for adenotonsillectomy in the context of OSA was approximate to be around 85 percent in a meta-analysis of the available material.[36] This number could be even lower, especially among obese children with OSA[37–41] or children with stringent OSA.[37,42] Following these findings, it is now recommended that patients undergo frequent nightly sleep studies following adenotonsillar surgery for OSA.[37] Though long-term outcomes in children with OSA are unclear, accumulating evidence suggests that OSA will recur in a subset of these patients,[42] particularly in those with craniofacial problems or a familial history of OSA.[42–46] The surgical technique employed for extirpation of lymphoid tissue (e.g., cold surgery, coblation, harmonic laser); and the requirement for tonsillectomy and adenoidectomy, either one of these two surgical operations alone, or tonsillectomy alone, are two more topics for which there is no definitive proof.[47–49] In the future, these difficulties will need to be solved. The only other interventional option for children whose T & A does not result in complete remission of OSA and whose residual severity of sleep-disordered breathing is moderate to severe (i.e., obstructive AHI. 5/h), is the administration of nasal continuous positive airway pressure (CPAP).[50–55] Despite a scarcity of size-appropriate masks, overall adherence rates appear to be reasonable,[56] and these rates could be enhanced further by providing behavioural interventions[57] and family logistic support. Those youngsters with an AHI greater than 1, but fewer than 5, incidents per hour of sleep are in the grey zone when it comes to OSA therapy. Indeed, despite the fact that these individuals are at high risk for associated morbidity, the risk-benefit ratio of surgical adenotonsillectomy has yet to be determined, and CPAP is less likely to be useful and effective when administered to an airway partially occluded by swollen lymphadenoid tissues. As a result of these factors, researchers are looking for therapeutic options. Topical intranasal administration of high-potency corticosteroids has been one strategy. Significant improvements in AHI and oxygenation have been shown in a cohort of children with OSA and AHI greater than 5 or children with enlarged adenoids in a series of trials.[58–61] Given the expression patterns of glucocorticoid receptors a and b in the upper airway, which imply favourable therapeutic responses to topical corticosteroid treatment in children with OSA, these findings are not surprising.[62] In addition, in children with OSA, the concentrations of inflammatory mediators such as leukotrienes and the expression of their receptors were shown to be higher,[63,64] and a leukotriene receptor antagonist was reported to be helpful in mild paediatric OSA.[63] Anti-inflammatory medication was also effective in reducing the amount of OSA that remained following adenotonsillectomy.[65] Oral appliances, also known as functional orthopaedic appliances, have been used to treat OSA in children with modest success.[66-68] However, it is unknown whether selection criteria should be used to identify patients who are likely to benefit from this therapeutic alternative, as well as what the long-term outcomes of this method would be. There is a paucity of information regarding short- and long-term results, as can be quickly deduced from the

existing compilation of the literature regarding the management of paediatric OSA. Future research, particularly in the context of type I, II, and III paediatric OSA categories, will be critical for optimising the care delivered to our kids and ensuring long-term health and quality of life improvements.[68]

CONCLUSIONS

The illness spectrum that includes snoring and OSA in children is linked to an increased frequency of a number of morbidities disturbing the CNS, cardiovascular, and endocrine systems. Obesity and OSA cohabitation appears to be associated with a distinct and overall predictable clinical phenomenology, as well as superior morbidity rates and poorer therapeutic outcomes. Since the early management techniques, therapeutic options have expanded to include not only surgical exclusion of hypertrophic adenoids and tonsils, but also nonsurgical therapies such as CPAP, antiinflammatory medication, and dental appliances. Conversely, recommendations based on well-designed, randomised trials are needed to determine the efficacy and most excellent purpose of all of these choices.

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