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Childhood Obstructive Sleep Apnea

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ABSTRACT

Obstructive sleep apnea (OSA) is a common condition in childhood and can result in severe complications if left untreated. It is showing a rising trend in India. A significant association with obesity has been observed; however, some children with enlarged tonsils and/or adenoids may even be underweight. The patient usually presents with snoring and other respiratory problems like mouth breathing, choking and gasping episodes in night. Poor school performance and neurocognitive deficits have been reported. Pulmonary hypertension and cor pulmonale are seen in severe cases. Besides the history and clinical examination, for definitive diagnosis an overnight polysomnographic evaluation is the gold standard. In all cases, the specific treatment ranges from simple lifestyle modifications and medications to surgeries like adenotonsillectomy. Early diagnosis is vital.

Key words: Childhood OSA, Obesity, adenotonsillar hypertrophy

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INTRODUCTION

Sleep apnea forms a part of spectrum of sleep disordered breathing. It is defined as cessation of respiration for ten seconds or more OR cessation of respiration for two or more respiratory cycles (1). It can be Central, Obstructive or Mixed Apneas. Obstructive Sleep Apnea Syndrome (OSAS) is a sleep disorder characterized by recurrent episodes of narrowing or collapse of pharyngeal airway during sleep despite ongoing breathing efforts. Normally during sleep, the muscles which control the tongue and soft palate hold the airway open. If these muscles relax, the airway narrows, causing snoring and breathing difficulties. If these muscles relax too much or if obstruction is present, the airway can become completely blocked, preventing breathing. These often lead to acute derangements in blood gas disturbances, surges of sympathetic activation and periodic arousal from sleep causing fragmented sleep.

In recent years, the epidemic of obesity that is affecting the pediatric population all over the world has led to the emergence of a phenotypic variant of OSA in children that closely resembles that of adults with the disease. Dayyat et al. proposed that two types of OSA disease exist, namely one associated with marked lymphadenoid hypertrophy in the absence of obesity (type I) and the other being primarily associated with obesity in the presence of only mild lymphadenoid hyperplasia (type II) (2).

Epidemiology:

Though it occurs in all age groups from newborn to adolescents, the prevalence of sleep-disordered breathing in pre-school and school age children varies considerably from 3.2% to 27% for snoring (in most studies, 1/3 of the children aged 2-6 years have been claimed to snore occasionally and 10-14% to snore frequently) and from 0.5% to 3% due to OSAS (3, 4). Although only few studies have been conducted in India, yet the incidence was found to be higher than western studies (5). Seasonal variation is seen which might be due to variation in incidence of upper and lower respiratory problems (e.g. chronic rhinitis and asthma).OSA is more common in African-American & Asian children due to anatomic features of upper airway (6).

Pathogenesis:

Upper airway obstruction can be partial causing snoring or complete leading to OSA. In normal children, arterial oxygen decreases and CO2 increases only slightly during sleep. Episodes of partial or complete airway obstruction result in impaired gas exchange with hypoxemia and hypercarbia which stimulates arterial chemoreceptors leading to arousal or partial awakening. The impaired gas exchange in conjunction with decreased airflow is a potent stimulus for increased ventilatory effort and upper airway muscle activity. Arousal is associated with increased respiratory efforts and airway muscle tone which leads to resumption of airway patency. Arousals may appear in the form of movements, increased muscle

tone or changes in EEG. Following the arousal, airflow is restored, blood gases are normalised and sleep resumes but the cycle of airway collapse starts again.

The most common functional process contributing to obstructive sleep apnea or hypoapnea is REM sleep, present in one-fourth of a typical night sleep. Apnea frequency, apnea duration and levels of hypoxemia are almost always more severe during REM sleep due to lack of "wakefulness" drive, decreased tone of pharyngeal muscles, intercostal and accessory muscle and depressed reflexes, minute volume and response to hypoxia.

Enlarged tonsils and adenoids are one of the common risk factors for OSA. Although one cause of OSA in children is adenotonsillar hypertrophy, yet tonsil size does not correlate with findings on sleep studies (7). One child with large tonsils may be without symptoms while another with modest tonsil enlargement may have significant symptoms. Increased resistance from swollen nasal turbinates or choanal stenosis places a greater negative collapsing pressure on the pharyngeal airway and may lead to worsening obstruction. Other anatomic factors such as micrognathia, retrognathia or macroglossia may force the tongue into the oropharyngeal portion of the airway and cause airway occlusion. Congenitally small midface or nasopharnyx also narrows the airway.

Diminished arousal responses can also impair the ability to restore upper airway patency. Children with CNS abnormality associated with impaired ventilator or arousal responses to hypoxemia, hypercapnia and/or airflow obstruction (e.g.Chiari II malformations) have increased vulnerability to severe OSAS and cardiorespiratory failure. Fat deposition from morbid obesity also narrows the airway. Sedative medicine or general anaesthesia can further compromise neural control of the upper airway.

Adenotonsillar hypertrophy, as a risk factor is now being replaced by obesity as a substantial cause of OSAS in children (8-13). For every increment in body mass index (BMI) of 1 kg/m^2 beyond the mean BMI for age and gender, the risk of OSAS increased by 12%. In obese, upper airway narrowing results from fatty infiltration of upper airway structures promoting pharyngeal collapsibility. Obesity reduces the intrathoracic volume and diaphragmatic descent during inspiration, particularly in the supine position, resulting in lower oxygen reserves and increased work of breathing during sleep. Obesity also results in blunted ventilatory responses to hypoxia and hypercapnea. Leptin, an adipocyte-derived hormone level appears to be determined by the degree of obesity. Leptin affect overall ventilatory drive, and influence peripheral chemoreceptor activity. Hypoxia induces an increase in both leptin gene expression and plasma leptin levels. Obesity is associated with peripheral and central leptin resistance. Thus, reduced bioavailability of leptin resulting in altered ventilatory responses also plays a role in the interaction between obesity and OSAS.

Sleep-disordered breathing (SDB) seems more common in boys (due to sex differences in airway structure and control of breathing) and African American children. Another putative risk factor is recurrent otitis media which is most likely related to chronic adenotonsillar hypertrophy. Disorders of the upper and lower respiratory system, including asthma and persistent wheezing are also risk factors. Environmental tobacco smoke exposure and maternal smoking during pregnancy not only exacerbate these other respiratory disorders but also have been shown to result in higher rates of snoring and likely SDB.

Symptoms (14-20):

Typically, loud snoring is the symptom that most disturbs and therefore alerts the parents. Though OSAS by definition is characterised by obstructive apneas terminated by arousals, OSAS in children present as partial continuous obstructive hypoventilation with fewer discrete obstructive apneas, fewer arousals and less disturbances of sleep architecture. For this reason day time hypersomnolence is much less frequent in children than adults. Children may sleep in unusual positions to help maintain a patent upper airway, e.g. with the neck hyperextended or prone with the bottom up in air. Thus the child presents with snoring, breathing pauses, choking or gasping arousal, restless sleep, nocturnal diaphoresis and enuresis at times.

Most children with OSA breathe normally while awake and have minimal day time symptoms. Daytime hypersomnolence results from sleep fragmentation that occurs when OSA is repeatedly terminated by arousals. So the child presents with morning headaches, excessive daytime sleepiness (EDS), dry mouth, chronic mouth breathing, poor appetite and failure to thrive. However, it is difficult to recognize EDS in young children who normally have daytime naps and early bedtimes. Most children do not present with daytime sleepiness and are more likely to be hyperactive or inattentive, often being diagnosed with Attention Deficit Hyperactivity Disorder.

Further, a child may have memory deficits and mood disturbances manifesting as poor school performance which is increasingly recognized as being associated with OSA. Lower academic performance has been described in young children as well as in adolescents who suffer from OSAS or from primary snoring. There is a debate as to whether the neurocognitive deficits are the results of poor night sleep secondary to frequent arousals (and hence inability to concentrate in school) or a result of spending every night in relative hypoxemia that could be predisposing the child to cerebral ischemia.

Signs (14-20):

The physical examination performed during wakefulness may be entirely normal and cannot be used to exclude OSAS when the clinical history suggests otherwise. Polycythemia and respiratory acidosis with metabolic

alkalosis support the diagnosis of OSAS when present but are absent in majority of patients. Systemic hypertension may occur in advanced cases. In the past cor pulmonale with heart failure was a common mode of presentation for OSAS in children, but is rare now. Although overt heart failure occurs now less often, yet asymptomatic degrees of pulmonary hypertension may be common.

As stated by Dayyat et al Type I OSA is associated with hyperactive behaviour, enlarged tonsils/adenoids and recurrent ear infections contrary to Type II OSA which shows excessive sleepiness, truncal obesity, enlarged neck circumference, depression, low esteem, social withdrawal, left ventricular hypertrophy, systemic hypertension, insulin resistance, dyslipidemia and elevated C-reactive protein (2).

Diagnosis:

When snoring is associated with nocturnal breathing difficulties and witnessed respiratory pauses, this triad of symptoms is highly suggestive of OSA in children. An approach to a case of snoring is depicted in **Figure 1**

Total number of episodes of apneas and hypopneas averaged per hour of sleep is regarded as apnoea/hypopnoea index.

AHI < 5 : No OSA
AHI 5-15 : Mild OSA
AHI 15-30 : Moderate OSA
AHI > 30 : Severe OSA

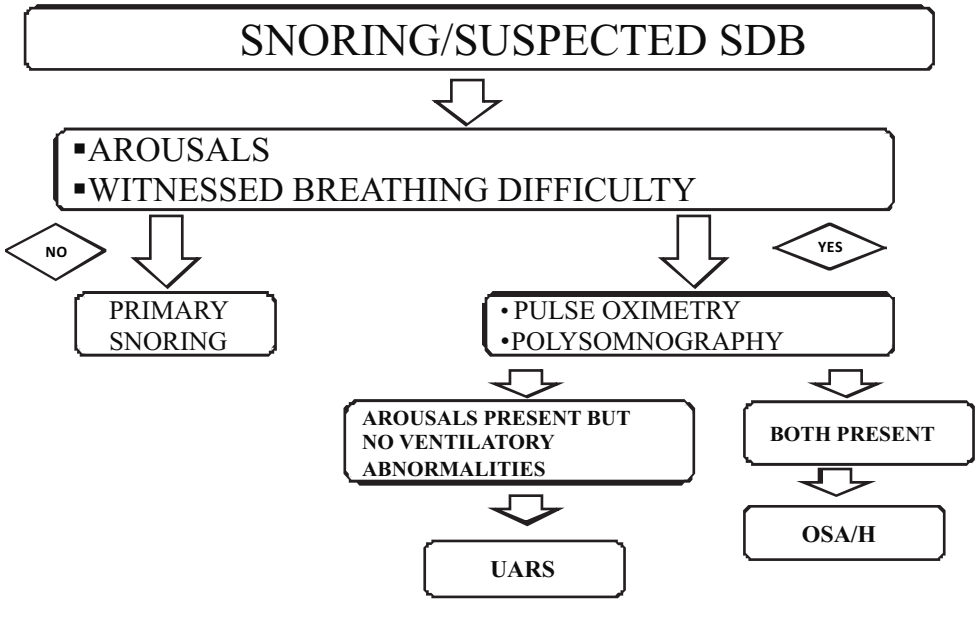

FIGURE 1. Diagnostic approach to a case of snoring

Polysomnography (PSG), an overnight recording of multiple physiologic sensors during sleep, is considered the gold standard for the diagnosis of OSA (20, 21). It is the only diagnostic technique shown to quantitate the ventilatory and sleep abnormalities associated with sleep disordered breathing. Variables that may be determined include:

1. EEG and EOG (for sleep state); EMG
2. Airflow at nose or mouth
3. End-tidal CO_2
4. Chest and abdominal motion (Impedance plethysmography)
5. ECG
6. Blood Pressure
7. Pulse oximetry
8. Esophageal Pressure (Intrapleural pressure)
9. Autonomic nervous system activity (Finger tonometer)

PSG can be performed satisfactorily in children of any age, provided that appropriate equipment and trained staff are available. Limitations of PSG are that it is a cumbersome, expensive, resource-intensive and inconvenient with limited accessibility and is an unreliable predictor of physical or psychological impairment in children with SDB.

As per International Classification of Sleep Disorders Edition 2 diagnostic criteria (1) for the childhood OSA is as follows:

Diagnosis = A + B + D or C + D

A. At least one of the following applies:
   i. Complaints of unintentional sleep episodes during wakefulness, daytime sleepiness, unrefreshing sleep, fatigue, or insomnia.
   ii. Awakenings with breath-holding, gasping, or choking.
   iii. Bed partner reports loud snoring and/or breathing interruptions during the patient's sleep.

B. Polysomnography shows the following:
   i. Scoreable respiratory events (apneas+ hypopneas + respiratory effort–related arousal RERAs)/hr of sleep ≥5/hr.
   ii. Evidence of respiratory effort during all or a portion of each respiratory event (in the case of RERAs, respiratory effort is best detected by esophageal manometry).

C. Polysomnography shows the following:
   i. Scoreable respiratory events (apneas+ hypopneas + RERAs)/hr of sleep ≥15/hr.
   ii. Evidence of respiratory effort during all or a portion of each respiratory event.

D. The disorder is not better explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder.

Overnight oximetry may be useful if it shows the pattern of cyclic

desaturation. Brouillette et al performed oximetry in a group of children with suspected OSAS and compared it with simultaneous full polysomnography (patients with complex medical conditions were excluded) (22). Compared with polysomnography, they found that oximetry was useful when results were positive. However patients with negative oximetry required full PSG for definitive diagnosis. False positive results were found in patients with mild coexistent medical problems, such as obesity and asthma, suggesting that this technique is useful only in otherwise healthy children. Other investigations that need to be done are:

Lateral neck radiograph: To evaluate the size of the adenoids.

Complete blood count: To detect polycythemia suggestive of chronic hypoxemia.

ECG & ECHO: For evidence of Cor Pulmonale or Right Ventricular Hypertrophy.

Treatment :

Lifestyle modifications include positional therapy (attaching a firm object, such as a tennis ball, to the back of a sleep garment to prevent the child from sleeping in supine position) and weight loss.

Previously, nonsurgical treatment, including the use of either intranasal or oral steroids, was used largely as a bridge to surgery. Medications are now are being considered as a viable, routinely used treatment option for those with mild SDB or snoring. However, pharmacologic management has only a limited role in paediatric OSAS patients. Treatment of nasal obstruction with topical nasal steroids can reduce snoring and OSAS severity in some children.

Steroids and antibiotics may be a useful adjunct in the acute management of infected pharyngeal tissues that have compromised upper airway patency. Nasal decongestants help in treatment of allergic rhinitis. Earlier beneficial effects of systemic corticosteroids on OSA were not proven but now a substantial decrease in the frequency of apnea and hypopnea events among children treated with fluticasone have been observed however no improvement in parents' symptom scores or reduction in tonsillar and adenoidal size is seen (23, 24).

Oral appliances: Continuous Positive Airway Pressure (CPAP) is useful as a treatment option. Though most common treatment in adults, medical management with nasal CPAP can be used in children also. It is indicated for patients with specific surgical contraindications, minimal adenotonsillar tissue, or persistent OSA after adenotonsillectomy or for those who prefer non-surgical alternatives. CPAP is delivered using an electronic device that delivers constant air pressure via nasal mask, leading to mechanical stunting of the airway and improved functional residual capacity in the lungs.

Among the surgical options available adenotonsillectomy is the most

common therapy for OSAS in children with adenotonsillar hypertrophy. When adenotonsillar hypertrophy is present, the majority of otherwise healthy children without major risk factors experience resolution or significant improvement after adenotonsillectomy. However, children with underlying problems, such as trisomy 21, craniofacial disorders, extreme obesity, or neuromuscular disorders, or who present before 2 yrs of age are at risk for incomplete resolution of OSAS even after adenotonsillectomy (25). Other surgical options used are as follows:

1. Uvulopalatopharyngoplasty
2. Tracheostomy
3. Mandibular distraction

Tracheostomy is treatment of choice if severe upper airway obstruction is present in both wakefulness & sleep (particularly when vocal cord dysfunction, impaired swallowing, or absent laryngeal protective reflexes exists). It may be necessary for severe OSA complicated by cor pulmonale when CPAP is unsuccessful or not tolerated. Alternative is mandibular distraction osteogenesis/maxillomandibular reconstruction surgery. The management approach to a case of sleep disorder breathing is depicted in **Figure 2**.

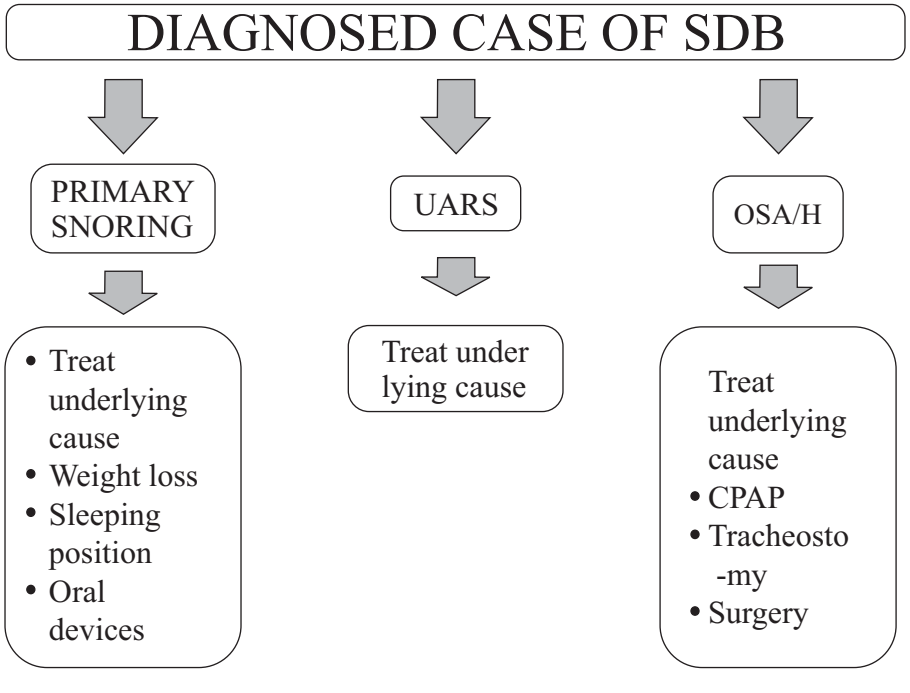

FIGURE 2. Management of case of Sleep Disorder Breathing

Conclusion:

Childhood OSA is on the rising trend in our country. Significant association is now being observed with obesity. Adenotonsillar hypertrophy remains the most important cause of this problem. It is vital to take a good history and conduct a thorough clinical examination of such children. Although polysomnography is the gold standard for making a definitive diagnosis yet in those centres where this facility is lacking, clinical evidence supported by basic investigations helps in making a diagnosis. Medical treatment should always be attempted before resorting to surgical treatment.

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