SPECIAL ISSUE 2: INVITED ARTICLE

Pediatric Obstructive Sleep Apnea

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Case Description

An 8-year-old girl brought by her parents to the pediatrician for snoring, restless sleep (tossing turning in bed in disarray), choking episodes at times and excessive sweating at night. Patient was under treatment for allergic rhinitis and wheezing. In the last one year, the growth chart is showing faltering, and her school grades had dipped. Of late, there have been complaints from the class teacher for her inattentiveness, and she has stopped participating in extracurricular [sports and social] activities as, she gets extremely fatigued by evening. On examination, BMI 15.35 kg/m^2 and blood pressure 118/88 mm Hg were recorded.

Dentofacial features: Adenoid facies, long face, narrow chin, mandible in the back position, nasal congestion, high arched palate, maxillary crowding and posterior cross bite. Oral examination showed enlarged tonsils and pale swollen inferior turbinates. PSG showed an AHI of 10/hr and lowest SPO_2–85%.

Sleep-disordered breathing (SDB) in paediatrics is a very prevalent disorder with serious repercussions, representing a spectrum of disorders from primary snoring to frank obstructive sleep apnea (OSA). Pediatic OSA is defined as "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,"[1] accompanied by symptoms or signs, as listed in Table 1. SDB can happen even immediately post birth and anytime in pediatric age group. However, maximum prevalence is seen in 1–8 years, the period of active lymphoid tissue growth. The overall incidence is around 2–4%.[2] Prevalence rates based on level I and II studies range from 1.2 to 5.7%.[3]

Pathophysiology

Pediatric OSA is a dynamic process. Thus, adult OSA features cannot be extrapolated on them due to the changing physiology and anatomy of a child. OSA in children happens due to primarily intrinsic obstruction in airway or due to increased airway collapsibility leading to a sustained inflammatory state in body (Fig. 1). Long-standing OSA may be associated with hypertension, cardiac dysfunction, and systemic inflammation.

Risk factors established as yet are adenotonsillar hypertrophy, obesity, craniofacial anomalies, neuromuscular disorders and premature birth. Adenotonsillar hypertrophy is currently the most common example of the former. Magnetic resonance imaging (MRI) studies have shown that the size of the adenoids and tonsils in children with OSA is significantly increased compared with healthy controls.[4] Other anatomical features resulting in upper airway narrowing such as micrognathia, macroglossia, and midface hypoplasia, are usually associated with craniofacial syndromes (e.g., Treacher Collins syndrome, Crouzon syndrome, Apert syndrome, Pierre Robin sequence), achondroplasia, trisomy 21 (Down syndrome), Beckwith Wiedemann syndrome, and mucopolysaccharidoses.[5] Obesity is yet another very important predisposing factor in the pediatric and peripubertal age group. Interestingly, increase in BMI by 1 kg/m^2 increases the risk by 12%. The factors implicated in obesity as a risk factor are pharyngeal fat deposition; decreased thoracic volume and diaphragmatic

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Table 1: Symptomatology in pediatric OSA[7]

Diurnal symptoms	*Nocturnal symptoms*
Headaches on awakening	Frequent snoring (≥3 nights/week)
Attention-deficit/ hyperactivity disorder	Labored breathing during sleep
Learning problems	Gasps/snorting noises/episodes of apnea
Behavioral problems	Sleep enuresis (especially secondary enuresis)
Daytime sleepiness	Sleeping in a seated position or with the neck hyperextended
Failure to thrive	
Poor scholastic performance	

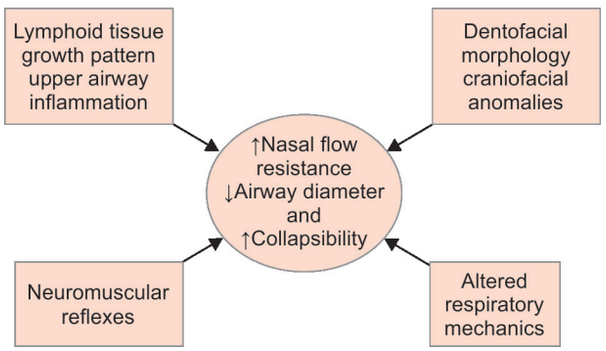

Fig. 1: Genesis of OSA in children

excursion secondary to visceral and abdominal fat deposition and blunted cortical ventilator drive. There is an increased incidence of recurrences of OSA in these patients as well as a high risk to develop postoperative complications.[6] Increased airway collapsibility as in asthma, allergic rhinitis and neuromuscular disorders also predisposes children to develop OSA and thus should be addressed with appropriate treatment as soon as possible.

Clinical Presentation

Pediatric OSA has peculiar features which are different from adult OSA (Table 1). In children the complaints are raised by the parents in comparison to the bed partners in adults. In daytime, neurocognitive impairment is much more pronounced with behavioral problems, failure to thrive, learning problems than daytime sleepiness. As the child grows older, daytime somnolence is seen more commonly especially if child is obese. In nighttime, enuresis is a feature restricted to pediatric OSA. Symptomatology in pediatric age group encompasses significant non respiratory complaints too. Normal sleep architecture is maintained thus patients do not experience excessive daytime somnolence. Also, movement arousals secondary to restless sleep is more common in the pediatric age group. Physical examination shows an underweight or overweight child with tonsillar hypertrophy, adenoidal facies, mid facial deficiencies, micrognathia, retrognathia, high-arched palate, dentopalatal aberrations, hypotonia and craniofacial anomalies.

In the syndromic phenotype, Down syndrome needs a special mention as they are prone to develop OSA for a variety of reasons—small upper airway, midfacial hypoplasia, micrognathia, relative macroglossia, lingual tonsil, decreased immunity and hypotonia; all these contribute to increase collapsibility of upper airway.[8] Hence, a PSG is a must in all Down's patient before 4 years of age with suggestive symptoms.

Diagnosis

When the symptomatology and signs point toward OSA, it is prudent to refer the child to a sleep consultant and otolaryngologist. The investigative modalities used in the study of sleep-disordered breathing are many but the gold standard is overnight attended PSG.[9] Diagnostic criteria need a clinical background to suspect and a PSG finding to confirm OSA (Fig. 2). AHI value is used for diagnosis and staging of pediatric OSA, where in mild OSA is defined as AHI of 1–5 per hour, moderate OSA is AHI of 5–10 per hour and severe OSA is AHI of more than 10 per hour. Lower cut-off in AHI criteria in pediatric age group in comparison to adults is due to the low functional residual capacity, relatively faster respiratory rate, low muscle mass and easy fatigability of the diaphragm.[10] Sleep questionnaire, video and audiotaping sleep and nocturnal oximetry are good screening tool in a resource restricted set-up. They are economical and easy to perform, but are only helpful if positive; as they do not record REM sleep, the stage of obstructive events, they may definitely underestimate the presence of SDB. Polysomnography features of pediatric OSA show a slight different trend than that in adults (Table 2). In pediatric age group, predominant obstructive apneas are seen which are REM phase phenomenon. Obese children with OSA behave like adult OSA patients.

In our case, the young girl has typical features of pediatric OSA affecting her scholastic performance and overall development. PSG demonstrates moderate OSA. Thus, this a classical case of pediatric OSA.

Criteria A and B must be met

A. The presence of 1 or more of the following:
1. Snoring
2. Labored/paradoxical/obstructed breathing during sleep
3. Sleepiness/hyperactivity/behavioral/learning problems

B. PSG demonstrates one or both of the following:
4. One or more obstructive/mixed apneas/hypopneas/ hour of sleep
5. A pattern of obstructive hypoventillation (Atleast 25% of TST with PCO_2 >50 mm Hg) in association with One or more of the following:
   —Snoring
   —Flattening of inspiratory nasal pressure waveforms
   —Paradoxicalthoracoabdominal motion

Fig. 2: Diagnostic criteria for pediatric OSA[9]

Consequences

Like in adults, OSA in pediatric age group also brings in debilitating consequences with the systemic inflammation all over. Long-standing pediatric OSA is associated with hypertension, cardiac dysfunction, metabolic complications and neurobehavioral compromises as depicted in Figure 3. These consequences are not that commonly seen in children as in adults.

Treatment

Definitive treatment is considered in children with moderate and severe grade of OSA. Mild OSA needs close monitoring with supportive care. Mainstay of treatment as explained above stays surgical intervention where indicated. Weight loss should always be encouraged in all children and even in postoperative phase. PAP therapy and oral appliances are helpful but compliance is a major concern. Compliance to PAP therapy can be improved by educating mothers and introducing the mask in a playful way to child. Family members can contribute by fulfilling the part of play therapist. Drug treatment which includes a trial of intranasal corticosteroids (mometasone or fluticasone or budesonide) and oral leukotriene receptor antagonist montelukast for a period of at least 3 months to reduce mucosal inflammation and adenotonsillar hypertrophy. Nutritional and physical rehabilitation is very important in growing children. Myofunctional therapy a physiotherapy of the orofacial and oropharyngeal muscles as an adjuvant to other therapies is recommended. It has role in lip and tongue posture which favors normal nasomaxillary unit development.[11] Behavioral therapy in selected patients should be considered. Sleep hygiene is equally important in this age group as well like in adults.

Surgery

With the understanding and plethora of literature to support, surgical intervention is the first line of treatment in those with evident adenotonsillar hypertrophy as a contrast to adult OSA where surgical intervention comes in picture once PAP therapy fails due to any reason. Adenotonsillectomy is the most commonly performed surgery followed by craniofacial surgeries and tracheostomy. Importantly, postoperative airway and anesthesia complications are a possibility in children with OSA because they already have a blunted ventilatory response to hypoxia and hypercarbia. Adenotonsillectomy is a common surgery done in children but needs to be carefully conducted in children with OSA. A subset of pediatric OSA is more prone to develop postoperative

Table 2: Childhood versus adult obstructive sleep apnea syndrome

Parameters	*Pediatric OSA*	*Adult OSA*
Age	Affects all ages, peaks at 1–8 years	Elderly age group
Gender	Male = Female (till puberty) Postpubertal, males have slight preponderance than females	Male > female (till menopause, then equal prevalence noted)
Risk factors	• *<1 year:* Craniofacial anomalies, syndromes, neurological disorders • *1–8 years:* Adenotonsillar hypertrophy • *>8 years:* Obesity	Obesity, craniofacial abnormalities, etc
Neurocognitive effects	ADHD, impaired concentration, performance, low IQ	Daytime somnolence, impaired vigilance and memory
Oral examination	Adenotonsillar hyperhertrphy	Crowded oropharynx
PSG (13) Apnoea Definition	Cessation of airflow (drop in the Peak signal excursion by ≥90%) for at least two respiratory cycles	Cessation of airflow (drop in the Peak signal excursion by ≥90%) ≥10 seconds
Obstruction	Persistent partial upper airway obstruction (hypoventilation)	Frank and cyclic partial or complete upper airway obstruction
Transcutaneous CO2/ End tidal CO2	Rise of PaCO2	In selected Cases, may be raised
Cortical Arousals	Low frequency of arousals secondary to respiratory events (20% of obstructive apnea are followed by cortical arousal)	Apnoeic episodes are followed by arousal
Sleep architecture	Normal sleep architecture	Fragmented sleep and decrease in sleep efficiency
State of OSA	Obstructive apnea and hypopneas are a REM related phenomena	Usually occur in both REM and NREM sleep
Severity of OSA	• *Mild:* 1–5 events /hour • *Moderate:* 5–10 events/hour • *Severe:* >10 events/hour	• *Mild:* 5–15 events/hour • *Moderate:* 15–30 events/hour • *Severe:* >30 events/hour
Surgical intervention	If adenotonsillar hypertrophy noted, surgical excision is mainstay in management	Surgical intervention should be opted for a very small subset where standard PAP therapy is not feasible

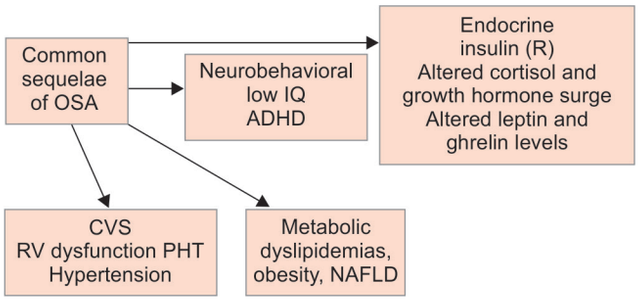

Fig. 3: Consequences in Pediatric OSA

complications like negative pressure pulmonary edema especially in those with severe grade, less than 3 years of age, premature, obese or failure to thrive, down syndrome, craniofacial anomalies, cardiac involvement (right ventricular dysfunction/pulmonary hypertension), chronic lung disease, neuromuscular disorder, sickle cell disease. These children are more prone to the depressive effects of sedatives and opioids used in induction and pain management during and following adenotonsillectomy.[12] Craniofacial surgeries are restricted to children with syndromic craniofacial anomalies. Tracheostomy is the most definitive intervention in obstructive OSA but rarely performed as it projects the child to a poorer quality of life.

Follow-up PSG

These corrective interventions can significantly reduce the grade of OSA but may not lead to a definitive cure. Thus, a repeat PSG is recommended postoperatively in these children to establish residual OSA. Generally, PSG is recommended 6 weeks after adenotonsillectomy, after 12 weeks of montelukast/nasal steroid treatment, 12 months after rapid maxillary expansion and after 6 months with orthodontic appliances. Children on CPAP or BPAP should be reevaluated at least every 12 months after initial titration.[13,14]

Pearls of Wisdom

- Pediatric OSA is a common disorder often underdiagnosed.
- Risk factors should be identified early.
- Symptomatology in pediatric age group is different than adults.
- Diagnosis relies upon suggestive symptoms and confirmatory supervised laboratory PSG like in adults.
- Adenotonsillectomy is the first line of treatment in children who are surgical candidates.
- PAP therapy is equally important but needs innovative ways to improve compliance with children.
- There is a definitive role of orofacial myofunctional therapy in nonobstructive sleep apnea: Focus on myofunctional therapy in obese pediatric OSA.

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