Postal Registration No. TN/CH/(C/59/2020 - 2022
Registered News Paper Posted at Egmore R.MS. Patirika Channel.
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RNI NUMBER : TNENG/2004/14197
RDISNo. 6342/98

Medi Quest BRS Hospital

A monthly News letter from BRS Hospital

OBSTRUCTIVE SLEEP APNOEA SYNDROME (OSAS) IN PEDIATRIC PATIENTS

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March - 2021

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OSAS is a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction with intermittent or complete obstructive apnoea that disrupts normal ventilation during sleep.

The types of Sleep Related Breathing Disorders (SBD) include Primary Snoring, Upper airway resistance syndrome, OSAS and Central Apnoea.

TERMINOLOGY:

- Apnoea : cessation of airflow for 10 sec
- Apnoea Index : number of apnoea episodes in 1 hour .
- Hypopnoea : reduction in airflow to 50% of Tidal volume associated with arousal as noted on EEG or drop in oxygen saturation.
- Apnoea-Hyponoea Index (AHI) : Number of Apnoea & Hypopnoea episodes in an hour.

Table 1: OSA severity by PSG defined by American Society of Anesthesiologists

OSA severity	AHI children	AHI adults
None	0	0-5
Mild	1-5	6-20
Moderate	6-10	21-40
Severe	>10	>40

PATHOPHYSIOLOGY :

Upper airway patency is determined by the interaction between respiratory dynamics, anatomic structures, and neuromotor tone; the degree of patency is determined by the balance between forces acting to collapse the airway (e.g. negative pressure during inspiration; size, shape, and floppiness of pharyngeal structures) and forces acting to maintain airway patency (e.g. pharyngeal dilator muscle tone, stiffness of pharyngeal structures

During the day, the increased activity of pharyngeal dilator muscles, holds airway open and prevents collapse. But during sleep, the pharyngeal muscle tone decreases and during REM sleep muscle tone further reduces leading to airway obstruction. Paediatric OSA is often a multifactorial disorder with overlapping contributions from airway narrowing , abnormal airway muscle tone, and genetics predisposing children to obstructed breathing during sleep. OSA is relieved by arousal from sleep.

Hypoxia stimulates carotid body causing arousal from sleep. Repeated episodes of arousal leads to sleep fragmentation leading to stress. Apnoea leads to hypoxia and hypercapnia, which in turn increases sympathetic output and acidosis .This causes peripheral vasoconstriction and systemic and pulmonary hypertension and cor pulmonale. Chronic cases develop left

ventricular hypertrophy. The increase in inflammatory marker C reactive protein triggers endothelial dysfunction and cardiovascular manifestations as well as metabolic and neurocognitive manifestations.

RISK FACTORS FOR OSA IN CHILDREN:

Table 1. Causes of obstructive sleep apnoea

Condition		Frequency of sleep-disordered breathing[2]
Uncomplicated causes	Enlarged tonsils Enlarged adenoids	
Complicated causes	Obesity	60%
	Craniofacial abnormalities — narrowing of upper airway • Cleft palate • Apert syndrome • Treacher Collins syndrome • Crouzon syndrome • Retrognathia (abnormal jaw positioning)	Up to 100% (depending on severity)
	Down's syndrome	70–100%
	Neuromuscular disease — decreased tone in upper airway • Cerebral palsy	42%
	Achondroplasia	42%
	Prader–Willi	25–75%
	Sickle cell disease	10–41%

OBESITY LEADING TO OSA:

In Obesity , increased tissue mass around neck and pharynx causes increased airway resistance. The respiratory effort thereby increases causing negative pressure and airway collapse. The Chubby Puffer Syndrome includes obesity, airway obstruction and somnolence in children.

DIAGNOSIS:

OSA should be suspected when nocturnal symptoms of snoring, gasping, increased work of breathing or paradoxical breathing, restless sleep, witnessed apnoeas , mouth breathing or nocturnal eneuresis are reported. Some children with OSA will also sleep with their neck in a hyperextended position to maintain their airway. Daytime symptoms of OSA are very non-specific, but together with nighttime symptoms may help alert clinicians to clinically significant obstructive sleep disordered breathing: hyperactivity, difficulty concentrating/learning difficulties, behavioural difficulties, excessive daytime sleepiness, and moodiness.

DIAGNOSTIC TESTS:

1. POLYSOMNOGRAPHY-

It is the Gold standard for diagnosis and quantitative description of OSA. The parameters assessed are – EEG, submental EMG, EOG, Oxygen saturation, ECG holter monitoring, Nasal Airflow, Chest/ abdominal movement, ET CO2, Snoring Sound recording, Sleep position detector.

Obstructive events during sleep in children with OSA have an inverse pattern in relation to sleep stage in comparison with that seen in adults with OSA. 80% of obstructive events in children with OSA occur during rapid eye movement (REM) sleep, whereas 80% of obstructive events in adults occur during non-REM sleep . Supine Body position is associated with obstruction compared to lateral or prone position.

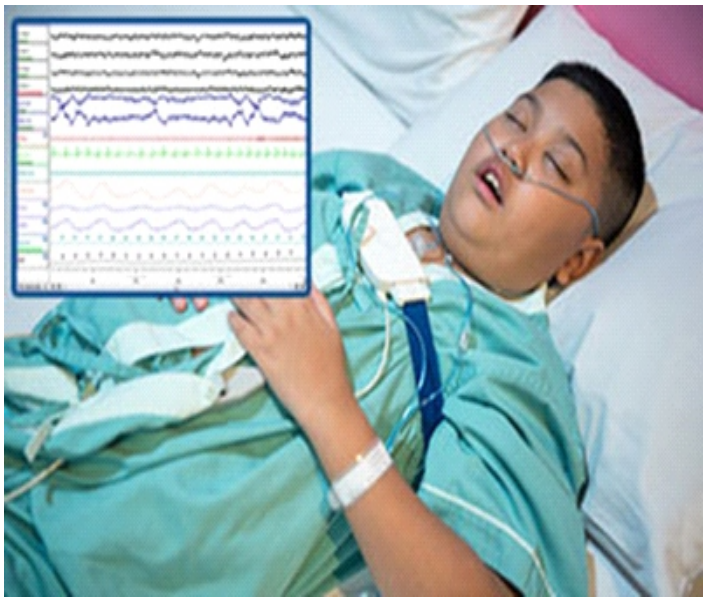

Fig 1: Child undergoing Polysomnography study

2. DISE-

Emination of Dynamic airway collapse can be done by Drug Induced Sleep endoscopy (DISE) wherein the patient is sedated with a drug such as dexmeditomidine (sedative effect mimics NREM sleep) and flexible fibreoptic endoscopy is done to determine the the levels of obstruction .

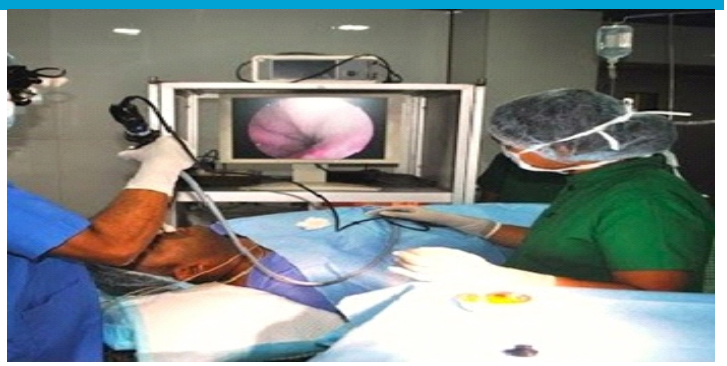

Fig 2: Diagnostic Sleep Endoscopic Study

DYNAMIC MRI:

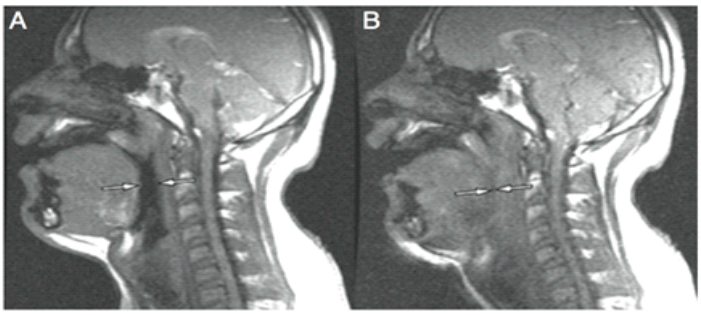

Fig 3: Dynamic MRI on inspiration the retroglossal airway is patent , on expiration glossoptosis and airway obstruction.

MANAGEMENT OF OSA IN CHILDREN:

CONSERVATIVE MANAGEMENT:

Continuous positive airway pressure (CPAP) or Bilevel positive airway pressure (BiPAP). CPAP pneumatically splints the upper airway and prevents collapse . The sleep study determines the positive pressure required to optimize airflow. The pressure is increased until the obstructive events , snoring, oxygen desaturation are minimized.

Fig 4: Continuous Positive airway pressure (CPAP)

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MEDICAL MANAGEMENT:

Children with OSA who present with mucopurulent discharge should be treated with antibiotics and intranasal steroidal spray. Obese children are advised to lose weight by diet and exercise.

SURGICAL TREATMENT:

The airway obstruction causing the OSA can be found at any of the 4 levels - Table 2: Level of obstruction Classification System: This helps to plan necessary surgical procedure and address areas of collapse.

Level	Site of Obstruction	Cause	Treatment
1	Velopharyngeal	Adenoids	Adenoidectomy
2	Tonsillar	Chronic Tonsillitis	Tonsillectomy
3	Tongue Base	Macroglossia	Nasopharyngeal airway, glossopexy, mandibular advancement splint, CPAP
4	Supraglottic	Laryngomalacia	Supraglottoplasty / Epiglottopexy

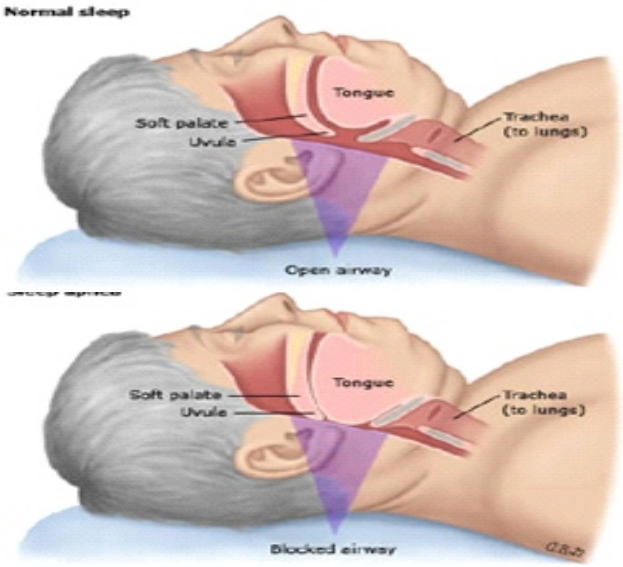

Fig 5: Levels of Airway collapse in OSAS

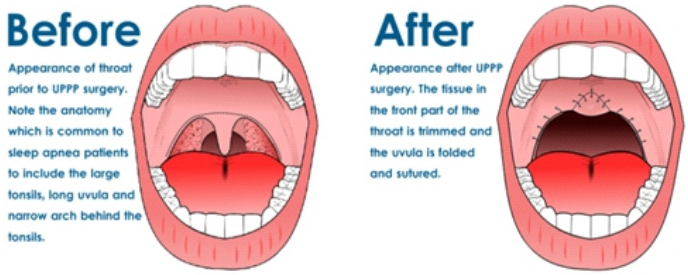

Fig 6: UPPP surgery for OSA

CONCLUSION :

Practitioners should be vigilant for paediatric OSA as it is common and may affect upto 1 in 30 children. OSA in children may present with behavioural problems and poor attention, which parents may not necessarily link to a sleep disorder. It is therefore important that the GP elicits a sleep history. Obesity is a risk factor and is thought to be responsible for rising levels of paediatric OSA. Any child with a history of snoring and any of the daytime or night-time symptoms of OSA should be referred; with treatment the prognosis is excellent.

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Owned and Published by Dr. Madhusudhan 28, Cathedral Garden Road,Chennai - 34.
Printed by S. Baktha at Dhevi Suganth Printers 52, Jani Batcha Lane, Royapettah, Chennai -14.
Publication on : Final Week of Every Month Posted on 29.03.2021