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Short Communication

Childhood Obstructed Sleep Apnoea and the Role of the ENT Surgeon

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Obstructed Sleep apnoea (OSA) is characterized by oxygen desaturation and reduced upper airway airflow despite preserved thoracic and abdominal effort. It occurs due to periodic partial collapse of the upper airway during sleep. It is typically associated with the elderly, especially males. The incidence of childhood OSA is much less (1 - 4%) [1] as compared to adults (20%) [2]. Yet the effect of prolonged poor sleep and inadequate oxygenation can have several deleterious effects on the child's health. OSA has been reported to cause poor feeding and failure to thrive, developmental delay, hyperactivity, learning difficulties, personality changes and depression in children. It has also been reported to result in hypertension and cardiac issues in the long term.

The causes of OSA in children are different than in adults. For children over 2 years - adenoid and tonsillar hypertrophy is the commonest cause, followed by craniofacial anomalies associated with hypoplastic maxilla or mandible and large tongue, neuromuscular disorders and obesity [3]. Other reported risk factors are a history of prematurity or multiple pregnancies, environmental exposure to smoking, asthma and allergic rhinitis and gross septal deformities [4]. In infants and very young children (< 2 years) laryngomalacia, unilateral choanal atresia, cleft palate and syndromes associated with craniofacial or neck abnormalities are the main causes.

Diagnosis is based on careful evaluation of history, clinical examination and eventually endoscopic and instrumental assessment. History and clinical examination have been reported to have positive predictive value for diagnosis of OSA of 65% and 46%, respectively [5]. OSA maybe suspected in a child if there is history of habitual snoring (> 3 days a week), restless sleep or apnoea episodes observed by caregivers, sleeping with mouth open, daytime somnolence or bed wetting [3].

The ENT specialist plays an important role in evaluating the cause of obstruction. A careful examination of the position and shape of the jaw bones, position of palate, the size of tonsils (graded from 0 (surgically removed) to 4 (in midline, kissing tonsils)) and the Mallampati score, to assess oropharyngeal narrowing, form part of the initial examination. A flexible nasopharyngolaryngoscopy is usually performed to look for evidence of nasal block, size of adenoids in relation to the choana and any pharyngeal or laryngeal abnormality. Neck circumference, BMI and BP should also be recorded. It is important to rule out pulmonary hypertension. The ENT specialist may perform a DISE (drug induced Sleep Endoscopy) to assess in detail the level/s of collapse during sleep. The role of DISE in the paediatric population is still controversial and it is not routinely performed. Boudewyns., *et al*. have found it useful to identify patients most likely to benefit from surgery [6].

Polysomnography is the gold standard for diagnosing OSA but the criteria are different for children than for adults. In children, the detection of a single apnoea episode or hypopneas per hour is considered pathological. OSA severity are identified according to the AHI (Apnoea Hypopnea Index): mild AHI 1 - 4, moderate AHI 5 - 9, severe AHI ≥ 10. The present polysomnographic classification also allows to identify children: 1) at risk of sequelae; 2) at risk of postoperative complications, which require strict clinical and instrumental follow-up; 3) at high risk of OSA even after adenotonsillectomy, requiring further investigations and treatments [3]. Polysomnography is an expensive test, not readily available and needs admission and overnight stay, at least for 1 night. Nocturnal pulse oximetry is a valid initial diagnostic test for SBD and OSA because of its high positive predictive value (97%), its easy applicability and low cost. It represents a good screening tool [7].

Management depends on treating the underlying cause of the condition and the ENT surgeon is involved in 3 main areas:

1. Treating the adenotonsillar hypertrophy. Surgery is the standard, best and the most effective option with more than 80% success rate [8]. It is found to be effective even in mild OSA cases. However, in these cases, especially in older, non-obese children, a wait and watch policy or trial of steroid nasal spray maybe considered. Needless to say, careful selection of patients and a complete resection of the adenoids under endoscopic vision is recommended. In very rare instances, adenoid regrowth is known to occur but not more than grade 1 and it does not cause nasal obstruction [9].
2. Though nasal obstruction is not a cause for OSA, it leads to increased negative inspiratory pressure downstream and thus contribute to the pharyngeal collapse. Turbinate hypertrophy and deviated nasal septum are two of the commonest obstructive nasal diseases. Turbinate hypertrophy is usually due to allergy and responds to medical management and steroid nasal spray. However, in refractory cases partial turbinectomy can be performed under general anaesthesia, with good results. Nasal septal defects are ideally not addressed surgically till adolescence so as to not affect bone growth. In severe septal deviation, limited endoscopic correction may be helpful without affecting future skeletal development. Unilateral choanal atresia, if present, is repaired endoscopically through the nose, again by the ENT surgeon.
3. Tracheostomy - may be needed for those OSAS children, secondary to craniofacial abnormalities, for whom the planned corrective surgical procedures carry a high mortality until the child is older. It is very effective but does carry higher risk in children than in adults.

For children who cannot be treated surgically, CPAP is an effective option but with low compliance rate. Unlike adults, CPAP is not the first line of treatment of childhood OSA and is reserved for select cases [10]. Other measures like treating obesity, allergies, oropharyngeal exercises [11] and avoiding risk factors are to be incorporated in the effective management of the OSA child.

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