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Tailoring therapy to improve the treatment of children with obstructive sleep apnea according to grade of adenotonsillar hypertrophy

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a common disease in children with the major causes of hypertrophy of adenoid or tonsil and nasal diseases. The treatment methods for this disease include the resection of adenoid or tonsil, and drug therapy as well. However, no agreement on the selection of treatment method is available to date.

Objective: To investigate the individualized treatment methods for children with OSA with different sizes of adenoids and tonsils.

Methods: Children with OSA (diagnosed by polysomnography) were included into groups A (adenoid/tonsil grade ≤III) and B (adenoid/tonsil grade = IV), and further subdivided into subgroups A1 (3-month medication), A2 (3-month medication and negative-pressure sputum aspiration [NPSA]), B1 (3-month medication plus NPSA), B2 (coblation adenotonsillectomy with preoperative/postoperative medication for 3 days/2 weeks) and B3 (coblation adenotonsillectomy with preoperative/postoperative medication for 2 weeks/3 months). Six-month outcomes included quality of life for children with obstructive sleep apnea-18 item (OSA-18), obstructive apnea index (OAI), apnea hypopnea index (AHI) and lowest oxygen saturation ($LSaO_2$).

Results: Three hundred and eighty six patients (310 male; 6.70 ± 2.44 years-old) were included. Preoperative OSA-18, OAI, AHI and $LSaO_2$ were not significantly different. At all postoperative time points, subgroup A2 had significantly lower OSA-18 than subgroup A1; postoperative improvements in OAI, AHI and $LSaO_2$ were also superior in subgroup A2 ($P < 0.05$). The initial decrease in OSA-18 was not maintained in subgroups B1 and B2, whereas subgroup B3 showed a sustained reduction at 6 months. OAI and AHI were more improved in subgroup B3 ($P < 0.05$). Surgical/anesthetic complications in subgroups B2 and B3 were 5.5% and 0%.

Conclusion: Conservative therapy could achieve satisfactory outcomes in children with grade III hypertrophy, while surgery and drugs could achieve good outcomes in grade IV.

1. Introduction

Obstructive sleep apnea (OSA) also known as obstructive sleep apnea hypopnea syndrome is a common pediatric disease (prevalence of 1–3%) most commonly caused by adenotonsillar hypertrophy [1–3]. OSA is characterized by reduced oro-nasal airflow and oxygen desaturation, and is clinically manifested by snoring, mouth breathing, periods of apnea, restless sleep, urinary incontinence, inattentiveness, daytime hyperactivity, mood swings and failure to thrive [3]. Since OSA is associated with neurobehavioral deficits and serious complications (including growth retardation, cardiac dysfunction, conductive deafness and craniofacial malformations [4,5]), timely diagnosis and treatment are important [6–8].

Adenoidectomy and tonsillectomy are the primary treatments for OSA [8–12], although symptoms in some children improve little or even recur [13]. Continuous positive airway pressure (CPAP) is an option [14], but it is reported that patients have a low compliance to this therapy [15]. Although drug therapy is beneficial [16], surgical intervention is still required in some patients. Despite the various options available, the management of children with OSA still lacks an international set of standardized criteria.

Previous studies showed that adenoidal hypertrophy, tonsillar hypertrophy, sinusitis and inflammatory strictures in the nasal

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cavity are important risk factors for OSA in children [4,17]. Tonsil and adenoid sizes are positively related to OSA risk and severity [18,19]. Tonsils and adenoids of grades III and IV can obstruct the airways and be a primary cause of OSA [4]. Therefore, tonsil and adenoid size should be considered when selecting treatment options. Additionally, hypopnea and apnea occur repeatedly in children with OSA, especially in those with moderate or severe disease. This often produces a hypoxic and hypercapnic state that causes dysfunction of vital organs, including the heart, lung, liver and kidney [4,20], and reduces the sensitivity of the respiratory center to elevated carbon dioxide concentrations, maintaining the central respiratory center excitability by stimuli from carotid and aortic body chemoreceptors. In this situation, removal of the upper airway obstruction by surgery can cause a sudden rise in oxygen partial pressure and respiratory depression [21–23], increasing the surgical and anesthetic risks. Adequate preoperative preparation can reduce these risks. Therefore, it is important to adopt an individualized and tailored therapeutic approach for children with OSA, based on the tonsil and adenoid grades and disease severity.

The aims of the present study were to retrospectively assess pediatric patients with OSA seen at our otolaryngology department over a 3-year period, and to explore the efficacy of individualized treatments tailored to the grade of their hypertrophy.

2. Materials and methods

2.1. Patients

This was a retrospective study of pediatric patients diagnosed with OSA and treated at the Department of Otolaryngology, Children's Hospital of Fuzhou (Fujian Province, China) between June 2008 and April 2011. The study was approved by the Ethics Committee of the Children's Hospital of Fuzhou. Individual consent was waived by the committee.

Inclusion criteria were: (1) patient aged 1–13 years, pre-pubescent, and had consistent living conditions; (2) patient had been monitored by polysomnography (PSG) and complied with relevant criteria [4]; (3) patient had been examined using a tongue depressor or video laryngoscopy, and assessed for body weight (obesity) and total IgE; (4) OSA was considered to be caused by rhinosinusitis, adenoid hypertrophy and/or tonsillar hypertrophy; and (5) patient had been screened using the obstructive sleep apnea-18-item (OSA-18) questionnaire [24,25], administered by a specialist. Children who had unbalanced grade of tonsillar and adenoidal hypertrophy (one was grade III and the other grade IV) were excluded because they could not be classified into one of the groups for the study. Children with serious anatomic abnormalities of the mouth, nose or pharynx, or a history of massive trauma or any other chronic disease affecting the heart, lung, liver, kidney or brain were also excluded.

2.2. Diagnosis of OSA

OSA was diagnosed by sleep monitoring using the Alice 5 Sleep Diagnostic System (Philips Healthcare, Best, The Netherlands), Alice PDx Portable Sleep Diagnostic System (Philips Healthcare) and Jaeger PSG (Jaeger Ausbau GMBH & Co Kg, Würzburg, Germany), according to published criteria [26]. The time of obstructive apnea index (OAI) >1/h or the apnea hypopnea index (AHI) >5/h in every night's sleep was considered as abnormal; hypopnea was defined as the peak signal of the oral and nasal air current decreased by 50% and the oxygen saturation decreased by >0.03 and/or wake up. The duration of respiratory events was defined as ≥2 respiratory cycles [26].

Tonsillar hypertrophy was diagnosed by visual inspection, using a tongue depressor: tonsil size was graded I to IV; a tonsil size of grade III or IV with the presence of clinical symptoms was considered as tonsillar hypertrophy [27]. Adenoidal hypertrophy was diagnosed using a Pentax VNL-1530T video laryngoscope (Pentax, Tokyo, Japan): adenoid size was graded I to IV; an adenoid size of grade III or IV with the presence of clinical symptoms was considered as adenoidal hypertrophy [28]. The diagnosis of rhinitis and rhinosinusitis were made on the basis of published criteria [29,30].

2.3. Grouping and therapy

2.3.1. Grouping

Patients were included into two groups according to their original diagnosis: group A, adenoid and tonsil grade ≤III; and group B, adenoid and tonsil grade IV. Then, according to the original treatment that was undertaken, Group A was further subdivided into subgroups A1 (drug therapy for 3 months) and A2 (drug therapy plus negative-pressure sputum aspiration [NPSA] for 3 months). Group B was subdivided into subgroups according to original treatments: B1 (drug therapy plus NPSA for 3 months), B2 (coblation adenotonsillectomy after 3 days of drug therapy, followed by postoperative drug therapy for 2 weeks) and B3 (coblation adenotonsillectomy after 2 weeks of drug therapy, followed by postoperative drug therapy for 3 months). In our clinical practice, children ≤3 years old with adenoid and tonsil grade IV according to Chinese guidelines are considered a high risk population for surgery, and do not undergo surgery; therefore, they were all included in group B1, alongside other children whose parents or guardians had decided against surgery [4,29].

2.3.2. Therapy

Local drug therapy, administered for 3 months, consisted of nasal inhalation of 1 mL budesonide suspension in children <3 years old [31], and mometasone furoate nasal spray in children ≥3 years old [32,33]. Systemic drug therapies included oral antibiotics (for 2 weeks), Sinupret drops (for 4 weeks; a mixture of herbal products; elder (*Sambucus nigra*, Caprifoliaceae) flowers, primrose (*Primula veris*, Primulaceae) flowers with calyx, common sorrel (*Rumex acetosa*, Polygonaceae) herb, European vervain (*Verbena officinalis*, Verbenaceae) herb, and gentian (*Gentiana lutea*, Gentianaceae) root) and montelukast sodium (for 3 months) [34]. The drug dosages were as recommended by the manufacturers.

NPSA was given as an assisted therapy prior to budesonide or mometasone. Aspiration frequency was adjusted to between 1/day to 2/week. For aspiration, the patient was reclined in the supine position with the head tilted backwards. Three drops of 0.5% ephedrine were instilled into both nostrils. After nasal mucosal engorgement had subsided and a no. 6 suction tube was inserted into the nasopharynx via the nasal cavity, secretions were aspirated (aspirator pressure, 200 mmHg), alternating between nasal cavities.

Surgical therapies included adenotonsillectomy under a low-temperature plasma system. General anesthesia was achieved via endotracheal intubation. The whole tonsil was resected from its lower pole along the capsule, using an Evac 70 radio-frequency knife (Arthrocare Corporation, Austin, TX, USA). The adenoid was endoscopically resected layer by layer from the posterior wall to the ceiling of the nasopharynx, until the bilateral choanae and tori were fully exposed.

2.4. Follow-up and evaluation indexes

Efficacy was assessed using the obstructive apnea index (OAI), apnea hypopnea index (AHI) and the lowest oxygen saturation ($LSaO_2$) [4] measured by PSG before and 3 months after treatment. OSA-18 [24,25] was assessed before treatment, and at 1 week, 2 weeks, 1 month, 2 months, 3 months and 6 months after treatment. OSA-18 scores ranged from 18 to 126 points, 18–36 being regarded as normal, 37–59 as mild, 60–80 as moderate, and 81–126 as