AM Li 李民瞻
S Hui 許漢琳
E Wong 黃銘聰
A Cheung 張育美
TF Fok 霍泰輝

Obstructive sleep apnoea in children with adenotonsillar hypertrophy: prospective study

扁桃腺肥大的兒童中的阻礙性睡眠呼吸窒息：預期研究

Objective. To determine clinical and baseline polysomnographic data on obstructive sleep apnoea secondary to adenotonsillar hypertrophy in Hong Kong Chinese children.
Design. Prospective study.
Setting. University teaching hospital, Hong Kong
Participants. Fifty children (35 boys and 15 girls) suspected to have obstructive sleep apnoea were recruited between January 1999 and December 1999.
Main outcome measures. Symptoms questionnaire, electrocardiogram, chest radiograph, and full-night polysomnography.
Results. All patients had symptoms suggestive of obstructive sleep apnoea. None were found to have clinical evidence of cor pulmonale. Forty-five (90%) of 50 children had obstructive sleep apnoea with a respiratory disturbance index of greater than five. Central and mixed apnoeas were rare. Tonsil size did not correlate with the severity of obstructive sleep apnoea.
Conclusion. Symptoms and signs suggestive of obstructive sleep apnoea can lead to a high detection rate and confirmation of obstructive sleep apnoea by polysomnography.

目的：確定香港華人兒童中，因扁桃腺肥大而繼發的阻礙性睡眠呼吸窒息的臨床和多功能睡眠圖像的基數資料。
設計：預期研究。
安排：香港，大學教學醫院。
參與者：在 1999 年 1 月至 12 月期間，被懷疑患有阻礙性睡眠呼吸窒息的 50 名兒童(包括 35 名男童及 15 名女童)。
主要結果測量：症狀問卷；心電圖；胸部放射性照片；整夜的多功能睡眠圖像。
結果：所有病人都有阻礙性睡眠呼吸窒息的症狀，但並未發現病人有臨床心肺病症。50 名兒童中 45 名(90%)的呼吸擾動指數大於 5，並患有阻礙性睡眠呼吸窒息。中樞及混合型呼吸窒息狀況屬罕見。扁桃腺的大小與阻礙性睡眠呼吸窒息的嚴重程度無關。
結論：透過多功能睡眠圖像顯示的阻礙性睡眠呼吸窒息的症狀和徵兆，更能偵察並確認病人是否患了阻礙性睡眠呼吸窒息。

Key words:
Polysomnography;
Sleep apnea;
Tonsil

關鍵詞：
多功能睡眠圖像；
睡眠呼吸窒息；
扁桃腺

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The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong:
Department of Paediatrics
AM Li, MRCP, MRCPCH
S Hui, MA, FHKAM (Paediatrics)
A Cheung, BSc
TF Fok, MD, FHKAM (Paediatrics)
Centre for Clinical Trials and Epidemiological Research
E Wong, MA

Correspondence to: Dr AM Li

Introduction

Obstructive sleep apnoea (OSA) in children is a frequently underdiagnosed condition. Studies have revealed an estimated prevalence of OSA in 1% to 3% of the paediatric population.[1,2] Many aetiological factors have been proposed in childhood OSA, including craniofacial anomalies, Down's

syndrome, obesity, neuromuscular disease, and laryngomalacia. Adenotonsillar hypertrophy remains the most common cause of OSA in children. In fact, the association between OSA and adenotonsillar hypertrophy has been well documented in studies conducted in western countries.[3-5] There is relatively little local data available, however. Since there may be geographic and racial differences, the application of overseas data to our population seems unacceptable. To better understand the problem locally, we studied a group of children with suspected OSA secondary to adenotonsillar hypertrophy who presented to the Paediatric Chest Clinic for assessment. The clinical presentation, polysomnography (PSG) findings, and complications were evaluated.

Methods

Patients referred to the Paediatric Chest Clinic with suspected OSA secondary to adenotonsillar hypertrophy were prospectively recruited. Children with a known syndromic diagnosis, chronic lung disease, or neurological or craniofacial abnormalities were excluded. The children in the study underwent physical examination and overnight PSG after informed consent had been obtained from the parents.

The clinical history of each child was obtained from the parents. The following information related to OSA was obtained using a standardised questionnaire: (1) night-time symptoms including loudness and frequency of snoring, difficulty in breathing, observed apnoea during sleep, necessity for parents to shake the child to breathe, restless sleep, varying sleep posture, enuresis, and night sweating; (2) daytime symptoms including excessive daytime sleepiness, behavioural problems, morning headache, and mouth breathing. The loudness of snoring was assessed as being just audible, clearly heard in the same room, clearly heard outside the room, and clearly heard in another room. The snoring frequency ranged from never, rarely, one to four times a month, more than once a week, to every night. Other symptoms were assessed as being present or absent.

Box. Five-point scale of tonsil size

0 No enlargement
1 Slight enlargement with the transverse air slit more than half the diameter of the palatal airway
2 Moderate enlargement with the air slit about half the palatal airway diameter
3 Profound enlargement with the air slit less than half the airway diameter
4 Total obstruction with tonsils touching each other at the midline

A complete physical examination was performed for each child to assess the physical status—in particular, growth parameters (height and weight), tonsil size, and signs of pulmonary hypertension or cor pulmonale. Tonsil size was graded using a five-point scale (Box). Chest radiograph and electrocardiogram were performed for each child for evidence of cor pulmonale.

An overnight PSG was performed in a dedicated room in the presence of the parent and a technician. We employed a 16-channel recorder (Oxford Medilog MPA-S, Oxford Instruments Ltd, Oxford, UK). In each PSG study, the stage of sleep was assessed by electroencephalogram, bilateral electro-oculogram, and submental electromyogram. Chest wall and abdominal movements were measured by respiratory impedance plethysmography, airflow through the nose was measured with a thermister, oxygen saturation was measured by pulse oximetry, and body motions were recorded by a body position sensor.

Sleep stage was scored by trained personnel and followed the standard criteria.[6] An obstructive event was defined as cessation of airflow with increase in irregular respiratory and abdominal movement for more than two consecutive breaths. Central apnoea was defined as cessation of breathing with no respiratory effort for more than 10 seconds. Apnoeic episodes with features of both obstructive and central events were categorised as mixed apnoeas. Hypopnoea was defined as more than 50% reduction in themistor signal associated with arousal, and/or desaturation of more than 4% or sustained values of less than 90%. Respiratory disturbance index (RDI) was defined as the number of apnoeic and hypopnoeic episodes per hour of total sleep. The number of episodes of oxygen desaturation by more than 4% from baseline per hour were also noted (oxygen desaturation index [ODI]).

Statistical analysis

The RDI and ODI formed skewed distributions and were presented as median values with interquartile range. The demographic data were presented as mean values with standard deviation. Spearman's rank correlation was used to assess the association between tonsil size and severity of OSA.

Results

Fifty children (35 boys and 15 girls) with a mean age of 6.45 years (standard deviation, 2.7 years) were included in the study. Their demographic data are

Table 1. Patients' demographic data

	Data* (n=50)
Age (years)	6.5 (2.7)
Sex	
male	35 (70%)
female	15 (30%)
Weight (kg)	21.7 (3.4)
Height (cm)	111.7 (4.2)
Ideal weight for height (%)	102.4 (8.5)

* Data are expressed as the mean (SD) except where indicated

shown in Table 1. None were considered to be obese (more than the median/ideal weight for height x 120%), but two children weighed below the third centile. The presenting symptoms are shown in the Fig. Forty-four (88%) children snored every night and the snoring could be heard in the same room for all the children. Other common presenting night-time symptoms included struggling to breathe during sleep (74%), restless sleep (74%), increased night sweats (58%), and sleeping with the neck extended or in the prone position (54%). Twelve (24%) children had apnoeic episodes during sleep but only four (8%) required stimulation to make them breathe again. Four (8%) children had secondary enuresis. The most common daytime symptom was mouth breathing (86%). Daytime somnolence was a feature in only 6 (12%) children.

All children had tonsillar hypertrophy on clinical inspection, with 43 (86%) children having a tonsil size of grade 2 or above. None had features of pulmonary hypertension or cor pulmonale on physical examination, chest radiograph, or electrocardiogram. One (2%) child had pectus excavatum.

All patients slept well in the sleep laboratory with an average total sleep time of 9 hours and an average sleep efficiency of 85%. The overall sleep architecture was similar to the existing data from western countries. The median RDI was 13.7 (interquartile range, 8.03-21.43) and the median ODI was 6.45 (interquartile range, 2.15-16.88). Obstructive events accounted for most of the apnoeic and hypopnoeic episodes. Five (10%) children had a RDI of less than five. Thirty (60%) had a RDI of greater than 10 and the RDI was greater than 20 for 14 of these children. An RDI of between 5 and 10 was recorded for 15 children. Twenty (40%) children had an ODI of less than five. Nineteen (38%) had an ODI of greater than 10 and 11 of these children had an ODI of greater than 20. Eleven (22%) children had an ODI of between 5 and 10. The frequency distribution for RDI on tonsil size is shown in Table 2. Using Spearman's rank correlation analysis, no association was found between tonsil size and RDI.

Discussion

Obstructive sleep apnoea is caused by a collapse of the upper airway during sleep when muscle tone of

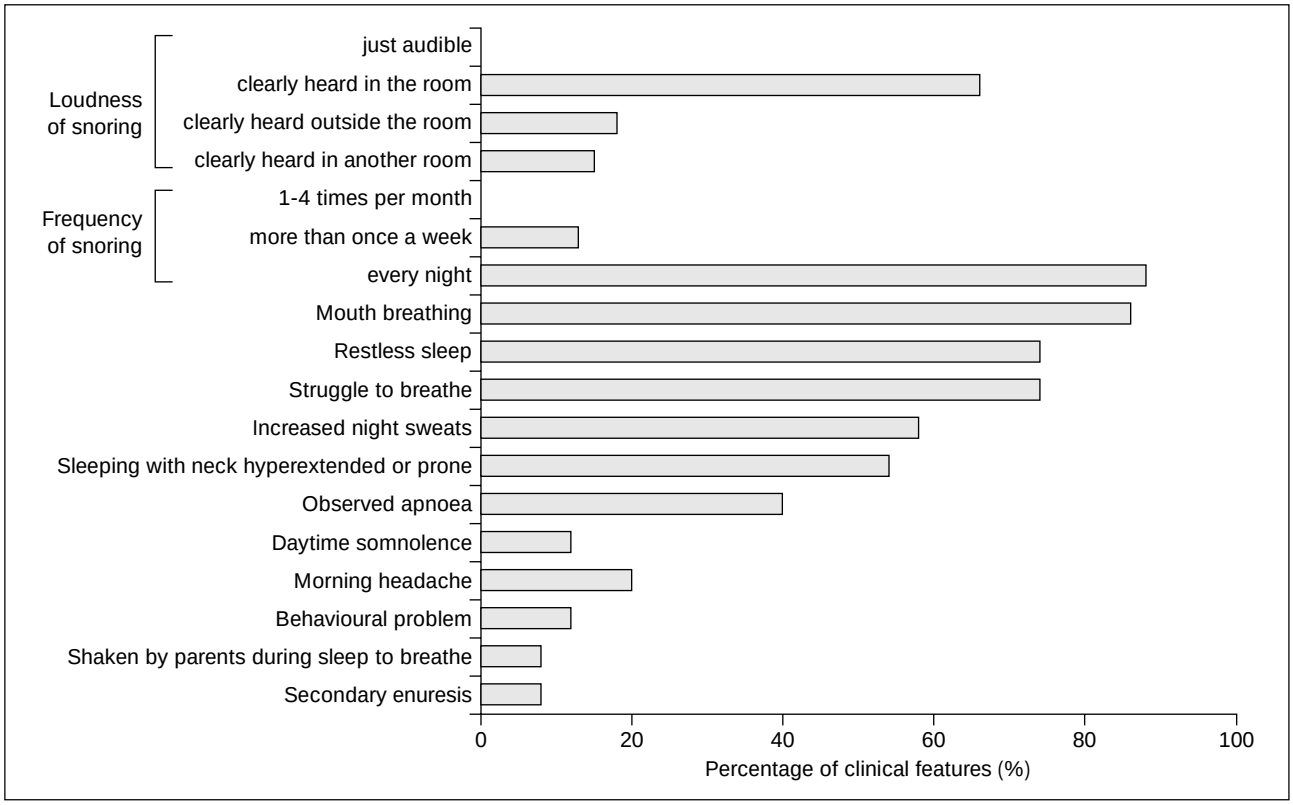

Fig. Frequency of presenting clinical features of obstructive sleep apnoea syndrome (n = 50)

Table 2. Frequency distribution for respiratory disturbance index on tonsil size*

	Respiratory disturbance index			
Tonsil size	≤5 No. (%)	6-10 No. (%)	11-20 No. (%)	>20 No. (%)
1+	2 (40)	3 (20)	2 (12.5)	
2+	1 (20)	5 (33)	5 (31.2)	5 (35.7)
3+	1 (20)	4 (27)	4 (25.0)	4 (28.6)
4+	1 (20)	3 (20)	5 (31.2)	5 (35.7)

* Linear by linear association Monte-Carlo estimate (100 000 tables sampled) of P>0.05

the upper respiratory tract decreases. It is thus prone to occur in patients with narrowing of the upper airway. In the paediatric population, the most common cause for this is adenotonsillar hypertrophy.

In Hong Kong Chinese children, the epidemiology of OSA secondary to adenotonsillar hypertrophy is unclear since it has rarely been reported. The epidemiology may be different from that in western countries with differences in facial morphology and race. A questionnaire survey for sleep problems among Hong Kong children found that the incidence of habitual snoring was 9.4%.[7] It had been suggested that loud snoring, difficulty in breathing, and apnoea observed during sleep were important symptoms pertaining to OSA.[8] Symptom scores for OSA had been derived although it was subsequently shown that they were poor predictors of the condition in children.[9]

We did not detect any association between tonsil size (observed on direct visual examination) and severity of OSA. This is, however, expected since the observed superior portion of the tonsil does not reflect its true size. Besides, the pathophysiology of OSA is considered to be multifactorial. Parameters of muscle tone, upper airway diameter, site and degree of obstruction, and sleep stage all seem to play a part.

The possible complications of OSA, if left untreated, include failure to thrive, cardiovascular impairment, cognitive dysfunction, and developmental delay. Sleep deprivation may manifest as daytime hypersomnolence in children, but this symptom is relatively less common than in adults.[10] Conversely, the child with OSA may exhibit hyperactivity in an attempt to keep them from falling asleep during the day.[11] Others may exhibit antisocial behaviour, poor attention spans, and delayed language acquisition.[12] Nightmares and sleepwalking, which have also been reported in association with OSA,[11] did not occur in the patients in this study. Nocturnal enuresis was reported in four of our patients. The exact underlying pathophysiology of this complication is still unknown but various factors have been proposed. They include increased secretion of atrial naturetic peptide,[13,14] higher fractional urinary flows with increased sodium and chloride excretion,[15] and a decrease in central inhibition.[16] Two of our patients weighed below the third centile. The corresponding RDIs were 12.0 and 14.6. Various factors have been proposed to explain the poor weight gain in patients with OSA. They include disturbed growth hormone release, low calorie intake, and increased energy expenditure during sleep. There is no evidence yet for any particular factor and it is most likely to be an interplay among them.

Pectus excavatum has been documented in childhood OSA. The association with disease severity has yet to be defined, however.[4] It is believed that the increased force of inspiration in upper airway obstruction during sleep can cause the sternum to buckle inwards during cartilage development. Only one of our patients had pectus excavatum and, on further questioning, it was found that the lesion had been there since birth.

This is the first report of paediatric OSA secondary to adenotonsillar hypertrophy in Hong Kong Chinese children. There is no doubt that when we begin to have a better understanding of the condition, more cases will be found. Currently, the gold standard for the diagnosis of OSA is overnight polysomnography. Patients presenting with symptoms and signs suggestive of OSA should be further investigated to exclude such a diagnosis.

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