A Case-Control Study of Obstructive Sleep Apnea-Hypopnea Syndrome in Obese and Nonobese Chinese Children*

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Background: **Obesity is a risk factor for obstructive sleep apnea-hypopnea syndrome (OSAHS) in adults. However, the prevalence of OSAHS in children is not clear, and the relationship between obesity and OSAHS remains controversial.**

Methods: **Obese children were recruited from the endocrinology, respiratory, and ear, nose, and throat clinics. Weight-matched, age-matched, and sex-matched children were recruited as control subjects. Standard questionnaires were administered, and a standardized physical examination was carried out. Lateral neck roentgenography, sleep polysomnography, full blood count, and arterial blood gas analysis were also performed. Children with body mass index z-scores of > 1.96 were considered to be obese. An adenoidal/nasopharygeal ratio of > 0.67 was considered to constitute adenotonsillar hypertrophy (ATH). OSAHS was defined as an apnea-hypopnea index (AHI) score of > 5 or obstructive apnea index (OAI) score of > 1.**

Results: **Ninety-nine obese children and 99 control subjects were recruited into the study. Obese patients had significantly higher AHI and OAI scores, and lower sleep efficiency and minimum arterial oxygen saturation ($MinSaO_2$) than control subjects. The prevalence of OSAHS was significantly higher in obese children with or without the ATH groups than their nonobese counterparts (odds ratio, 1.9 vs 108, respectively; 95% confidence interval, 1.21 to 4.7 vs 6.2 to 191, respectively). Obesity, tonsillar hypertrophy, and adenoid hypertrophy were independent risk factors for OSAHS ($p < 0.001$, $p = 0.042$, and $p = 0.004$, respectively). There was a positive correlation between the degree of obesity and AHI ($r = 0.535$; $p < 0.001$), and an inverse correlation between obesity and $MinSaO_2$ ($r = -0.507$; $p < 0.001$). End-tidal CO_2, $PaCO_2$, and bicarbonate levels were within the normal range.**

Conclusions: **Obesity is a risk factor for OSAHS, and the degree of obesity is positively correlated with the severity of OSAHS.** ***(CHEST 2008; 133:684–689)***

Key words: body mass index; children; obesity; obstructive sleep apnea; polysomnography

Abbreviations: AHI = apnea-hypopnea index; A/N = adenoidal-nasopharyngeal; AR = allergic rhinitis; ATH = adenotonsillar hypertrophy; BMI = body mass index; CAI = central apnea index; ENT = ear, nose, and throat; $ETCO_2$ = end-tidal carbon dioxide; IBW = ideal body weight; LgAHI = log-transformed apnea-hypopnea index; $MinSaO_2$ = minimum oxygen saturation; OAI = obstructive apnea index; OSAHS = obstructive sleep apnea-hypopnea syndrome; SDB = sleep-disordered breathing; WHtR = waist/height ratio

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a disorder characterized by narrowing of the pharyngeal airway, resulting in repeated episodes of airflow cessation, oxygen desaturation, and sleep disruption.[1] This can result in daytime sleepiness, morning headaches, and poor school performance, and longstanding OSAHS can lead to life-threatening cardiopulmonary problems such as pulmonary hypertension and cor pulmonale.

The prevalence of pediatric OSAHS is estimated to be between 5% and 6%.[2] Clinical experience has suggested that the risk factors, pathophysiology, clinical manifestations, diagnosis, and management of children with suspected OSAHS is different from that in adults.[3] The evidence for an association between obesity and OSAHS is well documented in adults.[4] Fewer studies are available in the pediatric population, and few have been done in China. Hence, obesity as a risk factor for OSAHS in children remains controversial.[5–8] As the prevalence of obesity is increasing worldwide in children,[9] and OSAHS may be associated with significant morbidity and

even mortality if undiagnosed, the relationship between obesity and OSAHS in children has become an important area of research. We hypothesized that obese Chinese children were more prone to have OSAHS than the normal population, and there was a positive correlation between the degree of obesity and the severity of OSAHS. In the current study, the prevalence of OSAHS in obese children was compared with control subjects of normal weight, and the relationship between severity of obesity and respiratory data was evaluated.

Materials and Methods

Subjects

Obese children who came to the endocrinology, respiratory, and ear, nose, and throat (ENT) clinics were recruited from April 2006 to January 2007. Those who had craniofacial anomalies, diabetes mellitus, chronic lung disease, and neuromuscular disease were excluded from the study. Normal weight-matched, age-matched, and sex-matched control children were also recruited from the ENT, ophthalmology, and dermatology departments at the same time. The institutional Ethics Committee approved the study and all parents gave written informed consent.

Clinical Data

Questionnaires and a standardized physical examination were carried out. Standard lateral neck roentgenography and polysomnography were performed. Height and weight were measured. Waist circumference was measured at the minimum circumference between the iliac crest and the rib cage. The waist/height ratio (WHtR) was calculated and was used to describe central obesity. The body mass index (BMI) was calculated as weight (in kilograms)/height (in square meters). In childhood, BMI changes with age and is influenced by gender. An absolute cutoff value to define obesity in children of different ages is therefore not feasible. We transformed an individual child's BMI value to a z-score based on the gender-specific and age-specific reference values for Chinese children.[10] Then, a BMI z-score of > 1.96 was considered to be obese.

The tonsil size was graded by direct visualization as follows: grade 0, not reaching tonsil pillars; grade 1, extending to the tonsil pillars; grade 2, enlarged beyond the pillars but not meeting the uvula; grade 3, meeting the uvula; and grade 4, touching in the midline. Tonsilar hypertrophy was defined as grade 3 or above. Adenoid size was determined by measuring the adenoidal/nasopharyngeal (A/N) ratio.[11] The A/N ratio was calculated as the ratio of adenoidal depths to the nasopharyngeal depths on lateral cephalometric radiographs (Fig 1). A full blood count was performed to exclude polycythemia. Polycythemia was defined as a hemoglobin level of > 16 g/dL. Arterial blood gas analysis, which included Pa_{O_2}, Pa_{CO_2}, and bicarbonate, was performed in the early morning.

Polysomnography

A standard overnight polysomnography was performed (Compumedics E-series; Compumedics Inc; Abbotsford, VIC, Australia), with the accompanying parent sleeping in the same room with the child. The monitoring duration was longer than 7 h for each subject. The following parameters were measured: four-channel EEG with bilateral central and occipital leads; electrooculograph; electromyograph with submental electrodes; ECG; airflow measurement by a nasal pressure cannula; and thoracic and abdominal piezoplethysmograph for respiratory effort measurement. Oxygen saturation in arterial blood was measured by pulse oximetry with a finger probe. From October 2006 to January 2007, end-tidal carbon dioxide (Et_{CO_2}) was monitored simultaneously with other parameters overnight for all obese subjects (Caponogard; Respironics Inc; Murrysville, PA).

Polysomnography was interpreted by a pediatrician trained in sleep medicine who was unaware of the clinical and radiographic findings. Sleep stages were assigned using the criteria of Rechtschaffen and Kales.[12] The term *sleep efficiency* refers to the percentage of the total recording time the patient spent asleep. An *obstructive apnea event* was defined as cessation of airflow with persistent respiratory and abdominal movement for more than two respiratory cycles. *Central apnea* was defined as the cessation of breathing with no respiratory effort for more than two respiratory cycles. *Hypopnea* was defined as a reduction in oronasal flow by $> 30\%$ associated with arousals and/or desaturations of $\geq 3\%$ for more than two respiratory cycles. *Apnea*

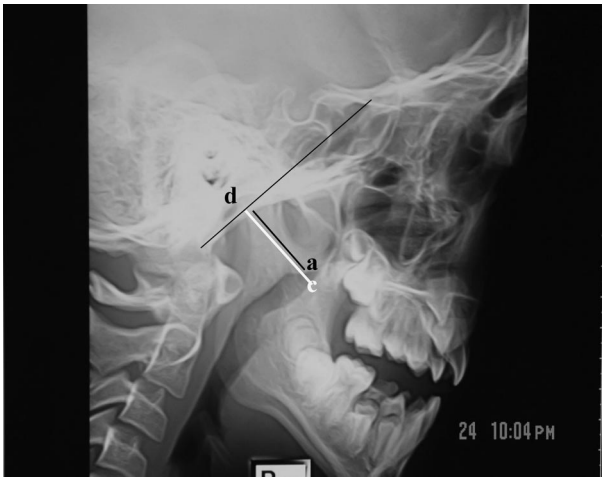

Figure 1. Measurement of the A/N ratio on lateral cephalometric radiographs. a = post prominent point of the adenoid; a-d = depth of the adenoid; c-d = diameter of the nasopharyngeal space. The A/N ratio was calculated as the ratio of adenoidal depths to the nasopharyngeal diameter.

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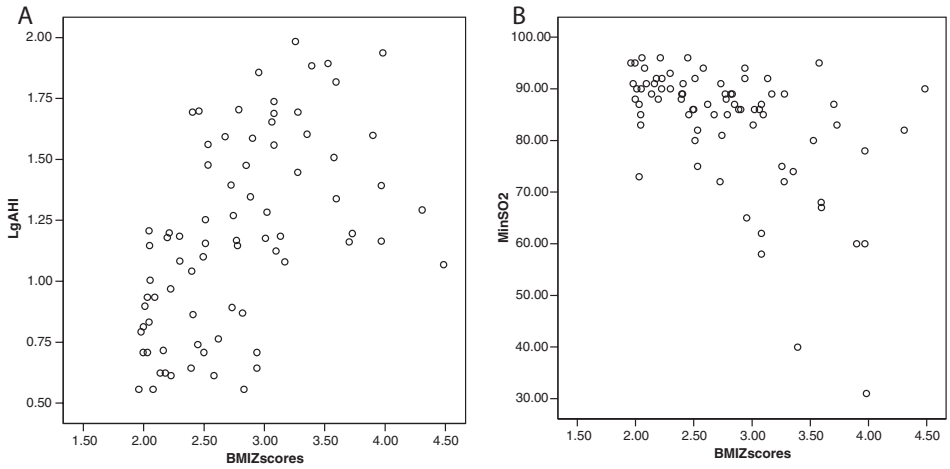

FIGURE 2. Relationships between obesity and OSAHS among obese children. *Left*, *A*: LgAHI positively related to BMI z-scores ($r = 0.535$; $p < 0.001$). *Right*, *B*: $MinSaO_2$ inversely related to BMI z-scores ($r = -0.507$; $p < 0.001$).

index was defined as the average number of apneas per hour of sleep. *Apnea-hypopnea index* (AHI) was defined as the average number of apneas plus hypopneas per hour of sleep. Sleep staging and respiratory events analysis were summarized by computer software (ProFusion 2; Compumedics Inc). The average and peak $ETCO_2$, the percentage of total sleep time with an $ETCO_2$ level of > 45 mm Hg and with an $ETCO_2$ level of > 50 mm Hg were analyzed also using the same software. OSAHS was defined as an AHI score of > 5 or an obstructive apnea index (OAI) score of > 1.

Continuous variables were reported as the mean $\pm$ SD. In univariate analyses, continuous data were compared by one-way analysis of variance and the Newman-Keuls test. AHI, OAI, and central apnea index (CAI) were log-transformed to correct for skewed distribution. The AHI was assigned as 0.05 if AHI was 0 when the data were log-transformed. The prevalence of obstructive sleep apnea was compared between obese children and control subjects by Fisher exact test. A general liner model was used for multivariate analysis to identify risk factors for OSAHS. The relationship between BMI z-score and the severity of OSAHS (*ie*, log-transformed AHI [LgAHI] and arterial oxygen saturation) was evaluated using the Pearson correlation test. All statistical analyses were performed with a statistical software package (SPSS, version 10.0; SPSS; Chicago, IL). All statistical tests were two-tailed with 0.05 as the threshold level of significance.

RESULTS

There were 124 obese children who met the inclusion criteria for our study. Seventeen parents refused the polysomnography offered to their children, and 8 patients were excluded because of incomplete clinical data such as, for example, neck circumference and A/N ratio. Therefore, 99 obese children and 99 control subjects of normal weight were finally recruited. All subjects were classified into the following four groups: obese only; obese plus adenotonsillar hypertrophy (ATH); normal weight only; and normal weight plus ATH (Table 1). Sleep efficiency, AHI, OAI, and minimum arterial oxygen saturation ($MinSaO_2$) were significantly different among the four groups. However, there was no difference with respect to the CAI among the groups.

Table 2 presents the relationship between the number of patients with obesity, ATH, and OSAHS. The risk of having OSAHS was significantly higher in obese children than in those with normal weight. By using a general linear model with gender, age, obesity, adenoid hypertrophy, tonsillar hypertrophy, allergic rhinitis (AR), and neck circumference as independent variables, and LgAHI as the dependent variable, OSAHS was determined to be significantly related to adenoid hypertrophy ($p = 0.004$), tonsil hypertrophy ($p = 0.042$), and obesity ($p < 0.001$); there were no relationship among age, sex, AR, neck circumference, and OSAHS (Table 3). For those OSAHS children who were obese, there was a positive correlation between BMI z-score and the AHI ($r = 0.535$; $p < 0.001$), and there was an inverse correlation between BMI z-score and the $MinSaO_2$ ($r = -0.507$; $p < 0.001$) [Fig 2]. A positive correlation was also found between the degree of central obesity measured by WHtR and the severity of OSAHS expressed by AHI and $MinSaO_2$ ($r = 0.341$; $p < 0.001$ vs $r = -0.345$; $p < 0.001$, respectively).

Of the 28 obese children who had $ETCO_2$ monitoring, 7 patients had $ETCO_2$ levels of > 45 mm Hg, and the longest duration of $ETCO_2$ of > 45 mm Hg occupied 10% of the total sleep time. The longest duration of the time spent at > 50 mm Hg occupied 2.5% of the total sleep time. The transient elevated CO_2 level was followed by increased ventilation and arousal. There was no persistent CO_2 retention documented (Table 4). $ETCO_2$ monitoring in our subjects was within the normal range according to

Table 1—*Demographic Data of Obese Children and Control Subjects of Normal Weight

Variables	Obese Only (n = 37)	Normal Weight Only (n = 37)	Obese + ATH (n = 62)	Normal Weight + ATH (n = 62)
Age, yr	10.3 ± 2.1	9.6 ± 2.4	8.6 ± 1.2	8.5 ± 1.4
Male/female ratio	2.4:1	2:1	3.1:1	2.6:1
BMI z-scores	2.90 ± 0.77	< 1.96	2.62 ± 0.59	< 1.96
Waist circumference,† cm	90.7 ± 11.7		84.4 ± 7.7	
Neck circumference, cm	33.4 ± 3.7	29.1 ± 2.8	32.3 ± 2.2	28.4 ± 1.6
WHtR†	0.61 ± 0.06		0.60 ± 0.05	
Symptoms				
Frequent snoring	31 (83.8)	0	59 (95.2)	61 (98.4)
Stop breathing during sleep	14 (37.8)	0	30 (48.4)	35 (56.5)
Daytime sleepiness	5 (13.5)	0	8 (12.9)	6 (9.7)
Recurrent sore throat	7 (18.9)	0	25 (40.35)	24 (38.7)
Polysomnographic data				
SE,‡ %	79.7 ± 7.4	83.1 ± 7.6	78.9 ± 8.0	86.5 ± 8.1
AHI‡§‖	16.4 ± 17.9 (1.1–86.5)	1.7 ± 1.0 (0.1–4.8)	21.9 ± 14.7 (1.5–96.3)	9.4 ± 9.4 (1.3–48.9)
OAI‡§‖	6.4 ± 7.9 (0–49.9)	0.4 ± 0.3 (0–0.9)	9.8 ± 8.1 (0–60.7)	4.1 ± 5.8 (0–23.4)
CAI	0.3 ± 0.3 (0–3.6)	0.3 ± 0.3 (0–1.4)	0.6 ± 0.6 (0–7.1)	0.4 ± 0.8 (0–2.5)
$MinSaO_2$,‡§ %	85.0 ± 9.3	93.4 ± 2.5	84.5 ± 7.3	88.3 ± 7.2

*Values are given as mean ±/SD, mean ± SD (range), or No. (%) unless otherwise indicated. SE = sleep efficiency.
†n = 59 in the Obese + ATH group.
‡Significant difference using Newman-Keuls test ($p < 0.05$; Obese + ATH group vs Normal Weight + ATH group).
§Significant difference using Newman-Keuls test ($p < 0.05$; Obese Only group vs Normal Weight only group).
‖Significant different using Newman-Keuls test ($p < 0.05$; Obese Only group vs Obese + ATH group).

American Thoracic Society criteria.[13] Arterial blood gas analysis in the early morning also showed normal PaO_2, $PaCO_2$, and bicarbonate levels. No signs of polycythemia were found in the full blood count measurement.

Discussion

In contrast to the adult population, the risk factors for childhood OSAHS are less well defined. Though obesity has been proven to be a risk factor for adult OSAHS, results conducted in the obese pediatric population have been conflicting.[5–8]

Marcus et al[14] reported that in a small study of 22 obese children, 46% had abnormal polysomnogram findings, and there was a positive correlation between the degree of obesity and the AHI. However, Sardon et al[8] reported no weight difference between OSAHS children and non-OSAHS children. Mallory et al[6] reported that in morbidly obese children the ideal body weight (IBW) was not associated with AHI and $MinSaO_2$. Conflicting results might be due to different patient groups and to the different criteria used for obesity and OSAHS in previous studies. AHI values of 3, 5, or 10 were used as indicators of OSAS in different studies, and both IBW and BMI were used to define obesity. Nevertheless, it must be pointed out that BMI in childhood changes substantially with age.[10,15] For instance, a BMI of 22 is normal for a 12-year-old child but will be considered obese for a 7-year-old child. Thus, defining obesity with a fixed cutoff point for a group of children of different ages is not possible. In the

Table 2—*Prevalence of OSAHS in Different Groups of Children

Variables	OSAHS†	OR (95% CI)	p Value‡
Obese only	22/37	108 (6.2–191)	< 0.001
Normal weight only	0/37		
Obese + ATH	55/62	1.9 (1.2–4.7)	0.001
Normal weight + ATH	40/62		

*OR = odds ratio; CI = confidence interval.
†Values are given as No. of patients with OSAHS/total No. of patients.
‡Fisher exact test.

Table 3—*Multivariate Analyses on Risk Factors for OSAHS in Children

Variables	Regression Coefficient	Adjusted OR	95% CI	p Value
Obesity	2.9	18.7	5.3–52.6	< 0.001
Adenoid hypertrophy	1.1	3.1	1.4–15.2	0.004
Tonsillar hypertrophy	1.7	5.2	1.3–28.2	0.042
Allergic rhinitis	1.5	4.6	0.8–26.6	0.089
Age	0.3	1.4	0.8–2.3	0.26
Gender	−0.2	0.8	0.5–2.7	0.52
Neck circumference	0.7	2.1	0.6–11.4	0.37

*General linear model. See Table 2 for abbreviations not used in the text.

Table 4—*$ETCO_2$ Findings (n = 28)

Variables	Mean (SD)	Range
Age, yr	10.2 (3.1)	4.9–15.3
Male:female ratio	2.1:1	
BMI z-scores	3.2 (0.7)	1.97–4.48
$ETCO_2$		
Average, mm Hg	38.4 (3.6)	32–45.1
> 45 mm Hg for duration of TST, %	1.3 (2.8)	0–10.3
> 50 mm Hg for duration of TST, %	0.2 (0.5)	0–2.5
Peak, mm Hg	42.8 (4.1)	35–51

*TST = total sleep time.

study by Redline et al,[5] obesity was defined as a BMI of > 28 kg/m^2 at any age, which might underestimate the rate of obesity in young children; while, in study by Wing et al,[7] all subjects were recruited from a clinic as a result of suspected OSAHS, which might overestimate the prevalence of sleep-disordered breathing (SDB). Furthermore, the most common cause of childhood obstructive sleep apnea is ATH. It is not uncommon for an obese child to have enlargement of the tonsils and adenoids. However, some previous studies did not control for the effect of upper lymphoid tissue when evaluating SDB in obese children.

In our study, we calculated and used the BMI z-score based on age-specific and gender-specific national survey data to define obesity. We used the A/N ratio to measure the upper airway blockage.[16]

The current study confirmed obesity to be a risk factor for OSAHS in children. In addition, obesity was positively related to the numbers of obstructive apnea/hypopnea events and was inversely related to arterial oxygen saturation drops in obese children. No persistent CO_2 retention was found. In the current study, 59.5% of otherwise healthy obese children had OSAHS. This rate was higher than the 46% reported by Marcus et al.[14] This might be due to the different ethnic groups and definitions of obesity. In the study by Marcus et al,[14] IBW was used to define obesity, and most of the subjects were white. It has also been reported[17] that Asians are prone to have obstructive respiratory events because of their cranial facial features.

AR might have an impact on OSAHS. However, no correlation between AR and OSAHS was found in our study. This might be because this was a cross-sectional study and only AR ("yes or no") has been used for analysis; therefore, our analysis could not differentiate current from past AR, intermittent vs persistent AR, and mild vs severe AR. A more precise evaluation of nose obstruction such as frontal cephalometrics should be used in future studies. No significant relationship was found in the current study between neck circumference and OSAHS. Although neck circumference was used as a predictor of OSAHS in adults, it seemed not to be useful for predicting OSAHS in children.

We demonstrated that in children with both OSAHS and obesity there was a positive correlation between the degree of obesity and the severity of OSAHS. Similar results were reported in other studies.[18] We speculated that weight reduction might be useful for the treatment of childhood SDB. Furthermore, for obese OSAHS patients who had enlargement of the adenoids and tonsils, the degree of reduction of AHI should be reevaluated after adenotonsillectomy.[19,20]

Our study is similar to that of Lam et al,[18] in which 482 children were reviewed and a positive correlation between AHI and BMI z-score was demonstrated. Our study included more polysomnography data and further supported the correlation between obesity and OSAHS. Furthermore, rather than using tonsil size only, our study also included A/N ratio to assess the upper airway. In our study, A/N ratio has been demonstrated to be a useful measurement for predicting OSAHS.

There are limitations in our study. First, the ATH control subjects of normal weight were referred from an ENT clinic rather than from the community. ENT doctors might send only children with suspected OSAHS to the sleep center for monitoring. Therefore, the prevalence of OSAHS might be overestimated in the control group. We assumed that if the control subjects had been from the general population, obese children would have a higher odds ratio of having OSAHS. Second, only some patients underwent $ETCO_2$ monitoring, and the absence of obstructive hypoventilation syndrome found in the current sample might well be due to the small sample size. A larger sample should be carried out for detecting obstructive hypoventilation syndrome.

Conclusion

Obese children are significantly more likely to have OSAHS, and the degree of obesity was positively related to the severity of OSAHS.

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