Severe Obesity Is a Risk Factor for Severe Obstructive Sleep Apnea in Obese Children

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Objective: *To determine the association between degree of obesity and severity of OSA in Thai children*
Material and Method: *The present retrospective study recruited obese children aged 3 to 15 years who had habitual snoring and underwent polysomnography (PSG) between January 2009 and June 2010. Obesity was defined as percentage of ideal weight for height (%W/H) ≥ 120 and was classified as mild (%W/H of 120-139), moderate (140-159), severe (160-199) and morbid (≥ 200). OSA was classified as severe (AHI ≥ 10) and non-severe (AHI < 10).*
Results: *Of 73 obese children, the mean age was 9.92 ± 3.42 years of which 60.3% were boys. The mean ± SD of BMI was 28.38 ± 5.99 kg/m^2 and %W/H ± SD was 162.63 ± 26.26. Gender, age, height, weight and BMI were not significantly different between severe and non-severe OSA groups. However, the %W/H of the severe OSA group (171.38% ± 29.54%) was significantly greater than the non-severe group (157.19% ± 22.68%) (p = 0.02). Severe to morbid obesity (OR 2.80, 95% CI 1.06-7.42; p = 0.038) and enlarged tonsils at least 3+ (OR 3.28, 95% CI 1.22-8.81; p = 0.018) were the risk factors for severe OSA.*
Conclusion: *Severe to morbid obesity was a predicting factor for severe OSA. These results suggested that severely obese children with snoring should have early recognition for severe OSA, which is highly contributing to multiple sequalae.*

Keywords: *OSA, Obese children, Obesity, Obstructive sleep apnea*

J Med Assoc Thai 2011; 94 (11): 1346-51
Full text. e-Journal: *http://www.mat.or.th/journal*

Obesity has become a global problem owing to obviously increased prevalence and its contribution to considerable adverse effects. Obstructive sleep apnea (OSA) characterized by intermittent complete or partial upper airway obstruction during sleep that disrupts normal ventilation and sleep pattern is an important consequence. Children with obesity appear to have a higher risk for OSA than normal-weight ones. The prevalence of OSA in the general population of children was 1 to 4%[1] but increased to 20 to 60% in obesity[2-7]. OSA leads to increased morbidity by impairing multiple systems such as cardiovascular, cognitive, and metabolic functions. In adult, the degree of obesity is strongly correlated with severity of OSA[8,9]. However, this correlation remains inconsistent in children. Some studies supported the role of degree of childhood obesity on severity of OSA[3,6,7,10-12] but others could not demonstrate this finding[2,4,5,13]. There are various factors such as ethnicity[7,11,12], age[14,15], and adenotonsillar enlargement[5-7] possibly affecting this correlation. Nevertheless, in Thai children, this association has not been reported. Therefore, the aim of the present study was to determine the association between degree of obesity and severity of OSA in Thai children. This relationship may help predict which level of obesity will predispose these children to severe OSA so that early investigation and appropriate treatment can be started in order to reduce complications and morbidity.

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Material and Method

Subjects

The present retrospective study recruited the obese children aged 3-15 years who had habitual snoring and underwent polysomnography (PSG) from January 2009 to June 2010 at the Siriraj Sleep Center,

Faculty of Medicine Siriraj Hospital. Obesity was defined as percentage of ideal body weight for height and gender (%W/H) ≥ 120. Degree of obesity was categorized as mild (%W/H of 120-139), moderate (140-159), severe (160-199) and morbid (200). The patients were also classified into two groups of severe (apnea-hypopnea index; AHI ≥ 10 events/hour) and non-severe OSA (AHI < 10 events/hour). Tonsil size 3+-4+ was determined as enlarged tonsils. The children with previous tonsillectomy or adenoidectomy, neuro-muscular diseases, craniofacial anomalies, airway anomalies, chromosomal disorders, or syndromic conditions were excluded. The patients' polysomnographic and demographic data including gender, age, weight, height and tonsil size were reviewed from medical records.

Polysomnography (PSG)

Overnight PSG was performed using a computerized PSG system (Sandman Elite® version 8 Tyco, healthcare, Canada). The following parameters were measured: electroencephalogram (F_4-M_1, C_4-M_1, O_2-M_1 and F_3-M_2, C_3-M_2, O_1-M_2), left and right electrooculogram, chin and leg electromyograms, electrocardiogram, airflow by oronasal thermistor and nasal pressure transducer, chest and abdominal effort using piezoelectric belts, body position, oxygen saturation using a pulse oximeter (Nellcor, Melville, CA) and end-tidal CO_2 by capnometer (Capnocheck® PLUS, SIMS BCI Inc., WI).

Children were continuously monitored by a pediatric sleep technician through an infrared video camera. Polysomnograms were manually scored by certified polysomnography sleep technicians and interpreted by certified sleep physicians. Sleep and associated events were scored using the standard criteria of the American Academy of Sleep Medicine (AASM)[16].

Obstructive apnea was defined as the absence of airflow on thermistor lasting for at least two consecutive breaths despite the presence of respiratory efforts. Central apnea was defined as the absence of airflow on thermistor along with the absence of respiratory efforts in combination with one of the following; 1) lasting for at least 20 seconds, 2) lasting for at least two consecutive breaths and resulting in 3% oxygen desaturation and/or arousals. Hypopnea was defined as a 50% reduction in airflow on nasal pressure lasting for at least two consecutive breaths and resulting in 3% oxygen desaturation and/or arousals.

Apnea-hypopnea index (AHI) was the number of apneas and hypopneas per hour of total sleep time (TST). The obstructive AHI of ≥ 1 event/hour was considered as the presence of obstructive sleep apnea (OSA). The severity of OSA was categorized as mild (AHI 1.0-4.9 events/hour), moderate (AHI 5.0-9.9) and severe (AHI ≥ 10)[17].

Statistical analysis

The data were expressed as mean ± SD or median (interquartile range, IQL) for normal or abnormal distribution data, respectively. Unpaired Student's t-test or the Mann-Whitney U test where applicable for quantitative data and Chi-square test for qualitative data were used to compare the data between the two groups of severe and non-severe OSA. Potential risk factors for severe OSA were determined by using logistic regression analysis. A p-value < 0.05 was considered statistically significant. All statistical analyses were performed by SPSS for Windows version 13.0.

The present study was approved by the Siriraj Institutional Review Board (Si 192/2010).

Results

The patients' characteristics are shown in Table 1. The present study recruited 73 children with

Table 1. Patients' characteristics

Characteristics	Mean ± SD (n = 73)
Age (years)	9.92 ± 3.42
Gender; boys: n (%)	44 (60.3%)
Height (cm)	144.22 ± 20.20
Weight (kg)	62.28 ± 26.41
Body mass index (kg/m^2)	28.38 ± 5.99
% weight for height	162.63 ± 26.26
Degree of obesity; n (%)	
Mild	14 (19.2)
Moderate	26 (35.6)
Severe	13 (17.8)
Morbid	20 (27.4)
Severity of OSA; n (%)	
Mild (AHI 1-4.9)	22 (30.1)
Moderate (AHI 5-9.9)	23 (31.5)
Severe (AHI ≥ 10)	28 (38.4)
Tonsil size; n (%)	
1+	17 (23.3)
2+	27 (37.0)
3+	27 (37.0)
4+	2 (2.7)

their mean (SD) age of 9.92 (3.42) years and 60.3% of them were boys. The mean (SD) of BMI was 28.38 (5.99) kg/m^2 and %W/H (SD) was 162.63 (26.26). There were 19.2%, 35.6%, 17.8% and 27.4% of children with mild, moderate, severe and morbid obesity, respectively. The PSG data revealed 30.1% of mild, 31.5% of moderate, and 38.4% of severe OSA. All obese children with snoring in the present study had OSA. 39.7% of these obese children had enlarged tonsils. The patients' polysomnographic data are shown in Table 2.

Table 2. Patients' polysomnographic data

Parameters	Median (IQL) (n = 73)
Total sleep time [TST] (hr)	7.0 (6.5-7.5)
Sleep efficiency (%)	89.8 (85.3-94.3)
Duration of REM stage (%TST)	17.2 (12.9-21.7)
Respiratory arousal index (events /hr)	4.1 (2.0-8.9)
AHI (events /hour)	7.3 (4.4-20.4)
SpO2 90-100% (%)	99 (97.0-99.8)
Mean SpO2 (%)	98 (97-98)
Minimum SpO2 (%)	86 (80-90)
Mean $P_{ET}CO2$ (mmHG)	35 (28-42)
Maximum $P_{ET}CO2$ (mmHG)	42 (38-50)

IQL = inter-quartile range

The factors analyzed as predictors for severe OSA are shown in Table 3. Gender, age, height, weight, and body mass index (BMI) were not significantly different between the children with severe OSA (AHI $\geq$ 10 events/hour) and non-severe OSA group (AHI < 10 events/hour). The %W/H of severe OSA group was significantly higher than non-severe group (171.38 $\pm$ 29.54% vs. 157.19 $\pm$ 22.68%; p = 0.02). The severe-morbid obesity was the significant risk factor for severe OSA (p = 0.038) with OR (95% CI) of 2.80 (1.06-7.42). Tonsillar enlargement was another significant predicting factor for severe OSA (p = 0.018) with OR (95% CI) of 3.28 (1.22-8.81).

Discussion

The present study demonstrated %W/H was associated with severe OSA. When stratified %W/H into degree of obesity, severe to morbid obesity (%W/H $\geq$ 160) was a significant predictor of severe OSA. Enlarged tonsils at least 3+ were also an independent risk factor for severe OSA. However, the

Table 3. Comparison of characteristics between severe and non-severe OSA

Factors	Severity of OSA		p-value	Odds ratio (95% CI)
	AHI < 10 mean $\pm$ SD (n = 45)	AHI $\geq$ 10 mean $\pm$ SD (n = 28)		
Gender; n (%)			0.95	
Boys	27 (61.4%)	7 (38.6%)		
Girls	18 (62.1%)	111 (37.9%)		
Age (years)	9.86 $\pm$ 3.69	10.03 $\pm$ 2.99	0.84	-
Height (cm)	144.69 $\pm$ 20.92	143.48 $\pm$ 19.34	0.81	-
Weight (kg)	60.93 $\pm$ 25.68	64.44 $\pm$ 27.87	0.58	-
Body mass index (kg/m2)	27.56 $\pm$ 5.56	29.71 $\pm$ 6.51	0.14	-
%weight for height	157.19 $\pm$ 22.68	171.38 $\pm$ 29.54	0.02*	-
Degree of obesity; n (%)				
Mild	10 (71.4)	4 (28.6)	-	1
Moderate	19 (73.1)	7 (26.9)	0.911	0.92 (0.22-3.92)
Severe	7 (53.8)	6 (46.2)	0.348	2.14 (0.44-10.53)
Morbid	9 (45.0)	11 (55.0)	0.133	3.06 (0.71-13.11)
Degree of obesity; n (%)				
Mild to moderate	29 (72.5)	11 (27.5)	-	1
Severe to morbid	16 (48.5)	17 (51.5)	0.038*	2.80 (1.06-7.42)
Tonsil size; n (%)				
Not enlarged (1+-2+)	32 (72.7)	12 (27.3)	-	1
Enlarged (3+-4+)	13 (44.8)	16 (55.2)	0.018*	3.28 (1.22-8.81)

severity of OSA was not related to gender, age, body weight, height, and BMI.

The studies by Marcus et al[3] and Brook et al[10] found the similar result of significant correlation between percentage of ideal weight for height and apnea-hypopnea index in obese children. Other studies using BMI Z score or BMI to determine the degree of obesity also revealed the correlation between degree of obesity and severity of OSA[6,7,11,12,18]. On the other hand, there were studies that failed to establish this correlation[2,4,5,13]. Unlike the consistent results in adults, the effect of obesity on severity of OSA in childhood remains inconclusive. These conflicting results may be due to the fact that association between obesity and OSA in children is influenced by multifactorial causes such as ethnicity, age and adenotonsillar size[19]. In addition, the criteria defined for obesity and OSA in each study was different. The present study used %W/H, instead of BMI, to reflect degree of obesity because there is standard reference of %W/H in Thai children. The standard BMI value for age and gender among Thai children is not available and using fixed cutoff point of BMI to define obesity as in adults is inappropriate because standard BMI in childhood changes with age.

Previous studies demonstrated strong association between body mass and OSA in Asian and African-American children. The significant correlation between degree of obesity and OSA was consistently reported among Chinese children[6,7,11]. Redline et al[18] and Rudnick et al[12] also found this association in African-American children. However, the studies among the white and Hispanic children revealed little or no association[13,20]. This racial effect is possibly attributed to the craniofacial structure[21]. The present study conducted in Thai children, therefore, supported the effect of Asian ethnicity on this association.

Age is another factor affecting the relation between obesity and OSA. The present study in which most subjects were older children with mean (SD) age of 9.92 (3.42) years, supported the previous studies that obesity increased the risk for OSA in older children and adolescent[14,15]. The study by Kaditis et al revealed that older children, aged > 6 years, who were obese had twice-greater risk for moderate to severe OSA when compared to non-obese children. Nevertheless, children ≤ 6 years old were at risk for moderate to severe OSA irrespective to body mass. Another study found BMI of OSA children was greater than of non-OSA children only for children aged > 8 years[15].

In the present study, tonsillar enlargement, defined as tonsil size 3+- 4+, was a predictor for severe OSA. This finding was in agreement with previous studies that assessed tonsil size by clinical inspection[5-7,11]. This may be the reason why adeno-tonsillectomy improves, even not cure, OSA in most obese children.

The strength of the present study is that it was conducted in Thai children. Prevalence of obesity has been increasing in Thailand as other countries and as mentioned, ethnicity is a factor contributing to the association between degree of obesity and OSA severity[12,18]. However, reports concerning this association in Thai obese children have not been available.

The limitations of the present study are retrospective in data collection. Consequently, some information such as detailed history of snoring, adenoid size, and neck, chest, abdominal, waist, or hip circumferences may be incomplete. In addition, the present study recruited obese children who had snoring symptom, not from general obese population. Furthermore, the authors' institute is a medical school hospital so that most children were referred from other medical centers and may experience more severe symptoms. This may be the reason why all obese children with snoring in the present study had OSA. Finally, evaluation of tonsil size might be varying because the assessment was performed by many physicians.

Conclusion

Among Thai obese children, tonsillar enlargement and %W/H were related to severity of OSA. Severe to morbid obesity as determined by %W/H ≥ 160 could be a predictor for severe OSA which may cause multiple consequences and leading to high morbidity and mortality. Therefore, children with severe to morbid obesity should be early detected and further investigations should be done to rule out severe OSA.

Acknowledgement

The authors wish to thank Associate Prof. Nuanchan Prapphal, MD for review and comment on this manuscript and Assist Prof. Dr. Chulaluk Komoltri for assistance in statistical analysis.

Potential conflict of interest

None.

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ภาวะอ้วนมากเป็นปัจจัยเสี่ยงต่อการเกิดภาวะหยุดหายใจขณะหลับขั้นรุนแรงในเด็กอ้วน

กนกพร อุดมอิทธิพงศ์, นิธิพัฒน์ เจียรกุล, พิมล รัตนาอัมพวัลย์, วัฒนชัย โชตินัยวัตรกุล, จักรพันธ์ สุศิวะ, ขุนพล มะโหฬาร, สุวัฒน์ ตั้งจิตยงศิวะ

วัตถุประสงค์: *เพื่อศึกษาความสัมพันธ์ระหว่างความรุนแรงของโรคอ้วนและความรุนแรงของภาวะหยุดหายใจขณะหลับในเด็กไทย*

วัสดุและวิธีการ: *เป็นการศึกษาย้อนหลังในเด็กโรคอ้วนอายุ 3-15 ปี ที่มีอาการนอนกรนประจำและได้รับการตรวจการนอนหลับโดยวิธี polysomnography ระหว่างมกราคม พ.ศ. 2552 ถึง มิถุนายน พ.ศ. 2553 ภาวะอ้วนใช้เกณฑ์ค่าร้อยละของน้ำหนักตามเกณฑ์ส่วนสูง (%weight for height; %W/H) ตั้งแต่ 120 ขึ้นไป และแบ่งระดับความรุนแรงของภาวะอ้วนเป็นอ้วนน้อย (%W/H 120-139) อ้วนปานกลาง (%W/H 140-159) อ้วนมาก (%W/H 160-199) และอ้วนรุนแรง (%W/H ≥ 200) ส่วนภาวะหยุดหายใจขณะหลับใช้ค่า AHI เป็นตัวกำหนดความรุนแรง และถือว่ามีภาวะหยุดหายใจขณะหลับรุนแรงมากเมื่อ AHI ≥ 10 ครั้งต่อชั่วโมง*

ผลการศึกษา: *มีเด็กโรคอ้วนจำนวนทั้งสิ้น 73 ราย อายุเฉลี่ย 9.92 ± 3.42 ปี เป็นเพศชายร้อยละ 60.3 ค่าเฉลี่ยดัชนีมวลกาย 28.38 ± 5.99 กก./ม^2 ค่าเฉลี่ยร้อยละของน้ำหนักตามเกณฑ์ส่วนสูง 162.63 ± 26.26 ไม่พบความแตกต่างของเพศ อายุ ความสูง น้ำหนัก และค่าดัชนีมวลกายระหว่างกลุ่มที่มีภาวะหยุดหายใจขณะหลับขั้นรุนแรงและไม่รุนแรง ส่วนค่าร้อยละของน้ำหนักตามเกณฑ์ส่วนสูงมีความแตกต่างอย่างมีนัยสำคัญทางสถิติระหว่าง 2 กลุ่ม (171.38% ± 29.54%, 157.19% ± 22.68%; p = 0.02) นอกจากนี้เด็กที่อ้วนมากหรืออ้วนรุนแรง (OR 2.80, 95% CI 1.06-7.42; p = 0.038) และทอนซิลโตอย่างน้อย 3+ ขึ้นไป (OR 3.28, 95% CI 1.22-8.81; p = 0.018) เป็นปัจจัยเสี่ยงต่อการเกิดภาวะหยุดหายใจขณะหลับขั้นรุนแรง*

สรุป: *เด็กที่อ้วนมากหรืออ้วนรุนแรงมีความเสี่ยงต่อการเกิดภาวะหยุดหายใจขณะหลับขั้นรุนแรง ดังนั้นเด็กกลุ่มนี้ที่มีอาการนอนกรนควรได้รับการสืบค้นเพื่อวินิจฉัยภาวะหยุดหายใจขณะหลับขั้นรุนแรง เนื่องจากภาวะดังกล่าวก่อให้เกิดผลแทรกซ้อนหลายอย่างตามมาได้*