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Risk of obstructive sleep apnea syndrome in Japanese men with a history of adenotonsillar hypertrophy

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Abstract

Objectives: Obstructive sleep apnea syndrome (OSAS) is an important cause of medical morbidity and mortality. Although adenotonsillar hypertrophy is linked to the pathogenesis of OSAS in children, the potential role of childhood adenotonsillar hypertrophy in the etiology of adult OSAS has not yet been examined.

Methods: We retrospectively evaluated 1,369 men aged ≥20 years with suspected OSAS who had undergone polysomnography at Fujita Health University Hospital in Japan. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated after adjusting for age and body mass index to evaluate the risk of development of OSAS in men with a history of adenotonsillar hypertrophy in childhood. The reference category for OSAS was non-OSAS.

Results: In total, 988 men were diagnosed with OSAS and 561 were diagnosed with severe OSAS (apnea–hypopnea index of ≥30). The adjusted ORs for a history of untreated adenotonsillar hypertrophy with OSAS and severe OSAS were 3.13 (95% CI, 1.18–8.27) and 4.31 (1.56–11.90), respectively. The adjusted ORs for a history of treated adenotonsillar hypertrophy with OSAS and severe OSAS were 1.31 (0.69–2.50) and 0.87 (0.41–1.90), respectively.

Conclusions: This study confirmed the risk of untreated childhood adenotonsillar hypertrophy in the development of adult OSAS. Our data also support the idea that abnormal dentofacial morphology induced by adenotonsillar hypertrophy in childhood is a critical factor in the pathogenesis of OSAS in adulthood.

Keywords: Obstructive sleep apnea, Adenotonsillar hypertrophy, Epidemiology

Introduction

Obstructive sleep apnea syndrome (OSAS) is a common disorder,[1,2] and its estimated prevalence in the Japanese population is 9.0% of men and 2.8% of women.[3,4] OSAS is characterized by repetitive upper airway collapse during sleep. Patients with OSAS have a reduced cross-sectional area of the upper airway lumen due to excessive bulk of the soft tissues, abnormal craniofacial anatomy, or both.[5] OSAS is an important cause of medical morbidity of hypertension, cardiovascular and cerebrovascular disturbances,[6-8] and the resulting increases in mortality.[1,9] In particular, severe OSAS significantly increases the risk of hypertension and fatal and nonfatal cardiovascular events.[10,11] Advanced age, anatomic variations, alcohol consumption, sex, and obesity are important factors in the development of OSAS.[1,12,13]

The most common cause of OSAS in children is adenotonsillar hypertrophy.[14,15] Although adenotonsillar hypertrophy in childhood may possibly cause OSAS in adulthood,[16,17] we found no studies on the effect of a history of adenotonsillar hypertrophy on the development of OSAS in adulthood. In the present study, we evaluated the risk of OSAS and severe OSAS associated with a history of adenotonsillar hypertrophy in Japanese men.

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Materials and methods

Patients

We retrospectively evaluated 1,481 men with suspected OSAS who had undergone polysomnography at Fujita Health University Hospital from September 1995 through June 2007. Women were not included because of the small number of these patients. Among the 1,481 men, 102 were excluded because they were aged <19 years and 10 were excluded because of insufficient data. Thus, 1,369 men aged ≥ 20 years were analyzed. All patients provided informed consent.

Polysomnography

The polysomnographic recordings were obtained using the Alice 3 Diagnostic Sleep System (Philips Respironics, Amsterdam, the Netherlands). Electroencephalogram leads, (C4-A2, C3-A1, O2-A1, and O1-A2), bilateral electro-oculograms, and submental electromyograms were used to monitor sleep. An anterior tibialis electromyogram was recorded to detect leg movements. A bipolar electrocardiogram was simultaneously recorded for cardiac monitoring. Blood oxygen levels were determined by finger pulse oximetry. Respiratory airflow was monitored using a nasal pressure cannula and a naso-oral thermistor. Respiratory effort was monitored using rib and abdominal piezoelectric strain gauges. The sleep stages and respiratory events were scored by registered polysomnogram

technicians at the Sleep Laboratory of the Fujita Health University Hospital using the standard diagnostic criteria of the American Academy of Sleep Medicine[18] and previously published criteria.[19] Apnea and hypopnea were defined as the cessation or reduction of respiration for ≥10 seconds caused by either airway obstruction or lack of respiratory effort that was associated with at least one of the following: (1) >90% airflow reduction, (2) >3% oxygen desaturation, and (3) subsequent arousal. The apnea–hypopnea index (AHI) was determined for each polysomnographic recording as the mean number of apneas and hypopneas per hour of total sleep time.

Event Definition

A self-administered questionnaire was used to determine the Epworth Sleepiness Scale (ESS) score,[20] medical history, and treatment or lack of treatment, including that for hypertrophy of the adenoids or/and tonsils. The diagnostic criteria in the second revision of the International Classification of Sleep Disorders were used to define OSAS, i.e., either an AHI of ≥ 5 events/h in combination with an ESS score of ≥11 or an AHI of ≥15 events/h indicated a positive OSAS diagnosis.[5] The severity of OSAS was defined by the AHI, where a frequency of ≥30 events/h was considered severe. Patients with a history of adenotonsillar hypertrophy were those with hypertrophy of the adenoid or/and tonsils, and patients with a history of treatment for adenotonsillar hypertrophy were those who had undergone adenotonsillectomy.

Data Analysis

Patient data were available for age, body mass index (BMI), AHI, ESS score, and history of hypertrophy of the adenoids or/and tonsils. The BMI was calculated as the body weight divided by the square of the height (kg/m^2). Data were evaluated using the Statistical Package for the Social Sciences (SPSS) ver. 17.0 (SPSS Inc., Chicago, IL, USA). Frequencies and average values of occurrence were analyzed using a χ^2-test and independent samples *t*-test. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated using logistic regression after adjusting for age and BMI to evaluate the risk of development of OSAS in men with a history of adenotonsillar hypertrophy in childhood. The reference category for OSAS was non-OSAS.

Ethical Considerations

This study was approved in November 2010 by the Ethical Review Board for Epidemiological and Clinical Studies of the Fujita Health University School of Medicine, Aichi, Japan (No. 10-183).

Results

Of all 1,369 men analyzed, 988 were diagnosed with OSAS and 561 were categorized as having severe OSAS (AHI ≥ 30). The remaining 381 men were diagnosed with non-OSAS (213 with snoring, 27 with narcolepsy, 23 with no abnormality, 16 with upper airway resistance syndrome, 14 with restless legs syndrome, and all that). The characteristics of all 1,369 men are shown in Table 1. The average age, BMI, AHI, and ESS score in patients with OSAS were significantly higher than in those without OSAS (Table 1).

The ORs and 95% CIs for the association between a history of adenotonsillar hypertrophy and OSAS are presented in Table 2. The ORs for a history of untreated adenotonsillar hypertrophy with OSAS and severe OSAS were significantly higher than those for non-OSAS (OR, 3.42; 95% CI, 1.34–8.72 and OR, 4.47; 95% CI, 1.72–11.60, respectively). Thus, OSAS and severe OSAS were significantly associated with a history of untreated adenotonsillar hypertrophy. Conversely, the ORs for a history of treated adenotonsillar hypertrophy with OSAS and severe OSAS were not significant (Table 2).

After adjustment for age and BMI, the ORs for a history of adenotonsillar hypertrophy with OSAS and severe OSAS

Table 1. Patient characteristics

Measurement	Patients with OSAS (n = 988)	Patients without OSAS (n = 381)	P value
Age, years	49.7 (13.6)	45.0 (15.8)	<0.001
BMI, kg/m^2	27.0 (5.4)	23.9 (4.0)	<0.001
AHI, events/h	37.1 (20.7)	5.2 (4.4)	<0.001
ESS score, points	10.0 (5.1)	8.1 (4.8)	<0.001

Data are presented as mean (standard deviation).
BMI, body mass index; AHI, apnea–hypopnea index; ESS, Epworth Sleepiness Scale; OSAS, obstructive sleep apnea syndrome (AHI ≥ 5 and ESS score ≥ 11, or AHI ≥ 15).

Table 2. Odds ratios and 95% confidence intervals for OSAS and severe OSAS associated with a history of adenotonsillar hypertrophy in Japanese men

History of adenotonsillar hypertrophy	Non-OSAS	OSAS			Severe OSAS		
	n	n	OR (95% CI)	P value	n	OR (95% CI)	P value
No history	363	891			504		
Untreated	5	42	3.42 (1.34–8.72)	0.006	31	4.47 (1.72–11.60)	0.000
Treated	13	55	1.72 (0.93–3.19)	0.100	26	1.44 (0.73–2.84)	0.374

OSAS, obstructive sleep apnea syndrome (OSAS was defined as AHI ≥ 5 and ESS score ≥ 11, or AHI ≥ 15; severe OSAS was defined as AHI ≥ 30); OR, odds ratio (calculated using logistic regression analysis); CI, confidence interval. The reference category for OSAS is non-OSAS.

Table 3. Risk of OSAS associated with history of adenotonsillar hypertrophy by adjusted odds ratios and 95% confidence intervals in Japanese men

History of adenotonsillar hypertrophy	Non-OSAS	OSAS			Severe OSAS		
	n	n	Adjusted OR (95% CI)	P value	n	Adjusted OR (95% CI)	P value
No history	363	891			504		
Untreated	5	42	3.13 (1.18–8.27)	0.021	31	4.31 (1.56–11.90)	0.005
Treated	13	55	1.31 (0.69–2.50)	0.414	26	0.87 (0.41–1.90)	0.722

OSAS, obstructive sleep apnea syndrome (OSAS was defined as AHI ≥ 5 and ESS score ≥ 11, or AHI ≥ 15; severe OSAS was defined as AHI ≥ 30); OR, odds ratio (adjusted for age and body mass index using logistic regression); CI, confidence interval. The reference category for OSAS is non-OSAS.

were still highly significant (OR, 3.13; 95% CI, 1.18–8.27 and OR, 4.31; 95% CI, 1.56–11.90, respectively). The ORs for a history of treated adenotonsillar hypertrophy with OSAS and severe OSAS were not significant (Table 3).

Discussion

In this study, we evaluated the association between a history of adenotonsillar hypertrophy in childhood and the presence of OSAS in men. This study showed that untreated hypertrophy of the adenoid or/and tonsil significantly increased the risk of OSAS in men compared with those with no history; the adjusted ORs for OSAS and severe OSAS associated with untreated adenotonsillar hypertrophy history were 3.13 (95% CI, 1.18–8.27) and 4.31 (95% CI, 1.56–11.90), respectively. Moreover, treated hypertrophy of the adenoid or/and tonsil did not increase the risk of OSAS compared with no history.

Several studies have shown an association between abnormal dentofacial morphology and the pathogenesis of OSAS in adults.[17,21,22] However, the effect, of a history of adenotonsillar hypertrophy on the development of OSAS in adulthood has not been shown. The most common cause of OSAS in children is adenotonsillar hypertrophy.[14,15] Adenotonsillar hypertrophy is a cause of mouth breathing due to impaired nasal breathing.[23] This condition leads to a posteriorly inclined mandible and hyoid bone caused by high intrapleural negative pressure reaching the upper airway secondary to pharyngeal airway obstruction.[24] That is, an abnormal dentofacial morphology is frequently observed in adult patients with OSAS.[21,22] Tonsillectomy or/and adenoidectomy are effective treatments for pediatric OSAS caused by impaired nasal breathing.[25] However, treatment efficacy decreases as the child ages,[26] and early adenotonsillectomy improves the dentofacial morphology.[27] The persistence of OSAS after adenotonsillectomy may be partly due to the smaller size of the mandible in children.[28] The present study shows that untreated hypertrophy of the adenoid or/and tonsil in childhood causes OSAS in adulthood. Untreated adenotonsillar hypertrophy leads to an abnormal dentofacial morphology and leads to probable OSAS in adulthood.

We adjusted for the main confounding factors, age and BMI. Although it is possible that the evaluated exposure to adenotonsillar hypertrophy was inaccurate due to recall bias, it is quite unlikely to obtain these relationships for that reason. We did not examine women because of the small number of affected female patients.

In conclusion, this study shows that untreated hypertrophy of the adenoid or/and tonsil in childhood increases the risk of OSAS and severe OSAS in men by 3.13 and 4.31 times, respectively, compared with men having no history of hypertrophy. The results of this study support the idea that an abnormal dentofacial morphology induced by untreated adenotonsillar hypertrophy in childhood is a critical cause of the development of OSAS and increases the risk of severe OSAS in adulthood.

Conflict of interests

The authors declare no conflict of interest.

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