Clinical Characteristics of Chinese Pediatric Obstructive Sleep Apnea Hypopnea Syndrome

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Abstract

We aim to analyze the clinical characteristics of obstructive sleep apnea hypopnea syndrome (OSAHS) in children. Polysomnography (PSG) and nasopharynx lateral film were performed, and clinical data were collected in 469 children who came to the outpatient department due to "snoring during sleep with mouth opening breathing, hard breathing or suffocation". Among the enrolled children, 123 (22.6%) were diagnosed with OSAHS, with 70 were mild and 53 were moderate-severe. Percentage of adenoid hypertrophy was higher in OSAHS patients ($p < 0.01$), instead of tonsil enlargement. The OSAHS children were aged 5 (4, 7). Compared with PS, the percentage of snoring, apnea, dyspnea, increased nocturia, and daytime sleepiness were significantly higher in moderate-severe patients ($p < 0.01$). In OSAHS groups, AHI, ODI, Longest time of apnea were increased, while minimum SpO₂ and mean SpO₂ during sleep were decreased significantly ($p < 0.01$) than PS. Time ratio of NREM1 was elevated in moderate-severe OSAHS patients ($p < 0.01$). Time ratio of REM was elevated in mild patients ($p < 0.01$). Compared with the preschoolers, the percentage of leg movement and sleepiness were significantly higher in school-agers ($p < 0.05$). The youngers had higher time ratio of NREM3 and better sleep efficiency ($p < 0.01$). However, AHI ($p < 0.05$) and ODI ($p < 0.01$) were higher in elder OSAHS significantly. Snoring (OR = 5.745, $p < 0.01$), adenoid hypertrophy (OR = 4.381, $p < 0.01$), apnea (OR = 2.670, $p < 0.001$), dyspnea (OR = 1.975, $p < 0.01$), and CRP (OR = 1.172, $p < 0.001$) were independent risk factors for OSAHS.

Conclusion AHI, ODI, Longest time of apnea, minimum SpO₂ and mean SpO₂ should be considered and analyzed simultaneously in diagnosis. The school-age OSAHS patients seems to more serious than the preschoolers. Snoring, apnea, dyspnea, adenoid hypertrophy, and CRP are risk factors for OSAHS.

Introduction

Obstructive sleep apnea hypopnea syndrome (OSAHS) is a frequent health problem in children with sleep disordered breathing. As reported, the incidence of OSAHS in children is as high as 1.2-5.7%, and the peak age is 2-8 years old[1-3]. OSAHS is a common disease characterized by the presence of recurrent episodes of increased upper airway resistance during sleep, leading to spells of apnea/hypopnea, hypercapnia, nocturnal sleep fragmentation, etc. It can also cause multiple problems, such as hypertension, glucose and lipid metabolism disorder, cognitive function damage, and growth and development delay. OSAHS in children is very much different from that in adult, and OSAHS in children is also variable in different age group. It is still complex to diagnose and manage in childhood, and AHI is considered insufficient to assess OSAHS. Pediatricians should attach more importance to the clinical manifestations and signs. In this study, we analyzed the clinical characteristics of OSAHS diagnosed in our sleep centre, and expected to be helpful for better diagnosis.

Materials And Methods

Patient

All 469 cases were consecutively recruited between December 2016 and November 2019, who met the following inclusion criteria: (1) aged older than 3 and younger than 15 ; (2) complaining about snoring, mouth breathing, dyspnea or apnea, labored or paradoxical breathing and gasping during sleep; (3) Patients with recent infectious diseases, known sleep apnea on regular therapy, maxillofacial dysplasia, autoimmune diseases, congenital pulmonary and heart disease, neuromuscular diseases, and other serious disease were excluded.

Data collection

The medical records of patients were reviewed retrospectively. Parental complaint was recorded which consisted of snoring, mouth breathing, salivation, labored breathing, bruxism, apnea, increased nocturia(IE), night enuresis(NE), nightmare, oppressive wake and so on. Physical examination, nasopharyngeal lateral film, and report of polysomnography were also integrated.

IE was defined as the frequency of urinating at night reaching three or more times after falling asleep. NE was defined as bed wetting for more than twice a week for 3 consecutive months, or a child 5–6 years old should have two or more bed-wetting episodes per month, and a child older than 6 years of age should have one or more bed-wetting episodes per month[4,5].

Tonsil and adenoid size assessment

Tonsil grade was determined using the Brodsky grading scale, in which the tonsils are assigned a grade from 1 to 4. This is dependent on the percentage of oropharyngeal airway occupied by the tonsils. Grade 1 = 25%, grade 2 = 26-50%, grade 3 =51-75%, and grade 4 >75%. The Clemens and McMurray adenoid grading system was used[6]. Grade 1 has adenoid tissue filling 1/3 the vertical height of the choana; Grade 2 up to 2/3; Grade 3 from 2/3 to nearly all but not complete filling of the choana and Grade 4 with complete choanal obstruction[7].

A lateral neck X-ray was used to determine the size of adenoid. Adenoid/tonsil size can be determined using adenoidal-nasopharyngeal (A/N) ratio. A: The perpendicular distance from the maximum convexity of the adenoid to the lateral surface of the occipital slope of the skull. N: The distance between the posterior end of the hard palate to the pterygoid and the intersection of the skull base is the width of the nasopharyngeal space[8]. $A/N \leq 0.6$ is normal. Between 0.61 and 0.7 is physiological hypertrophy. And $A/N \geq 0.71$ is Pathological hypertrophy[9].

Assessment of anthropometry

Height(m) and weight(kg) of the children were measured. According to the Body Mass Index Growth Curves For Chinese Children And Adolescents Aged 0 To 18 Years[10], obesity was defined as a body mass index (BMI, kg/m^2) >95th percentile of peers with the same age and gender.

Polysomnography

Overnight PSG (Compumedics E series and Greal series, Australia) was performed for all patients at the first night of admission starting from 10 p.m. to 6 a.m. next morning, and at least 7 h of recording time was considered a successful monitoring. Alcohol, tea, caffeine, sedatives or hypnotics were forbidden 24h before.

PSG included electroencephalography (4 channels: C3–A2, C4–A1, O1–A2, O2–A1), electrooculogram (2 channels: LOC-A2, ROC-A1), electromyogram (1 channels: mandibular muscle), electrocardiogram (1 channels), and recording of snoring and body position. The airflow was monitored with a thermistor and a pressure cannula. Respiratory efforts were detected with ribcage and abdominal piezo sensors. Arterial oxygen saturation was monitored by pulse oximetry. All PSG studies were analyzed by trained technicians and sleep physicians according to the criteria of the American Academy of Sleep Medicine 2012[11].

Diagnosis of OSAHS and grouping

According to the Draft guidelines for diagnosis and treatment of obstructive sleep apnea hypopnea syndrome in children (urumqi). Mild OSAHS was defined as $5 \leq \text{AHI} \leq 10$ or $1 \leq \text{OAI} \leq 5$, moderate was defined as $10 \leq \text{AHI} \leq 20$ or $5 \leq \text{OAI} \leq 10$, severe was defined as $\text{AHI} \geq 20$ or $\text{OAI} \geq 10$ [12]. Primary snoring (PS) was defined as $\text{AHI} \leq 5$ and $\text{OAI} \leq 1$, and other sleep related diseases were excluded.

Blood sampling and biochemical investigations

Samples of venous blood were collected after overnight fasting. C-reactive protein (CRP) was measured.

Statistical analyses

Variables exhibiting a normal distribution are represented as mean $\pm$ standard deviation (SD), whereas those with a non-normal distribution are represented by the corresponding nonparametric statistics. Statistical testings between the two groups (OSAHS vs. PS) were carried out by Student's t-test, non-parametric test (Mann-Whitney) or Chi-square test when appropriate. When the three groups (mild OSAHS, moderate-severe OSAHS and PS) were compared, variance analysis or non-parametric test (Mann-Whitney) were used appropriately. In addition, binary logistic regression analyses were performed to investigate the relationships between OSAHS against the clinical and PSG parameters. All statistical analyses were conducted using *SPSS 22.0* software for Windows. All probabilities were 2-tailed, and values of $p < 0.05$ were considered statistically significant.

Results

469 children were enrolled, ranging from 2 to 15 years old, with a median age of 5 (4, 7). There were 283 males and 162 females. 123(22.6%) cases were diagnosed as OSAHS. 70 were mild OSAHS, 20 were moderate OSAHS, and 33 were severe OSAHS. 322 were primary snoring (PS). 24 were excluded because of central sleep apnea syndrome, narcolepsy, restless legs syndrome, and so on.

The OSAHS patients were aging from 3 to 12 years old, with a median age of 5 (4, 7) (seen in picture 1). There were 86 boys and 37 girls. There was no significant difference between boy and girl with OSAHS.

Picture 1 The age distribution of the OSAHS patients

Clinical manifestations of OSAHS

Cases of the night performance of with OSAHS children was as follow: 120 cases (97.6%) with snoring, 104 cases (84.6%) with mouth breathing, 77 cases (62.6%) with salivation, 69 cases (56.1%) with labored breathing, 68 cases (55.3%) with IE, 52 cases (42.3%) with bruxism, 52 cases (42.3%) with leg movement, 46 cases (37.4%) with apnea, 27 cases (22%) with NE, 26 cases (21.1%) with nightmare, 24 cases (19.5%) with oppressive wake.

Cases of the daytime symptoms of with OSAHS children was in the order: 55 cases (44.7%) with difficulty getting up in the morning, 49 cases (39.8%) with mouth parched in the morning, 49 cases(39.8%) with hyperactivity, 47 cases (38.2%) with difficulty concentrating, 38 cases (30.9%) with morning fatigue, 29 cases (23.6%) with daytime sleepiness, 11 cases (8.9%) with social withdrawal, 9 cases (7.3%) with memory impairment, 5 cases (4.1%) with morning headaches.

Percentage of adenoid hypertrophy was higher in OSAHS patients ($p < 0.01$). 5 children had no adenoid hypertrophy, while 53 had slight adenoid hypertrophy, 35 had moderate adenoid hypertrophy, and 30 had severe adenoid hypertrophy. However, Percentage of tonsil enlargement was slightly higher in OSAHS patients.

The body mass index (BMI) of 123 children with OSAHS was ranged from 12.43-33.82 kg/m², with a mean value of 17.4±4.0 kg/m². 23 cases (18.7%) were achieving the standards for obese. There was no significant difference in BMI between OSAHS group and PS group (Table 1).

Table 1 Comparison of general information between OSAHS and PS

	OSAHS(N=123)	PS(N=322)	P value
Age(year)	5.0(4.0,7.0)	5.0(4.0,7.0)	0.739
male/female(N,%)	86(69.9)/37(30.1)	197(61.2)/125(38.8)	0.099
Disease course(month)	24.0(8.0,36.0)	12.0(6.0,36.0)	0.007
BMI(kg/m ²)	17.4±4.0	16.7±2.7	0.087
Tonsil enlargement(N,%)	105(85.4)	252(78.3)	0.110
Adenoid hypertrophy(N,%)	118(95.9)	275(85.4)	0.003
Rhinitis/sinusitis(N,%)	94(76.4)	259(80.4)	0.361
Asthma(N,%)	10(8.1)	44(13.7)	0.143
Cigarettes exposure(N,%)	51(41.5)	132(41.0)	1.000
Family snoring(N,%)	93(75.6)	251(78.0)	0.614
CRP(mg/L)	0.32(0.14, 0.79)	0.51(0.21, 2.12)	0.000

Data are expressed as mean ± SD, median (25%(C), 75%(D)), or as indicated.

Symptoms and PSG parametres of mild OSAHS, moderate-severe OSAHS and PS patients

Compared with PS, the percentage of snoring, apnea, dyspnea, increased nocturia, and sleepness were significantly higher in moderate-severe OSAHS patients ($p < 0.01$). Apnea($p < 0.05$) and dyspnea($p < 0.01$) were elevated in mild OSAHS patients, too (Table 2). In mild and moderate-severe group, AHI, ODI, Longest time of apnea, were increased, while minimum SpO₂ and mean SpO₂ during sleep were decreased significantly($p < 0.01$) than PS. Time ratio of NREM1 was elevated in moderate-severe OSAHS patients($p < 0.01$). Time ratio of REM was elevated in mild patients($p < 0.01$) (Table 3).

The moderate-severe patients were much more sleepness than PS ($p < 0.01$). However, there is no difference between the groups in common daytime symptoms, including difficulty in concentration, behavioral and emotional problems, and morning headaches.

Table 2 Comparison of Nocturnal and daytime symptoms

	PS(N=322)	Mild(N=70)	Moderate-severe(N=53)	P value
Snoring	283	67	53 [#]	0.006
Salivation	172	45	32	0.199
mouth breathing	239	59	45	0.068
dyspnea	105	36 ^{**}	33 ^{##}	0.000
apnea	46	50 [*]	26 ^{##+}	0.000
oppressive wake	36	11	13 [#]	0.026
night enuresis	44	15	12	0.101
increased nocturia	134	37	31 ^{##}	0.029
bruxism	158	33	19	0.204
leg movement	121	30	22	0.654
nightmare	69	16	10	0.865
sleepness	29	14	15 ^{##}	0.001

*PS & mild OSAHS $p < 0.05$ ** $p < 0.01$; #, PS & Moderate-severe OSAHS $p < 0.05$ ## $p < 0.01$; + mild & Moderate-severe OSAHS $p < 0.05$ ++ $p < 0.01$.

Table 3 Comparison of sleep and respiratory parameters PSG

	PS	mild	Moderate-severe	P value
Total sleep time	471.59±76.24	461.90±67.70	481.76±77.68	0.212
Sleep efficiency	82.85(73.68, 89.80)	82.10(72.88,89.32)	83.00(78.70,90.40)	0.262
Arousal index(AI)	1.88(1.36,2.64)	1.89(1.29,2.52)	1.86(1.28,2.52)	0.595
AI of REM	3.10(1.50,5.85)	3.60(1.88,8.13)	6.20(2.85,14.30) ^{##}	0.000
N1%	12.48±5.36	13.42±5.54	14.91±4.89 ^{##}	0.002
N2%	44.43±8.79	42.17±8.05	43.03±9.23	0.288
N3%	23.66±9.95	22.63±6.73	21.75±6.79	0.181
REM	20.04±4.50	21.80±4.63 ^{**}	20.01±4.85	0.971
AHI	1.10(0.60, 2.10)	6.35(4.80,8.00) ^{**}	21.70(14.90,33.60) ^{####}	0.000
OAI	0.00(0.00, 0.00)	1.40(0.50,2.82) ^{**}	10.00(2.35,15.75) ^{##}	0.000
Longest time of apnea	11.00(9.00, 13.00)	17.00(12.75,22.25) ^{**}	23.00(19.00,36.50) ^{##}	0.000
ODI	0.30(.010,0.60)	1.90(0.80,3.73) ^{**}	12.10(6.55,19.75) ^{####}	0.000
Minimum SpO2	91.45±2.91	86.59±5.48 ^{**}	77.57±11.06 ^{####}	0.000
Mean SpO2	97.11±0.70	96.74±0.85 ^{**}	94.65±3.09 ^{####}	0.000

Data are presented as mean ± SD and/or median (minimum, maximum).

REM, rapid eye movement; AHI, apnea hypopnea index; OAI, Obstructive apnea index; SpO2, pulse oxygen saturation; ODI, oxygen desaturation index.

Comparison of OSAHS children in preschool and school age

Children with OSAHS were divided into two group: Preschoolers: 3~6 year old (N=87) , and School-agers: 7~12 year old (N=36). Compared with preschoolers, the percentage of leg movement and sleepiness were significantly higher in school-agers ($p < 0.05$) (Table 4). The younger ones had higher time ratio of NREM3 and better sleep efficiency ($p < 0.01$). time ratio of REM was also elevated in younger children. However, AHI($p < 0.05$) and ODI($p < 0.01$) were higher in elder OSAHS (Table 5).

Among the patients over 5 years old, increased nocturia in moderate-severe OSAHS occurred more often ($p < 0.05$). Pearson correlation analysis had shown that nocturnal urination was positively correlated with OAI ($p < 0.01$) , and negatively correlated with average SpO₂ ($p < 0.01$) .

Table 4 Comparison of Nocturnal and daytime symptoms in different age group

	3~6 year old	7~12 year old	P value
Snoring	85 (97.7)	35 (97.2)	1.000 ^b
Salivation	53 (60.9)	24 (66.7)	0.683
mouth breathing	76 (87.4)	28 (77.8)	0.271
dyspnea	52 (59.8)	17 (47.2)	0.234
apnea	35 (40.2)	11 (30.6)	0.413
oppressive wake	18 (20.7)	6 (16.7)	0.632
night enuresis	18 (20.7)	9 (25.0)	0.163
increased nocturia	52 (59.8)	16 (44.4)	0.636
bruxism	38 (43.7)	14 (38.9)	0.691
leg movement	43 (49.4)	9 (25.0)	0.016
nightmare	19 (21.8)	7 (19.4)	0.814
sleepiness	15 (17.2)	14 (38.9)	0.012
headache	3 (3.4)	2 (5.6)	0.629 ^b
fatigue	24 (27.6)	14 (38.9)	0.284
difficult to get up	34 (39.1)	21 (58.3)	0.072

Data are presented as N (n%).

^b, Fisher exact test.

Table 5 Comparison of PSG results of OSAHS children in preschool and school age

	3~6 year old	7~12 year old	P value
Total sleep time	482.2±62.7	442.1±86.6	0.015
Sleep efficiency	84.4±78.0,90.7	76.3±13.3	0.004
Arousal index(AI)	14.0±10.0,18.0	17.0±10.5,22.8	0.142
AI of REM	5.0(2.8,10.6)	3.2(1.6,9.5)	0.135
N1%	68.3±27.2	59.5±21.7	0.083
N2%	194.9±44.7	207.0±55.4	0.206
N3%	23.2±6.4	19.1±16.2,22.1	0.001
REM%	21.5±19.0,24.6	19.1±4.4	0.010
AHI	8.3(5.8,17.6)	11.2(7.1,24.4)	0.022
Longest time of apnea	20.0(15.0,27.0)	19.0(14.3,25.8)	0.856
ODI	3.2(1.0,6.8)	5.7(3.1,13.8)	0.007
Minimum SpO2	85.0±80.0,88.0)	86.0±79.5,89.0)	0.940

Correlation of anthropometry and clinical variables with OSAHS

To investigate the relationships between anthropometry and clinical variables with OSAHS, Pearson correlation analysis was performed which demonstrated that OSAHS had been correlated with symptoms of snoring, mouth breathing, dyspnea, apnea, oppressive wake, increased nocturia and night enuresis, and signs of adenoid hypertrophy ($p < 0.05$). In addition, course of disease, CRP and BMI were positively correlated with OSAHS ($p < 0.05$). Regression analyses were performed among the anthropometry and clinical parameters and CRP, and it revealed that OSAHS was significantly associated with snoring (odds ratio:5.745, 95% CI: 1.616-20.424; $p < 0.01$), adenoid hypertrophy (odds ratio: 4.381, 95% CI: 1.619-11.858; $p < 0.01$), apnea (odds ratio:2.670, 95% CI: 1.554-4.587; $p < 0.001$), dyspnea (odds ratio:1.975, 95% CI: 1.223-3.190; $p < 0.01$), and CRP (odds ratio: 1.172, 95% CI: 1.077-1.276; $p < 0.001$).

Discussion

Obstructive sleep apnea hypopnea syndrome is one of the common type of sleep disordered breathing in children. It may cause multiple complications in organs and systems, affecting children's growth and development negatively. However, it is still complex to diagnose and manage in the pediatric age. This study retrospectively analyzed the clinical and PSG manifestations of OSAHS in children diagnosed in our centre.

In our study, the median age of OSAHS in children is 5 (4, 7) years old in this study, and 25.6% of the children recruited were diagnosed OSAHS, which was consistent with the reports[13]. Adenoidal and

tonsillar hypertrophy is recognized to be closely with OSAHS severity[14]. In our study, adenoidal hypertrophy were the prominent elements found in OSAHS patient, and it was independently related to OSAHS. But there was no significant difference in tonsillar hypertrophy between the groups. Researchers have thought that using tonsil size only in diagnosis was limited, even the clear parent-reported history of habitual snoring was more valuable[15,16].

Moderate-severe patients had higher frequency of rhinitis and/or nasosinusitis, which was obviously correlated with OSAHS. In our study, C-reaction protein was associated with OSAHS independently. Therefore, recurrent inflaming in upper airway must be treated effectively and in time and was considered to be related to OSAHS[17]. It may lead to a persistent systemic inflammatory response in the children. Inflammatory response is considered as an important pathophysiological process of OSAHS.

Daytime symptoms of OSAHS children usually included difficulty concentrating, behavioral and emotional problems, morning headaches, excessive daytime sleepiness, and dysplasia[18]. In our study, sleepiness was the only factor found to be different between the groups. We may need to expand the data sources through asking teachers from preschool or school.

The common nocturnal symptoms of children with OSAHS include snoring, excessive sweating, restless sleep, mouth breathing, apnea, wheezing, dyspnea or abnormality, and excessive neck extension during sleep. We found that the symptoms of snoring, dyspnea, apnea, nocturia, and oppressive wake were more discriminative. Increased nocturia was an interesting symptom of OSAHS, especially the moderate-severe ones. Nocturia has been shown to be a complication of OSAHS in children. A meta-analysis found a strong correlation between OSAHS and nocturia, and significant improvement in nocturia symptoms was found in treated children[19,20]. Su et al. had found that the risk of nocturia in children with OSAHS was increased markedly[21]. In our data, we found that the increased nocturia in moderate-severe OSAHS was particularly evident among the patients over 5 years old. Pearson correlation analysis had shown that nocturnal urination was positively correlated with hypoxia. This reminds us to pay special attention to the symptoms of increased nocturia in snoring children over 5 years old. Cardiopulmonary and renal reflex-induced neuroendocrine disorder may play an important role in the mechanism of night enuresis in children with OSAHS[22].

As we known, It was accepted that sleep architecture changed a lot in adult OSAHS patients. OSAHS initiates the sleep structure disorder, increase of shallow sleep, and decrease of deep sleep and REM sleep. For children, deep sleep is important for a variety of metabolic and endocrine processes, such as growth hormone secretion and other anabolic processes. Concludes of studies about OSAHS and sleep structure of children are inconsistent. Sun et al. found that the NREM3 sleep of school-age OSAHS patients was significantly reduced, and NREM1 sleep was prolonged[23]. In the study of durdik P et al., there were no significant differences in REM sleep, NREM2 sleep and sleep efficiency between the OSAHS and control group, but the NREM3 sleep of OSAHS children was significantly shorter, and the NREM1 sleep was significantly longer[24]. However, Goh DY found that children with *OSAHS* have normal sleep

stage distribution[25]. In our data, time ratio of NREM1 was elevated in Moderate-severe OSAHS patient, and REM was higher in mild OSAHS patients.

OSAHS in children is variable in different age groups. The school age group had significantly decreased proportion of NREM3 and REM, and increased AHI and ODI than the preschool one. It seems that the condition of elder children was much more serious, which is worthy of further exploring.

Limitation

Due to the limitation of sample size, no further analysis was made in different age. We will continue to collect cases.

Conclusion

We found that the median age of OSAHS in children was 5 (4, 7) years old. Compared with PS, snoring, dyspnea, apnea, oppressive wake, nocturia and daytime sleepiness were more common in OSAHS children. The school-age OSAHS patients seems to more serious than the preschoolers. Sleep structure disorder was found more often in children with OSAHS aged from 7 to 12 years old, mainly including shortening in the stage of NREM3 and REM. Children with snoring, apnea, dyspnea, adenoid hypertrophy and high level of CRP were more likely to be OSAHS patients. We hope to give clinicians better diagnosis basis.

Declarations

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Data availability All data generated or analyzed during this study are included in this published article.

Code availability N/A

Author Contributions Yuqing Wang and Xunwu Dou designed the study. Yanyu He and Xueyun Xu conducted and interpreted the statistical analysis and had the primary responsibility in the formation of the initial draft. All authors provided substantial contributions to the design of the work, or the acquisition, analysis, or interpretation of the data, revised the initial draft, approved the final manuscript, and agreed to be accountable for all aspects of the work.

Ethical approval The study protocol was approved by the Children's Hospital Affiliated to Soochow University (Approval number: 2020CS038).

Consent to participate N/A

Consent for publication N/A

Conflict of interest The authors declare that they have no conflict of interest.

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Figures

Figure 1

The age distribution of the OSAHS patients