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Obstructive sleep apnoea in children –a neglected entity

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ABSTRACT

Background: Obstructive sleep apnoea syndrome (OSAS) is a spectrum which includes primary snoring, upper airway resistance syndrome, obstructive hypoventilation and obstructive sleep apnoea. Sleep disordered breathing (SDB) is characterized by snoring, witnessed apnoea, frequent arousal, mouth breathing, restless sleep, irritability, recurrent upper respiratory tract infections etc.
Methods: This was a prospective observational study which included 20 children who presented to the otorhinolaryngology, pulmonary medicine, paediatric, oral and maxillofacial department were included in the study with clinical symptoms of obstructive sleep apnoea.
Results: 20 children, 13 (65%) males and 7 (35%) females were included. Snoring was the most common complaint.15 (75%) were obese, 5 had adenotonsillar hypertrophy(25%) as the risk factor for OSA. 15 out of the 20 children were graded under mallampati class 1(75%), 4 class 2(20%), 1under class 3(5%). There was no significant association in severity of OSA between 2 genders (p=0.82). Positive correlation (r=0.52) was found between AHI and BMI and is found to be statistically significant (p=0.02), which suggests that degree of obesity does predict the severity of OSA.
Conclusions: Obesity is the most significant risk factor among them followed by adenotonsillar hypertrophy. Hence children who snore should undergo polysomnography and necessary corrective measures should be prescribed.

Keywords: Paediatric obstructive sleep apnoea, Sleep disordered breathing

INTRODUCTION

Paediatric obstructive sleep apnoea (OSA) has been widely recognized as a cause of significant morbidity in children in the last few decades. Obstructive sleep apnoea syndrome (OSAS) is defined as a disorder of breathing during sleep characterized by prolonged airway obstruction and /or intermittent complete obstruction that interrupt normal ventilation during sleep and normal sleep pattern.[1,2] The spectrum of OSAS include primary snoring, upper airway resistance syndrome ,obstructive hypoventilation syndrome (Pickwickian Syndrome) and obstructive sleep apnoea syndrome. Sleep disordered breathing (SDB) in children is characterized by snoring, witnessed apnoea, frequent arousal, mouth breathing, restless sleep, irritability, recurrent upper respiratory tract infections etc.

Three factors which contribute to pathophysiology of SDB are anatomical variation, inflammation, neuromuscular tone. Children with SDB have a structurally narrow airway, increased collapsibility and resistance , during sleep there is further narrowing of the airway leading to reduced or absent airflow resulting in hypopnea and apnoea respectively.[3,4] Increased resistance to airflow can occur anywhere from nasopharynx to hypopharynx and can involve multiple sites. In children, adenotonsillar hypertrophy is the most significant factor.[5]

An anatomical obstruction at nasal and nasopharyngeal level in infants and children lead to chronic mouth breathing and affects the development of facial structures. With chronic mouth breathing, the tongue is unable to mould the palate and this results in narrow, high arched palate, poor maxillary development which can further narrow the airway, other anatomical abnormalities include retrognathic mandible with shorter maxilla and mandibular length ,large tongue, longer and thicker soft palate and more inferiorly placed hyoid bone.[6-8] Craniofacial anomalies are important in pathogenesis of OSA in non-obese children which may include Down syndrome, Pierre-Robin syndrome, cleft palate ,Treacher-Collin syndrome etc.

Untreated SDB can predispose to cardiovascular, neurocognitive complication and also disturbance of growth and mood. Cardiovascular complications probably due to sympathetic over activation, systemic hypertension, arrhythmias, heart failure etc. Increased risk of stroke, myocardial infarction which accounts for chronic intermittent hypoxia induced atherosclerosis, endothelial dysfunction, altered cerebral blood flow etc.[9] SDB can also affect short term concentration ability, short term memory. Increased work of breathing and load on heart require energy which may result in diminished weight and height gain in children.[10]

Despite of the recent advances in diagnosis and increased awareness of OSA, majority of those affected are still undiagnosed. Therefore, it is important to early recognize and treat the condition.

METHODS

This was a prospective observational study conducted in Justice K S Hegde Charitable Hospital attached to K S Hegde Medical Academy from January 2016 to June 2016, over a six month period. 20 children who presented to the otorhinolaryngology, pulmonary medicine, paediatric, oral and maxillofacial department were included in the study.

Inclusion criteria

Children who presented to us with clinical symptoms of OSA, who are under the age of 18, those who are willing to participate in the study.

Exclusion criteria

Patients who are 18 and above 18 years age, patients who had undergone previous craniofacial surgeries, patients receiving sedatives, patients who are not willing to participate.

Ethical clearance was obtained from institutional ethical committee board and written informed consent was taken.

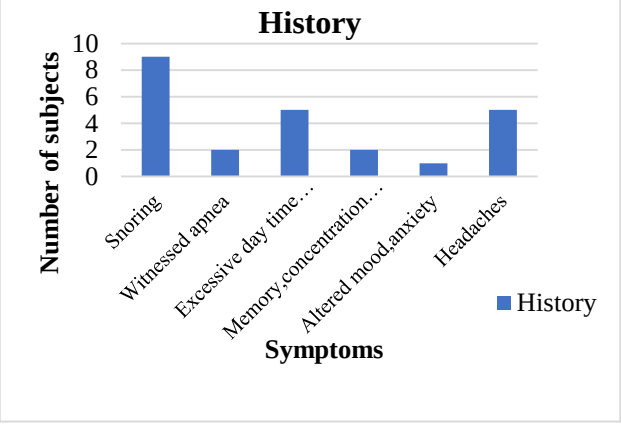
Serial paediatric age group patients who were undergoing sleep study (Polysomnography) were included to participate in the study. Patients having AHI (apnoea-hypopnea index)>5 were included in the analysis, based on AHI they were classified as mild, moderate and severe. The underlying causes of OSA were evaluated and corrected.

Laboratory polysomnography (PSG) was done for sleep study(Type 1 sleep study) . The following were monitored: central and occipital electroencephalogram (EEG), electrooculogram (EOG), submentalis electromyogram (EMG), nasal and oral airflow, thoracic and abdominal wall motion, anterior tibialis EMG, body position and electrocardiogram (ECG). Arterial oxygen saturation was monitored with a pulse oximeter. The tracing was scored using 30 second epochs. Hypopneas were scored per American Association of Sleep Medicine definition. Parameters to be evaluated in PSG include sleep state and stages, ventilator parameters like flow, effort and oxygen saturation. Severity of OSA is staged based on.

AHI	Grading
<5	Normal.
5-15	Mild OSA.
15-30	Moderate OSA.
>30	Severe OSA.

Staging of OSA

Obesity and overweight were defined using Body Mass Index (BMI) percentiles, BMI>97th percentile were considered as obese and BMI between 85th to 97th percentile were considered as overweight, they were marked using World Health Organisation(WHO) growth chart for children, charts were used separately for girls and boys.



Statistical analysis

The data obtained was entered in Microsoft excel and analysed using statistical package for social sciences SPSS software. A Fisher's exact test, student t test and Pearsons correlation test was used to analyse the

statistical significance of the data obtained. A p value of < 0.05 was considered as statistically significance.

RESULTS

Out of the 20 children under the study 9 of them (45%) of them had snoring symptoms, 2 of them (10%) had witnessed apnoea episodes as noticed by mother, 5 of them had excessive day time sleepiness(25%),2 had memory problems and learning difficulties(10%), 1 of them had mood related problems(5%), 5 of them complained about headaches(25%), 2 of them were habitual snorers (10%).

Table 1: Gender wise severity of OSA.

		Severity			Total	Fisher's Exact Test
		Mild OSA	Moderate OSA	Severe OSA.		P-value
Gender	F	5(38.5%)	2(40.0%)	0	7(35.0%)	0.82(NS)
	M	8(61.5%)	3(60.0%)	2(100.0%)	13(65.0%)	
Total		13(100.0%)	5(100.0%)	2(100.0%)	20(100.0%)	

*p<0.05 statistically significant, p>0.05 Non significant, NS.

Table 2: Severity of OSA in relation with BMI.

		Severity			Total	Fisher's Exact Test
		Mild OSA	Moderate OSA	Severe OSA.		P-value
BMI grade	Normal BMI	3(23.1%)	1(20.0%)	0	4(20.0%)	0.55(NS)
	Obese	3(23.1%)	3(60.0%)	2(100.0%)	8(40.0%)	
	Overweight	6(46.2%)	1(20.0%)	0	7(35.0%)	
	Underweight	1(7.7%)	0	0	1(5.0%)	
Total		13(100.0%)	5(100.0%)	2(100.0%)	20(100.0%)	

*p<0.05 statistically significant, p>0.05 Non significant, NS.

Table 3: Severity of OSA with age and anthrapometric measures.

	Severity of OSA	N	Mean	SD	Mean difference (95% CI)	T	df	p-value
Age(in yrs)	Mild	13	13.62	2.18	-0.10 (-2.08, 1.88)	-0.11	18	0.92(NS)
	Moderate/ severe	7	13.71	1.60				
BMI (kg/m2)	Mild	13	22.03	3.38	-2.90 (-6.02, 0.22)	-1.96	18	0.07(NS)
	Moderate/ severe	7	24.93	2.70				
Neck circumference (in cm)	Mild	13	31.23	4.63	-3.77 (-8.15, 0.62)	-1.81	18	0.09(NS)
	Moderate/ severe	7	35.00	4.08				
Abdominal girth (in cm)	Mild	13	60.85	3.58	-3.44 (-7.19, 0.31)	-1.93	18	0.07(NS)
	Moderate/ severe	7	64.29	4.23				
Thyromental distance(in cm)	Mild	13	5.615	0.33	0.14 (-0.26, 0.55)	0.75	18	0.46(NS)
	Moderate/ severe	7	5.471	0.53				

*Independent sample t test, p<0.05 statistically significant, p>0.05 Non significant, NS.

Out of 20 children under the study 15 of them were obese/ overweight (75%), 5 of them had adenotonsillar hypertrophy (25%) as the risk factor for OSA. 5 out of the 20 children had adenoid and / tonsillar enlargement on clinical examination (25%), one of the child had adenotonsillar hypertrophy (5%),4 of them had tonsillar enlargement (20%). 15 children were graded under mallampati class 1 (75%), 4 of them under class 2 (20%), one of them under class 3 (5%). Out of the 20 children 8 of them were obese (40%), 7 of them were overweight (35%), 4 of them were within normal BMI (20%), one of them was underweight (5%).

Out of 20 children, 13 (65%) of them were males and 7 (35%) of them were female. In severity of OSA's ,males are more likely to be affected as shown in the table, but when association between severity of OSA with gender tested by Fisher's exact test shows that there is no significant association in severity of OSA between 2 genders (p=0.82).

Among the children in mild OSA category 46.2% of them were overweight and 23.1% of them were obese. When tested by Fischer's exact test, no significant (p=0.55) association is found between BMI and OSA severity.

The severity of OSA is divided into mild and moderate/severe (as the number of severe OSA cases observed are minimum, this group is clubbed with moderate severity).

Table 4: Correlation between AHI and parameters studied.

		AHI
Age	R	0.14
	p-value	0.55(NS)
BMI (kg/m2)	R	0.52
	p-value	0.02*
Neck circumference (in cm)	R	0.49
	p-value	0.03*
Abdominal girth(in cm)	R	0.49
	p-value	0.03*
Thyromental distance(in cm)	R	-0.29
	p-value	0.20(NS)

*Pearsons correlation test, p <0.05 statistically significant, non significant, NS.

5 out of 20 children had adenotonsillar hypertrophy, 4 of them had mild OSA and one of them had moderate OSA. Hence moderate/severe OSA's clubbed together and Fischer's exact test for association is used and it is found that there is no significant association between the two (p=0.613) variables.

There was no significance difference between the mean age, BMI, neck circumference, abdominal girth, thyromental distance between the two groups mild OSA and mod/severe OSA.

There was no significant correlation (r=0.14) between age and AHI (p=0.55).There was a statistically significant positive correlation between AHI and other anthropometric parameters except thyromental distance. Positive correlation (r=0.52) was found between AHI and BMI and is found to be statistically significant (p=0.02), which suggests that degree of obesity does predict the severity of OSA .There is a negative correlation between AHI and thyromental distance (r=-0.29), but statistically not found to be significant (p=0.20).

DISCUSSION

Forty five percent of the children in our study had snoring as their main complaint .This is much higher than the comparative study by Chan J et al.[11] in which that 3-12% of the children had snoring as their main complaint and 1-10% of the children had 0SA.

In the study by Ersu et al, 10% of the children were found to be habitual snorer.[12] In our study, this value accounted for 7%.

Our study revealed that 10% of the children had memory and learning difficulties in their academics. This is in accordance with study done by Gozad et al, who reported that mid- school children who had low academic performance were likely to be snored in early childhood.[13]

Headache was the presenting symptom in 25% of the children .In their study, Miller et al, stated that migraine headache may also indicative of sleep disturbances.[14]

The mean age of children population in our study group was 13 years. The study done by Mallory et al.[15] on children and adolescents states that the mean age (SD) of the patient was 10.3 years.

Seventy five percent of the study population had overweight/ obesity which are a significant risk factor for OSA the study by Karla et al.[16] involving children of 2-18years old states that obesity was the most significant risk factor for OSAS.

In our study, neck circumference has positive correlation with AHI. Katz et al in their study on 6-17 year children stated that neck circumference >95th percentile for age and sex has got significantly increased risk of OSA [17]

The study reveals that the cases that are positive for SDB were in the order of obesity > adenotonsillar hypertrophy unlike the study conducted by Nanaware et al wherein positive SDB cases were in the order of craniofacial abnormalities > neuromuscular and skeletal disorders > adenotonsillar hypertrophy etc.[18]

Our study reveals that there is a positive correlation between AHI and obesity but the degree of obesity doesn't predict the severity of OSA in children which similar to study done by Tripuraneni et al.[19]

CONCLUSIONS

In our study, majority of the children who presented with snoring had coexistence of OSA. The presenting symptoms, in order of frequency of occurrence, were - excessive daytime sleepiness, headache, learning and memory deficit, witnessed apnoeas, mood related disturbances. Obesity was the most significant risk factor among them followed by adenotonsillar hypertrophy. The study also revealed that there is a positive correlation between obesity and Apnoea-Hypopnoea Index (AHI), which was statistically significant.

The study suggests that degree of obesity doesn't predict the severity of OSA. There is also a positive correlation between abdominal circumference and OSA.

Obesity is found to be a certain aggravating factor in children with OSA.

The results would have been more appropriate if the sample size was larger and if there is age and gender specific criteria for classifying severity of OSA.

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REFERENCES

1. American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med. 1996;153:866-78.
2. Medicine AAoS, editor. The international classification of sleep disorders, 2nd ed. Diagnostic and coding manual. Westchester, Illinois: American Academy of Sleep Medicine; 2005.
3. Gozal D, Burnside MM. Increased upper airway collapsibility in children with obstructive sleep apnoea during wakefulness. Am J Respir Crit Care Med. 2004;169:163-7.
4. Marcus CL, McColley SA, Carroll JL, Loughlin GM, Smith PL, Schwartz AR. Upper airway collapsibility in children with obstructive sleep apnoea syndrome. J Appl Physiol. 1994;77:918-24.
5. Arens R, McDonough JM, Costarino AT, Mahboubi S, Tayag-kier CE, Maislin G, et al. Magnetic resonance imaging of the upper airway structure of children with obstructive sleep apnoea syndrome. Am J Respir CritCare Med. 2001;164: 698-703.
6. Sullivan S, Li K, Guilleminault C. Nasal obstruction inchildren with sleep-disordered breathing. Ann Acad Med Singapore. 2008;37:645-8.
7. Peltomaki T. The effect of mode of breathing on craniofacial growth--revisited. Eur J Orthod. 2007; 9:426-9.
8. Johal A, Patel SI, Battagel JM. The relationship between craniofacial anatomy and obstructive sleep apnoea: a case controlled study. J Sleep Res. 2007;16:319-26.
9. Loffredo L, Zicari AM, Occasi F, Perri L, Carnevale R, Angelico F, et al. Endothelial dysfunction and oxidative stress in children with sleep disordered breathing: role of NADPH oxidase. Atherosclerosis. 2015;240(1):222-7.
10. Sinha D, Guilleminault C. Sleep disordered breathing in children. Indian J Med Res. 2010;131:311-20.
11. Chan J, Edman JC, Koltai PJ. Obstructive sleep apnea in children. Am Fam Physician. 2004;69:1147-54.
12. Ersu R, Arman AR, Save D, Karadag B, Karakoc F, Berkem M, Dagli E. Prevalence of snoring and symptoms of sleep-disordered breathing in primary school children in Istanbul. Chest. 2004;126:19-24.
13. Gozal D, Pope DW Jr. Snoring during early childhood and academic performance at ages 13 to 14 years. Pediatrics. 2001;107:1394-9.
14. Miller VA, Palermo TM, Powers SW, Scher MS, Hershey AD. Migraine headaches and sleep disturbances in children. Headache. 2003;43:362-8.
15. Mallory GB, Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. J Pediatr. 1989;115(6):892-7.
16. Narang I, Mathew JL. Childhood obesity and obstructive sleep apnea. J Nutr Metabol. 2012.
17. Katz S, Murto K, Barrowman N, Clarke J, Hoey L, Momoli F, Laberge R, Vaccani JP. Neck circumference percentile: a screening tool for pediatric obstructive sleep apnea. Pediatr Pulmonol. 2015;50(2):196-201.
18. Nanaware SK, Gothi D, Joshi JM. Sleep apnea. The Indian J Pediatr. 2006;73(7):597-601
19. Tripuraneni M, Paruthi S, Armbrecht ES, Mitchell RB. Obstructive sleep apnea in children. The Laryngoscope. 2013 May 1;123(5):1289-93.

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