Original Article

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Physiology Section

Polysomnographic Study to Evaluate Obstructive Sleep Apnoea Syndrome in Obese and Non Obese Adolescents

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ABSTRACT

Introduction: Obesity in adolescents is an emerging problem in developing countries like India, especially among higher socioeconomic status group. Obesity is the most important reversible risk factor for Obstructive Sleep Apnoea Syndrome (OSAS) in adolescents. Adolescent obesity with OSAS if not treated, can result in serious morbidity in cognitive, cardiovascular, somatic growth, development and metabolic disorders in future.

Aim: To compare the sleep pattern between the obese and non obese adolescents and to evaluate OSAS.

Materials and Methods: This was an observational study carried out at Life Style Laboratory, Department of Physiology, Bangalore Medical College and Research Institute, Karnataka, India. The study involved 30 obese and 30 non obese male adolescents, who were subjected to overnight Polysomnography (PSG) in the sleep laboratory. According to Kale's criteria, epochs were manually scored which were compiled and statistically analysed for parameters like Sleep Latency (SL), Actual Sleep Time (AST), wake after sleep onset, percentage of Non Rapid Eye Movement (NREM), Rapid Eye Movement (REM) sleep stages, Sleep Efficiency (SE). The number of apnoeas and hypopnoeas were also noted to calculate Apnoea Hypopnoea Index (AHI). These parameters were compared for statistical significance using student t-test. Adolescents with AHI ≥1 were diagnosed with OSAS.

Results: Mean age of obese adolescents was 17.7±0.97 years and their mean BMI was 28±0.73 kg/m^2. OSAS was found in 22 out of 30 obese (73%) and 14 out of 30 (46%) non obese adolescents. It was found that apnoeas (3±4.80 vs 1±0.89), hypopnoeas (27.36±26.5 vs 5.46±2.97) and AHI (4.17±3.90 vs 0.89±0.43) were significantly more among obese adolescents when compared with non obese adolescents respectively. PSG parameters like SL, Wake After Sleep Onset (WASO) were prolonged and AST, SE were reduced significantly in obese adolescents.

Conclusion: Adolescents with obesity had greater occurrence of OSAS, along with altered sleep architecture in them.

Keywords: Apnoea hypopnoea index, Body mass index, Sleep efficiency, Sleep latency, Wake after sleep

INTRODUCTION

Obesity is becoming a global pandemic in both industrialised and developing countries. In India, obesity is emerging as an important health problem particularly in urban areas, especially among the affluent class [1]. Over the last decade, the prevalence of obesity in children and adolescents has increased dramatically in many countries. The combined prevalence of 16% of childhood overweight and obesity reported in 2001 has significantly increased to 19.3% after 2010 [2]. School-based data from India indicate a prevalence of obesity to be 5.6% and 24% in children and adolescents, respectively [3]. This rise in obesity has caused concern on both rates and severity of sleep related breathing disorders, most notably obstructive sleep apnoea syndrome.

The OSAS is referred as frequent episodes of partial or complete obstruction of upper airway leading to frequent nocturnal awakening due to hypoxia. In case of obese children, OSAS independently causes cardiovascular and metabolic dysfunction, along with a wide range of cognitive, behavior and social problems [4,5]. The reported association between OSAS and body mass among children appears to vary according to factors such as ethnicity, the presence of adeno-tonsillar hypertrophy and, socioeconomic status [6-8].

The prevalence of upper airway obstruction among adolescents due to obesity may be much greater [9,10]. Reduction in upper airway tone and changes in anatomical structures may play a significant mediating role in any change in association between body mass and OSAS [11]. Obesity is the most important reversible risk factor for OSAS, with an estimated 50% prevalence of OSAS among western population with morbid obesity [12,13]. Few studies have shown a high index of suspicion for OSAS when evaluating obese adolescents, and have suggested PSG for these patients [14,15]. Adolescent obesity with OSAS if not treated, can result in serious morbidity in cognitive, cardiovascular, somatic growth and development and metabolic disorders in future. In light of increasing prevalence of adolescent obesity in India [2], it is particularly important to determine whether obesity in adolescent would precipitate OSAS and also alter the sleep pattern and educate them regarding consequences and complications of obesity to emphasise to adopt changes in lifestyle. Hence an attempt has been made to study the sleep pattern and evaluate OSAS in obese adolescents.

MATERIALS AND METHODS

This observational study was carried out from 1st August 2013 to 31st January 2015 at the Life Style Laboratary, Department of Physiology, Bangalore Medical College and Research Institute, Karnataka, India after getting approval from the ethical committee. Informed consent were taken from the parents and assent from the subjects.

Inclusion criteria: Study comprised of 30 obese and 30 non obese adolescents who were selected randomly from the community. Healthy male subjects in the age group of 10-19 years, with BMI ≥95th percentile (obese) and ≤85th percentile (non obese) for age and sex [16].

Exclusion criteria: Subjects with adenoid hypertrophy, muscular dystrophy, hypothyroidism, acromegaly, laryngomalacia, habit of smoking and, on drugs disrupting sleep and impact on respiratory dynamics (alcohol, narcotics, sedative hypnotics).

Study Procedure

Selected subjects were explained about the nature of the proposed study and written consent was obtained from each of them. Following all the sleep laboratory guidelines, individuals were subjected to

overnight PSG. The recording was done using RMS-PSG software on Microsoft window-based computer during the subject's normal habitual sleeping hours, under video monitored supervision. The following standard parameters were measured which was inclusive of Electroencephalogram (EEG; C3-A2 or C4-A1), left and right Electrooculogram (EOG), submental electromyogram (EMG) with skin surface electrodes, leg movements by motion detectors, heart rate by Electrocardiogram (ECG), respiratory movements of the chest using uncalibrated respiratory inductive plethysmography, arterial Oxygen Saturation (SpO_2) by pulse oximetry. The recordings were scored manually epoch by epoch of 30 second each according to Retchschaffen and Kale's manual [17].

Obstructive apnoeas were defined as the absence of airflow associated with continued chest and abdominal wall movement for duration of two or more respiratory cycles [11]. Obstructive hypopnoeas was defined as a 50% reduction in airflow signal associated with paradoxical chest/abdominal wall movement for duration of two or more respiratory cycles and followed with a 3% or greater oxygen desaturation [18]. The Obstructive Apnoea and Hypopnoea Index (OAHI) were calculated as the total number of obstructive apnoeas and obstructive hypopnoeas, divided by the total sleep time and expressed as the number of events per hour of sleep. An OAHI of one or greater was considered indicative of OSAS [11].

STATISTICAL ANALYSIS

Categorical data were compared between the groups using the Student's t-test. Data were analysed using Statistical Package for the Social Sciences (SPSS) software program, version 15.0. A value of $p<0.05$ was considered statistically significant.

RESULTS

It is evident from the [Table/Fig-1] that BMI significantly differed between the subjects but not the age of the participants. OSAS was found in 22 out of 30 obese (73%) and 14 out of 30 (46%) non obese adolescents. The [Table/Fig-2] shows that mean Oxygen Saturation (SpO_2) was reduced and apnoeas, hypopnoeas, AHI were increased significantly in obese group.

Variables	Obese	Non obese	p-value
Age (Years)	17.7±0.97	17.66±0.86	0.89
Weight (Kg)	78.7±6.53	63.2±4.94	<0.001
BMI (Kg/m^2)	28±0.73	21.85±1.6	<0.001

[Table/Fig-1]: Comparison of basic characters obese and non obese adolescents. p-value was calculated by Student t-test

Study variables	Obese	Non obese	Difference	t-value	p-value
SpO_2 (%)	95.5±0.95	96.3±0.73	0.8	+3.57	<0.001
Apnoea	3±4.80	1±0.89	-1.96	-2.16	0.03
Hypopnoea	27.36±26.5	5.46±2.97	-20.26	-4.04	<0.001
AHI	4.17±3.90	0.89±0.43	-3.06	-4.16	<0.001

[Table/Fig-2]: Comparision of parameters in obese and non obese adolescents.

[Table/Fig-3] shows that SL, WASO were prolonged and AST, SE were reduced in obese adolescents. Although REM sleep and SWS was reduced among obese adolescents it was not statistically significant [Table/Fig-4].

Study variables	Obese	Non obese	Difference	t-value	p-value
Total sleep time (min)	424.96±31.93	440.41±51	-15.45	-1.38	0.17
SL (min)	28.46±4.22	20.13±5.26	8.33	+6.65	<0.001
AST (min)	374.6±27.76	409.21±50.01	-34.61	-3.26	0.001
WASO (min)	22.1±8.37	10.86±6.99	11.23	+5.54	<0.001
NREM sleep %					
Stage 1	7±1.14	6.54±0.95	0.45	+1.65	0.10
Stage 2	47.71±1.12	47.25±1.39	0.46	+1.41	0.16
Stage 3	6.80±0.69	7.01±0.62	-0.21	-1.24	0.21
Stage 4	15.05±0.95	15.41±0.86	-0.35	-1.47	0.14
SE (%)	88.17±1.92	92.67±2.14	-4.49	-8.4	<0.001

[Table/Fig-3]: Comparison of PSG parameters other than AHI in obese and non obese adolescents.
SL: Sleep latency; WASO: Wake after sleep onset; AST: Actual sleep time; NREM: Non rapid eye movement sleep; SE: Sleep efficiency

Variables	Obese	Non obese	Difference	t-value	p-value
REM%	23.19±1.04	23.69±2.13	-0.49	-1.13	0.26
SWS%	21.86±1.12	22.42±1.21	-0.56	-1.84	0.07

[Table/Fig-4]: Comparison of Rapid Eye Movement (REM) and Slow Wave Sleep (SWS) in obese and Non obese adolescents

DISCUSSION

The present study intended to evaluate OSAS in obese adolescents and to compare sleep pattern between obese and non obese adolescents. PSG was done on 30 obese and 30 non obese adolescents. The present study showed that the average SpO_2 was reduced and apnoeas, hypopnoeas, AHI are increased significantly in obese group. This study is in agreement with similar other studies that showed increased prevalence of OSAS in obese adolescents with reduced SpO_2 and increased apnoeas, Hypopnoeas and AHI [11,19,20].

In obese adolescents, disordered breathing during sleep is a hallmark of OSAS. Breathing abnormalities include apnoea (cessation of air flow) and hypopnoea (decreased air flow). Upper airway obstruction may occur at one or more levels, including the nasopharynx, mouth, velopharynx (space behind the palate), retroglossal region (area behind the tongue), hypopharynx (region between the tongue base and larynx), and larynx. The upper airway is a pliant tube whose side walls consist of muscle and other soft tissues. During wakefulness, neural input to a number of small muscle groups in the pharynx maintains muscle tone and airway patency. With sleep, an increased resistance to airflow normally accompanies muscular relaxation of these muscle groups [21].

In obese individuals, there is excessive deposition of fat tissue within the muscles and tissue surrounding the upper airway leads to reduced airway size and increased airway resistance [11,22]. Reduced lung volumes and decreased central ventilatory drive also contribute to compromised upper airway patency [23].

The analysis of other PSG parameters in this study showed a prolonged SL, WASO and reduced AST, SE in obese adolescents. Similar studies compared obese adolescents with healthy controls using actigraphy, PSG. The findings showed that obese participants had later sleep onset, shorter sleep time, and more disrupted sleep than controls [24-26].

In normal subjects, waking and Stage 1 NREM sleep accompany increased cortisol level whereas in SWS there is modest inhibition of Hypothalamo-Pitutary-Adrenal (HPA) axis leading to decreased level of plasma cortisol [27,28]. In OSAS, because of frequent nocturnal awakenings and decreased SWS, this inhibition is reduced or lost resulting in increased plasma cortisol level which further disrupts the sleep architecture [29].

The OSAS is also associated with recurrent episodes of apnoeas and hypopnoeas, leading to hypoxaemia and stimulation of sympathetic nervous activity which in turn activates the norepinephrine projections [29]. This leads to arousals, sleeplessness as reflected in increased WASO and decreased sleep duration and SE.

The present study also found that REM sleep percentage and SWS were reduced among obese adolescents, though there was no statistical difference with the non obese group. This can

be attributed to increased tendency of upper airway obstruction during REM sleep in obese individuals due to decreased tone of muscles which leads to frequent arousals. These findings are similar to certain other studies where the REM sleep and the quantity of deep NREM sleep referred as SWS were reduced due to frequent sleep disruption [29,30].

Limitation(s)

The sample size was limited and the study population did not include females.

CONCLUSION(S)

Obese adolescents had very less SpO_2 with increased apnoeas, hypopnoeas, Apnoea Hypopnoea Index. PSG parameters like SL, WASO were prolonged, while AST, SE were reduced in obese adolescents. Though REM sleep percentage and SWS percentage were reduced in obese adolescents when compared to normal weight adolescents but they were not statistically significant.

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