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Is Metabolic Syndrome Associated with Obstructive Sleep Apnea in Obese Adolescents?

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Objective: To investigate whether there is an association between metabolic syndrome and obstructive sleep apnea syndrome (OSAS) in obese adolescents.

Methods: In total, 240 pubertal children or prepubertal children older than 11 y recruited consecutively from the pediatric endocrinology unit, obesity clinic. Patients with tonsillar and adenoid hypertrophy (grade 3/4), systemic illnesses, or chronic drug usage were excluded. After anthropometric measurement and laboratory study, patients were divided into two groups according to metabolic syndrome (MS): MS and non-MS. Overnight polysomnographic evaluation was performed and 104 subjects were included for statistical analysis. The two groups were compared in terms of sleep efficiency, number of awakenings per night, oxygen desaturation index, snoring time, and obstructive/central/mixed apnea-hypopnea index (AHI).

Results: Of the obese adolescents, 51 had MS and 53 did not. The AHI was ≥ 1 in 25 of the 53 non-MS children (47.2%) and in 25 of the 51 MS children (49%). The median obstructive AHI value was 0.9 (0.2–2.4) and total AHI was 0.9 (0.2–2.5) in the MS group; these values were 0.9 (0.25–3.55) and 0.9 (0.3–3.55), respectively, in the non-MS group. Obstructive, central, mixed, and total AHI values in the MS and non-MS groups were not statistically significantly different ($p > 0.05$).

Conclusions: In our study, we did not find an association between MS and sleep apnea in obese adolescents.

Keywords: metabolic syndrome, obesity, pediatric sleep apnea syndrome

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Obesity is an important public health problem and its prevalence increases seemingly day by day. The World Health Organization has described obesity as "The most ignored public health problem."[1] Its prevalence has increased 2.3–3.3-fold in United States over the past 25 years and 2.0–2.8-fold in England over the past 10 years.[2]

Obese children face many health problems, with physiological, neurological, pulmonary, cardiovascular, and endocrine system issues. Sleep disorders, especially OSAS, are also a problem in this population. Although pharyngeal airway fat deposition has been blamed for the occurrence of OSAS, the exact mechanism remains unclear. It is clear that repeated airway occlusions result in sleep disorders, and cyclic oxyhemoglobin desaturation and hypercapnia.[3,4] OSAS occurs in 2% of the general pediatric population[5,6] and up to one-third of obese children.[3,7–9] Obesity increases the risk of OSAS in children by 4.5-fold. Furthermore, the severity of OSAS is directly proportional to the degree of obesity.[5]

Metabolic syndrome is an appreciable issue in obesity and includes features of hypertriglyceridemia, low high-density lipoprotein (HDL) cholesterol levels, hyperglycemia, insulin resistance, diabetes mellitus (DM), abdominal obesity, hypertension, and prothrombotic-proinflammatory conditions.[10–12] To the best of our knowledge, no prospective randomized controlled study has assessed relationship between MS and OSAS in obese adolescents. Thus, in this study, we investigated the relation between MS and OSAS in obese adolescents by using polysomnography, the gold standard diagnostic method for sleep disorders.

BRIEF SUMMARY

Current Knowledge/Study Rationale: The literature about the relationship between obstructive sleep apnea syndrome (OSAS) and metabolic syndrome (MS) in obese adolescents is limited and inconsistent. The aim of this study was to investigate whether there is an association between MS and OSAS in obese adolescents.

Study Impact: We found no reported study that had investigated the effect of MS alone excluding the independent risk factor of obesity by examining study and control groups from obese adolescents. This prospective, randomized controlled study did not find a relation between MS and OSAS in obese adolescents.

MATERIALS AND METHODS

Subjects

Pubertal children or prepubertal children older than 11 y of age were recruited consecutively from the pediatric

Table 1—Tonsil and adenoid tissue size evaluation.

Tonsil size evaluation	
Grade 1	Tonsils are in the tonsillar fossa and hardly seen behind the anterior tonsillar plica.
Grade 2	Tonsils are seen clearly behind anterior tonsillar plica.
Grade 3	Tonsils reach three-fourths of the middle line.
Grade 4	Tonsils reach the middle line, closing the airway completely (also referred to as "kissing tonsils").
Adenoid tissue size evaluation	
Grade 1	Adenoid tissue does not touch other tissues.
Grade 2	Adenoid tissue touches the torus tubarius.
Grade 3	Adenoid tissue touches the torus tubarius and vomer.
Grade 4	Adenoid tissue touches the torus tubarius, vomer, and soft palate.

From Tagaya et al.[13]

Table 2—Metabolic syndrome criteria.

Criteria for metabolic syndrome
a. Hypertension (systolic ≥ 130 / diastolic ≥ 85 mm Hg)
b. Triglycerides > 150 mg/dL
c. HDL-cholesterol < 40 mg/dL
d. Impaired fasting glucose (fasting blood glucose ≥ 100 mg/dL) or impaired glucose tolerance (2-h oral glucose tolerance test, blood glucose 140–199 mg/dL) or type 2 diabetes mellitus.
e. Race/ethnicity-specific waist circumference > for age is over the 90th percentile.[17]

Subjects with at least three of these five criteria were included in the Metabolic Syndrome group.[18]

endocrinology unit, obesity clinic at a tertiary hospital between March 1, 2013 and March 1, 2014. The Bakirkoy Dr Sadi Konuk Training and Research Hospital Ethics Committee approved the study. Informed consent was obtained from the parents of each subject.

Clinical records of 240 patients were evaluated and patients who had systemic illnesses, endocrine diseases (e.g., Cushing syndrome), chronic lung diseases (e.g., asthma, sequelae of tuberculosis, cystic fibrosis), hereditary transmitted anemia (e.g., sickle cell anemia, thalassemia), craniofacial anomalies, neuromuscular diseases, and all genetic diseases, such as Down syndrome and Prader-Willi syndrome were not called for further assessment. Patients who were using drugs for obesity or other illnesses were also excluded. Children who were not eliminated by exclusion criteria and whose parents agreed to participate were called for otolaryngologic examination.

Otolaryngologic Examination

Both tonsil and adenoid tissue sizes were graded (**Table 1**) by an otolaryngologist.[13] Patients with tonsillar or adenoid hypertrophy (grade 3 and 4) were eliminated from the study.

Anthropometry

Waist circumference of the patients was obtained using an inelastic cloth measuring tape around the area of greatest girth of the abdomen. Their weight and standing height were measured with a calibrated weighing scale and stadiometer, respectively. Body mass index (BMI) was calculated as weight/height^2 (kg/m^2). Blood pressure was obtained using a calibrated sphygmomanometer. All anthropometrically analyzed subjects' BMI were bigger than 2 standard deviations (SD) and they continued to next step.

Laboratory Study

All subjects had blood samples taken in the morning, after an overnight fast, for the estimation of plasma glucose, serum insulin concentrations, and lipid profile (total cholesterol, triglyceride, HDL cholesterol, and low-density lipoprotein (LDL) cholesterol concentrations). Subsequently, a 2-h oral glucose tolerance test was performed.

According to the "Metabolic Syndrome Criteria, International Diabetes Federation, 2005," (**Table 2**) the obese adolescents were divided into two groups, MS and non-MS.[14,15]

Overnight Polysomnography

One hundred fifty-seven obese children were assessed for sleep study 2 months after starting the study.

A polysomnographic evaluation was performed using the American Thoracic Society's "Standards and Indications for Cardiopulmonary Sleep Studies in Children".[16] Children were assessed for 8 h in a quiet, darkened room with an ambient temperature of 24°C, in the company of one of their parents. No drug was used to induce sleep. Polysomnography (PSG) was started at the subject's regular night sleep time. Sleep parameters were evaluated using 12-channel PSG. We recorded the electroencephalogram (EEG), electrooculogram (EOG), submental, electromyelogram (EMG), thoracic, and abdominal wall movement-guided respiratory measurement bands, nasal airflow, and oxyhemoglobin saturation (SpO_2; Ohmeda Biox, Louisville, CO, USA).

Sleep study data were analyzed digitally (Remlogic software, Broomfield, CO, USA). During the analysis, children who slept over 2 hours or had at least one rapid eye movement (REM) sleep period were included. Thirty-two children could not fulfill sleep inclusion criteria and 21 children did not want to join sleep overnight PSG.

AHI ≥ 1 was defined as OSAS.[16–18]

Both the PSG technician and the PSG reading physician were unaware of the patients' group assignments.

The two groups (MS and non-MS) were compared in terms of sleep efficiency, number of awakenings per night, oxygen desaturation index, snoring time, and obstructive/central/mixed AHI.

Statistical Analysis

All data were managed and analyzed using the Number Cruncher Statistical System (NCSS) 2007 software (Kaysville, Utah, USA). Data were assessed by descriptive statistical methods (average, standard deviation, median, interquartile range) and by comparative statistical methods (for normally distributed variables, we used the independent *t*-test; for non-normally distributed variables, we used the Mann-Whitney *U*-test). A p value < 0.05 was considered to indicate statistical significance.

RESULTS

In total, 51 obese adolescents had MS and 53 did not. The average ages were 13.4 ± 1.4 y in the non-MS group and 13 ± 1.75 y in the MS group.

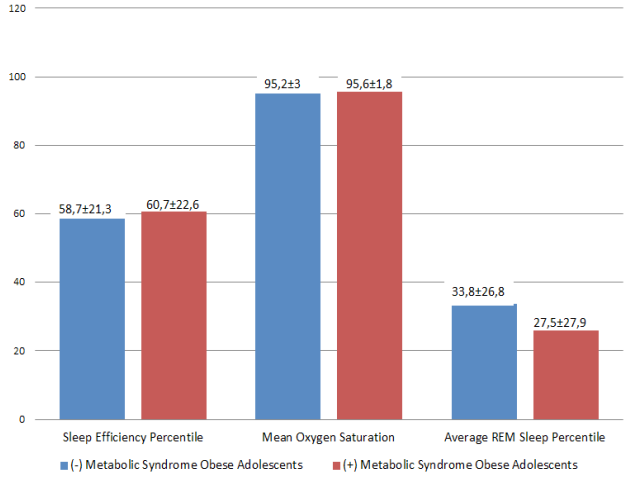
The average sleep time was 268.95 ± 109 min in the MS group and 259.17 ± 96.13 min in the non-MS group. Sleep efficiency was 60.74 ± 22.64% in the MS group and 58.71 ± 21.31% in the non-MS group (**Figure 1**). The number of awakenings per night was 8.9 ± 7.26 in the MS group and 10.85 ± 8.62 in the non-MS group. Total sleep recording time, total sleep time, sleep efficiency, and number of awakenings in the MS and the non-MS groups were not statistically significantly different (p > 0.05).

The REM sleep percentile was 27.5 ± 27.95 in the MS group and 33.82 ± 26.86 in the non-MS group (**Figure 1**). Total nonrapid eye movement (NREM) time, NREM percentile, Stage R latency from sleep onset, and REM percentile in MS and non-MS group were also not statistically significantly different (p > 0.05).

The AHI was ≥ 1 in 25 of 53 children (47.2%) in the non-MS group and 25 of 51 children (49%) in the MS group. AHI was ≥ 1 in 50 of the total 104 (MS and non-MS) children (48.1%). AHI was ≥ 5 in 6 of 53 children (11.3%) in the non-MS group, 5 of 51 children (9.8%) in the MS group, and 11 of the total 104 (MS and non-MS) children (10.57%). The median obstructive AHI value was 0.9 (0.2–2.4) and the total AHI value was 0.9 (0.2–2.5) in the MS group; the corresponding values were 0.9 (0.25–3.55) and 0.9 (0.3–3.55), respectively, in the non-MS group (**Table 3**, **Figure 2**). Obstructive, central, mixed, and total AHI rates in the MS and non-MS groups were not statistically significantly different. Comparison according to AHI ≥ 5 criteria in the MS and non-MS groups was not also significantly different (p > 0.05).

The numbers of oxygen desaturation events in 1 h were 1.4 (0.3–4.2) in the MS group and 1.5 (0.35–6.5) in the non-MS

Figure 1



Sleep efficiency percentile, mean oxygen saturation, and average rapid eye movement (REM) sleep percentile in (−) metabolic syndrome (MS) and (+) MS obese adolescents.

Table 3—Obstructive, central, and mixed apnea-hypopnea index value of metabolic syndrome (+) and metabolic syndrome (−) obese adolescents.

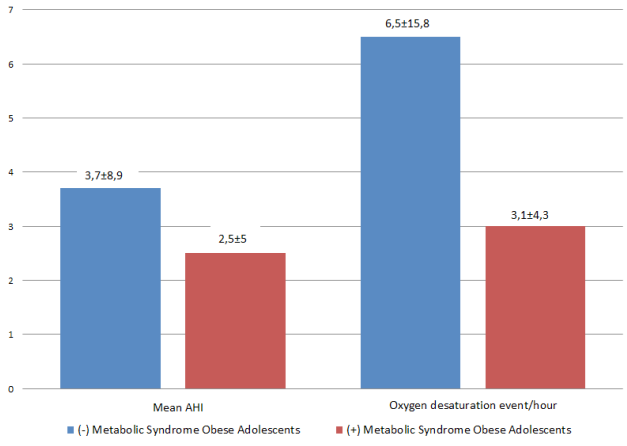
		MS (−) OSAS	MS (+) OSAS	p
AHI Obstructive	Average ± SD	3.73 ± 8.9	2.52 ± 4.96	0.652
	Median (IQR)	0.9 (0.25–3.55)	0.9 (0.2–2.4)	
AHI Central	Average ± SD	0 ± 0.01	0 ± 0	0.327
	Median (IQR)	0 (0–0)	0 (0–0)	
AHI Mixed	Average ± SD	0.02 ± 0.09	0.02 ± 0.11	0.706
	Median (IQR)	0 (0–0)	0 (0–0)	
AHI Total	Average ± SD	3.75 ± 8.89	2.54 ± 4.97	0.611
	Median (IQR)	0.9 (0.3–3.55)	0.9 (0.2–2.5)	

AHI, apnea-hypopnea index; IQR, interquartile range; MS, metabolic syndrome; OSAS, obstructive sleep apnea syndrome; SD, standard deviation.

group (**Figure 2**). Mean oxygen saturation was 95.66 ± 1.86 in the MS group and 95.2 ± 3.08 in the non-MS group (**Figure 1**). The minimum oxygen saturation was 89.1 ± 4.77 in the MS group and 88.11 ± 8.14 in the non-MS group. The mean desaturation event number was 4.65 (4.23–5.4) in the MS group and 4.5 (4–5.25) in the non-MS group (**Table 4**). Oxygen desaturation events, oxygen desaturation events/h, lowest oxygen desaturation, mean oxygen saturation, and average oxygen desaturation in the MS and non-MS groups were not statistically significantly different (all p > 0.05).

The snoring percentile was 3 (0.2–15.78) in the MS group and 3 (0.55–18.75) in the non-MS group (**Table 5**). Snoring time and relative snoring time in the MS and non-MS groups were not statistically significantly different (both p > 0.05).

Figure 2



Mean apnea-hypopnea index (AHI) and oxygen desaturation event/h value of (−) metabolic syndrome (MS) and (+) MS obese adolescents.

DISCUSSION

The prevalence of obesity has increased OSAS recently. From early infancy to the late childhood period, obesity-induced OSAS may be seen.[19–23]

Marcus and colleagues[21] found sleep disorders in 8 of 22 obese children (36%) (weight > 120% and subscapular skin fold > 85th percentile). They found a direct connection between apnea index and obesity. Kalra et al.[24] found the frequency of OSAS in morbidly obese patients to be 55%.

In the current study, 50 of 104 obese patients (48.1%) were found to have OSAS in the polysomnographic evaluation. According to these data, obesity carries a high risk for developing OSAS. Reasons for the differing results in the various studies may include variations in ethnicity and use of different diagnostic methods and criteria for OSAS and obesity.[5,21,24]

Metabolic syndrome is frequent in obese children; it includes hypertriglyceridemia, low HDL cholesterol, hyperglycemia, insulin resistance, diabetes mellitus, abdominal obesity, hypertension, and prothrombotic-proinflammatory conditions.[10–12] The prevalence of MS increases with the severity of obesity and reaches 50% in morbidly obese patients.[25]

Obesity is closely associated with OSAS[26,27] and MS.[25] However, studies of the association between MS and OSAS have reported controversial results. In a community-based study, Redline et al.[28] found a relationship between MS and OSAS. The probability of elevated metabolic markers was sevenfold higher in adolescents with sleep and respiratory disorders compared with normal adolescents. Blood pressure,

Table 4—Oxygen saturation of metabolic syndrome (+) and metabolic syndrome (−) obese adolescents.

		MS (−) OSAS	MS (+) OSAS	p
Oxygen Desaturation event	Average ± SD	29.64 ± 72.41	15.27 ± 32.52	0.508
	Median (IQR)	5 (1–28)	5 (1–20)	
Oxygen Desaturation event/h	Average ± SD	6.52 ± 15.86	3.05 ± 4.3	0.737
	Median (IQR)	1.5 (0.35–6.5)	1.4 (0.3–4.2)	
Average Oxygen saturation	Average ± SD	95.2 ± 3.08	95.66 ± 1.86	0.621
Lowest Oxygen saturation	Average ± SD	88.11 ± 8.14	89.1 ± 4.77	0.868
Average desaturation	Average ± SD	4.45 ± 2.21	4.62 ± 1.29	0.423
	Median (IQR)	4.5 (4–5.25)	4.65 (4.23–5.4)	

IQR, interquartile range; MS, metabolic syndrome; OSAS, obstructive sleep apnea syndrome; SD, standard deviation.

Table 5—Snoring time in metabolic syndrome (+) and metabolic syndrome (−) obese adolescents.

		MS (−) OSAS	MS (+) OSAS	p
Snoring time and relative snoring time	Average ± SD	23.4 ± 37.91	25.46 ± 31.96	0.898
	Median (IQR)	7 (1.25–30.4)	5.65 (0.75–46)	
% snoring time and relative snoring time	Average ± SD	10.21 ± 15.86	9.07 ± 10.64	0.746
	Median (IQR)	3 (0.55–18.75)	3 (0.2–15.78)	

IQR, interquartile range; MS, metabolic syndrome; OSAS, obstructive sleep apnea syndrome; SD, standard deviation.

insulin, and LDL cholesterol level were also higher in adolescents with sleep and respiratory disorders. However, in that study, one of the most important causes of OSAS, tonsillar and adenoid hypertrophy, was ignored. The subjects were also heterogeneous for MS and obesity. A small number of patients met the criteria for MS and overweight (approximately 25% of the sample was overweight and 19% met the criteria for MS). The BMIs of many patients in the MS group were already ≥ 95th percentile and whether the sleep/breathing disorders arose from MS or obesity was unclear.[28] Gozal et al.[29] did assess the effects of tonsillectomy and adenoidectomy on OSAS and metabolic markers in obese and nonobese children.[29] After surgery, C-reactive protein, Apolipoprotein B and LDL cholesterol levels decreased and HDL cholesterol levels increased in nonobese children. Triglycerides, total cholesterol, insulin, and glucose levels did not change significantly in that group. C-reactive protein, ApoB, LDL cholesterol, triglycerides, and insulin levels decreased, and HDL cholesterol levels increased after surgery in obese children. In this study, it was shown that tonsillectomy and adenoidectomy positively affected metabolic markers in children with OSAS but the effect of metabolic markers on pediatric OSAS was not determined preoperatively. Also, in this study, metabolic markers were assessed on their own, without reference to MS.[29] According to the study, OSAS treatment positively affected the systemic inflammation marker, C-reactive protein, in each patient group.[29] Although some studies have supported an association between OSAS and C-reactive protein,[30,31] others have not.[32,33] A study in China carried out by Li et al.[17] showed an increase in incidence of OSAS and insulin levels in obese adolescents. However, in that study whether insulin resistance caused further elevation of AHI was not determined. Tonsil and adenoid hypertrophy was not analyzed and metabolic markers were not assessed in the context of MS.[15] In contrast with those studies, Tauman et al.[31] found an association between BMI and AHI in children but no association among AHI and serum total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride levels. Katidis et al.[32] found an association between AHI and tonsil size in patients with similar BMI but no association between AHI and C-reactive protein, serum triglycerides, or cholesterol levels. Stefanini et al.[34] found no difference between nonobese children with OSAS and nonobese children without OSAS in terms of metabolic tests and blood pressure.

In our study, we excluded the most important OSAS causes, such as tonsil and adenoid hypertrophy and craniofacial and endocrine pathologies. By including solely obese patients in both groups, we also attempted to exclude the effects of obesity to identify any independent effect of MS on OSAS. In this obese patient population, the study group had MS and the control group did not. We found that MS was not associated with AHI, oxygen desaturation time and number, snoring time and percentile, total sleep time, REM and NREM sleep time, sleep efficiency, or wake-up number.

Although obesity may cause both MS and OSAS, we found no association between MS and OSAS in obese adolescents. However, our study was of a cross-sectional design and it is unclear which parameters will change during follow-up of these patients. Randomized controlled, longitudinal follow-up studies with larger numbers of patients are needed to further examine the relationship between OSAS and MS.

ABBREVIATIONS

AHI, apnea-hypopnea index
BMI, body mass index
DM, diabetes mellitus
EEG, electroencephalogram
EOG, electrooculogram
EMG, electromyelogram
HDL, high-density lipoprotein
LDL, low-density lipoprotein
MS, metabolic syndrome
NREM, nonrapid eye movement
OSAS, obstructive sleep apnea syndrome
PSG, polysomnography
REM, rapid eye movement
SD, standard deviations

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DISCLOSURE STATEMENT

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