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Screening for Sleep Disordered Breathing and Excessive Daytime Sleepiness in Adolescent Girls with Polycystic Ovarian Syndrome

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Abstract

Objective—To determine the prevalence and clinical and metabolic correlates of sleep disordered breathing (SDB) and excessive daytime sleepiness (EDS) in adolescent girls with polycystic ovarian syndrome (PCOS).

Study design—Standardized questionnaires were administered to subjects with PCOS and age-, sex-, ethnicity-, and BMI Z-score-matched controls. Medical records were reviewed for anthropometric and metabolic data.

Results—We studied 103 subjects with PCOS (16.9±1.5 years) and 90 controls (16.8±1.7 years). Compared with controls, girls with PCOS had a higher prevalence of SDB (45.6% vs. 27.8%, p=0.01) and EDS (54.4% vs.35.6%, p<0.01). Within PCOS, those with SDB had higher BMI Z-score (2.1±0.5 vs.1.7±0.6, p< 0.01), higher homeostatic model assessment (HOMA) (5.1±2.3 vs. 4.1±3.5, p<0.01), and higher prevalence of the metabolic syndrome (MetS) (42.6% vs. 16.1%, p=0.003), compared with those without SDB. Similarly, subjects with PCOS and EDS had higher BMI-Z score (2.0±0.6 vs.1.7±0.6, p=0.03), higher HOMA (5.1±2.9 vs.3.8±3.1, p=0.01), and higher rate of MetS (39.3% vs. 14.9% p<0.01), compared with those without EDS. MetS was independently associated with SDB (OR 3.2, CI-1.0–10.1) and EDS (OR 4.5, CI-1.2–16).

Conclusions—SDB and EDS are highly prevalent in adolescent girls with PCOS compared with matched controls. The MetS is independently associated with SDB and EDS in this group.

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Keywords

Pediatric Sleep Questionnaire (PSQ); Epworth Sleepiness Scale (ESS); metabolic syndrome (MetS)

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder of women of reproductive age [1]. It usually presents at puberty with irregular menstrual cycles and signs of hyperandogenemia such as acne and hirsutism. Women with PCOS are often obese and the prevalence of obesity is as high as 75% [2]. Patients with PCOS are also at increased risk of developing reproductive, metabolic and cardiovascular disorders, including infertility, insulin resistance, diabetes mellitus type-2, hypertension, and atherosclerosis [3].

In recent years, PCOS has also been recognized to be associated with sleep disordered breathing (SDB) as well as excessive daytime sleepiness (EDS) [4, 5]. In fact, a prospective case-control study, estimated that women with PCOS have a 30-fold higher prevalence of SDB than women in the general population [5]. The pathophysiological mechanisms leading to such high prevalence of SDB in PCOS have not yet been defined. However, possible causes include alterations in body fat composition due to excess androgen levels and/or the effects of metabolic syndrome [4, 6], the later of which has been previously associated with increased risk of SDB in patients without PCOS [7, 8]. Even though SDB is very prevalent in women with the disorder, the natural history of the disorder in adolescent girls and young women is unknown, mostly due to lack of knowledge about such an association. [9].

Thus, the main aims of the present study were twofold: First, to compare the prevalence of SDB and EDS among adolescent girls with PCOS with sex-, age-, race-, and BM-Z score-matched controls using standardized questionnaires [10] [11] Second, to evaluate the association of SDB and EDS with anthropometrics, demographics, metabolic, and endocrine profiles derived from electronic medical records, within the PCOS group.

METHODS

The study included a cross-sectional survey and a retrospective chart review. The study was approved by the Institutional Review Board (IRB) at the Albert Einstein College of Medicine. Informed assent was obtained from each subject. IRB requested that parental consent would not be obtained in order to maintain participant's confidentiality of diagnosis.

The study population included girls 13–18 year old, who were diagnosed with PCOS and subsequently followed at Children's Hospital at Montefiore, between January 2007 and June 2009. Subjects were first identified by Clinical Looking Glass (CLG), an interactive software application developed at Montefiore Medical Center. Accordingly, the PCOS ICD-9 code-256.4 was queried and the diagnosis was verified by reviewing each participant's "electronic patient file" (EPF).

Age-, race-, and BMI Z-score-matched girls, followed at adolescent and obesity clinics at Children's Hospital at Montefiore during the above time period were identified through the CLG. EPF of each individual controls were reviewed to exclude the diagnosis of PCOS.

A recruitment letter signed by the primary providers was sent to all the participants. Two weeks after the recruitment letters were mailed out, the participants who did not opt out were contacted via telephone. A maximum of three attempts were made to reach the participants. The questionnaire (see below) was administered by one of two investigators; KN or TS, though only TS was blinded to PCOS diagnosis. All responses were obtained from participants without help from their parents. Subjects with any significant co-morbid

conditions other than diabetes, hypertension or metabolic syndrome were excluded from the study. Subjects who were non-verbal, who were above 18 years of age at the time of survey, or could not communicate in English, were excluded from the study.

Screening for Sleep Disordered Breathing

The Pediatric Sleep Questionnaire- Sleep Related Disordered Breathing Scale (PSQ- SRDB) was used to screen for SDB [10]. This questionnaire has been previously validated in children between 2 and 18 years of age. Accordingly, the 22-item scale of PSQ-SRDB has 4 questions on snoring, 4 questions on sleepiness, 6 questions on attention/hyperactivity and 8 additional questions. The responses are coded yes = 1, no=0, don't know/missing = 0. The mean response from non-missing items creates a score between 0 and 1. A score greater than 33% (8 or more positive response out of 22 questions) has a sensitivity of 0.85 and specificity of 0.81 in diagnosing SDB.

Screening for Excessive Daytime Sleepiness

Excessive daytime sleepiness (EDS) was determined by using a modified version of the Epworth Sleepiness Scale (ESS) [11–13]. Though this version has not been validated in children, it is commonly used for both clinical and research purposes [11–13]. Accordingly, we have rated the probability of falling asleep in eight different situations on a scale of 0 (not likely at all) to 3 (extremely likely). The total scores range from 0–24 with a score greater than 10 being considered positive for EDS.

Electronic Patient File Review

We extracted the following data from each participant's EPF: (1) demographics: age, height, weight, BMI Z-score (obtained using the computerized software available from CDC website at the time of study), and race; and (2) history of adeno-tonsillectomy and history of previous overnight sleep studies. For subjects with PCOS, additional data were collected: (1) medications: prescribed during the survey period, particularly metformin and oral contraceptives, the two most commonly prescribed medications for PCOS. Presence of metabolic syndrome based on International Diabetic Foundation (IDF) criteria 2005: central obesity (if BMI>30 central obesity is assumed), and two of the following four factors: raised triglycerides, reduced HDL, raised blood pressure, and elevated fasting glucose/impaired glucose tolerance [14]; and (2) metabolic profile: fasting insulin, glucose, total and free testosterone done within 6 months of the study period. Homeostatic model assessment (HOMA) index, defined as the normalized product of fasting glucose and fasting insulin and used as a measure of insulin resistance (IR), was calculated from available insulin and glucose levels (HOMA-IR = fasting glucose (mg/dL) × fasting insulin (μU/mL)/405).

Statistical Analysis

Statistical analysis was conducted using SPSS version 18. We used proportions to estimate the prevalence of SDB and EDS respectively among girls with PCOS and control population. Means of continuous variables (age, BMI, insulin and testosterone levels) were compared between those with and without SDB and with and without EDS; using independent samples t-tests/Mann Whitney tests after checking for normality assumptions. Comparisons of proportions of categorical variables were assessed with Chi Square. Logistic regression analysis was conducted to assess whether free testosterone, HOMA index, or presence of metabolic syndrome was associated with SDB and EDS, while accounting for potential confounding factors including age, BMI and race. Hosmer-Lemeshow tests for model fit were examined and first order interactions between free total testosterone and other covariates were tested with interaction product terms. A two-tailed alpha of .05 was used to indicate statistical significance.

RESULTS

Out of 13,000 adolescent girls between 13–18 years of age followed during the study period in our Medical Center; we have identified 240 girls with PCOS. A telephone contact was established with 135 girls. Among those, 28 were not interested in participating in the survey and 4 were excluded because of mental retardation. The remaining 103 girls constituted the study sample. The study sample matched the rest of the sample (n=137) in terms of age (16.9±1.5 vs. 16.6±1.5), BMI Z-score (1.9±0.6 vs. 1.9±0.5) and race (predominantly Hispanic, 60% vs. 63%).

Age-, race-, and BMI Z-score-matched control girls (n=220) were identified from CLG. Telephone contact was established with 112 girls and 22 were not interested in participating in the survey. Final sample size was 90.

Demographic data, anthropometric data and medical history were compared between the PCOS and control groups and are shown in Table I. PCOS group had higher prevalence of SDB (45.6% vs. 27.8%, p=0.01) and EDS (54.4% vs. 35.6%, p<0.01) compared with age-, race-, and BMI Z-score-matched controls. There was no difference in the two groups in terms of prior history of adenotonsillectomy or prior sleep studies.

Within the PCOS group, demographic, anthropometric, medical and medication history was further compared between those with and without SDB and with and without EDS (Table II). Those with SDB had higher mean BMI Z-scores (2.1±0.5 vs 1.7±0.6, p<0.01), higher prevalence of MetS (42.6% vs. 16.1% p=0.003) and higher number of previous sleep studies (29.8% vs. 3.6% p=0.001), compared with those without SDB. Similarly, within the PCOS group, those with EDS had higher mean BMI Z-score (2±0.6 vs. 1.7±0.6, p=0.03) higher prevalence of MetS (39.3% vs. 14.9% p<0.01) and higher number of previous sleep studies (25% vs. 4.3% p=0.002) than those without EDS.

Metabolic and Hormone Profile of PCOS Group

Metabolic profile and hormone levels were available in 77% and 86% of subjects with PCOS, respectively (Table II). Those with SDB had higher unadjusted mean insulin levels (IU) (23.4±10.0 vs. 19.4±14.1 p=0.02) and higher HOMA index (5.1±2.3 vs. 4.1±3.5, p<0.01) compared with those without SDB. Similarly, those with EDS had higher unadjusted mean insulin levels (IU) (23.1±11.2 vs. 18.8±13.7, p=0.02) and higher HOMA index (5.1±2.9 vs. 3.8±3.1, p<0.01) compared with those without EDS (Table III). Logistic regression analysis, adjusting for age, BMI, race, free testosterone and HOMA index, showed that the presence of MetS is an independent predictor of SDB (odds ratio 3.2 CI-1–10.1, p= 0.04) and EDS (odds ratio 4.5 CI-1.2 to 16, p=0.02) (Table IV)

DISCUSSION

Our study suggests that SDB and EDS are present in a significantly higher proportion of adolescent girls with PCOS compared with age-, sex-, race-. and BMI Z-score-matched controls. Our data also shows that alterations in glucose metabolism are common in girls with PCOS who have SDB or EDS. In addition, MetS seems to be independently associated with SDB and EDS in this population.

The BMI Z-scores in both groups studied suggests that 92% of subjects were either overweight or obese. Therefore, an initial comment regarding obesity and SDB is most relevant to our study. The prevalence of obesity has almost tripled in the adolescent age group in the last two decades [15]. The association of SDB with obesity is well established in adults as well as pediatric population. Obese children and adolescents are at 4–5 fold

increased risk for development of SDB [16]. Though we do not know the exact prevalence of SDB in adolescent girls, polysomnography abnormalities are seen in up to 36% of moderately obese, inner-city, children and adolescents [17]. Our study is consistent with the above rate in our control group.

In regard to PCOS, both SDB and metabolic derangements related to glucose metabolism have been consistently reported in women with the disorder. Vgontzas et al showed that pre-menopausal women with PCOS have a 30-fold higher prevalence of SDB as compared with general population controls [5] and that insulin resistance was the strongest predictor for SDB when adjusted for age, BMI, and testosterone levels. Tasali et al [18] reported similar findings and showed higher fasting insulin and HOMA index in young women with PCOS and SDB. Our study extends and confirms the above findings to adolescent years when the diagnosis of PCOS is first made.

In recent years, the mechanisms leading to alterations in glucose metabolism in subjects with SDB in the general population have begun to unfold and have been linked to alterations in sleep architecture, presence of intermittent hypoxia, and increased sympathetic activity [19]. Other studies have demonstrated that SDB is an independent risk factor for the development of glucose intolerance, insulin resistance, and type 2 diabetes mellitus [20] [21].

It is plausible that SDB explains the propensity of subjects with PCOS to develop altered glucose metabolism by the above explained mechanisms. However, a recent study showed elevated fasting insulin and interleukin-6 levels independent of SDB or obesity in PCOS [22], suggesting a role of pro-inflammatory cytokines in the development of insulin resistance in this population.

Altered glucose metabolism noted in our subjects with SDB may indicate a broader perturbation linked to the MetS which is commonly associated with PCOS. MetS is characterized by abdominal obesity, glucose intolerance, dyslipidemia, hypertension and pro-nflammatory state, leading to increased risk of coronary heart disease.

Various studies link the MetS to SDB [7, 8, 23]. The mechanism could be related to increased abdominal visceral obesity altering chest wall and upper airway mechanics and reducing functional residual capacity making subjects more vulnerable to hypoxemia during sleep [24]. It has also been shown that SDB can independently induce MetS by decreasing insulin sensitivity in both animals and humans [25]. A recent study by Tasali et al in women with PCOS and SDB showed the reversal of key determinants of the MetS after 8-weeks of CPAP treatment including significant improvements in: insulin sensitivity, daytime diastolic blood pressure, heart rate variability, and daytime sleepiness [26]. Although, we cannot establish a causal relationship between PCOS, SDB, and the MetS in our study due to the cross-sectional design, our study demonstrates the need for routine screening and intervention for individuals with PCOS for SDB and EDS, especially those with associated MetS.

The characteristic finding of hyperandrogenemia in subjects with PCOS is another possible consideration explaining the high prevalence of SDB [4]. It has been speculated that presence of excess androgens in adult males may account for a higher prevalence of SDB in men as compared with women [27]. Differences in androgen levels may affect body composition, visceral adiposity, upper airway anatomy, ventilatory drive during sleep, and also insulin resistance [28]. In our study testosterone levels were similar between the subjects with and without SDB. Our study may have not been able to discern differences because the majority (70%) of our study population were prescribed hormonal contraceptives which can decrease the androgen levels, and testosterone levels are generally

much lower even in PCOS hyperandrogenemia than in males and so may have not been assayed with sufficient sensitivity.

In contrast to our findings of a high prevalence of SDB in adolescent girls with PCOS, a recent study by de Sousa et al did not find any difference in prevalence of SDB in adolescents with PCOS compared with normal and obese controls [29]. However, these authors did note altered sleep architecture between groups suggesting poor sleep efficiency and delayed sleep latency in the PCOS group. Such differences between the studies could relate to different methodologies as well as different sample size and populations.

Excessive day time sleepiness is one of the common causes for decreased academic performance and was noted to be twice more prevalent in the PCOS group compared with the control group. This finding is consistent with previously published reports, showing that EDS exists in women with PCOS independent of obesity and SDB [4, 5]. Various reasons can explain EDS in this population including sleep fragmentation and sleep deprivation secondary to SDB. Other causes may include insomnia with prolonged sleep latency and poor sleep efficiency as noted by de Sousa et al. [29]. Such disturbances in sleep architecture have been reported to be secondary to psychological stress and neurohormonal imbalances resulting from the disease [30]. Similar to our findings, EDS has previously been linked to insulin resistance[31]. Vgontzas et al showed that interlukin-6 was significantly elevated in women with PCOS and EDS, independent of obesity and SDB. They have postulated that cytokines may be one of the pathways leading to insulin resistance [22].

We would like to emphasize a few limitations of our study that are derived from the nature of the design. First, SDB and EDS were evaluated by screening questionnaires considered a standardized tool. However, polysomnography and multiple sleep latency test (MSLT) would be important methods to confirm our findings particularly because the version of ESS used has not been validated in children. Second, one of the two interviewers was not blinded to the primary diagnosis of PCOS. This may have confounded our results. However, comparison of scores between interviewers did not show any significant difference between them. Third, the MetS was defined by IDF criteria for adolescents/adults [14]. We chose these criteria as >80% of our participants were above 16 yrs of age. We did not use the Adult Treatment Plan report (ATP III) criteria [32], as the waist circumference for our participants was not reported in every medical record. Thus, the IDF definition may have over estimated the presence of MetS in the studied population. Nevertheless, it has been reported that prevalence of MetS using ATP III in overweight adolescents is about 28.7%, which is similar to our reports (28.2%) [33]. Fourth, the majority of our study participants were on hormonal contraceptives and metformin, which may have falsely lowered the prevalence of SDB and EDS; however, there was no difference in proportions of adolescent girls on these medications between the sub groups. Thus, based on our results, a prospective, longitudinal study in treatment naïve PCOS population is warranted to better understand the mechanism and natural history of SDB and EDS in this age group.

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TABLE 1

Population characteristics PCOS vs. Controls

	PCOS (n=103)	Controls (n=90)	p Value
Age in years (mean ± SD)	16.9 ± 1.5	16.8 ± 1.7	NS
Race:			
Hispanic (n) (%)	62 (60.2%)	48 (53.3%)	NS
African- American (n) (%)	25 (24.3%)	31 (34.4%)	NS
Others (n) (%)	16 (15.5%)	11 (12.2%)	NS
BMI Z-score (mean ± SD)	1.9 ± 0.6	1.8 ± 0.5	NS
Previous sleep studies (n) (%)	16 (15.5%)	11 (12.2%)	NS
History of adenotonsillectomy(n) (%)	7 (6.8%)	8 (8.9%)	NS
SDB positive (n) (%)	47 (45.6%)	25 (27.8%)	**0.01**
EDS positive (n) (%)	56 (54.4%)	32 (35.6%)	**<0.01**

TABLE 2

PCOS Characteristics

	SDB Positive (n=47)	SDB Negative (n=56)	EDS Positive (n=56)	EDS Negative (n=47)
Age in years (mean ± SD)	16.8 ± 1.5	16.9 ± 1.5	16.8 ± 1.5	16.9 ± 1.6
Race:				
Hispanic (n) (%)	29 (61.7%)	33 (58.9%)	36 (64.3%)	26 (55.3%)
African- American (n) (%)	11 (23.4%)	14 (25%)	13 (23.2%)	12 (25.5%)
Others (n) (%)	7 (14.9%)	9 (16.1%)	7 (12.5%)	9 (19.1%)
BMI Z-score (mean ± SD)	2.1 ± 0.5 *	1.7 ± 0.6	2 ± 0.6 *	1.7 ± 0.6
MetS (n) (%)	20 (42.6%) **	9 (16.1%)	22 (39.3%)*	7 (14.9%)
Previous sleep studies (n) (%)	14 (29.8%) **	2 (3.6%)	14 (25%)*	2 (4.3%)
History of adenotonsillectomy (n) (%)	5 (10.6%)	2 (3.6%)	5 (8.9%)	2 (4.3%)
Medications				
Metformin (n) (%)	15 (31.9%)	11 (19.6%)	14 (25%)	12 (25.5%)
Hormonal contraceptives (n) (%)	33 (70.2%)	37 (66.1%)	37 (66.1%)	33 (70.2%)

* p value<0.05

** p value<0.005

TABLE 3

Comparison of Fasting Insulin, Glucose, HOMA and Testosterone Levels in PCOS

	SDB Positive	SDB Negative	EDS Positive	EDS Negative
Fasting Insulin (IU) (mean ± SD)	23.5 ± 10* (n= 40)	19.4 ± 14.1 (n= 47)	23.1±11.2* (n= 49)	18.8± 13.7 (n= 38)
Fasting Glucose (mg/dl) (mean ± SD)	86.7 ± 14.7 (n= 41)	83.2± 14.9 (n= 48)	86.9 ± 17.9 (n= 50)	82± 8.7 (n= 39)
Free testosterone (ng/dl) (mean ± SD)	7.7 ± 5.4 (n= 36)	7.9 ± 5 (n= 44)	7.4 ± 4.7 (n= 44)	8.4± 5.7 (n= 36)
Total testosterone(pg/dl) (mean ± SD)	46.4 ± 23.9 (n= 38)	44.9 ± 22.3 (n= 50)	46.2 ± 26.4 (n= 46)	46.2± 26.4 (n= 42)
HOMA (mean ± SD)	5.1 ± 2.3* (n= 40)	4.1 ± 3.5 (n= 47)	5.1 ± 2.9* (n= 49)	3.8±3.1 (n= 38)

* p value<0.05

Data presented as mean ± SD

TABLE 4

Logistic Regression Analysis

	SDB Positive			EDS Positive		
	OR	**Adjusted OR (95% CI)**	**p Value**	**OR**	**Adjusted OR (95% CI)**	**p Value**
Age	0.9	0.6–1.3	0.61	0.9	0.6–1.3	0.51
BMI-Z score	2.3	0.8–6.8	0.12	1.6	0.6– 4.2	0.37
Race	1	0.5–2.1	0.89	0.9	0.4–1.8	0.71
HOMA	0.9	0.8– 1.2	0.9	1	0.9–1.2	0.7
Testosterone (Free)	1.0	0.9 –1.1	0.8	0.9	0.9– 1.1	0.7
MetS	3.2	1–10.1	0.04	4.5	1.2–16	**0.02**