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Gastroesophageal reflux and obstructive sleep apnea in childhood

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Summary

Objectives: To examine the relationship between gastroesophageal reflux (GER) and obstructive sleep apnea syndrome (OSAS) with polysomnographic alterations and symptom severity.
Patients and methods: Eighteen children aged from 6 to 12 years (8.14 ± 1.75) with adenotonsillar hypertrophy and OSAS were evaluated with the OSA-18 questionnaire nasofibrolaringoscopy and full overnight polysomnography performed simultaneously with esophageal pH monitoring.
Results: OSAS (Apnea-index (AI) ≥ 1/hour) was present in all cases. Reflux parameters did not correlate to OSAS severity and a temporal relationship between GER and apnea–hypopnea events was not observed. Body mass index was lower than 18 in 9 cases (52.9%) and 7 children (41.1%) presented a history of abnormal behavior during sleep. In most cases oxygen desaturation and reduction of sleep efficiency were mild. Sleep architecture was similar to the young adult pattern. Seven children (41.1%) presented pH monitoring values below 4 during more than 10% of total sleep time. pH monitoring values were correlated to emotional distress ($p = 0.008$) and to daytime problems ($p = 0.03$) as evaluated by the OSA-18.
Conclusions: GER is frequent and should be assessed in children from 6 to 12 years with OSAS. Emotional distress and daytime problems are correlated to increased GER severity.

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1. Introduction

It is estimated that 9—10% of children are habitual snorers or have sleep disordered breathing related illnesses [1,2]. Adenotonsillar hypertrophy is recognized as the most frequent cause of obstructive sleep apnea syndrome (OSAS) in childhood [3].

Despite a confirmed association between GER and OSAS in children as well as in adults the association between these two conditions remains controversial. Episodes of GER preceding apnea—hypopnea events in infants [4] and inducing SaO_2 desaturation [5] have been reported. Treatment of GER has been shown to improve OSAS [6] and OSAS therapy with CPAP has been demonstrated to reduce GER [7] confirming a close association between these two conditions. However GER has not been shown to contribute to apnea severity in pre-term infants [8]. Furthermore in older individuals a direct correlation between GER and OSAS was not found [9]. As adenotonsillar hypertrophy may be associated with more severe apnea in younger than in older children [10] it is possible that age plays a differential role on the manifestation of sleep abnormalities and daily symptoms associated with both OSAS and GER.

It is recognized that different approaches to the definition and quantification of sleep disordered breathing may lead to discrepant morbidity and treatment outcome results [11,12]. General criteria for diagnosis include the presence of obstructive apnea index of at least 1 or the apnea—hypopnea index of at least 5 events per hour [13]. However controversy remains about the use of a respiratory disturbance index to define various abnormalities and the secondary comorbidities expressed in association with them [14]. Sleep changes such as sleep fragmentation alteration of sleep microstructure and nocturnal oxyhemoglobin desaturation might also affect the manifestation of behavioral symptoms.

In the present study we investigated the association between OSAS and GER and its consequences on polysomnographic and clinical features and quality of life in children from 6 to 12 years of age.

2. Patients and methods

2.1. Study population

This was a cross-sectional study of children with adenotonsillar hypertrophy. Demographic and clinical data were recorded using a closed-question data collection instrument. All variables were measured concurrently. Cases were consecutively recruited from and Ear—Nose—Throat hospital based outpatient clinics. Twenty children of both genders aged from 6 to 12 years were initially enrolled. Simultaneous recording of polysomnographic parameters and esophageal pH monitoring was successfully obtained from 17 individuals. Two cases had unsatisfactory pH monitoring and one had insufficient polysomnographic data. Inclusion criteria were adenotonsillar hypertrophy grades 3 and 4 or adenoid hypertrophy associated with airway occlusion of 70% or more. The presence of neurological disorders craniofacial dysplasia and the use of sedative or antiepileptic drugs were considered exclusion criteria. The study protocol was approved by the local Research Ethics Committee and written informed consent was obtained in all cases.

2.2. Procedures

2.2.1. Clinical evaluation

After clinical examination nasofibrolaringoscopy for inspection of nasal mucosa adenoids and tonsillar tissue was performed. A questionnaire related to the presence of apnea in childhood was applied (OSA-18) [15]. It has been previously validated in Portuguese [16]. This questionnaire is divided into five components that assess sleep disturbance physical suffering emotional distress daytime problems and caregiver concerns. Scores vary from 18 to 126. Scores lower than 60 indicate small impairment of quality of life from 60 to 80 moderate impairment and greater than 80 high impairment. A visual analogical scale varying from 0 (bad quality of life) to 10 (good quality of life) is also part of this questionnaire. Additional questioning about the presence of symptoms such as halitosis, oral breathing, sore throat, sinusitis, irritable mood, stomach ache, frequent vomiting, anorexia and hoarseness was performed.

2.2.2. Polysomnography

Standard overnight polysomnography (PSG) was performed on an ALICE II digital polygraph (Respironics®). PSG was set to begin at 8:00 p.m. (lights-out) and end at 6 a.m. (lights-on). Monitored variables included electroencephalogram (EEG) (C3 C4 O1 O2 referenced to contralateral ear electrodes) electro-oculograms (EOG) submental electro-myogram (EMG) two-lead electrocardiogram (EKG) and pulse oximetry. Leg movements were monitored using a bilateral tibialis EMG and respiration using a nasal/oral thermocouple. Body position and thoracic and abdominal movements (inductance plethysmography) were also recorded. Sleep staging was performed by 30-s epochs according to standard procedures [17]. Polysomnography-derived parameters evaluated were apnea—hypopnea index (AHI) apnea index (AI) minimum oxygen saturation (SaO_2) sleep latency sleep efficiency REM latency

amount of REM sleep (% of Total Time of Sleep) amount of non-rapid eye movement (NREM) sleep (% of Total Time of Sleep) number of arousals and periodic leg movements. Arousal analysis and scoring of respiratory events during sleep were performed according to published criteria [17]. Obstructive apneas were defined as absence of oro-nasal airflow 80% decrease in airflow detected by thermistor for more than two respiratory cycles in the presence of continued respiratory effort lasting longer than two respiratory cycles and hypopneas as a reduction of inspiratory air flow of 50% or more associated with either oxygen desaturation of >3% or an arousal. Severity of sleep-disordered breathing was estimated by calculating the apnea index (AI; apneas per hour of sleep) and the apnea + hypopnea index (AHI; apneas plus hypopneas per hour of sleep) [18].

2.2.3. Esophageal pH monitoring

Prolonged esophageal pH monitoring was performed using an antimony pH electrode with a separate skin reference electrode (Sigma Instruments Belo Horizonte Brazil®). The data were stored on a portable digital recorder (Sigma Instruments Belo Horizonte Brazil). Before each study the pH probe was calibrated in buffer solutions of pH 7 and 1. An episode of acid reflux was defined as a decrease in esophageal pH to less than 4 during more than 10 s. Esophageal pH monitoring lasted for approximately 24 h. After a 4-h fast period the probe was placed transnasally into the stomach and then slowly withdrawn in such a way that the tip of the electrode would lie over the third vertebral body above the diaphragm [19]. Its position was confirmed by a chest X-ray. Mealtime changes in decubitus and occurrence of clinical symptoms were recorded. No nutritional restraints were imposed on the children during examination and all of them drunk a cup milk with chocolate around an hour before bedtime. The parameters analyzed by esophageal pH monitoring as well as their respective normal values [20] presented in parentheses included: total percent time of the presence of acid esophageal pH i.e. pH below 4 (<4.2%); total number of acid episodes (<50 episodes); number of reflux episodes longer than 5 min (3 or less) and duration of the longest reflux episode (<9.2 min). The same parameters were evaluated for the supine and standing positions. Polysomnographic measures and pH data were synchronized for the same time.

2.3. Statistical analysis

Data were examined for normality using the Shapiro—Wilk test and for homogeneity of variance with the Levene test. Mann—Whitney test was used for between-group comparison. Spearman correlation test was used to detect linear associations between quantitative variables. Statistical analysis was performed with the Statistic Package for Social Sciences (SPSS — Norusis 1993) software for Windows. The level of significance was set at $p < 0.05$.

3. Results

We studied 17 children aged 6—12 years (8.14 ± 1.75) with adenotonsillar hypertrophy grades 3 and 4 or adenoids with airway occlusion of 70% or more. Weight ranged from 18 to 46 kg (30.30 ± 7.99) height from 109 to 149 cm (128 ± 0.11) and BMI from 13 to 25 (18.27 ± 3.50). Ten children (58.8%) presented a BMI lower than 18 and the remaining seven had a normal BMI (range 18—25) Most commonly referred symptoms were snoring, frequent cold, coughing, halitosis, sinusitis, sore throat and oral breathing (Table 1). Seven children (41.1%) had a history of abnormal behavior during sleep. According to the OSA-18 evaluation 10 children (58.8%) showed a great impairment in quality of life (OSA 18 > 80) and this was not correlated to AHI severity. All studied children had an apnea index (AI) ≥1. Nine cases had an AHI ≥5. Table 2 shows the results of full-night polysomnography according to the criterion of ≥1 apnea/hour or ≥5 apnea—hypopnea/hour and no differences between groups were found. On average there was no difference in OSA-18 scores between children with AI≥1 and

Table 1 Most commonly reported symptoms in children with adenotonsillar hypertrophy and obstructive sleep apnea.

Sleep and respiratory symptoms	*N*	%
Snoring	18	94.7
Frequent cold	20	90.9
Cough	18	81.8
Sore throat	14	63.6
Sinusitis	14	63.6
Frequent crying or irritable mood	14	63.6
Oral breathing	10	52.6
Choking	9	47.4
Hoarseness	9	40.9
Abnormal sleep behavior	7	31.8
Repeated tonsillitis	5	26.3
Disturbed sleep	4	21.0
Gastric symptoms		
Halitosis	17	77.2
Stomach ache	11	50.0
Frequent vomiting	10	45.4
Anorexia	10	45.4
Dysphagia	7	36.8

Table 2 Polysomnography results of 17 children with obstructive sleep apnea and adenotonsillar hypertrophy.

Sleep measures	AI $\geq$ 1; AHI $\leq$ 5	AHI > 5	Mann–Whitney test *Z*; *p* value	Total
Total sleep time (min)				
Range	410.50–527.0	377.0–518.0	0.39	377–527.0
Mean ± SD	475.57 ± 46.46	453.27 ± 47.03		462.07 ± 43.9
Sleep efficiency (%)				
Range	92.2–99.5	84.4–99.8	0.79	84.4–99.8
Mean ± SD	95.21 ± 2.81	94.86 ± 4.65		95.2 ± 3.6
Sleep latency (min)				
Range	2–20.5	2–55.5	0.18	2–55.5
Mean ± SD	6.35 ± 6.54	14.61 ± 17.7		11 ± 13.7
NREM I (%)				
Range	0.14–0.90	0.19–2.33	0.18	0.14–2.33
Mean ± SD	0.51 ± 0.32	0.93 ± 0.75		0.7 ± 0.5
NREM II (%)				
Range	40.2–65.5	28.1–68.7	0.28	28.1–68.7
Mean ± SD	53.93 ± 9.4	46.57 ± 15.01		48.7 ± 13.4
NREM III–IV (%)				
Range	13.64–35.9	11.38–44.3	0.36	11.38–4439
Mean ± SD	26.20 ± 8.28	31.42 ± 11.65		30.1 ± 10.9
REM (%)				
Range	12.2–23.53	15.74–27.3	0.87	12.2–27.3
Mean ± SD	19.46 ± 4.13	20.54 ± 3.62		20.2 ± 3.9
REM latency (min)				
Range	18–123	20–150	0.42	18–150
Mean ± SD	84.85 ± 35.9	101.11 ± 42.63		90.5 ± 39.3
Arousal index (events/h)				
Range	4–27	5–45	0.42	4–45
Mean ± SD	10.88 ± 7.81	15.94 ± 13.48		13.12 ± 10.4
Minimum SaO_2				
Range	79–89	53–90	0.79	53–90
Mean ± SD	83.83 ± 3.76	80.12 ± 11.51		82.7 ± 8.2
AHI (events/h)				
Range	1.5–4.7	5.3–38.8	0.001	1.5–38.8
Mean ± SD	2.59 ± 1.05	9.91 ± 10.87		6.46 ± 8.58

Abbreviations: AI = apnea index; AHI = apnea–hypopnea index; min = minutes; h = hour; NREM = non-rapid eye movement; REM = rapid eye movement; SaO_2 = oxygen saturation.

AHI$\geq$5 (Fig. 1). Sleep efficiency was above 89% in all but one case. In general sleep architecture features were similar to the young adult pattern.

Gastroesophageal reflux was present in 15 children (88.2%). Eight children (47.0%) had more than 10% of total sleep time with pH monitoring values below 4. Nocturnal pH monitoring values were not different when comparing cases with less and more severe OSA as defined by AHI (Table 3). Also GER and AHI were not correlated (Table 4). Simultaneous recording of OSA and GER showed no relationship between the timing of these events.

Regarding gender no differences were found in pH monitoring results and in polysomnographic findings.

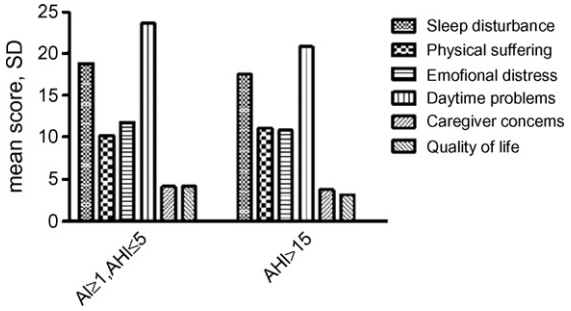

Fig. 1 OSA-18 measures are not different in children with adenotonsillar hypertrophy and OSAS grouped as apnea–hypopnea/hour $\leq$ 5 and apnea-index/hour $\geq$ 1 or apnea–hypopnea/hour > 5.

Table 3 Twenty-four hour pH monitoring results of 17 children with adenotonsillar hypertrophy and OSAS grouped as having apnea index >1 and apnea + hypopnea index ≤ 5 or apnea + hypopnea index >5.

	AI ≥ 1;AHI ≤ 5 ($N = 8$)	IAH >5 ($N = 9$)	Mann—Whitney test, p value
ST pHM (min)	638.75 ± 118.94	681.56 ± 154.16	0.56
ST GER (N)	21.00 ± 14.06	31.89 ± 24.71	0.47
ST GER >5 min (N)	2.25 ± 2.71	2.11 ± 2.08	0.88
ST Longest GER (min)	35.12 ± 35.18	14.55 ± 11.94	0.46
ST pH <4 (min)	76.12 ± 81.58	45.89 ± 34.14	0.56
ST pH <4 (%)	12.73 ± 13.10	7.20 ± 5.45	0.50

Abbreviations: pHM = pH monitoring; min = minutes; ST = supine time; GER = gastroesophageal reflux; N = number of events; % = percent.

Table 4 depicts the correlation matrix between polysomnographic measurements OSA-18 values and pH monitoring results. Polysomnographic measurements were compared with nocturnal pH monitoring results. Emotional distress as assessed by the OSA-18 was correlated to several measures indicative of GER. Daytime problems were also correlated to GER (Table 4). Overall polysomnographic parameters were not correlated to pH monitoring results. Age and BMI showed no correlation with pH monitoring results. Age was negatively correlated to sleep latency (0.04), to delta wave sleep (0.008) and positively correlated to the AHI ($p = 0.02$) and to REM latency (0.04). In this study the BMI was not correlated to any of the polysomnographic measures.

4. Discussion

Our data show that GER is common in 6—12-year-old children with obstructive sleep apnea syndrome (OSAS) associated with adenotonsillar hypertrophy. The increased frequency of GER in this population may have been secondary to the fact that we have studied only children with surgical indication of adentonsillectomy. The present findings cannot be generalized to the population given that the study population consisted of children looking for medical care for adenotonsillar hypertrophy and may have been over symptomatic. We also show that the severity of GER is correlated to emotional distress and daytime problems in this age group.

A temporal relationship between GER and apnea—hypopnea events was not observed. It should be noted that although a simultaneous recording was performed pH monitoring and polysomnographic registering were not integrated. Additionally since a pH probe and not a multichannel intraluminal impedance was used to assess GER it is possible that the true amount of GER may have been underestimated in these patients. No nutritional restraints were imposed which may have also affected the GER results in these children. However the same structural technique has been used before by other studies [4]. Furthermore the finding that OSA and GER severities were not related reinforces the soundness of our results. To the best of our knowledge a similar study has not been reported in this age group and part of the controversy on this issue may be secondary to the different ages of the study subjects. Previously the lack of a correlation between GER and OSA has also been reported in adults. In this study, undetected associations between OSAS and GER may have been due to the small sample size and a type II error cannot be excluded. In our subjects the most common symptoms were snoring and other respiratory manifestations in agreement with other reports [9]. Abnormal behavior during sleep present in more than a third of our cases corroborates the importance of sleep disordered breathing in the pathogenesis of parasomnia in this age group [21]. Gastric related manifestations were present in approximately 50% of cases also in agreement with other studies [8,22]. On average BMI was not increased in this study. In fact several children were below expected weight and height levels. Improvement in weight and height after adenotonsillectomy has been reported in children pointing to the fact that adenotonsillar hypertrophy and OSAS may lead to impaired growth and development [23,24]. In this study BMI was not associated with OSAS or GER severity.

The only gender related difference found in this study was a reduction of REM sleep latency in female children. At present we have no satisfactory explanation for this finding. On average our polysomnographic results were similar to what has been previously described for normal children from this age group [25]. In this study sleep architecture was similar to previous reported NREM 3—4 (23—34%) and REM (17—18%) sleep median values for children and adolescents [26]. This is in agreement with another study showing no major change on sleep micro or macrostructure in children with OSAS as compared to normal children [27].

Table 4 Spearman correlation test between OSA-18 results and polysomnographic measures with age BMI and pH monitoring findings.

		Age	BMI	TTGER	STGER	TTGER > 5 min	STGER > 5 min	TTLongGER	STLongGER	TTpH <4 (min)	STpH <4 (min)	TTpH <4 (%)	STpH <4 (%)
OSA results													
SD	*r*	−0.009	0.199	0.247	0.237	−0.114	0.018	−0.003	−0.012	−0.008	−0.097	−0.005	0.066
	p	0.07	0.44	0.33	0.35	0.66	0.94	0.99	0.96	0.97	0.77	0.98	0.80
PS	*r*	0.187	−0.058	0.173	0.166	0.446	0.354	0.258	0.277	0.339	0.032	0.334	0.362
	p	0.47	0.82	0.50	0.52	0.07	0.16	0.31	0.28	0.18	0.92	0.19	0.15
ED	*r*	−0.272	−0.422	0.622	0.620	0.604	0.529	0.478	0.486	0.590	0.407	0.596	0.499
	p	0.29	0.09	0.008**	0.008**	0.01*	0.02*	0.05	0.04*	0.01*	0.21	0.01*	0.04*
DP	*r*	−0.337	−0.073	0.435	0.524	0.486	0.396	0.116	0.106	0.196	0.022	0.196	0.072
	p	0.18	0.78	0.08	0.03*	0.04*	0.11	0.65	0.68	0.45	0.95	0.45	0.78
CC	*r*	0.409	0.308	0.089	0.140	0.085	0.277	0.369	0.361	0.282	0.340	0.280	0.329
	p	0.10	0.22	0.73	0.59	0.74	0.28	0.14	0.15	0.27	0.30	0.27	0.19
QL	*r*	−0.171	0.246	0.055	−0.024	0.049	−0.002	0.068	0.066	0.138	0.174	0.159	0.096
	p	0.51	0.34	0.83	0.92	0.85	0.99	0.79	0.80	0.59	0.60	0.54	0.71
PSG results													
AHI	*r*	0.531	0.306		0.070		0.076		−0.009		0.009		0.038
	p	0.02	0.23		0.79		0.77		0.97		0.97		0.88
SaO_2 min	*r*	−0.248	−0.256		−0.440		−0.455		−0.489		−0.490		−0.459
	p	0.39	0.37		0.15		0.13		0.10		0.10		0.13
SL	*r*	−0.651	−0.246		−0.310		−0.078		−0.022		−0.101		−0.052
	p	0.006**	0.35		0.22		0.64		0.93		0.69		0.84
N4 s	*r*	−0.630	0.126		−0.010		−0.122		−0.063		−0.163		−0.107
	p	0.009**	0.64		0.97		0.64		0.81		0.53		0.68
REM L	*r*	0.516	−0.224		0.053		−0.068		0.309		0.146		0.238
	p	0.04*	0.40		0.83		0.79		0.22		0.57		0.35
REM T	*r*	−0.131	−0.056		−0.144		0.225		0.122		0.226		0.228
	p	0.62	0.83		0.63		0.38		0.64		0.38		0.37
SE	*r*	−0.174	−0.159		−0.409		−0.251		−0.228		−0.246		−0.206
	p	0.21	0.55		0.10		0.33		0.37		0.34		0.42

Abbreviations: TT = total time; pHM = pH monitoring; min = minutes; ST = supine time; GER = gastroesophageal reflux; % = percent; SD = sleep disturbance; PS = physical suffering; ED = emotional distress; DP = daytime problems; CC = caregiver concern; QL = quality of life; PSG = polysomnography; AHI = apnea–hypopnea index; min = minutes; SaO_2 = oxygen saturation REM L = rapid eye movement latency; REM T = rapid eye movement total time; SE = sleep efficiency.

* $p < 0.05$.

** $p < 0.01$.

Poor quality of life was common in our children confirming previous reports [28]. Interestingly no significant difference in the OSA-18 domain clinical manifestations was found between children with more and less than five apnea—hypopnea events per hour of sleep.

In summary GER is frequent and not related to AHI in children from 6 to 12 years with OSAS. Adenotonsillar hypertrophy and OSAS are associated with low height and weight and therefore appropriate therapy is essential. Gastroesophageal reflux is related to emotional distress and daytime problems and should be actively searched in children with adenotonsillar hypertrophy and OSAS.

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