

Polysomnographic findings in persistent allergic rhinitis

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Abstract

Purpose Persistent allergic rhinitis (PAR) is characterized by a chronic, eosinophilic inflammation with nasal congestion and rhinorrhea. Nasal congestion can constitute to sleep-disordered breathing problems that range from simple snoring to obstructive sleep apnea syndrome (OSAS). The purpose of this study was to investigate the effect of PAR on sleep quality and severity of OSAS.

Methods The study included 150 patients presenting with typical symptoms of sleep apnea. Fifty-five patients were diagnosed as PAR (group-1) and were compared with age and body mass index matched 95 individuals (group-2) without any nasal disease. Skin prick tests and polysomnography were performed in all patients.

Results There were no differences between groups for polysomnographic findings including sleep architecture, arousal and respiratory index, and mean and minimal oxygen saturations. Simple snoring was determined in 41.8 % allergic and 32.6 % non-allergic patients. Mild OSAS was determined in

32.7 % allergic and 29.4 % non-allergic patients. Moderate OSAS was determined in 9 % allergic and 17.8 % non-allergic patients. Severe OSAS was determined in 16.3 % allergic and 20 % non-allergic patients. Their entire Epworth sleepiness scale index was also found similar.

Conclusions Our data pointed out that in patients with sleep-related breathing disorders symptoms, presence of PAR does not affect polysomnographic parameters compared with other patients without any nasal inflammation. Besides, prevalence of OSAS was similar between groups.

Keywords Allergic rhinitis · OSAS · Polysomnography · Apnea-hypopnea index

Introduction

Sleep-disordered breathing problems enclose a wide spectrum of sleep-related breathing abnormalities related to the increased upper airway resistance including snoring, upper airway resistance syndrome (UARS), and obstructive sleep apnea syndrome (OSAS). OSAS is a potentially life-threatening sleep disorder which is characterized by recurrent episodes of partial or complete airway obstruction during sleep due to repetitive obstruction of the upper airway, necessitating recurrent awakenings or arousals to re-establish airway patency, often with oxygen desaturation, fragmentation of sleep, and excessive daytime sleepiness [1]. In OSAS, underlying pathophysiology of upper airway obstruction is multifactorial.

Three interacting components take part of a serious function in obstruction of the upper airway in OSAS: the structural anatomy of the airway, the muscle activity of the pharyngeal airway, and the negative pressure

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produced during inspiration, which counters the activity of the dilator muscles [2, 3]. The biologic principle for nasal obstruction serves as a reason for sleep-disordered breathing [4]. Therefore, the nasal cavity is related to the part of the total respiratory system resistance [5]. Although, the main site of obstruction in patients with OSAS is believed to be the oropharyngeal–hypopharyngeal structural region. For this reason, the impairment of sleep quality in persistent allergic rhinitis (PAR) patients might not be completely described by the sleep-disordered breathing given rise to by nasal blockage.

Nasal obstruction can be caused by anatomic variations such as septal deviation, inferior turbinate hypertrophy, or mucosal congestion with nasal inflammation [6]. Lofase et al. [7] identified nasal obstruction as a risk factor for OSAS, and Suguira et al. [8] showed that nasal resistance is important for the initial acceptance of treatment by continuous positive airway pressure (CPAP) in OSAS patients.

Allergic rhinitis (AR) is an inflammatory disorder of the upper respiratory tract and affects more than 25 % of the population [9]. The common symptoms of AR are itching, sneezing, post nasal drip, decreased sleep quality, and substantially poor overall quality of life. PAR symptoms are mostly caused by indoor allergens such as house dust mites and animal dander from pets where the exposure occurs throughout the whole year. Chronic allergic nasal inflammation of the upper airway can cause lymphoid hypertrophy with prominence of adenoid and tonsil tissue that constitute a smaller upper airway size, so patients with AR are more likely to suffer problems about sleeping due to their nasal allergy symptoms [10]. In a recent survey, individuals with allergic rhinitis, 68 % of respondents with PAR and 48 % with seasonal allergic rhinitis (SAR) reported that their condition interfered with sleep [11]. The Nasal Allergy Survey Assessing Limitations (NASAL) determined that compared with the general population, more patients with AR reported that they had been extremely or moderately bothered by difficulty in getting to sleep (24 vs 8 %), at least moderately bothered by waking up during the night (31 vs 13 %), and extremely or moderately troubled by lack of a good night's sleep (26 vs 11 %) as a result of their nasal symptoms in the past week [12]. An inconsistent link between sleep problems and PAR has been reported in the literature for decades. In most studies, they compare AR patients with normal population. In our study, we aimed to investigate the effect of PAR on sleep characteristics, nocturnal oxygen levels, apnea-hypopnea index, and severity of OSAS in patients with sleep-related breathing disorders symptoms and compare them with non-allergic group with similar sleep symptoms.

Material and methods

Study design, subjects, and measurements

All consecutive newly diagnosed mild 55 PAR patients who do not use any medication but had sleep-disordered breathing symptoms like snoring, witnessed apnea, or excessive daytime sleeping were referred to full night polysomnography. Also, 95 age-matched non-allergic patients who were referred to full night polysomnography from our sleep disorders outpatient clinic were included as our control group. All patients were tested for an underlying nasal disease by patient's history, endoscopic nasal examination and if needed, computed tomography scans of paranasal sinuses. A general otorhinolaryngology examination, including nasal endoscopy, was performed to exclude subjects with relevant septal deviation, nasal polyps, acute or chronic rhinosinusitis, or any other underlying pathology that might affect nasal breathing or nocturnal sleep. Nasal inflammatory diseases, such as sinusitis, nasal polyposis, or vasomotor rhinitis, were excluded, and only patients with mild allergic rhinitis according to ARIA classification were included. Finally, patients with chronic cough, asthma, atopic dermatitis, or severe concomitant diseases were also excluded. The subjects were not allowed to use any anti-allergic medication during the course of the study, as well as other drugs that might affect sleep or daytime performance.

All anthropometric measurements were performed with the subjects wearing light clothes without shoes. Height was measured to the nearest 0.01 m using a wall-mounted stadiometer. Body weight was determined to the nearest 0.05 kg using a calibrated balance beam scale. BMI was calculated as weight (kg) divided by the height-squared (m^2). Body circumferences were measured with a flexible tape, with the subject in an upright position at the end of a gentle expiration, at the following levels: waist (midway between the lower rib margin and the superior anterior iliac spine) and hip (the widest circumference over the great trochanters). Neck circumference was determined at the level of the cricothyroid membrane.

The study was approved by the ethics committee of our medical faculty. The written informed consent for participation was obtained from all individuals.

Epworth sleepiness scale

All patients filled Epworth sleepiness scale (ESS) [13] to determine excessive daytime sleepiness (Table 1). The ESS is composed of eight situations requiring various degrees of vigilance. Ratings for each of the eight situations were totaled for each patient. ESS scores greater than ten are consistent with excessive daytime sleepiness.

Table 1 Epworth sleepiness scale

Likelihood to fall asleep in the following situation	
Situation	Chance of dozing (0–3)
1. Sitting and reading.....	
2. Watching TV.....	
3. Sitting, inactive in a public place..... (e.g., waiting room, a theater, or a meeting)	
4. As a passenger in a car for an hour without a break.....	
5. Lying down to rest in the afternoon when circumstances permit.....	
6. Sitting and talking to someone.....	
7. Sitting quietly after lunch without alcohol.....	
8. In a car while stopped for a few minutes in traffic.....	
0- Never dozing	
1- Slight chance of dozing	
2- Moderate chance of dozing	
3- High chance of dozing	

Allergy tests

A battery of seven inhalant antigens (*Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, specific grass, tree, mold, weed, and epidermal mixture) (Stallergens S.A.-Pasteur France) was searched by skin prick tests (SPT). Skin prick tests were performed in each subject using the above mentioned antigens on the volar side of the forearm, according to the guidelines of the subcommittee on skin tests of the European Academy of Allergy and Clinical Immunology (EAACI). Histamine hydrochloride (1 mg/ml) and normal saline solutions were used as positive and negative controls, respectively. The skin prick tests were read after 15 min, and a wheal of at least 3 mm greater than the negative control was considered positive. Atopy was defined as a positive response to at least one of the allergens tested. Perennial allergens were considered to be dog dander, cat dander, house dust mites, and mold mixture.

Polysomnographic (PSG) evaluation

Polysomnograms (Grass Technologies, Twin PSG Software) included eight channels of electroencephalogram, right and left outer canthus electrooculogram, submental electromyogram (EMG), nasal thermistor, airflow, thoracic and abdominal effort, oxygen saturation, anterior tibialis EMG, electrocardiogram, and snore microphone. Staging was performed according to the criteria of the American Academy of Sleep Medicine's manual for sleep scoring version-2 which was published in 2012 [14]. Respiratory events were scored as apneas or hypopneas. Apnea was defined as a 90 % reduction in airflow for at least for 10 s duration. Hypopnea was defined as a 30 % or greater decrease in flow lasting at least 10 s and associated with a 3 % or greater oxyhemoglobin desaturation.

Total number of apneas and hypopneas in 1 h was called the apnea-hypopnea index (AHI). The patients were classified according to the severity of OSAS (Table 2). Total number of apneas and hypopneas in 1 h at the supine position was called the supine apnea-hypopnea index (SAHI). Total number of apneas and hypopneas in 1 h at the non-supine position was called the non-supine apnea-hypopnea index (NSAHI). Total number of apneas and hypopneas in 1 h at the rapid eye movement (REM) sleep period was called the REM apnea-hypopnea index (RAHI). Total number of apneas and hypopneas in 1 h at the non-REM period was called the non-REM apnea-hypopnea index (NRAHI). Respiratory events were derived primarily from the nasal cannula/pressure transducer. The oxygen desaturation index (ODI) was defined as the total number of episodes of oxyhemoglobin desaturation ≥ 4 % from the immediate baseline, ≥ 10 s but < 3 min, divided by the total sleep time. Minimum oxygen saturation (MOS) levels were recorded.

Statistics

All results were expressed as means $\pm$ (SD) values. Significance of difference between groups was assessed by unpaired Student's *t* test or Mann Whitney *U* tests for continuous variables. The chi-square test or Fischer's exact test was used for testing prevalence between groups. The statistical analysis was performed using SPSS-11 program and *p* values < 0.05 are considered significant.

Results

A total of 150 patients (mean age 44.2 ± 11.4 years, 130 male) presenting typical symptoms of sleep apnea had polyso

Table 2 The Classification of the severity level of OSAS according to the American Academy of Sleep Medicine

Apnea/hypopnea index	Level
<5	Simple snorer
5–15	Mild OSAS
15–30	Moderate OSAS
≥ 30	Severe OSAS

mnography test for all night. Fifty-five of them were diagnosed as PAR (group-1). There were no differences for the average age, gender, body mass index, and neck circumference measurements between allergic and non-allergic patients (Table 3). The most common two allergens for group-1 were mites (37.2 %) and mold mix (36.3 %). Prevalence of skin test positivity was 37.2 % for *D. farinae*, 36.3 % for mold mix, 33.6 % for *D. pteronyssinus*, 18.6 % for animal dander mixture, 28.1 % for grass mix, 16.8 % for tree mix, and 11.7% for weed mix. Thirty-nine patients had allergy from more than one inhalant antigen. The mean total immunoglobulin E (IgE) level for allergic and non-allergic patients was 189.5 ± 13.2 and 32.4 ± 6.9 IU/ml, respectively.

Epworth sleepiness scale scores were similar for both groups. There were no significant differences between groups for polysomnographic findings including sleep stages, respiratory, and oxygen parameters (Table 4). The simple snoring was diagnosed in 23 allergic and 31 non-allergic patients. OSAS was classified as mild in 18 allergic and 28 non-allergic patients, moderate in 5 allergic and 17 non-allergic patients, and severe in 9 allergic and 19 non-allergic patients (Fig. 1).

Although we found that mean AHI values were higher in non-allergic group, this was not statistically significant. Also, non-allergic group seemed more supine-dependent, but also this was not statistically significant (Fig. 2).

Discussion

Allergic diseases are common conditions that may influence sleep through various mechanisms. In our study, we examine the effect of the presence of PAR on polysomnographic measurements of our patients with sleep-related breathing symptoms like snoring, witnessed apnea, or excessive daytime sleepiness. Nevertheless, a linear relation between nasal obstruction and the severity of sleep-disordered breathing was not detected. Our data pointed out that the presence of PAR did not affect polysomnographic parameters compared with other patients without any nasal inflammation. Also, according to our findings, presence of PAR did not increase the prevalence of OSAS. Therefore, by these results, patients with

Table 3 Demographic and anthropometric measurements and findings

	Allergic n: 55	Non-allergic n:85
Mean age (years)	43.2 ± 1.01	44.8 ± 1.2
Gender male/female	5/50	15/80
Body mass index	29.4 ± 4.6	30.2 ± 5.2
Neck circumferences (cm)	41.2 ± 2.4	41.0 ± 2.3
Epworth sleepiness score	11.5 ± 5.7	10.4 ± 5.04

persistent allergic rhinitis (PAR) could be protected from the effect of daytime use of anti-allergic medication in order to decline sleep-related breathing symptoms in clinic practice.

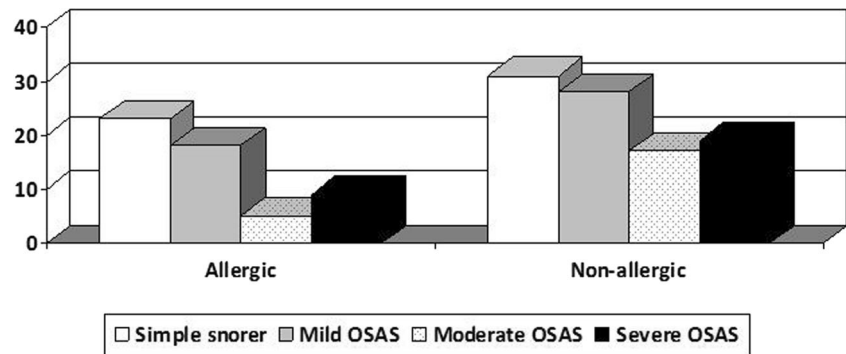
Although sleep-disordered breathing diseases are mostly multifactorial, the main problem behind these conditions is an increase in airflow resistance due to the decrease in airway diameter. At a certain point, high resistance inhibits airflow temporarily [7]. Also, increased upper and lower airway resistance due to circadian fluctuations in inflammatory cytokines and a dip in corticosteroid levels at night can be thought as a risk factor for apnea and sleep disturbances [15, 16]. Symptoms of PAR can be exacerbated during sleep because of close exposure to house dust mites in bed, and nasal blockage is worst in supine position [17]. Besides these conditions, PAR is rarely found in isolation and needs to be considered as a part of systemic comorbid conditions such as asthma and chronic sinusitis. The inflammatory mediators and cytokines that have been released as a part of a systemic disease process may disturb sleep and cause fatigue during the day. Higher levels of the cytokines interleukin (IL)-1 β , IL-4, and IL-10 in allergic patients compared with healthy individuals have been

Table 4 Polysomnographic findings

	Allergic	Non-allergic
Sleep stages (%)		
Stage-1	13.2 ± 8.9	13.8 ± 6.3
Stage-2	47.3 ± 9.6	47.6 ± 8.9
Stage-3	21.2 ± 9.7	21.1 ± 8.7
REM	18.2 ± 7.2	17.1 ± 6.7
Respiratory parameters		
Mean AHI	14.8 ± 2.1	20.6 ± 2.6
Supin AHI	24.8 ± 2.9	30.2 ± 3.2
Nonsupin AHI	6.6 ± 1.3	12.2 ± 1.9
Arousal index	18.7 ± 1.7	24.1 ± 2.3
Oxygen parameters		
ODI	15.0 ± 2.2	17.6 ± 1.9
Mean oxygen saturation (%)	94.2 ± 2.4	92.9 ± 3.1
Minimum oxygen saturation (%)	82.6 ± 1.1	81.7 ± 1.02

AHI Apnea-hypopnea index, ODI Oxygen desaturation index

Fig. 1 Sleep-disordered breathing problems in both groups



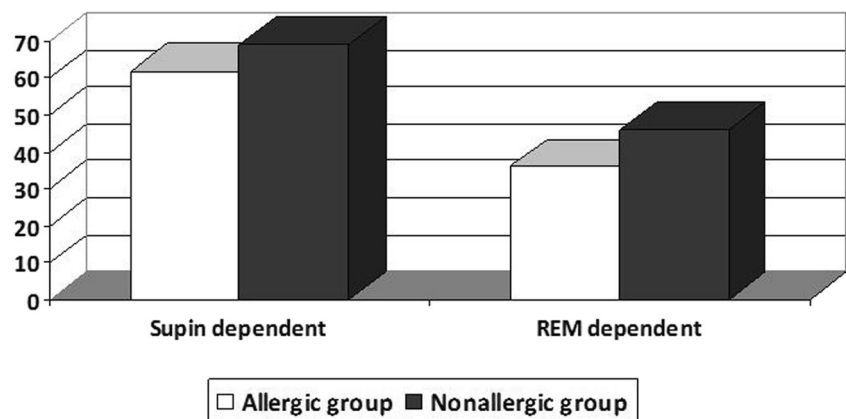
shown to correlate with increased latency to REM sleep, decreased time in REM sleep, and decreased latency to sleep onset [18]. We found that sleep quality, sleep maintenance, duration of REM and stage 3 sleep, AHI, and ODI ratios were similar between mild PAR and control group.

Leger et al. showed that the impact of severity of rhinitis but not its frequency was significant for sleep parameters [19]. Bousquet et al. found that the impact of severity of rhinitis on life quality was stronger than duration of rhinitis [20]. We could not find any influence of PAR on sleep latency and sleep stages. Although patients without PAR seemed to be more supine-dependent, this was not statistically significant. The main limitation of our study was that our patient number was relatively small because of our rigid inclusion criteria. We only included pure allergic newly diagnosed rhinitis patient in order to exclude influence of medicines. And also, we excluded patients with comorbid diseases, and this maintained us to test solely the role of PAR on sleep. Central H₁ receptors are classically associated with sedation. This effect is reduced in second generation H₁ antihistamines. Antihistamine oral decongestant combinations like pseudoephedrine are often used, and insomnia has been reported in these combinations [21]. When pretreatment and post-treatment apnea-hypopnea index (AHI) values in 80 patients with both OSAS and AR were compared, the most significant difference was observed in the nasal steroid plus antihistamine group ($p < 0.05$). The

ESS results and duration of oxygen saturation $<90\%$ were significantly decreased in the nasal steroid and nasal steroid plus antihistamine combination groups after treatment ($p < 0.05$) [22]. All of our patients were drug-free, so one of the strength of our study is that we exclude the effects of drugs on sleep and polysomnographic parameters.

The role of chronic nasal congestion at night as a risk factor for snoring was confirmed in a population-based cohort study ($n = 4916$) [23]. By reducing the internal nasal diameter and increasing airway resistance, patients with nasal congestion have 1.8 times more moderate to severe sleep-disordered breathing than in patients with AR without nasal congestion. Another limitation of our study was that we did not evaluate nasal congestion and measure nasal congestion objectively by nasal airflow (peak nasal inspiratory flow) or by airway resistance/conductance (rhinomanometry). Stuck et al. [24] searched daytime sleepiness and polysomnographic findings before and during pollen season in 25 patients with seasonal allergic rhinitis. Statistically significant differences were also found for AHI although the changes were only minimal, and all values were within normal ranges. In our study, we included perennial allergic patients. Meng et al. [25] showed that there were statistically significant differences between PAR patients and healthy controls in most polysomnographic parameters including sleep efficiency, arousal index, and average oxygen saturations. On the other hand, they found similar

Fig. 2 Distribution of OSAS subgroups



apnea-hypopnea index for the two groups. In another study, Kramer et al. [26] searched the influence of AR on sleeping behavior or polysomnography parameters in 119 patients with presenting typical symptoms of sleep apnea. They concluded that nasal obstruction due to allergic rhinitis did not constitute a risk factor for OSAS. Duchna et al. [27] detected increased positive allergy skin tests in 75 patients suffering from OSAS but failed to demonstrate important factors in pathologic sleep variables such as AHI, AI, or HI. Similarly, in our study, we also observed that patients with PAR have similar polysomnographic findings compared with the ones without PAR but same sleep symptoms.

As a conclusion, although lots of evidence suggests that patients with PAR might have worse sleep quality than normal healthy subjects without having effects on breathing, they might have similar sleep findings with the ones who had sleep-related breathing symptoms. The limited number of patients in this study meant that the statistical significance of the observed pathologic differences in sleeping parameters could not be elucidated. However, this group of patients would appear to be a promising target for further studies.

Compliance with ethical standards

Declaration of conflicting interests All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements) or non-financial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

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Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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