Original Article

Effects of continuous positive airway pressure on nasal and pharyngeal symptoms in patients with obstructive sleep apnea*

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Background: Nasal and pharyngeal symptoms are common in patients with obstructive sleep apnoea (OSA) treated with nasal continuous positive airway pressure (CPAP). However, these symptoms are common in OSA patients even before the treatment.

Objectives: Determine the impact of nasal CPAP on nasal and pharyngeal symptoms in OSA patients.

Method: Thirty-five adult patients (28 males), age 54±10 years old, with OSA diagnosed by polissonography. All patients answered to a questionnaire about the presence and quantification of sneezing, rhinorrhea, nasal pruritus, obstruction and bleeding, nasal and pharyngeal dryness. The questionnaire was answered before and after at least three months of CPAP therapy.

Results: The apnea-hypopnea index was 50±25 events per hour. Twenty six patients (74%) presented at least one naso-pharingeal symptom before treatment. Nasal obstruction was the most common symptom, being referred by 18 patients (51%). Among the patients that were initially assymptomatic (n=9), 78% developed adverse nasal reactions to CPAP. In contrast, among the patients that presented nasal symptoms before treatment, there was a significant reduction in nasal obstruction, nasal and throat dryness scores as well as nasal bleeding after CPAP therapy.

Conclusions: Nasal na pharyngeal symptoms are frequent in OSAS patients. CPAP therapy may originate nasal symptoms in patients previously assymptomatic, as well as reduce the intensity of these symptoms in patients that are previously symptomatic.

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Key words: Obstructive sleep apnea syndrome. Nasal obstruction. Continuous positive airway pressure, compliance.

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INTRODUCTION

Obstructive Sleep Apnea (OSA) is defined by recurrent episodes of obstructive apnea or hypopnea during sleep accompanied by daytime sleepiness or cardiovascular dysfunction[1]. In individuals with OSA, apnea or hypopnea episodes are caused by physiological relaxation of the pharyngeal musculature during sleep, with consequent narrowing or complete obstruction of upper airways[1]. Epidemiological data from the USA show that 2% to 4% of middle-aged individuals present OSA[2]. In addition to its high prevalence, the high morbidity observed makes OSA a growing public health problem[2].

The treatment of choice for moderate and severe OSA is nocturnal administration of continuous positive airway pressure (CPAP) through the use of a nasal mask[1]. Nasal CPAP virtually eliminates the respiratory events as soon as an appropriate pressure is established. Despite being highly effective, adherence to CPAP is variable, and intolerance to the mask is a common clinical problem[3]. Continuous airflow in the upper airways may cause discomfort, often associated with nasopharyngeal complaints, particularly nasal dryness[4].

Nasopharyngeal symptoms such as nasal and throat dryness, rhinorrhea and sneezing are common in patients with OSA, even prior to the initiation of CPAP treatment[5]. One of the most common symptoms in such patients is nasal obstruction. There are theories that implicate nasal obstruction in the genesis of OSA[6]. Nasal obstruction leads to an increase in the inspiratory effort, with an increase in negative intraluminal pressure. This leads to upper airway instability and a greater tendency for pharyngeal collapse[6]. Although nasal and pharyngeal symptoms are common in patients using CPAP[6,7], it is not clear whether the symptoms were present prior to the initiation of treatment. Brander[5] described an increase in the frequency and intensity of most nasal symptoms after the use of CPAP. In contrast, Lojander[7] found no significant alterations in the frequency of nasal symptoms after CPAP treatment.

The objective of this study was to determine the impact of nasal CPAP on the characteristics, frequency and intensity of nasopharyngeal symptoms in patients with moderate to severe OSA.

METHODS

We studied 35 adult patients with a mean age of 54 ± 10 years (mean ± standard deviation). The sample consisted of 28 men and 7 women treated consecutively at the Sleep Laboratory of the *Instituto do Coração* (Heart Institute), a sector of the *Faculdade de Medicina da Universidade de São Paulo* (University of São Paulo School of Medicine) *Hospital das Clínicas*. All patients underwent complete polysomnography, which included continuous monitoring of electroencephalography (2 central and 2 occipital channels), submental and tibialis anterior electromyography, electrooculography, air flow through nasal cannula and thermistor, thoracic and abdominal bands, snore sensor, position detector and pulse oximetry.

Only patients with moderate to severe OSA who had access to the use of CPAP were included. Those who discontinued regular monitoring were excluded. Once the polysomnographic diagnosis had been made, patients underwent another polysomnography evaluation with CPAP titration. According to the laboratory routine, all patients attended a respiratory physiotherapy session in order to adapt to CPAP. On this occasion, the basic concepts of the use of the device that generates positive pressure were explained and patients used CPAP with a nasal mask for approximately 60 minutes. During the same session (prior to the use of the machine), patients were asked to fill out a questionnaire regarding the presence and quantification of nasopharyngeal symptoms. The questionnaire comprised questions regarding sneezing, coryza, pruritus, dryness, nasal obstruction and nasal bleeding. The presence of throat dryness was also investigated. Each symptom was subjectively quantified by each patient in the following way: (0) absent; (1) mild: occasional symptoms, causing no impairment during daily activities; (2) moderate: frequent symptoms, causing discomfort during daily activities; or (3) severe: constant symptoms, impairing during daily activities. In addition, subjective sleepiness was determined using the Epworth Sleepiness Scale [8]. The subjective Epworth Sleepiness Scale evaluates, by means of a standardized questionnaire, the chance that an individual will doze off in 8 different situations. Scores range from 0 to 24 points, and values higher than 10 are considered indicative of excessive sleepiness.

Patients were systematically contacted via telephone (or through follow-up visits, if necessary) after one week, one month and three months of CPAP use. According to the laboratory routine, patients are then reevaluated every 6 months. The questionnaire regarding nasal symptoms was used

again in the form of a personal interview after at least 3 months of CPAP use. All patients were treated with a positive airway pressure generator (CPAP Sullivan® S5 and S6 - ResMed or Solo LX plus® - Respironics, Murrysville, PA, USA) with an integrated hour-of-use counter (hour meter), which registers the number of hours that the machine is in operation. Patient adherence to CPAP was estimated through determination of the number of hours registered by the pressure generator device divided by the number of days since the beginning of treatment. The protocol was approved by the ethics committee of the institution.

The Student's *t*-test was used for comparison of mean age and mean apnea-hypopnea index in patients with and without nasal symptoms. The Wilcoxon nonparametric test was also used to compare the mean scores of each nasal symptom prior to and after CPAP therapy. In two-tailed tests, the level of significance adopted was $p < 0.05$.

RESULTS

The apnea-hypopnea index was 50 ± 25 events per hour of sleep, ranging from 24 to 112 events per hour. The level of subjective sleepiness, determined by the Epworth Scale, was 15 ± 6. Of the 35 patients studied, 26 (74%) reported at least one nasopharyngeal symptom prior to the initiation of CPAP treatment. The most common symptom was nasal obstruction, which was observed in 18 patients (51%). Other symptoms were dry throat (16 patients), dry nose (10 patients), sneezing (9 patients), nasal pruritus (7 patients), coryza and nasal bleeding (both reported by 6 patients). Patients with and without nasopharyngeal symptoms prior to the beginning of treatment (n = 26 and n = 9, respectively) were similar regarding age (54 ± 9 and 55 ± 11 years, respectively, p = 0.82) and apnea-hypopnea index (39 ± 25 and 49 ± 26 events/hour, respectively, p = 0.34).

By the time of the second evaluation, the patients were using CPAP for 5.7 ± 1.9 hours per night. The CPAP level was 9.7 ± 1.5 cmH_2O (ranging from 5 to 13 cmH_2O). Re-evaluation was carried out 13 ± 21 months after the initiation of treatment. Table 1 shows the distribution and intensity of the various symptoms after nasal CPAP therapy in patients who were initially asymptomatic. Among the 9 patients initially presenting no nasopharyngeal symptoms, only 2 remained asymptomatic after the use of CPAP. Three patients developed 2 or more symptoms, and four patients developed 1 symptom. The most common symptoms were dry nose, coryza, dry throat and nasal obstruction. Bleeding was observed in only one patient.

Table 2 shows the intensity and evolution of nasopharyngeal symptoms in the 26 patients with at least one baseline symptom. In this analysis, each symptom was considered independently, and Table 2 also shows the number of patients with each specific symptom. In general, there was a tendency toward reduction in the mean intensity of nasopharyngeal symptoms. We found statistically significant reductions in the intensity of the symptoms obstruction ($p < 0.01$), dry nose ($p < 0.02$), nosebleed ($p < 0.01$), and dry throat ($p < 0.01$). Despite an overall reduction in the intensity of the symptoms, the effect of the CPAP was heterogeneous. In 13 patients, there was at least one new nasopharyngeal symptom after the use of CPAP. With regard to dry nose, for example, there was a significant decrease in its mean intensity, although there was an increase in the number of patients who reported it after CPAP therapy (10 before and 14 after) (Table 2).

DISCUSSION

The frequency of nasopharyngeal symptoms in our study sample prior to the beginning of CPAP treatment was high and similar to that previously described in the literature[4,5,7]. We found indices for obstruction, as well as for dry nose and dry throat, of nearly 50%. The impact of CPAP on nasopharyngeal symptoms varied widely. When analyzing only patients who were initially asymptomatic, we observed adverse nasopharyngeal reactions in a significant number of cases. However, the side effects were generally mild. Despite the fact that a large number of nasopharyngeal problems were seen in OSA patients on CPAP therapy, the symptoms were generally present prior to its use.

Our study was based on the clinical observation that OSA patients present nasopharyngeal symptoms, particularly nasal obstruction. In most cases, patients do not spontaneously report the symptoms and must be actively questioned. This observation is consistent with those of the study carried out by Ohki et al.[9]. The authors studied the relationship between mouth breathing and nasal obstruction in patients with and without OSA. Although nasal resistance is higher in OSA patients, most of the patients did not complain

TABLE 1
Occurrence of adverse nasopharyngeal symptoms after nasal continuous positive airway pressure treatment in initially asymptomatic patients (*n* = 9).

	Dry nose	Coryza	Dry throat	Obstruction	Pruritus	Sneezing	Epistaxis
Mild	1	2	–-	1	1	–-	–-
Moderate	1	1	1	1	–-	1	1
Severe	1	–-	1	–-	1	–-	–-

TABLE 2
Number of patients (and the respective percentages) and intensity of nasopharyngeal symptoms before and after the use of nasal continuous positive airway pressure in 26 initially asymptomatic patients

	PRE-CPAP Intensity					POST-CPAP Intensity				
SYMPTOM	n (%)	M	Mod	S	Mean score (Med)	n (%)	M	Mod	S	Mean score (Med)
Nasal Obstruction	18 (51)	6	6	6	2.0 (2.0)	14 (40)	8	5	1	1.0* (1.0)
Dry throat	16 (46)	4	4	8	2.3 (2.5)	14 (40)	9	3	2	0.7* (1.0)
Dry nose	10 (29)	1	3	6	2.4 (3.0)	14 (40)	9	4	1	0.8* (1.0)
Sneezing	9 (26)	2	4	3	2.0 (2.0)	11 (31)	6	2	3	1.3 (1.0)
Pruritus	7 (20)	2	2	3	2.0 (2.0)	9 (26)	8	0	1	1.0 (1.0)
Coryza	6 (17)	2	3	1	1.9 (2.0)	10 (29)	6	4	0	0.7 (1.0)
Epistaxis	6 (17)	4	1	1	1.5 (1.0)	2 (5)	1	1	0	0.0* (0.0)

CPAP: continuous positive airway pressure; *n*: number of patients; M: mild; Mod: moderate; S: severe; Med: median
*$p < 0.05$ when the mean score of post-CPAP symptoms was significantly lower than the initial mean score

of a sensation of nasal obstruction. Therefore, even though several OSA patients began to complain of this nasal symptom after the use of CPAP, the symptom was often present prior to the initiation of treatment[7].

Population studies suggest that there is a relationship between nasal obstruction and OSA. In a population analysis of 5000 patients, Young et al.[10] asserted that patients with symptoms of nasal obstruction had a greater chance of presenting snoring and excessive daytime sleepiness. In addition, patients with allergy-related nasal obstruction were 1.8 times more likely to present moderate to severe OSA. There are at least two mechanisms through which nasal obstruction may contribute to the genesis of OSA. Nasal obstruction leads to increased inspiratory effort and higher negative intraluminal pressure, thereby generating upper airway instability and a stronger tendency toward pharyngeal collapse[4]. Furthermore, as demonstrated by Fitzpatrick et al.[11], nasal obstruction may lead to mouth breathing, which, during sleep, significantly increases upper airway resistance. Maurice et al.[12] demonstrated that mouth opening in normal individuals aggravates the tendency towards airway collapse during sleep and may contribute to OSA. On the other hand, Miljeteig

et al.[13] found no correlation between the degree of nasal resistance and the severity of OSA. It has been recently postulated that nasal obstruction is not the major factor involved in the genesis of OSA but functions as an important co-factor[14,15]. Regardless of the actual role of nasal obstruction in the genesis of OSA, it is common and, when mild to moderate in intensity does not impede appropriate treatment with nasal CPAP.

Since the objective of our study was to determine the impact of nasal CPAP on nasopharyngeal symptoms, we only included patients with moderate to severe apnea. Since the use of CPAP is not financed by the government or reimbursed by private health insurance companies, patients paid for the machines with their personal resources, and therefore probably represent a sample of patients who were highly motivated to use the treatment. Another significant limitation of our study is that we only included patients in regular follow-up treatment. This may explain the high adherence to CPAP in our sample. Therefore, since we studied a selected population, we cannot make any inferences about the impact of nasal obstruction on patient adherence. Although it seems obvious that nasal obstruction would make it difficult to adapt to nasal CPAP, some authors found that there was no relationship between adherence to CPAP and nasal obstruction[16]. Lojander et al.[7] found that the frequency of nasopharyngeal symptoms prior to and after CPAP treatment was similar between patients who discontinued CPAP and those who continued the treatment.

An interesting finding of our study, which is consistent with clinical observations, was that many patients presented a decrease in the intensity of nasopharyngeal symptoms after CPAP treatment. Although there was a slight increase in the percentage of patients who reported nasal symptoms after the use of CPAP, the mean intensity of symptoms such as dry nose, dry throat and epistaxis decreased significantly after the beginning of treatment. Our hypothesis is that many OSA patients are mouth breathers and that, with the use of nasal CPAP, such patients begin breathing through their nose during sleep. Nasal breathing would restore upper airway normal physiology and could contribute to a reduction in the intensity of nasopharyngeal symptoms.

We concluded that the evolution of nasopharyngeal symptoms after CPAP treatment is variable in OSA patients. The CPAP therapy may provoke nasal symptoms in patients who were asymptomatic prior to treatment while reducing their intensity in initially symptomatic patients. Despite the high incidence of nasopharyngeal symptoms in OSA patients on CPAP therapy, it is difficult to attribute these symptoms to the treatment.

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