

The Main Symptoms in Dorsal Sleep Apnea - Hypopnea Syndrome

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Received:27.10.2013 / Accepted:26.11.2013 / Published online: 17.12.2013

Abstract

OSAHS is a chronic, multifactorial disease, accompanied by significant and complex symptoms. The aim of this study was to evaluate the relationship between OSAHS and dorsal AHI in order to improve early diagnosis of dorsal sleep apnea-hypopnea syndrome. There were significant statistical differences between: the dorsal AHI Mean of the group without excessive daytime sleepiness as opposed to the dorsal AHI Mean of the group with excessive daytime sleepiness; the dorsal AHI Mean of the group without snoring as opposed to the dorsal AHI Mean of the group with snoring; the dorsal AHI Mean of the group without restless sleep as opposed to the dorsal AHI Mean of the group with restless sleep; the dorsal AHI Mean of the group without dyspnea as opposed to the dorsal AHI Mean of the group with dyspnea; the dorsal AHI Mean of the group without night sweats as opposed to the dorsal AHI Mean of the group with night sweats; the dorsal AHI Mean of the group without irritability as opposed to the dorsal AHI Mean of the group with irritability and the dorsal AHI Mean of the group without nightmares as opposed to the dorsal AHI Mean of the group with nightmares. Through this study we highlighted that excessive daytime sleepiness and snoring are prevalent symptoms in dorsal OSAHS. The presence of these symptoms in patients with sleep disorders may improve early diagnosis and the choice of an appropriate treatment for dorsal sleep apnea- hypopnea syndrome, thus participating in improving the patient's life quality.

Keywords: Apnea; Hypopnea; Dorsal; Symptoms.

Introduction

Obstructive apnea-hypopnea syndrome is a chronic [1], multifactorial [2] disease, characterized by the partial or complete collapse of the upper airway during sleep [1] causing hypopnea and apnea, leading to chronic intermittent hypoxia, arousals during sleep, resulting in fragmentation and disturbance of its architecture [3]. At a person with obstructive sleep apnea- hypopnea syndrome (OSAHS), the upper airway muscles relax excessive during sleep. This allows the tissue supported by upper airway muscles (tonsils and nasal polyps) to be withdrawn in the airway with each breath, obstructing the air flow. Following this obstruction the airway efforts increase in order to defeat it, but the last arousal quickly shortens this process, which is followed by deep breaths [4]. A result of

these important events can also be the neuro-structural changes [3].

By sleep initiation the repetition of a vicious cycle occurs. During arousals, airway obstruction is evidenced by panting and loudly snoring [4]. The sound of snoring is the result of soft tissue vibrations in the pharynx, soft palate, uvula and epiglottis [5], which have specific acoustic characteristics, with a frequency of 5 to 136 Hz [4]. Snoring, most common in men than in women [6], reveals information about the position and the obstruction of the upper airway. The biological instability of the upper airway formation throughout the night, and especially during obstructive events, could lead to alterations of snoring properties [1]. Snoring is not a constant and fix occurrence, because it is influenced by: body position [1, 7], sleep stages, breathing airway (oral, nasal, or both), level and place of the upper airway narrowing. Snoring is often observed in inspiratory phase. Not all snoring episodes have the same characteristics and triggering mechanisms. In this way, it is vital to distinguish between two types of snoring: those which are successive and consecutive with respiratory cycles (regular snoring) and those that are separated by "non-snoring" breathing cycles and / or apnea (irregular snoring) [7].

The collapse of the upper airway has different consequences on the human body, ranging from noisy breathing (snoring) to a considerable cardiovascular morbidity [1]. Clinical studies show that OSAHS is strongly associated with serious chronic and life-threatening diseases such as: arterial hypertension, stroke, congestive heart failure, coronary heart disease, diabetes, obesity [4].

Like any other pathology OSAHS is accompanied by a complex symptomatology. These symptoms have an impact on both nighttime sleep and wakefulness. Among the nocturnal and diurnal symptoms of OSAHS can be listed: respiratory pauses, snoring, restless sleep, dyspnea, nocturia, night sweats, nightmares, excessive daytime sleepiness, morning headaches, irritability and behavior changes.

Excessive daytime sleepiness is a concern and an important public health problem for most patients with sleep disorders. The international classification of sleep disorders includes excessive daytime sleepiness as a key feature for three diagnoses: narcolepsy, hypersomnia and behaviorally induced insufficient sleep syndrome. This is associated with a wide range of pathologies, including psychiatric, neurological, pulmonary and cardiac diseases. Frequently, there may not be an identifiable cause and the only diagnosis may be of idiopathic hypersomnia. However the most common causes could be found in a quality and quantity of sleep disorders or other factors. Most commonly, insufficient sleep duration is responsible for the occurrence of this symptom [3].

Emotional regulation, cognitive, somatic and autonomic functions, including sleep stages and respiratory control are integrated into the structures of the limbic system (anterior thalamus, hypothalamus, hippocampus, fornix, and mammillary bodies). Chronic intermittent hypoxia and sleep fragmentation cause cell damage in the hippocampus that can contribute to neuropsychological impairment [2]. Neuropsychological impairment includes cognitive dysfunction, namely short-term memory dysfunction, which could damage including the remembering of dreams. OSAHS feature's sleep fragmentation is associated with an increased percentage of light sleep and repeated arousals that may increase the chances that the patient is aware of the dreaming process [8].

The process of dreaming occurs during Rapid Eye Movement (REM) sleep and is often present on awakening. This is a mental experience that can have both positive and negative consequences on feelings and emotional state of patients. The typical nightmare is a coherent sequence of the dream that seems real and is becoming increasingly worrying as it unfolds. The negative emotions that characterize nightmares usually involve anxiety, fear or terror, also including rage, roar, discomfort and disgust. Nightmares were differentiated from other vivid dreams by the dream's image feature expressed as an extreme perception of external reality. It contains mostly concentrations on the individual's immediate physical danger (threat of attack, fall, injury, death), but may also involve aggression towards others, potential personal failures, and other painful topics such as suffocation [9].

Symptoms of OSAHS have an important allure that can adversely affect the quality of patient's life. The position during sleep may also contribute to the increase of these symptoms. The medical literature describes various symptoms of OSAHS associated with polysomnographic parameters, but until now no study has reported relationships between them and the dorsal apnea-hypopnea

index (AHI). The present study aim was to assess the relationship between dorsal AHI and a part of the symptoms seen in OSAHS, in order to improve early diagnosis of dorsal sleep apnea-hypopnea syndrome.

Material and Method

The present study is a case - control study, conducted for a period of 6 months in the “Alpes-Leman” Sleep Laboratory in France.

Inclusion criteria: in the present study were included patients aged between 18 and 80 years, with a total sleep duration ≥ 180 minutes and patients with positive or negative diagnosis of OSAHS, but with at least one of the following related pathologies: cardiovascular diseases (arterial hypertension, stroke, arrhythmias, coronary artery disease), respiratory diseases (chronic obstructive pulmonary disease, asthma), metabolic and hormonal diseases (dyslipidemia, diabetes, hypothyroidism), otorhinolaryngology changes (micrognathism, retrognathia, enlargement of the tonsils / palate / uvula).

For each patient was made an individual assessment scheme, including demographic data (age, sex), various symptoms (excessive daytime sleepiness, snoring, restless sleep, dyspnea, night sweats, irritability, nightmares) and polysomnographic data (such as apnea type, total AHI, dorsal AHI). Patient's symptoms were collected from medical files. The same patient has had one or more associated symptoms.

The diagnosis of sleep apnea - hypopnea syndrome was performed using polysomnography (PSG). Polysomnography was performed with the "Morpheus hand-held with 34 channels recorded by Micromed s.p.a., which can be used in both hospital and ambulatory environment. Direct recording of signals in hospital involved the use of a fixed manner via the Micromed interface cable or fiber optic; Micromed Network interface through networking and Bluetooth wireless connection. Morpheus was connected to the “SystemPLUS EVOLUTION” version 1061, which integrates all the exams in a single application such as EEG (electro-encephalogram), video EEG, ambulatory PSG, video PSG, EMG (electromyogram), etc. Out of the 34 channels were used s: 8 channels for EEG, 2 channels for electro-oculogram, 2 channels for EMG chin and 4 for EMG legs, 3 channels for electro- cardiogram, pulse oximeter, larynx microphone for recording snoring, the respiratory flow nasal cannula, piezoelectric sensors for thoracic and abdominal respiratory movements, integrated sensor in patient unit for position during sleep, integrated light sensor to measure the time spent in bed. The analysis of each polysomnography was performed manually using the "Rembrandt Analysis Manager 7.5" programme. Sleep staging was interpreted into 30 seconds epochs and respiratory events within 4 minutes, according to the classification criteria of Rechtschaffen-Kales and the American Academy of Sleep Medicine. The diagnosis of OSAHS was established after performing polysomnography if the patient had an $AHI \geq 5$ / hour of sleep. The respiratory events (apnea and hypopnea) were defined using the internationally criteria. The assessment of the OSAHS's severity was established according to internationally validated criteria: mild: $AHI = 5-14$ / hour of sleep, moderate: $AHI = 15-29$ / hour of sleep, severe: $AHI \geq 30$ / hour of sleep.

Statistics. It was conducted a case-control study between dorsal apnea-hypopnea index and various symptoms associated to it. Descriptive analysis was performed of the individual parameters and the possible links between them: average groups / scatter / frequency charts (pie). In order to describe qualitative variables were used frequency tables, contingency tables, graphs or column type structure (pie). Comparison of the observed distribution theory was made with the following statistical tests: Chi2 test, Fisher's exact test. To describe quantitative variables we used the average (mean), standard deviation (SD), median (Me), frequency tables, histograms, box-plot charts. In order to compare means were applied: "t" Student test for independent samples or ANOVA variance analysis, in accordance with the conditions of independence, normality plots. The value of “p” was considered statistically significant when it was <0.05 .

Results

In this study were included 100 adult patients. Female gender was present in 43%, and the male in 57%.

61% of patients experienced excessive daytime sleepiness, 56% were snorers, 41% had asthenia, 25% restless sleep, 18% dyspnea, 17% night sweats, 11% irritability and 10% nightmares.

OSAHS was diagnosed in 60% of cases. Obstructive apnea was noted in 44% of patients, the dorsal obstructive apnea in 11% of subjects, and the central apnea in 5% of cases.

Among patients diagnosed with OSAHS: 5 patients had restless sleep, 3 night sweats, one dyspnea, one irritability and one patient nightmares.

46 patients had a dorsal AHI less than or equal to 8.3, 20 patients a dorsal AHI of between 8.4 and 16.6, 17 patients a dorsal AHI of between 16.7 and 24.9, and the remaining patients had a dorsal AHI over 24.5 / hour of sleep (Figure 1).

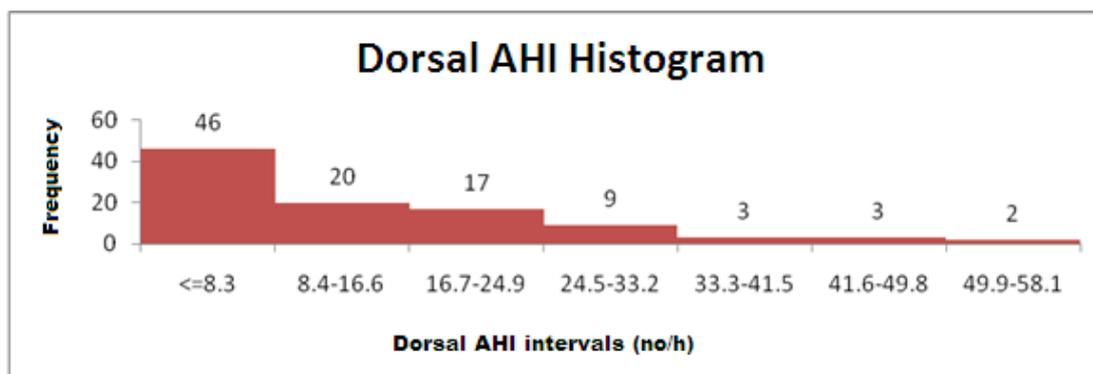


Figure 1. Distribution of patients on dorsal AHI periods

Between dorsal AHI mean of the group without excessive daytime sleepiness, and the dorsal AHI mean of the group with excessive daytime sleepiness was a statistical difference, represented in Table 1.

Table 1. Results of statistical tests between dorsal AHI means for the group without excessive daytime sleepiness versus the group with excessive daytime sleepiness

		Levene's Test		t-test for Equality of Means						
		F	P	t	df	p	m _{diff}	StErr _{diff}	95CI _{diff}	
Dorsal AHI (nr/h)	Equal variances assumed	1.498	.224	2.123	98	0.036	5.8776	2.7681	0.3844	11.3707
	Equal variances not assumed			2.235	93.509	0.028	5.8776	2.6299	0.6555	11.0996

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Comparing the dorsal AHI mean of the group without snoring, to the dorsal AHI mean of the group with snoring a statistical difference was demonstrated (Table 2). The variations of these groups were also equal.

Assessing the relationship between the presence of apnea and some of the symptoms mentioned above were highlighted statistical relationships with: restless sleep (Chi-square test: $p < 0.05$, OR = 11 (95% CI [3.29-39.07]), dyspnea ((Fisher's exact test: $p < 0.05$), night sweats (Fisher's exact test: $p < 0.05$), irritability ((Fisher's exact test: $p < 0.05$) and nightmares (Fisher's exact test: $p < 0.05$).

Table 2. Results of statistical tests between dorsal AHI means of the group without snoring versus the group with snoring

		Levene's Test		t-test for Equality of Means						
		F	p	T	df	p	m _{diff}	StErr _{diff}	95% CI _{diff}	
									Lower	Upper
Dorsal AHI (nr/h)	Equal variances assumed	0.416	0.521	2.121	98	0.036	5.7684	2.7201	0.3705	11.1663
	Equal variances not assumed			2.144	95.707	0.035	5.7684	2.6908	0.4269	11.1098

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Statistical differences were also found between: the dorsal AHI mean of the group without restless sleep as opposed to the dorsal AHI mean of the group with restless sleep; the dorsal AHI mean of the group without dyspnea as opposed to the dorsal AHI mean of the group with dyspnea; the dorsal AHI mean of the group without night sweats as opposed to the dorsal AHI mean of the group with night sweats; the dorsal AHI mean of the group without irritability as opposed to the dorsal AHI mean of the group with irritability and the dorsal AHI mean of the group without nightmares as opposed to the dorsal AHI mean of the group with nightmares. The comparison of variations in these groups was unequal (Tables 3-7).

Table 3. Results of statistical tests between dorsal AHI means for the group without restless sleep versus the group with restless sleep

		Levene's Test		t-test for Equality of Means						
		F	p	t	df	p	m _{diff}	StErr _{diff}	95% CI _{diff}	
									Lower	Upper
Dorsal AHI (nr/h)	Equal variances assumed	4.557	0.035	-3.694	98	0.000	-11.0361	2.9877	-16.965	-5.107
	Equal variances not assumed			-3.881	45.024	0.000	-11.0361	2.8434	-16.763	-5.309

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Table 4. Results of statistical tests between dorsal AHI means for the group without dyspnea versus the group with dyspnea

		Levene's Test		t-test for Equality of Means						
		F	P	t	df	p	m _{diff}	StErr _{diff}	95% CI _{diff}	
									Lower	Upper
Dorsal AHI (nr/h)	Equal variances assumed	16.208	0.000	-3.929	98	0.000	13.126	3.341	19.755	6.496
	Equal variances not assumed			6.494	68.059	0.000	13.126	2.021	17.159	9.093

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Table 5. Results of statistical tests between dorsal AHI means for the group without night sweats versus the group with night sweats

		Levene's Test		t-test for Equality of Means						
		F	p	T	df	p	m _{diff}	StErr _{diff}	95% CI _{diff}	
									Lower	Upper
Dorsal AHI (nr/h)	Equal variances assumed	12.662	.001	-3.392	98	0.001	-11.796	3.47756	18.697	4.895
	Equal variances not assumed			-5.619	59.463	0.000	-11.796	2.09914	15.996	7.596

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Table 6. Results of statistical tests between dorsal AHI means for the group without irritability versus the group with irritability

		Levene's Test		t-test for Equality of Means						
		F	p	t	df	p	m _{diff}	StErr _{diff}	95% CI _{diff}	
									Lower	Upper
Dorsal AHI (nr/h)	Equal variances assumed	13.768	0.000	-2.902	98	0.005	-12.290	4.235	-20.694	-3.886
	Equal variances not assumed			-6.857	64.42	0.000	-12.290	1.792	-15.870	-8.710

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Table 7. Results of statistical tests between dorsal AHI means for the group without nightmares versus the group with nightmares

		Levene's Test		t-test for Equality of Means						
		F	P	t	df	p	m _{diff}	StErr _{diff}	95% CI _{diff}	
									Lower	Upper
Dorsal AHI (nr/h)	Equal variances assumed	9.803	0.002	-2.497	98	0.014	-11.145	4.463	-20.001	-2.288
	Equal variances not assumed			-5.121	28.48	.000	-11.145	2.176	-15.599	-6.690

m_{diff} = mean difference; StErr_{diff} = standard error of the difference; 95%CI = 95% confidence interval

Discussion

The most common clinical cause of excessive daytime sleepiness is OSAHS. Daytime sleepiness is a sufficiently serious symptom that may be under-recognized. [3] This is a common public health problem [11] that is associated with car accidents and decreased efficiency at work. Sleep fragmentation, hypoxia and reduced perfusion resulted from episodes of apnea / hypopnea produce decreased cortical function, leading to the appearance of this characteristic symptom. Daytime sleepiness is correlated with the severity of OSAHS and can be assessed by the Epworth

questionnaire [2]. The factors that cause excessive daytime sleepiness in OSAHS are not well understood, usually interfering with the quality and quantity of sleep [3]. Sleepiness and its side effects could be multifactorial [10]. Excessive daytime sleepiness is present in the general population rate of 6 to 12% [11]. Only 34% of women experienced excessive daytime sleepiness [12]. In patients with severe obesity (body mass index > 35 kg / m²) prevalence of excessive daytime sleepiness is about 30% [11]. In 2002, the National Sleep Foundation reported sleepiness almost 7% every day and 9% several days a week. Patients with sleep apnea not only reported daytime sleepiness, but also declared they suffered of fatigue, asthenia and lack of energy; these complaints are more common than sleepiness in OSAHS [10].

In the current study, we decided to divide the patients after the absence or presence of symptoms, for example: the group without / with daytime sleepiness, the group without / with snoring, the group without / with restless sleep, etc. Patients who experienced excessive daytime sleepiness were found in a significant proportion, namely in 61% of cases. Between the dorsal AHI mean of the group without daytime sleepiness and the dorsal AHI mean of the group with excessive daytime sleepiness was found a significant statistical relationship. Another significant statistical relationship was also highlighted, between the dorsal AHI mean of the group without snoring as compared to the dorsal AHI mean of the group with snoring. This latter symptom - snoring - was present in 56% of cases.

The American Academy of Sleep Medicine has defined snoring as a sound originating in the upper airway which doesn't occur with apnea or hypoventilation, being caused by vibrations of different pharyngeal tissues. A person who snores more than 10-20% in a night, or more than 3 or 4 nights a week is classified as an ordinary snorer. An association between snoring and OSAHS was first observed in 1975. OSAHS is considered a progressive disease that usually begins early in life with regular snoring [4]. Snoring is a common and important clinical marker of OSAHS [7, 13], which occurs in 70-95% of patients [1]. It predicts the presence of OSAHS [14] and is one of the oldest and conscious symptoms [7]. Self-estimated prevalence of patient's snoring in the general population was between 16 and 18%, depending on the awareness, age, culture and the influence of their reporting bed partner [1]. Romero and colleagues found a positive predictive value of 84.7% for snoring and OSAHS, while Resta et al. found snoring to be a symptom present in 100% of patients diagnosed with OSAHS [14]. 51.9% of Taiwanese men older than 15 years had regular snoring [5]. Based on various epidemiological studies conducted between 1980 and 2007, the average prevalence of snoring in the general population was approximately 32% in men and 21% in women [4].

There are numerous studies designed to assess the severity of snoring [5]. Recently it was discovered that snoring vibration induces carotid vascular bed injury, mechanism involved in cardiovascular disease, particularly stroke. Hypothesis that snoring may precede or precipitate stroke is biologically plausible. In people who have already developed atherosclerosis, snoring could cause atheroma plaque rupture by mechanical stimulation. In addition, snoring could begin atherosclerosis. One study showed a strong correlation between the severity of snoring and carotid atherosclerosis prevalence: 20% for light snorers, 32% for moderate and 64% for severe snorers. However, the hypothesis that snoring alone causes cardiovascular or cerebrovascular diseases remains controversial in clinical and epidemiological terms. Snoring and sleep apnea are related but they are not completely overlapping phenomena [15]. In another study, laser Doppler perfusion monitoring combined with electrical stimulation (method used to test vascular reactivity) was conducted in the lining of the soft palate in patients with varying levels of upper airway obstruction and to control subjects. Regular snoring and mild OSAHS patients showed exaggerated vasodilation compared with the control group. This may be the result of minor injuries with consistently re-innervations that increase sensitivity to mechanical stimuli. In contrast, patients with severe OSAHS showed a significant decrease in vasodilatation compared with the control group, which could be due to complete loss of related C-fiber, representing a permanent injury. These disorders of microcirculation indicate a progressive damage at the local nerve to important snorers both with and without OSAHS [4].

Supine position during sleep is considered a cause of occurrence of snoring [16]. The dorsal AHI was significantly increased (> 8.4 / hour of sleep) at more than half of our study patients. Also

the partners of apnea or non-apnea snorers were the first to identify the role of body position on the severity of snoring or apnea to their bed partners. In 1984, Chest published a letter written by the wife of a patient. She cured her husband of his sleep apnea snoring problem by sewing a pocket on the back of his T-shirt, but leaving a hole in which she introduced light plastic balls and thus her husband avoided to sleep on the back. During the American War of Independence (1775-1783) and later during World War I (1914-1918), soldiers were advised to wear their backpacks (filled with a voluminous mass) during sleep, namely to avoid sleeping in the dorsal position in order to reduce snoring and thus to avoid highlighting their position in front of the enemy. Dorsal therapy, in any form, was found to have a significant influence on snoring and OSAHS severity [16].

Snoring, sleep apnea, sleep shortening and insufficient subjective sleep are independently associated with both falling asleep and with drowsiness during driving [13]. Snoring and OSAHS are the most prevalent diseases with respiratory problems during sleep. OSAHS is associated with morbidity and significant symptoms, such as daytime sleepiness, socially unacceptable snoring and deteriorating quality of life [16].

In this study the most common symptoms were excessive daytime sleepiness, snoring and asthenia, the latter being represented in 41% of cases. The other studied symptoms were highlighted in much lower percentages: 25% of patients had restless sleep, 18% dyspnea, 11% irritability and 10% nightmares. Correlating the dorsal AHI mean with various patient groups were shown statistically significant differences between dorsal AHI mean of the group without restless sleep as compared to the dorsal AHI mean of the group with restless sleep (Table 3) and between the dorsal AHI mean of the group without dyspnea as compared to the dorsal AHI mean of the group with dyspnea (Table 4). In the group of patients without restless sleep there was a risk of developing apnea 11 times higher than in the group with restless sleep. Statistically significant relationships were also highlighted between the presence of apnea and some of the studied symptoms, namely: restless sleep, dyspnea, night sweats, irritability and nightmares.

In a series of cross-sectional studies was documented an association between night sweats and a variety of related symptoms of sleep disorders, including general asthenia, restless legs syndrome, increased sleep latency, nocturnal arousals [17]. Possible causes of night sweats include malignancy, infection, endocrine diseases, neurological diseases, menopause, gastro-esophageal reflux disease, medication (antidepressants and antipyretics), substance abuse, and panic attacks and sleep disorders such as insomnia and OSAHS. In general population, night sweats were associated with increased daytime asthenia and sleep problems; this phenomenon is also seen in patients who come in sleep laboratories. At the general population, night sweats may be a marker of increased risk of OSAHS, and also of insomnia. A recent study found that 34% of 98 men with untreated OSAHS reported night sweats, which were reduced to 12% by applying CPAP (Continuous Positive Airway Pressure) therapy [18]. In our study, night sweats were reported in 17% out of 100 patients, and between the dorsal AHI mean of the group without night sweats as compared to the dorsal AHI mean of the group with night sweats (Table 5) were highlighted statistically significant relationships.

The main characteristics of OSAHS are nocturnal respiratory pauses interrupted by intermittent heavy snoring and excessive daytime sleepiness caused by sleep fragmentation [19]. OSAHS patients report a lower quality of life compared to control groups, namely: high levels of depression, cognitive deficits, irritability, mood swings and personality difficulties in personal relationships [9]. In this paper, between the dorsal AHI mean of the group without irritability as compared to the dorsal AHI mean of the group with irritability (Table 6) and the dorsal AHI mean of the group without nightmares as compared to the dorsal AHI mean of the group with nightmares (Table 7) were reported statistically significant relationships. There are previous studies that have reported that patients with OSAHS have negative dreams [19] at the awakening of apneic events, thus showing an increase in the frequency of nightmares [9]. Studies have suggested that severe sleep apnea can cause unpleasant dreams and adopting behaviors during dreams, symptoms that can be eliminated with CPAP treatment. Individuals diagnosed with OSAHS, complaining about recurring nightmares reported a significant improvement in both sleep and wakefulness due to CPAP treatment. Frequent nightmares are the most common symptom of posttraumatic stress disorder. Sleep apnea has been reported in more than 56% of patients with posttraumatic stress disorder. Compared to excessive daytime sleepiness, OSAHS is known to cause cognitive deficits that

include the decline of working memory and executive function deficits of the frontal cortex. One of the studies has demonstrated an increase in frequency of nightmares with increasing AHI. Understanding the relationship of sleep apnea - nightmares is important because the nightmares themselves can be a clinical issue that may contribute to lower overall quality of life of people with OSAHS. Nightmare distress may involve fear of falling asleep because of nightmares, with the difficulty of re-sleep after a nightmare, the latter interfering with sleep quality. It is plausible that lack of sleep recovery, resulted from OSAHS suffering, can be increased by the presence of nightmares and the fear of having nightmares, this combination worsening the welfare of the patient [19].

The relationship between the dorsal sleep apnea-hypopnea syndrome and the studied symptoms is important because it highlights the role of the supine position during sleep. The supine position is considered a potential risk factor in the development and emphasizing of symptoms of OSAHS. To determine its impact on patients with OSAHS, further studies, more precise, should be carried out, that should take into consideration the non-dorsal AHI. However, the present study revealed that some of the symptoms seen in OSAHS are significantly associated with the dorsal AHI, and therefore common with dorsal obstructive apnea-hypopnea syndrome.

Conclusions

According to our study, excessive daytime sleepiness and snoring could be considered predominant symptoms in dorsal OSAHS. Restless sleep is not a characteristic symptom for OSAHS and dorsal OSAHS.

About one in ten patients with dorsal OSAHS may present either irritability or nightmares. Nearly two out of ten patients who experience dyspnea or night sweats may have dorsal OSAHS. The relationship between symptoms of OSAHS and dorsal AHI has a double meaning as there is a mutual negative influence on them. This negative influence affects not only the patient's life quality, but also the morbidity and mortality associated with cardiovascular and metabolic diseases. This excessive daytime sleepiness and snoring in patients who come to sleep laboratories, participates in improving early diagnosis and proper treatment of dorsal OSAHS; reducing the occurrence of traffic accidents as well as improving both the life's quality of patients and their partners and the evolution of associated cardio-metabolic co-morbidities.

List of abbreviations

AHI = apnea-hypopnea index

CPAP = continuous positive airway pressure

EEG = electroencephalogram

EMG = electromyogram

OSAHS = obstructive sleep apnea-hypopnea syndrome

PSG = polysomnographic

REM = rapid eye movement

Conflict of Interest

The authors declare that they have no conflict of interest.

Acknowledgements

This research was conducted with partial financial support of European Social Fund, by the POSDRU 107/1.5/S/78702 project.

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