Obstructive sleep apnea syndrome and arterial hypertension – a complicated relationship? The role of controlling blood pressure values in patients with OSAS

Oana-Claudia Deleanu[1], Andra Elena Mălăuț[2], Ana Maria Nebunoiu[2], Miruna Mihaela Micheu[3], Florin Dumitru Mihălțan[1]

1. "Carol Davila" University of Medicine and Pharmacy, Bucharest, "Marius Nasta" Institute of Pneumophtisiology, Bucharest, Romania

2. "Marius Nasta" Institute of Pneumophtisiology, Bucharest, Romania

3. Clinical Emergency Hospital of Bucharest, Romania

Correspondence:
Oana-Claudia Deleanu, „Marius Nasta" Institute of Pneumophtisiology, Address: Șoseaua Viilor str. 90, Sector 5, Bucharest
e-mail: oanadeleanu@yahoo.com

Abstract

Background: *Arterial hypertension (HT) and obstructive sleep apnea syndrome (OSAS) are associated through cause-effect relationship. We aimed to study the effect of medication controlled hypertension on OSAS patients.*
Methods: *From 483 followed patients with OSAS, 252 associating HT; 142 patients of them (56.34%) received antihypertensive medication, 59 patients (41.54%) had controlled HT, 83 patients (58.46%) had uncontrolled HT. Demographic and anthropometric data, OSAS symptoms, comorbidities, apnea index (IA), apnea-hypopnea index (IAH), desaturation index, CPAP titration, CPAP failure rate were studiated regarding differences between patients with controlled and uncontrolled HT.*
Results: *Fifty nine patients with controlled HT were: 20 women (33.9%), 39 men (66.1%), with mean age of 56.08 years ± 11.33, with an average AHI of 53.61 ± 34.42/hour, an average of CPAP pressure prediction of 10.15 ± 2.43 cm H2O. Eighty three patients with uncontrolled HT were: 18 women (21.7%), 65 men (78.3%), with mean age 55 ± 9.06 years, with an average AHI of 61.91 ± 43.61/hour, an average of CPAP pressure prediction of 10.47 ± 2 cm H2O. Comparing with the controlled HT group, patients with uncontrolled HT reported morning headaches, morning fatigue and impotency in a higher rate (p=0.020, 0.018, 0.011 respectively); Epworth Sleepiness Scale was under 10 (cut-off for daytime sleepiness) in patients with controlled HT (p=0.001) and higher in those with uncontrolled HT. Patients with uncontrolled HT were diagnosed with HT for a longer period (p=0.006), had higher values of systolic and diastolic blood pressure at the time of the presentation. Statistically significant differences were found only for AHI post-CPAP (11.89/h vs. 22.30/h, p=0.013) and nocturnal desaturation index post-CPAP (6.03/h vs. 16.55/h, p=0.017), both higher in patients with uncontrolled HT. The hypothesis regarding existing differences related to the cardiovascular comorbidities was not supported.*
Conclusions: *Controlled blood pressure deletes sleepiness, a defining symptom for OSAS and reduces remaining symptoms (headaches, impotency and morning fatigue). Presence of OSAS symptoms is less common in the controlled HT group, making the OSAS more difficult to suspect. These patients may have a grater benefit from CPAP therapy – they have AHI post-CPAP and desaturations post-CPAP significantly lower than patients with uncontrolled HT.*
Keywords: *obstructive sleep apnea syndrome, controlled hypertension, uncontrolled hypertension*

Rezumat

Sindromul de apnee în somn de tip obstructiv și hipertensiunea arterială - o relație complicată? Rolul controlului valorilor tensionale la pacienții cu SASO
Ipoteze: *Hipertensiunea arterială (HTA) și sindromul de apnee în somn de tip obstructiv (SASO) sunt asociate printr-o relație de tip cauză - efect. Am studiat efectul hipertensiunii controlate medicamentos la pacienții cu SASO.*
Metodă: *Din cei 483 de pacienți cu SASO urmăriți, 252 asociau HTA; 142 dintre aceștia (56,34%) primeau medicație antihipertensivă, 59 pacienți (41,54%) prezentau HTA controlată, 83 de pacienți (58,46%) prezentau HTA necontrolată. Datele demografice și antropometrice, simptomele SASO, comorbiditățile associate, indicele de apnei (IA), indicele apnei-hipopnei (IAH), indexul de desaturare, titrarea CPAP, rata de eșec la titrarea CPAP au fost studiate comparativ între pacienții cu HTA controlată și HTA necontrolată.*
Rezultate: *59 de pacienți cu HTA controlată – 20 de femei (33,9%), 39 de bărbați (66,1%), cu vârsta medie de 56,08 ani ± 11,33, cu IAH mediu de 53,61 ± 34,42/oră, au necesitat o presiune CPAP medie de 10,15 ± 2,43 cm H_2O. 83 pacienți cu HTA necontrolată - 18 femei (21,7%), 65 de bărbați (78,3%), cu vârsta medie de 55 ± 9,06 ani, cu IAH mediu de 61,91 ± 43,61/oră, au necesitat o presiune CPAP medie de 10,47 ± 2 cm H2O. Comparativ cu grupul pacienților cu HTA controlată, pacienții cu HTA necontrolată au raportat cefalee matinală, oboseală matinală și impotență într-un procent mai ridicat (p=0,020, 0,018, respectiv 0,011). Scala de somnolență Epworth a fost sub 10 (limita pentru somnolență diurnă excesivă) la pacienții cu HTA controlată (p=0,001) și peste 10 la cei cu HTA necontrolată. Pacienții cu HTA necontrolată au fost diagnosticați cu HTA după o perioadă mai mare (p=0,006), au avut valori mai mari ale tensiunii arteriale sistolice și diastolice la momentul evaluării SASO. Diferențe semnificative statistic au fost înregistrate doar pentru IAH post-CPAP (11,89/oră vs. 22,30/oră, p=0,013) și indexului de desaturări nocturne post-CPAP (6,03/oră vs. 16,55/oră, p=0,017), ambele mai mari la pacienții cu HTA necontrolată. Ipoteza cu privire la existența diferențelor legate de comorbiditățile cardiovasculare nu a fost susținută.*
Concluzii: *Controlul valorilor tensionale duce la ștergerea somnolenței diurne, simptom definitoriu al SASO, și reduce restul simptomelor (cefalee, impotență, oboseală matinală). Simptomele SASO sunt mai puțin frecvente în grupul pacienților cu HTA controlată, SASO fiind mai greu de suspectat. Acești pacienți par a avea un beneficiu mai mare al terapiei CPAP – au IAH post-CPAP și indice desaturări post-CPAP semnificativ mai scăzut decât pacienții cu HTA necontrolată.*
Cuvinte-cheie: *sindrom de apnee în somn de tip obstructiv, hipertensiune controlată, hipertensiune necontrolată*

Abbreviations:
OSA = obstructive sleep apnea,
OSAS = obstructive sleep apnea syndrome;
HT = arterial hypertension;
sBP = systolic blood pressure;
dBP = diastolic blood pressure;
ESS = Epworth Sleepiness Scale;
BMI = body mass index;
AHI = apnea-hypopnea index;
AI = apnea index;
ODI = oxygen desaturation index;
CPAP = continuous positive airway pressure;

Introduction

Sleep apnea syndrome is characterized by obstruction of the superior airways, defined by the presence of more than 5 respiratory events per hour of sleep (apneas, defined as the total cessation of airflow in the superior airways, or hypopneas, a decrease of airflow to less than 50% of the initial amplitude, changes that must last for over 10 seconds per event). Obstructive sleep apnea is highly prevalent in general population, in adults with somnolence about 2-4%[1] as obstructive sleep apnea syndrome (OSAS) and 9-24%[2] in adults without somnolence, as obstructive sleep apnea (OSA). Newer studies found even higher prevalence, as the prevalence in India[3] (estimated to 7.5%) or that of one study from Brasil[4], where OSAS was observed in 32.8% of the participants. The prevalence in certain groups is even higher, as a prevalence of 30 to 55% in the Down populations[5], or the obese groups, where OSAS prevalence may reach 80% in males and 50% in females with morbid obesity[6], diabetes - up to 83% of patients with type 2 diabetes suffer from unrecognized OSA[7]. The prevalence of OSAS is higher in patients with cardiovascular disease[8, 9], up to 50%.

OSAS patients are mostly men, obese (especially with visceral fat and neck fat), they have poor quality of sleep due to nocturnal apnea (described sometimes by the bed partner and by polisomnography, showing a sleep fragmentation and arousals during each respiratory event[10, 11]), have excessive daytime sleepiness and some had road accidents as a consequence.

An association between OSAS and hypertension (HT) has been observed since the early clinical description of OSA in the 1970s[12-15]. Both OSAS and HT are common, and many individuals have both conditions. Now there is strong evidence that OSAS is an independent risk factor for HT than any other cardiovascular disease[8, 16-18], excluding from this pathogenic association the central sleep apnea syndrome, which counts up to 40% of cases of heart failure[19]. Because of the difficulty in accounting for multiple confounding factors, the association between OSAS and hypertension has remained controversial[20]. Studies estimated that about 50% of OSA patients are hypertensive, and an estimated 30% of hypertensive patients also have OSA, often undiagnosed[21] and up to 83% of patients with refractory hypertension have OSAS[22]. The association between OSAS and HT is now so clear that the US Joint National Committee on Hypertension puts OSAS first on the list of the causes of secondary hypertension[23]. Several multivariate analytical models indicate OSA as an independent risk factor for hypertension[24-26], while hypertension constitutes a significant predictor of cardiovascular deaths among patients with OSA[26].

Continuous positive airway pressure (CPAP) is the "gold-standard" treatment for OSAS[27, 28]. CPAP reduces or corrects the respiratory events in most patients. As OSAS is a risk factor for HT, it is to assume that treating OSAS will decrease blood pressure (BP) in patients with arterial hypertension. Studies have shown a slightly reduction of mean BP after 4-8 weeks of CPAP therapy[29]. Another study has shown a small and statistically non-significant decrease in 24 hours mean BP after 4 weeks of CPAP therapy, with no significant changes in systolic, diastolic, daytime, or night-time BP[30].

Despite overwhelming evidence linking OSAS to hypertension, current guidelines do not provide specific recommendation for pharmacological management of hypertensive patients with OSAS, due to limited data from prospective trials[31]. The logistic difficulties for an accurate delineation of which antihypertensive drug works best in OSAS patients are immense and require careful control for comorbidities and multiple other factors including the severity of sleep apnea, which can itself vary on different occasions[32].

The aim of our study was to compare how controlling arterial hypertension with antihypertensive drugs may influence obstructive sleep apnea syndrome: symptoms, comorbidities, OSAS severity, AHI before and after CPAP therapy, oxygen desaturation index (ODI) before and after therapy, and also CPAP failure.

Materials and methods

Study population

From November 2007 to March 2010 we enrolled 430 patients with OSAS who presented in the "Marius Nasta" National Institute of Pneumology, Bucharest, Romania for sleep study. From 430 patients, 252 associated arterial hypertension. From these 252 patients with OSAS and HT, 110 were untreated and 142 received antihypertensive treatment. We focused on the group with treated HT. From the 142 patients, 59 had controlled hypertension and 83 patients had uncontrolled hypertension (Figure 1). We defined controlled hypertension as values of systolic blood pressure less than 140 mmHg and diastolic blood pressure less than 90 mmHg under antihypertensive treatment[33]. Uncontrolled hypertension was defined as values of systolic blood pressure equal or more than 140 mmHg and diastolic blood pressure equal or more than 90 mmHg under antihypertensive treatment[33]. In the group of uncontrolled hypertension were included patients with resistant HT (22 patients). Resistant HT is defined as blood pressure that remains above goal in spite of using at least three different classes of antihypertensive drugs, one being a diuretic and all drugs being prescribed at optimal doses[31].

The study was approved by "Marius Nasta" Institute's Review Board, and each patient signed a written consent form. Patients were receiving their usual antihypertensive treatment, as previously prescribed. Patients were divided in the 2 groups (controlled and uncontrolled) according to the definitions specified above and they continued receiving other medication if needed, for associated comorbidities (diabetes mellitus, hypothyroidism, congestive heart failure and so on). Patients were treated for hypertension for at least 3 months previously to the inclusion in the study.

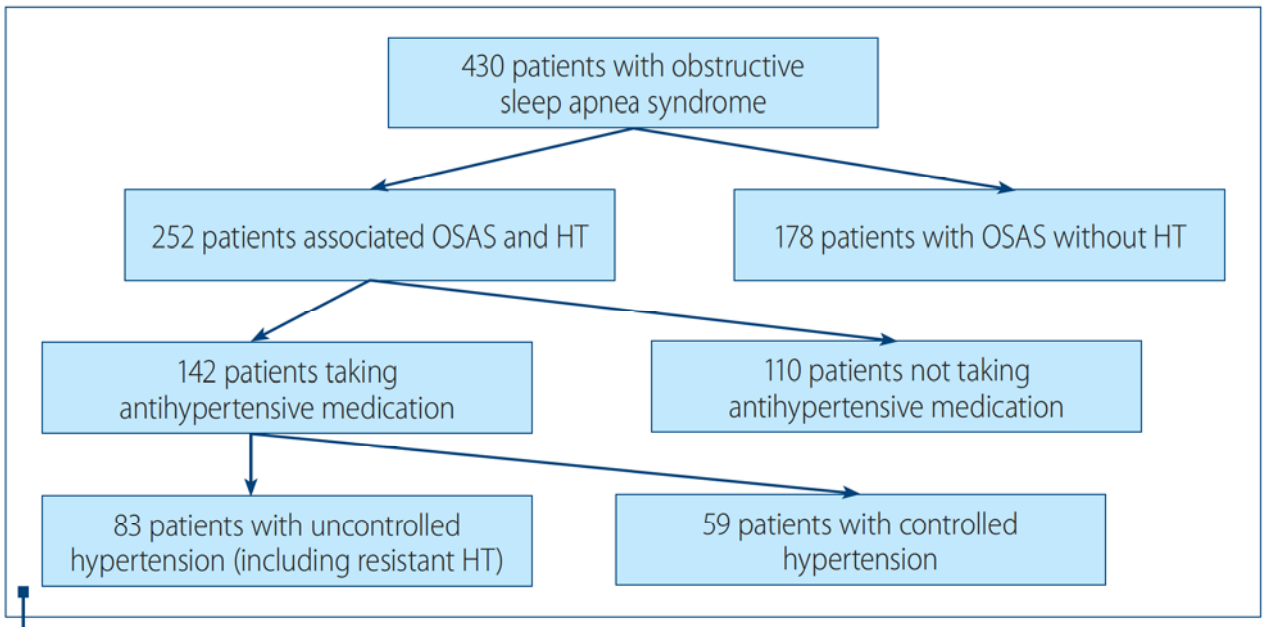

Figure 1. *Study design*

Measurements

Epworth Sleepiness Scale (ESS): The Epworth Sleepiness Scale (ESS) is a scale intended to assess daytime sleepiness that is measured by use of a very short questionnaire. This can be helpful in diagnosing sleep disorders. A number in the 0-9 range is considered to be normal while a number in the 10-24 range indicates that expert medical advice should be sought[34].

Body mass index (BMI): Body mass index is defined as the individual's body weight divided by the square of his/her height. The formulae universally produce a unit of measure of kg/m^2. BMI between 18.5 and 25 kg/m^2 is considered normal weight and overweight with BMI between 25-30 kg/m^2. Obese patients are defined the patients with BMI ≥ 30 kg/m^2, divided in three types of obesity: class I with BMI between 30-35 kg/m^2, class II with BMI 35-40 kg/m^2 and class III, or morbid obesity, BMI over 40 kg/m^2.[35, 36]

Blood pressure: Blood pressure was measured on the first visit - when sleep questionnaire was completed, a physical examination was performed and routine laboratory test were performed, (including ECG) and than in all other visits (before the diagnosis, before CPAP titration). Patients were asked to sit in a quiet room before measuring BP. There were at least two measurements, approximately 2 minutes apart from one another. Additional measurements were taken if the first two were very different. We used standard cuff (24-32 cm) for normal weight patients and a larger cuff (32-42 cm) for obese patients. We measured BP in both arms, and if differences were recorded the higher value was the reference one. We recorded the blood pressure as the average value of all measurements[33].

Sleep study: For the diagnosis of OSAS we used standard polysomnography (Alice 5 Diagnostic Sleep System, by Philips Respironics and SomnoStar® z4 Sleep System) from 22 PM to 6 AM, recording respiratory events (apnea or hypopnea) by termistor and flow canula, snoring by microphone, stages of sleep (electroencephalogram – EEG using standard criteria[37] for epochs of 30 seconds), eye and chin movements (electro-oculogram, chin electromyogram), body position, oxygen saturation, heart rate (pulse oximeter), limb movements (tibial electromyogram).

Study protocol

Apnea was defined as cessation of the oro-nasal airflow for more than 10 seconds, associating oxygen desaturation (> 3-4% of preceding baseline) and/or arousals (electroencephalogram). Obstructive apnea was defined as cessation of airflow with persistence of thoraco-abdominal movement. Hypopnea was defined as a reduction for more than 50% on the oro-nasal airflow, lasting more than 10 seconds, associating oxygen desaturation and/or arousals. Apnea-hypopnea index (AHI) was defined as the number of apneas and hypopneas per hour of sleep. Obstructive sleep apnea syndrome was defined as more than 5 AHI and a percentage of obstructive apnea > 50%[38]. Oxygen desaturation index (ODI) was defined as the number of oxygen desaturations per hour of sleep. Sleepiness was evaluated using the Epworth Sleepiness Scale[34] (ESS).

After the diagnosis night, patients were called for a second night in the sleep laboratory for CPAP titration (using GoodKnight 420G and REMstar Plus). The titration was performed with full-night polysomnography, and the goal was an optimal titration[39]. CPAP level was increased so respiratory events (apnea or hypopnea), snoring and oxygen desaturation were eliminated or maximally decreased maintaining a good CPAP tolerance. The goal was to eliminate all events described above for at least 15 minutes in REM sleep, in supine position, and having no arousals in this period. CPAP failure was the titration that does not reduce AHI to ≤ 10/h or more than 75% from baseline AHI are still present after titration[39].

Table 1 OSAS symptoms compared between the two groups (uncontrolled vs. controlled hypertension)

Symptoms	Uncontrolled hypertension	Controlled Hypertension	P value
Snoring	100%	96.6%	NS
Morning fatigue	81.9%	64.4%	0.018
Nocturnal apnea	90.4%	91.5%	NS
Morning headaches	51.8%	32.2%	0.02
Nocturia	77.1%	69.5%	NS
Nightmares	26.8%	30.5%	NS
Impotence	31.7%	13.8%	0.011

Table 2 Associated comorbidities in the two groups

Comorbidities	Uncontrolled hypertension	Controlled hypertension	P value
Dyslipidemia	66.3%	55.9%	NS
Diabetes mellitus	26.5%	23.7%	NS
Endocrine diseases	12%	16.9%	NS
Rhythm disorders	19.3%	18.6%	NS
Atrioventricular blocks	16,9%	18.6%	NS
Left heart failure	14.3%	5.2%	NS
Right hear failure	5.2%	0.0%	NS
Chronic obstructive pulmonary disease	7.3%	6.8%	NS

Table 3 Sleep study report of respiratory variables compared in the two groups

Polysomnography data	Uncontrolled HT	Controlled HT	P value
AHI	61.91 ± 43.61	53.61 ± 34.42	NS
ODI	54.24 ± 48.01	49.74 ± 38.41	NS
Maximum nocturnal desaturation (%)	73.16 ± 13.36	73.49 ± 11.96	NS
AHI post-CPAP	22.30 ± 10.86	11.89 ± 5.31	0.013
ODI post-CPAP	16.55 ± 2.91	6.03 ± 2.65	0.017
Maximum nocturnal desaturation post-CPAP (%)	81.09 ± 13.70	83.75 ± 16.46	NS
Mean CPAP value (cm H_2O)	10.47 ± 2	10.15 ± 2.43	NS
CPAP failure (%)	39.06	29.51	NS

*AHI = apnea-hypopnea index; ODI = oxygen desaturation index; CPAP = continuous positive airway pressure.
** Data presented as percent (%) or mean value ± SD.

Statistics

Statistical analysis was performed using SPSS statistical software program (version 17.0). Continuous variables are expressed as mean ± standard deviation (SD) and qualitative variables are expressed as a percentage. A two-tailed t test unpaired for independent samples was used to compare the two groups. For nominal data χ^2 test was used to compare baseline analysis in the two groups. The relationship between systolic blood pressure (sBP), diastolic blood pressure (dBP), ESS, BMI, AHI was assessed with Pearson correlation. Statistical significance was assumed at $p \leq 0.05$.

Results

We examined 483 followed patients with OSAS, 252 associating HT, from which 142 patient were treated with antihypertensive drugs.

The prevalence of the HT in the OSAS studied population was 58.6%, higher than the reported previous values. From all the hypertensive patients, 56.35% where treated in the moment of presentation.

We studied these 142 patients with OSAS and treated HT. The general characteristics of the entire studied population: they were middle-aged patients (mean age was 55.45 years), mostly men - 104 men (73.2%), obese (mean weight 103.08 kg, with mean BMI 35.03 kg/m^2), 41.1% were never smoked, 24.1% were active smokers and 34.8% were former smokers. Almost all of the patients 140 (98.6%) are snoring, 129 patients (90.8%) reported witnessed apnea by the bed partner, 116 patients (81.7%) complain from excessive daytime sleepiness, 106 (74.6%) from morning fatigue and 62 (43.7%) from morning headaches. Other symptoms found: 73.9% of patients reported nocturia, 64.8% nocturnal dyspnea, 37.5% insomnia and 24.3% impotence. Regarding associated comorbidities in the studied population: 62% had dyslipdemia, 35.9% ischemic heart disease, 25.4% diabetes mellitus, 14.1% endocrine diseases, 10.4% left cardiac failure and 7.1% chronic obstructive pulmonary disease. All 142 patients were diagnosed with hypertension for an average of 7.67 years having at the evaluation moment average blood pressure systolic and diastolic values above normal (mean sBP was 142.64 mmHg, mean dBP was 84.46 mmHg). After the diagnosis night we found that the mean AHI in the studied population was 58.64 ± 40.26, with a mean ODI of 52.51 ± 44.38. Regarding OSAS severity[40] - 63.4% had severe OSAS (AHI > 30/hour), 22.5% moderate OSAS (AHI = 15-29/h) and 14.1% mild OSAS (AHI = 5-14/h).

A higher percent of uncontrolled patients reported impotence (31.7% vs. 13.8%, p =0.011), they complained more from morning headaches (51.8% vs. 32.2%, p =0.02) and fatigue in the morning (81.9% vs. 64.4%, p =0.018). Regarding other symptoms there were no statistically significant differences (p>0.05) (Table 1). Patients with controlled HT do not have excessive daytime sleepiness as a quantitative measurement (ESS score below 10) comparing with those with uncontrolled HT (8.55 vs. 11.53 respectively, p=0.001).

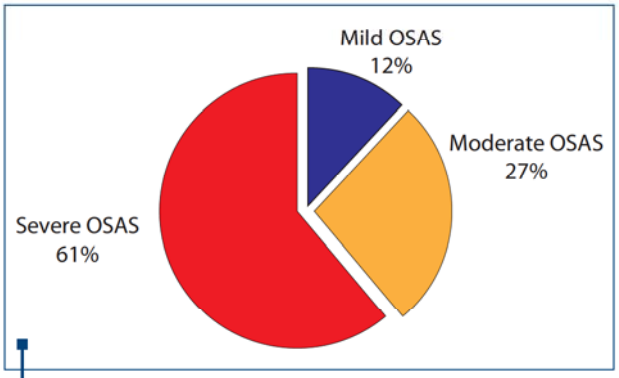

Figure 2. *Severity of OSAS in uncontrolled HT group*

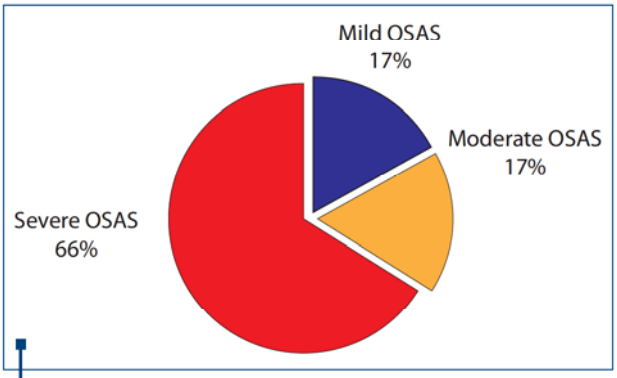

Figure 3. *Severity of OSAS in controlled HT group*

Patients with uncontrolled HT are fatter (107.39 kg vs. 97.03 kg, p=0.002) with a higher BMI (35.98 kg/m^2 vs. 33.88 kg/m^2, p=0.046) than patients with controlled HT. Regarding other comorbidities (cardiovascular and metabolic), there were no statistically significant differences (p>0.05, Table 2). As for BP values, patients with uncontrolled HT had higher values of both systolic and diastolic BP (as a criteria of inclusion in their group). Also, these patients have been diagnosed with hypertension for a longer period (9.20 years vs. 5.55 years, p = 0.006).

After the diagnosis night there were no differences between the two groups regarding sleep events (AHI, ODI, nocturnal desturation), but patients with uncontrolled HT had a higher AHI post-CPAP (22.3/h vs. 11.89/h, p=0.013) with a higher ODI post-CPAP (16.55/h vs. 6.03/h, p=0.017); they also showed a trend of higher CPAP failure rate. Optimal CPAP value that erases the respiratory events, snoring and oxygen desaturation is not different in the two groups (10.47 cmH_2O vs. 10.15 cmH_2O; Table 3).

No differences were found regarding OSAS severity in the 2 studied groups (p=NS) (Figures 2 and 3).

We studied the relationship between sBP, dBP and ESS, BMI, AHI, ODI, maximum nocturnal desaturation using Pearson correlations and we found that only ESS is correlated with diastolic BP measured at the evaluation moment; patients who reported excessive daytime sleepiness had a higher dBP (r=0.253, p=0.008).

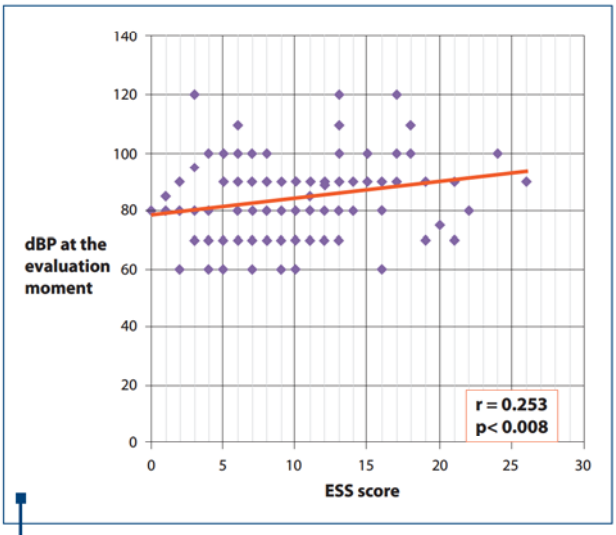

Figure 4. *Correlation between ESS and diastolic blood pressure*

Discussions

We studied a treated hypertensive group of patients identified in an OSAS population addressed to our sleep lab for a period of 2 years and a half. The prevalence of HT in the whole group was found to be 56.6%, above the values described in specialty literature[13]. We explain this by mostly addressability issues. In Romania, the addressability for sleep disorders is still weak (even if in great ascent) and most of the patients present for the sleep study late in the disease's evolution, so the OSAS at the diagnosis moment comes to be severe, and complication already installed.

Most of the patients with treated HT had uncontrolled HT (58.45%). This may be because of a weak medical adherence - which we could not objectively evaluate it and, as a result, we are not able to exclude it - or the presence of OSAS may have a certain contribution to the poorly controlled values of BP. There are certain antihypertensive which could be use preferentially in OSAS according to the physiopathogenic changes identified in OSAS. Periodic apneas are followed by intermittent hypoxia and sleep fragmentation. This activates inflammatory pathways, causes endothelial dysfunction and cardiovascular diseases trough sympathetic excitation, oxidative stress[41] and angiotensin II[42]. Angiotensin II is known as a proatherosclerotic factor and blocking of its receptor has been proved to lower the BP values and cardiac complications of OSAS[43]. Comparing to angiotensin II blockers, beta blockers have been proved to be as efficient in controlling the BP, but also lowered the heart frequency (applicable in selected patients)[44]. Spironolactone has been found efficient in the resistant HT associated to OSAS[45].

Interfering with all these pathogenic pathways, the antihypertensive drugs also diminish systemic inflammation, including that in the upper airways. The rationale of this study is that a good control of HT could also lead to a good control of local inflammation and therefore could modify the respiratory parameters of OSAS.

Many studies in literature[27, 46-48] have followed the impact of CPAP therapy on BP values, only few studied the impact of antihypertensive treatment on respiratory parameters, with no effect proved[49] until now; we aimed to verify the effect of controlled HT by medication on respiratory events in a population with OSAS and HT.

Between the two studied groups there were no statistically significant differences regarding age and gender distribution: mean age is 55 years (range 34 to 72 years) in patients with uncontrolled HT, and 56.08 years (range 31 to 85 years) in patients with controlled HT. Regarding gender distribution were 22% women and 78% men in uncontrolled group, 33% women and 66% men in controlled HT group. These findings are consistent with recent studies which demonstrated that OSAS is not a "male" disease, and at least 2% of middle-aged women suffer from OSAS[50-52], up to 9% in OSA cases (without somnolence)[2].

Patients with uncontrolled hypertension are fatter with a mean weight of 107.39 ± 19.03 kg (range 74 to 160 kg) vs. 97.03 ± 18.76 kg (range 58 to 149 kg) and, of course, a higher BMI (35.98 kg/m^2 vs. 33.88 kg/m^2, p=0.046) compared to patients with controlled hypertension. Obesity is one of OSAS's risk factor, obesity being known to account for approximately 30 to 50% of the variability in AHI[53]. Many studies demonstrated that increasing weight aggravates the AHI, which defines the severity of OSAS. It was also noticed that the rate of increase in apneas is exponential, with steeper elevations in AHI at higher levels of BMI[54]. Also, obesity is one of the hypertension's risk factors, and so, a confounding factor in our study. As the persons in the uncontrolled group weight more, they have a greater risk of having a severer and more difficult to control OSAS, explaining part of the results obtained.

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Our study is limited by using some anamnestic data or paper mentioned data (e.g. comorbidities). Also, for accurate blood pressure values, ambulatory blood pressure monitoring (ABPM) should be used. ABPM differentiate HT from white coat HT, knowing that white coat hypertension seems to occur in 24 to 39% of the general hypertensive population[55, 56]. 24-hour ABPM also has its errors. It has been shown that 24-hour ABPM causes arousals in 64% of the recordings and leads to an increase in systolic and diastolic blood pressure by 13.7 ± 15.9 mm Hg and 3.7 ± 8.2 mm Hg, respectively[57, 58].

The most important finding of our study is the fact that patients with uncontrolled HT reported more symptoms: morning headache (51.8% vs. 32.2%) and morning fatigue (81.9% vs. 64.4%) in a higher percent, and also they had the ESS score significantly higher (11.53 vs. 8.55). Patients with controlled HT having normal ESS score[38] (below 10), leading to the conclusion that controlling blood pressure values results in deleting excessive daytime sleepiness, a main symptom of OSAS. A lower AHI post-CPAP (11.89/h vs. 22.30/h, p=0.013) with a lower ODI post-CPAP (6.03/h vs. 16.55/h, p=0.017) was found in patients with controlled HT, so we can conclude that they may have a greater benefit from CPAP therapy, but the lack of daytime sleepiness makes difficult to suspect OSAS in these patients.

Conclusions

The clinical importance of this study is residing from the shown fact that patients with controlled HT can have OSA without symptoms, so they should be tested for OSA with night studies, while patients with uncontrolled HT (which had for a longer period HT without any suspicion of OSA) can be more difficult to be controlled regarding respiratory events.

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