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CPAP Effect in Patients with Hypertension: The Secret of Gods

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Abstract

Introduction: Obstructive sleep apnea syndrome (OSAS) is a respiratory sleep disorder characterized by episodes of occlusion of the upper respiratory tract, resulting in intermittent chronic hypoxia and sleep fragmentation. Clinical studies have described an association between OSAS and arterial hypertension (SAH), with CPAP being the treatment of choice. The use of CPAP in the treatment of OSAS makes it possible to avoid blocking the passage of air to the lungs. The primary underlying mechanism of hypertension in individuals with OSAS is the activation of the sympathetic nervous system triggered by episodes of apnea. Currently, there are no studies that correlate the use of CPAP and the decrease in the use of antihypertensive agents in patients with OSAS.

Aim: To investigate the relation between the use of CPAP in the treatment of secondary hypertension to OSAS and the concomitant reduction in antihypertensive drugs.

Methodology: 187 patients from the SAH consultation and cardiovascular risk factors carried out by Doctor Armindo Sousa Ribeiro, of which 31 had the diagnosis of OSAS, were analyzed. Sampling characteristics (mean, standard deviation, median, interquartile range and frequency analyzes) were calculated, and this analysis was complemented with the Wilcoxon non-parametric test at a significance level of 5% error. Hypothesis testing was considered unnecessary.

Results: The average age of patients treated with CPAP (70.6 years) is greater than the patients not treated with CPAP (53.19 years). 77.4% of the patients were obese, of which 48.5% were not treated with CPAP. Higher blood pressure levels were seen in patients with CPAP and a decrease in systolic blood pressure and diastolic blood pressure during the treatment of patients with OSAS, either in patients with or without indication for CPAP. There was a decrease in the prescription of Angiotensin Conversion Enzyme Inhibitors (ACEI), Angiotensin II Receptor Antagonists (ARA II) and Nitrates and an increase in the prescription of diuretics in patients with CPAP compared to patients without indication for CPAP.

Conclusion: Patients using CPAP are older, more obese, with more severe SAH and the use of CPAP allows a decrease in blood pressure values and a reduction in dosage, but not the number of antihypertensive drugs. CPAP should be started at earlier stages of SAH to allow a reduction in the number of active substances.

Keywords: CPAP; Hypertension

Introduction

The 2nd edition of the International Classification of Sleep Disorders (ICSD-2) classifies sleep disorders in 8 major categories. Within these, sleep breathing disorders stand out, of which apneas and hypopneas are part [1].

Sleep apnea is defined as the intermittent cessation of oro-nasal airflow, for an interval of more than 10 seconds, during which hypoxemia and hypercapnia occur until, upon awakening, the apnea episode ends with airway clearance [2].

The term Obstructive Sleep Apnea Syndrome (OSAS) is used to designate a chronic, progressive respiratory disorder with several systemic repercussions, characterized by frequent upper airway collapses during sleep, resulting in substantial reduction (hypopnea) or complete cessation (apnea) of the air flow to the lungs for about 10 seconds, with persistent respiratory efforts [3,4].

Once the suspicion is raised, the exam of choice for the diagnosis is nocturnal polysomnography [5]. This exam allows the diagnosis of OSAS and its characterization according to its severity [6]. According to the *American Academy of Sleep Medicine* criteria, the Apnea and Hypopnea Index (AHI) is considered mild when the AHI is ≥ 5 and ≤ 15 events per hour during sleep; moderate when the AHI is > 15 and ≤ 30 events / h; and severe when the AHI is> 30 events / h, establishing the severity of OSAS [7].

The lack of a normal breathing pattern during sleep causes multiple clinical manifestations that range from intermittent snoring and, usually intense to the point of causing awakening (snoring), to deep nocturnal hypoventilation that can evolve into apnea [8]. At a systemic level, arterial oxygen desaturation (hypoxemia), hypercapnia and various hemodynamic effects are observed, including changes in systemic and pulmonary blood pressure and changes in cardiac rhythm [9].

In Portugal, the General Directorate of Health and the National Institute of Health Doutor Ricardo Jorge carried out a cross-sectional study in 2013, which concluded that the prevalence of OSAS in the population aged 25 or over was 0.89% (95% CI: 0 , 80-1.00%), being higher in males 1.47% (95% CI: 1.30-1.67%) and in the age group between 65 and 74 years old (2.35%). Obesity (84.6%), hypertension (74.8%) and *mellitus* diabetes (38.7%) were the most frequent comorbidities in these patients [10].

Some studies report that the prevalence of hypertension secondary to OSAS is 30% and that individual with a diagnosis of arterial hypertension (SAH) may be about 2 times more likely to have OSAS than individuals without hypertension [11]. The relationship between OSAS and hypertension is mainly due to the role of OSAS in the persistent activation of the sympathetic nervous system (SNS), with a decrease in the sensitivity of baroreceptors, vascular hyper responsiveness and alteration in the metabolism of salt and water, contributing to the increase in blood pressure [6].

The activation of the SNS is triggered by episodes of apnea where patients with OSAS have episodes of intermittent hypoxemia, causing hemodynamic changes during sleep [5]. Cardiac output, heart rate and blood pressure tend to change due to changes occurring during the respiratory cycle (desaturation, hypercapnia, increased respiratory effort and micro-awakening) and subsequent variations in the sleep-wake state [12]. After apnea, breathing is normalized with subsequent return to normal sleep. All these stimuli, in particular desaturation-reoxygenation, are a source of stimulation of the sympathetic nervous system [13,14]. Patients with OSAS also exhibit changes in sensitivity at the level of the baroreceptors, which together with the decrease in vascular relaxation and the stimulation of the SNS, increase blood pressure during sleep [15-17].

The treatment of hypertension secondary to OSAS must be multidisciplinary, including behavioural change, with the aim of controlling symptoms and reducing cardiovascular risk [18]. Several studies report the benefit of OSAS treatment for lowering blood pressure (BP) due to the control of sympathetic activity associated with effective treatment with CPAP, being proportional to its time of night use [19]. CPAP works to keep the airway open during inspiration and expiration, promoting remission of altered respiratory events and, consequently, improving the sleep structure and quality of life of patients [13]. Results from 12 meta-analyses of randomized CPAP-placebo trials indicate a reduction of 1.69 mmHg in the mean blood pressure levels measured in 24-hour

outpatient monitoring [20]. However, it should be noted that the comparison of these studies should be interpreted with caution since these studies present variations in the definition of SAH and in the classification of the severity of OSAS, as well as in the time of use of CPAP and in the monitoring methods blood pressure [21].

In addition, no studies were found comparing the number of antihypertensive drugs indicated for patients with arterial hypertension and OSAS concomitantly before and after the start of ventilatory treatment.

Thus, the objective of this study is to understand whether OSAS therapy with CPAP will be associated with a decrease in blood pressure and antihypertensive medication.

Materials and Methods

Data collection

A retrospective study was carried out in an adult population aged 18 years or older who attended to the consultation of arterial hypertension and cardiovascular risk factors at the Unidade Local de Saúde do Litoral Alentejano, EPE, carried out by Doctor Armindo Sousa Ribeiro, between 2017 and 2020. Clinical data were obtained through the analysis of clinical processes using the SClinic software. Inclusion criteria were patients aged 18 years or older, diagnosed with resistant hypertension, hypertensive urgency, hypertension secondary to OSAS, or hypertension of etiology to be clarified, undergoing antihypertensive therapy and / or CPAP.

The exclusion criteria were the absence of computerized records of subsequent consultations, abandonment of antihypertensive treatment by the patient and the use of drugs that cause an increase in hypertension.

Resistant hypertension is defined as a SBP ≥ 140 mmHg and DBP ≥ 90 mmHg, despite therapy with 3 or more antihypertensive drugs including a diuretic.

Hypertensive urgency is defined as severe arterial hypertension (SBP ≥180 mmHg / DBP ≥120 mmHg) without signs and symptoms of acute target organ damage.

Blood pressure was measured using an ambulatory blood pressure monitoring exam (ABPM).

The diagnosis of OSAS was based on standard polysomnography performed using standard techniques and scoring criteria. OSAS was defined as an apnea-hypopnea index (AHI) of at least 5 events per hour. Awakenings were defined according to the recommendations of the Atlas of *American Sleep Disorders Association* task force. The stages of the sleep study are in accordance with the *Rechtschaffen and Kales* system.

Data processing

The results were obtained through the statistical application SPSS 24.

Sampling characteristics (mean, standard deviation, median, interquartile range and frequency analysis) were calculated. We chose to test the relationships between variables using non-parametric tests, since it is a relatively small global sample (31 patients), and when this sample is subdivided into groups of patients with and without the use of CPAP, it only contemplates 10 and 21 patients respectively; and the minimum required to use parametric tests is at least 30 elements in each subgroup.

Given the paired nature of the study, since we analysed numerical parameters (TA values; dosages of drugs; number of active substances) at two different times (first consultation and last consultation), the non-parametric test of choice is the Wilcoxon Test.

The relationships between the variables were tested with a 95% probability, resulting in a 5% significance level (a = 0.05). This level of significance allows us to affirm with a "certainty" of 95%, if the validity of the relationship between variables under study is verified, the existence of a causal relationship between them.

The decision criteria for testing these relationships between variables are based on the study of probabilities, confirming the relationship if the probability is less than 0.05 and rejecting it if greater than that value.

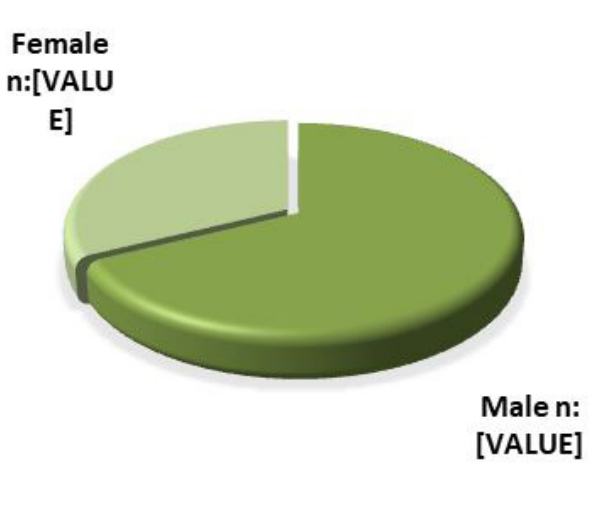
The data processing was done by computer using the SPSS.24 program.

Results and Discussion

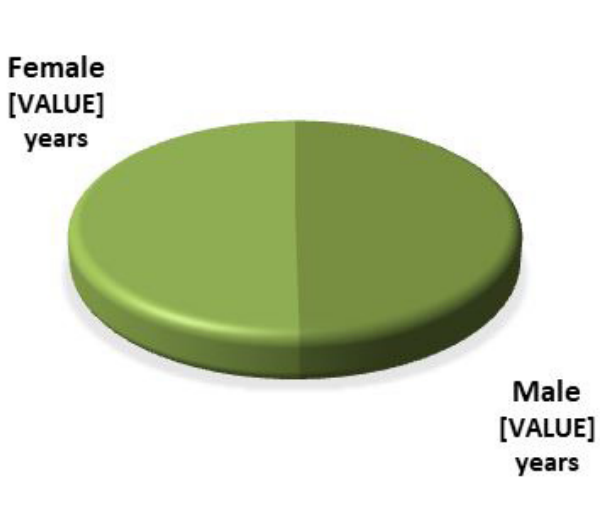
Descriptive Analysis

Demographic characterization: 181 patients from the consultation of arterial hypertension and cardiovascular risk factors, carried out by Doctor Armindo Sousa Ribeiro, at ULSLA, EPE, between the years 2017 and 2020 were analysed. Of these, 37 patients with arterial hypertension secondary to OSAS were diagnosed, presenting 6 patients exclusion criteria.

Of the sample, 21 patients were male (67.7%) whose average age was 59.85 years and 10 were female (32.3%), whose average age was 60.5 years (see graphs 1 and 2).



Grap7h 1: Distribution of patients according to sex



Graph 2: Average age in years of patients according to sex

In Table 1, we can conclude that the mean age of patients was 58.81 years, with an age range between 28 years and 88 years, and patients diagnosed with hypertension secondary to OSAS and who use CPAP, have a higher mean age (70.6 years) than those who do not use CPAP (53.19 years). The age varies between 28 and 88 years.

Age	N	Minimum	Maximum	Average	Dp	Kurtosis	Skewness
With CPAP	21	28	70	53,19	11,03	0,446	-0,647
Without CPAP	10	41	88	70,60	15,31	0,236	-0,849
Total	31	28	88	58,81	14,83	-0,148	0,096

Table 1: Distribution of the sample by age and need to use CPAP

Table 2 shows that 32.3% of patients with OSAS are in the age group between 51-65 years old (32.3%), followed by patients under the age of 50 years, which correspond to 29% of the cases, being that of these 6.5% of patients started CPAP. 28.7% of patients with OSAS are older than 65 years, with 25.8% starting therapy with CPAP.

The medical consultation is the referral most verified with 45.2% of the sample, followed by the CSP with 22.6%.Resistant hypertension is the main reason in 64.5%, with the majority (87.1%) having no personal history of OSAS.

VARIABLES	Without CPAP (n=21)		With CPAP (n=10)		TOTAL (n=31)	
	N	%	N	%	N	%
Age						
≤50 years	7	22,6	2	6,5	9	29,0
51-65 years	10	32,3	---	0,0	10	32,3
>65 years	4	12,9	8	25,8	12	28,7
Medical consultation	11	35,5	3	9,7	14	45,2
PCP	4	12,9	3	9,7	7	22,6
Internment	2	6,5	2	6,5	4	12,9
Other hospital	---	0,0	2	6,5	2	6,5
ES	4	12,9	---	0,0	4	12,9
Reason						
SAH secundary assoc with OSAS SAH resistent	1 13	3,2 41,9	2 7	6,5 22,6	3 20	9,7 64,5
Obesity	1	3,2	1	3,2	2	6,5
Metabolic syndrome	1	3,2	---	0,0	1	3,2
Hypertensive urgency	5	16,1	---	0,0	5	16,1
Personal history of OSAS						
No	20	64,5	7	22,6	27	87,1
Yes	1	3,2	3	9,7	4	12,9

Table 2: Distribution of users according to personal variables according to the use of CPAP

Clinical characterization: In the first consultation, ABMP, spirometry and polysomnography were requested, for evaluation in subsequent consultations. In Table 3a, the column "with CPAP" refers to patients who, after analysing the results of the complementary diagnostic tests, started treatment with CPAP. After evaluating the complementary diagnostic tests and depending on the results, patients were referred to the pulmonology clinic for follow-up and initiation of CPAP.

Analysing Table 3a, which corresponds to the distribution of patients according to clinical variables depending on the use of CPAP, we can see that 74.4% of patients are obese, with 38.2% having grade I obesity, 25.8% being obese grade II and 12.9% grade III obesity. Of the patients with grade I obesity, 16.1% needed to start CPAP, of the grade II obesity group, 6.5% started CPAP and of the grade III obesity group, 4.4% started CPAP.

We also found that most patients (58.1%) do not use *polypill* - a tablet that combines more than one active substance.

Regarding ABMP, 20 patients underwent a first blood pressure assessment using ABMP. It is concluded that, of the daytime evaluation

25.8% had SAH grade II, followed by SAH grade I with 12.9% of the patients. It was also found that 16.1% of patients did not show daytime hypertension. In the assessment of nocturnal values, it was found that 32.3% of the patients had SAH grade I, 9.7% SAH grade III and 3.2% grade II. It was found that 12.9% of the patients did not have nocturnal hypertension. In both daytime and nigh time assessment, 12.9% of patients had isolated systolic hypertension. A 48.4 % of patients were dippers. In the evaluation of spirometry, we found that 61.3% of hypertensive patients had normal values, and 29% had bronchial obstruction. Of these, 25.8% of patients started treatment with CPAP.

From the assessment of cardiorespiratory polysomnography, through the assessment of the AHI, we found that 48.4% of the patients had mild AHI, within which none started CPAP, 25.8% had moderate AHI, of which 19.4% started CPAP and 25, 8% had severe AHI, 16.1% of whom started CPAP. We can conclude that 35.4% of the patients who underwent cardiorespiratory polysomnography started therapy with CPAP.

VARIÁVEIS	Without CPAP (n=21)		Com CPAP (n=10)		TOTAL (n=31)	
	N	%	N	%	N	%
BMI						
Normal	3	9,7	1	3,2	4	12,9
Overweight	3	9,7	---	0,0	3	9,7
Obesity grade I	7	22,6	5	16,1	12	38,7
Obesity grade II	6	19,4	2	6,5	8	25,8
Obesity grade III	2	6,5	1	3,2	3	9,7
Morbid obesity	---	0,0	1	3,2	1	3,2
Polypill (initial)						
No	12	38,7	6	19,4	18	58,1
Yes	9	29,0	4	12,9	13	41,9
ABMP daytime (initial)						
Grade 1	2	6,5	2	6,5	4	12,9
Grade 2	6	19,4	2	6,5	8	25,8
Grade 3	1	3,2	---	0,0	1	3,2
Normal high	4	12,9	---	0,0	4	12,9
Great	1	3,2	---	0,0	1	3,2
Isolated systolic hypertension	1	3,2	1	3,2	2	6,5
ABMP night-time (initial)						
Grade 1	8	25,8	2	6,5	10	32,3
Grade 2	---	0,0	1	3,2	1	3,2
Grade 3	2	6,5	1	3,2	3	9,7
Normal high	---	0,0	1	3,2	1	3,2
Great	3	9,7	---	0,0	3	9,7
Isolated systolic hypertension	2	6,5	---	0,0	2	6,5
Dipper (initial)						
Absent	3	9,7	---	0,0	3	9,7
Inverted	1	3,2	1	3,2	2	6,5
Present	11	35,5	4	12,9	15	48,4
AHI						
Severe	3	9,7	4	12,9	7	22,6
Mild	15	48,4	---	0,0	15	48,4
Moderate	2	6,5	6	19,4	8	25,8

Table 3a: Distribution of users according to clinical variables according to the use of CPAP

Analysing Table 3b, we found that 54.8% of the patients were obese and 61.3% of the patients did not received polypills.

Regarding the ABMP of follow-up after antihypertensive therapy and CPAP, we found that was normalization of the DIPPER phenomenon in all 12 patients evaluated.

	Without CPAP (n=21)		With CPAP (n=10)		TOTAL (n=31)	
	N	%	N	%	N	%
Spirometry						
Slight restrictive ventilatory alteration	1	3,2	1	3,2	2	6,5
Moderate restrictive ventilatory alteration	---	0,0	1	3,2	1	3,2
Normal	14	45,2	5	16,1	19	61,3
Bronchial obstruction	1	3,2	---	0,0	1	3,2
Polypill (initial)						
No	12	38,7	7	22,6	19	61,3
Yes	9	29,0	3	9,7	12	38,7
ABMP daytime (initial)						
Grade 1 (140-159/90-99 mmHg)	2	6,5	---	0,0	2	6,5
Grade 2 (160-179/100-109mmHg)	2	6,5	---	0,0	2	6,5
Normal (<129/84mmHg)	1	3,2	---	0,0	1	3,2
Normal high (130-139/85-89 mmHg)	---	0,0	3	9,7	3	9,7
Grade 3 (> 180/110 mmHg)	1	3,2	3	9,7	4	12,9
ABMP night-time (initial)						
Grade 1 (140-159/90-99 mmHg)	1	3,2	---	0,0	1	3,2
Normal high (130-139/85-89 mmHg)	1	3,2	1	3,2	2	6,5
Grade 3 (> 180/110 mmHg)	3	9,7	5	16,1	8	25,8
Systolic Arterial Hypertension (> 140/<90 mmHg	1	3,2	---	0,0	1	3,2
Dipper (initial)						
Absent						
Inverted	6	19,4	6	19,4	12	38,7
Present						

Hematocrit values	Minimum	Maximum	Average	DP
Without CPAP	41	52	43.21	3.46
With CPAP	34	47	40.45	3.39

Table 3b: Distribution of users according to clinical variables according to the non-use / use of CPAP in subsequent consultations

Inferential Analysis

After the descriptive analysis of the data obtained described above, we then proceed to the inferential approach of the same, through analytical statistics. Thus, we proceeded to verify some relations between variables to answer our aim and the central question of our study.

Blood pressures values: By analysing Table 4, we found that, on average, those who use CPAP (SBP-171.20mmHg / DBP-85.10mmHg) have higher systolic values and lower diastolic values than patients in whom it was not necessary to use CPAP (SBP -165.33mmHg / DBP-97.83mmHg).

BP values		N	Minimum (mmHg)	Maximum (mmHg)	Average (mmHg)	Dp
Without CPAP	**SBP**	18	120	193	165,33	17,52
	DBP	18	78	134	97,83	16,50
With CPAP	**SBP**	10	140	207	171,20	18,00
	DBP	10	67	99	85,10	10,70

Table 4: Statistics on blood pressure values (initial) according to the use of CPAP

By analysing Table 5, we found that patients using CPAP (SBP-140.00mmHg / DBP-77.00mmHg) have higher systolic and diastolic values than patients in whom it was not necessary to use CPAP (SBP-13.67mmHg / DBP -75.94mmHg), however, there was a reduction in blood pressure in both groups.

BP values		N	Minimum (mmHg)	Maximum (mmHg)	Average (mmHg)	Dp
Without CPAP	**SBP**	18	110	145	131,67	11,97
	DBP	18	60	91	75,94	9,63
With CPAP	**SBP**	9	110	246	140,00	41,89
	DBP	9	54	98	77,00	13,49

Table 5: Statistics on blood pressure values (final) according to the use of CPAP

We also emphasize that, by the Wilcoxon Test result, when we compare the mean values of both systolic and diastolic blood pressure in both subgroups of our sample, we find the existence of statistically significant differences (p = 0.001) in the decrease in systolic blood pressure values. And diastolic in patients who did not use CPAP; statistically significant differences (p = 0.028) in the decrease in systolic values in patients using CPAP; and absence of statistical significance in decreasing diastolic values (p = 0.192).

BP values		Starting Average (mmHg)	Final Average (mmHg)	Negative rank	Positive rank	Tie	Wilcoxon	
							z	p
Without CPAP	**SBP**	165,33	131,67	15	1	0	-3,364	***0,001*** **
	DBP	97,83	75,94	15	1	0	-3,465	***0,001*** **
With CPAP	**SBP**	171,20	140,00	8	1	0	-2,196	***0,028*** *
	DBP	85,10	77,00	6	3	0	-1,304	0,192

*p<0,05 **p<0,01 ***p<0,001

Table 6: Wilcoxon tests comparing blood pressure values (initial and final) according to the use of CPAP

Calcium channel antagonists: Table 7 shows no difference between the use of calcium channel antagonists in patients with CPAP or without CPAP.

THERAPY	Without CPAP		With CPAP	
	Initial	Final	Initial	Final
Calcium channel antagonists	11	12	5	5

Table 7: Distribution of users in the initial and final phases of the study, according to the therapy with calcium channel antagonists, according to the use of CPAP

In Tables 8 and 9, there was an average reduction from 18.64 mg to 8.33 mg in patients without CPAP; and from 9 mg to 7 mg in patients using CPAP, with no statistically significant differences in both situations (p> 0.05).

Dosage (mg) of calcium channel antagonists		Minimum	Maximum	Average	Dp
Without CPAP	**Initial**	5	60	18,64	21,69
	Final	5	10	8,33	2,46
With CPAP	**Initial**	5	10	9,00	2,24
	Final	5	16	7,00	2,74

Table 8: Statistics on the dosage (initial and final) of calcium channel antagonists according to the use of CPAP

Dosage (mg) of calcium channel antagonists	Negative rank	Positive rank	Tie	Wilcoxon	
				z	p
Without CPAP	1	2	4	0,000	1,000
With CPAP	---	---	3	0,000	1,000

*p<0,05 **p<0,01 ***p<0,001

Table 9: Wilcoxon tests comparing the dosages (initial and final) of calcium channel antagonists according to the use of CPAP

Beta-blockers: Table 10 shows no difference between the use of beta-blockers in patients with CPAP or without CPAP.

THERAPY	Without CPAP		With CPAP	
Beta-blockers	Initial	Final	Initial	Final
	7	7	3	3

Table 10: Distribution of users in the initial and final phases of the study, according to the therapy with beta-blockers, depending on the use of CPAP

In Tables 11 and 12, we can see that there was an increase in both groups of beta-blocker dosages, an average increase from 7.5mg to 10mg in patients without CPAP; and from 2.92mg to 4.17mg in patients using CPAP, with no statistically significant difference in both situations (p> 0.05).

Dosage (mg) of beta blockers		Minimum	Maximum	Average	Dp
Without CPAP	**Initial**	3	25	7,50	7,77
	Final	3	25	10,00	10,31
With CPAP	**Initial**	1	5	2,92	1,91
	Final	3	5	4,17	1,44

Table 11: Statistics regarding the dosage (initial and final) of beta-blockers according to the use of CPAP

Dosage (mg) of beta blockers	Negative rank	Positive rank	Tie	Wilcoxon	
				z	p
Without CPAP	1	1	4	-0,447	0,655
With CPAP	0	0	2	0,000	1,000

*p<0,05 **p<0,01 ***p<0,001

Table 12: Wilcoxon tests comparing the dosages (initial and final) of beta blockers according to the use of CPAP

Diuretics: In Table 13, and regarding the use of diuretics, we see an increase in patients using furosemide in both CPAP users and non-users. Indapamide also increased in patients without CPAP, having decreased in patients with CPAP. As it reduced the use of hydrochlorothiazide (without CPAP) and chlorothalidone (with CPAP) Spironolactone and torasemide were prescribed at subsequent consultations in patients without the need for CPAP.

THERAPY	Without CPAP		With CPAP	
Diuretics	**Initial**	**Final**	**Initial**	**Final**
Chlorothalidone	2	1	1	---
Furosemide	2	3	3	5
Hydrochlorothiazide	3	---	---	---
Idapamide	7	6	2	1
Spironolactone	---	1	---	---
Torasemida	---	1	---	---

Table 13: Distribution of users in the initial and final phases of the study, according to diuretic therapy, depending on the use of CPAP

In Tables 14 and 15, and regarding the dosage statistics (mg) of diuretics, we found an average decrease from 14.57mg to 14.41mg in patients without CPAP; and an average increase from 24.17mg to 30.21mg in patients using CPAP, highlighting the absence of statistically significant differences in both situations (p> 0.05).

Dosage (mg) of diuretics		Minimum	Maximum	Average	Dp
Without CPAP	**Initial**	2	40	14,57	14,41
	Final	1	40	14,06	15,18
With CPAP	**Initial**	3	40	24,17	18,48
	Final	1	60	30,21	20,62

Table 14: Statistics on the dosage (initial and final) of diuretics according to the use of CPAP

Dosage (mg) of diuretics	Negative rank	Positive rank	Tie	Wilcoxon	
				z	p
Without CPAP	3	3	4	-0,105	0,916
With CPAP	1	2	1	-1,069	0,285

*p<0,05 **p<0,01 ***p<0,001

Table 15: Wilcoxon tests comparing the dosages (initial and final) of diuretics according to the use of CPAP

Angiotensin Conversion Enzyme Inhibitors (ACEI): In Table 16, and regarding the use of ACE inhibitors, we see an increased use of ACEI in both groups.

THERAPY	Without CPAP		With CPAP	
ACEI	**Initial**	**Final**	**Initial**	**Final**
	12	16	5	6

Table 16: Distribution of users in the initial and final phases of the study, according to ACEI therapy, depending on the use of CPAP

In Tables 17 and 18, and regarding the statistics of dosages (mg) of ACEIs, we found an average increase from 7.25mg to 7.81mg in patients without CPAP; and an average reduction from 12mg to 9.17mg in patients using CPAP, highlighting the absence of significant statistical differences in both situations (p> 0.05).

Dosage (mg) of ACE inhibitors		Minimum	Maximum	Average	Dp
Without CPAP	**Initial**	4	10	7,25	2,60
	Final	5	20	7,81	4,07
With CPAP	**Initial**	5	20	12,00	7,58
	Final	5	20	9,17	5,84

Table 17: Statistics on the dosage (initial and final) of the ACE inhibitors according to the use of CPAP

Dosage (mg) of ACE inhibitors	Negative rank	Positive rank	Tie	Wilcoxon	
				z	p
Without CPAP	2	3	6	-0,542	0,588
With CPAP	2	0	2	-1,342	0,180

*p<0,05 **p<0,01 ***p<0,001

Table 18: Wilcoxon tests comparing the dosages (initial and final) of the ACE inhibitors according to the use of CPAP

Angiotensin II Receptor Antagonists (ARA II): In Table 19, and about the use of ARA II, we see a decrease in its use in patients who do not use CPAP.

THERAPY	Without CPAP		With CPAP	
ARA II	Initial	Final	Initial	Final
	6	2	3	2

Table 19: Distribution of users in the initial and final phases of the study, according to ARA II therapy, depending on the use of CPAP

In Tables 20 and 21, and regarding ARA II dosage statistics (mg), we found an average increase from 40mg to 41.67mg in patients without CPAP; and an average reduction from 50mg to 35mg in patients using CPAP, highlighting the absence of statistically significant differences (p> 0.05).

Dosage (mg) of ARA II		Minimum	Maximum	Average	Dp
Without CPAP	**Initial**	20	80	40,00	21,91
	Final	5	80	41,67	37,53
With CPAP	**Initial**	20	80	50,00	30,00
	Final	20	50	35,00	21,21

Table 20: Statistics regarding the dosage (initial and final) of ARA II according to the use of CPAP

Dosage (mg) of ARA II	Negative rank	Positive rank	Tie	Wilcoxon	
				z	p
Without CPAP	1	0	2	-1,000	0,317
With CPAP	0	0	1	a)	a)

*p<0,05 **p<0,01 ***p<0,001
NOTE: a) inability to perform the Wilcoxon Test due to deficit of users in the group
Table 21: Wilcoxon tests comparing the dosages (initial and final) of ARA II according to the use of CPAP

Nitrates: In Table 22, and regarding the use of nitrates, we see an increase in the number of patients using the nitrates in both CPAP users and non-users.

THERAPY	Without CPAP		With CPAP	
Nitrates	Final	Initial	Final	Final
	1	6	3	5

Table 22: Distribution of users in the initial and final phases of the study, according to nitrate therapy, depending on the use of CPAP

In Tables 23 and 24, and regarding the dosage statistics (mg) of nitrates, we found an average increase from 5mg to 20mg in the only patient without CPAP; and an average reduction from 26.67mg to 24mg in patients using CPAP, highlighting the absence of statistically significant differences (p> 0.05).

Dosage (mg) of nitrates		Minimum	Maximum	Average	Dp
Without CPAP	Initial	5	5	5	---
	Final	20	20	20	---
With CPAP	Initial	20	40	26,67	11,55
	Final	20	40	24,00	8,94

Table 23: Statistics regarding the dosage (initial and final) of nitrates according to the use of CPAP

Dosage (mg) of nitrates	Negative rank	Positive rank	Tie	Wilcoxon	
				z	p
Without CPAP	0	1	0	a)	a)
With CPAP	0	0	3	0,000	1,000

*p<0,05 **p<0,01 ***p<0,001
NOTE: a) inability to perform the Wilcoxon Test due to deficit of users in the group
Table 24: Wilcoxon tests comparing the dosages (initial and final) of nitrates according to the use of CPAP

Adrenergic Alpha 2 Agonists: Regarding the use of alpha 2 adrenergic agonists, such a study was not possible to be carried out, as only one patient uses them (Methyldopa and Clonidine), maintaining the dosage from the 1st to the last consultation.

Table 25 shows that the mean heart rate is higher in the group of patients without CPAP than in those with CPAP

Heart Rate values	Minimum	Maximum	Average	DP
Without CPAP	56	103	85.42	9.59
With CPAP	58	104	84.57	8.4

Table 25: Statistics regarding the Heart Rate values according to the use of CPAP

Table 26 shows a statistically significant decrease in the mean hematocrit values of the Group of Patients with CPAP compared to the group of denontes without CPAP.

Hematocrit values	Minimum	Maximum	Average	DP
Without CPAP	41	52	43.21	3.46
With CPAP	34	47	40.45	3.39

Table 26: Statistics regarding the Hematocrit values according to the use of CPAP

Conclusions

SAH secondary to OSAS is more prevalent in male patients, with the average age being higher in female patients than in male patients. Age groups ≥ 65 years old and ≤ 50 years old started CPAP treatment.

In the initial assessment of the patients' BMI, it was found that 74.4% of the patients were obese. Patients with grade I obesity were those who started CPAP the most. Over time, there was a 19.6% reduction in obesity, accompanied by this decrease with the decrease in the use of CPAP. We can conclude that the decrease in BMI contributes to the reduction of the need to use CPAP.

Daytime and night-time SBP and DBP decrease over time, both in patients without using CPAP and in patients with CPAP, as seen in the literature.

In cardiopulmonary polysomnography and spirometry, it was found that patients with more severe indexes of AHI and bronchial obstruction started CPAP and with the use of it, improvement of parameters was observed, results compatible with that described in the literature.

It is concluded that in patients with SAH secondary to OSAS who started CPAP, there is a decrease in the dosages of the drugs in general, but that, despite the reduction in the SBP of about 30 mmHg and the DBP of about 8 mmHg, and because the initial blood pressures are very high, it was not possible to reduce the number of active substances. These variations are not statistically significant; however, they are valid conclusions for the sample, but they cannot be generalized for the general population. It is concluded that cardiac frequency and hematocrit decrease in the group of patients with CPAP. This study draws attention to the need to start treatment with CPAP at an earlier stage of hypertension in patients with OSAS so that there is a possibility of reducing the number of antihypertensive drugs.

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