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A Very Severe Obstructive Sleep Apnea Syndrome: In a Cause of Resistant Hypertension with One Sample Case

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Abstract

When blood pressure values remain above the target in a hypertensive patient treated concomitantly with three anti-hypertensive drugs including a diuretic, maximum well-tolerated doses, this is a resistant arterial hypertension. In this case, it is advisable to look for a secondary cause such as a drug intake that influencing the blood pressure or the presence of obstructive sleeping syndrome (OSAS). We report a clinical case of a patient with a high cardiovascular risk at the age of 50, hypertensive and diabetic, with dyslipidemia and obesity. He was on anti-hypertensive triple therapy at an optimal dose. Her diabetes was balanced with 6.4% glycated hemoglobin. Dyslipidemia has being treated. Despite healthy diet including a low sodium diet and weight loss, blood pressure target was not reached. With self-measurement, the mean arterial pressure was 180/110 mmHg and on ABPM it was 167/113 mmHg. The ventilatory polygraphy finds a severe OSA with an IAH = 56.6. Treatment with PCP (Continuous positive pressure) allowed this patient to control blood pressure. The search for OSA should be systematic in face of resistant hypertension, in particular in overweight or obese patients.

Keywords

Obstructive Sleep Apnea Syndrome (OSAS), Resistant Hypertension, Continuous Positive Airway Pressure (CPAP)

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is a condition characterized with the occurrence during sleep of apneas or hypopneas associated with upper airway collapse [1].

The association between high blood pressure and OSA is common. The main mechanism that explains this association is stimulation of sympathetic nervous system during apneas. Alongside this mechanism, the activation of renin-angiotensin-aldosterone system due to hypoxemia, that increase in preload and afterload linked to negative intrathoracic pressure and finally oxidative stress with dysfunction endothelial and other pathophysiological mechanisms that link arterial hypertension to OSA [1] [2] [3]. From an epidemiological point of view, approximately 50% of patients with OSA are hypertensive [3].

Arterial hypertension (hypertension) associated with obstructive sleep apnea syndrome exhibits several characteristics including high prevalence, diastolic and nocturnal predominance, non-dipper status and resistance to antihypertensive therapy [1].

Treatment of OSAS with continuous positive airway pressure (CPAP) may have beneficial effect on hypertension control when the patient is observant [1] [2].

The objective of this work was to relate the benefit of screening and treatment of severe OSA to resistant hypertension. The patient's that is free and inform his consent has been obtained.

2. Observation

This is a 50 year old patient with a well known hypertension since 2006 and we have been following him since 2013. He was treated with Perindopril 10 mg + Amlodipine 10 mg: 1 tablet per day for several years stable. After discovering his diabetes in 2015, with high blood pressure it was more difficult to control. It was added to his treatment Spironolactone 50 mg + Altizide 15 mg: 1 tablet per day. From June 2019, we added Rilménidine 2 mg per day to it.

The above treatment seemed to be effective. Several other therapeutic combinations had been tried without success, in particular the combination of Amlodipine 10 mg + Valsartan 160 mg, Irbesartan 300 mg + hydrochlorothiazide 25 mg.

Type 2 diabetes was treated with: Metformin 850 mg daily, in combination with Glimépiride 2 mg per day.

He was also treated for dyslipidemia with Rosuvastatin 20 mg per day.

After several months with this same treatment, his hypertension was not balanced.

Interviewing the patient at night while snoring, morning physical asthenia associated with episodes of daytime sleepiness, and erectile dysfunction.

The physical examination noted android-type obesity (abdominal circumference = 102 cm), weight = 79 kg; height = 168 cm; BMI = 27.99 kg/m^2; Blood

pressure = 180/110 mmHg.

The sounds of the heart was regular, the peripheral pulses was well perceived. The rest of the medical checkup was normal.

Biological medical test discovered: Blood sugar = 0.9 g/l; Glycated hemoglobin = 6.4%; Hemoglobin = 17.2 mg/dl; creatinine = 7.6 mg/l with clearance = 129 ml/min; dyslipidemia with LDL = 2.18 g/l, Total cholesterol = 1093 g/l, HDL = 0.55 g/l; triglycerides = 0.95 g/l. CRP = 31.4 mg/L Blood ionogram was normal. Micro albuminuria was found (240 mg/l).

The EKG showed a steady sinus rhythm at 70 beats per min; the layout was normal.

The chest x-ray was normal.

The echocardiogram was normal.

Ambulatory Blood Pressure Measurement (ABPM) showed unbalanced arterial hypertension with 24 hour means BP = 167/113 mmHg, that shows the patient is not dipper (Figure 1).

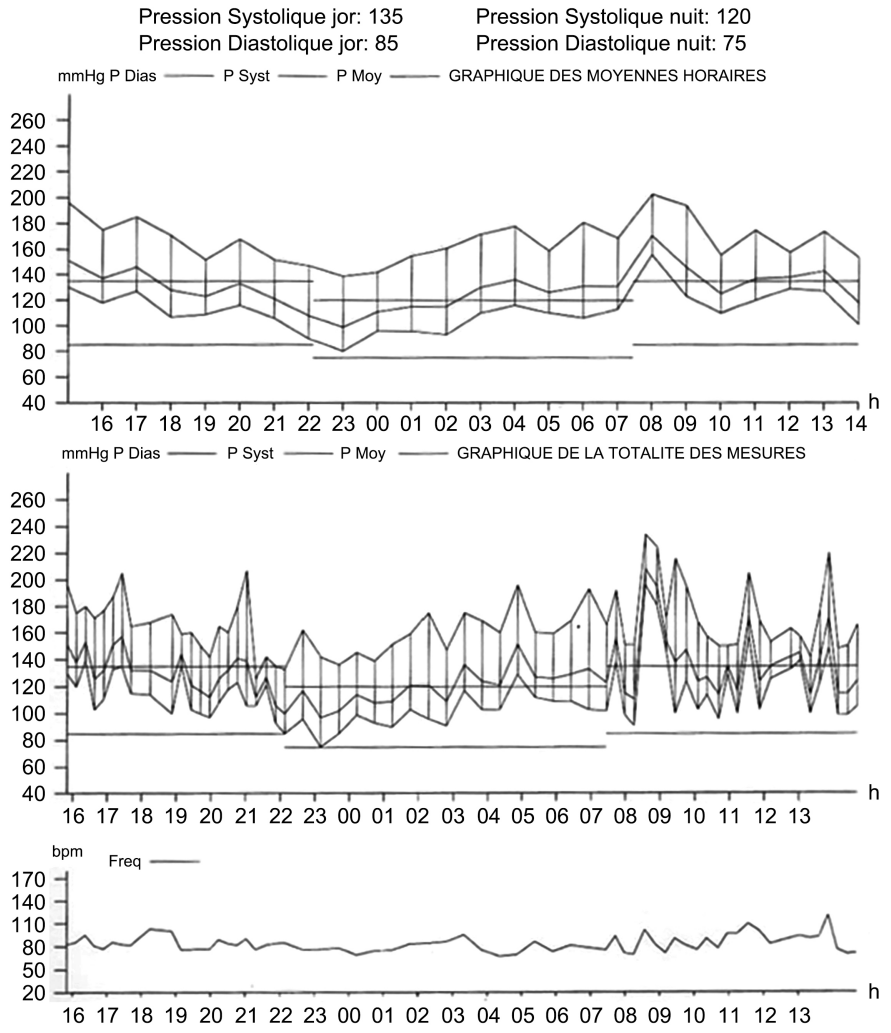


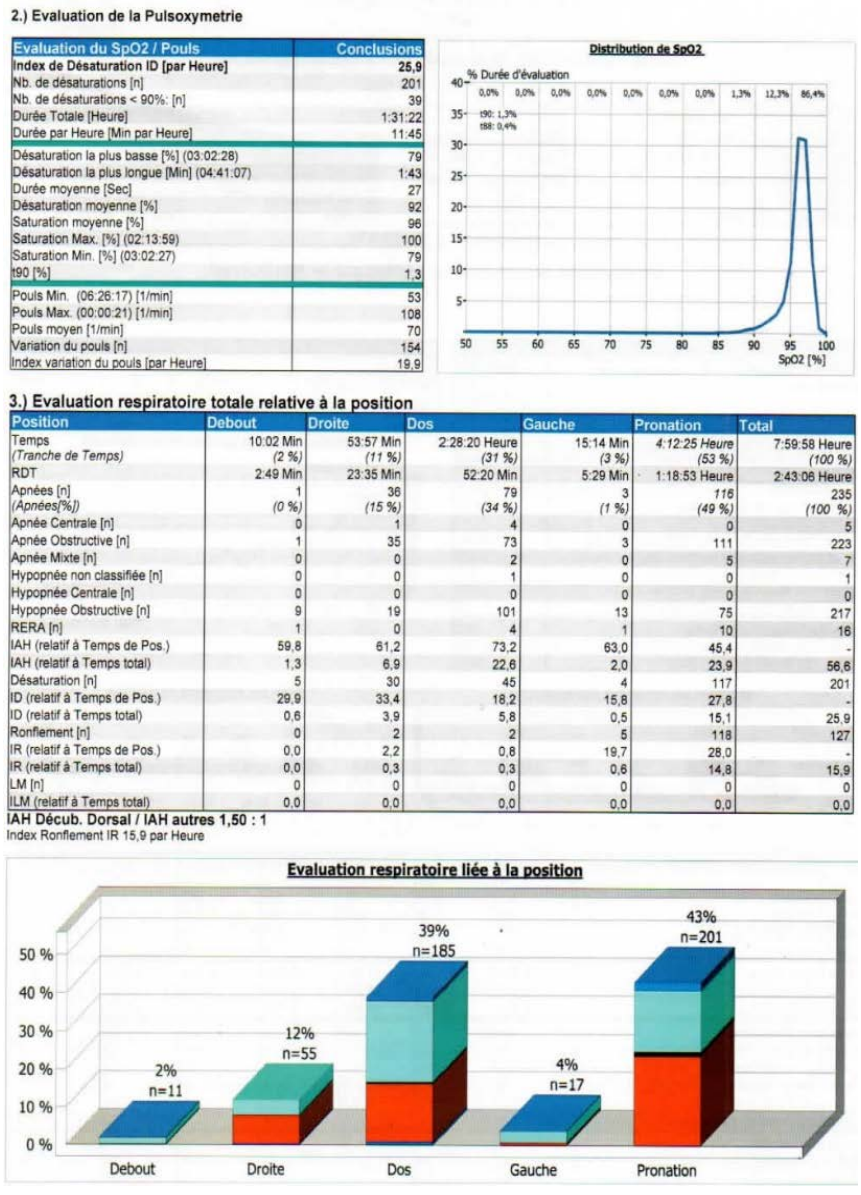
Figure 1. Ambulatory BP measurement, the graph indicates unbalanced hypertension with non-dipper status.

Tensional Self-Monitoring (TMA) was also in favor of uncontrolled arterial hypertenion (means BP = 180/110 mmHg).

In Totality, it is a resistant arterial hypertension in a patient with a high cardiovascular risk.

He was not administering any medication that could increase blood pressure. In this research for a secondary cause therefore turns to an obstructive sleeping apnea syndrome questioning.

Ventilatory polygraphy shows a severe obstructive apnea syndrome with (overall IAH at 56.6/H; RDI = 58.6) without positional character. The conclusion is that curves are presented in the appendix (Figure 2, Figure 3).



2.) Evaluation de la Pulsoxymetrie

Evaluation du SpO2 / Pouls	Conclusions
Index de Désaturation ID [par Heure]	**25,9**
Nb. de désaturations [n]	201
Nb. de désaturations < 90%: [n]	39
Durée Totale [Heure]	1:31:22
Durée par Heure [Min par Heure]	11:45
Désaturation la plus basse [%] (03:02:28)	79
Désaturation la plus longue [Min] (04:41:07)	1:43
Durée moyenne [Sec]	27
Désaturation moyenne [%]	92
Saturation moyenne [%]	96
Saturation Max. [%] (02:13:59)	100
Saturation Min. [%] (03:02:27)	79
t90 [%]	1,3
Pouls Min. (06:26:17) [1/min]	53
Pouls Max. (00:00:21) [1/min]	108
Pouls moyen [1/min]	70
Variation du pouls [n]	154
Index variation du pouls [par Heure]	19,9

3.) Evaluation respiratoire totale relative à la position

Position	Debout	Droite	Dos	Gauche	Pronation	Total
Temps *(Tranche de Temps)*	10:02 Min *(2 %)*	53:57 Min *(11 %)*	2:28:20 Heure *(31 %)*	15:14 Min *(3 %)*	*4:12:25 Heure (53 %)*	7:59:58 Heure *(100 %)*
RDT	2:49 Min	23:35 Min	52:20 Min	5:29 Min	1:18:53 Heure	2:43:06 Heure
Apnées [n] *(Apnées[%])*	1 *(0 %)*	36 *(15 %)*	79 *(34 %)*	3 *(1 %)*	*116 (49 %)*	235 *(100 %)*
Apnée Centrale [n]	0	1	4	0	0	5
Apnée Obstructive [n]	1	35	73	3	111	223
Apnée Mixte [n]	0	0	2	0	5	7
Hypopnée non classifiée [n]	0	0	1	0	0	1
Hypopnée Centrale [n]	0	0	0	0	0	0
Hypopnée Obstructive [n]	9	19	101	13	75	217
RERA [n]	1	0	4	1	10	16
IAH (relatif à Temps de Pos.)	59,8	61,2	73,2	63,0	45,4	-
IAH (relatif à Temps total)	1,3	6,9	22,6	2,0	23,9	56,6
Désaturation [n]	5	30	45	4	117	201
ID (relatif à Temps de Pos.)	29,9	33,4	18,2	15,8	27,8	-
ID (relatif à Temps total)	0,6	3,9	5,8	0,5	15,1	25,9
Ronflement [n]	0	2	2	5	118	127
IR (relatif à Temps de Pos.)	0,0	2,2	0,8	19,7	28,0	-
IR (relatif à Temps total)	0,0	0,3	0,3	0,6	14,8	15,9
LM [n]	0	0	0	0	0	0
ILM (relatif à Temps total)	0,0	0,0	0,0	0,0	0,0	0,0

IAH Décub. Dorsal / IAH autres 1,50 : 1
Index Ronflement IR 15,9 par Heure

Figure 2. Ventilatory polygraphy: oximetry and positional data showing a severe nocturnal desaturation (desaturation index per hour = 25.9; average saturation to 96 % for a basal 100% and minimal 79%).

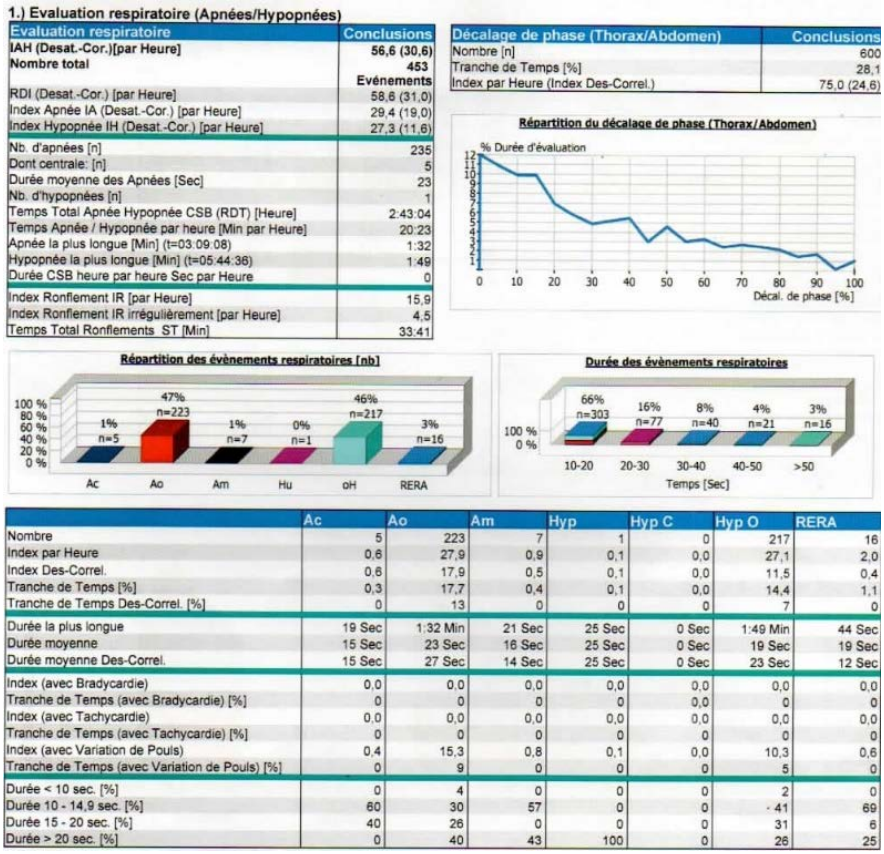


Figure 3. Ventilatory polygraphy: abnormal respiratory events.

Treatment of obstructive apnea syndrome by continuous positive pressure ventilation has been initiated to control high blood pressure. On self-measurement, the means of arterial pressure is 133/80 mmHg from the first month and it has remained stable for 5 months.

3. Discussion

In this present work, we reported a clinical case of a patient with resistant hypertension whose cause has been shown to be obstructive sleeping apnea syndrome (OSAS).

Resistant arterial hypertension (hypertension) is defined by blood pressure values above the therapeutic target despite a treatment combining 3 molecules including a diuretic, at maximum well tolerated doses. Its association with obstructive sleeping apnea (OSA) sleep is common. Thus, in a study on hypertension refractory to treatment, Bernard Waeber and François Feihl [4] showed that OSA was the most frequently found cause (64%). For Damiano *et al.*, The prevalence of OSA reached 83% of hypertensive patients resistant to treatment [3].

In our clinical case, this is a patient with high cardiovascular risk with age 50, hypertensive and diabetic, with dyslipidemia and obesity. Despite the hygieno-dietetic measures comprising a low-sodium diet, weight loss since he weighed more than 90 kg a few years previously (currently his weight is stable at 79 kg)

associated with an anti-hypertensive quadruple therapy at optimal dose, for several months, the blood pressure target was not reached (**Figure 1**). Faced with this scenario, it was recommended to look for a secondary cause, in particular OSA or drug intake that can influence blood pressure.

The questioning of the patient had made it possible to move towards a sleeping apnea syndrome in front of the notion of bulging associated with daytime sleeplessness. Adjoh *et al.* in Togo [6] found that 42.2% of patients were sleepy in their study.

Polysomnography is a reference examination for the search for OSA, combining respiratory polygraphy and recording of sleep [3] [5]. However, ventilatory polygraphy is an alternative to the gold standard. It makes it possible to confirm the presence of apneas and hypopneas, to identify the mechanism (obstructive or central character) and assess the severity. The severity is determined by the index of apnea-hypopnea (IAH) which is a number of apneas and hypopneas per hour. An AHI greater than 30 per hour defines a severe OSA [3]. For clinical case that has been reported, the AHI was 56.6 per hour, qualified as very severe (**Figure 3**).

Continuous positive airway pressure (CPAP) is the treatment of choice for OSA. The beneficial effect of this device on lowering blood pressure has been demonstrated [3] [7] [8]. Provided that the patient is observant. In this case of our patient, this beneficial effect was noted from the first months for his or her treatment mean arterial pressure for self-measurement is 133/80 mmHg. It remains stable for more than 5 months.

4. Conclusion

Resistant hypertension is often seen in overweight or obese patients. This association must systematically search for OSA, treatment of OSA is by CPAP which, when the patient is observant, can significantly lower blood pressure levels and improve the quality of life of patients.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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