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Resistant Arterial Hypertension and Obstructive Sleep Apnea Syndrome in Overweight Patient: A Case Report

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Abstract **Case Report**

Resistant hypertension represents about 5 to 10% of all hypertension. It should systematically lead to a search for a secondary cause. Its association with obesity should raise the possibility of obstructive sleep apnea syndrome (OSAS). This clinical case allows to put the point on the importance of the diagnosis of the OSA as etiology of the resistant HTA still under diagnosed. It allows to remind practitioners and interns in general medicine, cardiology and pneumology the importance of screening. We report the case of a young overweight patient with resistant hypertension admitted to the cardiology department for headaches and ringing in the ears. He underwent all complementary examinations in search of an etiology secondary to his hypertension, but they came back negative. The patient underwent several therapeutic adjustments without clear results on his blood pressure. After questioning the patient's family and friends (nocturnal snoring), we were able to determine that he had OSA, and from there he was able to achieve blood pressure control by regular physical activity and a low-calorie diet. OSA is easy to diagnose, the hardest thing is to know how to think about it in front of a resistant hypertension in overweight subjects. The treatment can be simple hygienic and dietary measures, without resorting to continuous positive pressure ventilation.

Keywords: Resistant arterial hypertension, sleep apnea, obesity, case report.

INTRODUCTION

Resistant hypertension is closely related to OSA (2), especially when the subject is overweight or obese. The simple fact of knowing how to think about it, allows to avoid a panoply of expensive check-ups for the patient and for the insurance companies. The choice of hygienic-dietary measures as treatment can contribute to the improvement of symptoms. We report the case of resistant hypertension revealing OSA in a young 26-year-old patient, followed for resistant hypertension whose etiology could not be diagnosed despite several paraclinical examinations. The family's contribution was of great importance, raising the notion of the patient's nocturnal snoring, which led us to the diagnosis of moderate-severity OSA that required a low-calorie diet and regular physical activity without recourse to continuous positive pressure ventilation.

CASE PRESENTATION

This is a 26-year-old patient admitted to the cardiology department for the appearance of morning headaches associated with intermittent ringing in the ears. He was not known to have heart or kidney disease and had not been taking NSAIDs, corticoids or licorice.

The patient is overweight and follows a sedentary lifestyle. The body weight was 105 kg for a height of 184 cm and the body mass index was 31 kg/m2 at admission.

The history of his illness dates back to 6 months with an incidental discovery of his hypertension during a usual consultation with a general practitioner. His blood pressure was 182/110. The patient was initially put on amlodipine 5mg and a low-salt diet and then lost to follow-up. A few months later, he presented with headaches and ringing in the ears associated with a blood pressure measured in consultation at 170/98, which required hospitalization in the cardiology department for an etiological assessment of secondary hypertension in our young patient.

On cardiovascular examination: the heart rate was 109 beats per minute. The peak shock was punctiform and in place. The heart sounds were well

perceived with the presence of a systolic murmur at the aortic focus rated 2/6. There was no pericardial rub or gallop sound. Auscultation of the neck and abdominal vessels did not reveal any murmur. The blood pressure on admission was 178/102 mm Hg symmetrical to the 4 limbs.

On pulmonary examination: The respiratory rate was 18 cycles per minute. Pulse oxygen saturation was 98%. On pulmonary auscultation, vesicular murmurs were well perceived with absence of rales or pleural effusion syndrome.

The thyroid gland was not palpable. There was no hair loss, skin changes or alteration of bowel movements. The neurological examination was unremarkable.

Blood count, renal function, fluid and electrolyte balance, liver transaminases, troponin level, lipid profile, fasting blood glucose and TSH were within normal limits. Urinary free cortisol, aldosterone and plasma renin in standing and lying position, urinary metanephrines were normal.

Electrocardiography: the rhythm was regular sinus. Absence of hypertrophy of the left ventricle and atrium. Absence of conduction and repolarization disorders.

Echocardiography: the left ventricle was of preserved size and systolic function, the ejection fraction was 67% with preserved global and segmental kinetics. The mitro-aortic valves were without abnormality. The atria were not dilated. The filling pressures were low. The rest of the ultrasound examination was within normal limits.

24-hour BP measurement: daytime and nocturnal grade 2 hypertension associated with blood pressure variability. The nocturnal decline in blood pressure was 3% (non-dipper profile).

Thoracic CT-angiography: aorta without notable abnormalities. CT angiography of renal arteries with adrenal section: no significant abnormalities.

After a negative etiological workup, the diagnosis of essential hypertension was retained. The patient was put on fixed, low-dose triple antihypertensive therapy combining perindopril 5mg, amlodipine 5mg and indapamide 1.25mg.

An ambulatory blood pressure measurement was performed 1 month after hospitalization showing a mean daytime and nighttime blood pressure of 155/88mmHg. A change in dosage was indicated with a switch to full-dose triple therapy with persistent high blood pressure. The hypertension was considered resistant.

The young patient was referred to the pneumology department for a ventilatory polygraphy in search of suspected OSA due to the nocturnal snoring reported by his family.

This diagnostic test confirmed a moderate OSA with 18.3 events per hour in the apnea-hypopnea index and the existence of an important ronchopathy with a snoring index of 477.9/h. After hygienic-dietary measures including regular physical activity and a hypocaloric diet for 3 months, our patient's body mass index decreased to 26.2kg/m2 with a stabilization of his blood pressure at 128/65mmHg with the previous pharmacological diet.

DISCUSSION

Resistant hypertension is defined as a blood pressure greater than 140/90 mmHg under optimal dose antihypertensive therapy including a diuretic [1]. There is a close relationship between OSA and treatment-resistant hypertension [2]. Marin *et al*., conducted a study on a sample of 21003 unknown hypertensive patients with suspected OSA. The results of this study concluded that patients with confirmed OSA had a higher rate of hypertension compared to patients without OSA [3]. In a study conducted in an African population, Konin *et al*., were able to demonstrate better control of hypertension in non-apneic patients than in apneic patients [4]. The sympathetic system is stimulated by apnea through baroreceptors and chemoreceptors. This system is further stimulated by asphyxia due to nocturnal awakenings. This results in hypertension and tachycardia. AH in OSAS is characterized by a non-dipper profile on 24-hour ambulatory blood pressure measurements (ABPM). A decrease in nocturnal blood pressure of less than 10% should raise suspicion of OSA [5]. In a Spanish study, 70% of 24-hour ABPMs confirmed treatment-resistant hypertension and a non-dipper blood pressure profile in patients with more than 30 apnea-hypopnea events per hour [6]. Obesity is a risk factor for OSA due to upper airway obstruction. The risk of developing OSAS in obese patients is 2.55 times higher than in non-obese patients [7]. The medical treatment of hypertension during OSAS can be ineffective with a poor control of blood pressure. Hence the interest of continuous positive airway pressure (CPAP) treatment. This interest is more important when it is a case of severe OSA with a good compliance of the antihypertensive treatment [8]. Nevertheless, in a meta-analysis performed on 3000 patients with OSA, it was shown that CPAP treatment was associated with a weight gain of 400g [9]. The American Thoracic Society advocates weight loss for all patients with OSA and overweight [10]. This was the case for our patient whose blood pressure figures were balanced after diet and hygiene alone.

CONCLUSION

Although resistant hypertension in young people requires a broad etiological exploration, its association with overweight should lead the practitioner to ask for a polysomnography first to look for OSA. This practice will avoid costly and inconclusive complementary examinations. An apnea/hypopnea index greater than 5 events per hour allows the diagnosis to be made and the resistance of the hypertension to be resolved by simple hygienic and dietary measures.

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