Severe hypertension in a young patient. What is the cause?

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Abstract

Obstructive sleep apnea syndrome (OSAS) is recognized nowadays as an exceedingly common cause of secondary arterial hypertension. Reduction in arterial oxygen pressure during each apneic episode activates reflexes that result in increased sympathetic and renin-angiotensin-aldosterone system activity, and consequently increased blood pressure and heart rate values after each apneic episode. We present a case of a young male hypertensive patient referred for a cardiologic evaluation due to an abnormal routine electrocardiogram recorded on admission in the neurology department for a second ischemic stroke. Due to the fact that the patient is young and hypertensive, a secondary cause of arterial hypertension is suspected. Following nighttime recordings of airflow, oxygen saturation, blood pressure and electrocardiogram, the diagnosis of obstructive sleep apnea syndrome is confirmed and suggested as the underlying cause of arterial hypertension. Further investigations reveal the cardiovascular complications of the obstructive sleep apnea syndrome and the secondary arterial hypertension: left ventricular hypertrophy, aortic aneurism requiring surgical treatment, ventricular arrhythmias. The two reported ischemic strokes might also be considered among the obstructive sleep apnea complications, superimposed with the smoker and obese status of the patient. Obstructive sleep apnea is a common cause of secondary arterial hypertension among young hypertensive patients and should be suspected especially in obese and/or active smoker patients as well as in diabetic patients and in those with COPD and/or severe CKD. If undiagnosed and untreated, OSA lead to uncontrolled hypertensive disease and target organ damage and an early onset of major cardiovascular events in young hypertensive patients.

Keywords: obstructive sleep apnea syndrome, hypertension, young, cardiovascular events

Introduction

Obstructive sleep apnea syndrome, defined through a combination of at least five episodes of obstructive breathing per hour of sleep and symptoms such as choking or gasping during sleep, recurrent awakening, unrefreshing sleep and daytime sleepiness, is recognized nowadays as an exceedingly common cause of secondary arterial hypertension (1). The elevated blood pressure values are a result of recurrent nocturnal hypoxemia, which impairs the sympathetic nerve activity as well as the renin-angiotensin-aldosterone system. Continuous positive airway pressure treatment results in a lowering of blood pressure values,
both systolic and diastolic, both during wakefulness and sleep.

Case report

A 46-year-old male with a previous medical history of hypertension (diagnosed at the age of 42 and treated daily with 50mg of metoprolol, 10mg of enalapril and 5mg of amlodipine) and a previous ischemic stroke 8-years ago, was referred for an evaluation in the cardiology department due to the presence of ventricular bigeminy on the electrocardiogram recorded on admission in the neurology department, accusing abrupt onset of weakness and numbness in the arm and leg on the left side of the body, being diagnosed with acute ischemic stroke in the territory supplied by the right middle cerebral artery.

The physical examination revealed no significant changes, apart from the increased body weight - 38.9 kg/m² body-mass index accounting for second degree obesity, with regular heart sounds at a heart rate of 86/min, a blood pressure value of 135/85mmHg, with no heart or vascular murmurs and without signs of pulmonary or systemic congestion, symmetric bilateral pulsatile arteries.

The transthoracic echocardiography revealed a concentric hypertrophied left ventricle without abnormal regional wall movement with preserved systolic function (LVEF=55%), mild diastolic dysfunction (impaired relaxation pattern) with normal filling pressures, and no criteria for pulmonary hypertension and no pericardial effusion. Furthermore, an ascending aortic aneurysm was identified with moderate aortic regurgitation (Figure 1).

In this context, a thoracic computed tomography was performed, revealing an ascending aortic aneurysm of 6 cm maximum dimension at the level of the aortic root, without evidence of aortic dissection (Figure 2).

In order to investigate the previously documented ventricular bigeminy, a 24-hour electrocardiogram (ECG) Holter monitoring was performed. This revealed sinus rhythm superimposed with monomorphic premature ventricular complexes accounting for approximately 11% of the total heart beats. The ventricular extrasystoles (VES) were organized as ventricular bigeminy, couplets, triplets and non-sustained ventricular tachycardia. No other arrhythmias or ST segment

Figure 1. Transthoracic echocardiography. PLAX view: concentric LV hypertrophy (IVS = 12mm, LV posterior wall - 13mm), dilated ascending aorta (59mm) with mild regurgitation at color Doppler examination. PLAX: parasternal long axis; LV: left ventricle; IVS: intraventricular septum.
or T wave changes were identified (Figure 3-5). The majority of the VES were recorded during nighttime.

Taking into account that hypertension onset was at an early age, a diagnosis of secondary arterial hypertension was suspected. In this respect, the most common causes of secondary arterial hypertension (renal parenchymal disease, renal artery stenosis, primary aldosteronism) were excluded due to the normal values of the laboratory tests and paraclinical evaluation.

Considering the night-time symptoms such as intermittent snoring and disturbed sleep, the presence of obesity in active smoker and the increased occur-

![Thoracic computer tomography](image1.png)

Figure 2. Thoracic computer tomography.

![Couplets on 24-hour ECG Holter monitoring](image2.png)

Figure 3. Couplets on 24-hour ECG Holter monitoring.
ence of VES during sleep period, a diagnosis of obstructive sleep apnea syndrome was speculated. Therefore, a polysomnography (recordings of airflow, oxygen saturation, blood pressure and electrocardiogram) was performed during nighttime. These revealed the presence of a severe sleep apnea syndrome with average apnea-hypopnea index (AHI) of 37.6 (Figure 6).

The coronary angiography evaluation, performed in order to exclude the ischemic origin of ventricular arrhythmias, revealed no significant coronary lesions.

The following diagnosis was established: aortic aneurism without evidence of aortic dissection and mild aortic regurgitation; hypertensive heart disease with preserved LV systolic function, monomorphic ventricular extrasystolic systematized arrhythmia (couplets,
triplets and non-sustained ventricular tachycardia) grade III secondary arterial hypertension and severe obstructive sleep apnea syndrome requiring nighttime CPAP.

Treatment with beta-blocker (bisoprolol), angiotensin-receptor blocker and non-dihidropiridinic receptor blocker fixed combination (olmesartan + amlodipine) targeting an heart rate of 60bpm and a BP value of 120/70mmHg, statine (atorvastatine), antiplatelet therapy (ASA) as well as and night-time CPAP was initiated.

Treatment with positive airway pressure resulted in improvement of sleep related symptoms and a significant decrease of ventricular arrhythmias on the 24-hour electrocardiogram monitoring and a decrease in AHI from 37.6 to 26.9 (Figure 7).

The evolution during hospitalization was towards improvement. As a consequence, the patient was discharged after 5 days. The recommended medical treatment at discharge was: antiplatelet medication (clopidogrel 75 mg q.d.), beta-blocker (bisoprolol 5 mg b.i.d.), calcium channel blockers and sartan fixed combination (amlodipine + olmesartan 5/20 mg q.d.), statine (atorvastatin 40 mg q.d.) and neurotropic medication (piracetam 600 mg q.d.) together with
As a result of the these changes in physiology, a number of cardiovascular complications have are related to untreated OSA, the association being more convincing for the subgroup of severe OSA (characterized by more than 30 episodes of apnea and hypopnea per hour of sleep).

Thus, OSA increases the risk of stroke independently of other cerebrovascular risk factors (7). It increases stiffness of large arteries and thus contributes to left ventricular remodeling, studies revealing that left ventricular hypertrophy is more prevalent in hypertensive patients without OSA, in normotensive patients with OSA, and even more in patients affected by both arterial hypertension and OSA (8). Furthermore, it promotes left ventricular dysfunction and progression towards congestive heart failure (9). There is also a correlation between OSA and arrhythmias, non-sustained ventricular tachycardia being one of the most prevalent arrhythmias related with this disorder (10).

The presented case confirms the abovementioned theories. A male patient is referred for cardiologic evaluation due to an abnormal routine electrocardiogram. Due to the fact that the patient is young and hypertensive, a secondary cause of arterial hypertension is suspected. Following nighttime recordings of airflow, oxygen saturation, blood pressure and electrocardiogram, the diagnosis of obstructive sleep apnea syndrome is confirmed and suggested as the underlying cause of arterial hypertension. Further investigations reveal the cardiovascular complications of the obstructive sleep apnea syndrome and the secondary arterial hypertension: left ventricular hypertrophy, aortic aneurism, ventricular arrhythmias. The two reported ischemic strokes might also be considered among the obstructive sleep apnea complications, superimposed with the smoker and obese status of the patient.

**Conclusions**

A cause of secondary arterial hypertension should be investigated in every young hypertensive patient. Obstructive sleep apnea is a common cause of secondary arterial hypertension among young hypertensive patients and should be suspected especially in obese and/or active smoker patients as well as in dia-
betic patients and in those with COPD and/or severe CKD.

If undiagnosed and untreated, OSA as well as other causes of secondary HT, lead to the progression of uncontrolled hypertensive disease towards target organ damage and an early onset of major cardiovascular events in young hypertensive patients.

References


