

## CASE PRESENTATION

# The ambulatory blood pressure measurement (A.B.P.M.) role in the “masked hypertension” diagnosis

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### SUMMARY

“Masked hypertension” is a relatively new entity in the high blood pressure, for which the only way to highlight it is the ambulatory blood pressure (BP) monitoring/24h in the context of a high clinical suspicion. For many of the today patients, the stress of the medical consulting which is the essence of the well-known “white coat hypertension” is much exceeded by the daily stress especially that related the work<sup>1</sup>. The patient we present is a typical example of “masked hypertension” due to a combination of stress and a disease which is becoming more common lately: sleep apnoea. Modern methods of diagnosis of sleep apnoea and also the accessibility made diagnosis possible in the context of a high clinical suspicion. Recent clinical studies proving important link between cardiovascular disease and sleep apnoea syndrome more than common risk factors, providing explanation of inexplicable nocturnal cardiovascular events: arrhythmia, night hypertension, sudden death<sup>2</sup>.

### CASE PRESENTATION

44 year old patient with no subjective complaints comes to a cardiology outpatient consultation after a periodic occupational health consulting which highlighted moderately increased blood pressure (BP 145/95 mmHg). The patient has no significant personal history and from the family history we notice that his father died suddenly at 51 years. As for his life style, we face a dynamic patient, authoritative, irritable, and permanently voyaging across the country and abroad, involved in multiple projects, heavy smoker, especially during the nights, chronic alcohol consumer (~150-200 ml alcohol/day). The patient also speaks about insomnia, nightmares linked to the payment of debts and to the many projects he is involved with a family climate threatened by stress, irritability, travels and lately “long sno-

re, stop breathing sometimes and wakes often” reported by his wife.

Clinical examination reveals a patient with moderate obesity (body mass index 32) and blood pressure between limits at the moment of the examination (BP 135/80 mmHg) with no other alterations. We notice that our patient is strongly influenced by the fact that he is approaching the age when his father suddenly died and he is obsessed of measuring blood pressure at home (all normal) (**Figure 1**).

The electrical circuit reveals Synusal Rhythm 70/min, axe QRS 60°, left ventricular hypertrophy (LVH) with secondary terminal modifications (**Figure 2**). The electrical aspect of the left ventricular hypertrophy is confirmed by the echocardiography examination that shows left cavities to the upper part of the normal limits, Left ventricle (LV) 41/60 mm, Left atrium (LA) 44 mm, concentric left ventricular hypertrophy, interventricular septum (IVS) 15 mm, left ventricle posterior wall (PWL) 14 mm without segmental kinetic disorders, with global systolic function to the lower part of

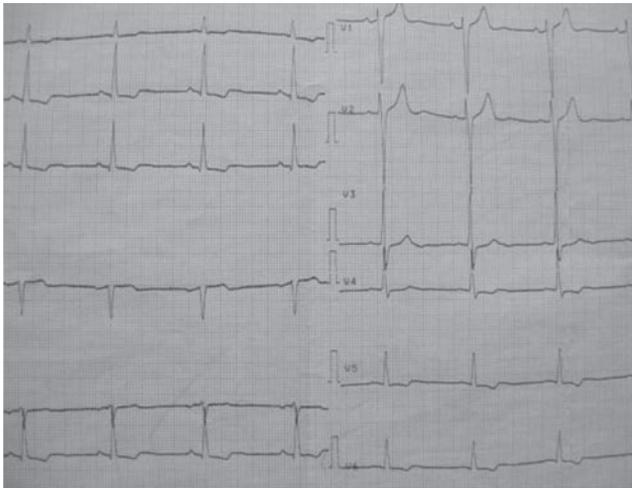
23 OCT	TA = 128/80	26 NOVEMBRI	135/85 mmHg
25 OCT	TA = 132/80	27 NOVEMBRI	137/80 mmHg
27 OCT	TA = 127/75	28 NOVEMBRI	134/72 mmHg
29 OCT	TA = 131/80	1 DECEMBRI	136/80 mmHg
1 NOVEMBRI	TA = 135/80	2 DECEMBRI	138/70 mmHg
3 NOVEMBRI	TA = 137/80	3 DECEMBRI	129/70 mmHg
5 NOVEMBRI	TA = 139/82	4 DECEMBRI	120/68 mmHg
7 NOVEMBRI	TA = 130/73	5 DECEMBRI	127/60 mmHg
9 NOVEMBRI	TA = 125/75	6 DECEMBRI	133/66 mmHg
11 NOVEMBRI	TA = 130/62	7 DECEMBRI	135/60 mmHg
13 NOVEMBRI	TA = 135/70	8 DECEMBRI	127/60 mmHg
15 NOVEMBRI	TA = 140/70 mm	9 DECEMBRI	129/60 mmHg
17 NOVEMBRI	TA = 127/70	10 DECEMBRI	135/60 mmHg
19 NOVEMBRI	TA = 125/60	11 DECEMBRI	120/60 mmHg

**Figure 1.** The results of auto blood pressure measurement, obsessive memorized in his notebook.

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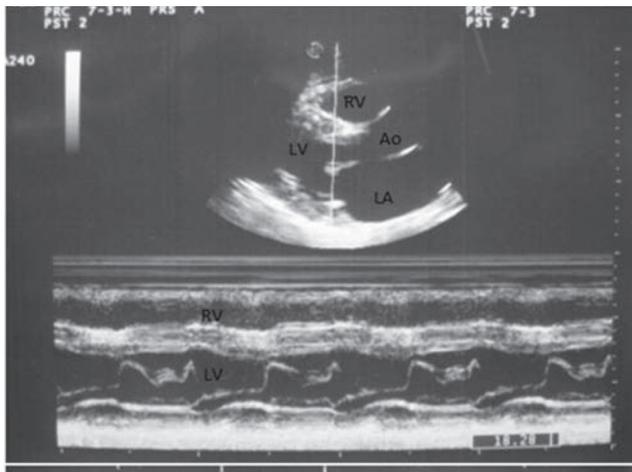
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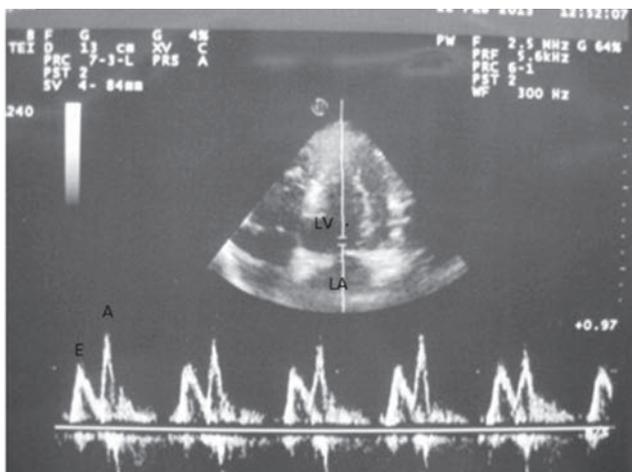


**Figure 2.** ECG with left ventricular hypertrophy and secondary repolarisation abnormality.

the normal limits (EF 55%), altered relaxation type diastolic dysfunction, moderate aortic atheromatosis,



**Figure 3.** Echocardiography, parasternal long axis, M mode, showing left ventricular hypertrophy and a preserved segmentary systolic function.



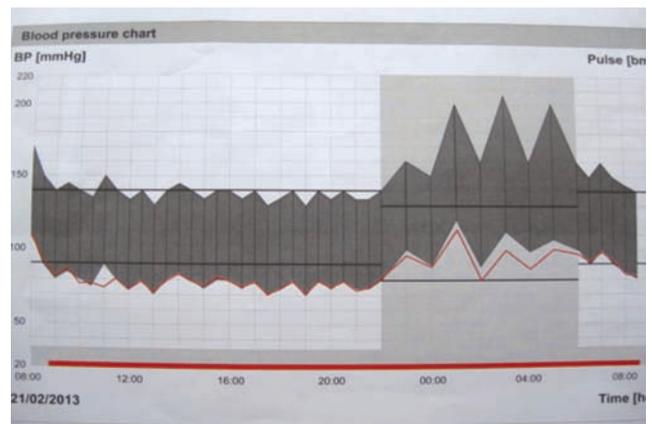
**Figure 4.** Mitral inflow velocities showing a pattern of grade 1 diastolic dysfunction (impaired relaxation and normal filling pressure).

right cavities non dilated, normal valvular apparatus, without pericardial fluid (**Figure 3, 4**).

Biological profile reveals carbohydrate and lipid metabolism in limits (glucose 0.96 mg%, Cholesterol 178 mg%, Low Density Lipoprotein (LDL) 110 mg%, triglycerides 145 mg%), creatinine 0.9 mg%, uric acid 6.8%, normal ionograma, micro albuminuria 140 mg/24h.

We are facing a patient with left ventricular hypertrophy electrical and in ultrasound examination, micro albuminuria but presenting normal blood pressure values during the clinical examination and also during the self-measurements made at home. As the hypertrophy of athlete was excluded from history, hypertrophic or dilated cardiomyopathy and aortic stenosis “clinical silent” were not identified by ultrasound, the clinical suspicion is masked hypertension, and for confirmation, it is necessary to monitor ambulatory BP/24 hours.

The blood pressure graphic obtained (**Figure 5**) points out an obviously non-dipper profile tension, with blood pressure values during the daytime of 142/84 mmHg and at night of 178/112 mmHg, hypertensive load during the day at 12% and at night at 100%. The tensional non-dipper profile is associated more frequently with left ventricular hypertrophy, stroke, peripheral arterial disease, albuminuria and cardiovascular mortality. From the circumstances frequently associated with non-dipper profile, some are obviously excluded in this case (preeclampsia, malignant hypertension, diabetic neuropathy), some are excluded after the targeted anamnesis (immunosuppressant such as medication use, corticosteroid, sympathomimetic). As for the secondary hypertension, this was not confirmed by abdominal ultrasound examination which revealed normal kidney size, symmetry, normal urinary ionograma and urinary metanephrines. Sleep apnoea is also one of the causes of secondary hypertension, often associated with non-dipper profile tension, also confirmed by



**Figure 5.** The blood pressure graphic/24h with non-dipper profile.

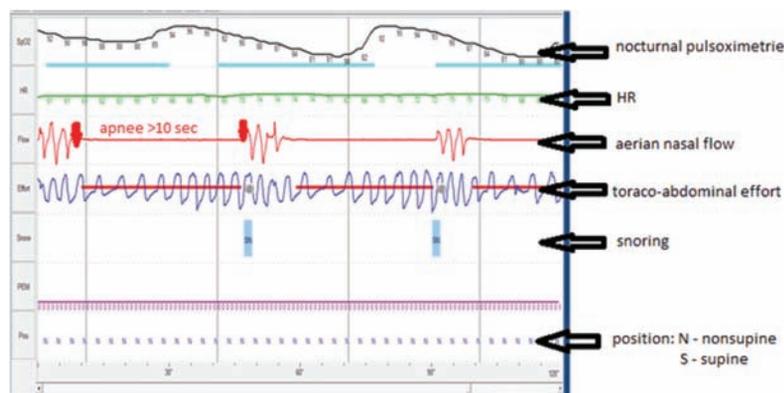


Figure 6. Nocturnal ventilatory polygraphy showing obstructive sleep apnoea with no aeriian nasal flow but movements toraco-abdominal.

the clinical data. The nocturnal ventilatory polygraphy recommended of the Department of Pneumology confirms the diagnosis of sleep apnoea syndrome (Figure 6) and establishes the indication of night ventilatory assistance with positive expiratory pressure<sup>3</sup>.

The diagnosis at this time is:

1. Essential 2<sup>nd</sup> degree hypertension, high added risk (masked hypertension)
2. Sleep apnoea syndrome
3. 1<sup>st</sup> Degree Obesity

The therapeutic recommendations implies first, hygienic-dietary measures, significant change in lifestyle including non-smoking, weight loss, physical exercise, reduced alcohol consumption. Taking into account that lifestyle change is extremely difficult to obtain and requires a perfect psychological balance and family support, his wife was called to understand the necessity of lifestyle changing and the importance of the family support. Psychological counselling sessions was also started<sup>4,5</sup>.

Hypertension medical treatment includes at first angiotensin receptor blockers (ARBs) given the secondary organ damage (micro albuminuria and left ventricular hypertrophy). Knowing that in patients with high blood pressure values and associated risk factors it is difficult to achieve blood pressure control with mono-

therapy, we preferred a combination with a calcium channel blocker, based on the results of Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT), ABPM subgroup considering its role in controlling night blood pressure<sup>6</sup>. Considering the patient's psychological profile and also his tension profile we preferred a fixed combination administered in small doses during the evening.

Regarding the sleep apnoea syndrome, it was initiated a night ventilatory assistance at home with positive expiratory pressure as recommended by the Department of Pneumology, but unfortunately not tolerated by the patient in spite of the repeated adaptation of the parameters.

As an additional advice we underline the effort of discouraging the obsessive self-blood pressure measurement at home, pointing out the doubts concerning the quality of the equipment used, and especially the patient's anxiety that can lead to the self-modifying of the treatment.

The patient's evolution was good, he adopted the hygienic-dietary measures and therapy at a satisfactory level, the awareness of the family support and the psychological counselling were very important given the family history. ABPM/24h control performed 3 months after the treatment started reveals a tension profile also non-dipper, but with a noticeably improved night profile (Figure 7).

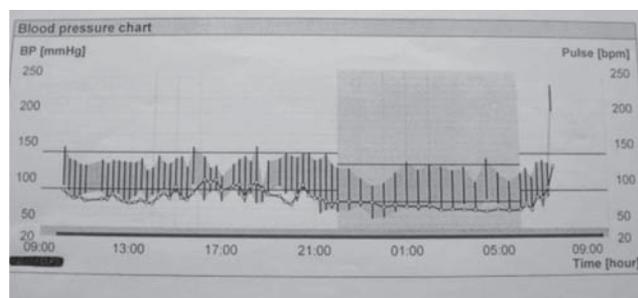


Figure 7. The blood pressure graphic/24h after 3 months of treatment.

## DISCUSSION

Masked hypertension is a relatively new subcategory of hypertension, diagnosed more and more often because both of stress increasing and of the access to modern methods of diagnosis. It should be considered every time we are in front of unexplained LVH. This form of hypertension is usually diagnosed late and therefore

the target organ is more severe damaged. Ambulatory BP monitoring is the only solution for these patients to confirm the suspected diagnosis, being necessary for initiating therapy and improved prognosis<sup>7</sup>. Sleep apnea may also be envisaged at patients with non-dipper profile tension and smokers patients to whom history can support targeted clinical vigilance.

**Conflict of interest:** none declared.

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