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Case Report

Resistant Hypertension: Don't Forget Subclinical Hypothyroidism

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Abstract

Subclinical hypothyroidism (SCH) is the most common thyroid dysfunction with a prevalence of 10% but remains under diagnosed and often neglected in primary care.

Among all specific cardiovascular manifestations associated to SCH, hypertension remains unusual and too controversial. Severe hypertension due to SCH is exceptional. It can be the first manifestation of the thyroid dysfunction representing a real diagnostic and therapeutic challenge for clinicians.

We report an original observation of severe and resistant hypertension revealing SCH in 38-year-old Tunisian man.

Keywords: *Hypertension; Subclinical Hypothyroidism; Cardiovascular Involvement; Hypothyroidism; Resistant Hypertension*

Introduction

Subclinical hypothyroidism (SCH) is the most common thyroid dysfunction; its prevalence exceeds 10% in the majority of studies and populations [1]. This prevalence can reach up to 20% in subjects over the age of 60, especially women [2,3].

The specific cardiovascular manifestations revealing SCH remain exceptional and little known, particularly in emergency situations [4,5]. Severe hypertension associated with this thyroid dysfunction remains unusual and too controversial [1,6,7].

Resistant hypertension is defined by blood pressure that remains above target despite treatment with three or more antihypertensive drugs, including a diuretic. Its prevalence is estimated at 10 - 20% of hypertensive patients and represents a real diagnostic and therapeutic challenge for clinicians [8,9].

We report the original observation of severe and resistant hypertension revealing SCH.

Case Report

38-year-old Tunisian man, followed for primary hypertension for five years, without degenerative complications, and was well balanced under acebutolol (400 mg/d) and furosemide (60 mg/d). For several consultations, his family doctor noted high systolic and diastolic blood pressures requiring therapeutic adjustment with candesartan cilexetil (16 mg/d), nicardipine (100 mg/day) and furosemide (60 mg/day) but without improvement. His blood pressure persisted high (160/90 mmHg) leading his doctor to refer him to our consultation.

The somatic examination was without significant anomalies. Blood pressure measurements at our outpatient clinics as well as an ambulatory blood pressure profile confirmed the diagnosis of resistant hypertension. No excess weight has been noted (body mass index at 22 kg/m^2) or consumption of toxic substances, alcohol abuse, excessive salt consumption, or self-medication that can induce iatrogenic hypertension.

Subsequent investigations concluded to isolated SCH: TSH at 6.25 µIU/ml and FT4 at 10 pmol/l. Cervical ultrasound was normal and anti-thyroid antibodies (anti-thyroperoxidase and anti-thyroglobulin) were negative.

The other explorations were without abnormalities: creatinine, uric acid, plasma ionogram, calcemia, fasting glucose, postprandial glycemia, lipid parameters, electrocardiogram, chest X-ray, cardiac ultrasound, renal Doppler ultrasound, and chest angio-CT.

The patient was treated with Levothyroxine in progressive doses up to 75 µg/d normalizing his TSH. The outcome was favorable with gradual stabilization of the systolic and diastolic blood pressures as the TSH normalized. After a month we were able to stop the nicardipine and the blood pressures remained at the objectives under combined dual therapy type candesartan cilexetil 8 mg/hydrochlorothiazide 12,5 mg/d.

Discussion

The cause and effect relationship between SCH and high blood pressure remains too controversial even in the most recent publications [6,10-12]. In fact, for some authors SCH is an indisputable cause of secondary hypertension, both systolic and diastolic [1,6]. This imputability seems clearer in late-onset hypertension (*de Novo* hypertension in subjects aged 65 and over) [7].

Piantanida E., *et al*. in a pilot study had shown a significantly increased prevalence of arterial hypertension in newly diagnosed hypothyroid patients, both those with overt and subclinical forms: 11.5% and 10.5% respectively versus only 8% in euthyroid controls, p = 0.003). Similarly, the prevalence of masked hypertension was also significantly increased in the two subgroups of hypothyroid patients compared to controls: 15.4% and 26.3% respectively versus only 10%, p = 0.05 [12]. In this study, thyroid hormone replacement therapy significantly lowered blood pressures [12].

During SCH, the rise in blood pressures mainly concerns diastolic pressure with a particular frequency of nocturnal hypertension and a "non-dipper" pattern [13]. This pathological status can be restored after hormone replacement therapy [13,14].

According to other authors, there is no cause and effect link between SCH and hypertension [10,15] and the increase in arterial pressures is only marginal and minimal during this thyroid dysfunction [15].

However, it should be noted that cases of hypertension, sometimes severe and even malignant, indicative of hypothyroidism have been reported [4,16]. This complication remains largely underestimated in the emergency departments (ED) since in Chen YJ., *et al.* series of unrecognized primary hypothyroidism in ED, high blood pressure revealing hypothyroidism was the reason for emergency visits in 14% of cases [4]. Similarly, in Dey A., *et al*. series, 16% of patients with SCH had stage 1 hypertension and 4% had stage 2 hypertension [17].

Conclusion

SCH is currently considered a possible cause of elevated blood pressures, and hormone replacement therapy can prevent and treat, if installed, hypertension in patients with SCH. Hormone replacement during SCH allows a clear reduction or even normalization of blood pressures levels.

A TSH test seems useful in front of any blood pressure anomaly, even if the patient does not have clinical signs/symptoms of hypothyroidism.

Conflicts of Interest

None.

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