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# GIANT HYPERFUNCTIONING THYROID GOITER CAUSING PULMONARY HYPERTENSION: CLINICAL CASE

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## ABSTRACT

The literature is full of information on the important cellular and hemodynamic effects of thyroid hormones and their important role in function and cardiac structure. Changes follow both hyperthyroidism and hypothyroidism in myocardial contractility, myocardial oxygen consumption, cardiac output, systemic or pulmonary vascular resistance. Due to the high frequency of hyperthyroidism, it is crucial to recognize its cardiovascular complications as a consequence of the high degree of mortality and morbidity that these complications present. Among the thyroid disorders that are associated with pulmonary hypertension are mainly forms of hyperthyroidism. The majority of patients with pulmonary arterial hypertension are elderly with toxic thyroid goiter. Thyroid hormone control should be considered in all patients with pulmonary hypertension. We present a case with a giant hyperfunctioning thyroid goiter which was considered as the cause of pulmonary arterial hypertension, which was resolved without any specific treatment for it, after thyroid treatment.

**KEYWORDS:** Hyperthyroidism, Pulmonary arterial hypertension, Goiter.

## INTRODUCTION

Both hyperthyroidism and hypothyroidism are followed by changes in myocardial contractility, myocardial oxygen consumption, cardiac output, systemic or pulmonary vascular resistance. Most cardiovascular changes are reversible by properly diagnosing and treating thyroid disorder.[1] Among the thyroid disorders that are associated with pulmonary hypertension mainly forms are those with hyperthyroidism. The majority of patients with pulmonary arterial hypertension (PH) are elderly with toxic thyroid goiter.[2,3] Recent findings suggest a direct effect of thyroid hormones on the vascular system through three possible mechanisms: 1) pulmonary vasoconstriction and a decrease in pulmonary vascular compliance as well as an increase in vascular resistance as a consequence of increased sensitivity to catecholamines; 2) significant increase in the metabolism of pulmonary vasodilating substances (prostacyclin, nitric oxide); 3) decrease in the metabolism of pulmonary vasodilating substances (endothelin 1, thromboxane and serotonin) (4). Thyroid hormone control should be considered in all patients with pulmonary hypertension.

## CASE PRESENTATION

We present a case of 78 years old female with a giant Multinodular Goiter (MNG) with a rapid increase in size within 10 years, with atrial fibrillation, palpitations, breathlessness on exertion and dyspnea. She is diagnosed with toxic nodular goiter since 40 years old, not regularly treated. In the physical examination, a thyroid giant goiter was observed (Fig 1). Her blood pressure was 115/70 mmHg, her heart rate was 116 beats per minute, and her oxygen saturation level was 93% without oxygen therapy. Both lungs were clear on auscultation and a cardiac examination shows normal findings. There was no edema in her legs, and except a diffuse goiter, the rest of the examination was normal.

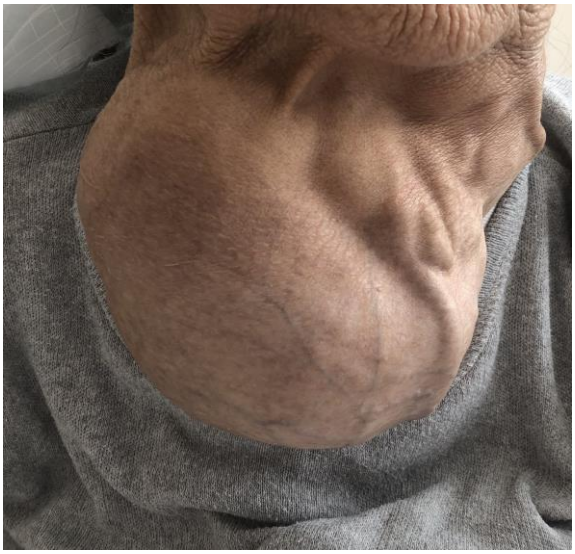

**Figure 1: Photo of patient's goiter.**

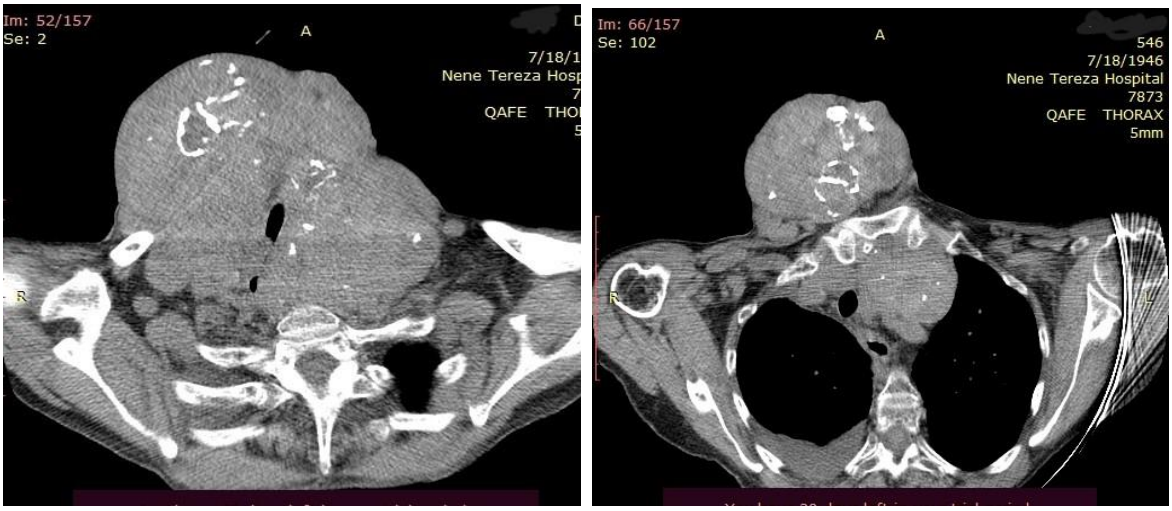


**Figure 2 (a,b): CT scan of thyroid gland.**

Her electrocardiography showed atrial fibrillation with a frequency of 116/min, without ST segment abnormalities. The patient's hemogram appeared normal, as well as the hepatic and renal balance and inflammation indices (PCR, ERS,). Computed tomography scan of the neck shows a gross enlargement of thyroid gland. (Fig. 2 a, b) Echocardiographic examination showed a severely dilated (basal diameter 50 mm) and hypokinetic right ventricle (TAPSE 10 mm) with severe pulmonary hypertension (PAP >80 mmHg), dilated left atrium and a nondilated left ventricle with preserved systolic ejection fraction. The D-dimer level was normal and a CT pulmonary angiogram showed no evidence of pulmonary embolism. A PH diagnosis was established with unknown cause. After that thyroid function tests showed hyperthyroidism (Table 1). The fine needle aspiration cytology suggested MNG with adenomatous nodules and toxic changes. The patient was started therapy with Methimazole 20 mg/day combined with diuretic and enalapril. The patient was followed up for a period of 12 months. After 4 months of therapy, thyroid hormones were normalized, while the improvement of pulmonary hypertension and resolution of right ventricular failure resolved completely after 8 months of the beginning of therapy. After establishing the euthyroid status, the patient was recommended for surgical treatment of the thyroid gland, a procedure that patient refused. The patient was followed periodically by an endocrinologist and a cardiologist.

**Table 1: Clinical follow-up of the patient.**

| Variable | Reference range | At presentation | After 2m | After 4 m | After 6 m | After 8 m | After 10 m | After 12 m |
|---|---|---|---|---|---|---|---|---|
| **Thyroid tests** | | | | | | | | |
| FT3 (pg/ml) | 2.6-5.1 | 5.2 | 5 | 4.7 | 4 | 3.8 | 3.1 | 3.2 |
| FT4 (ng/dl) | 1.0-1.81 | 3.0 | 2 | 1.5 | 1.3 | 1.5 | 1.4 | 1.7 |
| TSH (mlU/mL) | 0.27-4.2 | 0.008 | 0.01 | 0.2 | 1.5 | 1.9 | 1.8 | 2.1 |
| Medicamentation | | | | | | | | |
| Methimazole (mg/day) | 20 | 20 | 20 | 15 | 10 | 10 | 5 | 5 |
| Echocardiography | | | | | | | | |
| Pulmonary artery systolic pressure (mmHg) | 15-30 | 80 | 55 | 45 | 40 | 35 | 30 | 30 |

*FT3-Free T3; FT4-Free T4; TSH- Thyroid stimulation hormone*

## DISCUSSION

According to the latest ESC/ERS guidelines, pulmonary hypertension is divided into 5 groups according to etiology and physiopathological mechanism: Group 1-pulmonary arterial hypertension; Group 2-PH due to left heart disease; Group 3-PH due to lung disease or hypoxia; Group 4-chronic thromboembolic PH); and Group 5-PH due to unclear multifactorial mechanisms.[5] In our case it was considered PAH group 5 as a result of metabolic disorders, since it is difficult for PAH group 1 to pass without specific treatment,[2] while Group 2, 3, 4 were excluded even though detailed invasive examinations were not performed which measure the pulmonary vascular resistances or pulmonary capillary wedge pressure. The prevalence of PH is 35% to 47% in patients with hyperthyroidism,[6] so periodic examination of thyroid tests once a year or 2 years is recommended for patients with PH.[7] It remains doubtful whether the connection between PH and thyroid diseases is a coincidental finding or its cause, regardless of various studies where the effects of thyroid hormones (Hyper and hypothyroidism) were reported as the cause of the

increase in pulmonary arterial pressure.[6] The exact mechanism has not been fully investigated and may be multifactorial. Direct effects of thyroid hormones on pulmonary vascular proliferation, endothelial dysfunction mediated by autoimmunity, as well as chronotropic effects of thyroid hormones on the cardiovascular system, constitute the main proposed mechanisms.[8] The echocardiographic assessment of women with hyperthyroidism showed increased cardiac output and pulmonary vascular resistance, as in our case.[9] In patients with hyperthyroidism, there is a positive correlation between the estimated systolic pulmonary artery pressure with the levels of free thyroxine and the duration of hyperthyroidism.[10] In our patient, we have a decrease in the estimated systolic arterial pressure due to the decrease in free thyroxine levels. From the data that were extracted from a prospective study in which 17 patients with PH and hyperthyroidism were included, it resulted that all patients except one normalized the systolic pressures in the pulmonary artery for at least 9 months after the installation of a stable euthyroid status.[11] Similarly, in our patient, a decrease in pulmonary systolic arterial pressure, evaluated by echocardiography, as well as a significant improvement in dyspnea after the start of hyperthyroidism treatment, which were finally normalized about a month before the establishment of euthyroid status, were found.

## CONCLUSIONS

We present a case of PH as a consequence of giant hyper functioning thyroid goiter, which was successfully treated with thyroid therapy. Our clinical case highlights the importance of thyroid disease as one of the causes of PH, which can be best managed with proper treatment of thyroid disease.

### Conflicts of interest

No conflict of interest.

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