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Case Report

Right ventricular dysfunction secondary to hyperthyroidism with total reversal after thyroidectomy: case report

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Abstract: Thyrotoxicosis is a condition caused by an increase in thyroid hormone, commonly associated with Graves' disease and toxic multinodular goiter. Hyperthyroidism is known to increase the risk of pulmonary hypertension and right heart failure, although the underlying mechanisms are still unclear. The identification of these conditions is crucial, as they are potentially reversible with the achievement of euthyroidism. This case report describes a 40-year-old woman with right ventricular dysfunction and pulmonary hypertension due to hyperthyroidism, which was successfully treated with total thyroidectomy. The case highlights the importance of considering thyrotoxicosis as a potential cause of pulmonary hypertension and the need to rule out other underlying causes. Echocardiography is a valuable tool in assessing right ventricular dysfunction, and achieving euthyroidism can result in the reversal of structural and functional cardiac alterations.

Keywords: Pulmonary hypertension; Heart failure; Thyrotoxicosis; Hyperthyroidism.

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1. Introduction

Thyrotoxicosis is a clinical condition resulting from the inappropriate increase in the action of thyroid hormone in tissues, usually due to Graves' disease, toxic multinodular goiter, and toxic adenoma [1, 2]. Patients with untreated hyperthyroidism, regardless of coexisting diseases and by mechanisms that are still unclear, have increased risk of pulmonary hypertension and right heart failure [2–5]. The identification of these conditions due to thyrotoxicosis is relevant because structural and functional cardiac alterations are potentially reversible with the achievement of euthyroidism and have a good prognosis [2–6].

We report the case of a 40-year-old woman with right ventricular dysfunction and pulmonary hypertension secondary to hyperthyroidism with reversal after achieving euthyroidism with total thyroidectomy.

2. Case Report

A 40-year-old woman was admitted to the internal medicine ward because of a 3-week history of palpitations, progressive edema of the lower limbs and dyspnea on exertion. Her past medical history was significant only for Graves' disease diagnosed 5 years earlier treated with 20 mg daily methimazole and optimized atenolol 100 mg/day for 4 years, followed by radioiodine therapy. After radioiodine therapy, she used 10 mg daily methimazole with normalization of the thyroid profile, but treatment was interrupted about 1 year prior to this admission, coinciding with the new coronavirus pandemic.

She had no other comorbid conditions, no history of smoking and/or drug use, and no personal history of novel coronavirus (COVID-19) infection. No family history of heart disease or thyroid disease. On admission, the patient was tachycardic, afebrile, with normal blood pressure and oxygen saturation. On physical examination, she presented with diffuse enlargement of the thyroid gland (figure 1) with palpable thrill, exophthalmos, Plummer's nails, increased jugular venous pressure, left lower parasternal holosystolic murmur, unchanged pulmonary auscultation, and symmetrical edema of the lower limbs.

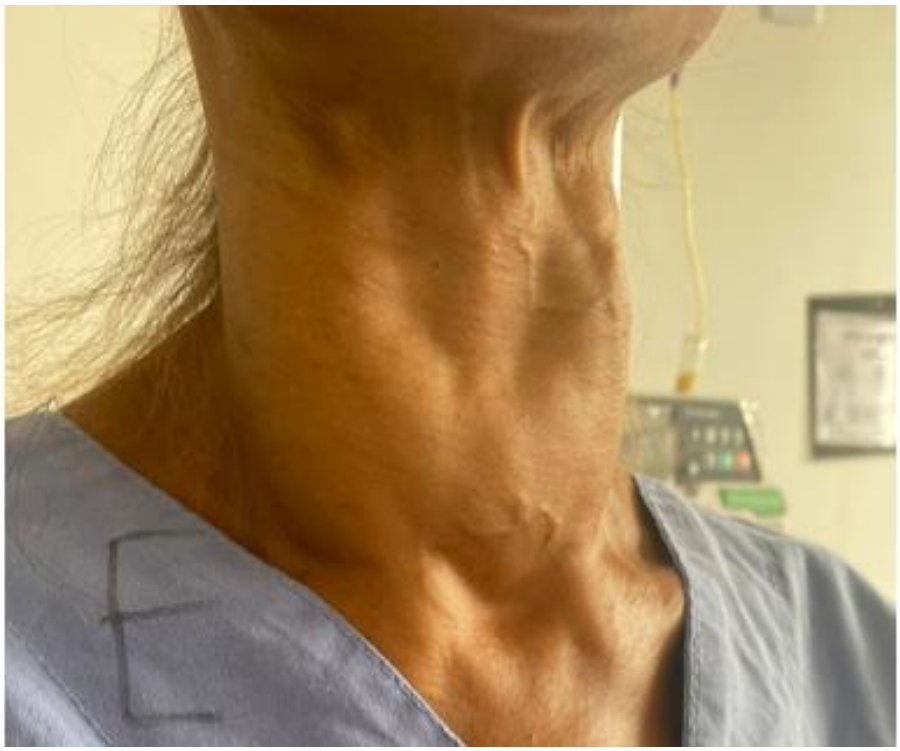

Figure 1: Diffuse enlargement of the thyroid gland on hospital admission.

The electrocardiogram showed sinus rhythm with alterations compatible with right ventricular overload and 1st degree atrioventricular block, while the chest X-ray revealed cardiomegaly at the expense of the right chambers. The 24-hour electrocardiographic monitoring showed an average heart rate of 88 beats por minute and 10 isolated supraventricular extrasystoles. The transthoracic echocardiogram showed normal systolic function (ejection fraction 58%), moderate enlargement of the atria and slight enlargement of the ventricles, mild mitral regurgitation, reduced right ventricular function, significant tricuspid regurgitation, and moderate pulmonary artery systolic hypertension with pulmonary artery systolic pressure 48 mmHg and tricuspid annular plane systolic excursion was 19 mm.

Laboratory tests documented significantly low thyroid-stimulating hormone (TSH) along with elevated free thyroxine. There was also a slight increase in N-terminal b-type natriuretic peptide (NT-proBNP). D-dimer and arterial blood gases were normal. Testing for COVID-19, viral serologies, serology for schistosomiasis and rheumatological tests were negative. Doppler ultrasonography of both lower limbs and contrast-enhanced computed tomography of the chest and abdomen ruled out deep venous thrombosis, pulmonary embolism, indirect signs of portal hypertension, and parenchymal abnormalities. Pulmonary function tests were negative for lung disease. Thyroid ultrasonography confirmed enlargement and hypervascularization of the thyroid gland, in addition to the presence of sparse nodular formations.

These findings were consistent with Graves' disease thyrotoxicosis with secondary pulmonary hypertension and isolated right ventricular heart failure. After evaluation of endocrinology, cardiology and head and neck surgery, total thyroidectomy was indicated. A total thyroidectomy was performed with preservation of bilateral recurrent laryngeal nerves and parathyroid glands, the surgery was performed without complications (Figure 2) with postoperative care in the intensive care unit. Serial calcium measurements were taken, and calcium replacement was administered intravenously, later transitioning to oral calcium (500 mg every 8 hours) along with vitamin D. Dexamethasone (4 mg every 8 hours) and levothyroxine (50 mcg) were initiated. Atenolol was reintroduced. The patitent did not developed dysphonia in the postoperative care, evaluated through speech therapy follow-up. The anatomopathological examination revealed multifocal lymphocytic infiltration.

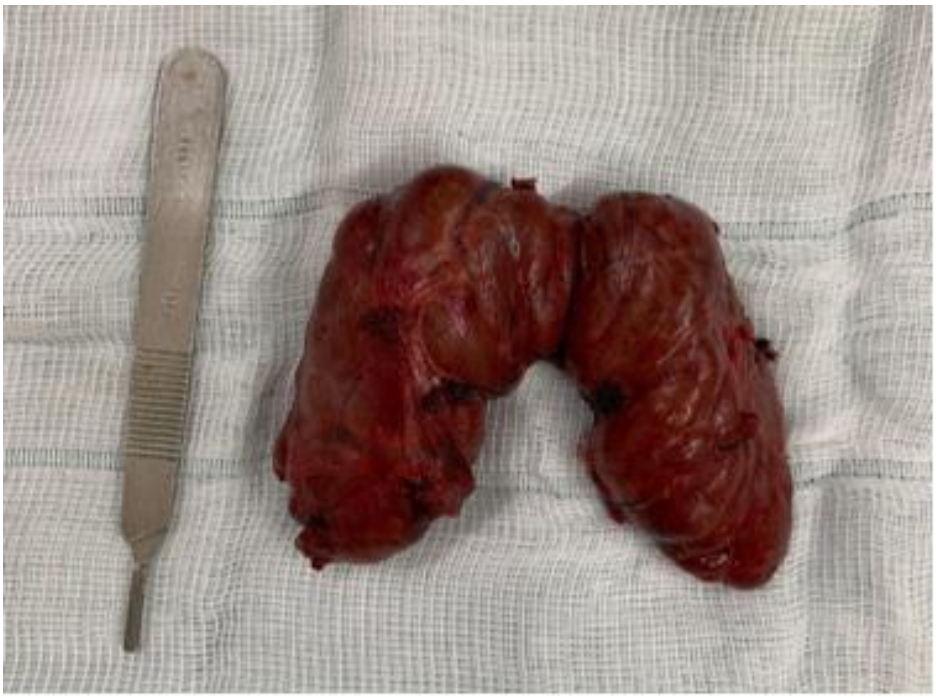

Figure 2: Total thyroidectomy product.

After 5 months of surgery, the echocardiogram and electrocardiogram were normal. At the 12-month follow-up, the patient remained without cardiovascular complaints (NYHA functional class I) or dysphonia, with normal calcium levels, and levothyroxine was adjusted to 100 mcg daily, with posterior laboratory results showing TSH levels of 2.11 and free T4 levels of 0.9. The patient continues to be monitored quarterly at the endocrinology outpatient clinic, maintaining stable TSH and free T4 levels, with no further need for adjustments in the levothyroxine dose.

3. Discussion

This case demonstrates that thyrotoxicosis can induce pulmonary hypertension and right heart failure independent of left heart disease. The most common cardiac manifestations include dyspnea and edema of the lower limbs, in addition to the presence of thyrotoxic symptoms [3–6]. The finding of clinically evident right heart failure is common.[6] Cardiovascular examination may reveal a systolic murmur of tricuspid regurgitation and increased jugular venous pressure. Atrial fibrillation may be present [1–3, 6]. The investigation must exclude classic causes of right ventricular dysfunction and pulmonary hypertension, which can be individualized based on the clinical analyses, personal and family

history [2–4, 6] It is critical to provide a resource-rational approach during the detection, prevention, and treatment of this condition [3, 4, 6].

Echocardiography is an important tool to characterize right ventricular dysfunction, including dilated right atrium or right ventricular chambers and tricuspid regurgitation. Left ventricular function, in this scenario, is preserved [2–6]. The echocardiographic measurement of pulmonary hypertension usually reveals systolic pulmonary artery pressure (PAP) >30 mmHg, mean PAP >25 mmHg or right ventricular systolic pressure >40 mmHg [2–5, 7] Not all patients undergo right heart catheterization to confirm pulmonary hypertension [2, 3, 6]. Some patients have been treated with specific therapy for pulmonary hypertension [6]. Proposed mechanisms include autoimmune endothelial dysfunction, endothelial damage from high cardiac output, alterations in the metabolism of vasodilators or vasoconstrictors, and sympathetic pulmonary vasoconstriction. Changes in the pulmonary vasculature contribute to hemodynamic overload of the right ventricle [2–7].

When evaluating patients with suspected Pulmonary Hypertension (PH), it is important to have a good understanding of etiologies and classification. Patients with PH are classified into five groups based on etiology and mechanism. Pulmonary arterial hypertension and right heart failure can coexist. Patients in group 1 are considered to have Pulmonary Arterial Hypertension (PAH), which has various causes (e.g., hereditary factors, medications, connective tissue diseases), whereas patients in group 2 (due to left heart diseases), group 3 (due to chronic lung disorders and hypoxemia), group 4 (due to pulmonary artery obstructions), and group 5 (due to unidentified or multifactorial mechanisms) are considered to have PH [8].

PH can also be classified as pre-capillary or post-capillary. Pre-capillary PH occurs due to a primary elevation of pressure solely in the pulmonary artery system (e.g., PAH), while post-capillary PH occurs due to pressure elevations in the pulmonary venous and pulmonary capillary systems (pulmonary venous hypertension, e.g., group 2). In practice, some patients exhibit mixed pre- and post-capillary characteristics [8]. In our case, we aimed to exclude relevant etiologies in accordance with the clinical history. In Group 1, the patient had no family history, hadn't used drugs or toxins that induce PAH, the rheumatological panel and clinical evaluation ruled out connective tissue disease, serology for HIV and schistosomiasis were negative, and abdominal ultrasound showed no changes consistent with portal hypertension. In Group 2, PH occurs due to left heart disease, accounting for nearly 70% of cases [8]. Here, the echocardiogram, apart from identifying PH, needs to assess the left chambers, valvular diseases, systolic and diastolic function of the left ventricle, which in our case excluded diseases from this group. In Group 3, spirometry and chest tomography ruled out the possibility of lung disease/hypoxia, such as chronic obstructive pulmonary disease, the second main cause of PH, and restrictive diseases. Another significant cause comes from Group 4, pulmonary artery obstruction due to chronic thromboembolism, evaluated by thoracic CT angiography. In our case, despite a low d-dimer level, we chose to perform the test to rule out this possibility. Lastly, Group 5 remained, which can encompass thyroid diseases like our case.

Graves' Disease can be established based on clinical parameters. If a patient presents with persistent symptoms for several months, along with any sign of orbitopathy and a diffuse enlargement of the thyroid gland, the diagnosis can be confirmed. The presence of nodules can be ruled out through ultrasound. Thyroid overactivity is diagnosed by measuring serum levels of TSH and free thyroxine. When the diagnosis cannot be confirmed solely based on clinical information and the results of TSH and free thyroxine, the detection of autoantibodies to the TSH receptor in the serum can confirm the diagnosis [9].

Once the diagnosis is established, appropriate treatment decisions can be made. If the autoantibody test yields a negative result, it is recommended to perform a radioiodine uptake and thyroid scintigraphy to confirm the diagnosis [9]. Euthyroidism can be achieved through antithyroid therapy, radioactive iodine, or thyroidectomy. The choice of treatment depends on the severity of thyrotoxicosis and patient preferences [2–4,6].

Patients had immediate resolution of symptoms and signs of right ventricular dysfunction after starting treatment for thyrotoxicosis. Echocardiography after restoration of euthyroidism usually shows resolution of right ventricular dysfunction, an important feature of this condition. Time required to normalize pulmonary artery pressure ang right ventricle function may vary according to disease severity and individual response to treatment [2–7].

4. Conclusion

Right ventricular dysfunction is an important complication in patients with untreated hyperthyroidism, and management includes antithyroid therapy, radioactive iodine, or thyroidectomy. The reestablishment of euthyroidism can reverse the disease.

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Research Ethics Committee Approval: We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

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Conflicts of Interest: None.

Supplementary Materials: None.

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