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Cardiothyreosis: Another Side of the Heart Failure

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Abstract **Case Report**

Thyrotoxic cardiac disease represent one of the multiple hormonal disorders that can affect the heart, understanding the physiologic effects of the thyroid hormone on the cardiovascular system is the key to suspect the condition in the first place and manage it secondly, the clinical presentation can be very tricky especially in naive patients. We are going to expose a rare case of cardiothyreosis resulting in congestive heart failure.

Keywords: Heart failure, cardiothyriosis, atrial fibrillation, hyerthyroidism.

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INTRODUCTION

Thyroid hormones regulate the basal metabolic rate in almost every tissue and organ in the body and especially the cardio-vascular system which makes the latter a direct target of any disorder that can affect the normal range of these hormones. Hyperthyroidism, which is a very common condition, can lead to heart failure in extreme cases. On the grounds of this, hyperthyroidism is considered as an etiology of cardiomyopathy in the WHO classification since 1995 [13]. We report in this observation a case of old women who was admitted for acute dyspnea presentation with history of neglected hyperthyroidism, we also report by this way the latest available literature reviews.

OBSERVATION

C.F a 71-year-old female, consulted our cardiology department for dyspnea and leg swelling. C.F was under treatment for hyperthyroidism supposedly controlled. She had no history of cardiac disease or known cardio-vascular risk factors. First examination found a very dyspneic patient (NYHA VI), afebrile, she did not report any kind of chest pain, BP= 90/60 mmhg, 110 Bpm, T= 37.5°, blood oxygen was 89-92% under 4L/min. Pulmonary auscultation found a reduction in vesicular sounds of the right lung, fine crackles at the base. ECG found an accelerated atrial fibrillation at 110 bpm with no repolarization anomalies. Chest radiography confirmed the presence of right pleural effusion. Trans-echocardiography objectified a dilated left ventricle, with an EF at 30%, and sPAP at 56mmHg.

With no etiological orientation between our hands, we proceeded to routine investigation tools to assess the origin of the cardiomyopathy, knowing our patient hormonal history, we measured TSH and surprisingly it was very low, the patient was put under antithyroid agents in the past but she interrupted them with no medical consultation. In the absence of other conditions that may explain the condition, we put our patient on traditional heart failure medical treatment in addition to Benzylthiouracile (150mg per 24h for 4 weeks followed by a dose reduction according to lab tests). The evolution was spectacular, a significantly clinical amelioration was observed, with an EF at 45% by the eighth week.

DISCUSSION

Cardiothyreosis (CT), or thyrotoxic heart disease, is defined as an association of hyperthyroidism with one or more heart complications [1]. Thyrotoxicosis results from excess thyroid hormone, whether endogenously produced, as in Graves' disease, multinodular goiter, and thyroiditis, or from an exogenous source, as in overtreatment with l-thyroxine therapy. It is a not well-defined disease entity given the lack of diagnostic criteria regarding the type of cardiac complication, that can include a wide range of manifestations, such as: rhythmic troubles with atrial fibrillation and sinus tachycardia in the lead, congestive heart failure and coronary insufficiency [2, 3]. Some authors also include atrioventricular blocks, left ventricular hypertrophy, diastolic dysfunction and more recently pulmonary hypertension [4]. CT frequency

varies widely according to the type of population studied and the used diagnostic criteria. It is associated with higher morbidity and mortality than the other forms of hyperthyroidism. The hemodynamic effects of hyperthyroidism consist of decreased systemic vascular resistance and increased resting heart rate, left ventricular contractility, and blood volume mediated by the activation of renin-angiotensin system in response to the peripheral vasodilatation, leading to an enhanced systolic and diastolic cardiac functions with increased ejection fraction and cardiac output.

In contrast to a fall in mean systemic arterial pressure, pulmonary artery hypertension is increasingly being recognized in hyperthyroidism [5]. This finding may be the result of an increased pulmonary blood flow unaccompanied by the same decrease in pulmonary vascular resistance as occurs in the systemic circulation. Pulmonary artery hypertension may in turn result in an increased load on the right ventricle, leading to right ventricular dilatation and a rise in right atrial and central venous pressure. About 6% of thyrotoxic individuals develop symptoms of heart failure, which may seem in the beginning paradoxal to the increased cardiac function that we already explained, this has led to the suggestion that there is a "thyrotoxic cardiomyopathy" [6]. Data from Forfar *et al*., [7] support such an entity; they reported the inability of the hyperthyroid heart to increase the left ventricular ejection fraction, as normally occurs with exercise. In addition to the fluid retention and the expanded plasma volume behind the congestion. But less than 1% develop dilated cardiomyopathy with impaired left ventricular systolic function [8]. Many mechanisms underlie the development of "true" heart failure in this setting. In patients with known or suspected underlying cardiac disease, the imposition of an increased workload or myocardial oxygen demand can lead to cardiac ischemia and/or impaired cardiac performance [9, 10].

Such patients are often older and have a history of hypertension or atherosclerotic coronary artery disease. Hyperthyroidism can also unmask valvular heart disease and produce worsening mitral or tricuspid regurgitation. Rate-related cardiomyopathy is the consequence of chronic sinus tachycardia and atrial fibrillation and it characterized by low cardiac output. Factors associated with CT have been analyzed in a limited number of studies. An advanced age and a preexisting cardiac disease are the most known risk factors. Conflicting data exist with respect to other factors, such as male gender, thyrotoxicosis severity, duration of the thyrotoxic state and the etiology of hyperthyroidism. Our patient clinical presentation was a rare one, with symptoms of heart failure, decreased ejection fraction and increased systolic pulmonary arterial pressure on echocardiography, that represent as previously mentioned less than 1% of hyperthyroidic patients. She had no history of cardio-vascular events, no underlying structural or vascular disease, but still she gathered some of the CT risk factors, such as the age, duration and severity of hyperthyroidism.

Etiological orientation is also present as she has mentioned during interrogation the interruption of all sort of medication of hyperthyroidism with no prior medical consultation. The presence of AF was also a strong indicator and the most constant one after sinus tachycardia. Patients presenting with AF are the more frequently subject to develop symptoms of heart failure according to data [11, 12]. As for the treatment, the goal is to simultaneously restore thyroid hormone levels to normal, while also managing cardio-vascular manifestations, upon achieving a euthyroid state, symptoms will resolve, more immediate effect can be obtained by beta-blockers, but their introduction on the setting of a heart failure with reduced EF should be monitored closely. The complete resolution of symptoms and the amelioration cardiac performances under appropriate treatment is another argument in favor of our initial diagnosis.

CONCLUSION

Thyrotoxicosis is a condition with many documented cardiovascular side effects, symptoms can vary from the classic chronic sinus tachycardia to heart failure. The challenging case we presented was an extreme form of the disease where the presentation was marked by a heart failure with reduced ejection fraction. Early recognition thanks to the presence of multiple risk factors and the pre-existing condition, early management using specific and non-specific treatments can reverse the cardio-vascular manifestations.

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