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Case Report

Hashitoxicosis, About a Clinical Case

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Abstract

Hashitoxicosis in a pathological entity considered as the second cause in frequency after Graves Basedow's thyroiditis, characterized by a state of hypermetabolism due to the increase in thyroid hormones due to an inflammatory reaction to the thyroid gland.

We present the case of a 34-year-old female patient with cardiac comorbidities who presented to the terations in the tests of hormonal function compatible with hiperthyroidism, however, the determination of antibodies allowed to establish the diagnosis of Hashimoto's Disease.

This clinical case report is made due to the limited information on the knowledge of the mixed thyroid autoimmune effect in the same patient.

With the description of the case it is intended to open a range of more research of this type and thereby improve the quality of life of patients who have this pathology.

Keywords: Hashimoto; Autoimmune; Hormonal; Hiperthyroidism; Hashitoxicosis

Introduction

Hyperthyroidism is a state of hypermetabolism caused by increased thyroid hormones. The first two causes are autoimmune diseases, Graves-Basedow disease (EG) in the first place, occurring in 95% of cases and Hashitoxicosis, which ranks second in frequency [1].

Hashimoto's thyroiditis is an autoimmune thyroid disease that commonly causes hypothyroidism, although patients may have a transient thyrotoxic called "Hashitoxicosis", this phase is prior to the hypothyroidism that ensues, this entity could be due to an excessive release of thyroid hormone from a inflamed gland that usually lasts one to two months [2].

Epidemiologically it is mentioned that in astudy of the NHANES database that analyzed about 18,000 cases in the U.S. it was observed that non-Hispanic Afro-descendants had hyperthyroidism of autoimmune origin almost three times more than non-Hispanic whites [3].

As stipulated by Shahbaz. A., *et al*. "Long-term follow-up of a child with autoimmune thyroiditis and recurrent hyperthyroidism in the absence of TSH receptor antibodies", atypical cases especially in the pediatric population draw attention for the understanding of the pathology in question, describes that "very rarely, at the time of diagnosis, they may present with signs and symptoms of short-lived hyperthyroidism, which tend to resolve within a few weeks and are followed by a euthyroid or hypothyroid state" [2].

Hashimoto's tir oiditis (HT), considered a more common autoimmune disease, was described more than a century ago as a pronounced lymphoid goiter thatpredominantly affects women [4]. 95% of cases of hypothyroidism are acquired primarily and can initially manifest as subclinical hypothyroidism, where mild symptoms can be found, which indicates that the early diagnosis of subclinical hypothyroidismis of great importance, since, although no important clinical manifestations are found, it can be the beginning of serious problems of thyroid origin and lead to clinical hypothyroidism withimportant clinical sequences [5]. (o linfocitos T helper 1).

The etiology of Hashitoxicosis is diverse, it has been associated with treatment for viral entities; thus, interferon therapy has been postulated can cause the immune reaction Th-1 (o linfocitos T helper 1) and, therefore, activation of CD4 lymphocytes which causes the release of interleukin-2, interferon gamma and tumor necrosis factor causing a vigorousagitation of autoimmunity [6].

In addition, in the treatment of pathologies that have nothing to do with thyroid damage, the drug use of epoprostenol as part of the therapy to combat Pulmonary Hypertension was seen to be induced thyrotoxicosis in a study of 59 cases in Japan [8].

In consideration of cases of variable etiology can occur in female patients with evidence of biochemistry and symptomatology of Hashitoxicosis whose cause may be induced by stress, ending in subclinical hypothyroidism, subsequently recovering their thyroid function after a couple of months [9].

Description of the clinical case

Female patient of 34 years, with pathological history of pneumonia by COVID-19 in July of the 20 20, severe mitral insufficiency, severe pulmonary hypertension, in sufficiency cardiac diagnosed 1 month ago of etiology not specified, hiperthyroidism in the same period of time. He goes for clinical evolution of 5 months of previous evolution of progressive dyspnea, at the moment NYHA.

Functional class 3, symptoms accompanied by náuseas that leads to vomiting for several occasions, a picture that is exacerbated 3 days ago referred to our institution.

On physical examination, hemodynamically without requirement of vasoactive amines, disoriented, Glasgow 7/10, bradipsiquy, subcostal pull, perioral cyanosis, anasarca. By committed ventilatory mechanics, immediate connection to mechanical ventilatory assistance is decided.

Complementary studies are carried out, considering alterations of thyroid function previously reported in reference, the analyzes demonstrate discordance between the presence of positive antibodies for hypothyroidism and the presence of hormonal levels suggestive of hyperthyroid pathology. Given the suspicion of Hashitoxicosis, for the above, the physical examination did not reveal signs or symptoms characteristic of any of the most typical autoimmune pathologies of the thyroid gland such as Graves Basedow's disease or H ashimoto's thyroiditis so it is determined at that time as subclinical hiperthyroidism.

From the cardiological, patient with heart failure with severe systolic dysfunction, diffuse hypokinesia, severe mitral insufficiency and moderate tricuspid insufficiency. Thyroid pathology was considered as a cause of cardiac decompensation.

Discussion and Conclusion

We must emphasize that Hashitoxicosis is a pathological entity considered as the second cause in frequency after Graves Basedow's thyroiditis categorized by a state of hypermetabolism whose main actors are the increase in thyroid hormones due to an inflammatory response to the thyroid gland that initially manifests itself with symptoms of hyperthyroidism but after 2 to 3 months can evolve to Hypothyroidism of course associated with antibodies in around an autoimmune process [1].

Regarding the determination of such anti-TPO antibodies, it is worth mentioning that they are present in approximately 90% of patients with Hashimoto's thyroiditis, and anti-TG antibodies are positive in approximately 60% of patients with chronic thyroiditis [2].

When antiperoxidase and thyroglobulin antibodies are present, the symptomatology that predominates in the long term is that caused by the hypothyroidism that is triggered. That is why in the presence of alteration of TSH and T4 with findings of hyperthyroidism the determination of one of the two options in principle is difficult and therefore the determination of the mixed pathology with the name of Hashitoxicosis is given [1].

Regarding the determination of a correct differential between the pathologies mentioned and how it is determined that "Hashitoxicosis is distinguished from Graves' disease by the absence of thyroid-stimulating immunoglobulins (TSI) and by a decrease in the uptake of radioactive iodine" and therefore the biochemistry involved in it should make us think in the initial analysis the only manifestation of a process of hyperthyroidism alone or as in our case an association of the same with hypothyroidism of an autoimmune nature [9].

As for the treatment, Hashitoxicosis will trigger the final appearance of hypothyroidism, manifesting symptoms of it and therefore hormonal restitution with levothyroxine in a range of 1.6 to 1.8 µg per kg of body weight will help improve clinical manifestations [4].

Hashitoxicosis requires treatment with beta blockers during the active phase of thyrotoxicosis. In case the diagnosis of a transient thyrotoxic phase of autoimmune thyroiditis cannot be confirmed with certainty, i.e. if the possibility of Graves' disease is suspected, a short course of antithyroid drugs may be prescribed, while being monitored with caution with periodic tests to prevent the occurrence of hypothyroidism [6].

As therapeutic alternatives it is worth mentioning that the definitive tool to correct thyroid hormone deficiency in a patient who does not have a functional thyroid gland would be a thyroid transplant [4].

We can conclude from the evidence correlated with our clinical case that Hashitoxicosis is a thyroid pathology of great care, due to the clinical alterations, acute and chronic hemodynamics that may occur, so it is necessary to discern within our diagnosis as an entity between 2 extremes that can be hyper and hypothyroidism and that in the end the therapeutic alternative will be carried out fundamentally in the application of hormonal restitution in most cases, or in the surgical excision of the thyroid gland combined with thyroid restitution hormone therapy.

Hematology	
Leukocytes	9.23x13.3/ul
Hemoglobin	13.7 g/dl
Hematocrit	43.9%
Platelets	112 x 10^3/ul
Urea	45 mg/dl
Creatinine	0.8 mg/dl
Total Bilirubin	3.7 cm g/dl
Total Bilirubin	01.4 mg/dl
Immunology	
Anti-microsomal TPO	22.3 Ul/ml
Anti - thyroglobulin	406.70 UL/ml
Anti-bodies anti TSH receptors	50.35 UL/ml
Cytomegalovirus	0.050
Toxoplasma	No reactive
Thyroid-stimulation hormone TSH	0.010 UL/ml
Free thyroxine T4	16.21 UL/ml

Table 1

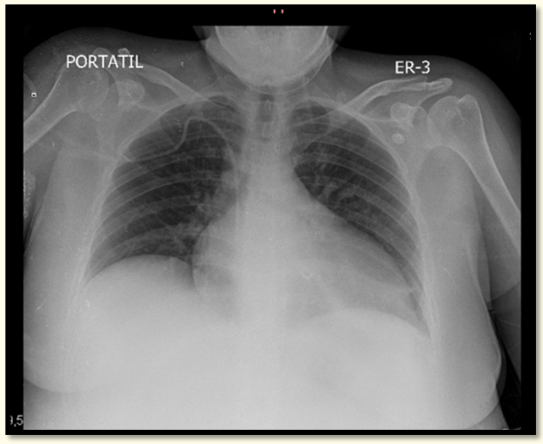

Figure 1: Cardiomegaly.

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