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Hashitoxicosis in children: A case report

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Abstract

Hashitoxicosis is a medical condition characterized by hypermetabolism that is thought to be an uncommon cause of hyperthyroidism in children. This phenomenon is due to excessive release of thyroid hormones in a patient previously diagnosed with Hashimoto's disease. Given the apparent rise in the number of patients with Hashimoto's thyroiditis and/or improvements in diagnosis, this may be more frequent than previously believed. The case study we report is an 11-year-old patient who was diagnosed with hyperthyroidism caused by Graves' disease based on clinical observations. Nevertheless, the initial investigations and the patient's subsequent development suggest Hashimoto's illness as the cause of the diagnosis.

Keywords: Hashimoto; Hyperthyroidism; Hashitotoxicosis.

Introduction

Hyperthyroidism is a state of hypermetabolism secondary to an increase in thyroid hormones. The two leading causes are autoimmune diseases, Graves-Basedow disease (MG) in first place (95%) and hashitoxicosis, which occupies second place in frequency [1]; This article aims to highlight the latter one.

Hashitoxicosis corresponds to the increased secretion of thyroid hormones caused by inflammation and destruction of thyroid cells. As thyroid cells die, previously synthesized and stored thyroid hormones are released into the bloodstream, this causing a hyperthyroid state. This phenomenon of inflammation and destruction occurs in response to antiperoxidases and antithyroglobulin characteristic of Hashimoto's disease first described by Hakaru Hashimoto in 1912 [2].

Children with Hashimoto's thyroiditis may present with euthyroid goiter, subclinical hypothyroidism, profound hypothyroidism with growth failure and delayed bone age, and, rarely, hashitoxicosis. The incidence and typical clinical course of Htx in pediatric patients have not been well characterized [3] it may be responsible for a delay in sexual maturation, although physical development is normal and skeletal growth can be accelerated [4].

Observation

Patient aged 11 years old boy, from a non-consanguineous marriage, without notable pathological ATCD who consulted the pediatric endocrinology department for hyperthyroidism with, clinical signs and symptoms of hyperthyroidism (tachycardia, distal tremors) an increased thyroid gland height without other signs on physical examination.

The results of the biological tests carried out report a TSH slowed to 0.03 Uui/ml; LT4: 2.92 (0.7-1.48); LT3: 12.08 (1.71-3.71); anti TPO: 246; TRAK: 1.25UI/l (negative<1.75)

Cervical ultrasound revealed an enlarged thyroid gland, pushing out the bilateral jugulo-carotid axes without however crossing the upper mediastinal orifice, measuring: isthmus: 4.7 mm; LD: 2.10x1.50x5.7 cm (9.35 ml); LG: 2.14x1.32x5.41 (8 ml), it has bumpy contours and a heterogeneous echo structure with diffuse hypoechogenicity predominating at the level of the apico-external regions, the limits with the healthy parenchyma creating a "candle spot" appearance. With a thickening and a hyperechogenic appearance of the interlobular septa, producing a reticulated "honeycomb" appearance. Presence of intense hypervascularization on Doppler of the thyroid parenchyma, diffuse and bilateral, creating an "inferno thyroid" appearance

with a pulsatile parenchyma and an increase in the caliber of the trunk of the thyroid arteries. On pulsed Doppler, we note the presence of a very high systolic velocity around 1 m/s, a choppy outline due to tremors and tachycardia, the presence of rapid diastolic flows and the entanglement of numerous spectra throughout the parenchyma. CC/ultrasound appearance in favor of Graves' disease.

On the basis of the above, the treatment consisted of the administration of synthetic anti-thyroid drugs (carbimazol 20 mg/day). 5 months later, the analyzes requested reported a TSH >100 Uui/ml, it was therefore decided to suspend the treatment. Then the introduction of L-thyroxine given the persistence of hypothyroidism. Currently the patient is on euthyroidism under regular clinical and biological monitoring every 6 months.

Discussion

Hashitoxicosis is a pathological entity that is thought to be an uncommon cause of hyperthyroidism in children, it is categorized by a state of hypermetabolism whose main actors are the increase in thyroid hormones due to an inflammatory response of the thyroid gland which initially manifests itself with symptoms. Hyperthyroidism can progress to hypothyroidism of course associated with antibodies around an autoimmune process [5].

Regarding the determination of these anti-TPO antibodies, it should be mentioned 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 [6].

Diagnosis of hashitoxicosis can be complicated, as the presenting features sometimes have significant overlap with Graves' disease. Therefore, in the presence of impaired TSH and T4 accompanied by hyperthyroidism, determining one of the two options is in principle difficult [5].

Data on the possible incidence of hashitoxicosis are scarce and observations are often published as case reports [7,8]. Wasniewska et al. Examined the outcomes of hashitoxicosis in 14 children [10]. The hyperthyroid phase was highly variable, always followed by complete resolution without relapse, which led to persistent euthyroidia or hypothyroidism. Four patients with more severe presentation received methimazole treatment and definitive resolution of hyperthyroidism was delayed.

Nabhan et al. [9] reviewed the medical records of children diagnosed with Hashimoto's thyroiditis between 1993 and 2002. Of 69 patients with autoimmune thyroiditis, eight were diagnosed with hashitoxicosis (11.69%). The duration of hyperthyroidism ranged from 31 to 168 days. Three patients became hypothyroid after a mean of 46±13.2 days and five patients became euthyroid after a mean of 112.8±59.8 days. Only one of the eight patients had been treated with methimazole; the others were treated with a beta-blocker or not at all.

The presentation in our patient was consistent with hashitoxicosis. It is usually treated symptomatically, but due to the significant signs and symptoms of hyperthyroidism, we started antithyroid medications in our patient. In subsequent follow-up visits, the patient gradually returned to baseline and correction of hyperthyroidism was achieved, and antithyroid treatment was gradually stopped.

The authors attempted to identify predisposing factors for the development of hashitoxicosis, such as gender, age, family history, thyroid hormone levels, anti-thyroid antibody titers, results of thyroid analysis and presenting characteristics. However, no risk factors were discovered.

There are no clear data indicating that its occurrence is a marker for more rapid progression to hypothyroidism in Hashimoto's thyroiditis.

Conclusion

Hashitoxicosis is a rare but significant cause of hyperthyroidism in children, with wide clinical variability. Diagnosis can be difficult because some features may be significantly similar to Graves' disease. To date no factors contributing to the development of hashitoxicosis have been found.

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