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Case Report

Marine-Lenhart Syndrome about a Case

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A B S T R A C T

The most common cause of hyperthyroidism is autoimmune (Graves Basedow disease). It usually presents with diffuse goiter, but between 25-30% have thyroid's nodules. Most of them are cold nodules on scintigraphy, therefore, hypo functioning and between 0.8 - 2.7% are hyper functioning1-5. The association of thyrotoxicosis due to Graves' disease (GBD) and toxic nodules is what is called Marine-Lenhart syndrome (MLS). It is rare and the prevalence ranges between 2.7 - 4.1%3,6.

It's characterized by the presentation of Graves' disease accompanied by hyper functioning's nodules with the following characteristics: a) thyroid scintigraphy performed with Tc-99m Pertechnetiate, which shows Graves' disease with one or more hyper functioning nodules; (b) hyper uptake in nodules after increase in TSH level due to radio-ablation; (c) return to nodal function after stimulation with endogenous or exogenous TSH; (d) benign histopathology. They present thyroid-stimulating antibodies. Although these characteristics are discussed7. This entity is more common in women, between 40 - 50 years old, with hyperthyroidism of several years of evolution and failed treatments with ATS.

Keywords: Hyperthyroidism; Graves basedow disease; Thyroid scintigraphy

Case Report

A 73-year-old woman, with no notable family history, a light smoker, who came for control of her thyroid disease (hyperthyroidism) after abandoning treatment for two years. From his history, diagnosis of GBD of 10 years of evolution, with grade III goiter, 3cm left thyroid nodule, clear limits, smooth surface, not painful. Bright eyes, conjunctival redness and chemosis in the eyes. On ultrasound, right thyroid lobe measuring 12 x 14 x 46 mm. We don't see nodules. Left lobe measuring 24 Isthmus without particularities. Scintigraphy with hyper enhancement nodule in the left lobe, with the rest of the parenchyma inhibited. He receives synthetic ant thyroid drugs (PTU for two years and methimazole for 4 more years). He refuses to receive iodine 131 at that time.

Two years after starting ant thyroid treatment, a repeat ultrasound showed a single nodule in the left lobe measuring 22 x 26 x 40mm. When eufunction is achieved, a fine-needle aspiration puncture of the left thyroid nodule is requested and a Bethesda II cytogram is obtained.

At the time of the consultation, he presented insomnia, palpitations, and had lost 10 kg of weight in recent months. The examination revealed grade III goiter, an irregular surface with a nodule in the left lobe of approximately 4 cm. Regular rhythm of 120 cycles/minute. Discrete exophthalmos, lacrimal gland hypertrophy, bilateral chemosis. Positive Moebius sign. From the laboratory TSH: 0.09 uU/l (0.27-4.7), T4l 1.9 ng/dl, T3l: 3.9 ng/dl (0.93-1.7). Thyroperoxidase antibodies (TPO) and thyrotropin-stimulating antibodies (TSI): positive.

Thyroid ultrasound: total volume of 26 cc, right lobe 18 x 19 x 42 mm and left lobe 24 x 27 x 48. Nodule 22 x 25 x 39 mm, peripheral vascularization with little central. No lymph nodes (**Figure 1)**.

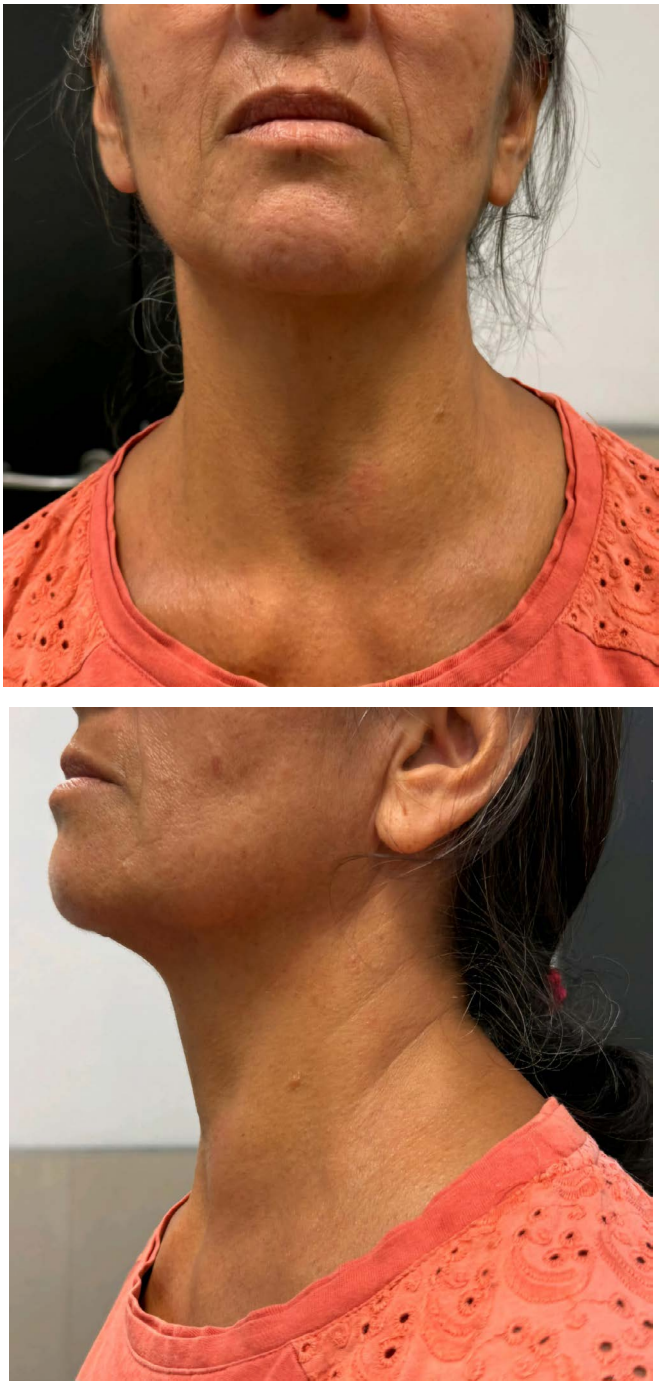

Figure 1. Front and profile photo showing the major thyroid nodule in the left thyroid lobe

Thyroid scintigraphy with CT-99: irregular hyper uptake, highlighting a cap-shaped area of hyper uptake, with a thyroid nodule in the left lobe with characteristics of multi-nodularity and functional blockage of the rest of the glandular parenchyma (**Figure 2**).

Orbital tomography: bilateral exophthalmos. Optic nerves and intraorbital, intra and extraconal fat without alterations. Increased thickness of both internal rectus muscles. Methimazole 10 mg/day and propranolol 20 mg every 12 hours were started. After 2 months he was euthyroid, prednisone 0.5 mg/kg/day was started for a month and a therapeutic dose of Iodine 131, 25 mCi was administered without complications. Two months later he developed hypothyroidism and was started to be replaced with levothyroxine.

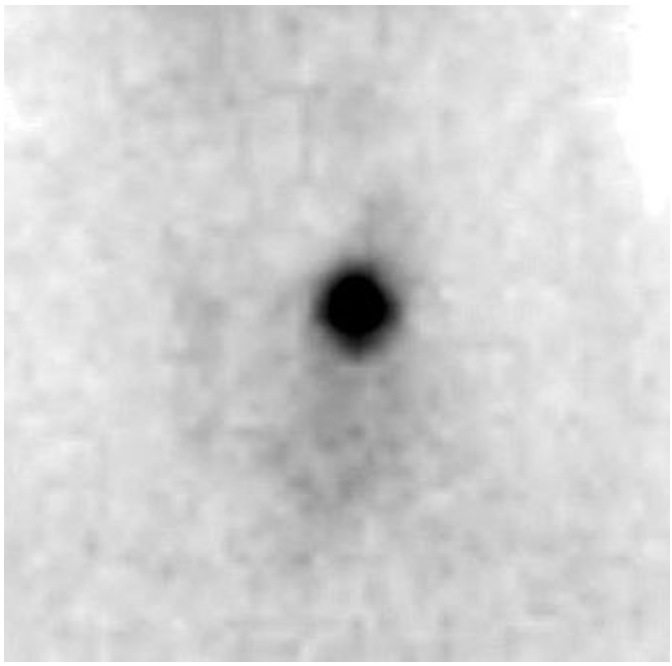

Figure 2. Thyroid scan compatible with Marine Lenhart' syndrome.

Discussion

SML is a rare pathology that associates Graves' disease with hyper functioning nodules. Diagnostic criteria are not well established. It can be done in a classic way in which the nodules on the scintigraphy present the "cold" appearance described by Charkes, and in a variant in which cases are characterized by nodules with a "hot" appearance. This last subtype includes cases of functioning nodules with thyroid carcinomas and cases in which Graves' disease and functioning nodules can appear at different times in the evolution, such as after treatment with synthetic antithyroid drugs or radioactive iodine. The last presentation is the one present in our patient due to hyperuptake in multinodularity.

The uptake in the characteristic scintigraphy is given by the expression of NIS in the thyroid follicles, which increases, stimulated by TSH-stimulating antibodies in Graves' disease (independent of endogenous TSH), and cold-hypofunctioning nodules have expression of NIS TSH dependent. And in the physical examination, exophthalmos and acropathies have been reported. Series of SML have been reported in which exophthalmos was present in up to 50% of patients[7].

It is not clear if the hyperfunctioning nodules represent a form of localized - autoimmune Graves' disease or if they could be an acquired and localized mutation in the TSH receptor gene, producing a constitutive activation of the TSH receptor, which leads to the development of a toxic adenoma[8].

The activity of toxic thyroid nodules can be enhanced by stimulators such as TSH or antibodies against the TSH receptor. In summary, our case presents a variant of SML characterized by ocular disease and hyperuptake in the thyroid scintigraphy. Treatment can be with radioiodine, surgery, and synthetic antithyroid drugs.

Surgery is preferred in suspected malignancy, moderate to severe orbitopathy, compressive symptoms, symptomatic goiter and patient preference. Synthetic antithyroids are preferred in

severe hyperthyroidism, advanced age, to achieve euthyroidism before definitive treatment. Radioactive iodine is a safe, definitive therapy that requires that the patient does not have active thyroid ophthalmopathy, in which patients are generally left with thyroid hypofunction - hypothyroidism and therefore must be replaced with exogenous thyroid hormone[9] as is the case presented.

There are reports in which radiotherapy in SML induces hypothyroidism more frequently than in multinodular goiter, reported by Danno, D. et al, (42.9 vs. 9.0%, p = 0.005)[10]. Remember that this pathology should be suspected if, before stopping treatment with synthetic antithyroid drugs, the patient presents a relapse, accompanied by autonomous functioning nodules by scintigraphy. We must remember that this pathology is not malignant and can be accompanied by thyroid carcinoma, but it is the minority of cases, so follow-up is essential in these patients.

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