

A rare case of central hyperthyroidism and autonomous nodule of the thyroid

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A 63-year-old woman with autoimmune thyroiditis (AIT) and hyperthyroidism caused by an autonomous nodule of the left thyroid lobe was admitted to a nuclear medicine department for radioiodine therapy in 2022.

In 2018, AIT and concomitant focal lesions were diagnosed, laboratory tests revealed euthyroidism and increased concentration of antithyroid peroxidase antibodies (586 IU/ml; reference range [RR] <35 IU/ml). After 4 years, elevated free thyroid hormone values were observed with normal thyroid-stimulating hormone (TSH) level. Then, thyroid technetium scintigraphy was performed, which showed an autonomous tumor of the left lobe (FIGURE 1A) and iodine uptake of 32.4%. Radioiodine therapy was planned, but due to the hormonal test results (inadequate TSH levels concerning free triiodothyronine [FT3] and free thyroxine [FT4]), this treatment was abandoned.

The patient was referred to the Department of Endocrinology, Diabetes, and Isotope Therapy for further diagnostics. The overall clinical picture, as well as the results of hormonal tests (unsuppressed TSH concentration of 3.5 μ IU/ml; RR, 0.35–4.94 μ IU/ml, with elevated FT4 concentration of 19.88 pmol/l; RR, 9.01–19.05 pmol/l; lack of adequate TSH stimulation in the thyrotropin releasing hormone test [0 min, 3.61 μ IU/ml, 30 min, 4.25 μ IU/ml, 60 min, 4.45 μ IU/ml], increased concentration of the α subunit of glycoprotein hormones of 7.83 IU/l [RR, 0–1.3 IU/l], insulin-like growth factor 1 concentration of 71.1 ng/ml [RR, 75–212 ng/ml], normal level of sex hormone binding globulin and anti-TSH receptor antibodies), and imaging studies (FIGURE 1B–1D) indicated thyrotropinoma

(TSHoma). Hyperprolactinemia (96.7 ng/ml; RR, 6–29.9 ng/ml) was most likely a result of the tumor pressure on the pituitary infundibulum, but mixed adenoma could not be excluded. Pituitary magnetic resonance imaging showed a tumor measuring 1.15 cm \times 1.65 cm \times 1.9 cm with the suprasellar and left cavernous sinus infiltration, optic chiasm modeling, and local adhesion to the left optic nerve (FIGURE 1E). During hospitalization, lanreotide was administered subcutaneously as a single dose of 120 mg, and the patient was qualified for transsphenoidal neurosurgery of the pituitary macroadenoma. Following lanreotide administration, normalization of peripheral thyroid hormone concentration was not obtained. Histologic examination revealed pituitary adenoma with multihormonal structure of growth hormone (+), prolactin (+), TSH (+), α subunit of glycoprotein hormones (+), and Ki-67 approximately 1%. Ultrastructural features of TSHoma with significant polymorphism of neuroendocrine granules were found on electron microscope preparations. After neurosurgery, normalization of FT4 concentration (13.6 pmol/l) with a normal TSH level (1.01 μ IU/ml), and a minor reduction in the dimensions of the lesions in the thyroid were observed. Due to goiter extending below the sternal notch and the size of the focal lesion in the left thyroid lobe, the patient was qualified for strumectomy.

TSHoma is a rare cause of hyperthyroidism accounting for 0.5% to 3% of all types of pituitary adenomas.¹ Clinical manifestations of this disease include hyperthyroidism and goiter associated with increased concentrations of free thyroid hormones and unsuppressed TSH. Additionally, there are symptoms resulting

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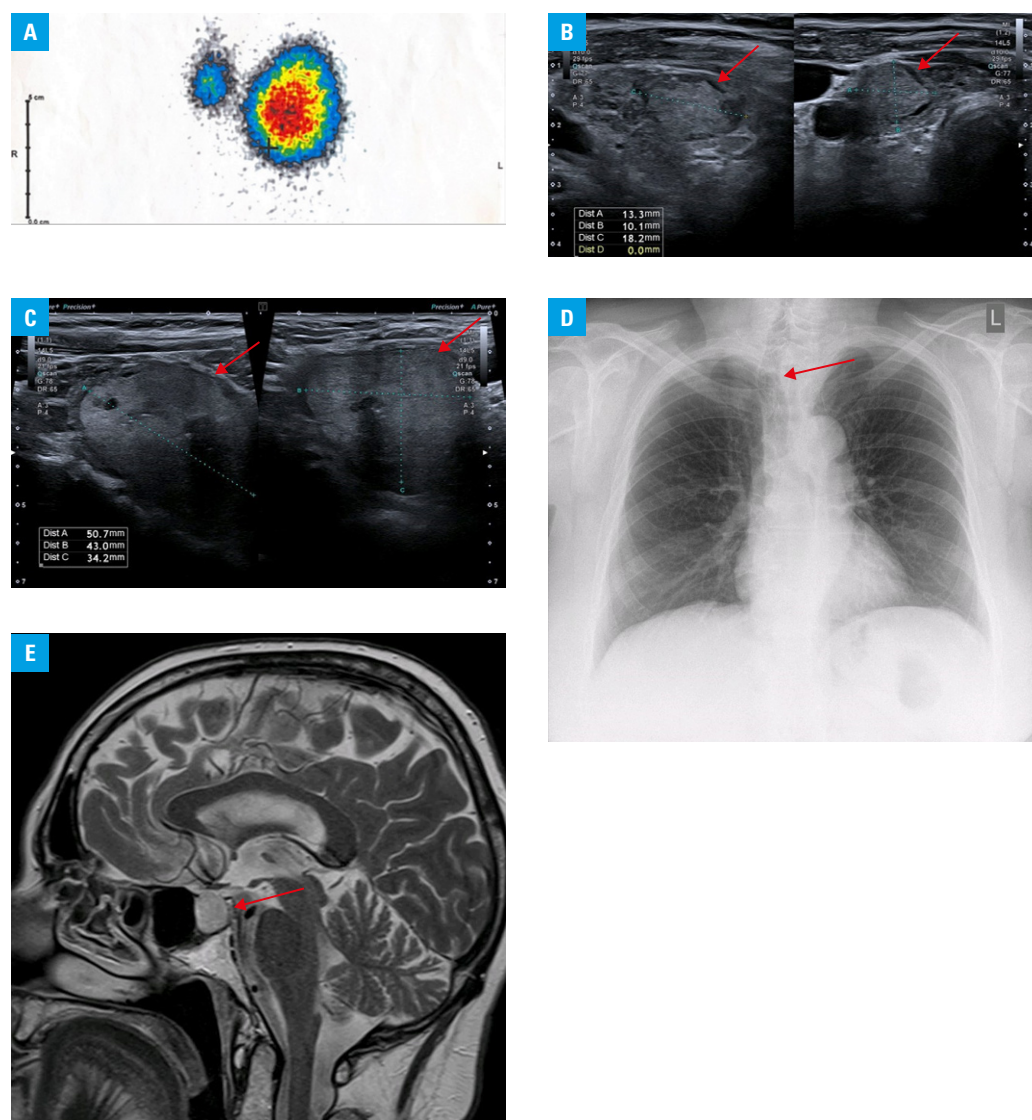


FIGURE 1 **A** – thyroid scintigraphy planar acquisition after intravenous administration of 80 MBq ^{99m}Tc showing high isotope uptake by the left lobe of the thyroid gland (a scintigraphy hot area). Image of the autonomous nodule of the left thyroid lobe; **B** – ultrasound examination showing an isoechoic solid-fluid lesion located in the right lobe of the thyroid gland, measuring 13 mm \times 10 mm \times 18 mm, with a hypoechoic halo and peripheral vascularization (arrows); **C** – almost entire left lobe of the thyroid gland is filled with an isoechoic, solid-fluid focal lesion measuring 43 mm \times 34 mm \times 50 mm, with a hypoechoic halo, peripheral type of vascularization, and single, linear calcifications (arrows). **D** – chest X-ray showing slight displacement of the trachea (arrow); **E** – sagittal T2-weighted magnetic resonance imaging of the pituitary gland showing macroadenoma (1.15 cm \times 1.65 cm \times 1.9 cm) with suprasellar and left cavernous sinus invasion, modeling of the optic chiasm, and localized adhesion to the left optic nerve. The pituitary infundibulum is displaced to the right side (arrow).
Abbreviations: L, left; R, right

from the mass effect of the tumor and clinical signs of concomitant hypersecretion of other pituitary hormones (acromegaly, galactorrhea / amenorrhea).² The first-line treatment for TSHoma is resection of the pituitary adenoma followed by irradiation in the case of non-radical surgery. Additionally, somatostatin analogs effectively reduce TSH secretion in over 90% of cases, which leads to restoration of euthyroidism.^{3,4} There are studies that indicate that AIT can induce proliferation of thyroid-stimulating cells and lead to TSHoma formation.⁵ However, most of the available research works are case studies. Therefore, diagnostic

vigilance necessary for an appropriate therapeutic process should be maintained.

ARTICLE INFORMATION

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