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A marked goiter involved in Marine-Lenhart syndrome

Kosuke Oka MD, PhD[1] | Miho Yasuda MD[1] | Tadahiko Shien MD, PhD[2] | Fumio Otsuka MD, PhD[1]

[1]Department of General Medicine, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan

[2]Department of Breast and Endocrine Surgery, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan

Correspondence
Fumio Otsuka, Department of General Medicine, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan.
Email: fumiotsu@md.okayama-u.ac.jp

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A 41-year-old female had a complaint of palpitation and sweating. Serum free thyroxine (FT4 > 5 ng/dL) and free triiodothyronine (FT3 > 20 pg/mL) levels were excessive, and TSH level was undetectably lowered (<0.001 μIU/mL). A huge goiter (Figure 1A) with the increased flow was shown by ultrasound (Figure 1B), and a tracheal shift by a 10-cm mass was observed by CT (Figure 1C). $^{99m}TcO4^-$ was accumulated in the right nodule (Figure 1D) with degenerative cold spots as shown by ultrasound (Figure 1B), indicating an autonomously functioning thyroid nodule (AFTN), and the accumulation in the marginal thyroid indicated coexisting Graves' disease (Figure 1D). Serum level of TSH-receptor stimulating antibody (TSAb) was markedly high at 5324%, and both of TgAb (25.3 IU/mL) and TPOAb (957.3 IU/mL) were also positive. Following intensive treatment with methimazole (40 mg), iodine (100-200 mg), and propranolol (30 mg), total thyroidectomy was performed. The tumor in the right lobe was pathologically adenomatous nodule (Figure 1E), while the left lobe was compatible to Graves' disease consisting of enlarged follicular cells with lymphocyte infiltration (Figure 1F).

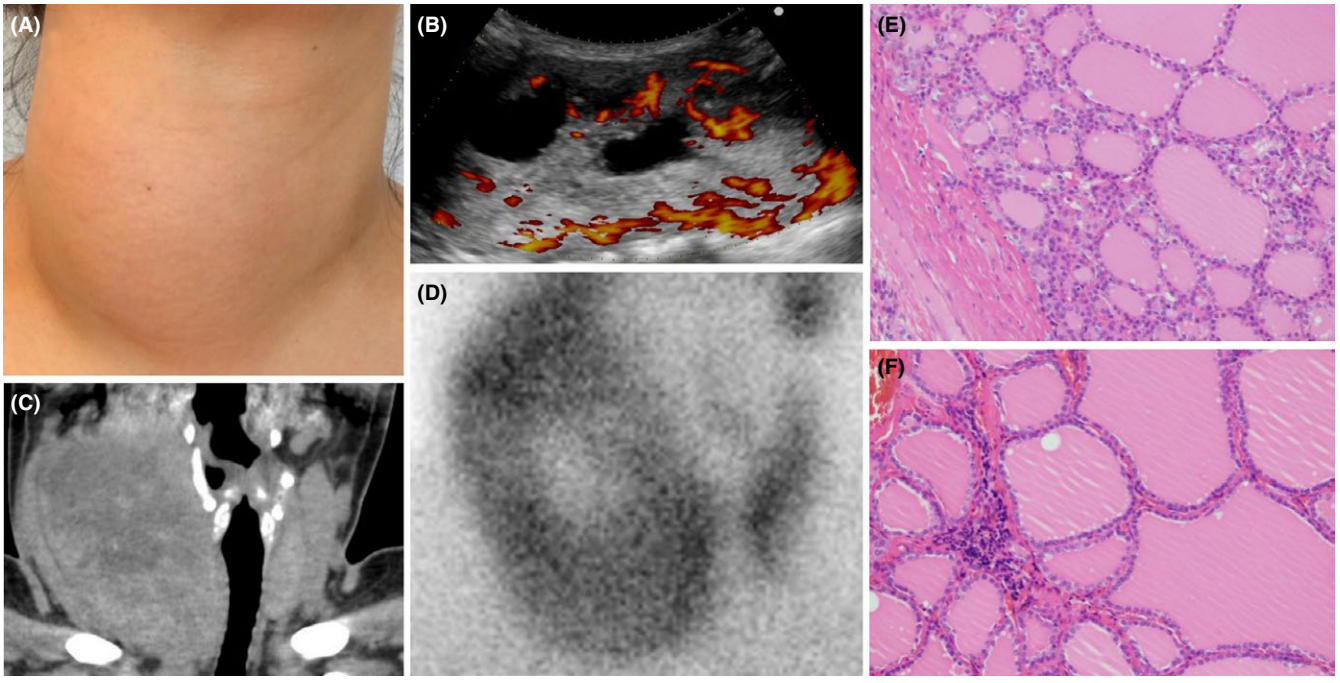

FIGURE 1 A, Appearance of the neck. B, Doppler ultrasonography of the thyroid. C, Cervical CT scan. D, $^{99m}TcO4^-$ scintigraphy of the thyroid. E, Pathology of the resected autonomously functioning thyroid nodule in the right lobe of thyroid. F, Pathology of the resected left lobe of Graves' disease

Marine-Lenhart syndrome is defined as the co-existence of AFTN and Graves' disease.[1] This disease entity has been still controversial, since some cases that had developed Graves' disease after radiotherapy for AFTN were inappropriately included in this syndrome.[2] Nevertheless, as a common pathophysiology of these two disorders, it is of interest that the release of thyroid hormones from AFTN tissues can be accelerated in response to TSH-receptor stimulation.[3] Both scintigraphy and autoimmune testing should be performed for appropriate diagnosis.

Furthermore, family physicians should also be careful for handling the patients with hyperthyroidism.[4] If the patients relapse quite earlier after the cessation of oral antithyroidal therapy, the physician needs to suspect the possibility of coexistence of AFTN and consider the choice of radioactive iodine therapy or surgical treatment for hyperthyroidism.

CONFLICT OF INTEREST

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

ORCID

Kosuke Oka http://orcid.org/0000-0002-7996-2354

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