

The oldest case of Marine-Lenhart syndrome?

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DECLARATIONS

Competing interests

None declared

Funding

No funding was required in the preparation of this manuscript

Ethical approval

Written informed consent to publication was obtained from the patient

Guarantor

Dr Dhatariya acts as the guarantor for this work

Contributorship

All authors contributed equally to this article

Acknowledgements

None

Reviewer

Christopher Ball

Marine Lenhart syndrome occurs rarely at any age, we present a case of this unusual condition occurring in an octogenarian.

Introduction

Thyroid dysfunction is relatively common, with hypothyroidism in women reported to have a prevalence of 4.1 per 1000 population, and 0.8 per 1000 having hyperthyroidism. Unlike hypothyroidism which becomes more prevalent with age, Grave's disease – autoimmune hyperthyroidism – seems to not do this, with the incidence remaining relatively steady at 1 in 1000 per year between the ages of 35 and 60 years.

Thyroid nodules are also very common, being palpable in approximately 5%, and being detectable on ultrasound in up to two thirds in the general (iodine replete) population.² The prevalence has been shown to be higher with increasing age and in women.³ The vast majority of these nodules are non-functioning, and cause no symptoms. Thus the presence of autonomously functioning nodules within a Grave's thyroid is rare, occurring in 0.8% to 2.7% of cases of Grave's disease.^{4,5} We present such a case of Marine Lenhart syndrome – functioning nodules within an overactive thyroid gland – in a lady of advanced age.

Case Report

An 81-year-old woman was admitted in January 2007 with worsening symptoms of heart failure. She had a past history of mitral regurgitation due to bi-leaflet mitral valve prolapse and had several

previous admissions due to heart failure. Her medication list included calcium and vitamin D, furosemide, ramipril, and warfarin. She was also on sotalol which had been started 2 days prior to admission by her general practitioner. On examination she was short of breath at rest and in atrial fibrillation with a fast ventricular rate. She also had evidence of peripheral oedema, a raised jugular pressure and lung crepitations. Palpation of her thyroid gland was unremarkable.

Her blood tests showed that she was biochemically thyrotoxic with a thyroid stimulating hormone (TSH) of 0.02 mIU/L (0.35-3.5), a free thyroxine (fT4) of 24 pmol/L (8-21) and a free triiodothyronine (fT3) of 4.2 pmol/L (3.8–6.0). She was started on carbimazole 20 mg twice daily by her admitting team and her heart failure medication was optimized. Over the following 5 days she improved and was discharged with an endocrinology outpatient appointment. When she was seen 3 months later, her TSH was 12.34, fT4 11. She was advised to decrease her dose of carbimazole to 20 mg once daily and was referred for radioactive iodine treatment. A CT scan of her neck was also arranged because of a history of choking whilst eating and a feeling of neck fullness. This showed that she had retrosternal extension of her goitre on the left, with no evidence of tracheal compression.

Six weeks later, 100 micrograms of thyroxine was added as part of a block and replace regime whilst waiting for her radioactive iodine and her thyroid function test results to normalize with a TSH of 0.6 and a fT4 of 22, a further 6 weeks later.

In our institution, a Tc-99m thyroid scan is performed prior to the administration of radioactive iodine. This showed 2 foci of decreased tracer accumulation at either lower pole of the thyroid with generalized intense tracer uptake within an enlarged thyroid gland (Figure 1). This was consistent with a diagnosis of Marine-Lenhart syndrome on a background of Grave's disease. When last checked in November 2011, her thyroid function tests were normal.

Discussion

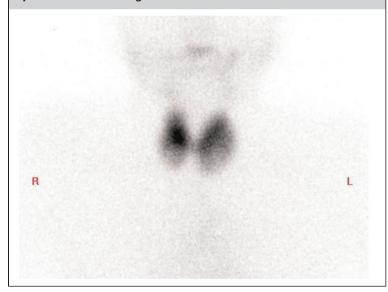
Marine-Lenhart Syndrome was first described in 1911.⁶ The tissue within the functioning nodule

becomes suppressed by the overactivity of the remaining gland and therefore does not take up tracer or radioiodine. However, as the remainder of the gland is treated with oral medication and becomes progressively less active, the nodules become more active.

There has been controversy regarding how to diagnose Marine-Lenhart syndrome, depending on what imaging techniques have been used to identify the presence of a thyroid nodule. If ultrasound scanning has identified a nodule with no differences in uptake on Tc-99m scanning, or if no ultrasound scan is done, it is difficult to justify the diagnosis.^{7,8,9}

Figure 1

There is intense tracer accumulation seen within an enlarged thyroid gland. There are two foci of decreased tracer accumulation at either lower pole consistent with Marine Lenhart syndrome on a background of Graves.



References

- 1 Vanderpump MP, Tunbridge WM, French JM, et al. The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham Survey. Clin Endocrinol (Oxf) 1995;43:55-68
- 2 Cooper DS, Doherty GM, Haugen BR, et al. Revised American Thyroid Association management guidelines for patients with thyroid nodules and differentiated thyroid cancer. *Thyroid* 2009;19:1167–214
- 3 Tan GH, Gharib H. Thyroid incidentalomas: Management approaches to nonpalpable nodules discovered incidentally on thyroid Imaging. Ann Intern Med 1997;126:226–31
- 4 Carnell NE, Valente WA. Thyroid nodules in Graves' disease: classification, characterization, and response to treatment. *Thyroid* 1998;8:647–52
- 5 Wiest PW, Hartshorne MF, Inskip PD, et al. Thyroid palpation versus high-resolution thyroid ultrasonography in the detection of nodules. J Ultrasound Med 1998;17: 487–96
- 6 Marine D, Lenhart CH. Pathological anatomy of exophthalmic goitre. *Arch Intern Med* 1911;8:265–316
- 7 Cakir M. Diagnosis of Marine-Lenhart syndrome. *Thyroid* 2004:14:555
- 8 Braga-Basaria M, Basaria S. Marine-Lenhart syndrome. *Thyroid* 2004;**14**:1107
- 9 El-Kaissi S, Kotowicz MA, Goodear M, Wall JR. An unusual case of Marine-Lenhart syndrome. *Thyroid* 2003;13:993–4

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