lesson of the month

Hypothyroid cardiomyopathy due to hypopituitarism: a diagnostic dilemma

A case of hypothyroid cardiomyopathy secondary to hypopituitarism posed a diagnostic challenge. This lesson describes the patient's presentation with simultaneous acute heart failure and neurological signs; the investigations and imaging findings which initially suggested myocardial infarction (MI) and/or infiltration; and the response to treatment, with biochemical, sonographic and clinical resolution of the cardiomyopathy following thyroxine replacement therapy. This lesson illustrates the cardiovascular sequelae of severe hypothyroidism, and physicians are reminded of the difficulties involved in investigating putative coronary events in hypothyroid patients, since hypothyroidism itself may mimic MI.

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Lesson

A 46-year-old woman with type 1 diabetes mellitus presented with double vision on right lateral gaze and a history of long-standing lethargy. Examination showed reduced proximal muscle strength bilaterally, pedal oedema, a convergent right eye which failed to abduct, and full visual fields to confrontation. She was hypertensive at 170/88 mmHg.

Initial investigations revealed microcytic anaemia (haemoglobin 9.2 g/dL), poor diabetic control (HbA1c fraction 12.5%), and normal renal and liver function. Thyroid function was abnormal, with low free thyroxine (2.1 pmol/l, normal range 9.0–22.5) and free tri-iodothyronine (0.78 pmol/l, normal range 3.5–6.5). Thyroid stimulating hormone (TSH) was only mildly elevated (5.45 mU/l, normal range 0.35–5.0). Further endocrine tests confirmed hypopituitarism, although imaging of the brain (computed tomography) and pituitary fossa (magnetic resonance imaging (MRI)) was normal.

Serum creatine kinase (CK) was markedly elevated (632 U/l, normal range 22–269), as was troponin T (Tn-T) (0.25 ng/ml, normal range <0.1). The electrocardiogram (ECG) showed sinus rhythm, flat T waves infero-laterally and marked low voltage complexes (<0.3 mV) (Fig 1). Though pain-free, the patient was treated for acute coronary syndrome as poor diabetic control made silent myocardial ischaemia a possibility. Subsequent coronary angiography, however, was normal. Echocardiography revealed a thickened myocardium, but cardiac MRI showed no evidence of myocardial infiltration. One week later the Tn-T level remained elevated, but a simultaneous troponin I (Tn-I) level was normal. Creatine kinase isoenzyme analysis revealed an elevated absolute and fractional level of CK-MB.

Following thyroxine replacement, the patient reported much improved energy levels. Two months after discharge the Tn-T level had fallen to 0.13 ng/ml; at four months the CK level had normalised. At six months, the ECG voltage had more than doubled and repeat exams showed a reduction in left ventricular wall thickness to 1.2 cm.

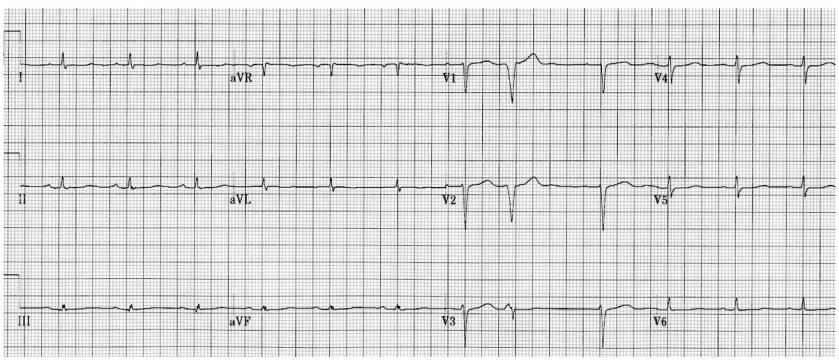

Fig 1. ECG at presentation. A 12-lead electrocardiogram showing sinus rhythm at 75 bpm, low voltage in the limb leads (mean <0.3 mV) and lateral leads, poor R-waves anteriorly and a prolongation of the QT interval (QTc 477 msec).

Discussion

At presentation, the patient had an abnormal ECG with elevated CK and troponin levels. These abnormalities resolved with thyroxine replacement therapy, and coronary angiography was normal. Hypothyroid cardiomyopathy has been described previously, including ECG changes suggesting ischaemia which have reversed with thyroxine replacement.[1] While this report describes hypothyroid cardiomyopathy secondary to hypopituitarism, it is interesting to note that the presentation was similar.

Serum CK can be elevated in up to 90% of patients with hypothyroidism;[2] while this is mostly due to the skeletal CK-MM isoenzyme, CK-MB can also be elevated without MI.[3] This case shows that Tn-T can be elevated in hypothyroidism, even as part of a hypopituitary picture. A previous laboratory study reported that 65% of patients with hypothyroidism with markedly elevated TSH also had elevated CK, and 13.5% had elevated CK-MB, but Tn-I levels were normal in all samples.[4] While Tn-I may therefore be regarded as the best humoral marker for myocardial damage in these patients, it is likely that most myocardial enzyme markers are of questionable reliability in hypothyroidism. Furthermore, many hospitals have access to only one troponin assay. Therefore, coronary angiography may need to be considered earlier in these patients.

Conclusion

Physicians will often investigate patients with hypothyroidism for putative acute coronary syndromes, especially as they are at higher risk of coronary atherosclerosis.[5] In these cases, extreme care is required when interpreting investigations, since many of the ECG and enzyme changes of myocardial ischaemia and/or infarction can be mimicked by hypothyroidism itself, whether it is primary hypothyroidism or, as our case now adds, secondary to pituitary failure.

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Competing interests

No financial or non-financial competing interests.

Authors' contributions

ANS collected the laboratory data, performed the literature review, and wrote the original manuscript. SWD performed the echocardiography and made substantial revisions to the manuscript. DT collected follow-up data on the patient's response to treatment and made significant contributions to the manuscript.

References

1 Gunduz H, Arinc H, Yolcu M *et al.* A case of hypothyroidism mimicking acute coronary syndrome. *Int J Cardiovasc Imaging* 2006;22:141–5.
2 Goldman J, Matz R, Mortimer R, Freeman R. High elevations of creatine phosphokinase in hypothyroidism. An isoenzyme analysis. *JAMA* 1977;238:325–6.
3 Nee PA, Scane AC, Lavelle PH, Fellows IW, Hill PG. Hypothermic myxedema coma erroneously diagnosed as myocardial infarction because of increased creatine kinase MB. *Clin Chem* 1987;33:1083–4.
4 Cohen LF, Mohabeer AJ, Keffer JH, Jialal I. Troponin I in hypothyroidism. *Clin Chem* 1996;42:1494–5.
5 Boelaert K, Franklyn JA. Thyroid hormone in health and disease. *J Endocrinol* 2005;187:1–15.

Erratum

Mir FA, Brown MJ, Appleton DS. Lessons in the diagnosis and management of Conn's syndrome. *Clin Med* 2007;7:530–2.

Please note that the arrow in Fig 1 on page 530 was covering the 2 cm right-sided adenoma rather than pointing at it. The correct image is reprinted here.