BRITISH JOURNAL OF HOSPITAL MEDICINE

CASE REPORT

What lies beneath: hypothyroid heart and valvular disease

Holly Morgan[1]

Melanie Nana[2]

Kofi Obuobie[2]

Shawmendra Bundhoo[1]

Author details can be found at the end of this article

Correspondence to:
Holly Morgan;
morganhp7@gmail.com

How to cite this article:
Morgan H, Nana M, Obuobie K, Bundhoo S. What lies beneath: hypothyroid heart and valvular disease. Br J Hosp Med. 2020. https://doi.org/10.12968/hmed.2020.0195

Introduction

This article describes the case of a 54-year-old man who presented with severe hypothyroidism and cardiac compromise. Echocardiography revealed a large pericardial effusion which was attributed to the hypothyroidism after other secondary causes were excluded. There were no signs of cardiac tamponade requiring invasive intervention.

Case report

A 54-year-old man presented to his GP with a 6-month history of shortness of breath, lethargy, low mood and constipation. The breathlessness was worse on exertion and lying flat, and was accompanied by progressive leg swelling. He denied any chest pain, cough, fevers or weight loss. His past medical history included hypertension for which he was taking ramipril 7.5 mg. He was a non-smoker who drank minimal amounts of alcohol.

On examination he had a raised jugular venous pressure, his heart sounds were quiet with a soft early diastolic murmur. He had bi-basal crackles with peripheral pitting oedema. There was loss of the lateral third of his eyebrows, dry skin, proximal myopathy and delayed relaxation of reflexes of the upper arm. His heart rate was 58 beats per minute, blood pressure 142/58 mmHg and he was apyrexial. An electrocardiogram showed sinus bradycardia, with small voltage complexes.

Full blood count, liver function and bone profile were normal. He had mild hyponatraemia (sodium 130 mmol/litre, normal range 133–146 mmol/litre), otherwise his electrolyte profile was unremarkable. Thyroid function tests revealed profound hypothyroidism (thyroxine (T4) <5.2 pmol/litre (9.0–19.1 pmol/litre), thyroid-stimulating hormone (TSH) 186.51 mU/litre (0.3–4.4 mU/litre) and anti TPO antibodies 289.3 U/ml (<6 U/ml). He underwent a chest X-ray which demonstrated cardiomegaly, and a transthoracic echocardiogram which revealed a large global pericardial effusion measuring 5.7 cm, with intermittent right atrial collapse and exaggerated respiratory variation on tricuspid and mitral valve Doppler tracings (Figure 1). Moderate aortic regurgitation and impaired left ventricular function, with an estimated ejection fraction of 30%, was also observed.

In addition, he had a computed tomography aortogram which did not reveal any evidence of aortic dissection, aortic root dilatation or mitotic lesions.

He was admitted promptly for further assessment. His blood pressure and heart rate remained within normal limits. The hypothyroidism was deemed the likely cause of the pericardial effusion as no other secondary causes (including neoplastic, bacterial and inflammatory) were identified. He was started on levothyroxine 100 mg and the dose was uptitrated over 2 months. As there was no clinical evidence of cardiac tamponade, he was discharged home with a follow-up focused echocardiogram organised for 1 week post discharge.

The patient had weekly echocardiograms and regular thyroid function tests which reassuringly demonstrated that the effusion was resolving and his TSH and T4 were normalising (Figure 2). At 6 weeks the frequency of follow up was reduced.

A repeat echocardiogram carried out at 4 months following thyroxine replacement demonstrated an effusion of less than 1 cm. At this stage it was noted that resolution of the effusion had allowed the left ventricle to expand (measuring 70 mm in diastole and 50 mm in systole), unveiling underlying severe aortic regurgitation with a functionally bicuspid valve (Figure 3). His ejection fraction was noted to be 50–55%, rather than hyperdynamic as would be expected.

He was therefore discussed at the cardiothoracic multidisciplinary team meeting and has been accepted for aortic valve replacement. In terms of his hypothyroidism, his symptoms resolved with normalisation of his thyroid function.

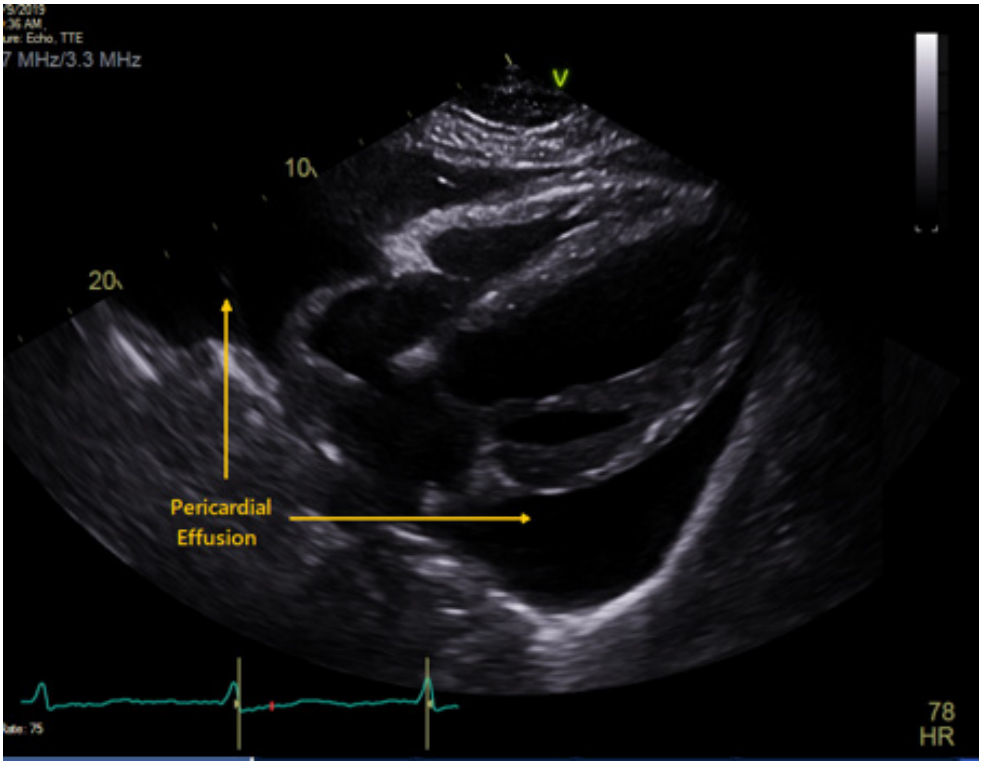

Figure 1. Parasternal view showing large pericardial effusion.

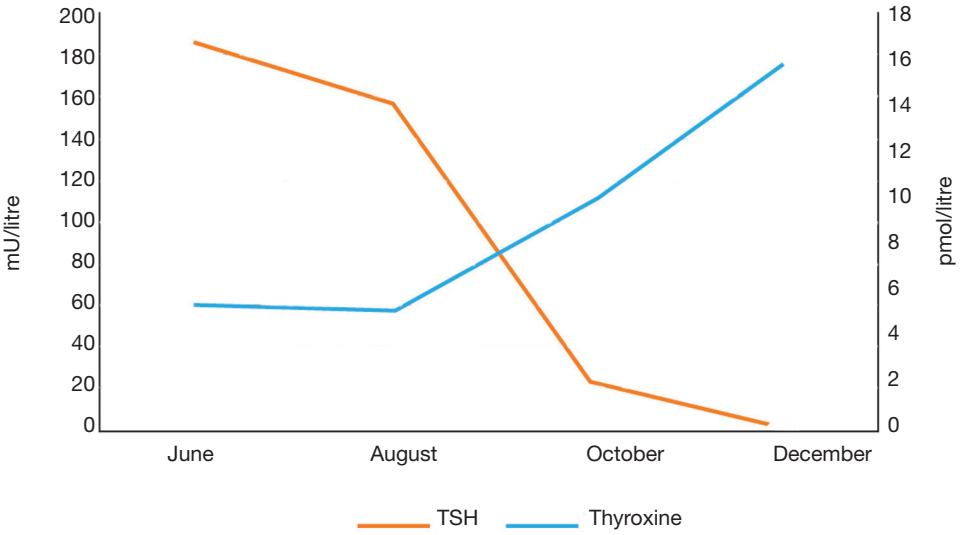

Figure 2. Thyroid function normalising over time. TSH = thyroid-stimulating hormone

He was promptly started on thyroid hormone replacement and the effusion was monitored using transthoracic echocardiography. The effusion resolved over a 4-month period and the patient's thyroid function normalised. The echocardiogram had also revealed aortic regurgitation, and on complete resolution of the effusion it became apparent that it had masked severe ventricular dilatation and thus an indication for surgery. The patient has since been listed for an aortic valve replacement.

Discussion

Cardiac manifestations of hypothyroidism are well documented, including cardiomyopathy, arrhythmia and pericardial disease (Klein and Danzi, 2016). In addition, recent evidence

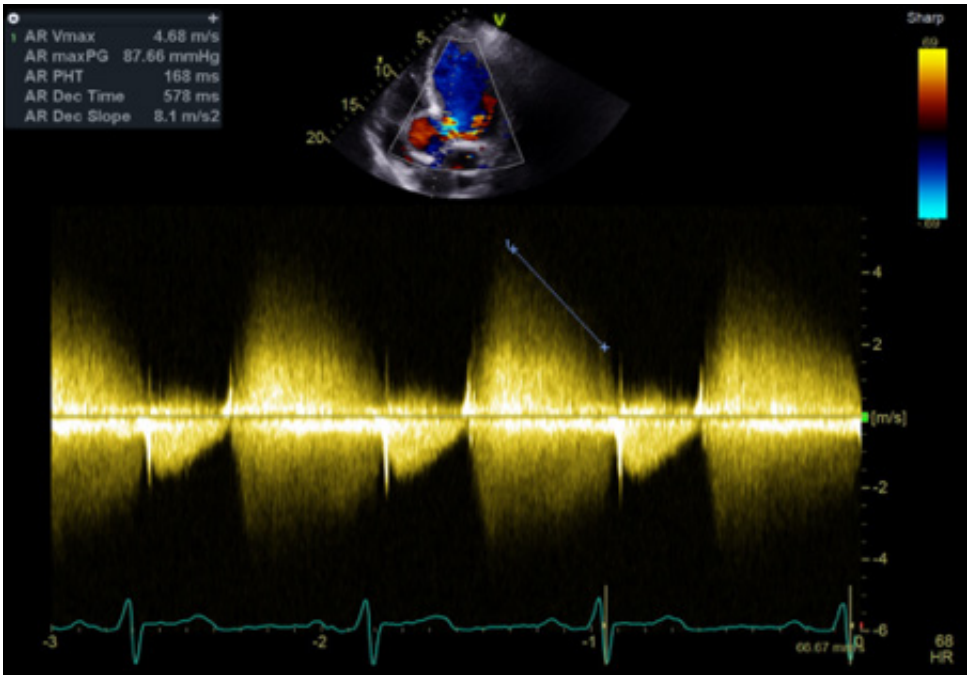

Figure 3. Echocardiogram demonstrating severe aortic regurgitation on Doppler assessment.

demonstrates direct stimulation of the ventricular cardiomyocytes by triiodothyronine (T3) and T4 (Ohba et al, 2020).

The incidence of pericardial effusion secondary to hypothyroidism is reported as ranging between 3 and 37%, with increased incidence in those with severe disease (Nouh et al, 1991). Accumulation of pericardial fluid is driven by the increased permeability of epicardial capillaries, which results in the efflux of protein-rich fluid and glycosaminoglycan, as well as decreased lymphatic draining of albumin (Chahine et al, 2019). Diagnosis relies on the presence of abnormal thyroid hormone levels and exclusion of other secondary causes.

Management includes thyroid hormone replacement, judicious monitoring of the effusion and clinical assessment for evidence of cardiac tamponade. Increased thyroid-stimulating hormone levels or features of tamponade are poor prognostic indicators in terms of resolution of effusions (Hardisty et al, 1980). Case series have documented that the effusion begins to decrease in size as soon as thyroid hormone replacement therapy is commenced (Khaleeli and Memon, 1982). In this case the effusion resolved over 4 months, which is in keeping with the literature (Chahine et al, 2019).

Aortic regurgitation can result from disease of either the valve leaflets or the aortic root (Lancellotti et al, 2010). National guidelines recommend serial transthoracic echocardiogram monitoring of patients with aortic regurgitation to assess severity, cardiac chamber size and function (Vahanian et al, 2012). Visual and Doppler assessments of severity are significantly affected by the left ventricular diastolic pressure gradient and left ventricular compliance. Surgery is recommended in asymptomatic patients with severe aortic regurgitation when the ejection fraction is <50% or the end systolic diameter is >50 mm (Vahanian et al, 2012).

When the effusion resolved, in the case described here, the left ventricular diameter increased, revealing the indication for surgery. The authors therefore highlight the importance of being vigilant for factors which may limit ventricular dilatation during work up of such patients to avoid underestimation of disease severity.

Author details

[1]Cardiology Department, Royal Gwent Hospital, Wales, UK

[2]Endocrinology Department, Royal Gwent Hospital, Wales, UK

Learning points

- Hypothyroidism has a range of cardiac complications, including pericardial effusion.
- In such cases clinical assessment is important to ensure there are no signs of clinical tamponade requiring invasive management.
- Prompt replacement of thyroid hormone results in rapid resolution of the effusion in the majority of cases.
- Factors which limit ventricular dilatation may lead to the underestimation of disease severity in patients with aortic regurgitation.
- Clinicians should be vigilant for these factors in the work up of such patients.

References

Chahine J, Ala CK, Gentry JL, Pantalone KM, Klein AL. Pericardial diseases in patients with hypothyroidism. Heart. 2019;105(13):1027–1033. https://doi.org/10.1136/heartjnl-2018-314528

Hardisty CA, Naik DR, Munro DS. Pericardial effusion in hypothyroidism. Clin Endocrinol. 1980;13(4):349–354. https://doi.org/10.1111/j.1365-2265.1980.tb03395.x

Khaleeli AA, Memon N. Factors affecting resolution of pericardial effusions in primary hypothyroidism: a clinical, biochemical and echocardiographic study. Postgrad Med J. 1982;58(682):473–476. https://doi.org/10.1136/pgmj.58.682.473

Klein I, Danzi S. Thyroid disease and the heart. Curr Probl Cardiol. 2016;41(2):65–92. https://doi.org/10.1016/j.cpcardiol.2015.04.002

Lancellotti P, Tribouilloy C, Hagendorff A et al. European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 1: aortic and pulmonary regurgitation (native valve disease). Eur J Echocardiogr. 2010;11(3):223–244. https://doi.org/10.1093/ejechocard/jeq030

Nouh MS, Famuyiwa OO, Sulimani RA, Al-Nuaim A. The spectrum of echocardiographic abnormalities in hypothyroidism and the effect of hormonal treatment. Ann Saudi Med. 1991;11(6):611–615. https://doi.org/10.5144/0256-4947.1991.611

Ohba K, Okada E, Goto Y et al. Influence of thyroid dysfunction on brain natriuretic peptide level in health examination participants. Endocr J. 2020;67(4):449–454. https://doi.org/10.1507/endocrj.EJ19-0380

Vahanian A, Alfieri O, Andreotti F et al. Guidelines on the management of valvular heart disease (version 2012). Eur Heart J. 2012;33(19):2451–2496. https://doi.org/10.1093/eurheartj/ehs109