

SHORT CASES IN CARDIOLOGY

Dilated cardiomyopathy in thyrotoxicosis

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Heart failure is a rare manifestation of thyrotoxicosis in patients without heart disease.¹⁻³ We report two cases of thyrotoxicosis and no previous heart disease presenting with severe heart failure. Successful treatment of the thyrotoxicosis resulted in progressive improvement of cardiac function.

Case 1

A 31 year old woman was admitted with pulmonary oedema. Two months earlier she had

complained of fatigue, palpitation, and chest discomfort. She had no risk factors for coronary artery disease, had regular menses, and denied smoking or alcohol consumption. There were no symptoms of intercurrent infection.

On physical examination she looked very ill. Her heart rate was 120 beats/min and blood pressure 130/80 mm Hg. She had a small goiter and typical signs of thyrotoxicosis including warm, moist skin and a fine tremor. There was an audible S3 on cardiac auscultation. ECG showed sinus tachycardia, increased voltage, and ST depression in leads I, II, aVL, and V4-6.

Chest radiography was consistent with interstitial pulmonary oedema and moderate cardiomegaly. Echocardiography showed four chamber dilatation and depressed left ventricular (LV) function with an ejection fraction of 34% (fig 1). Routine tests were normal as was erythrocyte sedimentation rate. She had mild anaemia (haemoglobin 115 g/l). Thyroid function tests were available later: thyroid stimulating hormone < 0.2 MU/l (normal range 0.3-4.5), total triiodothyronine 11 pmol/l (normal range 1.0-2.7), and free thyroxine 60 nmol/l (normal range 8.9-24.3).

She was treated with intravenous frusemide and morphine. As thyroid function test results became available treatment with dexamethasone, propylthiouracil, and potassium iodide solution was added. A few hours later she developed atrial fibrillation, which converted to normal sinus rhythm after a short course of amiodarone treatment. She was discharged on oral propylthiouracil, propranolol, and captopril. One year later thyroid function tests were normal, she was free from any symptoms of heart failure, and continued only propylthiouracil treatment. Repeated echocardiography was normal (fig 2).

Case 2

A 46 year old man, who had a history of partial thyroidectomy because of Grave's disease, was admitted with pulmonary oedema. The clinical and laboratory results were typical of thyrotoxicosis. Following successful treatment of thyrotoxicosis with propylthiouracil and β blockers his left ventricular ejection fraction increased from 34% to 50%. He remains clinically well and euthyroid on maintenance propylthiouracil treatment.

Discussion

The association of thyrotoxicosis and cardiovascular morbidity is well established. There are several cardiac manifestations of thyrotoxi-

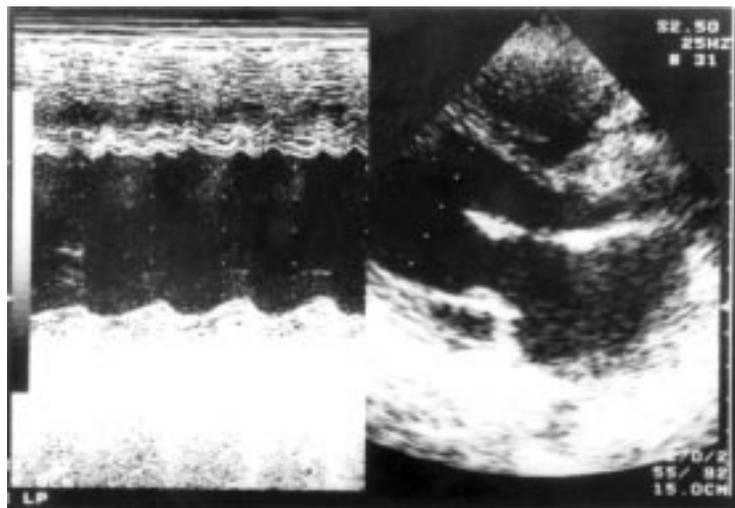


Figure 1 Parasternal long axis view (B mode and M mode) showing reduced systolic left ventricular function. End diastolic diameter is 5.0 cm; end systolic diameter is 4.2 cm; estimated ejection fraction is 34%.

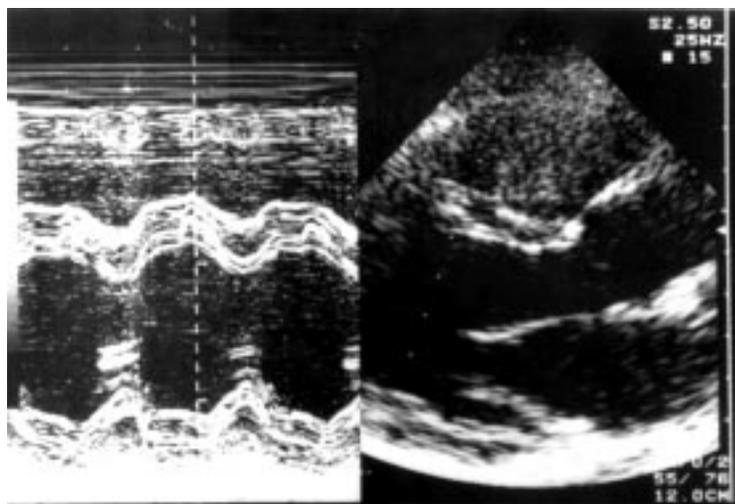


Figure 2 Parasternal long axis view showing normal left ventricular diameter and function. Estimated ejection fraction is 62%.

cosis, including enlargement of the heart, atrial fibrillation, high output heart failure, hypertrophic cardiomyopathy, anginal syndrome without evidence of coronary artery disease, and sudden death.¹⁻³ The high output state in thyrotoxicosis results from direct enhancement of heart rate and contractility by thyroid hormones, indirectly increasing blood volume and causing peripheral vasodilatation. High output heart failure develops despite increased cardiac performance.³ Low output heart failure is an extremely rare manifestation of thyrotoxicosis. There have been a few recent reports of dilated cardiomyopathy and congestive heart failure, especially in children and elderly patients with apathetic thyrotoxicosis.^{4,5} Most of the patients had complete or near complete recovery of cardiac function after treatment.^{4,5} However, Ebisawa *et al* reported that cardiomyopathy in patients with thyrotoxicosis may be irreversible even 15 years after successful treatment of their thyrotoxicosis.⁵ There has also been a report of sudden death in one

young patient with thyrotoxicosis and dilated cardiomyopathy.⁶ There were no specific abnormalities in myocardial biopsy specimens taken from these patients.

We describe two young patient with thyrotoxicosis who developed dilated cardiomyopathy and pulmonary oedema as the first presenting symptom. Patients presenting with heart failure and dilated cardiomyopathy may have thyrotoxicosis as the underlying cause. Treatment of the thyrotoxicosis can restore left ventricular function. Awareness of this possible presentation of thyrotoxicosis may help identify patients with reversible dilated cardiomyopathy.

- 1 Woeber KA. Current concepts. *N Engl J Med* 1992;327:94-8.
- 2 Thyrotoxicosis and the heart: something old and something new [editorial]. *J Clin Endocrin Metab* 1993;77:332.
- 3 Polikar R, Burger AG, Scherrer U, *et al*. The thyroid and the heart. *Circulation* 1993;87:1435-41.
- 4 Umpierrez GE, Challapalli S, Patterson C. Congestive heart failure due to reversible cardiomyopathy in patients with hyperthyroidism. *Am J Med Sci* 1996;410:99-102.
- 5 Ebisawa K, Ikeda H, Maruta M. Irreversible cardiomyopathy due to thyrotoxicosis. *Cardiology* 1994;84:274.
- 6 Manger JA, Clark W, Allenby P. Congestive heart failure and sudden death in a young woman with thyrotoxicosis. *West J Med* 1988;149:86-91.

SHORT CASES IN CARDIOLOGY

Non-surgical CHOP cures right ventricular outflow obstruction

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A 27 year old banker was referred by his general practitioner with constitutional symptoms and a murmur. He had a four month history of weight loss, lethargy and dry cough. On

examination there was a systolic thrill and a loud ejection murmur in the pulmonary area.

Chest radiography suggested a large anterior mediastinal mass, which was confirmed by thoracic computed tomography (CT). Echocardiography showed a 2.5 × 1.5 cm mass in the right ventricular outflow tract (RVOT) (fig 1) and colour flow mapping showed turbulent flow around its free edge. Flow velocity through the RVOT, assessed by continuous wave Doppler, was 4 m/s estimating a pressure gradient of 64 mm Hg (fig 2). The right ventricle was mildly dilated and an anterior extracardiac soft tissue mass was seen.

CT guided biopsy failed to get suitable tissue for analysis but a sample was obtained mediastinoscopically, histology of which confirmed sclerosing mediastinal non-Hodgkin's lymphoma.

The patient was treated with six cycles of cyclophosphamide, doxorubicin—formerly hydroxydaunorubicin, vincristine—oncovin, and prednisolone (CHOP) chemotherapy with excellent clinical and radiological response.

Repeated echocardiography one month after treatment showed complete resolution of the RVOT mass (fig 3) with normal spectral and colour Doppler assessment.

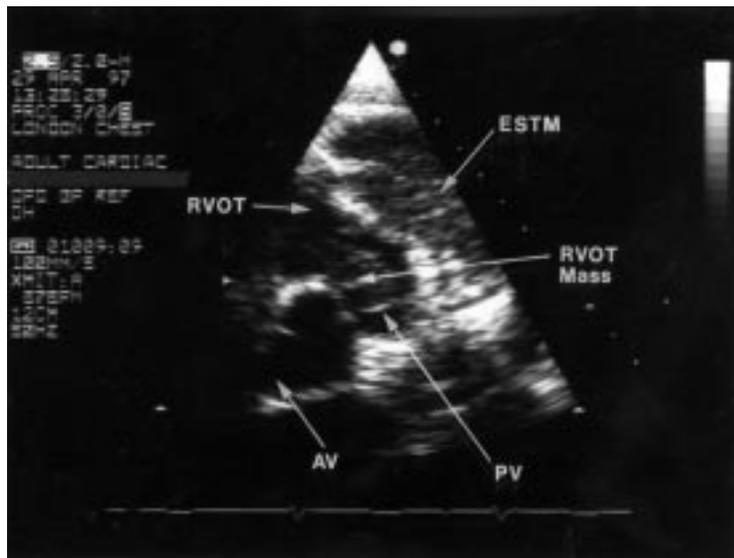


Figure 1 Parasternal short axis echocardiogram. RVOT, right ventricular outflow tract; PV, pulmonary valve; AV, aortic valve; ESTM, extracardiac soft tissue mass.

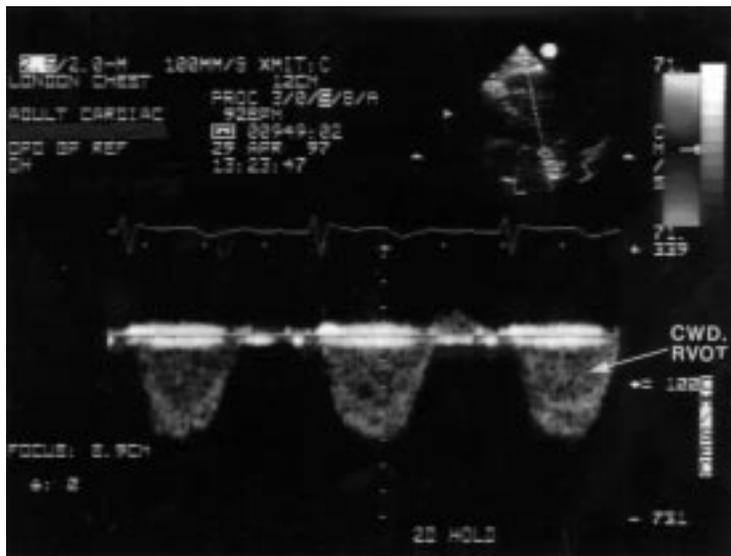


Figure 2 Continuous wave Doppler interrogation across the RVOT showing a peak velocity of 4 m/s (pressure gradient of 64 mm Hg).

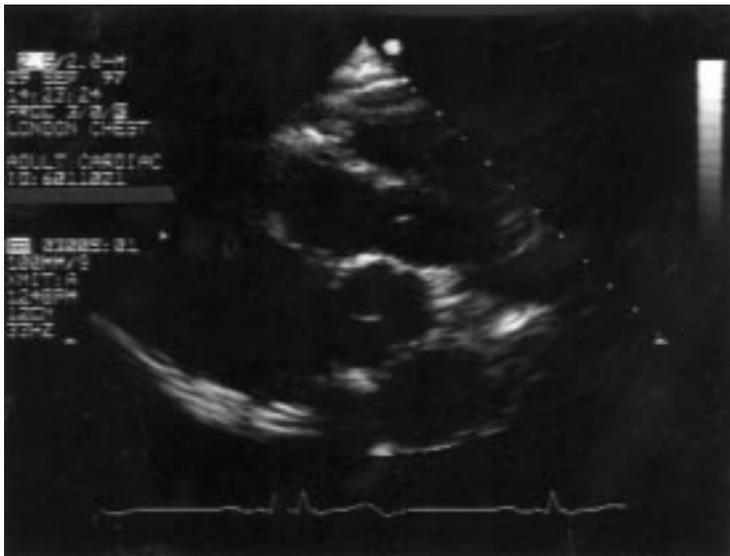


Figure 3 Repeat parasternal short axis scan one month after completion of chemotherapy showing resolution of the RVOT mass and reduction in extracardiac soft tissue shadowing.

Discussion

The differential diagnosis of an intracardiac mass includes vegetation, thrombus, and both primary and secondary neoplasm. Although the pericardium is the most usual site for cardiac secondary tumours, myocardial deposits are not infrequent. Lymphoma is noted as one of the more common tumours to spread to the myocardium.¹⁻³ The RVOT is a well recognised site of cardiac involvement with lymphoma.^{4 5}

Our case illustrates the value of echocardiography in the assessment of such patients. The effect of an appropriate regimen of chemotherapy is clearly demonstrated with complete resolution of the tumour echocardiographically and radiologically.

I thank Ms D Harrington and Ms S Fergy for producing these informative images.

- 1 Lam KY, Dickens P, Chan AC. Tumours of the heart. A 20 year experience with a review of 12,485 consecutive autopsies. *Arch Pathol Lab Med* 1993;117:1027-31.
- 2 Abraham KP, Reddy V, Gatuso P. Neoplasms metastatic to the heart: review of 3314 consecutive autopsies. *Am J Cardiovasc Pathol* 1990;3:195-8.
- 3 McDonnell PJ, Mann RB, Bulkley BH. Involvement of the heart by malignant lymphoma: a clinicopathologic study. *Cancer* 1982;49:944-51.
- 4 Mills P, Wolfe C, Redwood D, et al. Non-invasive diagnosis of subpulmonary outflow tract obstruction. *Br Heart J* 1980;43:276-83.
- 5 Johnstone ID, Popple AW. Right ventricular outflow tract obstruction secondary to small intestinal lymphoma. *Br Heart J* 1980;43:593-6.