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Dilated Cardiomyopathy Following Total Thyroidectomy Complicated by Severe Mitral Regurgitation: A Case Report and Literature Review

Nadia LOUDIYI[1], Siham BELLOUIZE[2], Hicham FALIOUNI[1], Mohamed MALKI[1], Najat MOUINE[1], Atif BENYASS[1]

[1]Department of Cardiology, Mohammed V Military Teaching Hospital, Rabat, Morocco
[2]Department of Cardiovascular Surgery, Mohammed V Military Teaching Hospital, Rabat, Morocco

ABSTRACT : Cardiac dysfunction following thyroid and parathyroid surgery is rare but clinically significant. Hormonal disturbances, including iatrogenic hypothyroidism and calcium metabolism disorders, may adversely affect myocardial function and the mitral valve.

We report the case of a 56-year-old woman who underwent thyroidectomy and parathyroidectomy in 2024. One year later, after discontinuation of thyroid hormone replacement and calcium supplementation, she progressively developed dyspnea, peripheral edema, and heart failure. Transthoracic echocardiography revealed hypokinetic dilated cardiomyopathy (left ventricular ejection fraction [LVEF] 32%) associated with severe functional mitral regurgitation. Laboratory tests showed marked hypothyroidism (TSH 95 µIU/mL, free T4 2.39 pmol/L), hypocalcemia (total calcium 43 mg/L), and elevated NT-proBNP levels (7,850 ng/L).

Management combined guideline-directed medical therapy for heart failure, gradual thyroid hormone replacement, calcium supplementation, and discussion of potential surgical/valvular intervention. After six months of follow-up, a significant improvement in cardiac function and regression of mitral regurgitation were observed.

This case highlights the importance of cardiologic and endocrinologic surveillance after thyroid and parathyroid surgery. Early recognition of hormonal abnormalities and cardiac dysfunction is crucial to optimize management and improve prognosis.

KEYWORDS: dilated cardiomyopathy, thyroidectomy, parathyroidectomy, mitral regurgitation, ventricular dysfunction, heart failure

INTRODUCTION

Dilated cardiomyopathy (DCM) is characterized by left ventricular dilation and impaired systolic function. Among secondary causes, endocrine disorders—particularly hypothyroidism and calcium metabolism abnormalities—represent potentially reversible but uncommon etiologies. Thyroid and parathyroid surgery may induce acute or subacute hormonal disturbances, such as iatrogenic hypothyroidism and hypocalcemia, which can affect both the myocardium and cardiac valves. We report a case of DCM occurring after thyroidectomy and parathyroidectomy, complicated by severe mitral regurgitation, and discuss the pathophysiological mechanisms, management, and relevant literature.

CASE PRESENTATION

A 56-year-old woman with no prior cardiovascular history was admitted for progressive exertional dyspnea, lower limb edema, and fatigue.

She had undergone total thyroidectomy combined with parathyroidectomy one year earlier for toxic multinodular goiter with secondary hyperparathyroidism. The patient had discontinued thyroid hormone replacement therapy and calcium supplementation six months before admission.

On physical examination, blood pressure was 95/60 mmHg (well tolerated), heart rate was 122 bpm and regular, with a grade 5/6 systolic murmur consistent with mitral regurgitation. Bilateral lower limb edema and bilateral mid-lung crackles were present, along with signs of hypocalcemia (muscle cramps and paresthesias).

Electrocardiography showed sinus tachycardia at 124 bpm with narrow QRS complexes. Chest radiography revealed cardiomegaly with pulmonary congestion.

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Transthoracic echocardiography demonstrated left ventricular dilation (LV end-diastolic diameter 68 mm) with global hypokinesia and a reduced LVEF of 32%. Severe functional mitral regurgitation was present (effective regurgitant orifice area 50 mm², regurgitant volume 55 mL), with a dilated mitral annulus (43 mm) and an estimated pulmonary artery systolic pressure of 57 mmHg.

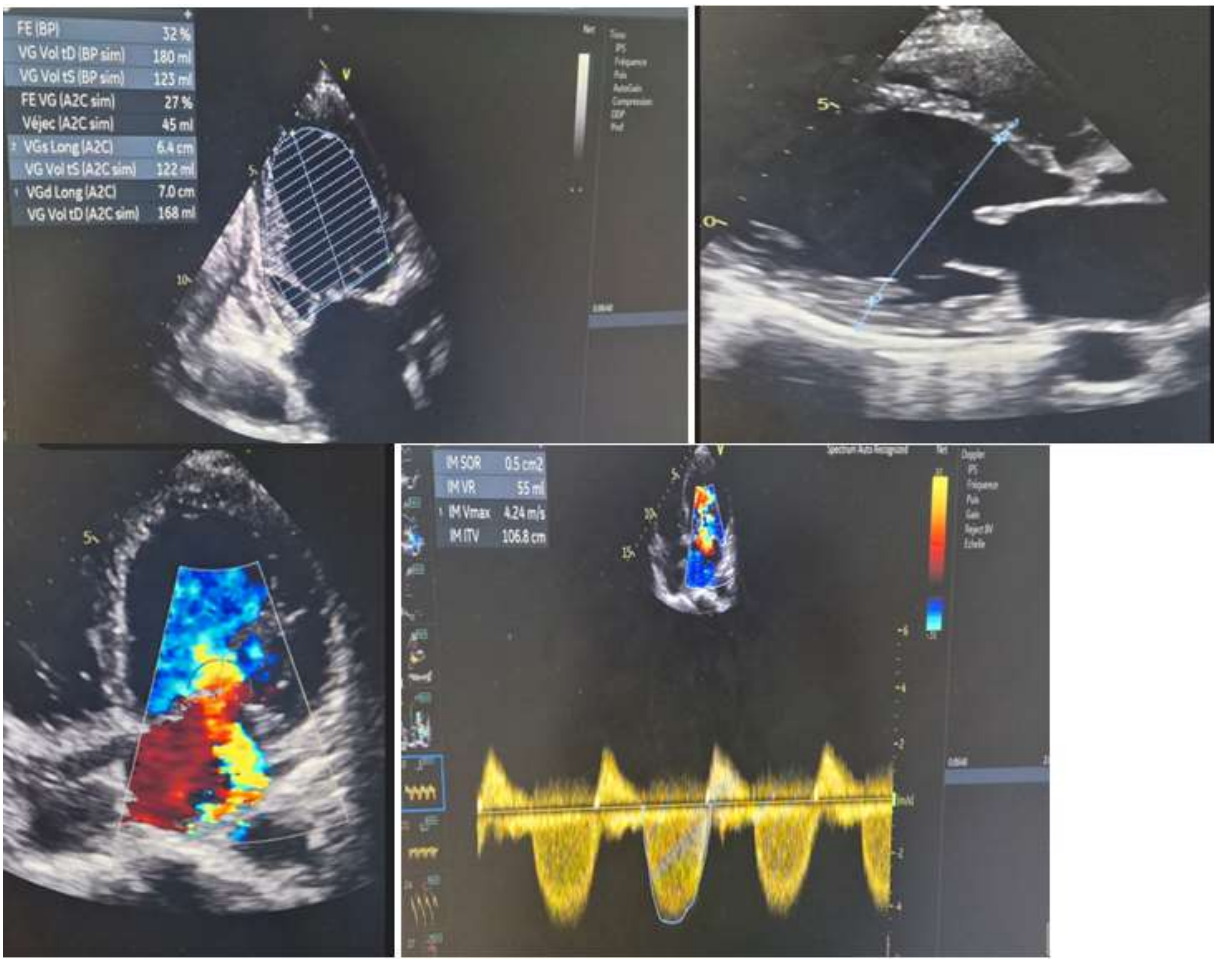

Figure 1: Echocardiography showing left ventricular dilation with systolic dysfunction and severe mitral regurgitation.

Coronary angiography revealed angiographically normal coronary arteries.

Cardiac magnetic resonance imaging (CMR) showed global left ventricular dilation (LV end-diastolic volume indexed to 126 mL/m²) with diffuse hypokinesia and an LVEF of 30%, without late gadolinium enhancement, suggestive of a non-ischemic and potentially reversible etiology.

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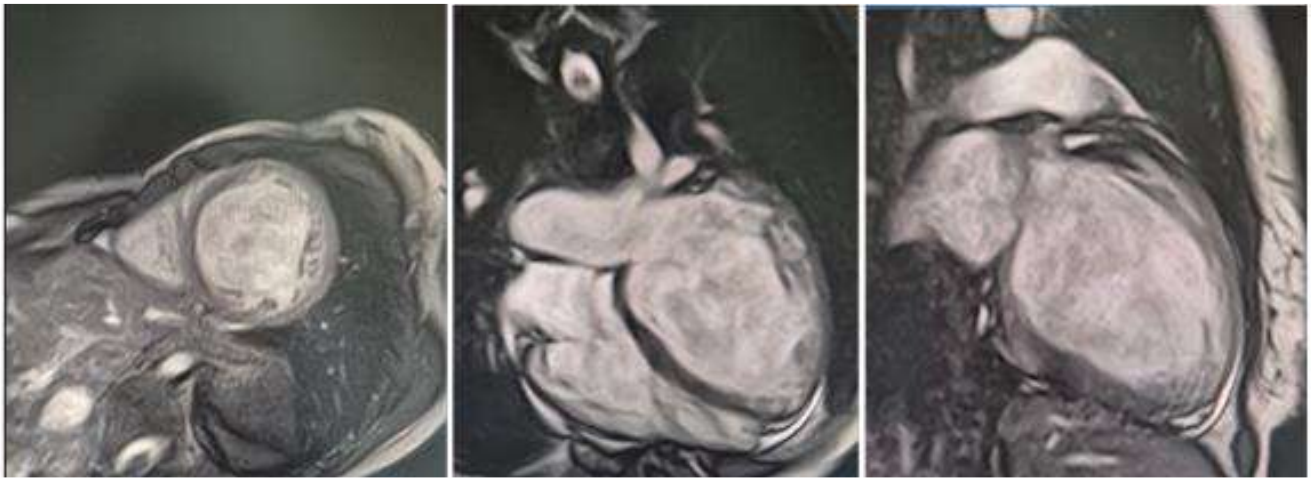

Figure 2: Cine CMR sequences showing a dilated left ventricle with global hypokinesia.

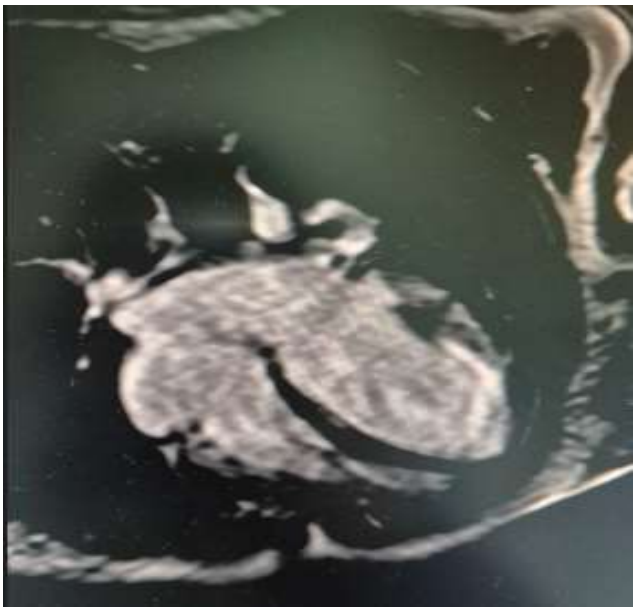

Figure 3: Short-axis late gadolinium enhancement sequence showing absence of myocardial enhancement.

Laboratory tests revealed severe hypothyroidism (TSH 95 µIU/mL, free T4 2.39 pmol/L), hypocalcemia (total calcium 43 mg/L), and elevated NT-proBNP (7,850 pg/mL). C-reactive protein, blood glucose, serum creatinine, and electrolytes were within normal limits.

The patient was treated with calcium and vitamin D3 supplementation, thyroid hormone replacement with levothyroxine, and guideline-directed medical therapy for heart failure (diuretics, beta-blockers, SGLT2 inhibitors, mineralocorticoid receptor antagonists, and angiotensin receptor–neprilysin inhibitor). After six months of treatment and normalization of calcium levels, follow-up echocardiography showed normalization of cardiac chamber dimensions, recovery of LVEF, and significant regression of mitral regurgitation severity (effective regurgitant orifice area 20 mm², regurgitant volume 30 mL).

DISCUSSION

Dilated cardiomyopathy secondary to postoperative metabolic disturbances following thyroid or parathyroid surgery is a rare but clinically relevant entity.

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In our case, the occurrence of severe DCM complicated by functional mitral regurgitation after total thyroidectomy and parathyroidectomy illustrates the complex cardiovascular consequences of prolonged hypothyroidism and hypocalcemia.

1. Pathophysiological mechanisms

Thyroid hormones exert positive chronotropic and inotropic effects on the myocardium. They regulate the expression of contractile proteins (α-myosin heavy chain, SERCA2, phospholamban) and promote diastolic relaxation and ventricular compliance. Hypothyroidism reduces these effects, leading to bradycardia, decreased cardiac output, increased systemic vascular resistance, and, over time, left ventricular dilation due to volume overload and altered myocardial metabolism (1).

Concomitantly, parathyroid hormone deficiency results in persistent hypocalcemia, impairing myocardial contraction by reducing intracellular calcium influx and altering excitation–contraction coupling (2). Severe hypocalcemia may cause acute or chronic heart failure, which is reversible after correction (3).

Thus, the coexistence of hypothyroidism and hypocalcemia synergistically exacerbates myocardial dysfunction and promotes the development of severe DCM, as observed in our patient.

2. Echocardiographic and CMR findings

Echocardiography is the first-line imaging modality to assess systolic dysfunction severity and detect functional valvular disease, such as mitral regurgitation secondary to left ventricular dilation. In our case, ventricular dilation led to severe functional mitral regurgitation, further worsening hemodynamic status.

CMR plays a key role in differentiating metabolic DCM from ischemic or inflammatory cardiomyopathies. The absence of late gadolinium enhancement is typical of a non-necrotic, reversible process (4). In our patient, the absence of enhancement confirmed the functional and potentially reversible nature of myocardial involvement.

3. Reversibility with replacement therapy

The favorable evolution under hormonal and calcium replacement therapy constitutes a strong diagnostic argument. Several similar cases have reported complete normalization of left ventricular function after correction of endocrine abnormalities.

Sharma et al. described recovery of LVEF from 25% to 60% after six months of levothyroxine therapy (5). Similarly, Dorr et al. reported dramatic improvement following calcium supplementation in hypocalcemia-induced DCM (6). In a series published by Biondi and Cooper, correction of hypothyroidism led to hemodynamic improvement within weeks, associated with reduced ventricular dilation (7,8).

These data confirm the potentially reversible nature of metabolically induced DCM, provided that correction is prompt and adequate.

4. Prognostic value and clinical implications

The prognosis of metabolic DCM is generally favorable when diagnosis is made early. Delayed correction of hormonal and electrolyte abnormalities, however, may result in irreversible myocardial fibrosis and persistent systolic dysfunction. This underscores the importance of close endocrinological follow-up after thyroidectomy and parathyroidectomy.

Moreover, severe functional mitral regurgitation, as observed in our case, should be considered a mechanical consequence of ventricular dilation. It often regresses in parallel with myocardial recovery, thereby avoiding unnecessary valvular surgery.

CONCLUSION

Metabolic dilated cardiomyopathy should be considered in any case of unexplained heart failure, particularly following thyroid or parathyroid surgery. Prompt correction of hypothyroidism and hypocalcemia allows complete recovery of cardiac function. This case highlights the necessity of close cardiologic and endocrinologic follow-up after thyroidectomy and parathyroidectomy.

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