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Myxoedematous tamponade as initial presentation of Hashimoto's thyroiditis

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SUMMARY

Cardiac tamponade as the initial presentation of hypothyroidism is extremely rare. We report the case of a 48-year-old man admitted for acute respiratory distress, with cardiac ultrasound showing compressive pericardial effusion. Percutaneous pericardiocentesis was performed leading to a rapid clinical improvement. Laboratory tests confirmed severe hypothyroidism related to Hashimoto's disease. Despite hormone replacement therapy, pericardial effusion recurred after 3 weeks, requiring surgical drainage. Pericardial histology highlighted slight chronic fibrous pericarditis. The cardiac ultrasound scan performed 4 months later showed a well-tolerated chronic pericardial effusion. In conclusion, hypothyroidism should be suspected in case of cardiac tamponade especially in the absence of tachycardia, or in winter when myxoedema is prone to decompensation. Prognosis is generally good under hormone replacement therapy but ultrasound monitoring should be carried out at least until euthyroidism is achieved.

BACKGROUND

Hypothyroidism is a common endocrine disorder that can involve many organs. It can be asymptomatic, but its complications can be fatal. Hypothyroidism-related serous fluid effusion is well documented (pericardium, pleura and peritoneum)[1] and is due to the increased permeability of capillaries to albumin.[2] Prevalence of pericardial effusion in hypothyroidism is relatively low (3%–6%) and cardiac tamponade as the initial presentation of hypothyroidism is rare[3] due to slow fluid accumulation and pericardial distensibility.[4] Intercurrent viral infection or cold conditions, both of which frequently occurred in winter, can explain decompensated myxoedema thus precipitating tamponade evolution.[5] We report the case of a patient presenting this serious complication of undiagnosed severe hypothyroidism.

CASE PRESENTATION

A 45-year-old man with a history of diabetes, dyslipidaemia and hypertension was admitted to the intensive care unit (ICU) in December 2019 with acute respiratory distress after collapsing while driving his car. He had also mid-thoracic pain associated with rapidly progressive dyspnoea over a period of several weeks. Questioning revealed increasing sluggishness, weight gain and also facial oedema progressing over 3 months. On admission to the ICU, the patient was in respiratory distress, sitting on his bed, with oxygen saturation <85%, a respiratory rate of 45 breaths/min and blood pressure of 90/60 mm Hg. The clinical examination found a slight goitre on palpation; without compressive sign. In addition to the signs of hypothyroidism, the patient presented a clinical condition of hydrops, with swelling leg; no signs of ophthalmopathy or vitiligo. The ECG showed hypervoltage QRS complexes with repolarisation disorder in the anterolateral territory (figure 1). The heart rate was 70 beats/min. The chest X-ray showed an increased cardiothoracic index (figure 2). Transthoracic cardiac echocardiogram (TTE) revealed circumferential fibrinous pericardial effusion, 30 mm thickness, with the right atrial and right ventricular flaps (figures 3 and 4). Left pleural effusion was also apparent. Left ventricular ejection fraction was assessed at 35% and was indicative of hypertrophic cardiomyopathy. In view of the patient's poor clinical tolerance, emergency percutaneous pericardiocentesis was performed via subxiphoidal biopsy under echographic/fluoroscopic guidance using a 'Peel Away SJM' 10.5F drain. Collection of 1000 mL of a pale yellow fluid led to rapid clinical improvement.

BIOLOGICAL FINDINGS AND IMAGING

Laboratory test results highlighted signs of severe hypothyroidism with thyroid-stimulating hormone (TSH) at 93 µIU/mL, low FT3 (2.23 pmol/L) and FT4 (<5.41 pmol/L) with highly positive antithyroid peroxidase antibodies (578 IU/mL) due to Hashimoto's thyroiditis. Albumin rate (43 G/L) and renal function (creatinine clearance: 62 mL/min, urea concentration of 7.1 mmol/L) were normal. TSH receptor antibody and antineutrophil cytoplasmic antibodies were negative. There was evidence of influenzae infection with positive nasopharyngeal PCR. Cytological analysis of the pericardial fluid revealed non-inflammatory haemorrhagic fluid, with 243 nucleated elements/mm^3, and no malignant cells. Troponin levels of 228 pg/mL were recorded together with brain natriuretic

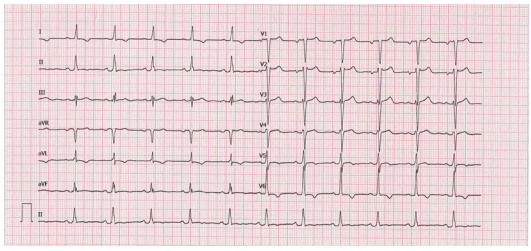

Figure 1 ECG at admission with hypervoltage QRS complexes and repolarisation disorder in the anterolateral territory.

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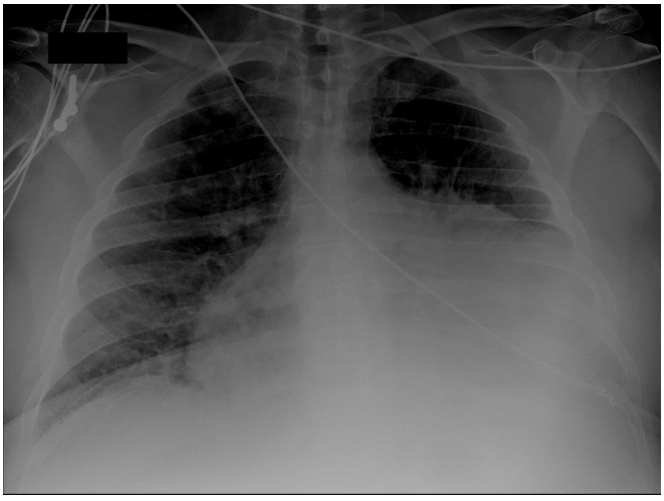

Figure 2 Chest X-ray at admission with increased cardiothoracic index.

peptide levels of 630 pg/mL and C reactive protein at 1 mg/dL. The blood count was normal. With regard to the hypertrophic cardiomyopathy assessment, serum protein electrophoresis was normal and there was no evidence of Bence Jones proteinuria. The patient tested negative for Fabry's disease.

Regarding imaging, the ultrasound scan of the thyroid gland revealed thyroiditis compatible with autoimmune origin. Coronary angiography secondary to altered left ventricular ejection was normal. Cardiac MRI confirmed hypertrophic cardiomyopathy with septum and lateral wall thicknesses of up to 26 mm and 21 mm, respectively, with no enhancement after gadolinium injection (figure 5). The biphosphonate myocardial scintigraphy did not show signs of amylosis.

TREATMENT

Post-pericardiocentesis, levothyroxine treatment was started at the initial dosage of 25 µg/day for 4 days, with upward titration of 25 µg every 4 days, up to a final dose of 75 µg.

OUTCOME AND FOLLOW-UP

The cardiac ultrasound scan performed on discharge confirmed an estimated effusion of 200 mL. Despite a decrease in TSH, effusion increased on a weekly basis with evidence of poor

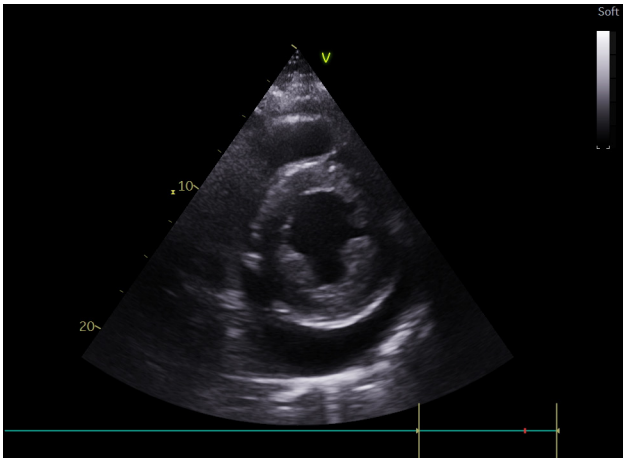

Figure 3 Transthoracic cardiac ultrasound with circumferential fibrinous pericardial effusion (parasternal short axis view).

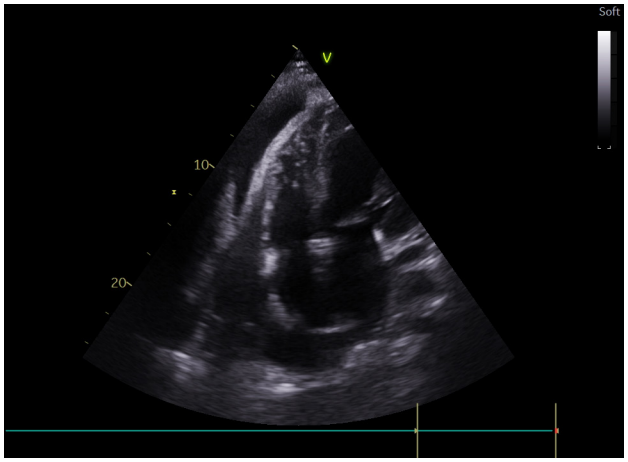

Figure 4 Transthoracic cardiac ultrasound with circumferential fibrinous pericardial effusion (apical four-chamber view).

tolerability on the TTE. The patient was therefore rehospitalised for surgical drainage associated with pericardial biopsy indicative of slight chronic fibrous pericarditis without inflammation or malignancy. A positron emission tomography scan did not detect any suspicious hypermetabolic activity, in particular on the pericardium. The patient was subsequently referred to a cardiac rehabilitation unit. Pericardial effusion remained stable after euthyroidism was achieved: the 3-month follow-up ultrasound scan showed stable circumferential effusion of 10 mm around the right atria, and 13 mm around the left ventricle, with no sign of compression. TSH was 5.8 µIU/mL.

DISCUSSION

This clinical case reminds us that myxoedematous tamponade is an entirely diagnostic entity. Indeed, undiagnosed severe hypothyroidism can present as an inaugural tamponade. As our patient's case shows, this rare and serious complication may be exacerbated during the winter months by the increased prevalence of viral infections and cold conditions that can precipitate myxoedema.[5 6] In the presence of tamponade, myxoedema should be suspected in patients without sinus tachycardia.[7] Indeed, patients with hypothyroidism frequently present bradycardia because their heart rate is unable to accelerate at a normal

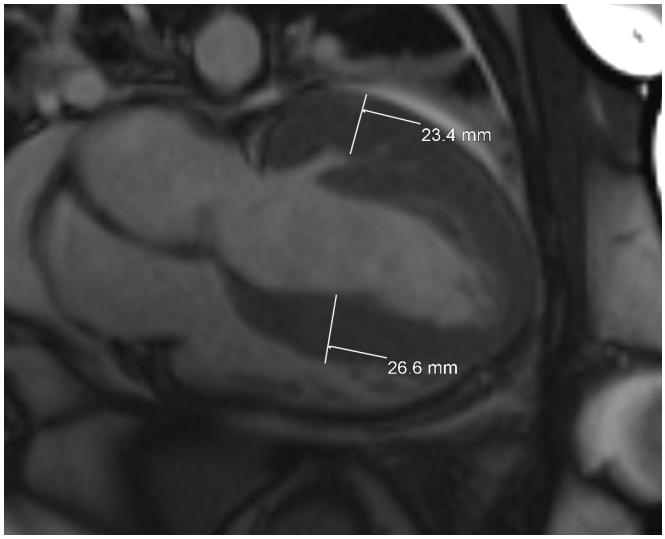

Figure 5 Cardiac MRI with hypertrophic cardiomyopathy.

level, although the right chambers are compressed.[8] ECG presentation was even more atypical in this patient with hypertrophic heart disease leading to hypervoltage of QRS complexes, whereas tamponade is usually associated with diffuse microvoltage. Syndromic association between autoimmune hypothyroidism and hypertrophic heart disease was ruled out following a multidisciplinary discussion. Myxoedematous tamponade is a medical emergency. After conducting bedside TTE, the first treatment is pericardiocentesis followed by hormone replacement therapy. In high-risk patients with cardiovascular disease, an ischaemia assessment may be justified when starting thyroid hormone therapy given the increased risk of atherosclerosis associated with hypothyroidism-related hypercholesterolaemia.[8] The prognosis is good after hormone replacement therapy. Pericardial effusion generally resolves in 2–12 months.[9 10] However, there is a significant risk of recurrent effusion in the first few weeks if euthyroidism is not achieved, as observed in our patient. It is therefore important to ensure stringent ultrasound monitoring until thyroid function returns to normal. In addition, the presence of chronic effusion justifies prolonged follow-up to ensure that the condition does not progress to constrictive chronic pericarditis.

Learning points

- Classical symptoms of tamponade may be absent in patients with hypothyroidism.
- Hypothyroidism should be suspected when cardiac tamponade is not associated with sinus tachycardia, especially in winter.
- Prognosis is generally good under hormone replacement therapy.
- Ultrasound monitoring should be carried out at least until euthyroidism is achieved given the risk of recurrent effusion.

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