MASSIVE PERICARDIAL EFFUSION ASSOCIATED WITH HYPOTIROIDISM

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MASSIVE PERICARDIAL EFFUSION ASSOCIATED WITH HYPOTIROIDISM (Abstract): The diagnosis of hypothyroidism is difficult because hypothyroidism in adults and especially the elderly, classic, has an insidious onset with a range of nonspecific symptoms which may delay diagnosis for months or even years. Old age seems to represent trigger factor for autoimmune diseases, including hypothyroidism. Clinical features in hypothyroidism, such as weight gain, fatigue, intolerance of the cold, constipation, dry skin, edema and muscle weakness, and decreased osteo-tendinous reflexes are usually subtle and can be overlooked. Thyroid dysfunction may be associated with a negative impact on the cardiovascular system. Pericardial, pleural and peritoneal effusions are common findings in hypothyroidism. This case report represents a typical primary hypothyroidism (autoimmune) and shows the clinical features of this disease. Basically we talked about a severe myxedema with the involvement of internal organs in an elderly woman and the euthyroidism restoration, under thyroid replacement therapy, was correlated with the clinical improvement and cardiovascular and neurological status, with radiographic remission and regression to extinction of pericardial effusion at repeated echocardiographic evaluations. Keywords: AUTOIMMUNE THYROIDITIS, HYPOTHYROIDISM, PERICARDIAL EFFUSION, LEVOTHYROXINE.

Thyroid dysfunction may be associated with a negative impact on the cardiovascular system. The prevalence of hypothyroidism increases with age (1,4), especially in women (2,3) and is often difficult to diagnose clinically in elderly patients (1,5). Old age seems to represent a trigger factor for autoimmune diseases, including hypothyroidism (3).

Incidence of pericardial effusion in hypothyroidism is related to the severity and duration of disease, being reported to be in approximately one-third of the cases, as low as 3% in the early mild stage and as high as 80% when myxedema is present (6).

Pericardial, pleural and peritoneal effusions are common findings in hypothyroidism. A few cases have been reported in which pericardial fluid evolved into tamponade. A variety of conditions can cause pericardial effusion. Early recognition of etiologic factors is important to improve outcome and avoid unnecessary invasive investigations. Once a diagnosis set, pericardial effusion slowly regresses and eventually disappears after several months of
replacement therapy. We here present the case of a patient with massive pericardial effusion associated with hypothyroidism, which responded to levothyroxine replacement therapy after 6 months.

**CASE REPORT**

A 84-year-old woman presented at the emergency department of our hospital, with profound dyspnea, pronounced asthenia, somnolence, vertigo, paresthesia, symptoms emphasized about 2 weeks prior hospitalization, in terms of ambulatory detection by echocardiographic exploration of an important pericardial effusion (fig. 1).

The patient has husky voice, slow speech, and periods of disorientation in time and space and visual hallucinations. Physical examination revealed cool and dry skin, eyelid edema and edema of the extremities, faint heart sounds with a heart rate of 54 beats / minute, TA= 125/88 mmHg, bilateral basal crackles and reduced vesicular murmur, with a respiratory rate of 20 breaths / min and a temperature of 35.4 °C, coprostasis. Neurological examination revealed delayed deep tendon reflexes, dysdiadochokinesia, depressive mood.

![Fig. 1. Ultrasound 2D parasternal long axis section, the objectification of pericardial fluid disposed in the posterior wall of the left ventricle in diastole of 18.31 mm.](image)

Laboratory tests revealed blood count and C-reactive protein (CRP) values within normal limits, hypercholesterolemia 373 mg/dl, an LDL-cholesterol of 250 mg/dl, HDL cholesterol 47 mg/dl and triglycerides 193 mg/dl.

An electrocardiogram revealed sinus bradycardia, with a low P wave and QRS complexes and first degree atrioventricular block (PQ was 0.24 sec). Chest radiograph identified cardiac enlargement and bilateral pleural effusion.

The echocardiogram revealed massive pericardial effusion, disposed circumferentially with fibrin deposits in fluid or on epicardial surface, with measurement of the
pericardium separation foils, with a maximum of 28 mm and an approximate estimated liquid volume 600 ml, without signs of tamponade (fig. 1, 2, 3).

**Fig. 2.** Ultrasound 2D parasternal short shaft section outlining the pericardial fluid in large quantities ordered in the posterior wall of the left ventricle, with dimensions of 21.17 mm, containing fibrin deposits.

**Fig. 3.** Ultrasound 2D apical 4-room section, which highlights the pericardial fluid disposed in the anterolateral wall of the left ventricle of 19.25 mm and the free wall of the right atrium of 15.33 mm without ultrasound resemble tamponade.
A computed tomography (CT) scan of the chest revealed no evidence for acute lung injury, aortic dissection, or tumor lesions. Infectious, neoplastic, traumatic, uremic, post heart attack pericarditis or aortic dissection being excluded, thyroid dysfunction was taken into account and, in this regard, we dose serum thyroid-stimulating hormone (TSH) was 96.8 μUI/ml, free T3 2.9 ng/dl, free T4 0.3 pg/ml, anti-peroxidase (TPO) 372U/ml, and anti-thyroglobulin 834U/ml with a thyroid ultrasound without changes of position, size or structure. Once diagnosis settled, in the absence of signs of tamponade, pericardiocentesis was not performed (fig. 3).

Treatment was started with low doses of levothyroxine initially with 12.5μg and increased at 25μg after the first week and fitting according to thyroidian parameters. The patient was evaluated at 1, 3 and 6 months.

Restoring euthyroidism was correlated with clinical improvement and cardiovascular and neurological status, radiographic remission and regression to extinction of pericardial effusion on repeated echocardiogram.

DISCUSSION

Chronic autoimmune thyroiditis may eventually cause hypothyroidism, mainly via destruction of thyrocytes. The serological hallmark of Hashimoto’s disease is the presence of high titers of thyroid peroxidase (TPO) auto antibodies. Elevated titers of anti-TPO simply show the presence of an autoimmune reaction against the thyroid and may be associated with hypothyroidism (most common) as well as with hyperthyroidism.

The diagnosis of hypothyroidism is difficult because hypothyroidism in adults and especially the elderly, classic, has an insidious onset with a range of nonspecific symptoms which may delay diagnosis for months or even years. Clinical features in hypothyroidism, such as weight gain, fatigue, intolerance of the cold, constipation, dry skin, oedema and muscle weakness, decreased osteo-tendinous reflexes (due to thyroid hormone action on the nervous system) are usually subtle and can be overlooked (7). The diagnosis is rarely made solely on clinical grounds, especially the elderly, who are often faced with either depression or dementia.

The occurrence of pericardial effusion in hypothyroidism appears to be related to the severity and duration of disease. The incidence of pericardial effusion is reported as 3% in the early mild stage reaching up to 80% of cases when myxedema is present (6).

In contrast to the frequent occurrence of pericardial effusion in patients with hypothyroidism, massive pericardial effusions are rarely associated with tamponade. Cardiac tamponade in patients diagnosed with hypothyroidism is probably as rare as it is due to pericardial distensability and the slow accumulation of fluid, allowing significant fluid accumulation without hemodynamic compromise (8). A variety of medical conditions, including malignant tumors, may lead to pericardial effusion. The presumptive mechanism underlying exudative pericardial effusions is extravasation of hygroscopic mucopolysaccharides into the pericardial space along with increased capillary permeability, decreased lymphatic drainage, and increased retention of salt and water (8). Due to its insidious onset, a pericardial effusion secondary to hypothyroidism frequently occurs without hemodynamic change, and is apparent only on echocardiography (9). Such findings in
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an asymptomatic elderly patient can be misleading and may lead to unnecessarily invasive procedures such as pericardiocentesis (9). Using echocardiography it can be detected a pericardial effusion with a sensitivity of 96%, specificity of 98% and overall accuracy of 97.5% (10). Suggestive clinical signs of hypothyroidism and thyroid function evaluation in patients are crucial for an early diagnosis.

Therefore, hypothyroidism should be excluded in all patients with unexplained pericardial effusion etiology, not only in patients with clinically evident hypothyroidism or elder. Finally, once the diagnosis has been established, treatment with thyroid replacement hormones usually leads to the resolution of the effusion over 2–12 months without sequel.

CONCLUSIONS

This case report represents a typical primary hypothyroidism (autoimmune) and shows all the clinical aspects of these diseases. Basically we talked about a severe myxedema with the involvement of internal organs in an elderly woman and the euthyroidism restoration, under thyroid replacement therapy, was correlated with the clinical improvement, cardiovascular and neurological status, with radiographic remission and regression to extinction of pericardial effusion, at 6 months without sequela.

REFERENCES