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Pericardial diseases in patients with hypothyroidism

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Overview

Incidence And Prevalence: Pericardial effusion

The incidence of pericardial effusion due to hypothyroidism ranges from 3% to 37% and most commonly found in states of severe hypothyroidism. Fortunately, a minority (less than 30%) of the effusions are large.

Cardiac tamponade and acute pericarditis

No study to date has determined the prevalence or incidence of cardiac tamponade or acute pericarditis in hypothyroidism.

Pathophysiology: Pericardial effusion I

It is well known that myxoedema is the result of the accumulation of acid mucopolysaccharides in the skin causing water retention. However, this mechanism does not explain the oedema and effusion caused by severe hypothyroidism outside of the skin, as there is a lack of acid mucopolysaccharides buildup in many organs like the heart and pericardial space. Effusions and oedema in these organs are attributed to increased vascular permeability to albumin.

Pericardial effusion II

Usually, the pericardium is impermeable to proteins, and the physiological pericardial fluid is formed by a fluid transudation at the arterial end of the epicardial capillaries. Part of this fluid will be reabsorbed at the venous end of those capillaries and the rest drained by the lymphatic vessels of the parietal pericardium. In hypothyroidism, the increased albumin permeability of the pericardial capillaries along with decreased albumin drainage into the lymphatic vessels increases the pericardial colloid pressure.

Pericardial effusion III

The aetiology of the increased albumin permeability is proposed to be related to mastocyte release of histamines induced by the low thyroid state or by the direct effect of hypothyroidism on the endothelial layers of pericardial capillaries. The lymphatic drainage is proposed to be impaired due to pulmonary hypertension induced by hypothyroidism and subsequent elevation of right heart pressures. An additional theory for the decreased lymphatic drainage is hypothyroidism induced decrease in circulating catecholamines (whose role is to increase the flow of the lymph).

Cardiac tamponade

The occurrence of pericardial tamponade due to hypothyroidism is not common but has been reported to occur in severe hypothyroidism, most likely due to an increase in albumin capillary leak and impairment in lymphatic drainage resulting in a significant accumulation of fluids over time and resulting in pericardial tamponade.

Acute pericarditis

At this time, there is not enough data to suggest/refute that hypothyroidism can directly cause acute pericarditis and further studies are needed.

The role of autoimmunity in pericardial diseases caused by hypothyroidism

There does not seem to be an autoimmune explanation for the pericardial diseases in hypothyroidism for two reasons. First, pericardial diseases occur in autoimmune and non-autoimmune hypothyroidism alike: they have been reported in autoimmune thyroiditis, as well as in hypothyroidism due to iodine deficiency and postsurgical hypothyroidism. Second, to date, no specific antibodies have been proven to be the culprit of pericardial diseases in hypothyroidism.

Clinical Manifestation I

Most pericardial effusions, even if large (up to 30% of hypothyroid effusions), are asymptomatic. In hypothyroidism, the pericardial fluid accumulates slowly allowing the pericardium to accommodate via stretching and thus tolerate higher fluid volumes without a significant rise in pericardial pressures. This phenomenon is best demonstrated by a J-shaped pressure–volume curve.

Clinical Manifestation II

As the pericardial fluid slowly accumulates, the intrapericardial pressures remain low; however, in the rare instance that the pericardial stretch limit is reached, the pressures in the pericardium will rise at a steeper curve even with smaller increases in pericardial fluid volume and thus cardiac tamponade may occur. Fortunately, cardiac tamponade is infrequent in hypothyroidism.

Clinical Manifestation III

The clinical manifestations of cardiac tamponade include pulsus paradoxus, increased jugular venous pressure, muffled heart sounds and even haemodynamic instability. Surprisingly, many case reports have described atypical presentations of tamponade when caused by hypothyroidism such as syncope, weakness, shortness of breath, lower limb oedema or even gastrointestinal manifestations.

Diagnosis

Hypothyroidism can be deemed the aetiology of pericardial effusion or cardiac tamponade if a high TSH level has been found, after excluding other secondary causes like a neoplastic, bacterial or an inflammatory process.

Electrocardiography I

Sinus bradycardia is traditionally associated with severe untreated hypothyroidism. A statistically significant lower heart rate was found in hypothyroid patients with pericardial effusions when compared with euthyroid patients with pericardial effusions.

Electrocardiography II

Thus, hypothyroidism should be considered in cases of tamponade in the absence of rapid heart rate. Low voltage EKG was seen in only 42%–50% of patients with pericardial effusion due to hypothyroidism.
Low thyroid hormones levels by themselves might induce a low voltage on the EKG independently from the presence of pericardial effusion.
T-wave flattening and inversions have been commonly seen in pericardial effusions due to hypothyroidism.

Imaging:Chest X-ray

Cardiomegaly might be indicative of a pericardial effusion but also lacks sensitivity and specificity with a false negative rate of around 30% when hypothyroidism is the aetiology

Imaging:Echocardiography

Echocardiography remains the most important test in the diagnosis of pericardial effusions and pericardial tamponade and must be considered in the first 24hours of acute pericarditis.

Imaging:Other imaging

Cardiac CT and cardiac MRI can be helpful in the evaluation of pericardial effusions as they can accurately estimate the amount of pericardial fluid present, evaluate the presence and extent of pericardial thickening and possibly distinguish exudates from transudates and inflammatory from malignant effusions.

Laboratory tests

TSH
Free T4
Creatine Kinase
CRP

Treatment I

According to the 2014 American Thyroid Association guidelines for the treatment of hypothyroidism, levothyroxine is the drug of choice to treat hypothyroidism. The clinical situation, the severity of hypothyroidism (ie, extent of TSH elevation), the severity of underlying illness, the age of the patients and their comorbidities need to be considered when deciding on a dose of levothyroxine replacement, particularly in the acute setting.

Treatment II

Generally, levothyroxine should be started at 1.6µg/ kg body weight per day in overt hypothyroid patients with a TSH more than 10 uU/mL and lower doses (25–50µg per day) if TSH between 5 uU/mL and 10 uU/mL or in subclinical hypothyroidism.

Treatment III

However, extra caution must be used to avoid inducing iatrogenic hyperthyroidism that can be detrimental to the elderly and patients with a cardiac history. Thus, a lower dose may need to be considered in patients with coronary artery disease (12.5–25µg per day) and in the elderly (up to 50µg per day)

Treatment VI

The principal risks of iatrogenic hyperthyroidism reside in an increased risk of arrhythmias like atrial fibrillation, acute coronary syndromes and osteoporosis. TSH level must be rechecked in 4–6 weeks and adjusted by increments of 12.5–25µg per day if needed. This should be repeated until the TSH level is within the normal reference range (0.4–4.0 mIU/L).

Pericardial effusion

Achieving a euthyroid state is the definitive treatment and in most cases the only treatment that is needed for pericardial effusions due to hypothyroidism. The effusions may take several months to resolve completely.

Cardiac tamponade

Wang et al have demonstrated that giving thyroid replacement therapy without pericardial drainage can be done in some cases of pericardial tamponade caused by hypothyroidism in haemodynamically stable echocardiographic tamponade physiology without clinical cardiac tamponade (mainly without pulsus paradoxus).

Acute pericarditis

Non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine should be initiated for patients with acute pericarditis. Additionally, TSH must be obtained, and hypothyroidism should be treated if present. Anti-inflammatory medications should be started immediately and continued until the patient becomes asymptomatic and the inflammatory markers normalise.

The End