

## Case Report

# An unusual case of pericardial tamponade in primary hypothyroidism

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### ABSTRACT

Pericardial effusion is not so uncommon in hypothyroidism, but pericardial tamponade is very rare entity. Hypothyroidism complicated by pericardial tamponade is very rarely referenced in the medical literature. Here we report a rare case of a 70 year old female, who presented with breathlessness and was found to have hypothyroidism with large pericardial effusion complicating into pericardial tamponade. Management included urgent pericardiocentesis followed by L-thyroxine supplementation.

**Keywords:** Hypothyroidism, L-thyroxine, Pericardial tamponade, Pericardial effusion, Pericardiocentesis

## INTRODUCTION

Pericardial effusion due to hypothyroidism was reported as early as 1918 and subsequently in 1925.<sup>1</sup> The total incidence of pericardial effusion secondary to hypothyroidism varies in different studies from 30% to 80%.<sup>2</sup> Cardiac tamponade as a complication of hypothyroidism is very rare.<sup>3</sup> Until 1992, there were less than 30 cases described and even more recently there are only few cases found in the world literature. This low incidence is most likely due to slow accumulation of fluid and gradual pericardial distention.<sup>4</sup>

Hypothyroidism is characterized by low metabolic demands and therefore, despite a depressed cardiac contractility and cardiac output, cardiac function remains sufficient to sustain the workload imposed on the heart. Another feature that distinguishes cardiac tamponade caused by hypothyroidism from the other causes of tamponade is the absence of sinus tachycardia as a mechanism to maintain cardiac output.<sup>5</sup> Here we report a case of 70 years old female who presented with large pericardial effusion complicating into life threatening cardiac tamponade, questioning the need of 2D echo as a

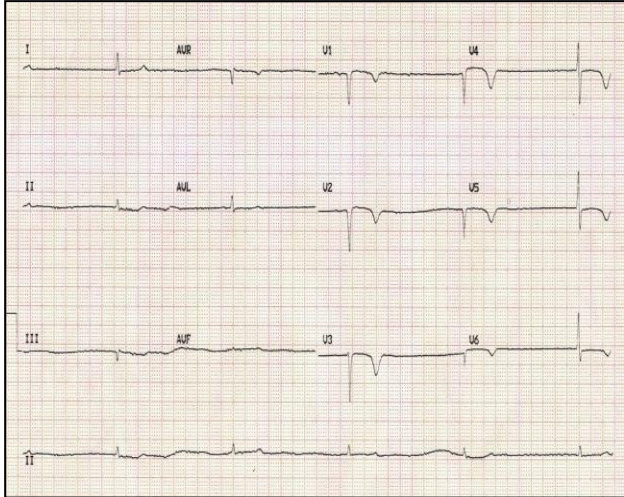
routine work up in hypothyroid patients at high risk of cardiac complications.

## CASE REPORT

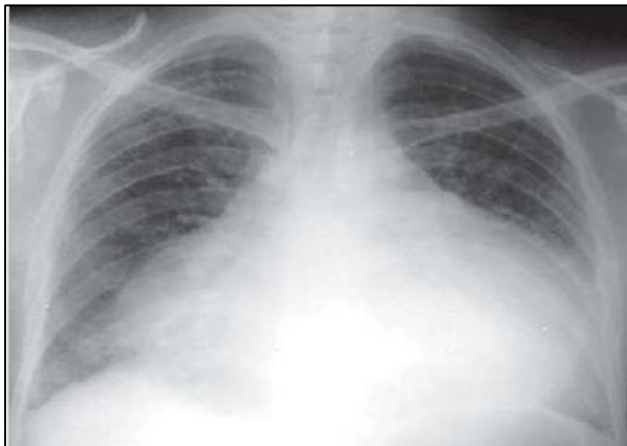
A 70-year-old female presented to the emergency department with shortness of breath for 2 weeks. Patient was asymptomatic 2 months back when she developed distension of abdomen and swelling of whole body, reduced urine output and weight gain. She had no significant past history or drug history. On admission vital signs were pulse 54 bpm, blood pressure 120/70 mm Hg, respiratory rate 18 breaths/min. Body mass index was 24.76 kg/m<sup>2</sup>. Physical examination showed a confused female with swollen face and extremities, dry skin, mild pallor, non-pitting edema of extremities, thyroid gland was non-tender and no nodules palpable. Patient was hypothermic (35.8°C). Cardiac sounds were faint and distant with raised jugular venous pulsation. Deep tendon reflexes were delayed.

Laboratory analysis revealed: Hb 9.9 g/dl, Rbs 126 mg/dl, blood urea 24 mg%, serum creatinine 1.1 mg%, lipid profile: total cholesterol 269 mg/dl, triglycerides

220 mg/dl, HDL cholesterol 44 mg/dl. Serum sodium 142 meq/L, serum potassium 3.8 meq/L. viral markers were negative. ECG showed low voltage, sinus bradycardia (Figure 1). Chest x-ray showed cardiomegaly with globular silhouette ‘water bottle configuration’ (Figure 2).



**Figure 1: ECG of the patient at admission with low voltage complexes, bradycardia and t wave inversion in precordial leads.**

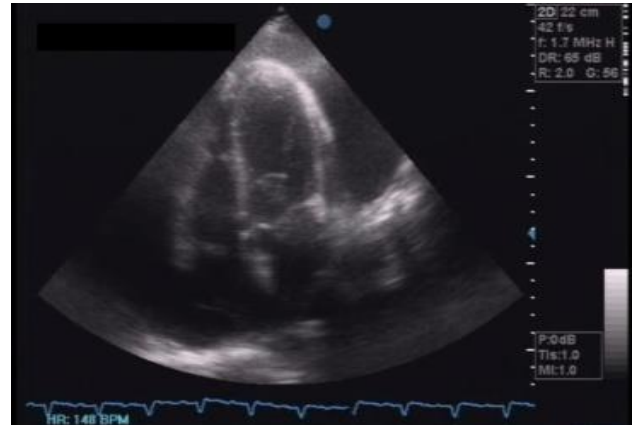


**Figure 2: Chest x-ray suggestive of cardiomegaly (water bottle configuration).**

On 2<sup>nd</sup> day of admission patient became drowsy, hypotensive (systolic BP 70 mm of Hg), with ECG suggestive of low voltage complex with electrical alternans. Patient was immediately shifted to intensive care unit and put on inotropic support and fluid resuscitation.

Bedside 2D echo was done which was suggestive of massive pericardial effusion, small sized all four chambers and characteristic right ventricular diastolic collapse. There was swinging motion of heart within large effusion, prominent respiratory alteration of right

ventricular dimension with RA and RV collapse during diastole (Figure 3).



**Figure 3: Large pericardial effusion with small sized cardiac chambers with characteristic right ventricular diastolic collapse.**

Sub-xiphoid pericardiocentesis was performed under 2D echo guidance and 300cc of yellowish fluid was aspirated which resulted in dramatic improvement in blood pressure and general condition. Pericardial fluid analysis showed sugar 152 mg%, protein 6.1 g%, RBC's 180 cells/mm<sup>3</sup>, WBC's <5 cells/mm<sup>3</sup> with predominant lymphocytes. Thyroid function test revealed T3 0.195, T4 0.485 and TSH 35.91. Diagnosis of hypothyroidism was made and cardiac tamponade secondary to hypothyroidism was established as the cause. Patient was put on l-thyroxine in a dose of 100 mcg/day. There was gradual improvement in all her symptoms.

**DISCUSSION**

Cardiac tamponade rarely occurs with severe hypothyroidism and myxedema.<sup>5</sup> Our case is unique due to the fact that patient presented with cardiac tamponade as a complication of hypothyroidism.

The normal pericardial space is filled by serous fluid which is not more than 15 to 30 mL.<sup>6</sup> The layer visceral is composed of mesothelial cells and the parietal is formed of collagen fibers and a small amount of elastin.<sup>7</sup> The pericardium limits acute cardiac dilation and enhances the mechanical interaction of the cardiac chambers as a result of its relatively inelastic properties. In response to long-standing stress, the pericardium dilates, shifting the pericardial pressure-volume relation substantially to the right.<sup>7</sup>

When there is slow accumulation of fluid, the parietal pericardium compliance increases. The increasing amounts of fluid increase at the same time the intrapericardial pressure, while the central venous pressure responds by increasing to maintain a gradient that allows cardiac filling.

When the pericardial compliance reaches its maximal capacity, the intrapericardial pressure equalizes with the right ventricular diastolic pressure and later with the left. At this point, the cardiac output drops and the circulation is maintained by an increase in heart rate, contractility and peripheral vasoconstriction.<sup>6</sup> If the fluid accumulates slowly, large amounts can occupy the space but in case of rapid fluid accumulation, the compliance capacity is exceeded. The amount of fluid in the pericardial effusions associated with hypothyroidism can be as large as 5 - 6 L.<sup>8</sup> Inadequate lymphatic drainage may be the mechanism by which the exudates are produced in the serous cavities of patient with hypothyroidism.<sup>9</sup>

The primary phenomenon in tamponade is a compression of all cardiac chambers after the pericardial content reaches the limit of pericardial reserve volume. With smaller cardiac chambers, the myocardial diastolic compliance is reduced and cardiac inflow becomes limited, ultimately equalizing mean diastolic pericardial and chamber pressures. This equalization of pressures is the hall-mark of cardiac tamponade.<sup>10</sup> Clinical features depend on the rapidity of accumulation of fluid in the pericardial space and include chest pain and dizziness. Classic findings include Beck's triad (raised venous pressure, arterial hypotension and distant heart sounds).<sup>7,10</sup>

Bradycardia and systemic hypertension, with narrow pulse pressure and slightly increased mean arterial pressure, and some degree of exercise impairment are the most common findings in patients with overt hypothyroidism. Systemic hypertension in overt hypothyroidism is likely due to the remarkable increase in peripheral vascular resistance and increase in arterial stiffness, likely resulting from myxedema of the arterial wall.<sup>11</sup>

In hypothyroidism there is an increased capillary permeability and impaired lymphatic drainage with protein leakage into the interstitial space, resulting in pericardial effusion. Furthermore there is increased salt and water retention.<sup>4,8</sup> Bradycardia in hypothyroid cardiac tamponade has been evident in several studies and is suggesting to be related to a decreased sympathetic activity, but indirect measurements of sympathetic activity showed that is elevated. There are also some evidence of blunted sympathetic excitatory and tachycardia response to hypotension and depressed arterial baroreflex with elevated dependence on the resting sympathetic tone that can explain the presence of bradycardia.<sup>12</sup> The fluid characteristic in hypothyroid cardiac tamponade is typically an effusion, with a color that goes from clear to golden hue, with predominance of lymphocytes and high protein content, which was evident in our case.

Treatment depends on the hemodynamic state of the patient. Removal of even a small amount of pericardial fluid (about 50 ml) produces considerable hemodynamic

and symptomatic improvement because of the steep pericardial pressure volume relationship. A majority of the cases will require surgical drainage procedure. Therapeutic measures that reduce venous filling pressures or effective cardiac output should be avoided.<sup>13</sup> Pericardiocentesis is well tolerated by patients, is a quick procedure and can be performed even in unstable patients. The success rate is about 97% and the preferred method of approach is the sub-xiphoid region.<sup>14</sup>

## CONCLUSION

Hypothyroidism with cardiac tamponade as a presenting symptom is a rare condition. Cardiac tamponade is a life-threatening condition that clinicians should have high degree of suspect in relevant case scenario. Supplementation with l-throxine is sufficient for small pericardial effusions in hypothyroidism patients. However, pericardiocentesis becomes a lifesaving procedure if pericardial tamponade develops. However, this may be questionable that whether or not 2D echo should be included in routine workup of patients with hypothyroidism. High index of suspicion must be kept in hypothyroid patients at high risk of cardiac complications. This may require further large scale studies to reevaluate the need for 2D echo in workup of hypothyroid patients.

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