

Asymptomatic Hypothyroidism with Concomitant Viral Pericarditis Presenting as Acute Cardiac Tamponade

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Abstract

A case of a young woman who presented to emergency department with severe abdominal pain and shock is reported here. The patient was found to have pericardial tamponade due to massive pericardial effusion. On further evaluation, the etiology of this effusion was found to be secondary to hypothyroidism with concomitant acute viral pericarditis leading to a fulminant tamponade. The presentation, differential diagnosis and management of pericardial effusion and tamponade secondary to hypothyroidism and viral pericarditis are discussed. The diagnosis of hypothyroidism in conjunction with acute viral pericarditis should be considered in patients presenting with unexplained pericardial effusion and tamponade.

Introduction

In the past, pericardial effusion was thought to be a common finding in the hypothyroid patient, estimated to occur in as many as 30% of cases (1), but recent studies using echocardiography show that only 3-6% of hypothyroid patients have pericardial effusion (2). Pericardial tamponade is a rare presentation of hypothyroidism (2,3) and has been found in most cases only after many years of symptomatic hypothyroidism (1,3-6). Although viral infection is thought to be most common cause of acute pericarditis, it rarely progresses to cardiac tamponade (7).

Our case is unique as compared to previously reported cases, in that our patient had no previous

diagnosis, or chronic symptoms of hypothyroidism, and also had a proven viral pericarditis causing tamponade.

Case Report

A 25 year old Hispanic woman was brought into the emergency department by ambulance with the complaint of nausea, vomiting, and diffuse abdominal pain for 2 days. On the day of admission, the patient became dizzy and fell to the floor. When EMS arrived she was severely hypotensive. One liter of normal saline was rapidly infused, the patient improved markedly and was transported to the emergency department. She denied having any chest pain, difficulty breathing, palpitation

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or trauma. She had no fever, upper respiratory tract or urinary symptoms. She denied any history of sexual contact, vaginal bleeding or discharge, but her menses were usually irregular. She did not give a history of weight gain, weakness, cold intolerance, hoarseness or changes in her skin, hair or bowel habits. There was no past history of any significant medical illness and she was not taking any medication. She denied any drug or alcohol abuse.

On examination, the patient was well developed, well nourished and not in any distress. Vital signs included a blood pressure of 102/60 mm of Hg pulse of 78/mt, respiratory rate was 18/mt. and a rectal temperature of 98.3F. There were no orthostatic changes. The examination of her head was normal with normal pupils and fundus. Neck examination revealed distended jugular veins but no masses. Her lungs were clear with bilaterally equal breath sounds. Her heart sounds were distant. There was no rub or murmur. Abdomen was soft with minimal right upper quadrant and upper epigastric tenderness. There was no rebound tenderness or guarding and no significant organomegaly. The rectal examination was normal with brown stool negative to occult blood. Pelvic examination revealed a non-tender cervix, normal sized uterus and no adnexal masses. Her neurological examination was normal, with normal mentation and reflexes. Laboratory examinations revealed white blood cell count of 5,800 cells/mm³, a hematocrit of 29.8% with normal indices and platelets, normal electrolytes, normal serum glucose and normal renal functions. Coagulation studies, amylase, lipase and liver function tests were normal. Her urine analysis, pregnancy and toxicology tests were negative. The sedimentation rate was 51 mm/hr, cholesterol 310 mg/dl, and CPK 1145u/l and the CKMB was 3 ng/ml. Her chest x-ray revealed massive cardiomegaly without infiltrate or pleural effusion (Figure 1). Abdominal x-rays were normal. The

ECG strip revealed low voltage complexes with electrical alternans (Figure 2).

An echocardiogram was done in the emergency department which demonstrated massive pericardial effusion with right ventricular diastolic collapse (Figure 3). The ultrasound of the gallbladder done simultaneously was normal. The patient was then taken to the operating room for a pericardial window which released 1300 cc of straw colored fluid. The pericardium was closed with a drain left in place, which was subsequently removed three days later. From the operating room, the patient was transferred to the Cardiac Care Unit in stable condition.

Pericardial fluid showed many lymphocytes and polymorphonuclear cells but smears and cultures were eventually negative for bacteria, mycobacteria and fungi. Pericardial biopsy demonstrated a moderately dense and acute inflammatory infiltrate consistent with acute pericarditis. Lyme, rheumatoid and syphilis serological tests were all negative. A workup for anemia only revealed iron deficiency. CD 4/CD 8 counts were normal. Tests for Human Immune Deficiency Virus and cytomegalic virus were negative, but IgG titers for the Epstein Barr virus were moderately positive. The patient's PPD was negative. Thyroid function tests showed severe hypothyroidism with a TSH greater than 150 uIU/ml, T3 of 9ng/dl, T4 less than 3ng/dl, but negative antithyroid antibodies. The patient was started on intravenous hydrocortisone and oral Synthroid. The patient responded very well to this therapy and was discharged home in two weeks with the final diagnosis of cardiac tamponade secondary to pericardial effusion due to hypothyroidism. The tamponade was thought to have been triggered by a concomitant acute viral pericarditis, secondary to Epstein Barr virus. When the patient was seen in the clinic ten days later, she was still doing very well and a chest x-ray at that time was normal (Figure 4).

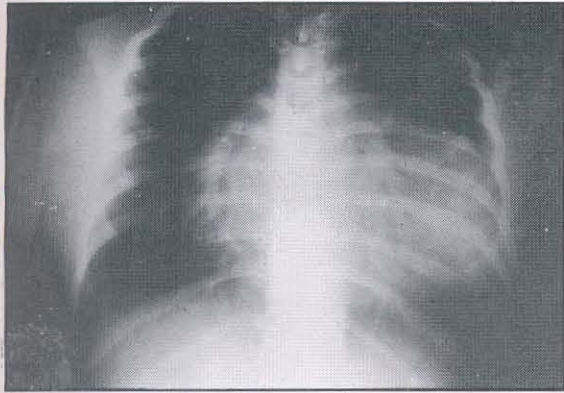


Fig. 1. Chest x-ray of the patient revealing massive cardiomegaly.



Fig. 2. EKG strip revealing low voltage complexes with electrical alternans.

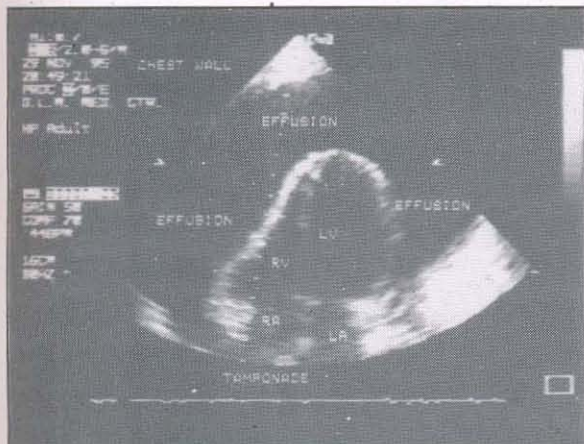


Fig. 3. Echocardiogram demonstrating massive pericardial effusion with right ventricular diastolic collapse.



Fig. 4. Follow up normal chest x-ray.

Discussion

Hypothyroidism can cause effusion in various body cavities including the peritoneum, pericardium, pleura, middle ear, uvea, joints and scrotum (8). These effusions are exudative and the mechanism is mainly extravasation of hygroscopic mucopolysaccharide into the body cavities along with increased capillary permeability, decreased lymphatic drainage, and increased retention of salt and water (2,3,9). The serum and effusions in patients with hypothyroidism usually have high levels of cholesterol and cholesterol pericarditis causing tamponade has been reported (10).

Although pericardial effusion is a common finding in hypothyroidism, pericardial tamponade has been found in most cases only after many years of being symptomatic or having been diagnosed and treated for hypothyroidism (1,3-6). It has been thought that the size of the pericardial effusion depends on the severity and duration of hypothyroidism and most cases of tamponade have been reported in the elderly (2,11). There have been few cases of massive pericardial effusion due to hypothyroidism reported in children (12-14).

However, hypothyroid patients with hemodynamically significant pericardial effusions may not always have prominent symptoms and signs of hypothyroidism, such as weight gain, weakness, edema, slow mentation etc. This is clearly demonstrated by our young patient without obvious symptoms or signs of hypothyroidism. Therefore, we believe that hypothyroidism must be ruled out in all patients with unexplained pericardial effusion and not just in patients with clinically obvious hypothyroidism or the elderly.

The classical signs of cardiac tamponade like hypotension, muffled heart sounds, and dilated necks veins (Beck's Traid) are not always present (7). Pulsus paradoxus is usually but not always present (7). The expected compensatory tachycardia in a patient with



tamponade is usually absent in patients with hypothyroidism as in our case (6,7,9). A pericardial rub is usually not present with large effusion (7). The electrocardiogram can have low voltage complexes with electrical alternans, which can be due to either myxedematous heart disease or pericardial effusion (2,7,9).

Diagnosis of the pericardial effusion is usually made by chest x-ray and is confirmed by echocardiography, which is the diagnostic procedure of choice with an extremely high sensitivity and specificity (2,7,11). The echocardiogram signs of cardiac tamponade, all of which were present in our patient, include right ventricular end diastolic collapse, right atrial compression, and a bowing of the interventricular septum into the left ventricle upon inspiration (2,7). With adequate medical treatment of the hypothyroidism with thyroid hormones and steroids the vast majority of pericardial effusions will resolve slowly but completely and rarely surgical intervention is needed (1,2,9). Pericardiocentesis or surgery is necessary only in the cases of pericardial tamponade where the patients are hemodynamically unstable (2,7).

The occurrence of cardiac tamponade in hypothyroidism is very rare due to the slow accumulation of the fluid and pericardial distensibility. When tamponade occurs it may be due to provocative factors such as concomitant viral pericarditis (2,3). This might be the case in our patient, whose pericardial biopsy demonstrated acute inflammation. Also, our patient's EBV titers were acutely elevated and the Epstein-Barr virus has been implicated as a cause of acute pericarditis (7). It is recommended that the diagnosis of pericardial tamponade should always be considered as a possible etiology in patients with unexplained shock and that underlying hypothyroidism in conjunction with acute

viral pericarditis is considered in patients with unexplained pericardial effusion and tamponade.

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