Brief Communication

A swinging heart

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ABSTRACT

We present a case of young female presenting with clinical features of cardiac tamponade. On initial investigation, the etiology of cardiac tamponade could not be made. The presence of bradycardia with cardiac tamponade prompted us to perform thyroid function test which lead to the diagnosis of hypothyroidism.

Key words: Hypothyroidism, pericardial tamponade, pericardial effusion

Introduction

While hypothyroidism is common and its features are well-recognized, pericardial tamponade is a rare presentation of hypothyroidism. Prompt diagnosis is vital as cardiac tamponade can be life-threatening. Treatment with creation of a pericardial window and drainage of pericardial fluid leads to hemodynamic improvement. Hypothyroidism should be treated and other causes of pericardial effusion should be excluded in such patients.

Case Report

In July 2011, a 27-year-old female was admitted because of increasing dyspnea and orthopnea of 20 days duration with sudden worsening 3 days back. She also noted puffiness of face and swelling in feet. She denied history of fever, rash, cough, hemoptysis, arthralgia, and chest pain. Her urinary habits were normal. On examination, she was severely dyspnoeic with respiratory rate of 35 breaths/minute. Her pulse rate was 84 beats/minute, feeble, blood pressure was 88/50 mmHg, pulses paradoxus was present, and jugular venous pressure was raised 12 cm above sternal angle. Facial puffiness and mild pitting pedal edema was present. There was no cyanosis and lymphadenopathy. The cardiovascular examination revealed a silent precordium. Cardiac apex impulse was neither visible nor palpable. On percussions, the right cardiac boarder was 2 cm to the right of sternal boarder and left cardiac border extending up to mid-axillary line. On auscultation, heart sounds were soft, distant, and there was no murmur. The chest examination was normal. Abdominal examination showed moderate tender hepatomegaly. The electrocardiograph (ECG) showed low voltage complexes with electrical alternans. X-ray chest [Figure 1] showed gross cardiomegaly and normal lung fields. Echocardiography (ECHO) revealed intrinsically normal heart, large pericardial effusion with a swinging motion of heart within effusion, and diastolic collapse of the free right ventricular wall suggestive of lemonade. Doppler study revealed a plethoric and non-collapsible

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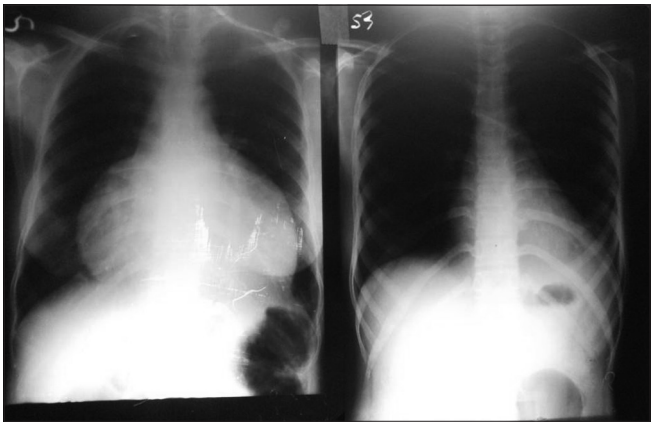

Figure 1: Chest X-ray prior to treatment and then after treatment

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inferior vena cava during inspiration with reversal of expiratory hepatic-vein flow. Ultrasonography-guided needle pericardiocentesis was performed and 200 ml of straw-colored fluid was removed. The fluid showed 300 white blood cells (WBC) with 45% neutrophils, 55% lymphocytes, 250 red blood cells (RBCs), 6.5 gm% total protein, sugar 65 mg%, and total cholesterol 230 mg%. Pericardial fluid gram stain, culture, and acid fast stain were all negative. Adenosine deaminase (ADA) in the fluid was negative. Her hemogram, liver and renal function tests, and urinalysis were normal. Montoux test and antinuclear antibody was negative. Polymerase chain reaction (PCR) was negative for mycobacterium. As the cause of temponade was unclear and keeping in mind the absence of tachycardia in the presence of persistent hypotension, thyroid function was performed. Thyroid function was: Thyroxine (T4)-2.5 ng/dl (4-12 ng/dl), triiodothyronine (T3)-160 μg/dl (180-650 μg/dl), and thyroid-stimulating hormone (TSH)-135 uIU (0.5-5.5 uIU). Thyroid replacement therapy was initiated with levothyroxine 100 μg. She improved and was shifted from cardiac care unit to general ward. On 3th day in the medical ward, again her dyspnoea worsened and she became hemodynamically unstable. Repeat ECG showed increased pericardial fluid with severe cardiac tamponade. An ultrasonography-guided pigtail catheter was inserted and total 1,200 ml of straw colored fluid was drained in next 24 hours. She improved clinically and discharged on levothyroxine 100 μg. At 3 month follow up, she was asymptomatic and her thyroid function and repeat ECG was normal.

Discussion

Hypothyroidism is a common disease with multi-system involvement. The prevalence of pericardial effusion in hypothyroidism has been reported between 30% and 88%; however,[1] it is extremely rare for hypothyroidism to present as a cardiac temponade.[2] The rarity of cardiac temponade in hypothyroidism is attributed to slow accumulation of fluid in the pericardial cavity and marked distensibility of pericardium, allowing significant fluid accumulation without hemodynamic compromise. However, the severity and duration of hypothyroidism determine the size and rate of accumulation of pericardial effusion in hypothyroidism. Emergency, needle percardiocentesis is recommended to relieve hemodynamic instability. Serial ECHOs are recommended to detect early relapse, after pericardiocentesis.[3] In our case, early relapse was suspected clinically and confirmed on ECHO after needle pericardiocentesis, even though the etiology was a benign one. Our case highlight that even though hypothyroidism is very uncommon cause of cardiac temponade, it is ought to be included among different diagnosis of cardiac temponade, even in the absence of classical symptoms of hypothyroidism. Clinical problem solving often forces physicians to consider numerous diagnostic possibilities including infections, malignancy, connective tissue, and autoimmune disorders. Occasionally, the initial clinical evaluation reveals a distinct finding that fits into a more circumscribed problem. In the present case, the diagnosis was reached through a systemic process of eliminating commoner causes of cardiac temponade and interpreting the clinical sign of an absence sinus tachycardia in the presence of ongoing persistent hypotension which collectively fit best with hypothyroidism.[4]

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