Subacute Cardiac Tamponade In A Patient With Myxedematous Pericardial Effusion

T Mukhithi, R Cordoves, J Zaldivar

Citation

T Mukhithi, R Cordoves, J Zaldivar. *Subacute Cardiac Tamponade In A Patient With Myxedematous Pericardial Effusion*. The Internet Journal of Cardiology. 2020 Volume 15 Number 1.

DOI: 10.5580/IJC.55470

Abstract

Hypothyroidism is a recognized cause of pericardial effusion but as a cause of cardiac tamponade it is rare, especially in the sub-Saharan Africa where tuberculosis is the commonest cause. When clinically suspected, confirmation of cardiac tamponade with echocardiography is mandatory; technic that will assist in the execution of an ultrasound-guided, lifesaving, pericardiocentesis. We report a case of a patient who presented with cardiac tamponade secondary to myxedema and Wolf-Parkinson-White pattern as incidental finding.

INTRODUCTION:

There are multiple causes of pericardial effusion (PE): infections, neoplasms, autoimmune diseases, trauma, uremia, myxedema, iatrogenic and others; any of them might cause a cardiac tamponade (CT).[1] In developed countries idiopathic (presumably viral), neoplastic and iatrogenic are the leading causes of PE but in developing countries, especially sub-Saharan Africa, tuberculous pericarditis is by far the commonest, situation that has worsen with the HIV epidemic, however others unexpected causes surprise us sometimes.[2] [3]

The pericardial sac can stretch to accommodate large volumes of fluid, up to four liters or more, if it develops slowly. At any given moment the pericardium behaves as a rigid container; in such a way that a sudden increment of pericardial volume will elevate the intrapericardial pressure opposing cardiac chambers pressure and impeding compliance during diastole; when pericardial and myocardial pressures equalize CT develops.[2]

Acute CT develops within minutes due to rupture of the heart or aorta, trauma or as a complication of an invasive procedure; its presentation resembles a cardiogenic shock. Subacute CT develops over days to weeks and can be associated with PE of any aetiology. Patients present with the classical picture of CT: shortness of breath, chest discomfort, peripheral odema, hypotension, engorged jugular veins, pulsus paradoxus, etc.

The diagnosis of CT demands a high index of suspicion, regardless of its aetiology, because it resembles severe right side cardiac failure or shock. An immediate decompression of the heart is mandatory in order to avoid the imminent patient death.

PE in hypothyroidism is commonly recognized but as a cause of CT it is rare, presumably because fluid slow accumulation allows the pericardial sac to accommodate large quantities of it.

Echocardiography revolutionized the diagnosis of CT. After suspecting this complication from the medical history and physical findings, mainly the presence of pulsus paradoxus, the visualization of diastolic collapse of right cardiac chambers makes this diagnosis certain demanding the immediate execution of a lifesaving pericardiocentesis or pericardial window.

We report a patient with primary hypothyroidism who presented in cardiac tamponade and who had a WPW pattern type B as incidental finding.

CASE REPORT

A 56-year-old female patient was brought by her relatives to the emergency department because of one month history of weakness and prostration. She gave a vague history of poor appetite and constipation for “a long time”. She complained of feeling weak and easy fatigue when walking for about a month; her weakness became worse since a week prior to

admission to the point that she was confined to bed the whole day. She denied orthopnea, paroxysmal nocturnal dyspnea, chest pain or palpitations. In further inquiry, she denied cold intolerance. Her relatives reported facial and lower limbs swelling for several months. She never took medication for any prior chronic condition neither she reported cardiac rhythm disturbance of any kind.

On physical examination she looked critically ill with periorbital edema. She was awake but responded during the interview with a low pitch hoarse voice. She showed a slow mentation and a facies characteristic of myxedema. Her skin was cold, thick and dry with a rough texture. Her temperature was 36.2° Celsius. She was pale but not jaundiced. She looked in respiratory distress with a respiratory rate of 28 breaths per minute; no crackles, wheezes or pleural rub were heard. Her cardiovascular examination was remarkable by the impossibility to find a point of maximal impulse and the heart sounds were distant. There was no murmur or pericardial rub. The jugular veins were distended up to the jaw angle. The brachial pulse was weak at a rate of 88 beats per minute; remarkably, it became imperceptible during inspiration. We could not quantitate the pulsus paradoxus due to lack of manual sphygmomanometer in the hospital, only electronic monitors were available. Her blood pressure was 70/42 mm of Hg. A tender hepatomegaly could be felt eight cm bellow the costal margin; she also had a moderate ascites. Symmetrical lower limbs pitting edema extended up to the knees. Deep tendon reflexes were absent in all limbs therefore Woltman's sign could not be elicited.

A supine chest x-ray showed an increased cardiac silhouette and clear lungs fields. The EKG showed sinus rhythm, the QRS was wide measuring 158 milliseconds with morphology suggestive of LBBB in lead I. The PR interval was short, 80 ms. PJ interval measured 240 ms (normal < 270 ms). All leads showed clear delta waves, indicative of WPW pattern (Figure 1).

An informal echocardiogram, performed in casualty by a general physician, showed a four cm pericardial effusion with partial diastolic collapse of right atrium and ventricle suggestive of cardiac tamponade (Figure 2).

An ultrasound-guided pericardiocentesis was performed using the paraxiphoid approach, 380 mL of light straw-yellow color pericardial fluid were removed with immediate improvement of patient clinical condition, blood pressure rose to 95/73 mm of Hg. A catheter was left in the pericardial cavity for 48 hours, a total of 1220 mL were removed before withdrawing the catheter.

The pericardial fluid analysis showed a protein content of 48 g/L, compatible with an exudate, LDH: 152 U/L, cholesterol: 1.4 mmol/L, ADA: 3.9 u/L, erythrocytes: 38/mm³, lymphocytes: 24, polymorphs: 0/mm³, molecular diagnostic test (Gen Xpert) and culture for Mycobacterium tuberculosis were negative. The full blood count, liver and renal function were normal. Other laboratory results can be seen in table 1.

The patient was treated with levothyroxine 50 mcg/day initially increased later to a maintenance dose of 100 mcg/day. Her thyroid function normalized six months after the initial visit, TSH: 2.11 mIU/L and a T4: 8.2 pmol/L, but small pericardial effusion persisted, (Figure.3). She died suddenly at home 14 months after the initial presentation.

Figure 1

Electrocardiogram showing sinus rhythm, short PR (80 ms), wide QRS (158 ms), delta waves, normal PJ interval (240 ms) and one extrasystole at beat 8 that shows a delta wave.

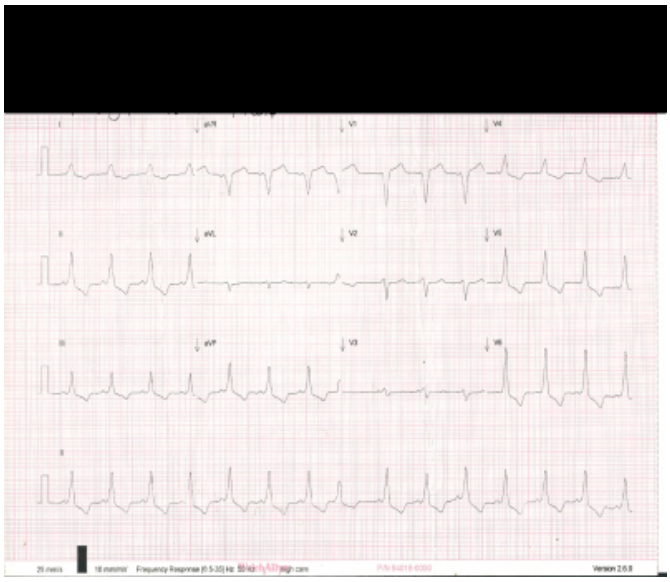

Figure 2

Echo showing pericardial effusion with partial diastolic collapse of right atrium and ventricle.

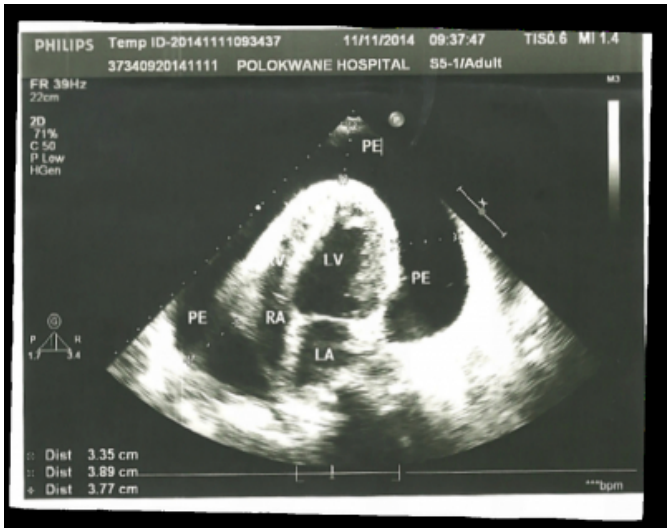

Figure 3

Echo showing persistence of small pericardial effusion.

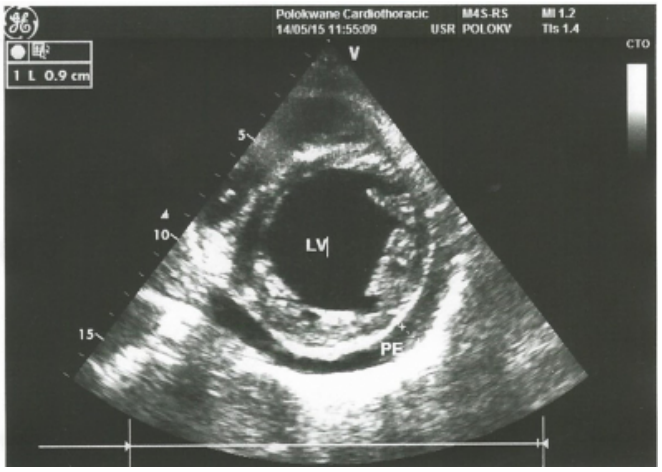

Table 1

Lipids and thyroid function.

Test	Result	Normal value
Total Cholesterol	3.99 mmol/L	< 5.2 mmol/L
LDL cholesterol	1.64 mmol/L	<3 mmol/L
HDL cholesterol	1.67 mmol/L	>1.2 mmol/L
Triglyceride	1.49 mmol/L	<1.7 mmol/L
TSH	> 100 mU/L	0.5-4.5 mU/L
T4	1.2 pmol/L	12-22 pmol/L
T3	1.3 pmol/L	3.9-6.7 pmol/L

DISCUSSION:

The patient that we present is interesting for two reasons, first because myxedematous PE is a rare cause of CT and second due to the coexistence of a WPW pattern.

In developed countries the leading causes of PE, and therefore tamponade, are acute pericarditis (presumably viral), trauma, and rupture of the heart.[1] Corey et al reported, out of 57 patients, that malignancy (23%), viral infection (14%), and radiation-induced inflammation (14%) were the most common causes of medical PE; no effusion caused by hypothyroidism was identified.[2] Regarding CT, Cornily and coworkers found 114 patients, in a 10 years period, of whom 74 (65%), 11 (10%) and 4 (3%) represented cases of malignant PE, viral pericarditis and hemopericardium due to anticoagulants respectively; again, no case of myxedematous PE was reported.[2] The reports of these different aetiologies will depend on the characteristics of the population investigated, the different types of departments (general versus specialized), the study protocol employed and the prevalence of the different aetiologies in a given geographic area.[1]

In Sub Saharan Africa cardiac tamponade is most frequently caused by tuberculous pericarditis; in fact when facing a patient with this complication, if there is no evidence of trauma or malignancy, this should be the most likely diagnosis, regardless of the evidence of tuberculous infection, pulmonary or otherwise.[3] In our case this aetiology was ruled out by a negative genXpert test, relevant microscopy and culture for tuberculosis, even the ADA level did not suggest this cause.

Pericardial effusion in hypothyroidism has been reported

from 30 to 64% from early reports to just 3 to 6% in more recent studies.[11,12] The occurrence of pericardial effusion in hypothyroidism appears to be dependent on the severity of the disease; a reduction of the diagnosis of mixedematous PE is attributed to an earlier diagnosis of hypothyroidism due to wider availability of laboratory facilities and modern techniques of hormones measurements.[13] PE can take months to disappear despite clinical and humoral evidence of euthyroidism. The case that we present showed a typical clinical and humoral picture of advanced myxedema. Eight months after the initial event and normal thyroid function test values small pericardial effusion was still present, (Figure 3).

The diagnosis of CT is both difficult and urgent; a high index of suspicion is needed. This syndrome is characterized by the following triad: arterial hypotension, venous hypertension (distended neck veins), "quiet heart" (distant or inaudible heart sounds), also known as Beck's triad, and pulsus paradoxus. Most patients show tachycardia; its absence might indicate hypothyroidism or uremia, the former was the explanation for a normal heart rate in our patient.[1] Pulsus paradoxus has been defined as a fall in inspiratory systolic blood pressure of 10 mm of Hg or more during normal breathing, it can be measured by intra-arterial catheterization or by manual sphygmomanometer, being the former more accurate but usually not available in the emergency room. Manual sphygmomanometers has virtually disappeared from South African hospitals, electronic monitors have replaced them, they are more accurate, if well calibrated, but useless for detecting postural hypotension or pulsus paradoxus; that is why we had to use the inspiratory disappearance of the radial pulse as a sign of pulsus paradoxus, the same criterion used by Kussmaul almost 150 years ago, a physical sign still valid but much less sensitive.

An enlarged cardiac silhouette on chest x-ray is common in CT, as it was the case in our patient; however, its absence does not rule out the condition, specifically in acute CT, since more than 200 mL of fluid are needed for the cardiac silhouette to enlarge. [1]

The typical electrocardiographic findings of CT are low voltage and electrical alternance, features that our patient electrocardiogram did not show, although neither of them is very sensitive. [1] It also showed a remarkable finding, a WPW pattern, type B in this case. We classified it pattern, rather than syndrome, because there was no history of palpitations or documented arrhythmias. We consider this electrocardiographic abnormality just an incidental finding.

WPW type B resembles LBBB, rarely these two conditions can coexist. [1,] Short PR interval, delta waves and normal PJ interval (< 0,270 ms) favor the former whereas the morphology of R wave in lead DI, slurred almost round, made us to consider the coexistence of the later. Hypothyroidism has been associated to higher risk of atherosclerosis, presumably due to dyslipidemia, that our patient did not have, but also caused by diastolic hypertension and endothelial dysfunction. LBBB could be expression of coronary heart disease, silent due to decreased physical activity and low oxygen demand. The patient sudden death could also suggest an acute episode of myocardial ischaemia. Unfortunately, we couldn't perform electrophysiological studies to gather evidence in favor of our hypothesis, mainly, that this was a case of WPW pattern and LBBB rather than just WPW type B alone. Facilities to perform electrophysiological studies were not available in our hospital and the patient rejected the idea of going to another institution alleging that she was feeling well after treatment.

Ultrasonic visualization of the heart has revolutionized the approach to CT. This technique enables us to objectively observe the pericardial fluid and the collapsing of the right ventricle and, or, right atrium. It also provides guidance when performing the pericardiocentesis. A formal echocardiogram, although desirable, is not mandatory; an informal study performed by anyone with just basic knowledge of the technique can provide a quick change in patient outcome. In most developing countries a cardiologist may not be available in the right place at the right time, but the removal of just 200-300 ml of pericardial fluid under echocardiogram guidance can save many live. Several lessons can be learned from this case:

1. Myxedematous pericardial effusion is an uncommon cause of cardiac tamponade but this aetiology should be considered if the clinical picture suggests hypothyroidism and other commoner causes are excluded.
2. There is still a place for manual sphygmomanometer in modern medical practice otherwise pulsus paradoxus and postural hypotension will never be detected.
3. An informal echocardiogram could be a lifesaving diagnostic tool in the emergency room and the technique should be learned by any practitioner not only cardiologists.
4. Although we could not prove it with this patient, it is important to remember that WPW pattern, or syndrome, and bundle branch blocks can coexist in the same patient.

References

1- Spodick D H. Acute Cardiac Tamponade. N Engl J Med 2003; 349:684-690.
2- Sagristà-Sauleda J, Mercé J, Permanyer-Miralda G, Soler-Soler J. Clinical clues to the causes of large pericardial effusions. Am J Med. 2000; 109:95–101.
3- Sliwa K, Mocumbi AO. Forgotten cardiovascular diseases in Africa. Clin Res Cardio 2010; 99:65–74.
4- Reddy PS, Curtiss EI, Uretsky BF. Spectrum of hemodynamic changes in cardiac tamponade. Am J Cardiol 1990; 66: 1487-1491
5- Spodick DH. Threshold of pericardial constraint: the pericardial reserve volume and auxiliary pericardial functions. J Am Coll Cardiol 1985; 6:296-297.
6- Klein I, Danzi S. Thyroid disease and the heart. Circulation 2007; 116:1725–1735.
7- Naqvi SY, Luebbert JJ, Rosen SG. Thyroid storm in a patient with Wolff-Parkinson-White syndrome. BMJ Case Rep Published online: 2015
8- Corey GR, Campbell PT, Van Trigt P, Kenney RT, O'Connor CM, Sheikh KH, et al. Etiology of large pericardia effusions. Am J Med 1993; 95:209–213.
9- Cornily J et al. Cardiac Tamponade in Medical Patients: A 10-Year Follow-Up Survey. Cardiology. 2008; 111:197-201.
10- Sagristà-Sauleda J, Mercé AS, Soler-Soler J. Diagnosis and management of pericardial effusion. World J Cardiol 2011; 3(5): 135-143
11-Crowley WF Jr, Ridgway EC, Bough EW, et al. Noninvasive evaluation of cardiac function in hypothyroidism. Response to gradual thyroxine replacement. N Engl J Med. 1977;296 (1):1-6.
12- Kerber R, Sherman B: Echocardiographic evaluation of pericardial effusion in myxedema. Circulation 1975; 52,823-827
13-Kabadi UM, Kumar SP. Pericardial effusion in primary hypothyroidism. Am Heart J 1990; 120:1393.
14-Cooper JP, Oliver RM, Currie P, Walker JM, Swanton RH. How do the clinical findings in patients with pericardial effusions influence the success of aspiration?. Br Heart J. 1995;73 (4):351–354
15- Hoit, Brian D. Cardiac Tamponade. Up to Date. May 2020
16- P.A. Chiale, M. Elizari. The electrocardiographic diagnosis of intraventricular blocks coexisting with ventricular preexcitation. J Electrocardiol 2012;45:515-524
17- Yang Chen, Renguang Liu, Zhaolong Xu. Wolff-Parkinson-White Syndrome: Could a Normal PJ Interval Exclude Bundle Branch Block? Rev Esp Cardiol. 2014.67 (2):153-155
18- Reydel B, Spodick DH. Frequency and significance of chamber collapses during cardiac tamponade. Am Heart J 1990; 119:1160-1163.

Author Information

T Mukhithi, MBChB, Dip HIV Man(SA),Senior Medical registrar
Department of Internal Medicine, Pietersburg-Mankweng Academic Hospital and School of Medicine, Faculty of Health Sciences, University of Limpopo
South Africa

R Cordoves, MD, consultant
Department of Internal Medicine, Pietersburg-Mankweng Academic Hospital and School of Medicine, Faculty of Health Sciences, University of Limpopo
South Africa

J Zaldivar, MD,Consultant
Department of Internal Medicine, Pietersburg-Mankweng Academic Hospital and School of Medicine, Faculty of Health Sciences, University of Limpopo
South Africa