

Cardiac tamponade. Presentation of two cases and review of the literature.

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Abstract

Objective: Describe two patients, who presented with cardiac tamponade attributable to malignancies and a review of the literature relative to clinical presentation..

Methods: We present clinical and imaging data for the two patients

Results: The two cases presented at the Accident and Emergency Department of our institution. The first case was a 48 year old male patient who was under investigation for a mediastinal mass, and the second, a 47 year old male patient who had been previously diagnosed with mesothelioma. Echocardiography showed in the two cases large pericardial effusion and diastolic right ventricular collapse. Emergency pericardiocentesis was performed on the two patients. The first patient was mechanically ventilated almost immediately after the procedure, and died 72 hours after admission as a consequence of sepsis. On the postmortem examination, large cell carcinoma of the lung with metastases to the trachea and brain was confirmed. The second case had a successful recovery and was discharged to be followed up as an out-patient.

Conclusion: Cardiac tamponade is not an unusual presentation in late stage malignancies and the role of echocardiography is primordial for the diagnosis, treatment and follow up of these patients. A high index of suspicion must be maintained for timely diagnosis of cardiac tamponade followed by prompt intervention.

Key words

Cardiac tamponade, echocardiography, pericardial effusion, pericardiocentesis, late-stage malignancies

■ INTRODUCTION

The complexity of some cases and the way we make diagnosis and provide appropriate treatment depends on several factors; firstly on the clinical skills, on how to match and recognize the clinical presentation of different diseases and conditions in our patients, and secondly on the availability of technology that can help us to confirm and establish their diagnosis and prognosis. The Karl Heusner Memorial Hospital in Belize City is our national referral center and for the past years, thanks to the presence of new technology and with the use of team work, we have been able to diagnose, confirm, and treat medical conditions that are more complex. In this article, we are reporting two cases of cardiac tamponade that

we managed in our institution, and a review of the literature.

■ CARDIAC TAMPONADE CASES

Patient number 1 was a 48 year old male patient admitted through the Accident and Emergency Department with history of malaise, and respiratory distress 24 hours before admission. This patient was under investigation at the outpatient clinic due to cough and radiological findings of a wide mediastinum. Two weeks prior to admission, he underwent an open biopsy to rule out a possible malignancy. On admission we found the subject with severe respiratory distress, and his vital signs at that time were: pulse of 120 per minute, blood pressure 90/60 torr, respiratory rate of 50 per minute. His neck had bilateral jugular distention, the chest was symmetrical, and there was evidence of the use of respiratory muscles. The lungs had the presence of bilateral crepitant sounds and wheezing.

Cardiac sounds were muffled; but there were no murmurs or pericardial rub, his abdomen was soft, depressible and without visceromegaly and his extremities were normal. Chest X Ray was performed before pericardiocentesis.

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Figure 1. X Ray before pericardiocentesis (patient 1)



Figure 3. X ray after pericardiocentesis (patient 1)

A bedside echocardiogram was performed (figure 2 A & B).

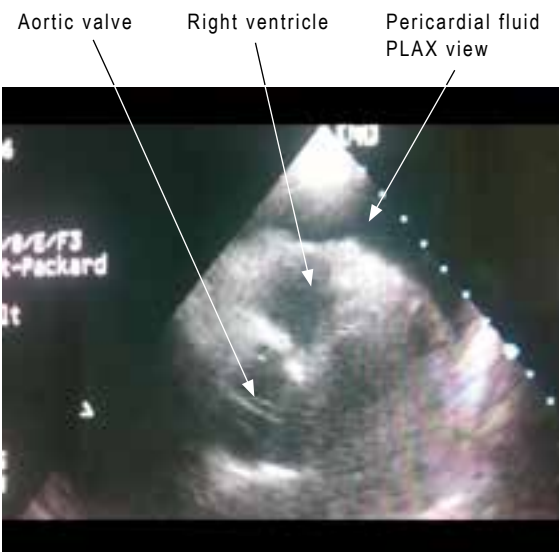
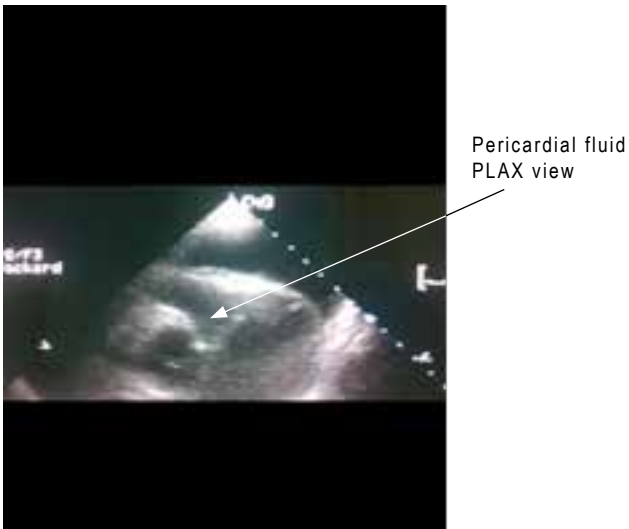


Figure 2 A & B. Echocardiogram (patient 1)

thoracic surgeon was immediately contacted. Emergency A diagnosis of cardiac tamponade was made and the cardio-bedside pericardiocentesis using Seldinger technique guided by echocardiogram was performed on this patient and a total of 500cc of a sanguinolent fluid was obtained slowly. A catheter was left in place to allow drainage of more fluid (figure 3).

The patient's respiratory distress showed no improvement and the blood pressure fell, so he was orotracheally intubated and connected to a mechanical ventilator. The possibility of a septic process was considered and antimicrobials with the use of vasopressors were added to his treatment. The patient was admitted to the hospital and presented cardiac arrest 48 hours after.

Necropsy was performed with the following findings: bilateral occlusion by thrombi of the external jugular veins (Figs. 4A & B), presence of a mediastinal mass with possible metastatic lesions to the trachea (Figs. 5 & 6), the left lung was found to have the presence of emphysematous changes in its apex, the right lung had changes suggestive of a neoplastic lesion in its upper lobe (Fig. 7), and this area was surrounded by purulent material, the pericardium had macroscopic changes suggestive of a "bread and butter" pericarditis (Fig. 8), and the brain had multiple hemorrhagic lesions.

The postmortem investigation confirmed large cell carcinoma of the lung with metastases to the brain and trachea, and the presence of a fibrinous type of pericarditis.



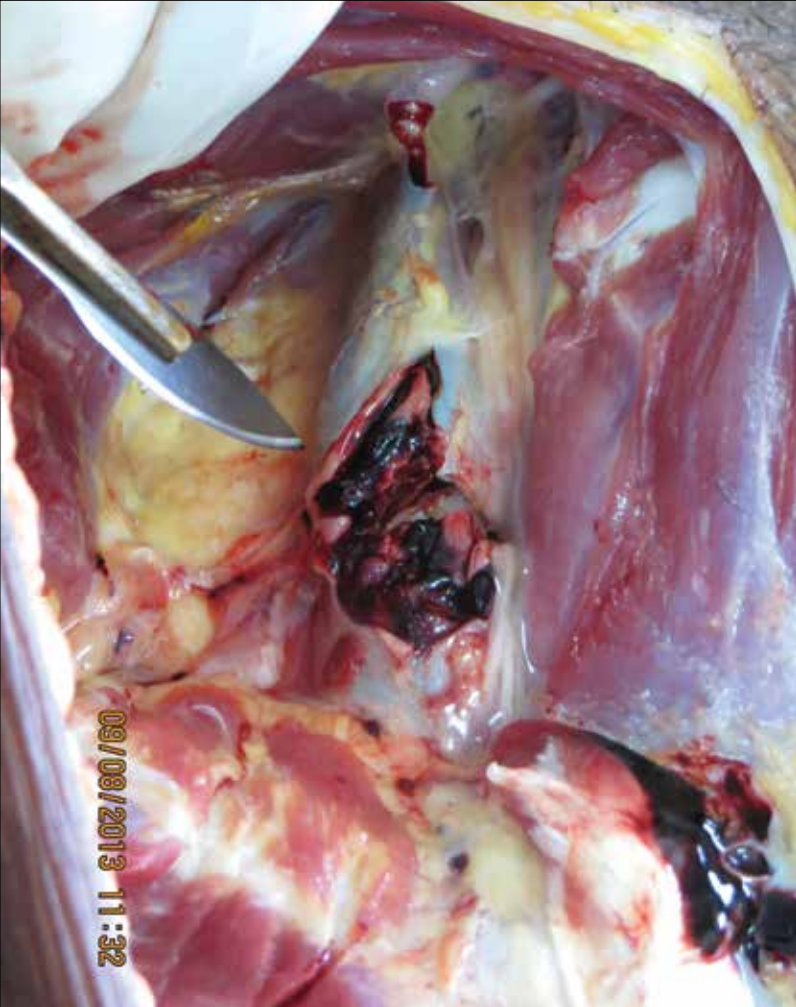


Figure 4 A & B. Jugular veins occluded with clots (patient 1)

Figure 6. Trachea with metastases (patient 1)

Figure 5. Mediastinal mass (patient 1)





1 Figure 7. Neoplastic lesion to the upper lobe of the lung (patient 1).



Figure 8. "Bread and butter" pericarditis (patient 1).

Patient number 2 was a 47 year old patient referred from one of the district hospitals due to respiratory distress. This patient had already been diagnosed to have mesothelioma. On admission, he was diaphoretic with moderate to severe respiratory distress and the following vital signs: B/P 90/60 torr, pulse 114, RR 30.

There was evidence of bilateral jugular distention; the chest was symmetrical but with evidence of the use of respiratory muscles; there was tachycardia with no murmurs, no pericardial rub, and the cardiac sounds were muffled; the abdomen was soft, depressible, and without visceromegaly; his extremities were normal.

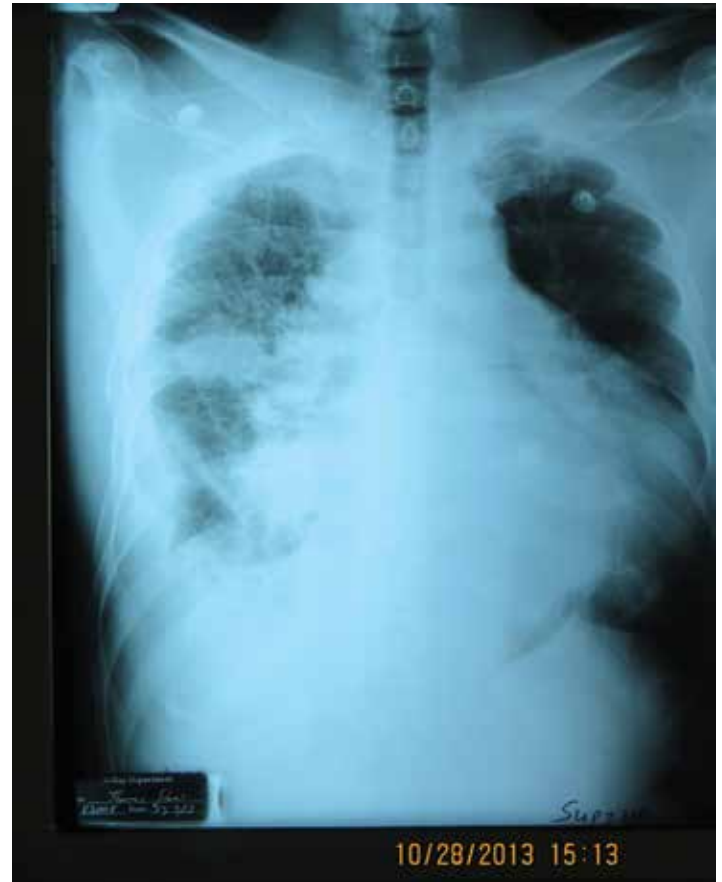


Figure 9. Chest X ray on admission (patient 2).

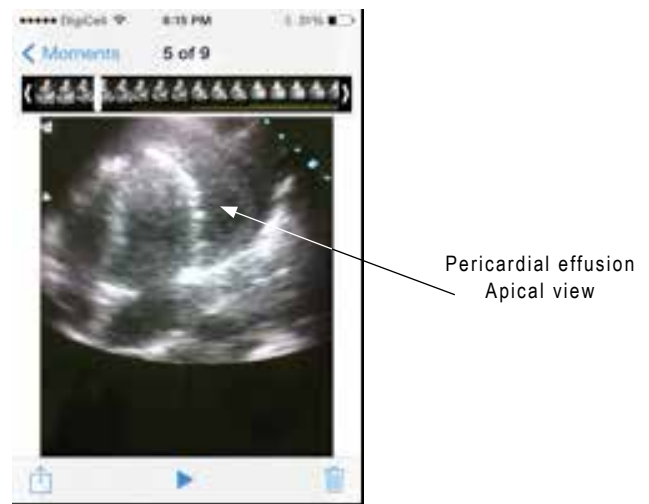


Figure 10. Echocardiogram (patient 2).

The echocardiogram (figure 10) showed presence of pericardial effusion with signs of strain to the right ventricle, and a "swinging heart". Pericardiocentesis was performed on this patient using Seldinger technique guided by echocardiography obtaining a total of 1500 cc of a sanguinolent fluid. A flexible catheter

was left in place for slow drainage. The patient was monitored with echocardiography on a daily basis to evaluate the amount of fluid and to perform intermittent drainage. After 5 days the catheter was removed and the patient was discharged to be followed as outpatient.

■ REVIEW OF CARDIAC TAMPONADE

Cardiac tamponade is a hemodynamically significant cardiac compression caused by pericardial fluid, which may be blood, pus, effusion (transudate or exudate), which will cause a constraint on chamber filling, reducing their diastolic pressures.

The incidence of cardiac tamponade is 2 cases per 10,000 inhabitants in the United States and approximately 2% of penetrating injuries are reported to result in cardiac tamponade. Also 24% of all the pericardial effusions are related to HIV infection.

Subacute cardiac tamponade occurs over days to weeks and can be associated with neoplastic, uremic, or idiopathic pericarditis. Symptoms include dyspnea, chest discomfort or fullness, peripheral edema, and fatigability.

Low pressure (occult) cardiac tamponade, a subset of subacute cardiac tamponade, occurs in patients who are severely hypovolemic. These patients with severe reduction in volume may have intracardiac and pericardial diastolic pressures as low as 6 to 12 mmHg.

Regional cardiac tamponade occurs when a loculated, eccentric effusion or localized hematoma produces regional cardiac tamponade in which only selected chambers are compressed.

ANATOMY OF THE PERICARDIUM

The pericardium is a bilayered sac: the thicker parietal pericardium is the outer fibrous layer, the thinner visceral pericardium is the inner serous layer, and the space in between is called the pericardial space that contains small amounts of pericardial fluid (20-50 ml). At the base of the heart, the serous pericardium reflects up and around the great vessels, forming the pericardial sinuses and recesses. The external layer is attached to the diaphragm by the pericardiophrenic ligament and to the sternum by the sternopericardial ligament. The posterior aspect is bound to the structures of the posterior mediastinum.

The arterial supply is given by the pericardiophrenic artery, a branch of the internal thoracic artery. The venous drainage is through the pericardiophrenic veins which are tributaries of the brachiocephalic veins.

Its functions are: a.- limitation of intrathoracic cardiac motion; b.- balancing left and right ventricular output; c.- suction filling; d.- limitation of acute dilatation during exercise and hypervolemia; e.- lubricant effects and f.- lymphatic and immunological functions

The normal pericardium has a small capacitance reserve (150-250cc). Once this is exceeded, it will result in sharp increments in intrapericardial pressure (IPP).

The IPP reflects the following: chamber volumes, plus fluid, plus clots and/or masses in the intracardial space. However,

1000cc of fluid may accumulate over a long period of time causing no hemodynamic effects. In general for any magnitude of effusion, the interval over which it accumulates is a critical determinant of hemodynamic impairment.

Three phases of hemodynamic changes have been described in cardiac tamponade:

Phase I: Initially, there is accumulation of fluid that causes an increase in stiffness of the ventricle, requiring higher filling pressure. At this point the left atrial and ventricular filling pressures are higher than the intrapericardial pressure.

Phase II: After more fluid accumulation, the pericardial pressure will be above the ventricular filling pressure, reducing cardiac output.

Phase III: Finally there is more reduction in the cardiac output due to the equilibrium of pericardial and left ventricular pressures.

CAUSES OF CARDIAC TAMPONADE

Hemorrhagic

A. Trauma

- a. Penetrating and blunt chest trauma
- b. Cardiac catheterization, pacemaker insertion, central venous cannulation, percutaneous coronary artery angioplasty
- c. Postoperative thoracotomy
- d. Pericardiocentesis



- e. External cardiac compression
- B. Myocardial Infarction
 - a. Rupture
 - b. Anticoagulation, thrombolytic therapy, antiplatelet agents
- C. Dissecting aortic aneurysm

Serous / Sero – sanguinous

- a. Neoplastic
- b. Connective tissue diseases: systemic lupus erythematosus, rheumatoid arthritis, rheumatic fever, polyarteritis, idiopathic thrombocytopenic purpura
- c. Uremia
- d. Radiation therapy
- e. Idiopathic

Purulent

Infectious

- a. Bacterial
- b. Viral
- c. Mycobacterial
- d. Yeast

Chylous

The most common etiologies are: a.- malignant disease – 30-60% of cases; b.- uremia – 10-15% of cases; c.- idiopathic pericarditis – 5-15% of cases; d. infectious diseases – 5-10%

of cases; e.- anticoagulation – 5 – 10% of cases; f.- connective tissue disease – 2-6%, and g.- Dressler or postpericardiotomy syndrome – 1-2 %

Cornily and coauthors reported a 1-year mortality rate of 76.5% in patients, whose tamponade was caused by malignant disease, compared with 13.3% in patients with no malignant disease. The investigators also noted a median survival of 150 days in patients with malignant disease.

CLINICAL PRESENTATION

Symptoms of cardiac tamponade will depend on the size and severity of the tamponade. The following have been described: dyspnea, fatigue, light-headedness, tachypnea, tachycardia, pallor, cool clammy extremities, pericardial rub, pulsus paradoxus, Beck's triad, hypotension and shock, although hypertension can occur. Sometimes the patient is found leaning forward or sitting in knee-chest position.

Pulsus paradoxus is an inspiratory decrease in systolic blood pressure greater than 10 mmHg and is characteristically found in acute cardiac tamponade. However, it can be found in right ventricular infarction, severe congestive heart failure, myocarditis, emphysema, asthma, hypovolemic shock, pulmonary embolism, extreme obesity and ascites.

Pulsus paradoxus may be absent under the following conditions: atrial septal defect, aortic regurgitation, severe aortic stenosis, uremia and in patients with severe hypotension accompanying advanced tamponade.

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Beck's triad consists of: muffled heart sounds, hypotension and jugular vein distention that has been classically reported in patients with cardiac tamponade. However, in some patients the triad could be absent.

The clinical auscultation of the lungs could be normal, but a large pericardial effusion may cause what is called Ewart's sign (dullness to percussion and/or the presence of bronchial breathing at the left lung base due to compression of the same area).

INVESTIGATIONS

Echocardiography

It is the most reliable way of diagnosing pericardial fluid, clots, and the hemodynamic effects of the effusion, and it helps determine the distribution of the effusion (loculated or concentric)

This test can confirm it even before clinical signs appear or an effect on cardiac output is found.

The findings may be: a.- abnormal septal motion: both ventricular and atrial septa move sharply leftward on inspiration and expiration); b.- right atrial and ventricular diastolic collapse; c.- reduced respiratory variation of the inferior vena cava and d.- swinging of the heart in its sac

Electrocardiogram

Its findings are: sinus tachycardia, with low voltage complexes and non-specific ST and T wave changes. It is possible to find presence of "electrical alternans", which is characterized by beat to beat alterations in the QRS complex and other electrocardiographic waves that reflect the swinging of the heart in the pericardial fluid.

Chest X ray

The results could be within normal limits especially in acute tamponade; but could also show: a- enlarged globular cardiac shadow with loss of the hilar waist (pericardial effusion >250 cc) or the so called water bottle-shaped heart; b- prominence of the superior vena cava; c- pleural effusions; d- epicardial fat pad sign (a radiolucent line between the epicardial fat and the mediastinal fat and represents the pericardium, and it should be 2mm or less) and e- pericardial calcifications.

Computed Tomography

In the CT scan, Gold and coauthors reported compression of the coronary sinus observed as an early marker for cardiac tamponade in 46% of patients.

TREATMENT

Definitive treatment is the removal of the cardiac diastolic restriction using pericardiocentesis or thoracotomy. Acute tamponade requires pericardiocentesis; the acute removal of as little as 50 cc of fluid is often enough to correct acute hypotension.

Pericardiocentesis is the removal of pericardial fluid by percutaneous catheterization of the pericardial sac. The sub-xiphoid approach has been the traditional procedure: he

patient is placed in a supine 30-45 degree head-up position, using a 16- to 18-gauge, 6 inch (15 cms) or longer over the needle catheter, attached to a 30-60 cc empty syringe with a three way stopcock. The skin is punctured 1-2 cms lower to the left of the xiphochondral junction at a 45 degree angle to the skin. The needle is advanced cephalad and aimed toward the tip of the left scapula. The needle can be used as a chest ECG lead to detect myocardial contact indicated by ST segment elevation, which is the so called "current of injury". When blood is aspirated from a cardiac chamber, it clots, unlike blood withdrawn from the pericardial sac. Checking the level of hemoglobin may also be done on the aspirated fluid and comparison to the one from a peripheral vein of the patient.

Another option is to apply the Seldinger technique, passing a guide wire through the needle into the pericardial space, removing the needle and passing a 14 gauge flexible catheter over the guide wire, which will be withdrawn and attached to a three way stopcock.

Presently pericardial aspiration should be performed under transthoracic echocardiographic guidance to determine if there is the presence of a large clot, or if it is a loculated effusion. The percutaneous site that is selected should avoid the internal mammary artery that is located 3-5 cms from the parasternal border and the vascular bundle at the inferior margin of each rib.

The first 100-200 cc of fluid should be drained rapidly. The remainder is drained slowly using an indwelling "pigtail" catheter, since fast removal of large volumes of pericardial fluid (>500 cc) may result in a "decompressive syndrome", causing pulmonary edema. If there is no drainage after 24 - 72 hours and evaluation, using echocardiography, shows no reaccumulation of fluid the catheter can be removed. When performed as an emergency, this procedure is associated with a reported mortality rate of approximately 4% and a complication rate of 17%.

Indications for thoracotomy are: a.- when tamponade follows a coronary artery bypass; b.- grafting; c.- cardiac rupture; d.- penetrating or closed cardiac trauma; e.- aortic dissection; f.- when a large pericardial clot is present; g.- failure of pericardiocentesis.

■ CONCLUSIONS

The prognosis of the patient suffering from acute cardiac tamponade will depend on the ability to demonstrate its presence and the speed to perform decompression of the pericardial sac. Long term prognosis will depend on the underlying condition that is causing the problem.

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