

CARDIAC TAMPONADE AND IT'S ECHO FINDINGS

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Abstract

Cardiac tamponade results from an accumulation of pericardial fluid under pressure, leading to impaired cardiac filling and haemodynamic compromise. A compression of the heart due to pericardial effusion. Cardiac tamponade typically leads to crisis by decreasing venous return, which impairs diastolic ventricular filling. If the tamponade is untreated, hemodynamic compromise can lead to the diminishing cardiac output. Therefore, as a continuum, cardiac tamponade constitutes a medical emergency. The main causes of cardiac tamponade include percutaneous cardiac interventions, malignancies, infectious/inflammatory causes, mechanical complications of myocardial infarction and aortic dissection. The diagnosis of cardiac tamponade is a clinical cab be done by worsening dyspnoea, distended jugular veins, muffled heart sounds , and should be confirmed by echocardiography. Cardiac tamponade is a life-threatening syndrome that requires urgent treatment by pericardiocentesis. Pericardiocentesis is an interventional technique to drain pericardial fluid by percutaneous route.

Keywords - Cardiac Tamponade, pericardial, Palpitation, iatrogenic, Hypotension.

CHAPTER NO. 1**1.INTRODUCTION**

Cardiac tamponade is a pericardial syndrome characterised by impairment of the diastolic filling of the ventricles causing reduction of cardiac output, usually producing signs and symptoms of cardiac arrest, if untreated. The impairment of diastolic filling is continuous through the entire diastole. The size of the effusion as well as its distribution may be variable. The pericardium is relatively stiff, thus the speed of accumulation of pericardial in the determination of the time course of cardiac tamponade. Rapidly accumulating pericardial fluid is responsible for a rapid increase in pericardial pressure, and cardiac tamponade can be reached soon by 200–300 ml of fluid (e.g. blood for haem pericardium in aortic dissection). On the contrary, slowly accumulating pericardial fluid can be asymptomatic and cardiac tamponade can be reached larger volumes upto 1-2l. This pathophysiology explains why cardiac tamponade is a last drop phenomenon and how small volumes of pericardial fluid may cause tamponade, as well as how it can be dramatically relieved by the aspiration of small amounts of fluid during urgent pericardiocentesis. In clinical practice, cardiac tamponade is generally caused by moderate to large circumferential pericardial effusion.(1,2,3,25)

CHAPTER NO. 2**2.SYMPTOMS AND CAUSES**

Acute cardiac tamponade is usually associated with low blood pressure (<90 mmHg) but slightly reduced in subacute, chronic tamponade. Hypertensive patients may have normal to mildly elevated blood pressure concomitant to cardiac tamponade. Classic signs include neck vein distention with elevated jugular venous pressure at bed side examination, pulsus paradoxus and diminished

heart sounds on cardiac auscultation. Pulsus paradoxus is due to exaggerated ventricular interdependence occurring in cardiac tamponade when the overall volume of cardiac chambers becomes fixed and any change in the volume on one side of the heart causes the opposite changes on the other side (i.e. inspiratory increase of venous return and right chambers with decreased left chambers volume and reduced systemic blood pressure).

Pulses and Paradoxus

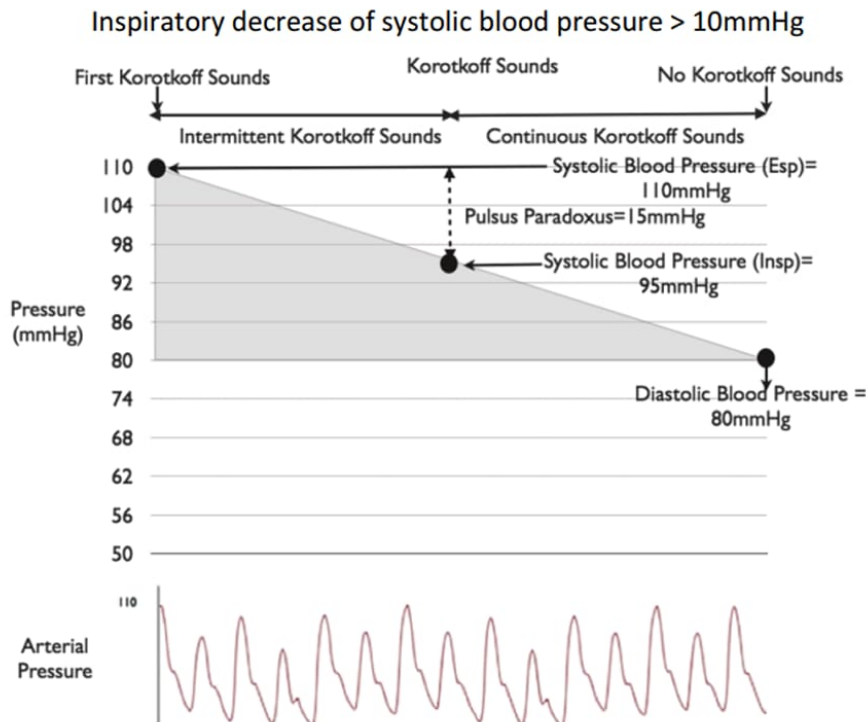


Fig: Showing the mechanism of pulses and paradoxus

On ECG, the patient usually shows tachycardia, low QRS voltages and electrical alternans. Both ECG signs are nonspecific and are considered related to the damping effect of pericardial fluid and swinging of the Heart. Sharp pain in the chest. The pain may also radiate or extend to nearby parts of your body like your arm, back, neck or shoulder. It may also get worse when you breathe deeply, lie flat or cough. Trouble breathing or breathing rapidly. Fainting, dizziness or light headedness. Changes in skin color, especially pale, gray or blue-tinted skin. Heart palpitations (where you become unpleasantly aware of your heartbeat). Altered mental status. A person with cardiac tamponade won't act like themselves and may act confused or agitated. When the condition happens more slowly, you may also have these cardiac tamponade symptoms: Shortness of breath. Swelling in your abdomen or legs. Fatigue or tiredness. Chest discomfort that typically gets better when you lean forward or sit up. All the causes of pericardial effusion are also possible causes of cardiac tamponade. In clinical practice the most common aetiologies include: cancer, tuberculosis and purulent infections, trauma, iatrogenic complication of cardiovascular interventions (e.g. ablation of arrhythmias, pacemaker implantation, percutaneous coronary intervention), acute aortic disease, systemic inflammatory diseases and renal failure. Blunt trauma, such as a fall or a car crash. Penetrating trauma, like a stab wound from a knife. Aortic dissection, Advanced cancer, Heart attack, Tuberculosis (TB), Inflammation or infection of the pericardium, Chronic immune diseases like lupus, rheumatoid arthritis and scleroderma, Heart or kidney failure, Hypothyroidism. In some cases, cardiac tamponade can also happen after a medical procedure. Some of these include: Surgery on heart or near the pericardium. Catheter-based procedures that involve your heart or surrounding blood vessels. Placement of a device like a pacemaker. Radiation therapy near your heart or pericardium. In recent series of cardiac tamponade, reported aetiologies include: percutaneous cardiac

interventions (upto 36% of all cases), malignancies(upto23%), infectious/inflammatory causes(upto15%) and mechanical complications of myocardial infarction(upto12%).¹⁴ An additional life-threatening cause of cardiac tamponade is aortic dissection. In a recent series, cardiac tamponade was reported in 76 out of 308 cases (24.7%) with acute aortic dissection.^(4,5,6,28)

CHAPTER NO. 3

3.DIAGNOSIS

Patients with cardiac tamponade present similar to patients with other forms of cardiogenic or obstructive shock. They may endorse vague symptoms of chest pain, palpitations, shortness of breath, or in more severe cases, dizziness, syncope, and altered mental status. They may also present in a pulseless electrical activity cardiac arrest. The classic physical findings in cardiac tamponade included in Beck's triad are hypotension, jugular venous distension, and muffled heart sounds. Pulsus paradoxus, which is a decrease in systolic blood pressure by more than 10 mm Hg with inspiration is an important physical exam finding that suggests a pericardial effusion is causing cardiac tamponade.

Pulsus paradoxus may be absent in patients with ASD, elevated diastolic pressures, pulmonary hypertension and aortic regurgitation.

The Kussmaul sign – a paradoxical elevation in JVP and pressure during inspiration is sometimes seen in cardiac tamponade.

In patients with large pericardial effusions, the Ewart sign may be present. This is an area of dullness with bronchial breath sounds heard just below the left scapula.

The JVP tracing may reveal an absent 'y' descent due to the elevated intrapericardial pressure that prevents the filling of the ventricles.

When fluid compresses the heart and impairs filling, the interventricular septum bows toward the left ventricle during inspiration due to increased venous return to the right side of the heart. This further decreases the size of the left ventricle leading to decreased left ventricular preload and stroke volume. The challenge with making the diagnosis of tamponade with clinical signs alone is difficult since they are neither sensitive nor specific.

The diagnosis of cardiac tamponade can be suspected on history and physical exam findings. ECG may be helpful, especially if it shows low voltages or electrical alternans, which is the classic ECG finding in cardiac tamponade due to the swinging of the heart within the pericardium that is filled with fluid. This is a rare ECG finding, and most commonly the ECG finding of cardiac tamponade is sinus tachycardia. In severe cases, one may note electrical alternans.

A chest x-ray may show an enlarged heart and may strongly suggest pericardial effusion if a prior chest radiograph with a normal cardiac silhouette is available for comparison. CT chest can also pick up pericardial effusion.

Echocardiography is the best imaging modality to use at the bedside, whether it is a point-of-care echo or a cardiology echo study. Echocardiography can not only confirm there is a pericardial effusion, but determine its size, and whether it is causing compromise of cardiac function (right ventricular diastolic collapse, right atrial systolic collapse, plethoric IVC). The medical literature is replete with studies that show clinicians (noncardiologists) with limited training using point-of-care echo can perform focused echocardiograms to answer specific questions such as whether there is a significant pericardial effusion.

Blood work that may assist with the diagnosis include creatine kinase levels, renal profile, coagulation profile, antinuclear antibody tests, ESR, HIV testing and PPD skin test.(7,8,9,10)

CHAPTER NO. 4

4.ECHO FINDINGS:-

4.1 Large pericardial effusion with swinging heart.

4.2 Chamber collapse

a) The lower pressure cardiac chambers (atria) are affected before the higher pressure cardiac chambers (ventricles).

b) Compressive effect is more likely to be seen in the phase of cardiac cycle when filling pressure within a cavity is lower, as occurs during systole for the atria and during diastole for the ventricles.

C) Right atrial chamber collapse (inversion)

- Right atrial chamber collapse begins first in late diastole.
- Commonly preceding typical clinical signs.
- It is sensitive but not specific sign of cardiac tamponade.
- However, the specificity of this sign improves if the duration of right atrial collapse exceeds 30% of the cardiac cycle.

D)Right ventricular chamber collapse (inversion).

- Right ventricular wall inversion occurs typically in early diastole, when intracavitary RV pressure/volume is at a nadir.
- As in right atrial wall collapse, the right-ventricular wall inversion will extend further into diastole (longer duration) as the hemodynamics of tamponade worsen.
- This echo finding is often best seen in the parasternal long-axis view, with transient “dimpling” of the right ventricular outflow tract anterior wall noted when the mitral valve opens.

E)Left atrial and left ventricular chamber compression

- Exclusively been described related to loculated collections occurring post cardiac surgery.

- However, circumferential pericardial effusion leading to left ventricular diastolic compression has rarely been reported in the setting of severe pulmonary hypertension

4.3 Duration of diastolic collapse of RA as ratio on the cardiac cycle length >0.34

4.4 Caveats

Tamponade in patients with high intracardiac pressure Ventricular interdependence doppler signs can be helpful in some cases. Hepatic vein expiratory diastolic flow reversal.

4.5 Echocardiographic or Doppler Signs of Increased Ventricular Interdependence

a) Mitral flow velocities:

- Mitral inflow E velocity will decrease significantly with inspiration (vs. expiration)
- A drop of more than 25% is considered consistent with significant tamponade physiology.

b) Tricuspid flow velocities:

- Tricuspid inflow E velocity will decrease significantly with expiration (vs. inspiration)
- A drop of more than 40% is considered consistent with significant tamponade physiology.
- This change should be noted on the first beat with expiration versus the first beat with inspiration

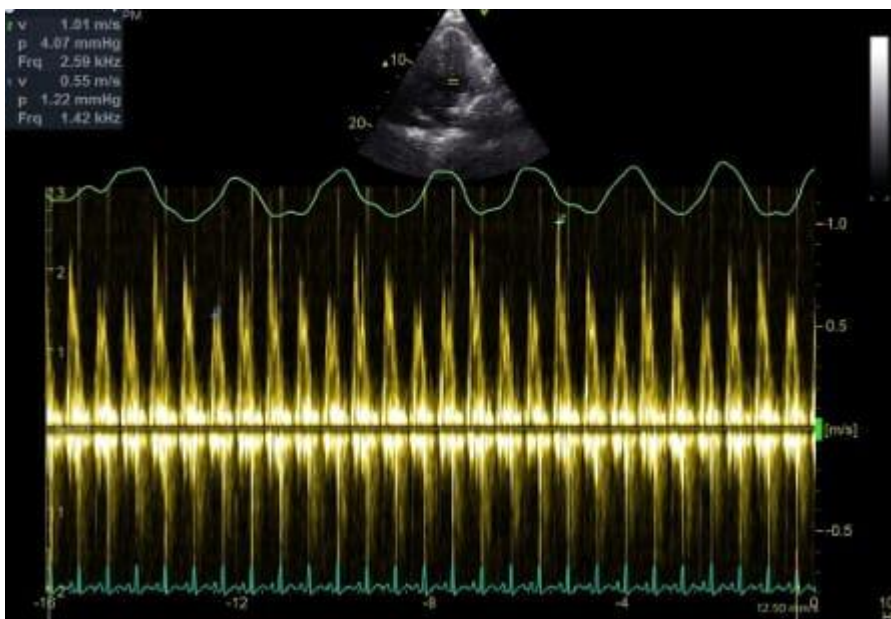


Fig 4.5 (a) Showing mitral flow velocity

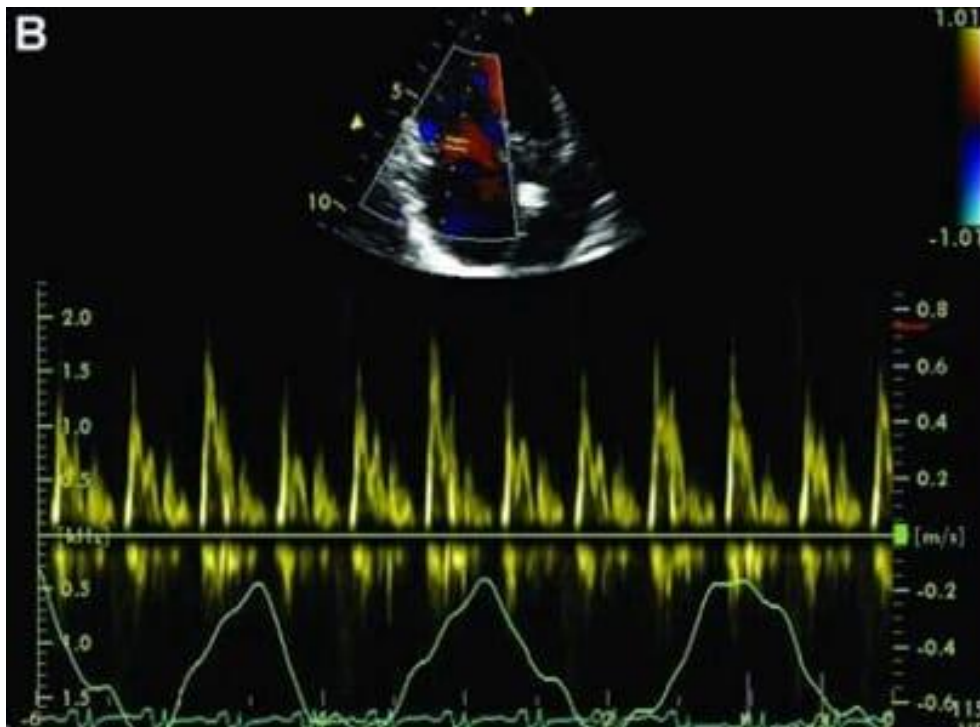


Fig 4.5 (b) Showing tricuspid flow velocity

4.6 Inferior vena cava

Plethora(dilatation>20mm and <50% reduction of diameter with respiratory phases).

4.7 Large pleural effusion with pericardial effusion:

- Occasionally been described as causing tamponade physiology sometimes with echo signs such as right ventricular diastolic collapse.
- In these situations, pericardial effusion is often present, and it can be difficult to decide which collection is more significant.
- Clinical experience usually favors first draining the more accessible pleural fluid, and then reassessing both clinically and by echocardiography.(26,26,27,30)

CHAPTER NO. 5**5.TREATMENT**

The treatment of cardiac tamponade is drainage of the pericardial fluid, preferably by needle pericardiocentesis with the Use of echocardiographic or fluoroscopic guidance that should be performed without delay in unstable patients. Alternatively, drainage is performed by a surgical approach, especially in some situations such as purulent pericarditis, or urgent situations with bleeding into the pericardium.

The standard technique for pericardiocentesis is guided echocardiography fluoroscopy under local anaesthesia. Blind procedures must not be used to avoid the risk of laceration of the heart or other organs, except in very rare situations that are immediately life threatening. An experienced operator and staff should perform pericardiocentesis in facility equipped least for echocardiographic, haemodynamic and ECG monitoring.

Before rushing to decompression of the pericardium, the patient should be provided with oxygen, volume expansion and bed rest with legs elevated. If possible, positive pressure mechanical ventilation should be avoided as it may further decrease venous return and aggravate the symptoms.

The treatment of cardiac tamponade is the removal of pericardial fluid to help relieve the pressure surrounding the heart. This can be done by performing a needle pericardiocentesis at the bedside, performed either using traditional landmark technique in a sub-xiphoid window or using a point-of-care echo to guide needle placement in real-time. Often the removal of the first small amounts of fluid can make a large improvement in hemodynamics, but leaving a catheter within the pericardium can allow for further drainage.

Surgical options include creating a pericardial window or removing the pericardium. Emergency department resuscitative thoracotomy and the opening of the pericardial sac is a therapy that can be used in traumatic arrests with suspected or confirmed cardiac tamponade. These options are preferable to needle pericardiocentesis for traumatic pericardial effusions.

Volume resuscitation and pressor support may be helpful; however, these are temporizing measures that should be performed while preparing for definitive treatment with one of the above procedures.(12,13,17,19)

CHAPTER NO. 6**6.PROGNOSIS**

The prognosis of cardiac tamponade is essentially related to the aetiology. Cardiac tamponade in with cancer and metastatic involvement of the pericardium have short-term prognosis, because it is a sign of advanced cancer. On the contrary, patients with cardiac tamponade and a final diagnosis of idiopathic pericarditis generally have a good long-term prognosis. The outcomes of chronic large idiopathic pericardial effusions have recently been reviewed in a cohort study of 100 consecutive cases.

The reported evolution is usually benign, with reduction of the size of the effusion in the majority of cases, and regression in about 40% of cases. The risk of cardiac tamponade is 2.2% per year (lower than previously reported) and recurrence/ complications and survival was better in patients treated conservatively without interventions.²¹ On the basis routine drainage of pericardial effusion is questioned in clinical practice in the absence of specific indications for aetiology search or symptoms.^(21,22,23)

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