Aetiology and Management of Acute Cardiac Tamponade

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Objective: *To review current concepts in the aetiology and management of patients with cardiac tamponade.*

Data sources: *A review of articles reported on acute cardiac tamponade.*

Summary of review: *Cardiac tamponade is defined as a haemodynamically significant cardiac compression caused by pericardial fluid. The fluid may be blood, pus, effusion (transudate or exudate) or air and treatment involves correction of the cardiac diastolic restriction by removing pericardial fluid during either pericardiocentesis or thoracotomy.*

Pericardiocentesis is usually performed for urgent management of an acute tamponade. A thoracotomy is required when a tamponade exists following coronary artery bypass grafting, cardiac rupture, penetrating or closed cardiac trauma and aortic dissection or where a pericardial clot is likely. Removal of pericardial fluid by percutaneous catheterisation of the pericardial sac traditionally uses the subxiphoid 'blind' approach., However, pericardial aspiration is often currently performed under transthoracic echocardiographic guidance with the commonest site of needle entry being on the chest wall at or near the apex and the needle directed perpendicular to the skin. The pericardial fluid is drained using an indwelling 'pigtail' catheter, preferably with close monitoring of the pulmonary artery wedge pressure, as rapid removal of large volumes of pericardial fluid (> 500 mL) may result in a 'decompressive syndrome' causing pulmonary oedema. The fluid is sent for culture and Gram-stain and analysed for glucose, protein, lactate dehydrogenase, haemoglobin and white cell count. If appropriate the fluid is also analysed for amylase, and cholesterol and sent for cytology, serology and parasitic studies and viral, mycobacterial and fungal cultures.

Conclusions: *Cardiac tamponade may present as an acute or subacute syndrome of elevated central venous pressure and hypotension. Pericardiocentesis using echocardiographic guidance and right heart catheter control is often the method of choice for acute removal of fluid.* (**Critical Care and Resuscitation 2004; 6: 54-58**)

Key words: Cardiac tamponade, pericardiocentesis, pericardial infection

The pericardium consists of a thin serous membrane covering the epicardial surface (visceral pericardium) and a serous membrane-lined fibrous sac (parietal pericardium). They are fibrous structures with limited elastic properties. The pericardial space separates the two layers and contains approximately 20 mL of fluid.

Congenital absence of the pericardium or pericardiectomy are not associated with deleterious cardiovascular effects, indicating that the pericardium is not essential for life. Nevertheless, it does have several protective functions.[1] For example, it prevents acute atrial dilation of the heart during exercise and hypervolaemia, prevents acute atrial-ventricular valve regurgitation, lubricates the moving heart, and prevents spread of infection to the myocardium from surrounding structures.[2] An acute accumulation of pericardial fluid of greater than 100 mL will produce haemodynamic effects of tamponade whereas a chronic pericardial collection of fluid up to 2000 mL may occur without imposing any effect upon cardiac output.[3]

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Cardiac tamponade is defined as a haemodynamically significant cardiac compression caused by pericardial fluid.[4] The fluid may be blood, pus, effusion (transudate or exudate) or air.[5] The principal haemodynamic effect is a constraint on atrial filling with a reduction in atrial diastolic volume,[6] which causes an increase in atrial diastolic pressure.[7] During the early stages of cardiac tamponade, cardiac output and organ perfusion are maintained by an increase in the ejection fraction to 70% - 80% (the normal ejection fraction ranges from 50% - 70%), tachycardia and peripheral vasoconstriction.[3]

Causes

The causes of a cardiac tamponade include an acute accumulation of pericardial fluid from a ruptured myocardium (following myocardial infarction, blunt or penetrating cardiac trauma or cardiac perforation following cardiac catheterisation), proximal dissecting aortic aneurysm, carcinomatous infiltrate of the pericardium and acute pericarditis. As the fluid may be blood, blood stained (sero-sanguinous), pus, transudate or exudates, the aetiology of the tamponade may be classified as haemorrhagic, sero-sanguinous, serous or purulent (Table 1).

Clinical features

The classical presentation of a cardiac tamponade is an elevated venous pressure, decreased systemic arterial pressure and a quiet heart (i.e. Beck's triad).[8] However, in one study of 56 patients with cardiac tamponade the triad was often absent (most did not have a quiet heart and the blood pressure was often well maintained).[9]

The major symptoms are often dyspnoea (the patient often is found leaning forward or sitting in the knee-chest position to relieve the breathlessness), fatigue and light-headedness.

The signs include tachypnoea, tachycardia, hypotension (although hypertension may occur, particularly in patients with pre-existing hypertension),[10] shock (i.e. pale, cool clammy extremities and poor peripheral perfusion), faint heart sounds, pericardial rub and pulsus paradoxus. Pulsus paradoxus describes a pulse that demonstrates a systolic blood pressure reduction greater than 10 mmHg during quiet inspiration. It represents an exaggeration of the normal decline in pulse pressure on inspiration, which occurs due to a reduction in left ventricular stroke volume caused by a decrease in preload and increase in afterload. It is characteristically found in acute cardiac tamponade[9] although, it can also be found in right ventricular infarction, severe congestive cardiac failure, myocarditis, emphysema, acute asthma, hypovolaemic shock, pulmonary embolism, extreme obesity, ascites and pregnancy. Pulsus paradoxus may also be prevented from occurring in patients with cardiac tamponade if the patient's tidal volume is reduced (e.g. the patient becomes obtunded) or is mechanically ventilated or if severe left ventricular dysfunction, or a regional tamponade, atrial septal defect or aortic regurgitation coexist.[10-13]

Table 1 Causes of cardiac tamponade

Haemorrhagic
Trauma
Penetrating and blunt chest trauma
Cardiac catheterisation, pacemaker insertion, central venous cannulation, percutaneous coronary artery angioplasty
Post operative thoracotomy
Pericardiocentesis
External cardiac compression
Myocardial infarction
Rupture
Anticoagulation, thrombolytic therapy, antiplatelet agents
Dissecting aortic aneurysm
Anticoagulant therapy
Serous or sero-sanguinous
Neoplastic
Lung or breast cancer, lymphoma
Connective tissue diseases
Systemic lupus erythematosus, rheumatoid arthritis
Rheumatic fever, polyarteritis
Idiopathic thrombocytopenic purpura
Uraemia
Radiation therapy
Idiopathic
Purulent
Infectious
Bacterial
Viral (coxsackie B, influenza, infectious mononucleosis)
Mycobacterial, yeast

The central venous pressure is characteristically elevated (although it may not be elevated if the tamponade is localised to the left atrium or ventricle)[13] and does not increase on inspiration (i.e. Kussmaul's sign is negative).[14] The lungs are usually clear, although a large pericardial effusion may produce Ewart's sign (i.e. dullness to percussion and bronchial breathing at the left lung base due to compression of the left lung base). However, this is a non-specific finding and may occur in any patient with cardiomegaly.[15]

While cardiac tamponade may be suspected clinically in a patient with dyspnoea, a clear chest, elevated systemic venous pressure, tachycardia and paradoxical arterial pulse,[13] the patient may present with hypotension or shock without any of the characteristic clinical features.

Investigations

Echocardiography. This is the most reliable and convenient way of diagnosing pericardial fluid, clot and the haemodynamic effects of the effusion. It can detect signs of tamponade before clinical signs occur or an effect on cardiac output is found.[16] For example tamponade will be detected by an abnormal septal motion (both ventricular and atrial septa move sharply leftward on inspiration and reverse with inspiration), right atrial and right ventricular diastolic collapse (indicative of an elevated intrapericardial pressure that transiently exceeds the intra-cavity pressure with the collapse of the atrial and ventricular walls), and reduced respiratory variation of the diameter of the inferior vena cava (Doppler assessment of intracardiac flows and their relationship to respiration may also play a useful role in the diagnosis).[17] In patients without heart disease the ejection fraction is normal or increased.

In addition to detecting an effusion, it can assess the distribution of the effusion (e.g. loculated or concentric). Trans-oesophageal echocardiography will facilitate detection of a tamponade if a mitral valve or aortic valve prosthesis is present or a localised posterior clot exists in the post cardiac surgery patient.

Other investigations include

ECG. This may reveal sinus tachycardia, low-voltage complexes and non-specific ST segment and T wave changes or ST segment elevation due to pericarditis. Electrical alternans (due to a pendular swinging motion of the heart within a pericardial effusion) is characteristically seen only with a large effusion.3 Combined P and QRS alternation is said to be pathognomonic for tamponade.[18] Nonetheless, an ECG is largely used to rule out other causes of hypotension rather than confirm the diagnosis of cardiac tamponade.

Chest X-ray. The chest X-ray may be within normal limits although features suggestive of pericardial fluid include an enlarged globular cardiac shadow with loss of the hilar waist (if the pericardial effusion is greater than 250 mL) with a normal pulmonary vascular pattern. Other signs include prominence of the superior vena cava (reflecting an elevation in the central venous pressure), pleural effusions (transudates which may be caused by a raised central venous pressure) and the epicardial fat pad sign (which is best seen on the lateral chest radiograph as a radiolucent line between the epicardial fat and the mediastinal fat and represents the pericardium. It should be 2 mm or less, so any increase suggests fluid or thickening in the pericardium).

Haemodynamic monitoring

a) Arterial catheter. Direct assessment of the arterial pressure is useful as it allows identification of an abnormal pulsus paradoxus.
b) Right heart catheterisation. The characteristic haemodynamic changes associated with cardiac tamponade include:[19]
   - a right atrial pressure wave with a prominent 'x' descent (i.e. forward venous flow occurs only during systole) but not a prominent 'y' descent
   - equalisation of diastolic pressures (to within 3 - 4 mmHg) in the right atrium, right ventricle, pulmonary artery and left atrium (i.e. all average diastolic pressures at 12 - 25 mm/Hg in the absence of pulmonary oedema).[18]
   - pulsus paradoxus in the pulmonary artery pressure that is out of phase with the systemic pulsus paradoxus.

The differences between the two cardiac restrictive disorders of cardiac tamponade and constrictive pericarditis are listed in table 2.

Table 2. A comparison of the features of cardiac tamponade and constrictive pericarditis

	Tamponade	*Constrictive pericarditis*
Pericardial effusion	Present	Absent
Pericardial calcification	Absent	Often present
Dip and plateau pattern	Inconspicuous	Prominent
RAP waveform	Prominent 'x' descent	Prominent 'x' and 'y' descent
Pulsus paradoxus	Prominent	Inconspicuous
Kussmaul's sign	Absent	Often present
Diastolic 'knock'	Absent	Often present

Treatment

The patient is initially resuscitated with intravenous fluids to promote maximum filling of the heart. However, increasing the intravascular volume is usually only helpful in hypovolaemic patients as intravenous fluids in normovolaemic or hypervolaemic patients may only increase right-ventricular filling at the expense of the left ventricle and has had disappointing results in clinical trials.[20] In general, inotropic agents that increase the stroke volume and support systemic resistance are also used, although one reviewer recommended isoprenaline, as it reduces the cardiac size and diminishes the effective degree of tamponade while increasing cardiac output.[3]

The definitive treatment of cardiac tamponade is the removal of the cardiac diastolic restriction by either pericardiocentesis or thoracotomy. Pericardiocentesis is usually performed for urgent management of an acute tamponade (the acute removal of as little as 50 mL of fluid is often sufficient to correct the acute hypotension). A thoracotomy is often required when a tamponade exists following coronary artery bypass grafting, cardiac rupture, penetrating or closed cardiac trauma and aortic dissection, where continuous blood loss occurs and the source of the bleeding often requires surgical correction or where a pericardial clot is likely, which cannot be easily aspirated. It is also indicated when pericardiocentesis has failed to relieve the tamponade.

Pericardiocentesis involves removal of pericardial fluid by percutaneous catheterisation of the pericardial sac. The subxiphoid 'blind' approach to pericardiocentesis has been the traditional approach which involves placing the patient in a supine 30º - 45º head-up position to facilitate the movement of the effusion towards the anterior chest wall. A large-bore needle is inserted at the right side of the xiphisternum and advanced subcostally, aiming towards the tip of the left shoulder, with continuous suction applied to a syringe which is attached to the hub of the needle. The needle can be used as a chest ECG lead to detect myocardial contact indicated by ST segment elevation (although ECG monitoring may provide misleading results and is not uniformly recommended).[18] If blood is aspirated from a cardiac chamber, it clots, unlike blood withdrawn from the pericardial cavity; also, bloody pericardial fluid on a gauze swab separates, with a central deep red spot and a peripheral halo which is less red, whereas blood from a cardiac cavity spreads uniformly throughout the gauze swab. A haemoglobin estimation may also be performed on the aspirated fluid: if it is less than the intravenous haemoglobin estimation, it is likely to be haemorrhagic pericardial fluid.[3]

However, blind pericardial aspiration is not recommended as a routine procedure as it has an unacceptably high complication rate with a recorded mortality and technical complication rate as high as 6 and 50%, respectively.[21,22] Currently, pericardial aspiration performed under transthoracic echocardiographic guidance is the treatment of choice for percutaneous aspiration of the pericardial effusion.[21,23] The echocardiogram will not only detect the pericardial fluid, but will determine whether there is any haemodynamic effect (i.e. tamponade) whether it is largely clot (where a thoracotomy and surgical evacuation rather than pericardiocentesis may be required), whether it is loculated, where the fluid is predominantly found, the chest surface location closest to the effusion and the angle and distance the needle has to travel (without piercing intervening vital structures e.g. lung) before it enters the effusion. Using this approach the commonest entry of the needle is on the chest wall at or near the apex and directed perpendicular to the skin.[24] Any percutaneous site that is selected should avoid the internal mammary artery (3 - 5 cm from the parasternal border) and the vascular bundle at the inferior margin of each rib.[21]

Continuous intra-arterial blood pressure and pulmonary artery pressure measurements should be performed during pericardial aspiration. When pericardial entry occurs, the first 100 - 200 mL of fluid should be drained rapidly. Thereafter the remainder is drained slowly using an indwelling 'pigtail' catheter (inserted over a guidewire), preferably with close monitoring of the pulmonary artery wedge pressure, as rapid removal of large volumes of pericardial fluid (> 500 mL) may result in a 'decompressive syndrome' causing pulmonary oedema.[25] The fluid is sent for glucose, protein, lactate dehydrogenase, haemoglobin, white cell count, culture and Gram-stain. If appropriate the fluid is also sent for amylase, cholesterol, cytology, serology (rheumatoid factor, antinuclear factor, complement levels), viral, mycobacterial and fungal cultures and parasitic studies.

If there is no drainage after 24 - 72 hr and a repeat echocardiogram demonstrates no reaccumulation of fluid, the pigtail catheter is removed from the pericardial cavity.

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