Cardiac Tamponade

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KEYWORDS

• Pericardial effusion • Cardiac compression • Cardiac tamponade • Echo-Doppler findings

KEY POINTS

- Cardiac tamponade is caused by an abnormal increase in fluid accumulation in the pericardial sac, which, by raising intracardiac pressures, impedes normal cardiac filling and reduces cardiac output.
- The clinical manifestations typically reflect elevated right-sided filling pressures, reduced cardiac output, pulsus paradoxus, and imaging evidence of dynamic chamber compression.
- Treatment consists of needle pericardiocentesis, preferably guided by echocardiography, or surgical drainage.

Video content accompanies this article at http://www.cardiology.theclinics.com.

INTRODUCTION

Cardiac tamponade is caused by an abnormal increase in fluid accumulation in the pericardial sac, which, by raising intracardiac pressures, impedes normal cardiac filling. When the fluid accumulation is rapid, a marked increase in intrapericardial pressure can reduce inflow gradients to such low levels that compensatory reflexes that are activated to maintain cardiac output and blood pressure are overwhelmed and cardiogenic shock and death can occur. Less frequently, cardiac tamponade occurs due to localized, severe compression of cardiac chambers due to a mass, or a localized hematoma after cardiac or thoracic surgery.

In both instances, patients present with tachypnea, tachycardia, and hemodynamic instability with an elevated systemic venous pressure. A clinical hallmark of cardiac tamponade is pulsus paradoxus, or an abnormal decrease in systolic blood pressure with inspiration. Emergency echocardiography using both imaging and Doppler techniques is the primary method for rapid diagnosis and assessing the severity of tamponade. When possible, pericardiocentesis using echo guidance is used to drain pericardial effusions because of its high success rate and low procedural morbidity.

PATHOPHYSIOLOGY

Normal Pericardial Layers and Function

As discussed in other articles in this issue, a small amount of fluid (15–35 mL) exists in the pericardial sac between the smooth surfaced visceral and parietal pericardial layers to facilitate lubrication in the ever-beating heart.[1,2] The serosal visceral pericardium, which is the outermost layer of the myocardium, is thin, elastic, and translucent; and the fibrous parietal pericardium is thicker and inelastic. The visceral pericardium appears to aid diastolic elastic recoil and suction, whereas the parietal pericardium, with attachments to the sternum, diaphragm, vertebrae, and pleura, holds the heart in optimal

Conflicts of Interest: None.

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orientation for filling and ejection, limits excessive motion, and prevents excessive dilation from acute adverse events, such as mitral chordal rupture or right ventricular (RV) infarction. The parietal pericardium also provides a barrier to contiguous pulmonary infection or cancers.

The parietal pericardium envelopes the heart snugly, contributing more than 50% resting right atrial (RA) pressure and distributes this effect variably over the cardiac chamber pressures. Yet, due to pericardial reflections, irregular spaces between the pericardial layers exist, like the transverse and oblique sinuses (Fig. 1), that provide a pericardial reserve volume, which allows for modest changes in cardiac volume with inspiration and changes in body position to occur without the parietal pericardium raising intracardiac pressures (Fig. 2). When this reserve volume is exceeded by markedly increased right heart filling on inspiration (chronic obstructive pulmonary disease exacerbation or acute respiratory distress), or acute cardiac chamber dilation, both intrapericardial and intracardiac pressures rise due to the steep nature of the pericardial pressure-volume relation (see Fig. 2). This pericardial restraint increases all diastolic intracardiac pressures and results in enhanced and competitive filling between the right and left heart that alternates with the phase of respiration. This is why pulsus paradoxus can be observed in conditions such as status asthmaticus.

Pericardial Effusions

These exist when there is an increased volume of pericardial fluid of any type in the pericardial space. They can present acutely with symptoms or, when chronic, are more likely to be found incidentally after computerized tomography or MRI of the heart or chest or echocardiography ordered for a separate indication. A general rule is that any patient with unexplained jugular venous distension should have an echocardiogram to exclude pericardial effusion. Idiopathic asymptomatic small, medium, and even large pericardial effusions can be seen without tamponade (Fig. 3). When these are present, it can be assumed that they have accumulated slowly enough that the fibrous parietal pericardium added additional cells (stretched) so that pericardial pressure is normal or minimally raised.[1,2] The initial focus in idiopathic effusions, if there is no obvious systemic disease, is on ruling out malignancy such as lymphoma, breast, lung, or another type of cancer. Although the diagnostic yield of needle pericardiocentesis for malignancy is low, it can usually been done safely under echo guidance.[3] Alternatively, the pericardium can be biopsied as part of surgical pericardial window creation.

Pericardial fluid can be serous as seen in idiopathic pericarditis, serosanguineous as seen in other inflammatory or malignant conditions, purulent due to infections, or bloody as seen after inadvertent cardiac perforation from an invasive

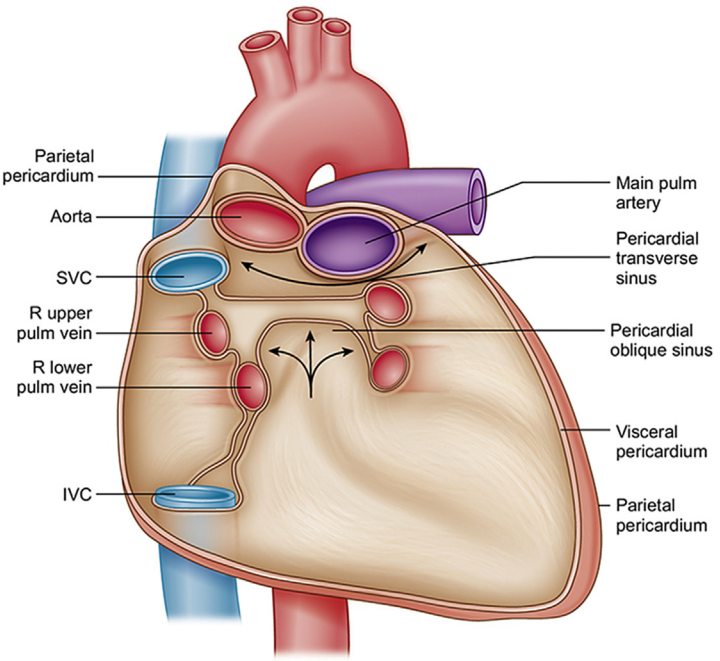

Fig. 1. Anatomic drawing of pericardial space with the heart removed showing the pericardial attachments to the great vessels and pericardial reflections, or transitions from parietal to visceral pericardium. The latter create irregular spaces between the pericardial layers such as the transverse and oblique sinuses as shown. IVC, inferior vena cava; SVC, superior vena cava.