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CHEST

Tamponade

Hemodynamic and Echocardiographic Diagnosis

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Cardiac tamponade is a medical emergency that can be readily reversed with timely recognition and appropriate intervention. The clinical diagnosis of cardiac tamponade requires synthesis of a constellation of otherwise nonspecific features based on an understanding of the underlying pathophysiological characteristics. Although echocardiographic examination is a central component of diagnosis, alone it is insufficient to establish the physiological diagnosis of hemodynamically significant cardiac tamponade. The hemodynamic diagnosis of cardiac tamponade requires clinical evidence of low cardiac output and stroke volume in the setting of elevated cardiac filling pressures, with evidence of increased sympathetic tone (eg, tachycardia, peripheral vasoconstriction), and exclusion of other causes of shock as the primary problem (particularly cardiogenic shock). The hemodynamic features of tamponade are revealed by considering the effects of pericardial constraint. Pulsus paradoxus and loss of the normal "y" descent of a jugular venous pressure waveform may be appreciated on clinical examination. When a pulmonary artery catheter is placed, equalization of diastolic pressures across all chambers is observed. Echocardiographic examination confirms the size, location, and other characteristics of the causal pericardial collection. Several echocardiographic features support the hemodynamic diagnosis of tamponade, including early diastolic collapse of the right ventricle, late diastolic collapse of the right atrium, respiratory variation in mitral valve inflow (akin to pulsus paradoxus), and decreased early filling (E wave) of mitral valve inflow (related to loss of the y descent). Echocardiographic examination then supports decisions about the early treatment and drainage of the tamponading effusion. CHEST 2017; ■(■):■-■

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Cardiac tamponade occurs when distributed or localized intrapericardial collections (and occasionally extrapericardial factors) raise intrapericardial pressure or locally constrain cardiac filling sufficiently to impact hemodynamics. Cardiac tamponade is classically considered "obstructive shock,"[1] although physiologically it involves restriction of cardiac chamber filling. The rate at which intrapericardial collections develop is critical, as the fibrous pericardium is acutely noncompliant but chronically can adapt to accommodate very large volumes of fluid without hemodynamic effect. The principal clinical manifestations of cardiac tamponade are reduced stroke volume, elevated filling pressures, and attendant compensatory changes associated with increased sympathetic tone (eg, tachycardia and

ABBREVIATIONS: CVP = central venous pressure; SHOCK = septic, hypovolemic, obstructive, cardiac

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increased systemic vascular resistance). These manifestations are not specific for cardiac tamponade[2,3] and can be further complicated in ICU settings, in which multiple cardiac and noncardiac causes may account for the observed physiological abnormalities, and causes of shock can overlap. Furthermore, patients in the ICU are frequently mechanically ventilated, receiving vasoactive medications, and occasionally supported by circulatory-assist devices. These factors also contribute to difficulty in interpreting echocardiographic findings associated with cardiac tamponade.[4] Nevertheless, cardiac tamponade is primarily a clinical diagnosis, and clinicians must be familiar with its hemodynamic and echocardiographic features. Prompt diagnosis and treatment are essential to improving patient outcomes.[5]

This review focuses on the early clinical examination, which raises the possibility of cardiac tamponade, and then the hemodynamic and echocardiographic diagnostic features, based on an understanding of the underlying pathophysiological characteristics. The list of conditions that may lead to cardiac tamponade is long (Table 1) and is touched on only peripherally.

Clinical Findings of Cardiac Tamponade

Rapid Bedside Assessment/Clinical Suspicion

Timely recognition of obstructive shock due to cardiac tamponade and exclusion of other causes of shock is facilitated by stepping through the "SHOCK" mnemonic at the bedside.[6] Septic (S) or distributive shock is distinguished from other forms of shock by evidence of increased cardiac output—warm shock rather than cold. Hypovolemic (H) shock is then separated from the remaining forms of shock by evidence of low cardiac filling pressures (jugular venous pressure, central venous pressure [CVP], tissue turgor, history of volume loss) vs high cardiac filling pressures associated with obstructive (O) and cardiogenic (C) shock. Obstructive shock is then distinguished from cardiogenic shock by evidence that the lungs are clear on physical examination and chest radiography.[7] Pulmonary embolism, pneumothorax, and cardiac tamponade are the most common causes of obstructive shock, so the physical examination next focuses on distinguishing these causes. Using this approach, the possibility of cardiac tamponade can be recognized quickly at the bedside of the hypotensive or hemodynamically unstable patient.

TABLE 1] Causes of Pericardial Effusion or Increased Juxtacardiac Pressures, and Tamponade

Increased pericardial pressures
Hemopericardium
Aortic dissection (including traumatic)
Ventricular rupture (most commonly post-MI)
Postthoracotomy/pericardiotomy
Postcoronary intervention, biopsy, pacemaker placement
Post-transcatheter aortic valve replacement
Post-myocardial infarction, post-cardiotomy syndrome (Dressler syndrome)
Malignant effusion
Postradiation
Renal failure
Inflammatory diseases (eg, systemic lupus erythematosus, rheumatoid arthritis, mixed connective tissue disease)
Infection (eg, viral pericarditis, TB, purulent)
Hypothyroidism
Idiopathic pericardial effusion
Pneumopericardium (tamponade almost exclusive to pediatric patients)
Extrapericardial increase in pressure
Pleural effusion
Mechanical ventilation
Tension pneumothorax
Increased intraabdominal pressure

Physical Examination Findings

First, a relevant history pointing toward an underlying cause (Table 1) can be invaluable. In view of the preceding rapid bedside assessment, a patient with hemodynamically significant cardiac tamponade will classically display physical examination findings consistent with low cardiac output and stroke volume with high right-sided (venous) filling pressures and compensatory increased sympathetic tone. Evidence of a low cardiac output, and hence a low stroke volume, include a low mean arterial pressure and a low pulse pressure, which also manifests as a "thready pulse" on palpation. Other features include central and peripheral cyanosis, delayed capillary refill, and mottled and cool skin. Evidence of an increase in sympathetic tone includes tachycardia, anxiety, diaphoresis, and poor peripheral perfusion.

During inspiration, increased filling of the right-sided chambers can further reduce the limited space within the pericardium so that the left-sided chambers are

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