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GUEST EDITOR'S PAGE

Spectrum of Pericardial Tamponade

A New Look at an Old Problem

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Pericardial tamponade encompasses the symptoms and signs of heart compression most typically caused by pericardial effusion. Hemodynamically, an increase in intrapericardial pressure due to pericardial effusion results in an increase in intracardiac pressures with pressure equilibration. It is a persistent misconception that pericardial tamponade is an all-or-none phenomenon (1). In clinical practice, echocardiography commonly serves as a tool to diagnose and quantify pericardial effusion as well as assess its hemodynamic effects. Because of the widespread use of echocardiography and variability of clinical presentation of patients with pericardial effusion, a variety of terms have emerged that attempt to cover the whole spectrum of pericardial tamponade: "pre-tamponade," "impending tamponade," "echo tamponade," and "hemodynamic tamponade," among others. These terms propagate confusion among clinicians, creating difficulties in clinical decision making.

COMPENSATORY MECHANISMS

The degree of heart compression with any degree of pericardial effusion depends on the balance between pericardial compliance and the rate of fluid accumulation. Intrapericardial pressure increases significantly with rapid fluid accumulation, while the rise is blunted in slowly accumulating effusion because of the adaptive increase in pericardial compliance ("pericardial stretch") (2). The compression effect of pericardial effusion causes predictable changes in cardiac hemodynamics: pressures within cardiac chambers increase and equilibrate with intrapericardial pressures, systemic venous returns becomes markedly systolic predominant, and diastolic filling of the right and left chambers shows exaggerated respiratory dependency. These changes can be assessed and quantified using echocardiography. Chamber collapse, inferior vena cava engorgement, and flow variation across the atrioventricular valves have been well described as echocardiographic evidence of pericardial tamponade. The systemic compensatory response to pericardial tamponade includes sympathetic and neurohumoral activation and renal sodium retention, but no increase in natriuretic peptides is typically seen (2). Importantly, the overall clinical picture can be compounded by comorbid conditions and the overall cardiovascular reserve.

CLINICAL PRESENTATION

Pericardial tamponade is not a binary phenomenon but rather a spectrum of hemodynamic abnormalities. This has been elegantly demonstrated in the study by Reddy et al (3), which relied on a comprehensive hemodynamic assessment of patients with pericardial effusion undergoing diagnostic or therapeutic pericardiocentesis. The investigators defined 3 groups of patients based on hemodynamic findings before the drainage: those in whom: 1) intrapericardial pressure was less than right atrial and pulmonary arterial wedge pressures; 2) intrapericardial pressure was equilibrated with right atrial but not pulmonary arterial wedge pressure; and 3) intrapericardial pressure was equilibrated with right atrial and pulmonary arterial wedge pressures. Obviously, the hemodynamic improvement following pericardiocentesis was greatest among group 3 patients, but even patients in group 1 had favorable hemodynamic changes, that is, a decrease in the right atrial pressure and pulmonary arterial wedge pressure (3).

Clinically, pericardial tamponade describes 2 distinct presentations: acute and subacute

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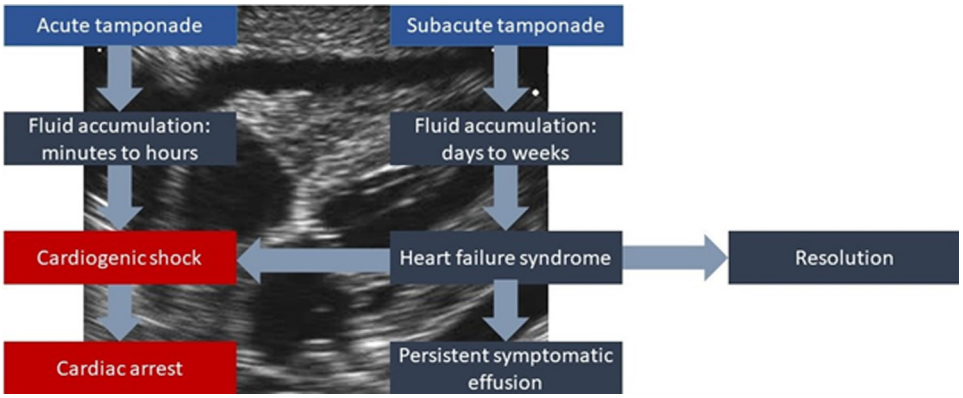
FIGURE 1 Natural History of Pericardial Tamponade Without Pericardial Drainage as 2 Distinct Clinical Presentations, Acute and Subacute Pericardial Tamponade



tamponade. Acute tamponade results from pericardial fluid accumulation within minutes to hours. Chamber perforation during a cardiac procedure is a typical example. Rapid fluid accumulation in an incompliant pericardial sack has an immediate compression effect that overwhelms the compensatory mechanisms. Acute tamponade results in cardiogenic shock due to the sudden decrease in cardiac output, commonly with a small to moderate amount of pericardial fluid, progressing to cardiac arrest if not corrected. On the other hand, subacute tamponade due to pericardial fluid accumulation within days to weeks typically presents as heart failure syndrome caused by an increase in cardiac filling pressures. Slower accumulation of moderate or large pericardial effusion commonly allows the full range of compensatory mechanisms. Dyspnea on exertion is almost invariable. Most patients are tachycardic on examination but are not hypotensive. To the contrary, many patients with subacute pericardial tamponade are actually hypertensive on admission. In studies of pericardial tamponade, the mean systolic blood pressure ranged from 127 to 144 mm Hg (4). In one study, blood pressure readings upon initial presentation did not show a significant association with markers of effusion severity or the need for drainage in patients with moderate and large pericardial effusion (5).

Subacute pericardial tamponade can progress to frank cardiogenic shock with continued fluid accumulation and failure of the compensatory mechanisms, but that is not the invariable outcome (**Figure 1**). Because pericardial tamponade is a spectrum, some patients experience complete resolution without pericardiocentesis, especially with a clearly reversible cause of effusion (6). Clinical findings in subacute tamponade are notoriously nonspecific (7). Echocardiography (more so than other imaging modalities) is an essential noninvasive tool that is used to diagnose and quantify pericardial effusion as well as assess its hemodynamic impact. At the same time, echocardiographic findings have known limitations, and overreliance on echocardiography may lead to unnecessary procedures. As an example, a study by Merce et al (8) demonstrated that one-third of patients with pericardial effusion but without clinical features of pericardial tamponade had at least 1 chamber collapse on echocardiography. Therefore, an integrative approach based on clinical features, size of the effusion, and echocardiographic findings is reasonable in identifying high-risk patients with subacute tamponade that would benefit from pericardial drainage as opposed to a "wait and see" approach (1).

One potential strategy to risk stratification of patients with suspected subacute tamponade is based on a pericardial effusion scoring index (9). Similarly, a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases suggested a model that is, in essence, a 3-step algorithm based on etiology, clinical presentation, and imaging findings (6). Although these approaches lack robust clinical validation, they provide a framework for future studies in this field (10).

In conclusion, pericardial tamponade is not a uniform entity, and it includes 2 distinct clinical presentations. An integrative approach combining

clinical and imaging findings is essential in treating patients with subacute pericardial tamponade.

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