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EARLY MANAGEMENT OF PERICARDIAL TAMPONADE AT THE EMERGENCY DEPARTMENT; REVIEW

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Abstract:
This paper examines the advantages and disadvantages of emergency department (ED) imaging modalities that have facilitated a more rapid diagnosis of pericardial effusions and cardiac tamponade. With earlier detection, the necessary treatments can be administered. Therefore, accurate and quick diagnosis is essential. Our search was conducted using an electronic search of MEDLINE, Embase, PsychInfo, Cochrane, and CINAHL databases until May 2022. Our search strategy included the phrases pericardial tamponade AND in conjunction with pericardial effusions, diagnosis, and emergency department (ED). Cardiac tamponade is a common cardiac emergency that requires prompt diagnosis and treatment. Interventional cardiologists must have a comprehensive understanding of the spectrum of professional and hemodynamic changes in patients with pericardial effusion. Clinicians must recognize the physiology of cardiac tamponade, especially in cases without a large pericardial effusion, and correlate clinical tamponade symptoms with echocardiographic findings. The draining of cardiac tamponade saves lives. Emergency medical professionals have been shown to have a high degree of diagnostic accuracy, reducing the need for time-consuming or costly detailed or consultative echocardiography.

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INTRODUCTION:

Cardiac tamponade is a fatal complication of pericardial effusion for which prompt diagnosis and treatment are crucial for a positive outcome [1].
Pericardial effusion is frequently observed in clinical practice, either as an incidental observation or as a sign of a systemic or cardiac disease. From moderate, asymptomatic effusions to cardiac tamponade, the spectrum of pericardial effusions extends. In addition, pericardial effusion can develop gradually or abruptly [1,2]. Cardiac tamponade is defined as a substantial hemodynamic cardiac compression caused by pericardial fluid [3]. The fluid may be blood, pus, effusion, or air [3]. The primary hemodynamic effect is a restriction on atrial filling due to a decrease in atrial diastolic volume, resulting in an increase in atrial diastolic pressure [4]. During the early phases of cardiac tamponade, cardiac outcome and organ perfusion are supported by an increase in the ejection fraction from 50% to 70% to 70% to 80%, tachycardia, and peripheral vasoconstriction [3].

In addition to intrathoracic pressure, the intrapericardial pressure varies from 5 to +5 mmHg during the breathing cycle. The natural pressure-volume curve of pericardium is a J-shaped curve with an original shallow segment, which allows the pericardium to stretch somewhat in response to volume or postural modifications, and a high section indicating pressure increases. This suggests that the abrupt accumulation of a restricted amount of fluid faster than it can be absorbed may lead to a large increase in pericardial pressure and, eventually, cardiac tamponade. On the other hand, a slow-moving but persistent pericardial distension may result in the accumulation of a large amount of fluid, such as 1- 2 l, with only a little increase in pericardial pressure and no hemodynamic implications [1].

Although cardiac tamponade is primarily a medical diagnosis based on elevated systemic venous pressure, tachycardia, dyspnea, and paradoxical arterial pulse, which is frequently accompanied by hypotension, echocardiography remains the gold standard for confirming the existence and hemodynamic effects of the tamponade [6].

The current method of choice for pericardiocentesis is echocardiography-guided pericardiocentesis, which has the highest rate of procedural success and the lowest rate of significant complications compared to blind or surgical procedures [6].

The goal of this review was to examine the presenting medical characteristics and diagnostic procedures of patients with pericardial tamponade in the emergency department. Diagnostics of pericardial tamponade in the emergency department (ED) are essential for proper patient management, as untreated early pericardial tamponade can lead to worsening hemodynamic instability and a possible heart attack if left untreated.

In this literature review, we discuss the provided diagnostic approaches as clinical background and evaluation, which, despite being the mainstay of initial analysis, are supplemented by very early analysis investigation, especially when clinical symptoms are subtle and/or life-threatening conditions are suspected. Due to this, we examine the advantages and downsides of emergency department (ED) imaging modalities that have facilitated a more faster detection of pericardial effusions and cardiac tamponade. With earlier detection, the necessary treatments can be administered. Therefore, accurate and quick diagnosis is essential.

METHODOLOGY:

We did an electronic search for this review in MEDLINE, Embase, PsychInfo, Cochrane, and CINAHL databases through May, 2022. Our search strategy included the phrases pericardial tamponade AND coupled with pericardial effusions, diagnosis, and emergency department (ED). Our search was limited to English-language, human-subject literature, and we only considered studies that included data pertinent to the diagnosis of pericardial tamponade.

DISCUSSION:

- **Etiologies:**

Cardiac tamponade is a life-threatening condition that results from the sudden and/or excessive buildup of fluid in the pericardial space. The condition restricts the appropriate filling of the cardiac chambers, disrupting normal hemodynamics and ultimately causing hypotension and heart attack [1].

Pericardial disorders may exist independently or as part of a systemic disorder. Cardiac tamponade is a dangerous disorder characterized by reduced or rapid pericardial fluid buildup and subsequent heart compression [1]. The majority of cases of pericarditis are due to idiopathic causes, which account for roughly 90% of cases. Other causes include infections caused by viruses, bacteria, or TB. Medical and clinical causes of cardiac tamponade, generally indicating acute and chronic onsets respectively, can be distinguished. **Table 1** provides a summary of the causes of cardiac tamponade.

Table 1. Causes of cardiac tamponade [7].

Cause	Onset
Bleeding following cardiac surgery	Immediate/acute; may be up to 2 weeks
Trauma	Immediate
Aortic dissection	Acute
Malignancy	Chronic
Idiopathic	Chronic
Infective	Acute/chronic
Pericarditis	Chronic
Massive pleural effusion	Acute/chronic

- **Types of cardiac tamponade**

Low-pressure tamponade occurs at diastolic pressures between 6 and 12 mm Hg and is typically limited to patients with hypovolemia and severe systemic disease, bleeding, or malignancy, or hypovolemia after diuresis [8]. Individuals are often weak and normotensive, with dyspnea on exertion and no definitive pulsus paradoxus, but with characteristic changes in transvalvular diastolic Doppler flows of the respiratory system. The low-pressure effusion equilibrates initially only with right-sided diastolic pressures during inspiration (" inspiratory tracking") [9]. A fluid challenge with one liter of warm saline can enhance the kinetics of tamponade [10].

Extreme beta-adrenergic drive is attributable to hypertensive cardiac tamponade, which has all the typical characteristics of tamponade and occurs at high and actually high arterial blood pressures (including over 200 mm Hg). Damaged individuals often have a history of hypertension [11]. Regional cardiac tamponade occurs when any cardiac region is pressured by loculated effusions, which are typically accompanied by local pericardial additions, particularly following cardiac surgery [8]. Occasionally, the regular hemodynamic abnormalities are only detected in compressed chambers or regions. However, loculation can also generate classic tamponade, most likely by constricting the uninvolved pericardium; for instance, loculated effusions following cardiac surgery may contain hematomas across the right atrium and atrioventricular groove [12]. Localized right atrial tamponade may also cause right-to-left shunting in the presence of a patent foramen ovule or atrial septal abnormalities [13].

After a right ventricular infarction, loculated effusion can cause a selective right-heart tamponade in which the right atrial pressure is greater than the left atrial pressure [12]. The absence of pulsus paradoxus (Table 2) makes it difficult to identify this kind. Given that a constrictive epicarditis underpins the pericardial effusion, effusive- constrictive pericarditis is characterized by integrated clinical, imaging, and hemodynamic symptoms. In some patients with inflexible parietal and natural pericardium, tamponade can be accompanied by a modest fluid collection. These patients are diagnosed as effusive-constrictive pericarditis when drainage of pericardial fluid does not cause intracardiac pressures to return to normal [14].

Table 2. Conditions Leading to the Absence of Diagnostic Pulsus Paradoxus in Cardiac Tamponade [12-14].

Condition	***Consequence***
Extreme hypotension, as in shock, and even severe tamponade	May make respiration-induced pressure changes unmeasurable
Acute left ventricular myocardial infarction with occasional effusion causing tamponade	—
Pericardial adhesions, especially over the right heart	Volume changes impeded
Local (usually postsurgical) pericardial adhesions	Local cardiac compression by loculated fluid
Pulmonary vein and left ventricular diastolic pressures and left ventricular stiffness markedly exceed those of the right ventricle	Reduced effects of respiration on right-heart filling
Right ventricular hypertrophy without pulmonary hypertension	Causes right-sided resistance to the effects of breathing
Severe aortic regurgitation, with or without severe left ventricular dysfunction	Produces sufficient regurgitant flow to damp down respiratory fluctuations
Atrial septal defects	Increased inspiratory venous return balanced by shunting to the left atrium
Some cases of low-pressure tamponade	Makes marked respiratory changes in blood pressure diagnostically insignificant

- **Diagnostic methods:**

Based on the typical signs of cardiac tamponade, thoracic expert Beck characterized this condition in 1935 as involving hypotension, increased jugular venous pressure, and a muffled heart [16]. This triad has been observed in "surgical tamponade" resulting from intrapericardial hemorrhage caused by trauma, myocardial or aortic tear. Patients with gradually accumulating pericardial fluid have a shortage, which is one of the major contributors [17]. Absolute and relative hypertension are the two different forms of hypertension. Acute cardiac tamponade is typically associated with low blood pressure (100 mmHg), whereas subacute and chronic tamponade is associated with slightly higher blood pressure. Due to the enhanced adrenergic tone and distribution of catecholamines, [18] hypertensive patients may have a consistent or even mild increase in blood pressure concurrent with cardiac tamponade. Infectious or immune-mediated pericarditis may be correlated with a high body temperature (**Box1**).

During the physical examination, it is important to identify main distinctive signs, such as neck blood vessel dilation with elevated jugular venous pressure, pulsus paradoxus, and diminished heart sounds on cardiac auscultation. Kussmaul first reported pulsus paradoxus in 1873 as a noticeable decrease of radial pulse on inspiration in patients with cardiac tamponade ("waxing and waning" of the outer pulse, in contrast to the unchanging endurance of the apical cardiac impulse) [19]. Pulsus paradoxus is defined as an inspiratory fall in systolic blood pressure of at least 10 mmHg. Recording the systolic stress at which Korotkoff sounds are first audible and the systolic stress at which they are audible throughout the full breathing system cycle is straightforward.

Box 1. Physical exam findings include [15]:

- sinus tachycardia;
- elevated jugular venous pressure;
- pulsus paradoxus;
- pericardial friction rub (from pericarditis, if present);
- distant heart sounds (from heart sound muffling related to the pericardial effusion); and
- Kussmaul's sign (rarely) – increase in jugular venous pressure during inspiration.

ECG may reveal sinus tachycardia, low voltage complexes, nonspecific ST segment and T wave changes, or ST segment elevation due to pericarditis. Electrical alternans (caused by a pendular swinging movement of the heart within a pericardial effusion) is typically observed only in the presence of a large effusion [23]. According to [24], dual P and QRS rotation is pathognomonic for tamponade. However,

an ECG is typically used to rule out other causes of hypotension rather than confirm a cardiac tamponade diagnosis.
If the pericardial effusion is greater than 250 mL, the upper body X-ray may be within normal limits, but suggestive of pericardial fluid are a larger globular cardiac shadow with loss of the hilar waist and a normal pulmonary vascular pattern [19]. Other indicators include prominence of the superior vena cava (reflecting an increase in the central venous pressure), pleural effusions (transudates that may be caused by an increase in the central venous pressure), and the epicardial fat pad indicator (best seen on the side upper body radiograph as a radiolucent line between the epicardial fat and the mediastinal fat and representing the pericardium). It must be 2 mm or less, therefore any increase signals pericardial fluid or thickening) [24].

- **Transvalvular pulse wave Doppler**

Using Doppler mode, changes in blood flow throughout the heart valves that suggest cardiac tamponade can be detected. As a result of the breathing cycle, there is typically some variation in flow across the valves. This respiratory system difference in blood circulation across the valve is accentuated during cardiac tamponade. This is best visualized by imaging the tricuspid valve and searching for a greater blood inflow during inspiration compared to expiration [25]. Although these new echocardiographic findings may aid in the medical diagnosis of acute tamponade, severe hypovolemia or ventricular hypertrophy may mask these signs.

Cardiac tamponade must be distinguished from other causes of diminished cardiac output, such as cardiogenic shock, in which a primary myocardial disease renders the heart incapable of producing sufficient cardiac output to sustain systemic perfusion. Massive myocardial infarction is one of the most typical causes of cardiogenic shock.
The majority of individuals with cardiac tamponade (> 70%) have been documented to have five characteristics: dyspnea (85-90%), cardiomegaly on breast radiograph (89%), pulsus paradoxus (82%), tachycardia (77%) and high jugular venous pressure (76%) [26]. In the context of a significant pericardial effusion, the presence of pulsus paradoxus (inspiratory decrease in systolic blood pressure > 10 mmHg) increased the chance of cardiac tamponade (probability proportion of 3.3) [27]. Surprisingly, hypotension and quiet heart sounds (respectively 26% and 28%) were insensitive. Due to the fact that cardiac tamponade is primarily a medical diagnosis, echocardiographic indicators are only confirming [26].

Large pulmonary emboli and stress pneumothorax are two other potential diagnoses. Infrequently, a tension pneumopericardium can mimic acute cardiac tamponade, albeit with a distinctive mill-wheel murmur. This issue can manifest after a chest wall penetrating injury, a burst esophagus, and a bronchopericardial fistula [26]. In individuals with a subacute onset of symptoms, the differential diagnosis should rule out constrictive pericarditis, congestive heart failure, severe liver disease with cirrhosis, and Ebstein anomaly almost never.

- **Prognosis**

Cardiac tamponade is one of the medical emergencies. The diagnosis is affected by quick recognition and care of the problem as well as the underlying causes of tamponade. Without treatment, cardiac tamponade has quick and widespread fatal outcomes.
Haneya et al. (2005-2011) investigated the effects of timing and indicator of reexploration for blood loss or tamponade following cardiac surgical treatment in 209 patients and found that reexploration was related with greater mortality and morbidity rates [28]. Multivariate analysis revealed that the adverse consequences of reexploration (e.g., blood loss, transfusion requirements) rather than the treatment itself were independent risk factors for mortality. Those whose reexploration was delayed and who dealt with cardiac tamponade were more likely to have poor results [28].
Le et al. concluded, based on prior research findings, that the use of several mediastinal breast tubes during cardiac surgery has no advantage over the use of a single chest tube in preventing return to the operating room for blood loss or tamponade [29].
To prevent recurrence, all persons must receive treatment for the underlying cause of the disease in addition to treatment for the tamponade.

In a study of patients with cardiac tamponade, Cornily et al observed a 1-year mortality rate of 76.5% in patients whose tamponade was caused by a fatal disease, compared to 13.3% in those without a fatal disease. The detectives kept in mind a typical survival time of 150 days for patients with cancer [30].

CONCLUSION:

Ventricular tamponade is the buildup of pericardial fluid, blood, pus, or air within the pericardial area, which causes an increase in intra-pericardial pressure, hence restricting cardiac filling and diminishing cardiac output. Cardiac tamponade is a cardiac

emergency requiring hospitalization and is lethal if not discovered and treated immediately. The medical diagnosis is supported by clinical suspicion and echocardiographic evidence of hemodynamic compromise.

Cardiac tamponade is a common cardiac emergency that requires prompt diagnosis and treatment. Interventional cardiologists must have a comprehensive awareness of the spectrum of professional and hemodynamic changes in patients with pericardial effusion. Clinicians must understand the physiology of cardiac tamponade, especially in patients without a substantial pericardial effusion, and correlate clinical tamponade symptoms with echocardiographic findings. The draining of cardiac tamponade saves lives. Emergency medical personnel have been shown to have a high degree of diagnostic accuracy, reducing the need for time-consuming or costly detailed or consultative echocardiogram. Co-occurring morbidity and mortality are reduced when a high index of suspicion is paired with adequate diagnostic criteria.

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