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AN OVERVIEW OF PROPER DIAGNOSIS OF PERICARDIAL TAMPONADE AT THE EMERGENCY DEPARTMENT (ED).

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Abstract:

This paper focus on available imaging modalities in the emergency department (ED) which have allowed a more rapid diagnosis of pericardial effusions and cardiac tamponade, their benefits and disadvantages. With earlier discovery, the required treatments can be implemented. Consequently, precise and prompt diagnosis is crucial. Our search was developed an electronic search in MEDLINE, & Embase, PsychInfo, Cochrane, CINAHL databases, through the time up to December 2019, our search strategy used several terms including; ***pericardial tamponade*** *AND combined with; pericardial effusions, diagnosis, emergency department (ED). Cardiac tamponade is a typical cardiac emergency needing timely diagnosis and intervention. A thorough understanding of the spectrum of professional and hemodynamic modifications in patients with pericardial effusion is crucial for interventional cardiologists. Clinicians need to recognize the physiology of cardiac tamponade, particularly in cases without large pericardial effusion, and associate the indications of clinical tamponade along with the echocardiographic results. The drainage of cardiac tamponade is life preserving. Emergency medical professionals have been revealed to have a high degree of accuracy in making the diagnosis, decreasing the requirement for the time or expense of detailed or consultative echocardiography. A high index of suspicion with appropriate diagnostic arcades decreases the concomitant morbidity and death.*

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INTRODUCTION:

Cardiac tamponade stands for a lethal problem of pericardial effusion where fast identification and management are important for a desirable result [1]. Claudius Galen (131-201 AD) first defined pericardial effusions in gladiators struggling with stab wounds to the chest and the English doctor, Richard Lowe (1669) first explained its physiology [2]. It took an additional two hundred years for the term "cardiac tamponade" to be coined by the German surgeon Edmund Rose [2]. Today cardiac tamponade is acknowledged as a crucial diagnosis to omit during heart attack in global advanced life support algorithms.

Cardiac tamponade is determined as a hemodynamically important cardiac compression leaded by pericardial fluid [3] .The fluid could be blood, pus, effusion (transudate or exudate) or air [3] .The principal hemodynamic effect is a constraint on atrial full of a reduction in atrial diastolic quantity, which creates a raise in atrial diastolic pressure [4] .During the early stages of cardiac tamponade, cardiac outcome and organ perfusion are supported by an elevation in the ejection portion to 70% - 80% (the regular ejection fraction varies from 50% - 70%), tachycardia and peripheral vasoconstriction [3].

Variations from − 5 to +5 mmHg is the physical intrapericardial pressure, in addition to intrathoracic pressure, during the breathing cycle. The natural pressure- volume curve of pericardium is a J-shaped curve with an original shallow section, which enables the pericardium to slightly stretch in response to volume or postural adjustments, and a high part showing increases in pressure. This indicates that sudden accumulation of restricted amount of fluid, faster than it can be absorbed, may cause a significant increase in pericardial pressure and ultimately to cardiac tamponade. On the other hand, a slow-moving but consistent pericardial distension may result in accumulation of substantial amount of fluid, such as 1- 2 l, with just a small raise in pericardial pressure and no hemodynamic consequences [1].

Although cardiac tamponade is basically a medical diagnosis, on the basis of elevated systemic venous pressure, tachycardia, dyspnea, and paradoxical arterial pulse, which is regularly accompanied by hypotension, echocardiography is still the criterion of care to confirm the existence and hemodynamic effects of the tamponade [6] .Echocardiography-guided pericardiocentesis is the present method of option, which has the greatest rate of procedural success and the most affordable rate of major complications compared with blind or surgical approaches [6].

The purpose of our research study was to investigate the presenting medical features and diagnosis techniques of emergency department patients with pericardial tamponade. ED diagnostics of pericardial tamponade is important to appropriate patient care, since if left unmanaged, early pericardial tamponade can cause intensifying hemodynamic instability and feasible heart attack.
In this literary works review we discuss the provided diagnostic approaches as clinical background and evaluation, although they are the mainstay of first analysis, it is complemented by very early analysis investigation particularly if clinical indicators are subtle and/or life-threatening conditions are thought. Due to that we focus on available imaging modalities in the emergency department (ED) which have allowed a more rapid diagnosis of pericardial effusions and cardiac tamponade, their benefits and disadvantages. With earlier discovery, the required treatments can be implemented. Consequently, precise and prompt diagnosis is crucial.

METHODOLOGY:

Our search was developed an electronic search in MEDLINE, & Embase, Psych Info, Cochrane, CINAHL databases, through the time up to December 2019, our search strategy used several terms including; **pericardial tamponade** AND combined with; pericardial effusions, diagnosis, emergency department (ED). Articles search was limited to English published, and Human subject related articles, we included only those trails which discussing evidence related to the diagnosis of pericardial tamponade.

DISCUSSION:

- **Causes of Cardiac Tamponade**

Cardiac tamponade is a serious ailment that takes place after abrupt and/or excessive accumulation of fluid in the pericardial space. The state limits the suitable filling of the cardiac chambers, disturbing regular hemodynamics, and eventually triggering hypotension and heart attack [1].

Pericardial diseases may be separated or part of a systemic ailment. Cardiac tamponade is a serious condition due to reduce or quick pericardial accumulation of liquid with succeeding compression of the heart [1]. The root cause of pericarditis is mostly idiopathic that makes up around 90% of instances. Various other causes are infections triggered by infections, bacteria or tuberculosis. Causes of cardiac tamponade can be separated into medical and clinical reasons, commonly representing acute and chronic beginnings respectively. The root causes of cardiac tamponade are summed up in Table 1.

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

- **Variant forms of cardiac tamponade**

Low-pressure tamponade happens at diastolic pressures of 6 to 12 mm Hg and is essentially restricted to individuals with hypovolemia and excessive systemic illness, hemorrhage, or cancer, or in patients with hypovolemia after diuresis [8]. Individuals are weak and usually normotensive, with dyspnea on effort and no diagnostic pulsus paradoxus, however with characteristic breathing system modifications in transvalvular diastolic Doppler flows. The low-pressure effusion equilibrates only with right-sided diastolic pressures and does so at first only throughout inspiration (" inspiratory tracking") [9]. A fluid challenge with a liter of warm saline can boost tamponade dynamics [10].

Hypertensive cardiac tamponade with all the standard attributes of tamponade, takes place at high and actually high arterial blood pressures (also over 200 mm Hg) and is attributed extreme beta-adrenergic drive. Damaged patients typically have had antecedent high blood pressure [11]. Regional cardiac tamponade occurs when any kind of cardiac area is compressed by loculated effusions, which are generally accompanied by local pericardial additions, especially after cardiac surgical treatment [8]. Occasionally the regular hemodynamic irregularities are discovered only in the compressed chambers or areas. Nonetheless, loculation can in addition produce classic tamponade, most likely by tightening up the uninvolved pericardium; for example, loculated effusions after cardiac surgical procedure may include hematomas over the right atrium and atrioventricular groove [12]. Localized right atrial tamponade may also create right-to-left shunting with a patent foramen ovule or defects of atrial septal [13].

After right ventricular infarction, loculated effusion can trigger selective right-heart tamponade in which right atrial pressure is higher than left atrial pressure [12]. The lack of pulsus paradoxus (Table 2) makes this type difficult to recognize. Effusive-constrictive pericarditis is characterized by consolidated clinical, imaging, and hemodynamic signs, given that a constrictive epicarditis underlies the pericardial effusion. In some patients with significant, rigid parietal and natural pericardium, tamponade can come with a small of accumulation of fluid. Effusive- constrictive pericarditis is disclosed in these patients when water drainage of pericardial fluid does not trigger intracardiac pressures to go back 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

- **Clinical diagnosis**

In 1935 thoracic specialist Beck based on the conventional findings of cardiac tamponade defined this health problem as an including hypotension, enhanced jugular venous pressure, and a muffled heart [16]. This triad has been identified in "surgical tamponade" with acute haemodynamic collapse as a result of intrapericardial hemorrhage brought on by trauma, myocardial or aortic tear. Among the main factors is shortage in patients with gradually accumulating pericardial liquid [17]. There are 2 types of hypertension: absolute or relative. Acute cardiac tamponade is mostly gotten in touch with low high blood pressure (<100 mmHg) however blood pressure is just little less in subacute, chronic tamponade. Individuals with hypertense may have regular or even light rise of high blood pressure concomitant to cardiac tamponade because of the improved adrenergic tone and distributing catecholamines [18]. High temperature is a general indicator that may associate with infectious or immune-intermediated pericarditis.

During physical examination it worth to discover major characteristic indicators including neck blood vessel distention with enhanced jugular venous stress, pulsus paradoxus, and minimized heart audios on cardiac auscultation. In 1873 Kussmaul described initially pulsus paradoxus as a palpable decrease of radial pulse on inspiration in people with cardiac tamponade ("waxing and going away" of the outer pulse, in contrast to the unvarying endurance of the apical cardiac impulse) [19]. Pulsus paradoxus is specified as an inspiratory decrease of at the very least 10 mmHg of the systolic blood pressure. It can be easily found recording the systolic stress at which Korotkoff sounds are first distinct and the systolic stress at which they are distinct with the entire breathing system cycle.

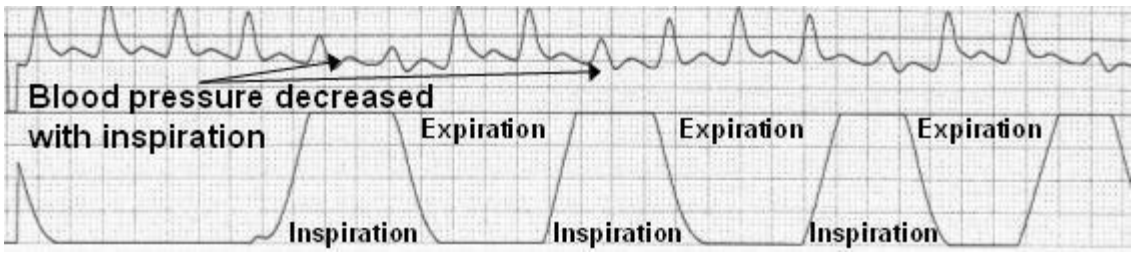

Figure 1. Illustration of pulsus paradoxus [15].

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.

- **Echocardiography**

In 1935 thoracic specialist Beck based on the conventional findings of cardiac tamponade defined this health problem as an including hypotension, enhanced jugular venous pressure, and a muffled heart [16]. This triad has been identified in "surgical tamponade" with acute haemodynamic collapse as a result of intrapericardial hemorrhage brought on by trauma, myocardial or aortic tear. Among the main factors is shortage in patients with gradually accumulating pericardial liquid [17]. There are 2 types of hypertension: absolute or relative. Acute cardiac tamponade is mostly gotten in touch with low high blood pressure (<100 mmHg) however blood pressure is just little less in subacute, chronic tamponade. Individuals with hypertense may have regular or even light rise of high blood pressure concomitant to cardiac tamponade because of the improved adrenergic tone and distributing catecholamines [18]. High temperature is a general indicator that may associate with infectious or immune-intermediated pericarditis.

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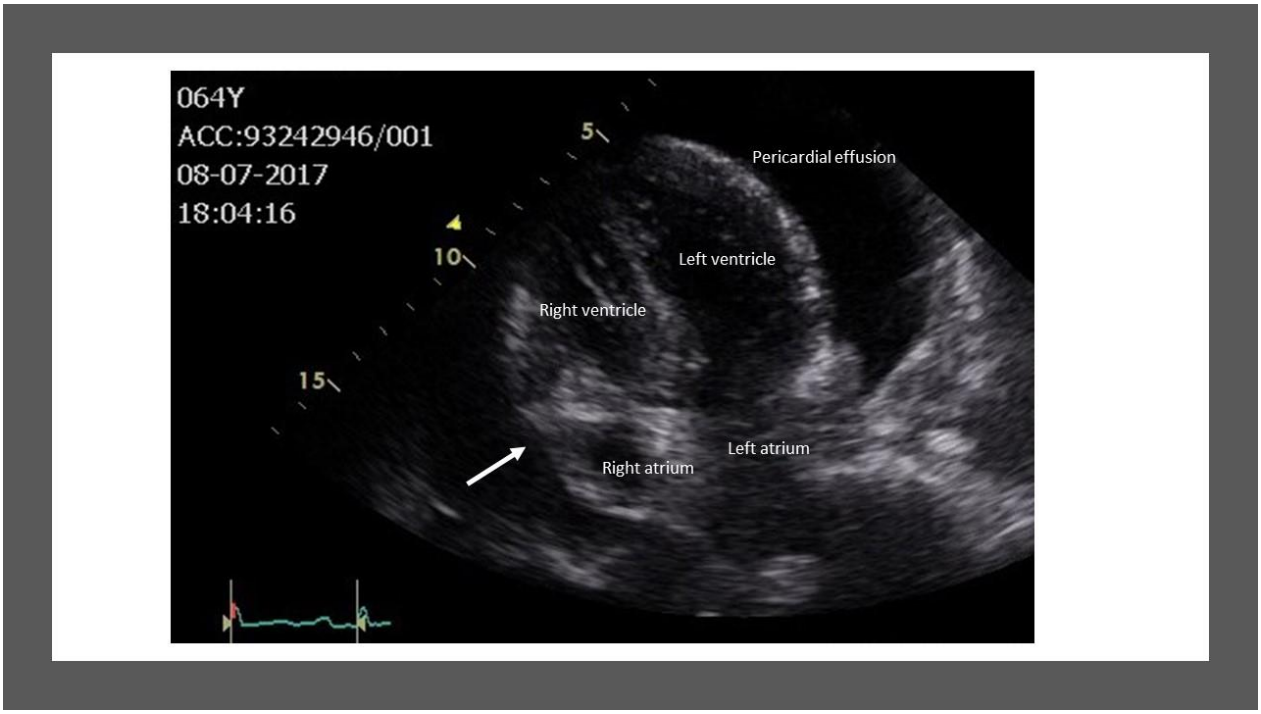

Figure 2. Two-dimensional echocardiogram illustrating cardiac tamponade with right atrium collapse or indentation (arrow) [5].

- **ECG**

This might disclose sinus tachycardia, reduced voltage complexes and non-specific ST segment and T wave modifications or ST segment elevation due to pericarditis. Electrical alternans (as a result of a pendular swinging movement of the heart within a pericardial effusion) is characteristically seen only with a big effusion [23]. Combined P and QRS rotation said to be pathognomonic for tamponade [24]. However, an ECG is mainly utilized to rule out other sources of hypotension instead of confirm the medical diagnosis of cardiac tamponade.

- **Chest X-ray**

The upper body X-ray might be within typical limitations although characteristics suggestive of pericardial fluid consist of a bigger globular cardiac shadow with loss of the hilar waist (if the pericardial effusion is greater than 250 mL) with a normal pulmonary vascular pattern [19]. Various other indications consist of prominence of the remarkable vena cava (reflecting an elevation in the central venous pressure), pleural effusions (transudates which may be caused by a raised main venous pressure) and the epicardial fat pad indicator (which is best seen on the side upper body radiograph as a radiolucent line in between the epicardial fat and the mediastinal fat and stands for the pericardium. It must be 2 mm or less, so any increase suggests liquid or thickening in the pericardium) [24].

- **Transvalvular pulse wave Doppler**

Using Doppler mode, modifications in blood flow throughout the cardiac valves can be seen that indicate cardiac tamponade. Usually, there is some variant in flow throughout the valves as a result of the breathing cycle. Throughout cardiac tamponade, this respiratory system variation in blood circulation across the valve is exaggerated. This can be best imagined by imaging at the level of the tricuspid valve and looking for a bigger inflow of blood throughout motivation as contrasted to expiry [25]. While these innovative echocardiographic findings might be helpful in the medical diagnosis of acute tamponade, extreme hypovolemia or appropriate ventricular hypertrophy might obscure these indicators.

- **Differential diagnosis**

Cardiac tamponade ought to be differentiated from other root causes of reduced cardiac outcome failure, such as cardiogenic shock, in which primary myocardial disorder means the heart is incapable to produce enough cardiac result to maintain systemic perfusion. One of the most usual reason for cardiogenic shock is enormous myocardial infarction.

In a methodical review of the literary works, 5 elements have actually been reported in the majority of patients with cardiac tamponade (> 70%): dyspnea (85-90%), cardiomegaly on breast radiograph (89%), pulsus paradoxus (82%), tachycardia (77%), and elevated jugular venous pressure (76%) [26]. In the setting of a large pericardial effusion, the presence of pulsus

paradoxus (inspiratory decline of systolic blood pressure > 10 mmHg) increased the probability of cardiac tamponade (likelihood proportion 3.3) [27]. Surprisingly, hypotension and soft heart sounds were insensitive (respectively 26% and 28%). Given that cardiac tamponade is mostly a medical diagnosis, echocardiographic indicators are just confirmatory [26].

Other differentials consist of large pulmonary emboli and stress pneumothorax. Rarely, a tension pneumopericardium can replicate acute cardiac tamponade, but with a characteristic mill-wheel murmur. This problem can be seen after penetrating chest wall injury, ruptured oesophagus and bronchopericardial fistula [26]. In patients with a subacute beginning of symptoms, the differential medical diagnosis ought to exclude constrictive pericarditis, congestive heart failure, advanced liver illness with cirrhosis, hardly ever Ebstein anomaly.

- **Prognosis**

Cardiac tamponade is one of the emergent medical situations. Immediate recognition and prompt management of the problem along with underlying causes of the tamponade impact on the diagnosis. Untreated, cardiac tamponade is rapidly and widely leading to fatal consequences.

Haneya et al retrospectively (2005-2011) assessed the impacts of timing and indicator of reexploration for blood loss or tamponade following cardiac surgical treatment in 209 individuals and discovered that reexploration was associated with higher rates of mortality and morbidity [28]. Multivariate evaluation showed it was the unfavorable effects of reexploration (eg, blood loss, transfusion needs) instead of the treatment itself that were independent risk aspects for fatality. Probability of obtaining negative results in those whose reexploration was delayed and who dealt with cardiac tamponade was higher [28].

Based on other research studies of Le et al suggested that adhering to cardiac surgery, there is no benefit for making use of several mediastinal breast tubes over a solitary chest tube in protecting against return to the operating room for blood loss or tamponade [29].

Along with treatment for the tamponade, all individuals need to obtain treatment for the condition's underlying cause in order to protect against recurrence.

In a research of patients with cardiac tamponade, a 1-year mortality rate of 76.5% in patients whose tamponade was brought on by deadly ailment was reported by Cornily et al, compared to 13.3% in individuals without fatal disorder. The detectives lkept in mind a median survival of 150 days in individuals suffered from malignant illness [30].

CONCLUSION:

Cardiac tamponade is the accumulation of pericardial fluid, blood, pus, or air within the pericardial area that creates a rise in intra-pericardial pressure, limiting cardiac filling and lowering cardiac result. Cardiac tamponade is a cardiac emergency that needs a hospital stay and can be fatal if it is not swiftly detected and treated promptly. The medical diagnosis is based on clinical suspicion and sustained by evidence of hemodynamic compromise on echocardiography.

Cardiac tamponade is a typical cardiac emergency needing timely diagnosis and intervention. A thorough understanding of the spectrum of professional and hemodynamic modifications in patients with pericardial effusion is crucial for interventional cardiologists. Clinicians need to recognize the physiology of cardiac tamponade, particularly in cases without large pericardial effusion, and associate the indications of clinical tamponade along with the echocardiographic results. The drainage of cardiac tamponade is life preserving. Emergency medical professionals have been revealed to have a high degree of accuracy in making the diagnosis, decreasing the requirement for the time or expense of detailed or consultative echocardiography. A high index of suspicion with appropriate diagnostic arcades decreases the concomitant morbidity and death.

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