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A REVIEW ON PERICARDIAL EFFUSION

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AUTHORS' CONTRIBUTIONS

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Review Article

ABSTRACT

Background: The collection of extra fluid in the pericardial sac surrounding the heart is referred to as a pericardial effusion. The pericardial sac contains between 15 and 50 milliliters (mL) of serous fluid in a healthy person. Infectious organisms or malignant cells may be present in this fluid, which can be transudative, exudative, or sanguineous. Infection, inflammation, or direct blood filling of the pericardial sac from a defect in the myocardium (iatrogenic or traumatic injury or heart wall rupture) or backfilling from an ascending aortic dissection that dissects into the pericardium are all possibilities. This activity looks at when this condition should be on a differential diagnosis list and how to properly evaluate for it, as well as the role of the interprofessional team in caring for patients with this illness.

Conclusion: The goals of this review article are to describe the symptoms of a pericardial effusion, outline the testing that should be done in patients who present with pericardial effusions, explain pericardial effusion management considerations, and summarise interprofessional team strategies for improving care coordination and communication to improve outcomes for patients with pericardial effusions.

Keywords: Cardiac tamponade; echocardiography; pericardial effusion; pericardiocentesis; pericarditis.

1. INTRODUCTION

The collection of fluid in the pericardial sac surrounding the heart is known as a pericardial effusion. The pericardial sac is made up of two layers: a thin visceral pericardium that adheres to the cardiac epicardium and a thicker, fibrous parietal pericardium made up of collagen and elastin that adheres to the lungs, diaphragm, sternum, great vessels, and other mediastinal structures surrounding the heart. The pericardial sac contains between 15 and 50 mL of serous fluid in a healthy person [1].

2. ETIOLOGY

Pericardial effusions have a variety of causes, which can be classified into various groups: Infectious: Infection with a variety of viral, bacterial, fungal, and parasitic diseases can cause pericardial effusion. Pericardial effusion can be caused by a variety of auto-immune diseases, including systemic lupus erythematosus, rheumatoid arthritis, and Sjogren's syndrome. Pericardial effusions can be caused by both metastatic illness and original cardiac malignancies. The most common cause of the malignant pericardial effusion is lung cancer. Trauma: Injury to the

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myocardium, aorta, or coronary arteries that is blunt, penetrating, or iatrogenic can cause blood to accumulate in the pericardial sac. Post-myocardial infarction (also known as Dressler Syndrome), cardiac surgery, and heart wall rupture are all examples of cardiac conditions. Vascular: Cardiac tamponade can worsen type A aortic dissection. Idiopathic: Many pericardial effusions are caused by unknown causes. Radiation, chronic kidney disease, and renal failure, congestive heart failure, cirrhosis, hypothyroidism leading to myxedema, ovarian hyperstimulation syndrome, and drug-induced pericardial effusion are among the other causes [2].

3. PATHOPHYSIOLOGY

Pericardial effusion is an accumulation of fluid in the pericardial cavity that can be transient or persistent. Effusion can be sanguineous, exudative, or transudative. Because the pericardium has low flexibility, just 100 mL to 150 mL of fluid is required to elicit cardiac tamponade in acute situations. The fluid buildup in the pericardial sac increases pressure, causing compression of the heart, particularly the right heart, which has a thinner wall. Venous congestion is caused by a problem with the right heart's diastolic filling. The stroke volume is reduced when the diastolic filling of the left ventricle is reduced. The initial compensatory reaction mediated by adrenergic stimulation to sustain cardiac output is tachycardia and increased contractility. Blood pressure and cardiac output, on the other hand, gradually decrease with time. As long as the accumulation is gradual and the parietal pericardium has enough time to stretch and accommodate the increased volume, the pericardial effusion can grow to one to two liters in size before causing cardiac tamponade in chronic circumstances (Fig. 1) [3].

4. HISTORY AND PHYSICAL

Pericardial effusion can manifest as anything from a clinically insignificant incidental finding to life-threatening cardiac tamponade. This vast range is attributable in large part to the varied rate of pericardial fluid buildup. With as little as 100 mL of fluid, acute accumulation can cause reduced heart-filling and decreased cardiac output, whereas chronic and slow accumulation can develop to substantial effusions of one to two liters with no major hemodynamic effects [5].

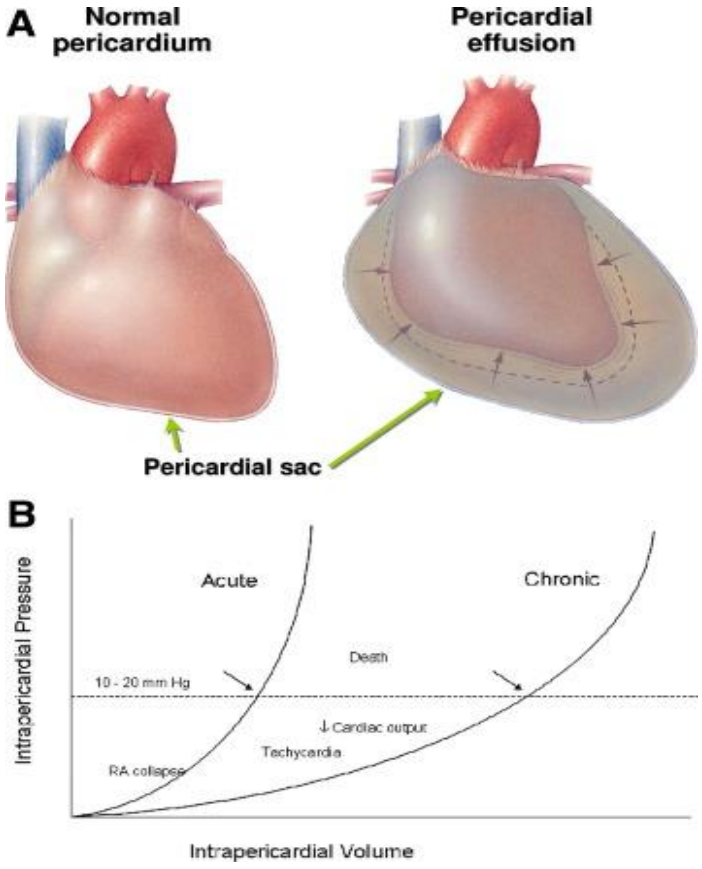

Fig. 1. Hemodynamics of Tamponade: Pericardium and pressure volume curves [4]

History: Owing to the inflamed pericardium contacting nearby structures, individuals with pericardial effusion due to pericarditis frequently appear with chest discomfort and dyspnea, with symptoms that improve when sitting upright and worsen when lying flat. Patients may also have symptoms such as dyspnea, edema, and weariness that are not specific to pericardial effusion. Questions about recent illnesses, cancer, tuberculosis history, and vaccination status, auto-immune disorders, history of chronic kidney disease or renal failure, or history of congestive heart failure, hypothyroidism, or liver disease are also important parts of the history [6].

Physical exam: For individuals in cardiac arrest or with vital sign anomalies such as hypotension and tachycardia, pericardial effusion leading to pericardial tamponade should be considered. Only a small percentage of patients have the traditional Beck's triad (hypertension, jugular venous distension, and muffled heart sounds). Ewart's sign is another physical exam finding that is specific to pericardial effusion (dullness to percussion at the base of the left inferior scapular border in conjunction with tubular breath sounds and egophony). Patients with cardiac tamponade should have their blood pressure checked for pulsus paradoxus, which is defined as a reduction in systolic blood pressure of more than 10 mmHg during inspiration caused by the left ventricle collapsing at the expense of the right ventricle. This causes the interventricular septum to bow and increased compression of the left side of the heart, resulting in lower filling volumes, stroke volume, and systolic blood pressure [7].

While the history and physical examination are still important parts of the evaluation for pericardial effusion and cardiac tamponade, other modalities such as echocardiography are increasingly used to confirm the diagnosis [8].

5. INVESTIGATIONS

The extent to which fluid analysis should be used to evaluate pericardial effusions is still up for dispute. The likelihood of myocarditis or pericarditis should be examined in a patient with a fresh pericardial effusion, and the initial diagnostic investigation should be aimed toward these disorders. In the setting of pericarditis, all patients with pericardial tamponade, suspected purulent effusion, or poor prognostic signs should have a diagnostic pericardiocentesis performed. Fluid analysis may be necessary for those who have recurring effusions or significant effusions that do not resolve with the therapy of the underlying ailment. Because electrocardiographic (ECG) alterations are a part of the diagnostic criteria for acute pericarditis, an ECG should be conducted right away. Because echocardiography can be performed quickly and in unstable patients, it is the imaging modality of choice for the diagnosis of pericardial effusion (Fig. 2) [9].

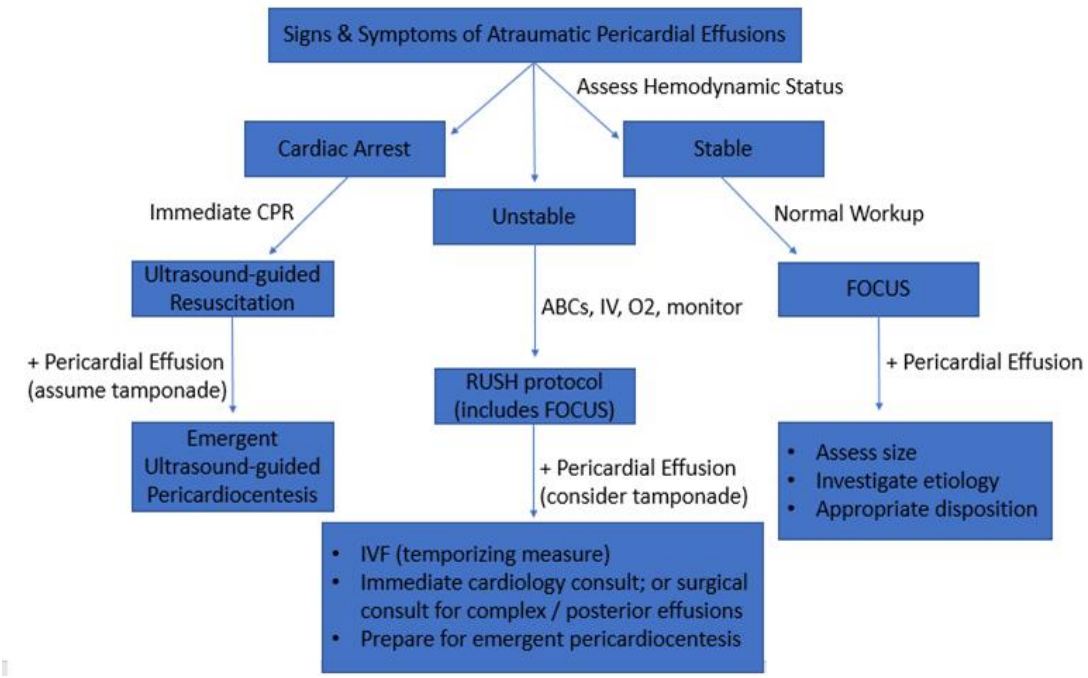

Fig. 2. Diagnostic Approach for Pericardial Effusion [10]

6. LAB STUDIES

In individuals with a suspected pericardial effusion, the following lab tests may be performed: Electrolytes are used to check for metabolic problems (eg, renal failure), Complete blood count (CBC) with differential - Leukocytosis for infection evidence and cytopenias for indicators of underlying chronic disease (eg, cancer, HIV), Biomarkers of the heart, Other inflammatory markers include erythrocyte sedimentation rate and C-reactive protein. -Reactive protein - While these cannot be used to pinpoint specifics, they can be used to determine whether anti-inflammatory drugs (corticosteroids, colchicine, and NSAIDs) are needed, particularly in the case of recurring effusions. Thyroid-stimulating hormone - Hypothyroidism Thyroid-stimulating hormone test In the presence of systemic inflammatory response syndrome (SIRS) or fever, blood cultures are recommended; however, specific viral tests have a limited yield and are therefore not recommended. Specific infectious disease testing, based on clinical suspicions, such as (1) tuberculin skin testing or QuantiFERON-TB assay; (2) rickettsial antibodies if there is a high index of suspicion for tick-borne disease; and HIV serology - in suspected rheumatologic causes, and Rheumatoid factor, immunoglobulin complexes, antinuclear antibody test (ANA), and complement levels (which would be diminished) [11].

7. CARDIAC ENZYMES

In acute pericarditis, the troponin level is frequently marginally raised, usually in the absence of a high total creatine kinase level. This is most likely owing to the inflammatory process involving the epicardium. Although an elevated troponin level can lead to a misinterpretation of acute pericarditis as a myocardial infarction, most patients with an elevated troponin and acute pericarditis do not show angiographic signs that are consistent with the acute coronary syndrome. In acute pericarditis, an increased troponin level usually returns to normal within 1-2 weeks and is not linked to a worse prognosis [12].

8. PERICARDIAL FLUID ANALYSIS

It's worth noting that standard biochemical and cell-count analyses have a low success rate in determining the etiology of effusion. Gram stain and culture, on the other hand, can reliably determine the cause. It is routine for the following tests to be considered part of the standard pericardial fluid analysis: Total protein, lactic (acid) dehydrogenase (LDH) - The Light criteria (for exudative pleural effusion) were shown to be as reliable as the Light criteria (for transudative pleural effusion) in discriminating between exudative and transudative effusions: (1) total protein fluid-to-serum ratio >0.5, (2) LDH fluid-to-serum ratio >0.6, (3) LDH fluid level surpasses two-thirds of normal serum level Other signs of exudate include specific gravity >1.015, total protein >3.0 mg/dL, LDH >300 U/dL, and a glucose fluid-to-serum ratio of less than one. Although inconsistent, an elevated leukocyte count (ie, >10,000) with a neutrophil preponderance suggests a bacterial or rheumatic etiology. Gram stain is a sensitive (but not specific) sign of bacterial infection. Cultures can be used to detect and identify the source of infection. Fluid hematocrit for bloody aspirates - hemorrhagic fluid hematocrits are typically lower than simultaneous peripheral blood hematocrits - and pericardial fluid cytology to look for cancer cells [13].

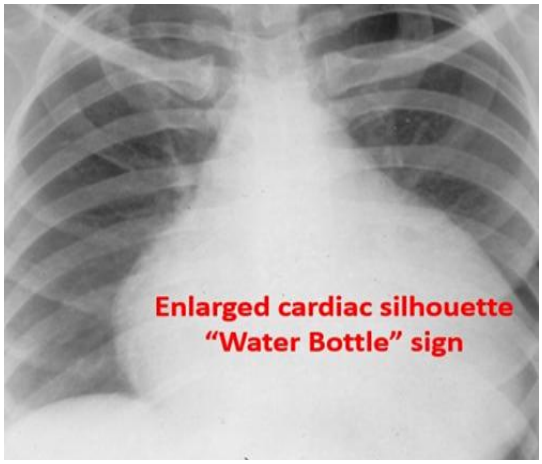

Fig. 3. This image is from a patient with malignant pericardial effusion. Note the "water-bottle" appearance of the cardiac silhouette in the anteroposterior (AP) chest film [16]

9. SPECIAL TESTS

These pericardial fluid examinations should be evaluated on an individual basis, based on the pre-test probability of the suspected concomitant ailment. The following are some of them: Viral cultures and adenosine deaminase; polymerase chain reaction (PCR); tuberculosis culture; acid-fast bacilli smear in patients with HIV who are suspected of tuberculosis infection. The presence of tubercle bacilli in pericardial fluid or a histologic slice of the pericardium is required for a definitive diagnosis of tuberculous pericarditis. Probable tuberculous pericarditis is defined as TB elsewhere in a patient with otherwise unexplained pericarditis, a lymphocytic pericardial exudate with high adenosine deaminase or gamma interferon (IFN) levels, and/or a

positive response to an antituberculosis chemotherapy trial. CEA levels in the pericardial fluid that are elevated show a good specificity for malignant effusion. Perform a pericardial biopsy, especially if you suspect a malignant pericardial effusion. When paired with pericardiotomy, this can be more diagnostic [14].

10. CHEST RADIOGRAPHY

An enlarged cardiac profile (so-called water-bottle heart) and a pericardial fat stripe can be seen on chest radiography. A pleural effusion affects one-third of patients. In establishing or rejecting a diagnosis of pericardial effusion, radiography is unreliable (Fig. 3) [15].

11. ECHOCARDIOGRAPHY

Because echocardiography can be performed quickly and in unstable patients, it is the imaging modality of choice for the diagnosis of pericardial effusion. Most notably, echocardiography can be used to assess the contribution of pericardial effusion to total cardiac enlargement, as well as the relative roles of tamponade and myocardial dysfunction in changed hemodynamics. Patients with viral cardiomyopathy, especially in the acute setting, may present with an enlarged heart on chest radiographs, similar to those with pericardial effusion. The distinction between enlarged heart chambers and pericardial effusion can be easily distinguished using echocardiography (Figs. 4, 5, 6, 7) [17].

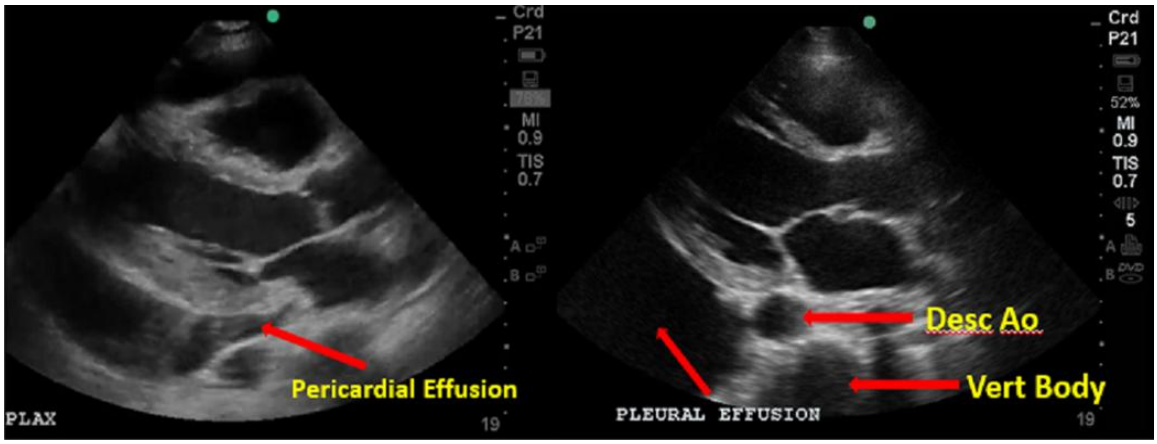

Fig. 4. Differentiation of pericardial effusion from pleural effusion [18]

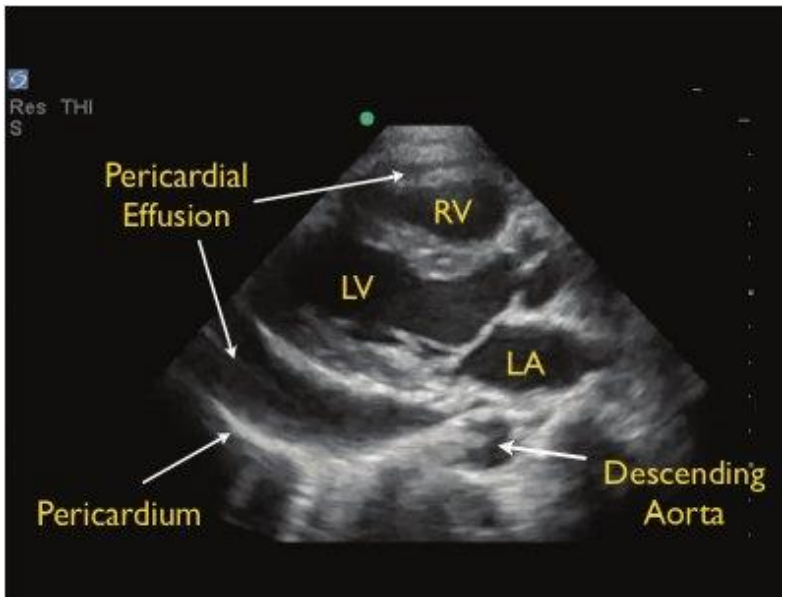

Fig. 5. Pericardial effusion, parasternal long axis view. RV: right ventricle, LV: left ventricle, LA: left atrium [19]

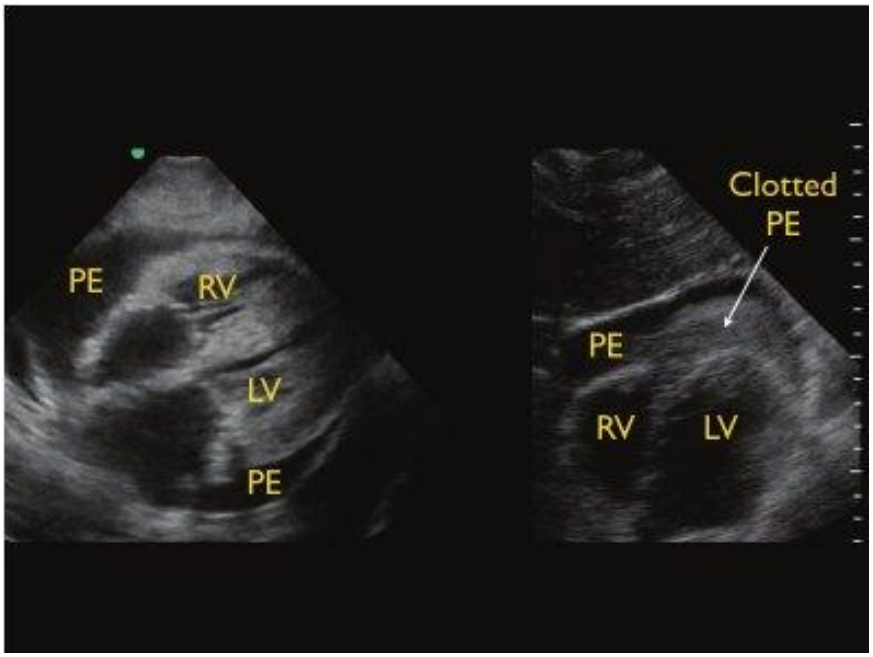

Fig. 6. Types of pericardial effusions, subxiphoid cardiac view. Left image: typical effusion, right image: clotted e ff usion. RV: right ventricle, LV: left ventricle, PE: pericardial effusion [20]

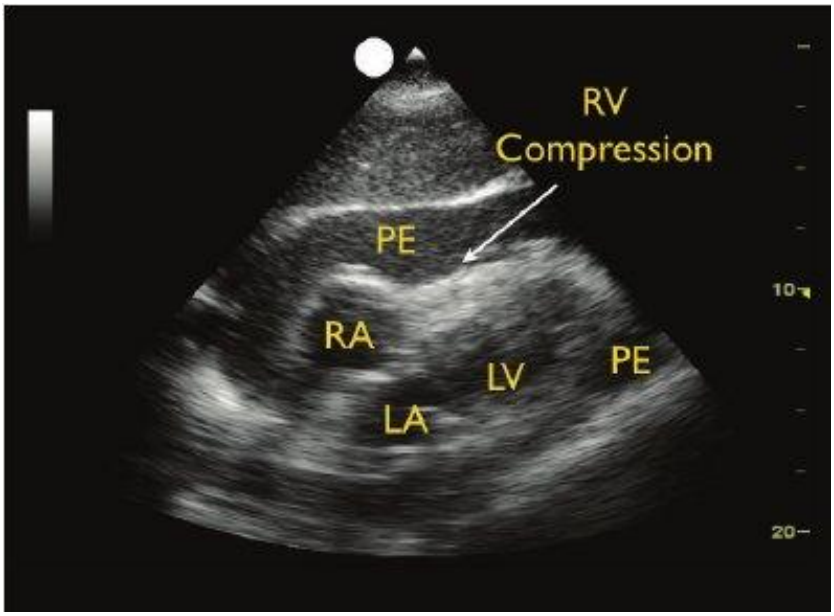

Fig. 7. Cardiac tamponade, subxiphoid view, RV: right ventricle, RV: right atrium, LV: left ventricle, LA: left atrium, PE: pericardial effusion [21]

12. TWO-DIMENSIONAL ECHOCARDIOGRAPHY

Between the visceral and parietal pericardium, pericardial effusion appears as an echo-free area. Because of the expansive posterior/lateral pericardium, early effusions tend to accumulate posteriorly. Excessive motion within the pericardial sac, often known as swinging, is a symptom of large effusions. Small effusions are detected posteriorly and have an echo-free space of less than 10 mm. The diameter of moderate effusions is 10-20 mm and they are circumferential. A significant effusion is indicated by an echo-free space of more than 20 mm. A pericardial effusion with fluid close to the right atrium is a warning sign (Fig. 8) [22].

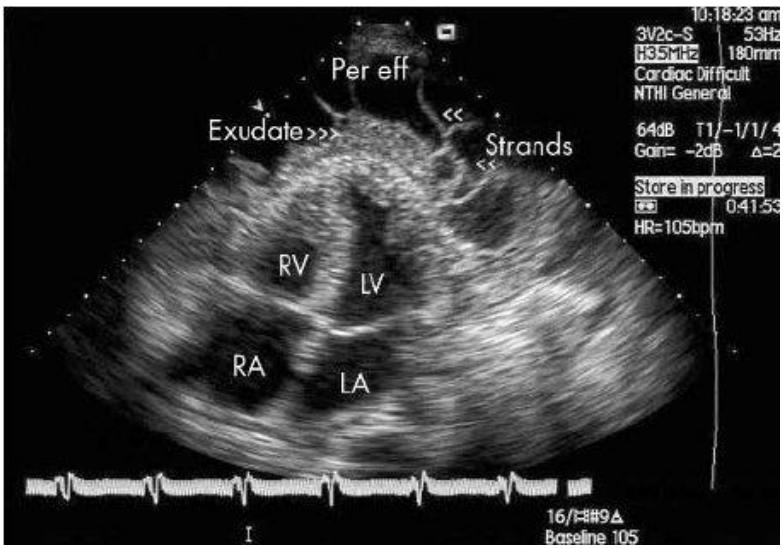

Fig. 8. Apical four-chamber view of a two-dimensional echocardiogram of a patient with tuberculous pericardial effusion showing multiple fibrin strands as linear or band-like structures crossing the pericardial space or protruding from the epicardium or parietal pericardium and exudates. LA, left atrium; LV, left ventricle; Per eff, pericardial effusion; RA, right atrium; RV, right ventricle [23]

Although echocardiography can detect characteristics that suggest hemodynamically substantial cardiac tamponade, this is a clinical diagnosis rather than an echocardiographic one. The following are some of the findings: Right atrial collapse, especially if it lasts for a third of the cardiac cycle, is dangerous. The right ventricle's diastolic collapse, The left atrium, and the left ventricle may also show indications of collapse in hypovolemic patients. In some cases, echocardiography can be used to determine the cause of the effusion. The echocardiographic results listed below may be useful: A bloody pericardial effusion is indicated by the presence of coagulum in the pericardial cavity (aortic dissection, postoperative, or after other catheter-based procedures), The presence of a pacemaker lead in the proximity or inside an area of localized effusion, indicating lead perforation as a cause, and the presence of a pacemaker lead in the vicinity or inside an area of localized effusion, indicating lead perforation as a cause, and In the pericardial space, there is a tumor (primary or secondary) [24].

13. M-MODE ECHOCARDIOGRAPHY

For the identification of pericardial effusion, M-mode echocardiography is used in conjunction with two-dimensional (2-D) imaging. M-mode can be used to classify effusions according to Horowitz et al's proposed system: Type A - No effusion, Type B - No effusion, Type C - No effusion Type B - Epicardium and pericardium separation Type C1 - Pericardial systolic and diastolic separation, Type C2 - Pericardial systolic and diastolic separation, attenuated pericardial motion, Type D - Pronounced pericardium and epicardium separation with significant echo-free space. Discordant changes in right and left ventricular cavity size in the parasternal long-axis and apical 4-chamber views can indicate substantial interventricular dependency, as well as an echocardiographic "substrate" for tamponade. It's worth noting that these alterations happen outside of the cardiac cycle (as these are dependent on respiration) (Fig. 9) [25].

14. DOPPLER ECHOCARDIOGRAPHY

To check for respiratory fluctuation, look at the transmitral and transtricuspid input velocities. Reduced flow during inspiration (transmitral >25%) or expiration (transtricuspid >40%) may raise concerns about clinically substantial interventricular dependency and tamponade physiology. Altogether in the face of a hemodynamically significant pericardial effusion, these symptoms may be less obvious or even absent in patients on mechanical breathing. With hemodynamically substantial effusions, pulmonic vein inflow may indicate a decrease in early diastolic flow. Increased right atrial pressures can be indicated by a plethoric inferior vena cava that collapses less than 50% on inspiration. Another characteristic symptom of ventricular interdependence is hepatic vein diastolic flow reversal on expiration (Fig. 10) [27].

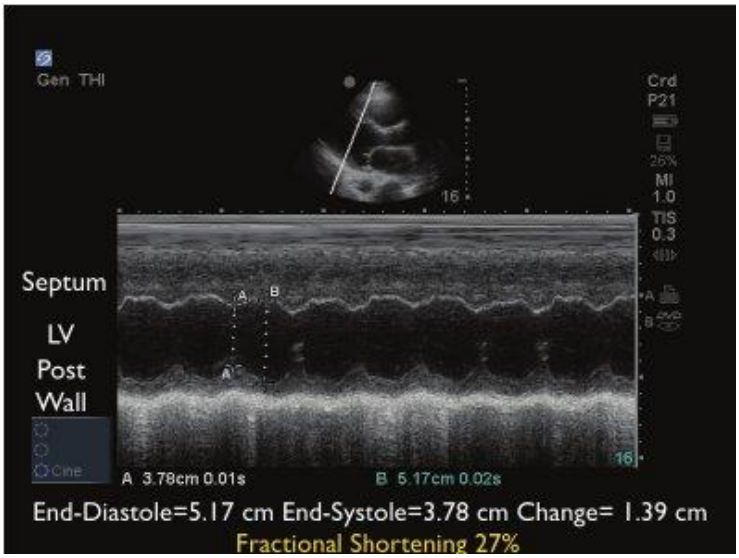

Fig. 9. M-mode tracing demonstrating poor contractility, LV: left ventricle [26]

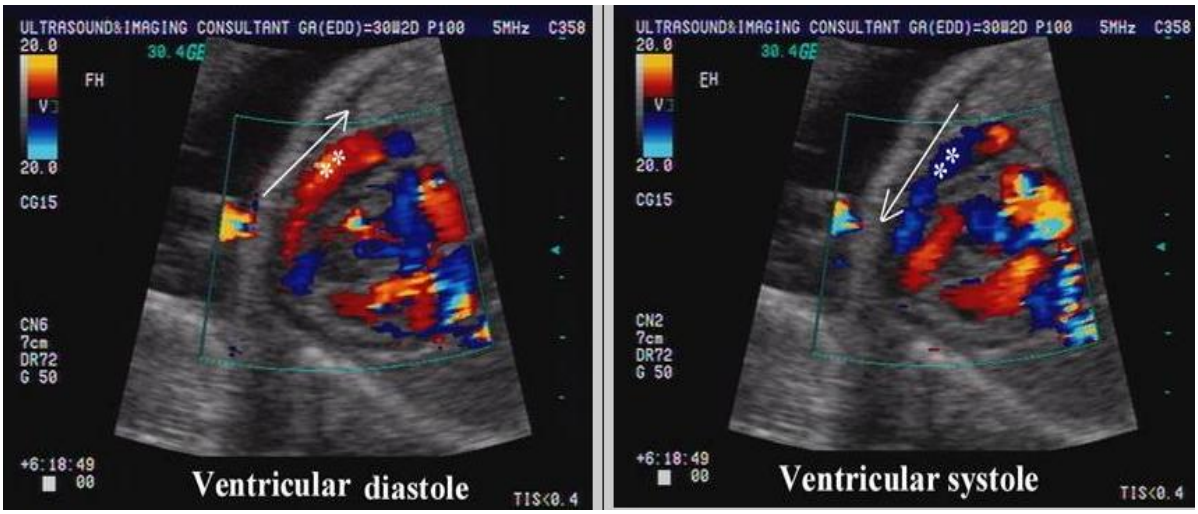

Fig. 10. During ventricular diastole: pericardial space disappears as the ventricles expand resulting in the fluid moving out of this space. During ventricular systole: pericardial space around the ventricles expand and pericardial fluid moves into this space [28]

15. TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Tamponade can occur in the absence of any of the conventional 2-dimensional or Doppler symptoms, such as a postoperative mediastinal hematoma. Transesophageal echocardiography has all of the benefits of transthoracic echocardiography and is excellent for determining the location of loculated effusions. However, due to hemodynamic instability, this imaging scan may be challenging to execute in patients with symptomatic effusions, making the requisite sedation more difficult (Fig. 11) [29].

16. INTRACARDIAC ECHOCARDIOGRAPHY

In most cases, intracardiac echocardiography (ICE) is used to assess pericardial effusion during a percutaneous interventional or electrophysiologic technique. 2-D and Doppler's interrogations are possible with phased-array ICE devices (Fig. 12) [31].

17. FALSE-POSITIVE FINDINGS

Pleural effusions, pericardial thickness, increased epicardial fat tissue, atelectasis, and mediastinal lesions can all cause false-positive echocardiographic

results. Pericardial cysts, which are typically benign, can be observed in the right cardiophrenic angle and are often mistaken for pericardial effusion. Fat tissue in the epicardial cavity is more apparent anteriorly, although it can also present circumferentially, giving the appearance of effusion. Fat is slightly echogenic and moves in lockstep with the heart, two traits that separate it from an effusion, which is echolucent and stationary. Imaging from the low to mid posterior thorax can provide extra diagnostic echocardiographic images in patients with pericardial effusion and should be employed in patients for whom conventional images are technically challenging or require additional information [33].

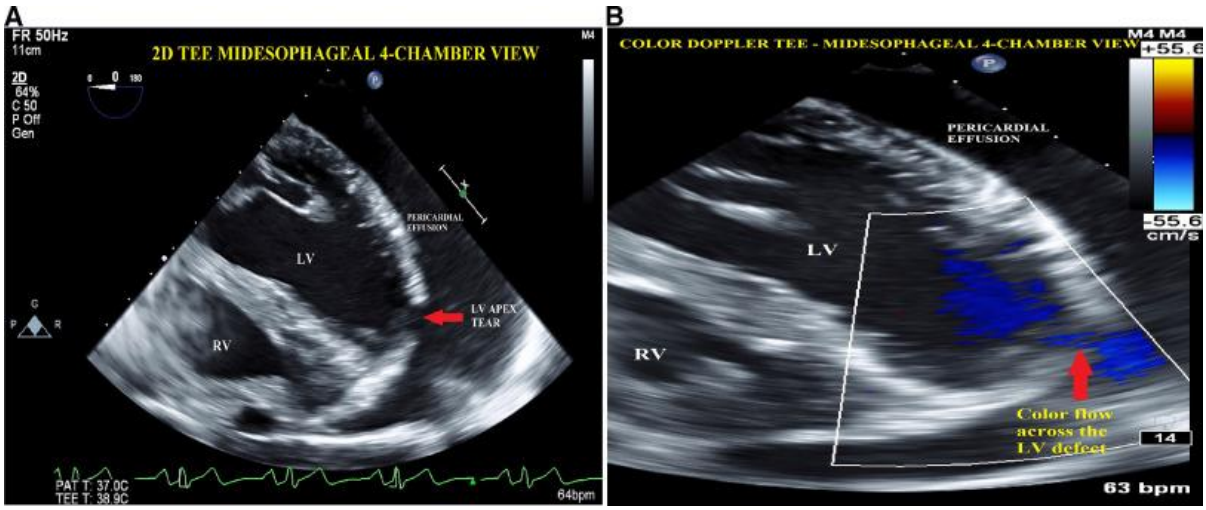

Fig. 11 (A) Two-dimensional TEE examination in midesophageal 4-chamber view shows massive PE and a tear in the wall of LV apex. (B) Color Doppler examination shows color flow across the defect in LV apex. LV, left ventricle; PE, pericardial effusion; RV, right ventricle; TEE, transesophageal echocardiography [30]

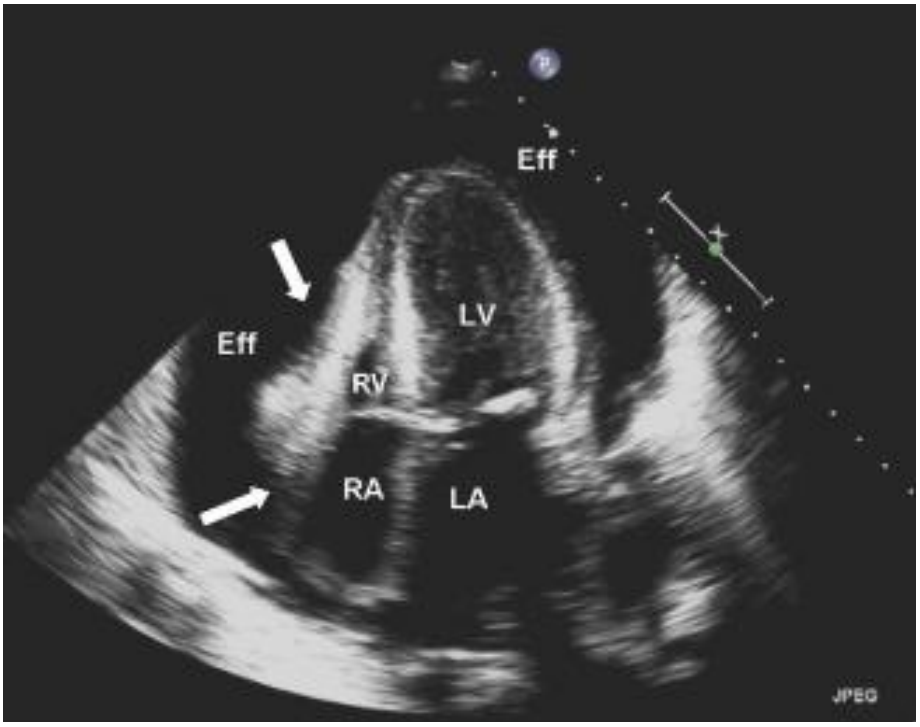

Fig. 12. Iatrogenic Pericardial Effusion and Tamponade in the Percutaneous Intracardiac Intervention [32]

18. CT SCANNING AND MRI

CT scanning and magnetic resonance imaging (MRI) may be more effective than echocardiography in detecting loculated pericardial effusions, particularly when they are anteriorly positioned. Furthermore, because these modalities allow for better imaging of the thoracic cavity and nearby structures, they may be able to detect anomalies related to the effusion's origin [34].

19. CT SCANNING

CT scanning has the potential to assess the fluid composition and can detect as little as 50mL of fluid. Pericardial calcifications, which can be symptomatic of constrictive pericarditis, can also be detected with this technique. Compared to echocardiography, CT scanning produces fewer false-positive outcomes. However, considering the amount of time required to transfer patients to and from the scanner and complete the test, it can be troublesome in unstable patients. Dilated venae cavae, contrast reflux into the azygos vein and inferior vena cava, distortion or compression of the heart chambers, and bowing of the interventricular septum are all classic CT symptoms of tamponade (Fig. 13) [35].

20. MRI

Pericardial fluid as small as 30 mL can be detected with MRI. Hemorrhagic fluids have a high signal intensity on T-1 weighted images, whereas non-hemorrhagic fluids have a low signal intensity. On MRI, nodularity or irregularity in the pericardium could indicate a malignant effusion. Given the length of time, the patient must remain in the scanner, MRI is more challenging to do acutely than CT scanning. Late gadolinium enhancement can indicate areas of inflammation, which can aid in the diagnosis of effusive-constrictive pericarditis and help decide on anti-inflammatory therapy in recurrent pericarditis (Fig. 14) [37].

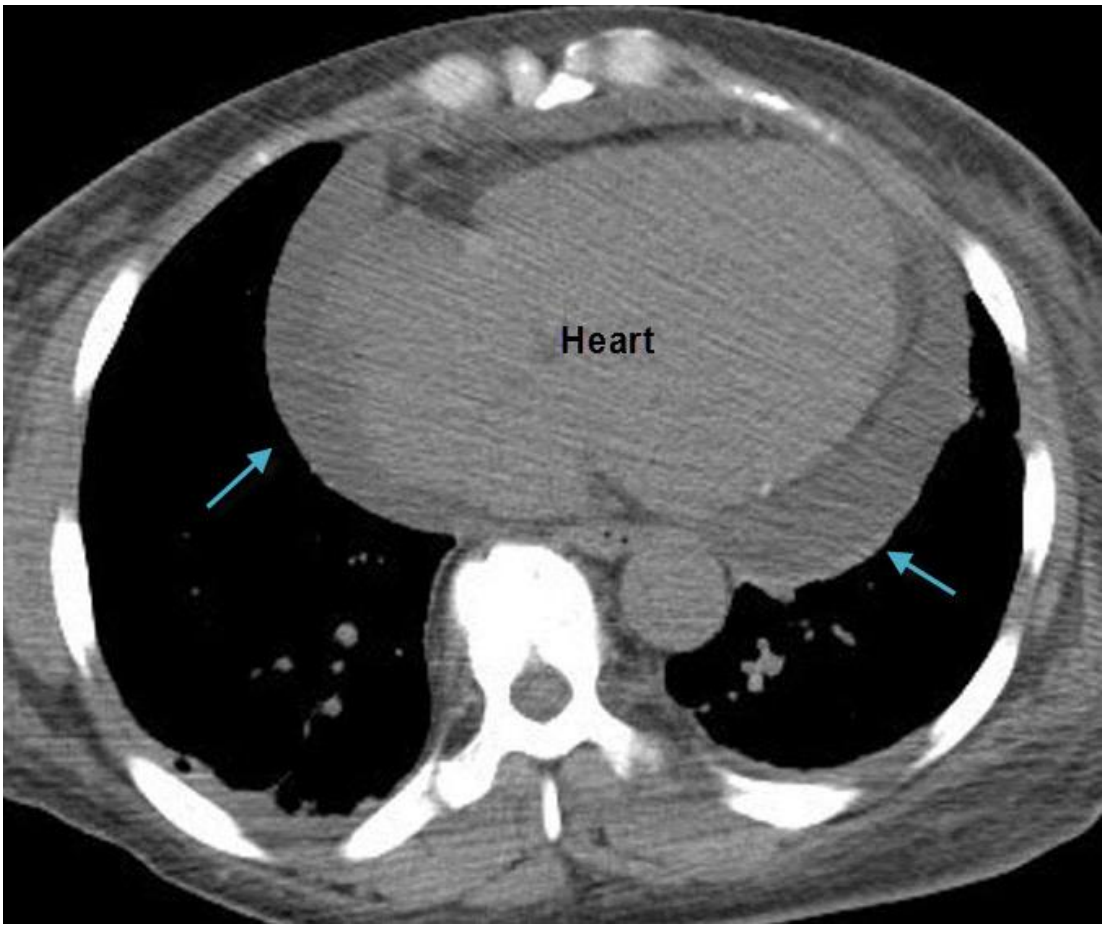

Fig. 13. Pericardial effusion CT [36]

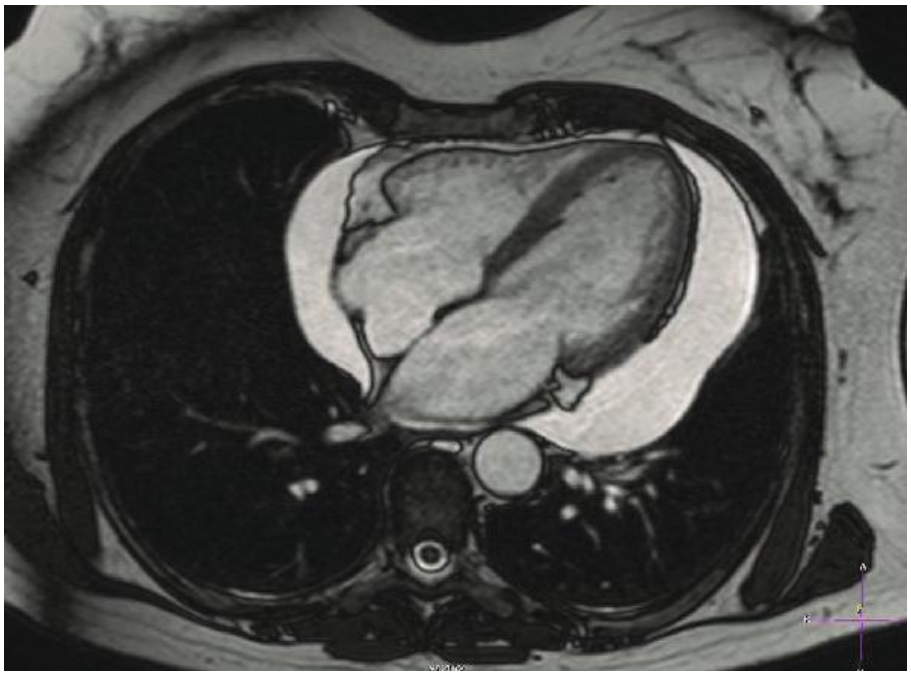

Fig. 14. Magnetic resonance imaging (4-chamber view, turbo field echo [TFE]) confirmed the extended pericardial effusion without signs of cardiac tamponade, and a slightly thickened pericardium [38]

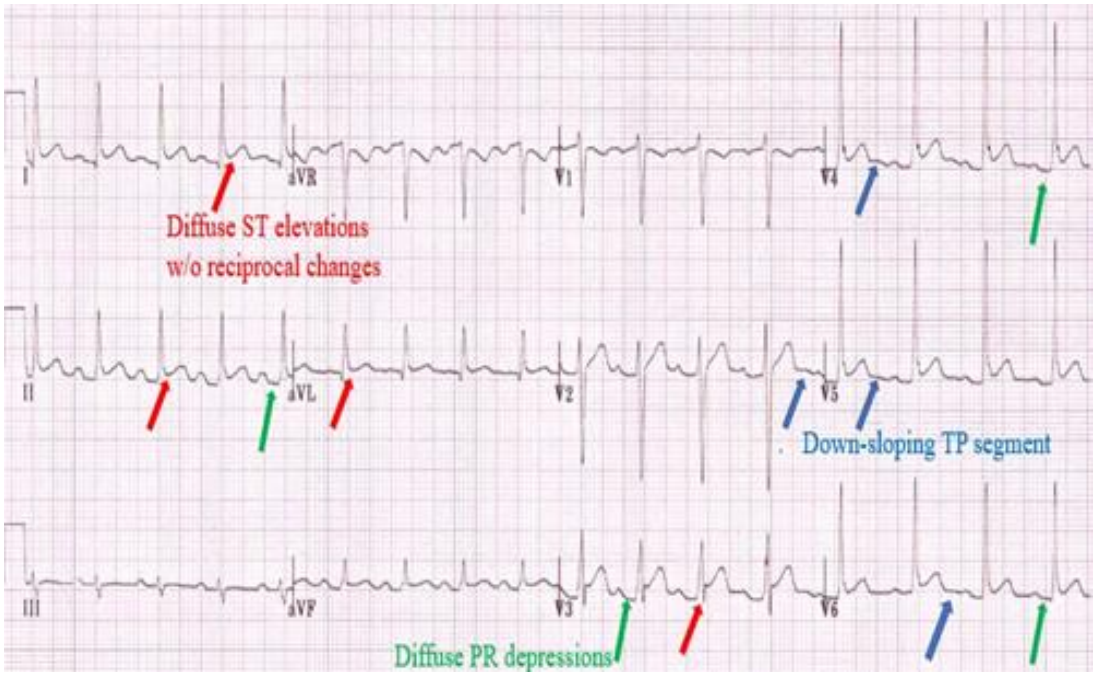

Fig. 15. This electrocardiogram (ECG) is from a patient with pericardial effusion [40]

21. ELECTROCARDIOGRAPHY

The ECG usually shows generalized ST elevation with PR depression early on in the course of acute pericarditis. Except for aVR, ST elevation is normally evident in all leads, while the alterations in postmyocardial infarction pericarditis may be more localized. (Electrocardiographic abnormalities are typically absent in patients with uremic pericarditis) (Fig. 15, 16) [39].

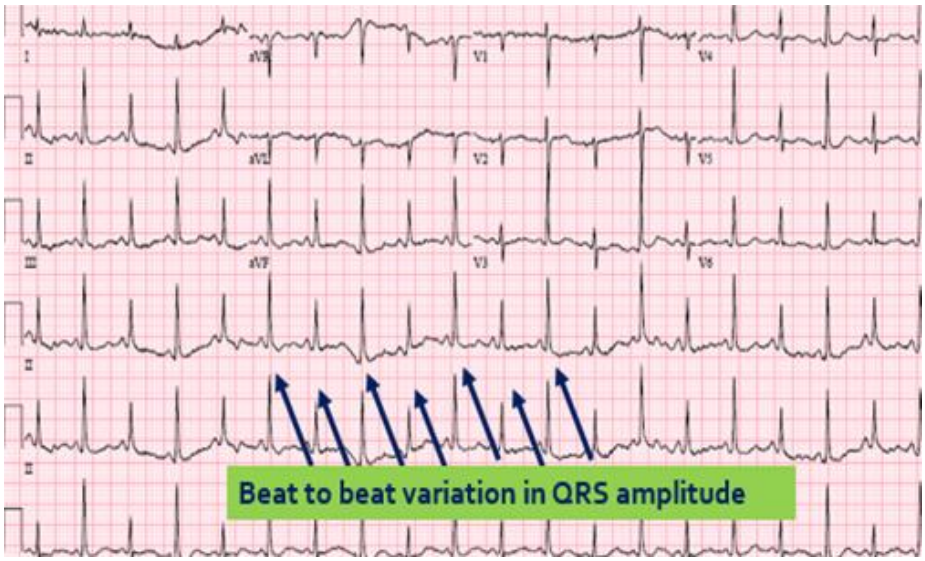

Fig. 16. Electrical Alternans [41]

The electrocardiographic alterations of acute pericarditis have traditionally progressed through four stages: Diffuse ST-segment elevation and PR-segment depression in Stage I. Stage II - ST and PR segment normalization, Stage III - Widespread T-wave inversions: This is critical to distinguish from myocardial infarction because T-wave inversions in pericarditis usually occur after ST-segment normalization, whereas in myocardial infarction, T-wave inversions usually occur after ST-segment normalization, Stage IV - Normalization of the T waves. The electrical hallmark of the "swinging" of the heart in the pericardial fluid is electrical alternans, which is the beat-to-beat fluctuation in the direction and amplitude of the QRS complex. It can involve both the P and T waves in extreme circumstances. It can be noticed in massive pericardial effusions and is specific but not sensitive for tamponade. Large effusions and tamponade can both show low-voltage QRS complexes, which are defined as the total amplitude of the QRS complex less than 0.5 mv in the limb leads and less than 1 mv in the precordial leads. According to one study, limb lead criteria are more specific for tamponade than for effusion [42].

22. PERICARDIOCENTESIS AND PERICARDIOSCOPY

Pericardiocentesis is a procedure that can be used for both diagnostic and therapeutic purposes. Unless emergency treatment is required, the use of echocardiographic guidance is gaining popularity. Impending hemodynamic compromise (ie, pericardial tamponade), probable infectious or neoplastic etiology, and unclear etiology are all reasons for pericardiocentesis. Pericardioscopy is a procedure that is not always available. In cases of unexplained pericardial effusions, it may improve diagnostic sensitivity, especially for neoplastic illness. It allows for pericardium imaging and pericardial biopsies [43].

23. TREATMENT

The treatment for pericardial effusion varies greatly depending on the presumed etiology and ranges from watchful waiting to emergency intervention. If necessary, minor effusions with no indication of hemodynamic compromise are monitored using a serial echocardiogram, unless the effusion is tiny enough that no follow-up is required. If the patient has associated symptoms such as dyspnea, chest discomfort, pulmonary or lower extremity edema, or decreased exercise tolerance, a diagnostic pericardiocentesis may be performed to determine the etiology. If the patient has associated symptoms such as dyspnea, chest discomfort, pulmonary or lower extremity edema, or decreased exercise tolerance, the effusion may be drained to provide symptomatic relief. Emergently, at the bedside, in the cardiac catheterization lab, or the operating room, effusions that have collected quickly enough or grown large enough to cause hemodynamic instability or collapse are addressed. Percutaneous balloon pericardiotomy, emergent thoracotomy, and pericardiotomy, as well as surgical pericardial window via subxiphoid, anterior mini-thoracotomy, or video-assisted thoracoscopic surgery (VATS) approach are all options for drainage. The sort of intervention chosen is determined by the cause of the pericardial effusion, the patient's clinical situation at the time of the intervention, and the projected clinical course of the patient [44].

There is a risk of pericardial decompression syndrome (PDS) during pericardiocentesis in patients with significant pericardial effusions and underlying ventricular dysfunction. Following a simple pericardial evacuation for cardiac tamponade physiology, pericardial decompression syndrome (PDS) is a rare yet life-threatening consequence. PDS is characterized by paradoxical hemodynamic instability and/or pulmonary edema after a routine pericardial drainage procedure. Physicians should be aware of PDS prevention techniques and provide close clinical surveillance to vulnerable patients, particularly those having pericardial drainage for large malignant effusions with suspected tamponade. Massive amounts of pericardial fluid should not be drained in one session, especially in the event of large pericardial effusions. The most reasonable approach would be to remove just enough pericardial fluid to resolve cardiac tamponade physiology (which can be easily accomplished using hemodynamic or echo-doppler monitoring) and then use prolonged pericardial drainage to remove additional pericardial fluid slowly and gradually. When the daily fluid return is less than 30-50 ml, pericardial drainage may be discontinued [45].

24. EPIDEMIOLOGY

Pericardial effusion can occur at any age and in any demographic. The origin of the effusion varies depending on demographic factors such as age, location, and comorbidities. The prevalence and incidence of pericardial effusions are poorly understood. In the industrialized world, viral pericarditis with effusion is the most common cause. Pericardial effusion caused by Mycobacterium TB is common in impoverished countries. Bacterial and parasite infections are rare. Multiple malignant neoplasms can cause pericardial effusion in non-inflammatory pericardial effusions. Malignancy accounts for between 12% and 23% of pericarditis cases in patients with pericardial effusion. Pericardial effusion was found in 5-43 percent of HIV patients, depending on the inclusion criteria, with 13% having moderate to severe effusion. Post-cardiac surgery (54 percent), neoplasia (13 percent), renal (13 percent), idiopathic or viral pericarditis (5 percent), and rheumatologic (5 percent) were the most common causes of pericarditis and pericardial effusions in children, according to a study [46].

25. PROGNOSIS

The majority of people who have acute pericarditis recover without complications. The following are indicators of a poor outcome: A fever of more than 38°C, Symptoms that appear over weeks and are associated with an immunosuppressed state Pericarditis caused by a traumatic event, Pericarditis in a patient using anticoagulants, a significant pericardial effusion (>20 mm echo-free space or indications of tamponade), and nonsteroidal anti-inflammatory medication failure (NSAIDs). In a study of 300 individuals with acute pericarditis, 254 (85%) had no high-risk features and experienced no significant consequences. When these low-risk patients did not respond to aspirin, 221 (87%) were handled as outpatients, while the remaining 13% were admitted to the hospital. Short-term death rates are high in patients with symptomatic pericardial effusions caused by HIV/AIDS or malignancy [47].

The etiology and concomitant factors influence the morbidity and mortality of pericardial effusion. In most cases, idiopathic effusions are well tolerated. During long-term follow-up, up to 50% of patients with large, chronic effusions (effusions lasting more than 6 months) were found to remain asymptomatic. In 86 percent of cancer patients with symptomatic effusions, pericardial effusion is the primary or contributing cause of mortality. Patients with HIV and symptomatic pericardial effusion have a 36 percent six-month survival rate and a 19 percent one-year survival rate [48].

The equalization of diastolic filling pressures, which signals pericardial tamponade, can cause severe hemodynamic compromise and mortality. Expansion of intravascular volume (little quantities of crystalloids or colloids may help, especially in hypovolemic individuals) and prompt pericardial drainage are used to treat it. If at all feasible, avoid positive-pressure breathing, which reduces venous return and cardiac output. Vasopressors aren't very useful in the clinic [49].

26. DISCUSSION

Pericardial effusion is defined as an abnormal amount of fluid in the pericardial space with an aberrant nature. It can be caused by several different local and systemic illnesses, or it could be idiopathic. Acute or chronic pericardial effusions exist, and the progression of the condition has a significant impact on the patient's symptoms. Treatment varies, but it normally focuses on removing pericardial fluid and treating the underlying cause, which is usually diagnosed through a mix of fluid studies and concomitant conditions [50].

Embryology: During the fourth week of development in the human embryo, the pericardial cavity develops from the intraembryonic celom. The pericardial cavity communicates with the pleural and peritoneal cavities

at first, but by the eighth week of development, they are isolated. The visceral and parietal pericardium are both made up of mesoderm, however, they come from distinct areas of the embryo. As cells from the sinus venous stretch out throughout the heart, the visceral pericardium develops from the splanchnic mesoderm. The lateral mesoderm that covers and accompanies the growing pleuropericardial membrane, which will eventually separate the pleural and pericardial compartments, gives rise to the parietal pericardium. The pericardium covers the heart and major vessels in healthy people, except the left atrium, which is only partially covered [51].

The pericardium can be absent from birth, and it can be partial or complete. This disorder is frequently clinically asymptomatic, but it can result in excessive cardiac motion (in the case of complete absence), causing vague chest pain or dyspnea, or strangulation of the heart muscle and death (in the case of partial absence with substantial abnormalities) [52].

Physiology: The pericardial area generally contains 15-50 mL of fluid that lubricates the pericardium's visceral and parietal layers. This fluid, which is essentially an ultrafiltrate of plasma, is assumed to come from the visceral pericardium. Total protein levels are normally low; but, due to albumin's low molecular weight, its concentration in a pericardial fluid is elevated. The pericardium and pericardial fluid contribute to heart function in a variety of ways, including The parietal pericardium is responsible for the majority of the pressure in the right atrium and ventricle during resting diastole. The ability of the pericardial structures to evenly transfer stress around the heart helps to ensure that the myocardium contracts uniformly [53].

27. CONCLUSION

Pericardial effusions are a common complication. Acute effusions in symptomatic individuals, unlike chronic effusions, require immediate treatment. Pericardial effusions are treated by a multidisciplinary team that includes a cardiologist, radiologist, and cardiac surgeon. While aspiration can help most patients with symptoms and hemodynamic compromise, those who have had a malignancy may need a pericardial window, which can be done open or by thoracoscopy. Symptomatic pericardial effusions with inadequate treatment have a significant death rate.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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