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ETIOLOGY, INVESTIGATIONS, AND TREATMENT IN CASES OF CONSTRICTIVE PERICARDITIS

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AUTHORS' CONTRIBUTIONS

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Review Article

ABSTRACT

The fibroelastic sac that covers the heart is known as the pericardium. It has an effect on cardiac hemodynamics and serves as a protective barrier. Constrictive pericarditis is a condition in which the pericardium grows granulation tissue, causing a loss of pericardial elasticity and ventricular filling limitation. Although it is usually a chronic condition, subacute, transient, and occult variants have been reported. The pathogenesis, symptoms, and diagnosis of constrictive pericarditis, as well as the role of the interprofessional team in its treatment, are covered in this exercise. This review article aims to describe the pathophysiology of constrictive pericarditis, review a patient's presentation with constrictive pericarditis, summarise constrictive pericarditis options for treatment, and discuss the importance of improving care coordination among interprofessional team members to enhance constrictive pericarditis patient outcomes. The 10-year survival rate for patients who get a pericardiectomy is around 50%. Medical treatment alone results in a short lifespan.

Keywords: *Cardiac MR; constrictive pericarditis; echocardiography; heart failure.*

1. INTRODUCTION

The pericardium is a fibroelastic sac that surrounds the heart. It has an effect on cardiac hemodynamics and serves as a protective barrier. Constrictive pericarditis is a condition in which the pericardium grows granulation tissue, causing a loss of pericardial elasticity and ventricular filling limitation. Although it is usually a chronic condition, subacute, transient, and occult variants have been reported [1].

2. ETIOLOGY

Chronic constrictive pericarditis has a number of causes and clinical signs that differ depending on the severity of the condition. It takes a long time to develop, which in many cases, no cause is ever discovered. In about 10% of cases, there is a history of acute pericarditis. Other instances of constriction are likely to have been preceded by a subclinical or hidden form of pericarditis. Any sort of pericarditis can cause pericardial constriction. They are classified

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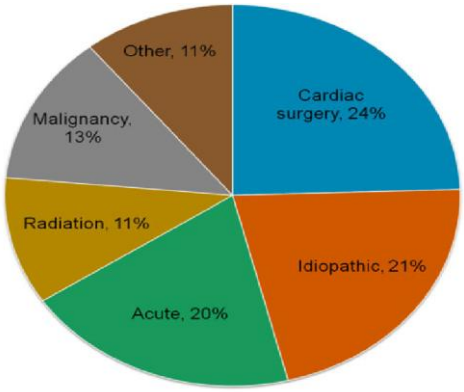

Fig. 1. Presumed etiology of constrictive pericarditis among 506 patients undergoing pericardiectomy at Mayo Clinic, Note that the two most common categories are postoperative the constriction and idiopathic etiology [3]

into three categories: common, uncommon, and uncommon. According to one study, the primary three causes of constrictive pericarditis are unexplained (probably viral), cardiothoracic surgery, and radiation therapy, which account for 46 percent, 37 percent, and 9 percent of cases, respectively (in patients who underwent surgical therapy of their constriction) (Fig. 1) [2].

3. COMMON FORMS

Idiopathic: There is no prior diagnosis in many cases, especially in rich countries. Certain types of situations are referred to as idiopathic. Many investigations have indicated that previously identified or undiagnosed viral pericarditis is connected to a substantial percentage of unexplained episodes of constrictive pericarditis. Although tuberculosis is most frequent cause of constrictive pericarditis in developing nations, it only accounts for a small fraction of cases in the United States and other developed countries. As a result of bacterial infections, purulent pericarditis has become less common. The most common bacterial source presentation used to be purulent pericarditis associated with pneumococcal pneumonia. The widespread use of antibiotics, on the other hand, has significantly altered the incidence and spectrum of purulent pericarditis, with cardiac surgery now being most common presenting symptom. It should be noted that the progression from acute pericarditis to constrictive pericarditis can occur more rapidly after bacterial pericarditis (eg, ≤6 months in some cases) [4].

It's possible to isolate a growing number of gram-positive organisms, particularly staphylococci-resistant strains. Gram-negative rods, such as Pseudomonas species, Escherichia coli, and Klebsiella species, have been detected, as well as Group A and B streptococci. Despite the fact that tuberculous and bacterial pericarditis are becoming less common, they are nonetheless connected to constrictive pericarditis. Per a prospective study, the prevalence of constrictive pericarditis after severe idiopathic/viral pericarditis was 0.76 cases per 1,000 person-years, while acute tuberculous pericarditis had 31.7 cases per 1,000 person-years and purulent pericarditis had 52.7 cases per 1,000 person-years. Viral illnesses that can induce constrictive pericarditis include coxsackievirus, hepatitis, adenovirus, and echovirus [5].

The long-term consequences of thoracic and mediastinal radiotherapy (used to treat hematologic, breast, and other malignancies) are becoming clearer. Radiation-induced heart disorders frequently include microcirculation injury, such as endothelial damage, capillary rupture, and platelet adhesion. An inflammatory response ensues, which can either dissolve or form contractures between the visceral and parietal pericardium. This series of events does have the ability to constrain the flow of blood. Radiation-induced constrictive pericarditis often develops 5-10 years after treatment and is often accompanied by a pericardial effusion. The median time between radiation and pericardiectomy, according to Bertog's research, was 11 years, with a range of approximately to 30 years. These findings were consistent with previous research [6].

Cardiac surgery: Any surgery or invasive procedure that opens, tries to manipulate, or destroys the pericardium might result in an inflammatory response, leading in constrictive pericarditis (postpericardiotomy syndrome). The most frequent cause of previous coronary artery bypass grafting (CABG) is constrictive pericarditis, which occurs when only a piece of the pericardium is removed [7].

4. LESS COMMON FORMS

Infection (fungal): Fungal infections are a rare condition of constrictive pericarditis in immunocompetent people. Nocardia species can be causal organisms, especially in endemic areas like the Ohio Valley. Aspergillus, Candida, and Coccidioides species are common infections in HIV patients and other immunocompromised individuals. Neoplasms can cause a malignant pericardial effusion (with or without tamponade) or an enclosing heart with thickness of both visceral and parietal layers, resulting in constrictive physiology. Although a number of neoplasms have been related to constrictive pericarditis, the most common metastatic malignancies are breast and lung carcinomas, and also lymphomas. Other malignancies that commonly affect the pericardium include melanoma and mesothelioma. Long-term hemodialysis can result in constrictive pericarditis, which would be habitually by a pericardial effusion [8].

Connective tissue illnesses: Pericardial autoimmune disorders are prevalent, and they usually manifest themselves as a mild pericardial effusion or an acute pericarditis episode. Chronic pericardial involvement is unusual, however it can occur in rheumatoid arthritis patients, and it's typically preceded by subcutaneous nodules. Systemic lupus erythematosus (SLE) or scleroderma can also produce constrictive pericarditis, with a poor prognosis in the latter case. Drugs: Procainamide and hydralazine-induced constrictive pericarditis have been related to a drug-induced lupus-like sickness. The use of methysergide has also been connected to constrictive pericarditis. As a result of traumatic and piercing injuries to the chest wall, constrictive pericarditis has been reported, most likely due to an inflammatory process. Traumatic pericarditis with constrictive pericarditis is uncommon. Post-MI constrictive pericarditis has been documented after myocardial infarction. After thrombolytic therapy, the patient usually develops Dressler syndrome or hemopericardium [9].

5. RARE FORMS

Constrictive pericarditis is a rare but very well complication following the implant of an epicardial pacemaker or an automated implanted cardiac defibrillator. Dwarfism, constrictive pericarditis, deformed fundi, and fibrous dysplasia of the long bones are all symptoms of Mulibrey nanism, an autosomal recessive disorder. In some persons, sclerotherapy for esophageal varices can result in constrictive pericarditis. Chylopericardium, a rare disorder, causes constrictive pericarditis [10].

6. COVID-19 AS A POSSIBLE CAUSE OF PERICARDITIS

SARS-CoV-2 (Severe Acute Respiratory Syndrome-Coronavirus-2) is a one-of-a-kind dilemma in the world of medicine. COVID-19 is most commonly associated with respiratory illness, which can vary from moderate flu-like symptoms to severe pneumonia and potentially lethal acute respiratory distress syndrome. SARS-CoV-2 infection begins with a protein termed SPIKE (S protein) generated in the viral coat attaching the virus to the membrane-bound version of angiotensin-converting enzyme 2 (ACE2), accompanied via primed by the serine protease TMPRSS2, which promotes virus uptake. ACE2 is a membrane-bound peptidase present in the lungs, heart, arteries, kidney, brain, and gut, among other places (Fig. 2) [11].

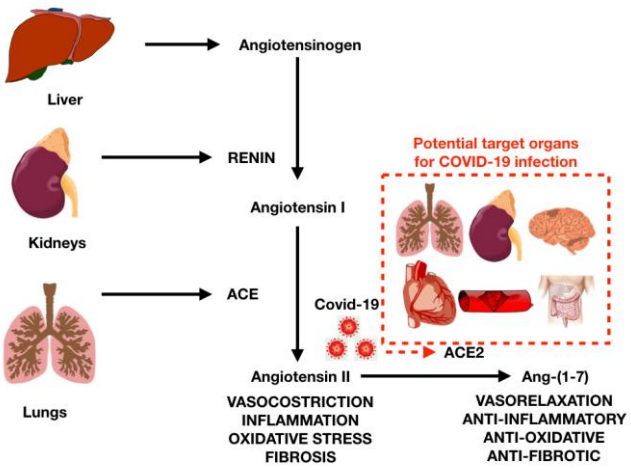

Fig. 2. the renin-angiotensin system (RAS), ACE and ACE2 (target of COVID-19) with potential target organs for COVID-19 infection [12]

7. PATHOPHYSIOLOGY

After the healing of the an acute episode of fibrinous or serofibrinous pericarditis or the resolution of a chronic pericardial effusion, granulation tissue obliterates the pericardial space, causing chronic constrictive pericarditis. The heart is encased with granulation tissue, which can become calcified as it contracts throughout time. The inflexible thickening pericardium limits ventricular filling because the elastic limit of the sick pericardium is considerably lower than that of a healthy pericardium. Unlike cardiac tamponade, which causes ventricular filling to be impeded throughout diastole, ventricular filling is undisturbed in early diastole and is only inhibited when the pericardium reaches its elastic limit. End-diastolic volume, stroke volume, and cardiac output are all lowered as a result. The thickened and damaged pericardium prevents the heart chambers from receiving the typical inspiratory drop in intrathoracic pressure [13].

There is a distinction between intrathoracic and intracardiac pressures. With inhalation, venous return decreases as pulmonary venous pressure falls. With inspiration, however, the left atrium pressure remains constant, as well as the flow from the pulmonary veins to the left atrium (LA) decreases. This separation of intrathoracic and intracardiac pressures distinguishes this situation from cardiac tamponade, in which intrathoracic pressure changes are still communicated to the heart and systemic venous return rises with inspiration. Both disorders equalise the pressures in the right atrial (RA), right ventricular (RV), left ventricular (LV), and pulmonary wedge; even so, in cardiac tamponade, the pressure decreases with inspiration, whereas in constrictive pericarditis, the RA pressure stays constant while the pulmonary wedge pressure drops (Fig. 3) [14].

8. HISTORY AND PHYSICAL EXAMINATION

Long-term symptoms are common among patients. Their symptoms could be caused by volume overloads, such as gaining weight and puffiness, or by a decline in cardiac output, such as fatigue and dyspnea while exercising. They may also feel discomfort or a rise in abdominal girth. Abdominal symptoms are caused by ascites or congestive hepatomegaly. On physical examination, the jugular venous pressure (JVP) is usually high, but it can be acceptable in early constrictive pericarditis. When JVP somehow doesn't decrease with inspiration, this is a sign of Kussmaul. Kussmaul's sign can also be detected in tricuspid valve dysfunction and right-sided heart failure. Pulsus paradoxus (a drop of more than 10 mm Hg in systolic blood pressure during inspiration) can be noted, however it is more common among patients with cardiac tamponade. Pericardial knock, an increased heart sound, is detected in approximately half the patients even before third heart sound. During an abdominal examination, ascites or hepatomegaly may well be detected. Depending on the aetiology, other signs of chronic illness, such as muscular atrophy, may be present. Peripheral swelling is also a possibility [16].

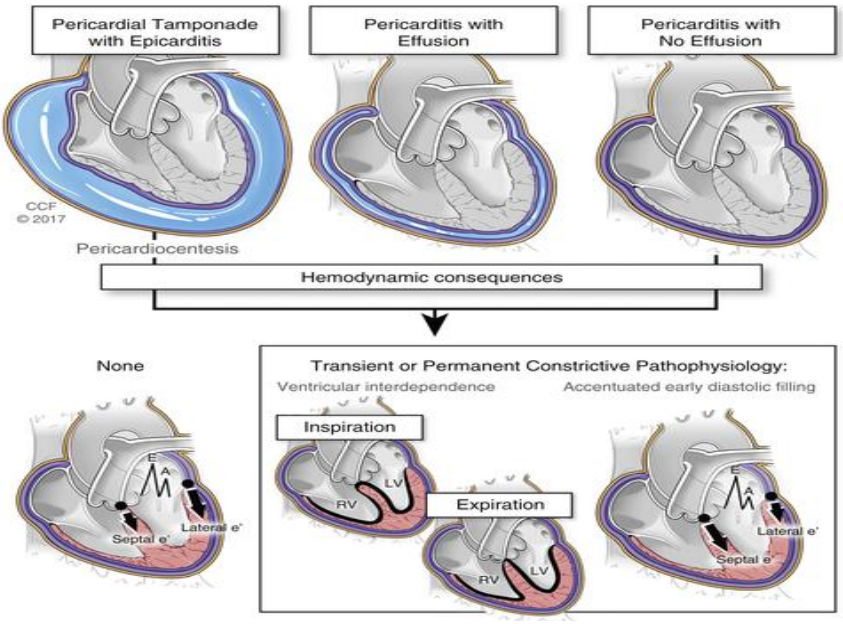

Fig. 3. Pathophysiology of constrictive pericarditis [15]

9. COMPLICATIONS

Early detection and treatment of pericarditis reduces the likelihood of long-term complications. The following complications can occur as a result of pericarditis: Pericarditis is characterised by the presence of fluid surrounding the heart, that can leads to serious complications. Pericarditis is closely linked with fluid around the heart, that can lead to more serious consequences. Pericardial effusion: Pericarditis is commonly associated with fluid all around heart, which can develop to more serious complications. Pericarditis constrictive chronic: Permanent pericardial thickening and scarring develop in some people with long-term (chronic) pericarditis, preventing the heart from effectively filling and draining. This uncommon complication is characterised by severe edema of the legs and abdomen, as well as shortness of breath. Cardiac tamponade is indeed a potentially dangerous disorder that happens when the pericardium fills with too much fluid. An excess fluid exerts pressure on the heart and prevents it from adequately filling. Blood pressure drops dramatically when less blood leaves the heart. Cardiac tamponade necessitates immediate medical attention [17].

10. EPIDEMIOLOGY

Constrictive pericarditis' clinical range has expanded, as was the case with many other diseases that were once mostly infectious. Constrictive physiology affects about 9% of people who develop it as a consequence of acute pericarditis. Assuming that acute pericarditis is detected clinically in only 1 in 1000 hospitalisations, constrictive pericarditis is likely to be identified in less than 1 in 10,000 enrollment. In a 2019 analysis of annual trends of patients admitted in US institutions for constrictive pericarditis from 2005 to 2014, researchers found a consistent prevalence of 9-10 occurrences per million. Infectious infections are still more prevalent in developing countries (tuberculosis has the highest total incidence) [18].

Age, gender, and race demographics: Despite the lack of paediatric data for epidemiologic studies, it is clear that the illness is unusual in adults and even rarer in children. In all age groups, patients who have been hospitalised or who have had cardiac surgery have a greater frequency. Cases have been reported in people as young as eight years old and as old as seventy years old. The predisposition is most likely a reflection of the underlying disease. The median age is 45 years old, according to historical surveys, however more recent research show a typical age is 61 years old (possibly reflecting ongoing demographic changes). There appears to be a male predominance, with some studies claiming a male-to-female percentage of 3:1. This illness has no racial predisposition. Patients with constrictive pericarditis who had pericardiectomy were younger, more likely to be male, and had fewer comorbidities than those who received medical therapy between 2005 and 2014 [18].

11. PROGNOSIS

Prognosis information is restricted due to the rarity of constrictive pericarditis. It is a possibly treatable disease if caught early, but if not, it can be fatal. Undiagnosed patients have such a ninety percent chances of death. The outlook is influenced by the nature of the illness. In to one research, postpericardiectomy survival rates were 71 percent and 52 percent after five and ten years, respectively. The prognosis for medical therapy alone is bad in the long run. Children who are not treated and patients who develop symptoms quickly have a reduced life expectancy [19].

Prognostic indications that have an impact on the outcome include: Greater medial mitral annulus early diastolic velocity (e') is connected to better outcomes in constrictive pericarditis. Separately, in a 2021 study looking at the pre - operative model for end-stage liver disease (MELD) (n = 175) and MELD-XI (exclude international normalised ratio) score (n = 226) as predictors of outcomes in patients having undergone pericardiectomy for constrictive pericarditis, researchers discovered an 8.7% 90-day death rate in patients undergoing pericardiectomy for constrictive pericarditis. MELD and MELD-XI scores that are higher Both ratings were connected to a higher likelihood of renal failure, as well as more intubations and blood transfusions. A high MELD-XI score was also connected to greater intubation and extended stays in the intensive care unit and hospital [19].

Between 2005 and 2014, in-hospital mortality for individuals having constrictive pericarditis was 7.3 % for those who had a pericardiectomy and 6.8% for those who got medicinal therapy. Lengthy survival after pericardiectomy is determined by the underlying cause. The greatest prognosis of the common causes is idiopathic constrictive pericarditis (average percentage survival at 7 years), following by constriction due to cardiac surgery (66 percent at 7 years). The worst postpericardiectomy result is postradiation constrictive pericarditis (27 percent survival at 7 years), that is likely owing to confounding comorbidities. Whenever the source of a constriction is unknown, cardiac dysfunction might

develop over time. Of older years, poor renal function, abnormal left ventricular systolic function, elevated pulmonary artery systolic pressure, decreased serum sodium level, worsening NYHA classification, and, as previously mentioned, radiation therapy as the reason of constrictive pericarditis, long-term outcomes of patients with constrictive pericarditis treated surgically are less favourable. The quantity of calcification in the pericardium has little bearing on survival [20].

Morbidity: When traditional medical treatment for heart failure fail, a lengthy diagnostic process usually follows, eventually leading to the diagnostic of constrictive pericarditis. A reduction in function is caused by low cardiac output with conditions such as heart failure, and also morbidity from prolonged systemic venous congestion. Multisystem failure can progress to an advanced stage of illness if global tissue hypoxia causes severe metabolic academia [20].

12. INVESTIGATIONS

The diagnosis of constrictive pericarditis can always be made only on the basis of test findings. However, as a result of the nearly universal observations of chronically elevated right-sided atrial pressure and passively congestion of the liver, kidneys, and gastrointestinal (GI) tract, following problems may be evident (see Laboratory Studies below). Only a few instances include elevated conjugated and unconjugated bilirubin levels, hepatic transaminases, and serum creatinine. To aid in the diagnosis, a variety of ancillary tests are typically required. These examinations include chest radiography, computed tomography (CT), magnetic resonance imaging (MRI), echocardiogram, and invasive hemodynamic measurements. Due to the invasiveness of several diagnostic tests, inpatient therapy is frequently required during the workup. As previously said, one of the most prevalent challenges that arise from diagnostic testing is determining the difference between restrictive cardiomyopathy and constriction [21].

13. LABORATORY STUDIES

A complete blood count (CBC) may reveal signs of dilutional anaemia if congestive heart failure (CHF) is present. Leukocytosis may be apparent if the pathogenesis is infectious, bacterial, or rheumatologic, or if patients are on steroid medication. Patients getting chemotherapy medicines for cancer treatment may develop leukopenia. As a result of the dilution produced by CHF, hyponatremia or pseudohyponatremia might develop. Contractile alkalosis (hypochloremia with hypercarbia) may occur when diuretics are used often. Renal insufficiency is marked by short-term increases in blood urea nitrogen (BUN) and long-term increases in serum creatinine. On arterial blood gas measures, metabolic acidosis (low pH and low bicarbonate) is commonly detected in right-sided CHF, even without compensating respiratory alkalosis (lower partial pressure of carbon dioxide). As a consequence of passively hepatic congestion induced by cor pulmonale, transaminase levels may rise. Protein-losing enteropathy (PLE) causes hypoalbuminemia when the portal system of people with hepatomegaly and ascites sees an increase in central venous pressure (as well as proteinuria that may approach the nephrotic range). If PLE is suspected, the levels of stool 1 antitrypsinase should be checked [22].

If active or chronic inflammation is present, nonspecific signs such as an elevated sedimentation rate (ESR) or normocytic normochromic anaemia may be observed. In postpericardiotomy syndrome, both the ESR and C-reactive protein (CRP) values may be elevated. The level of brain natriuretic peptide (BNP), a cardiac hormone released in response to increasing ventricular wall strain, is often slightly higher (usually below 150 ng/L) in constrictive pericarditis. BNP levels are levels of alt in restrictive cardiomyopathy (specific if greater than 650 ng/L) and therefore can assist distinguish between the two diseases. If a collagen vascular disease is suspected, antinuclear antibody (ANA) or rheumatoid factor (RF) levels must be examined. In cases of tuberculous pericarditis, the results of a pure protein derivative (PPD) skin test should be positive (unless the patient is anergic). Cytologic examination of the pericardial fluid, if present, helps diagnose a malignant cause (if such a cause is not otherwise apparent) [22].

14. CHEST RADIOGRAPHY

The vast majority of radiography findings are disappointing. Traditional signs, while not specific for the existence of constrictive pericarditis, were indicative of the diagnosis when presented in a compatible clinical context. For example, severe pericardial calcification is found in 20-30% of patients, but it is not diagnostic and does not suggest pericardial constriction. According the European Society of Cardiology, all clinically suspected constrictive pericarditis must receive chest radiography (frontal and lateral) having suitable technical features. If there is no significant pericardial effusion, the cardiac silhouette can look normal. It's possible that the azygos vein, superior vena cava, then both are dilated. Pleural effusions are fairly common, and they almost invariably affect both sides of the body. Pulmonary edema is a rare symptom that could indicate a heart or lung problem (Fig. 4) [23].

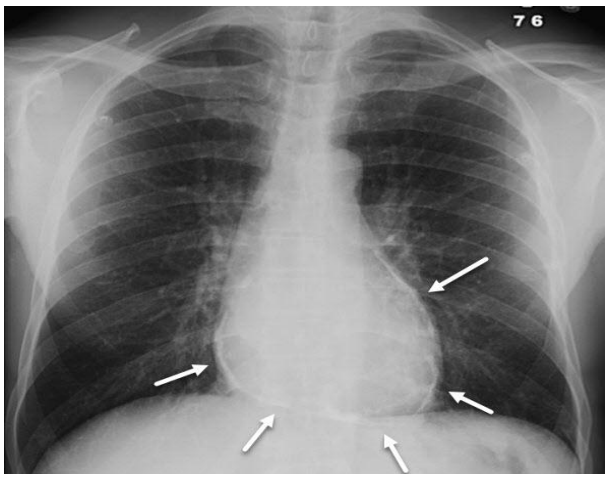

Fig. 4 Pericardial Calcifications, Constrictive Pericarditis [23]

15. ECHOCARDIOGRAPHY

Echocardiography was used to help detect constrictive pericarditis and differentiate it from restrictive as well as other cardiomyopathies for many years. Unfortunately, constriction does not have a pathognomonic echocardiographic result. When all of echocardiographic information is pooled in a clinical environment, however, the probability of constriction can usually be accurately diagnosed. Pericardial imaging with echocardiography is indeed not sensitive and, as a rule, is not considered a reliable method of viewing the pericardium. Although the pericardium can be echodense at times, it is not always the case. Computed tomography (CT) scanning and magnetic resonance imaging are the procedures of choice for visualising the pericardium (MRI). TEE is more reliable than TTE for detecting a thickened pericardium, especially if the pericardium is thick or extremely echogenic, although it is not as accurate as CT scanning or MRI. The European Society of Cardiology recommends TTE in all patients with suspected constrictive pericarditis (Fig. 5, 6) [24].

According to Welch et al research, .'s echocardiography can be used to differentiate constrictive pericarditis with restrictive myocardial disease and severe tricuspid regurgitation. The researchers looked at echocardiograms from 130 people who had constrictive pericarditis and 36 people who had one of the other two disorders. If the ventricular septal shift was present with a medial mitral annular e' velocity of 9 cm/sec or a hepatic vein expiratory diastolic reverse ratio of 0.79 or above, constrictive pericarditis could be diagnosed with a sensitivity of 87 percent and a specificity of 91 percent. The diagnoses could be made with ninety percent specificity and only 64 percent sensitivity if all three criteria have been met [26].

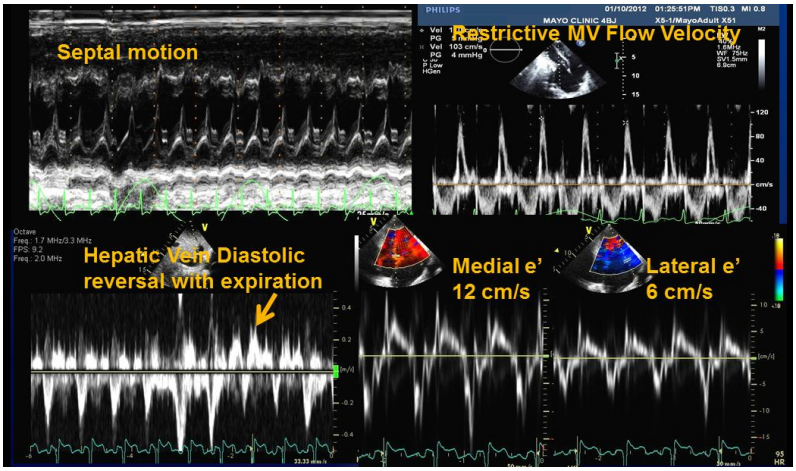

Fig. 5. Echocardiography Diagnostic Criteria for Constriction [25]

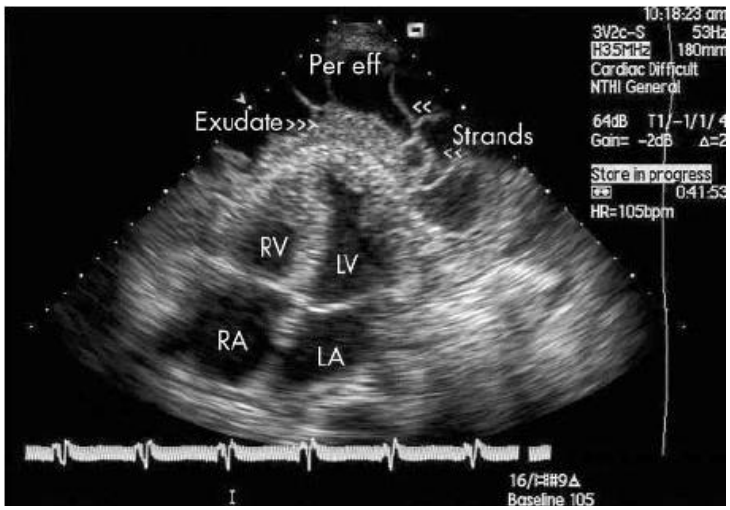

Fig. 6. Apical four chamber view of a two dimensional echocardiogram of a patient with tuberculous pericardial effusion showing multiple fibrin strands as linear or band like structures crossing the pericardial space or protruding from the epicardium or parietal pericardium and exudates. LA, left atrium; LV, left ventricle; Per eff, pericardial effusion; RA, right atrium; RV, right ventricle [25]

Two-dimensional echocardiogram: On two-dimensional echocardiography, systemic veins may seem dilated. Reduced intraventricular volumes can be detected via echocardiography. This technique is quite effective at detecting pericardial effusions. An interventricular septal motion can appear paradoxical, or the septum may seem flattened, as an indication of ventricular dependency. The inspiratory rise in chamber size was larger in individuals with constrictive pericarditis than all those with restrictive cardiomyopathy. The posterior displacement of the interventricular septum relative to some less compliant ventricular walls correlates with the auscultatory pericardial knock (which can be observed on M-mode and two-dimensional echocardiography as just an early diastolic septal notch or "septal bounce") (which are encased by the pericardium). This septal bounce has been regarded a finding consistent with constrictive physiology, with a sensitivities of 62% and specificity of 93%. On two-dimensional echocardiography, evidence of right-sided pressure overload, including such atrial septal displacement to the left with inspiration, can indeed be visible. With inspiration, the inferior and superior venae cavae, and the hepatic veins, may expand and collapse less. These really are vague symptoms that can occur as a result of a multitude of conditions in right-sided heart failure [26].

Doppler echocardiography is a type of echocardiography that can provide critical hemodynamic information. A variety of Doppler observations can indicate pericardial constriction when they are present, but their lack, like so many echocardiographic signals, does not exclude out constrictive hemodynamics. Early fast diastolic filling can be detected by measuring forwards flow at the mitral and tricuspid valve levels. Both E (early filling) and A (atrial filling) waves are the consequence of this process. The transtricuspid velocities have the opposite pattern as the transmitral velocities. The tricuspid valve's velocity increases with inspiration and decreases with expiration, whereas the mitral valve's velocity decreases with inspiration and increases with expiration. The decreased deceleration duration from these peak velocities appears to match the dip-and-plateau hemodynamics found with limited early diastolic flow [26].

According the pulmonary vein Doppler flow pattern, the diastolic input is greater than that of the systolic inflow, and the difference may even reverse. During constriction, the pulmonary venous flow pattern reveals systolic and diastolic forward flow, with diastolic flow decreasing on inspiration and increasing on expiration. This measurement can be utilised to evaluate if the mitral inflow trace has a diastolic pattern that is pseudonormalized. Pulsed-wave Doppler of a hepatic venous flow mimics the right-sided atrial pressure curves. Diastolic flow reverse is common in constriction, and it rises during expiration relative to inspiration. In severe constriction or a mix constrictive-restrictive picture, hepatic diastolic flow reversal can occur both in inspiration and expiration. In restrictive cardiomyopathy, however, hepatic flow reversal is more noticeable during inspiration [27].

Though it might be difficult to distinguish among constrictive and restrictive heart physiology, Doppler ventricular inflow patterns can help. Constriction

reduces ventricular filling thus improves ventricular interaction from a Doppler standpoint; restriction, on either hand, typically inhibits ventricular distensibility. Due to the inherent intraventricular septum, constriction generally generates more respiratory variation that restriction, with changes typically exceeding 25%. When the restriction is imposed, the E/A ratio is usually greater than 2, the deceleration duration is less than 150 ms, as well as the relaxation period is even less than 60 ms. Unfortunately, when certain Doppler evidence is missing, diagnostic reliability falls. Some breathing fluctuations may be understood if a pericardial effusion was present [27].

Tissue Doppler echocardiography is used to measure endocardial and epicardial tissue velocities (TDE). Because myocardial relaxation is preserved in pure constrictive pericarditis, the initial relaxation myocardial velocities (Ea, also known as Em) is normal, whereas it is abnormal in limitation (when the intrinsic myocardial disease is present). Given that a normal Ea is larger than 10 cm/s, a near-normal Ea (about 8 cm/s) favours constriction, whereas a much lower Ea favours restriction. A unique method for assessing cardiac longitudinal and radial distortion is speckled tracking of B-mode images. Circumferential deformation was shown to be reduced in patients with constrictive pericarditis (however, this may also be decreased in restrictive cardiomyopathy). In constriction, longitudinal strain and longitudinal early diastolic velocity are conserved, while in restriction, they are reduced [28].

Doppler interrogation may be limited if patients can't change their breathing enough, or if they have myocardial disease, atrial fibrillation, or serious lung disease (e.g., chronic obstructive pulmonary disease [COPD]), which can lead to false-positive findings. Other variations must be studied since COPD patients can now have Doppler transmitral input respiratory fluctuation. In contrast to COPD, constriction does not result in a considerable increase of inspiratory superior vena cava systolic flow. Load affects diastolic function measures (ie, dependent on preload and afterload). When atrial and ventricular filling pressures were low, Doppler interrogation findings may be falsely negative. If the atria and ventricles filling pressures both are high, respiratory fluctuation may be hidden. Preload can be lowered in such circumstances with medicine or dynamic actions like tilting the patient's head up or having them sit. These techniques have the potential to reveal respiratory variance [28].

16. COMPUTED TOMOGRAPHY SCANNING

Conventional computed tomography (CT) scanners may struggle to see the parietal pericardium. A high-resolution CT scan, on either hand, can clearly show the parietal pericardium. The thickness of the pericardium, the extent of calcification, and the spread of these findings are all easily quantifiable. The pericardium is typically 1-2 mm thick. If indeed the pericardial thickness is 3-4 mm or more, it is considered abnormal. Thickness of the pericardium greater then 4 mm aids in the differentiation of constrictive disease versus restrictive cardiomyopathy, and thickness greater than 6 mm adds even more specificity to the diagnosis of constriction. CT scans are the most effective way to identify pericardial calcification. Even though it is neither completely sensitive nor specific, pericardial calcification is usually linked to constriction. Ascites or hepatosplenomegaly are all symptoms of a difficulty with right ventricular filling, as are dilated vena cava, hepatic vein, and right atrium. Cine imaging may reveal early diastolic interventricular septal bouncing or inflammatory pericardial tethering (from a retrospective gated investigation) (Fig. 7) [29].

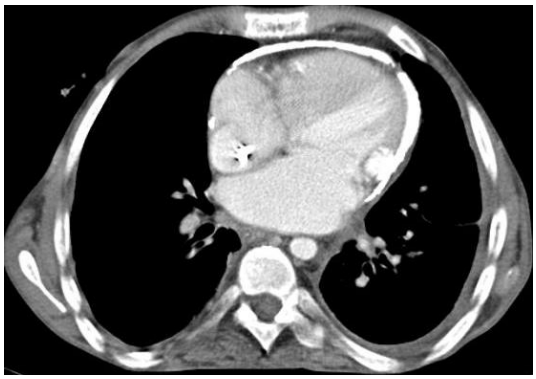

Fig. 7. Calcific constrictive pericarditis on CT [29]

False-negative results can occur if there is a long skinny pericardial scar with no significant thickening. In other words, normal pericardial thickness doesn't really rule out pericardial restriction, as well as the clinical situation must always be taken into account. Even if pericardial imaging yields normal results, if indeed the hemodynamics and presentations are normal, constrictive pericarditis must be explored. In patients with a verified diagnosis of constriction, CT scanning could be helpful for preoperative planning. This is especially true for people who had previous cardiothoracic operations, as it helps identify anatomic links, scar tissue, and bypass graft implantation. Coronary calcium, as well as potential coronary stenoses, can be detected. The location and intensity of pericardial calcification may guide the surgeon to a certain technique or indicate areas of the pericardium that require special attention during resection. CT scanning may also be used to identify associated radiation lung injury in radiation-induced constriction [29].

17. MAGNETIC RESONANCE IMAGING

MRI has become a sensitive approach for imaging the pericardium thanks to the advances of true, high-resolution magnetic resonance imaging (MRI) as well as the capacity to collect images in 50 milliseconds or less. The European Society of Cardiology recommends computed tomography (CT) as well as magnetic resonance imaging (MRI) as second-level imaging techniques again for assessment of constrictive pericarditis, such as assessing calcifications (CT scanning), pericardial thickness, and the degree and extent of pericardial involvement, with a level of evidence of class I, level C. Cardiac MRI has good diagnostic accuracy in the case of recurrent pericarditis and in identifying people who are at higher risk of complications (Fig. 8) [30].

The thickness, calcification, and other anatomic abnormalities of the pericardium can be assessed using MRI, just as they can with CT scanning. On MRI, constriction is associated with a long, narrow right ventricle ("tubing of the ventricle"), atrial dilatation, as well as a distinctive intraventricular septal "bounce" in early diastole. The septal bounce is linked to ventricular interdependence. A sigmoid septum is a septum that has a sigmoid form. True steady-state free-procession (SSFP) imaging, which allows for the analysis of changes in the ventricular septal shape and motion throughout the breathing cycle, can be used to investigate ventricular coupling. Gated MRI has an advantage in determining if pericardial fluid is hemorrhagic. Whenever pericardial calcification is severe, CT scanning may be the best option [31].

A larger pericardium does not always mean constrictive pericarditis is present; it must be clinically related. In the same way, the lack of pericardial thickening does not rule out the potential of hemodynamically significant restrictive pericarditis; constriction can happen in a scarred fibrous pericardium of normal thickness. MRI and gadolinium injection can be used to classify patients having reversible constrictive pericarditis. In a small study employing gadolinium MRI to measure cardiac inflammation, greater pericardial thickness and a greater standard of qualitative late gadolinium enhancement were reported to be linked with reversible constrictive pericarditis. Gadolinium enhancement and increased pericardial thickness were thought to be linked to increased inflammation that'd react to the anti-inflammatory medicine (nonsteroidal anti-inflammatory drugs [NSAIDs] and corticosteroids). Cardiac MRI may be useful in the diagnostic workup of patients who may have inflammatory constriction that would resolve with anti-inflammatory medication [31].

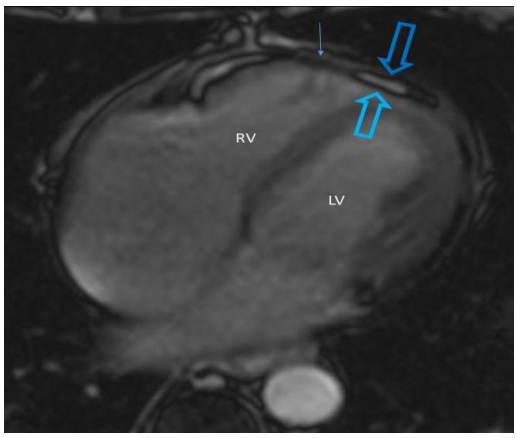

Fig. 8. Constrictive pericarditis on MRI [30]

Cremer et al looked studied pericardial delayed hyperenhancement as just a predictor of clinical improvement in individuals with constrictive pericarditis. Researchers are able to quantify the improvement in people who had increased pericardial delayed hyperenhancement and were using anti-inflammatory medicines for the first time. When it comes to differentiating constrictive pericarditis from restrictive cardiomyopathy, Amaki et al claim that left ventricular mechanics utilising both cine MRI–based tissue tracking or two-dimensional echocardiography–based tissue tracking can provide comparable diagnostic information [32].

18. ELECTROCARDIOGRAPHY

Constriction is not indicated by electrocardiographic signs. The ECG frequently exhibits nonspecific ST-T segment anomalies during constriction. The diagnostician can be tempted to look for signs and symptoms which resemble pericarditis. Chronic pericarditis, on either hand (which can be followed with constriction), is not associated with the electrocardiographic (ECG) symptoms that characterise acute pericarditis. A characteristic of acute pericarditis is diffuse concave ST-segment elevation, that must be distinguished from those other causes of ST elevation with PR depression. The ST elevation in the lateral V leads is usually larger that one-fourth of the T-wave height in most cases of acute pericarditis. If you have a history of these symptoms, you should be concerned about the possibility of constrictive pericarditis later on. The patient with restrictive cardiomyopathy, on the other hand, may have diffuse low-voltage tracings, bundle-branch obstruction, or AV conduction anomalies (Fig. 9) [33].

Although if chronic pericarditis evolves over time, there are no obvious ECG abnormalities. It's possible that the inverted T waves may persist or that all ECG values will normalise. Long-term circumstances can cause atrial fibrillation, however this isn't always the case. If a pericardial effusion occurs, a low QRS voltage in the limb and chest leads will be seen. Other causes of low voltage, including such long-term myocardial infarction (MI), pleural effusion, postoperative state, or other cardiomyopathies, must be distinguished. Electrical alternans (a beat-to-beat cyclic shift in the QRS axis which may include the P and T waves) must be evaluated when cardiac tamponade is present [34].

19. RIGHT-SIDED HEART CATHETERIZATION

Despite information from the history, physical symptoms, laboratory test results, other noninvasive procedures, a correct assessment of constrictive pericarditis may be difficult to make. When a diagnosis isn't certain with all of the data, invasive techniques like right cardiac catheterization can help to make or rule it out. The conventional hemodynamic criterion for constrictive pericarditis are as follows: The left and right ventricles' elevated diastolic pressures were normalised within 5 mm Hg. Right ventricular systolic pressure less than 55 mm Hg, mean right arterial pressure greater than 15 mm Hg, with right ventricular end-diastolic pressure larger than one-third of right ventricular systolic pressure are all signs of heart failure (narrow pulse pressure). Restricted pericarditis is by far the most likely diagnosis in the absence of these features. In addition, an accentuated x fall with a steep y descent on right artery pressure waveforms (W sign), and the square root sign (dip-and-plateau) on right or left ventricular tracings, would distinguish this diagnosis from cardiac tamponade. Another hemodynamic measure is the Kussmaul sign, which is described as the inability of the right arterial pressure to fall during inhalation. This can manifest itself in a variety of ways, including right-heart failure, acute tricuspid regurgitation, including systemic venous congestion [34].

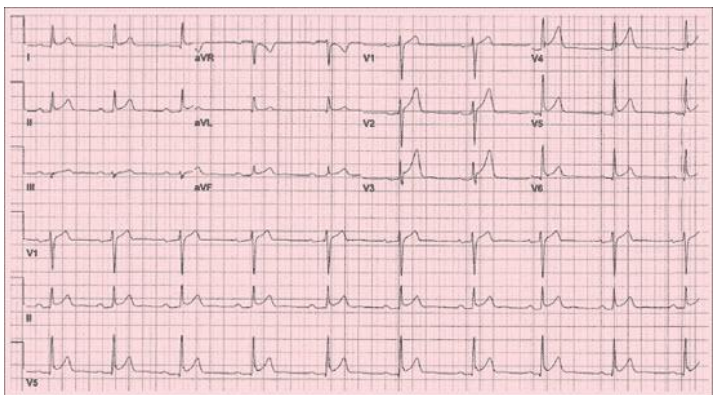

Fig. 9. ECG Changes in Pericarditis [33]

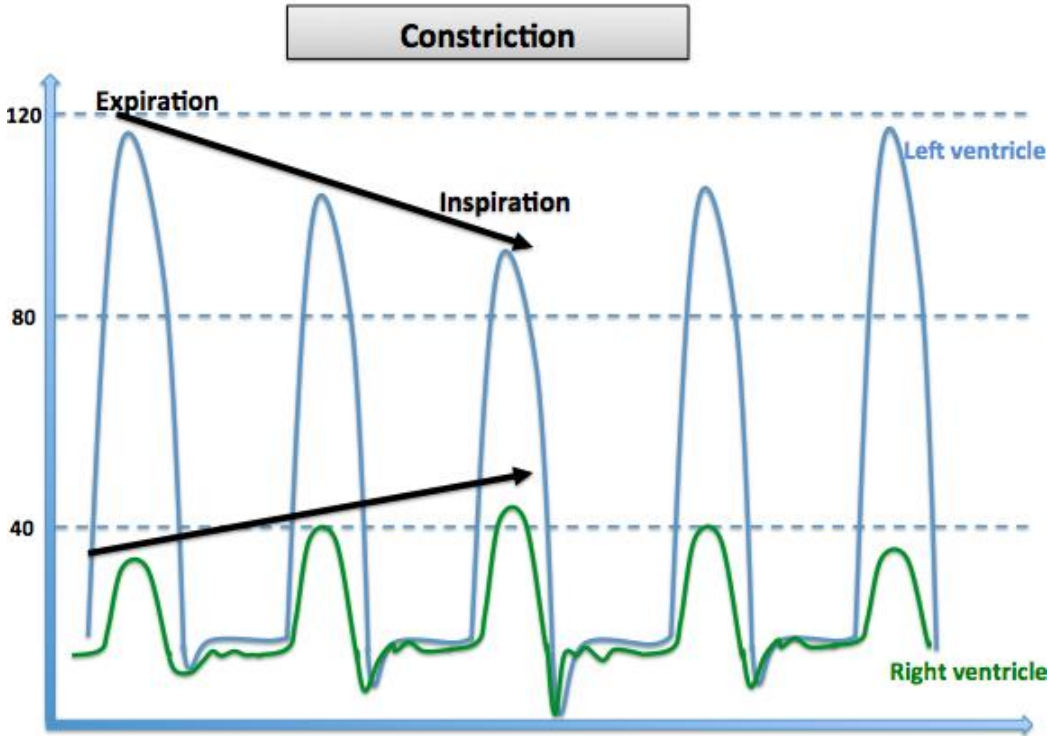

Fig. 10. Constrictive pericarditis: During inspiration the left ventricle becomes smaller and the right ventricle becomes larger in cardiac catheterization [36]

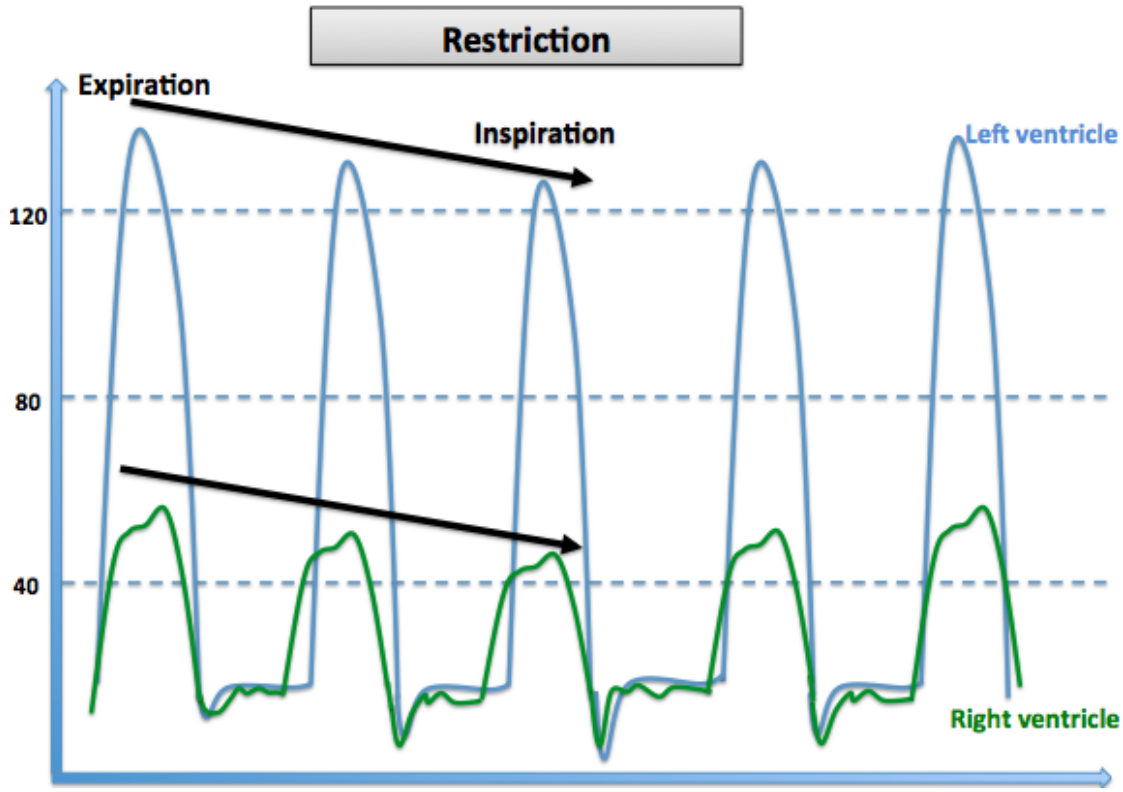

Fig. 11. Restrictive pericarditis: During inspiration both the right and the left ventricle become smaller in cardiac catheterization [36]

The respiratory variations in intrathoracic pressures really aren't transferred to the heart chambers during constriction, resulting in much less left-side filling on inspiration than right. Talreja et al discovered that constriction with a systolic area index larger than 1.1 showed 100 percent positive predictive accuracy, 97 percent sensitivity, and 100 percent specificity in a research analysing the areas beneath the right and left ventricular curves during inhalation and exhalation (comparing expiration with peak inspiration). Because this was not a randomised controlled clinical trial, there was selection bias; nevertheless, it could become a clinical screening criterion in the future. Although these markers are useful, there is always a degree of uncertainty when applying them in practise to diagnose constrictive pericarditis. Low-fidelity tracings are common with fluid-filled catheters, that can result to hemodynamic data misinterpretation. Because of the fluctuating RR intervals, irregular rhythms such as atrial fibrillation can affect ventricular filling pressures (Figs. 10, 11) [35].

Because diastolic filling pressures might affect hemodynamic data, some authors advocate infusing isotonic sodium chloride solution to show occult constrictive pericarditis if indeed the patient's left ventricular end-diastolic pressure is much less than 15 mm Hg. Small pressure variations in the lungs may be disregarded if indeed the filling pressures are much too high. Diastolic pressure equalisation can be caused by restrictive cardiomyopathy, cardiac tamponade, chronic obstructive pulmonary disease (COPD) as well as pneumothorax (pulmonary hyperinflation), dilated cardiomyopathy (that also, unless severe, could indeed cause all filling pressures to be significant), atrial septal defect, and volume depletion (when all filling pressures are low) [37].

20. PERICARDIAL AND ENDOMYO-CARDIAL BIOPSY

In rare cases, direct inspection and pericardial biopsy may well be required to detect restriction. Histologic abnormalities in the myocardium include fibrotic thickening, persistent inflammation, granulomas, and calcification. If constriction is widely suspected on clinical grounds, direct surgical inspection, biopsy, & pericardiectomy may well be indicated (despite imaging and hemodynamic evidence). Only after a comprehensive review to verify or eliminate out the diagnosis should this be done. Despite best efforts, it may be impossible to confirm the diagnostic of constrictive pericarditis until the surgical examination. Patients and families should be aware of this fact and understand that in some cases, surgery may be considered exploratory [37].

21. TREATMENT

The most common method of definitive treatment is surgery (ie, pericardiectomy). Surgical treatment usually improves hemodynamics and clinical outcomes quickly. Medical intervention, such as watchful observation or symptomatic treatment, has been advised in less severe instances; nonetheless, this choice is debatable. Rilonacept, an interleukin-1 (IL-1) cytokine trapping, was approved by the US Food and Drug Administration (FDA) in March 2021 to treat pericarditis and minimise the risk of recurrence in patients over the age 12 and up. In the early stages of the condition, diuretics were used to reduce pulmonary and systemic congestion. They should, however, be used with caution since any reduction in fluid overload can lead to a reduction in cardiac output. Complications may occur if constrictive pericarditis (as well as any associated explanation) is not diagnosed or treated adequately. Outpatient care may be appropriate in the early stages, especially if the diagnostic is a surprise and the symptoms are relatively stable. A low-salt, low-fluid diets is most likely to work. Even if no specific constraints are required, symptoms can typically considerably limit activity [38].

22. PHARMACOLOGIC THERAPY

Because the patient is frequently referred for surgical therapy after a definite diagnostic of constrictive pericarditis was made, no medicines are required. To help maintain a euvolemic state, diuretics and afterload-reducing medications are used with caution; decreasing preload as well as afterload can cause increased heart compression and sudden cardiac decompensation, especially when general anaesthetic agents are administered before the pericardiectomy. Rilonacept, an interleukin-1 (IL-1) cytokine capture, was approved by the FDA in March 2021 for the treatment of pericarditis and the reduction of the risk of recurrence in patients aged 12 or up. The RHAPSODY clinical trial has been used to support rilonacept's approval [39].

Medical treatment is ineffective in the vast majority of cases and if there is an inflammatory responses component. Nonsteroidal anti-inflammatory medicines (NSAIDs), cyclooxygenase (COX)-2 inhibitors, colchicine, corticosteroids, or even a mixture of these medications may be effective in constrictive pericarditis, but not in acute pericarditis. Even after excellent treatment for acute pericarditis, constriction might develop over time. People with a diagnoses of constriction who are clinically stable may well be given a fair trial of conservative treatment (NSAIDs and/or steroids) for 2-three

months because transitory constrictive pericarditis has been documented. Only persons with a decent volume status and controllable symptoms should explore this. The FDA authorized Rilonacept, an interleukin-1 (IL-1) cytokine trap, in March 2021 to treat pericarditis and reduce the risk of recurrence in patients aged 12 and up. The RHAPSODY clinical trial (N = 61) was used to support the approval of rilonacept. Pericarditis recurrence was seen in 2 of 30 patients (7%) who received rilonacept, compared to 23 of 31 patients (74%) in the placebo group [39].

Other things to consider while addressing constrictive pericarditis therapeutically include: Subacute constrictive pericarditis could respond to steroids if given before pericardial fibrosis develops. Diuretics (particularly loop diuretics) are the gold standard for alleviating congestion and optimising clinical volume status; however, they can lower preload to the point of reducing cardiac output, necessitating careful monitoring. Any other medications to treat constrictive pericarditis patients would've been customised to the exact cause of the pericardial condition. Any treatment that targets the underlying condition (such as antituberculosis medication) is acceptable. Treatment is required for complications (such as atrial arrhythmias). Because the sinus tachycardia that is prevalent in constrictive pericarditis does have a compensatory function, maintaining cardiac output inside a setting of fixed stroke volume (secondary to fixed diastolic filling), beta-blockers and calcium channel blockers should be avoided in general. Persons with extremely advanced related symptoms to constriction may derive little advantage from pericardiectomy; these are most often the same patients who have particularly high survivability [39].

23. PERICARDIECTOMY

A full pericardiectomy is the only treatment that can guarantee a cure. The results are generally better when treatment is started earlier in the course, whenever there was less calcification and the danger of aberrant myocardium or severe heart failure is reduced. Because patients in NYHA class I (i.e., asymptomatic) or slightly earlier NYHA stage II characteristics may remain clinically stagnant for years, considerable judgement is required. Pericardiectomy is a night before going to bed and often technically challenging procedure. The two most prevalent methods are an anterior thoracotomy and a median sternotomy. The pericardium should be decorated as thoroughly as possible, especially in the diaphragmatic-ventricular contact zones. An excimer laser can be used to treat severe adhesions between both the pericardium and epicardium. Complications include bruising, atrial and ventricular arrhythmias, and ventricular wall ruptures. In published articles, surgical mortality has indeed been reported to range from 5% to 15%, with one study estimating a 6.1 percent 30-day intraoperative mortality. Among the causes of death are progressive heart failure, sepsis, renal failure, respiratory failure, or arrhythmia. Approximately 80% to 90% of individuals who do have a pericardiectomy postoperatively achieve NYHA class I or II (Fig. 12) [40].

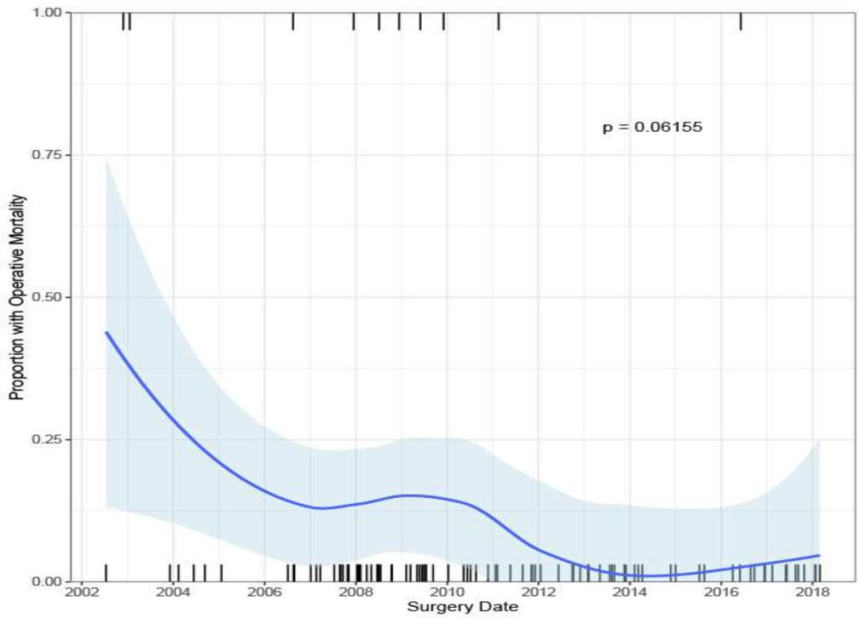

Fig. 12. Outcomes of Pericardiectomy for Constrictive Pericarditis following Mediastinal Irradiation [40]

Researchers looked studied the clinical outcomes of 99 consecutive patients who already had pericardiectomy just at Montreal Heart Institute over the course of 20 years. They discovered that hospital mortality was 7.9% in single pericardiectomy patients, and that patients who were performed on within 6 months of symptom onset had a lower mortality risk. The percent of late survivors improved their functional capacity after pericardiectomy, and preoperative clinical features and comorbidities are important in determining mortality risk. Despite the fact that pericardiectomy usually relieves symptoms, evidence of abnormal diastolic filling (that can be connected to clinical status) is constantly left behind. According to one study, only 60% of patients had complete restoration of cardiac hemodynamics. Although some patients healed over time, most with a longer history of preoperative complaints were more likely to develop chronic diastolic filling abnormalities, supporting the case for early surgery in symptomatic patients. Patients who continue to have symptoms after one successful pericardiectomy may well have a mixed constrictive-restrictive image. Thirty percent of 58 individuals who had complete pericardiectomy for constriction always had significant symptoms after four years. These individuals were more likely to have a prolonged restrictive or constrictive trend in their transmitral or transtricuspid Doppler signals, as determined by respiratory monitoring [41].

In a research of 25 patients who already had pericardiectomy due to symptomatic chronic constrictive pericarditis (decreased exercise capacity and sleep-disordered breathing), there was an improvement in peak increase in oxygen uptake, quality of life, and sleep, but no significant change in sleep-disordered breathing. Different approaches of reaching the pericardial area, such as video-assisted thoracoscopy, are being investigated. Further development of such gadgets may help to improve pericardial disease patients' diagnostic and therapy choices [41].

Myocardial atrophy or fibrosis, as seen via computed tomography (CT) scanning, seems to be connected to cardiac mortality and morbidity before surgery. By removing these patients, the mortality rate is maintained at the lower end of the range (5 percent). Patients who are weary and also have edema or other symptoms of fluid retention may experience low cardiac output after surgery. In individuals with low cardiac output, maintain high left atrial pressure, sympathomimetic infusions, or may both be necessary. Mechanical circulatory support, including such extracorporeal membrane oxygenation (ECMO) or intra-aortic balloon counterpulsation, can be used in critically ill patients [41].

24. GUIDELINES SUMMARY, EUROPEAN SOCIETY OF CARDIOLOGY (ESC)

Updated guidelines for the assessment and therapy of constrictive pericarditis were issued by the European Society of Cardiology (ESC) in 2015, which are summarised here. All of these proposals are founded on class I, level C evidence unless otherwise noted. A transthoracic echocardiography, and also frontal and lateral chest radiography with acceptable technical features, should be performed on any and all patients suspected of having constrictive pericarditis. To examine calcifications (CT scanning), pericardial thickness, and the degree and degree of pericardial involvement, second-level imaging techniques including such computed tomography (CT) scanning and/or cardiac magnetic resonance imaging (CMRI) are advised. Cardiac catheterization is used when noninvasive diagnostic methods fail to produce a definite diagnosis of constriction. A most prevalent treatment for chronic constriction is pericardiectomy. To prevent constriction advancement, medical therapy is essential for certain etiologies of pericarditis, as well as advanced instances or in the presence of higher risk of surgery, or mixed types with cardiac involvement. Empiric anti-inflammatory therapy may be considered in cases of transient or new diagnosis of constriction with concurrent evidence of pericardial inflammation on CT/CMRI or results of laboratory studies suggesting inflammation (eg, elevated C-reactive protein [CRP] or erythrocyte sedimentation rate [ESR]) (Class IIb, level C evidence) [42].

25. DISCUSSION

Constrictive pericarditis occurs when a thicker, fibrotic pericardium obstructs regular diastolic filling for whatever reason. The parietal pericardium is the most typical site of involvement, however the visceral pericardium also can be impacted. Acute or subacute pericarditis (which may or may not be symptomatic) can deposit fibrin, resulting in a pericardial effusion. Pericardial inflammation, fibrotic scarring that persists, calcification, and reduced heart filling are all common complications. Myocardial infarction (MI), aortic dissection, pneumonia, influenza, other connective tissue disorders all have symptoms that are comparable to constrictive pericarditis. This overlap can cause even the most skilled diagnostician to get perplexed. Increased suspicion of constriction pushes constrictive pericarditis to the top of a broad list of differential diagnoses, enabling for a more precise diagnosis and faster treatment. The difficulty in separating constrictive pericarditis from restrictive

cardiomyopathy and other illnesses associated with high right-sided pressure, which all share similar symptoms, clinical findings, and hemodynamics, is a classic diagnostic challenge [42].

Although a thorough history and physical examination remain the cornerstones of evaluation, technological advances have made diagnosis easier, especially with the proper the use Doppler echocardiography, high-resolution computed tomography (CT), magnetic resonance imaging (MRI), and invasive hemodynamic measurement. Pericardiectomy is by far the most commonly definitive treatment. Hemodynamic and symptom improvements happen soon. Medical intervention, such as watchful observation or symptomatic treatment, has been advised in less severe instances; nonetheless, this choice is debatable. The underlying disease usually determines the prognosis. Malignancy and heart failure symptoms classified as class III or IV by the New York Heart Association (NYHA) are connected to a poor prognosis [42].

26. CONCLUSION

In the United States, constrictive pericarditis is infrequent, and long-term evidence is quite rarer. Anecdotal evidence suggests whenever a diagnosis is established early, the results are good. However, death rates of greater than 90% are common when the condition is diagnosed or left untreated. Patients who have a pericardiectomy have a 50 percent 10-year survival rate. A limited lifetime is the effect of medical therapy alone. The cause of constrictive pericarditis determines the prognosis. Radiation-induced constrictive pericarditis patients had the worst outcomes, while open-heart surgery patients have the best. Negative prognostic signs include advanced age, renal failure, a low ejection fraction, and elevated pulmonary artery pressures. Most patients experience multiorgan failure, hypoxia, including metabolic acidosis if they are not treated. Patients should be sent to a tertiary care centre that specialises inside the treatment of constrictive pericarditis if treatment of constrictive pericarditis isn't really available at the presenting hospital.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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