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A Review Article on Pericarditis

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HUMAN

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ABSTRACT

Pericardial inflammation is a hallmark of acute pericarditis. It is associated with a high mortality rate compared to myocardial tamponade. It can be subdivided into bacterial, fungal, opportunistic, traumatic, and metabolic causes. The diagnosis of this ailment is crucial, as prompt and suitable treatment is linked to a positive prognosis. Despite advances in diagnostic techniques, individuals affected by this chronic and debilitating condition can now be accurately diagnosed and potentially restored to normal life through timely resection procedures. Surgical resection is the treatment of choice for patients with this condition. This paper aims to provide a comprehensive review of the current literature on the diagnosis and treatment of this condition.

INTRODUCTION:

Pericardial inflammation is a hallmark of acute pericarditis. It is a frequently encountered condition seen in both primary and secondary healthcare facilities, arises from multiple causes, leading to its diverse presentation. It contributes to up to 5% of emergency department visits due to pain in the chest not linked to myocardial infarction and is detected in about 0.1% of hospital stays. Inside the pericardium, the visceral layer and the outer parietal layer are affected by an inflammatory process that causes pericarditis. Every single layer is roughly 1-2 mm thick. Within these two compartments exists a serous liquid named pericardial fluid, which has a volume of approximately 15–35 mL [1]. Compared to myocarditis, Younger males exhibit a higher prevalence of pericarditis. Men and women appear to be more prone to pericarditis in distinct ways due to differences in sex hormone levels, particularly testosterone. Tragically, there are instances in which problems might develop over a long time such as enduring constrictive pericarditis, halfway (recurrent illness), or in a brief period as Cardiac Tampnode if neglected and not addressed immediately. Complication rates vary significantly based on the root cause which includes treatment approaches of glucocorticoid utilization and specific patient attributes such as age and gender. Within the framework of a systemic ailment or as a stand-alone illness, acute pericarditis could occur. 15% to 30% of individuals are found to experience their initial relapse following their first attack of acute pericarditis between 25 and 50% face a second, and 20 to 40% encounter a third [2]. Acute, ongoing, recurrent, and chronic pericarditis are the different types that can occur. Though pericarditis tends to be idiopathic, it can arise from both viral and not transmissible sources. Signs, prognosis, and stages of pericarditis and clinical manifestation may differ dramatically. Acute pericarditis often resolves on its own and poses minimal threat to life, however it can result in severe short-term impairment and Complications. Despite being largely resolving themselves and not fatal, this condition may still result in substantial short-term impairment and complications [3].

CLINICAL PRESENTATION:

The following are the chief clinical signs of acute pericarditis:

1. Pain in the chest:

Bending forward and raising up may soothe chest pain, which is usually severe and pleuritic. Pericardial membrane inflammation and higher deterioration are the causes of it [4].

2. Friction rub on the pericardium:

A specific sound heard during auscultation, when using a stethoscope over the chest left sternal border which is characterized by a scratchy or grating noise. The three distinct patterns of friction rubs are the diastole of the ventricular muscle, the arterial systole, and the systole of the ventricular [4]. Pericarditis can cause sporadic or temporary friction rub, but it generally lasts for many hours to several days [6].

3. Changes in ECG:

Broad ST elevation or PR decline [4]. Acute myocardial infarction, early repolarization, and athletic heart have to be differentiated from the abnormal ECG findings that accompany acute pericarditis [6].

4. Mild Level – 60% of an effusion in the pericardium [4].

A temperature greater than 38 degrees Celsius indicates a particular etiology of pericarditis, but sinus tachycardia and low-grade fever are typical. Since the pericardium is crossed by the nerve known as the phrenic nerve, which connects the trapezius ridge, discomfort radiating to this location is typically indicative of pericarditis. The spectrum of manifestations encompasses those of major right-sided heart failure as well as alterations in effort tolerance [5].

People who have acute bacterial, extremely sensitive, or autoimmune-induced pericarditis typically have chest pain. Depending on causing it, the pain could start suddenly or gradually, but it usually feels like a severe piercing. The discomfort associated with pericarditis worsens with exercise, much like atherosclerosis does. To prevent their condition from getting worse, people usually remain still and inhale gently [6].

TYPES OF PERICARDITIS:

Viral Pericarditis:

Viral infection stands as the primary cause of pericarditis among children. Typically, patients experience a preceding period of 10 to 14 days marked by respiratory or gastrointestinal symptoms. Coxsackievirus, echovirus, adenovirus, Epstein-Barr virus, influenza virus, and human immunodeficiency virus are among the most prevalent agents responsible for this condition. Those affected by viral pericarditis commonly exhibit symptoms such as fever and

chest pain, often accompanied by a friction rub. Notably, unlike other infectious disorders, viral pericarditis frequently coexists with myocarditis [7].

Acute Pericarditis:

It is an inflammation that can stem from diverse causes, with infection, autoimmune diseases, rheumatic fever, uremia, malignancies, drug reactions, or post-cardiac surgery situations being the most frequent triggers. Surprisingly, around one-third of cases lack a clearly identifiable cause and are labeled as idiopathic [7].

Bacterial Pericarditis:

Bacterial pericarditis, while less prevalent, is linked to a higher mortality rate compared to viral infections. Patients often exhibit signs of toxicity, including elevated temperatures, irritability, and chest pain. Cardiomegaly is commonly observed in chest X-rays. The infection can be primary, occurring after thoracic surgery, or secondary, stemming from the spread from another site such as pneumonia. Staphylococcus aureus and Haemophilus influenza stand as the two most frequent bacterial pathogens associated with this condition [7].

Purulent Pericarditis:

It is primarily caused by bacterial pathogens, accounting for approximately 5% of instances. The bacteria can reach the pericardium through hematogenous spread or via extension from neighboring organs, particularly the lungs or pleural space [8].

The incidence of purulent pericarditis is expected to increase further. Changes in the population at risk have significantly altered the spectrum of organisms causing this condition. Recognizing this ailment is crucial, as prompt and suitable treatment is linked to a positive prognosis. Yet, in numerous documented cases, the diagnosis was confirmed only during post-mortem examinations [9].

Tuberculous Pericarditis:

In developed countries, M. tuberculosis contributes to approximately 4% of acute pericarditis cases and is involved in 7% of presentations with tamponade. Its significance persists in developing nations and among immune-compromised individuals. Diagnosing tuberculosis-related pericarditis may necessitate a pericardial biopsy. Additionally, this condition can lead

to pericardial effusion or constriction in as many as 50% of cases, adding to its complexity [8].

While infrequent, still occurs among immune-compromised individuals and in regions where tuberculosis is widespread. Usually spreading either directly from mediastinal lymph nodes or through the bloodstream from a distant site, tuberculous pericarditis often manifests without the typical pulmonary or extra-pulmonary symptoms of tuberculosis. Patients affected by this condition frequently develop significant pericardial effusions, with cardiac tamponade being a common outcome. Before the availability of antituberculous medications, this infection was fatal in the majority of cases [7].

Constrictive Pericarditis:

It refers to the thickening of the pericardium with dense fibrous tissue, leading to persistent compression of the heart. This condition restricts the heart's ability to fill during relaxation (diastole), resulting in signs such as venous congestion and reduced cardiac output. While rare, the understanding of its underlying mechanisms has significantly improved. Thanks to advancements in diagnostic techniques, individuals with this chronic and debilitating condition can now be accurately diagnosed and potentially restored to normal life through timely pericardial resection procedures [10].

Neoplastic Pericarditis:

Pericarditis related to neoplasms commonly arises as a secondary condition, often due to local tumor invasion or spread through the lymphatic or hematogenous routes. Instances of primary malignant diseases affecting the pericardium are infrequent [8].

HISTORY:

Temporarily, pericarditis can be further classified as "chronic pericarditis" if it persists for more than three months, "incessant pericarditis" if it lasts for more than three months, and "acute pericarditis" if it lasts for less than four weeks. The phrase "recurrent pericarditis" refers to an episode that happens following a 4-6 week period without symptoms in between episodes [11].

It is possible to distinguish between ischemic pain, which is exacerbated by emotion and activity and relieved by rest or nitroglycerin, and pericardial pain. Additionally, the latter is

usually non-positional, non-pleuritic, and non-palpably reproducible. A clinical distinction is often challenging due to the multitude of atypical presentations of ischemic pain. It is more likely that a pulmonary disease is secondary to pleuritic chest pain if it is accompanied by respiratory symptoms like coughing or sputum production and does not go away when the patient sits up. Conversely, lower chest pain that is related to food intake, non-pleuritic, and improves with forward-leaning should be referred to an abdominal cause, such as acute pancreatitis or esophagitis [12].

CAUSES:

Pericarditis can be caused by multiple causes. It can be separated into infectious and non-infectious pericarditis. Infectious pericarditis can be subdivided into bacterial, tuberculosis, viral, fungal, rickettsial, mycoplasma, parasitic and others. Non- infectious pericarditis can be subdivided into immunoreactive, neoplastic, traumatic and metabolic causes. The main causes of acute pericarditis include idiopathic, vasculitis and connective- tissue disease, disease in adjacent structures, metabolic disorders, neoplastic disorders, trauma and other symptoms include postmyocardial and pericardial injury syndromes, inflammatory bowel disease, stevens- Johnson syndrome, acute pericarditis, etc [13]. The causes of transient constrictive pericarditis include postpericardiotomy, viral, bacterial, idiopathic, collagen vascular disease, trauma, malignancy and tuberculosis [14]. Many of the studies show that pericarditis can be caused by impair diastolic cardiac function leads to cardiac failure manifested as systemic without the pulmonary congestion [15].

PATHOPHYSIOLOGY:

The fibrous pericardium consists of the outer sac and inner sac, which is the interior double-layered sac, make up the pericardium. The proximal portions of the great vessels are found in the pericardial sac. In this pericardium provides mechanical support to the heart, reduced the friction between the heart and other structures, and limit the distention of the heart. Regularly the function is achieved via the presence of a small amount of pericardial fluid (25-50 ml) produced by the visceral pericardium. Intrapericardial pressure is equal to intrapleural pressure. The cause of infectious and non- infectious are responsible for the inflammation of pericardial layers leading to increased production of pericardial fluid as exudate [16].

The three major effects of pericardial inflexibility of pathophysiological hallmarks are

1. Dissociation of Intrathoracic and intracardiac pressures with respiration

2. Right and left ventricular interdependence and

3. Impaired diastolic filling and heart rate

1. Dissociation of intrathoracic and intracardiac pressures with respiration:

The understanding of intracardiac pressure pathophysiology is very complicated. Because the pulmonary veins are intrathorial, the influence of respiration on the direction of change in pressure within the heart chamber is mirrored. The stimulation drop of intrathoracic pressure often travels to every heart to the pulmonary veins and chambers. The impact factor between the left sided pulmonary veins and the cardiac structures are largely remains unaltered. The pericardium effectively separated the heart chambers from variations. The pressure of the pulmonary veins and left ventricle can decreases with inspiration, resulting in an inspiratory reduction in the velocity of diastolic flow in pulmonary veins and reduction in left sided filling [16].

2. Right and left ventricular independence:

In Contrictive pericarditis, the ventricles are pathologically linked and operate inside a noncompliant pericardium. The overall volume of blood entering the heart during the respiratory cycle is rather constant in patients with constrictive pericarditis. As a result, the septum shifts to the left and the inspiratory decrease in left ventricular filling is accompanied by an increase in right ventricular diastolic filling. With expiration, the opposite physiological effects on the ventricles filling gradients and septal shift are observed. The cardiac chambers are not affected by the rise in intrathoracic pressure that occurs during expiration. As a result, during expiration the septum moves to the right and flow into the left ventricle, increasing the diastolic hepatic venous flow and decreasing the flow velocity in the venacava [16].

3. Impaired diastolic filling and heart rate:

All the cardiac chamber of diastolic filling is restricted by unyielding fibrotic encasement of the heart, which elevates end-diastolic pressures. Many variables including heart rate, atrial systolic force, loading conditions and myocardial relaxation affect diastolic filling. Due to the significant limitation of filling in late diastole, arise in heart rate becomes effective means of maintaining cardiac output [16].

DIAGNOSIS:

An echocardiography, chest X-ray, and electrocardiogram should be performed on all patients who are suspected of having acute pericarditis. It's also important to get cardiac inflammatory and injury markers like troponins, CRP, and ESR (Class I, LOE C). Since most cases respond quickly to empirical treatment, this workup may be sufficient in developed countries where tuberculosis infection is not suspected and additional diagnostic testing is not necessary. More testing that is geared towards a particular etiology may be necessary if a particular cause is suspected [17].

If additional work up is sought, baseline tests like a complete blood count, basic metabolic panel, liver function tests, and thyroid stimulation hormone level are advised as they may point to a specific etiology like uremia or infection. Blood cultures, viral seromarkers, and tuberculosis testing (like PPD or quantifier TB essays) may also be carried out on a subset of patients. Since there is a strong correlation between an immune-compromised state and fungal or tuberculosis infection, patients who test positive for HIV using antibody/antigen testing or nucleic acid testing (NAT) should also be tested for opportunistic infections. Getting anti-nuclear antibody (ANA) serologies and doing focused testing for a suspected systemic illness like sarcoidosis are two possible next steps in the diagnostic process [17].

TREATMENT:

Treating the underlying cause is the first step in treating acute pericarditis. Individuals with malignancy and tuberculosis should receive therapy targeted at the main disease process, whereas patients with uremic pericarditis should receive more frequent dialysis. Standard treatment for tuberculosis involves the use of quadruple antibiotics (rifampicin, isoniazid, pyrazinamide, and ethambutol) for at least two months, then isoniazid and rifampicin for a total of six months, either with or without the previously mentioned adjunctive high-dose prednisolone [18].

The majority of patients will have idiopathic acute pericarditis, which is safely treated as an outpatient condition with medication alone. It's also advisable to limit your activities beyond leading a sedentary lifestyle until your symptoms go away or your CPR returns to normal. Patients who exhibit indicators of a poor prognosis or who do not improve after a week of treatment ought to be admitted and given more attention. The presence of a large pericardial effusion (>20 mm in thickness), a fever (> 38 C), subacute or recurrent presentation, or

cardiac tamponade physiology on echocardiogram (such as right ventricular diastolic collapse, transmitral flow respirophasic variation more than 25% throughout the respiratory cycle, and a dilated inferior vena cava with inspiratory collapse < 50% indicating elevated right atrial filling pressures) are some of these markers [19].

If NSAIDS/ASA and colchicine are not recommended, low to moderate doses (e.g., prednisone 0.2–0.5 mg/kg/day or equivalent) with a gradual taper may be used instead. Although the latter frequently provides quick clinical improvement, there is a lot of evidence to suggest that using steroids raises the risk of recurrent pericarditis following therapy discontinuation. For most patients, corticosteroids are therefore not advised as first-line treatment unless an autoimmune cause for acute pericarditis is found. Once symptoms have subsided and CPR has returned to normal, the initial dosage should be continued and then gradually reduced.

Although symptom relief is the primary clinical metric used to evaluate treatment response, repeated CRP measurements can also be useful. If the effects of NSAIDs or anti-inflammatory drugs (ASA) are insufficient [20].

Steroid-sparing immunosuppressive drugs, such as azathioprine, IVIG, or anakinra (an IL-1 receptor antagonist), may be utilized as third line therapy for corticosteroid-dependent recurrent pericarditis. Pericardiectomy will always be the last option [21].

Small studies have linked an increased risk of hemorrhagic pericardial effusion and cardiac tamponade to specific forms of acute pericarditis, including iatrogenic and uremic pericarditis. Therefore, in those patients, it might be wise to discontinue anticoagulation when it is practical, even in the absence of compelling evidence. Nevertheless, there are no official guidelines, and the suggestions are solely supported by professional judgement. Additionally, unless there is a clear indication, concurrent use of ASA should be avoided (such as recent stent placement, or post-acute coronary syndrome) [21].

CONCLUSION:

A thorough examination is stressed for patients with specific symptoms like ascites, liver enlargement, and raised jugular venous pressure, particularly for ascites precox, to investigate the possibility of constrictive pericarditis. Signs such as weak cardiac pulsations, fluid presence in serous cavities, pericardial calcium deposits, abnormal ECG results, atypical echocardiogram findings, distinctive changes in hemodynamics, and increased pericardial

thickness observed in CT or MRI scans are crucial for confirming the diagnosis. These meticulous investigations are vital because an accurate diagnosis of constrictive pericarditis may lead to a potentially curative surgical intervention.

REFERENCES:

1. Sheth S, Wang DD, Kasapis C. Current and emerging strategies for the treatment of acute pericarditis: a systematic review. Journal of inflammation research. 2010 Nov 25:135-42.
2. Lazarou E, Tsioufis P, Vlachopoulos C, Tsioufis C, Lazaros G. Acute pericarditis: update. Current Cardiology Reports. 2022 Aug;24(8):905-13.
3. Chiabrando JG, Bonaventura A, Vecchié A, Wohlford GF, Mauro AG, Jordan JH, Grizzard JD, Montecucco F, Berrocal DH, Brucato A, Imazio M. Management of acute and recurrent pericarditis: JACC state-of-the-art review. Journal of the American College of Cardiology. 2020 Jan 7;75(1):76-92.
4. Imazio M, Gaita F, LeWinter M. Evaluation and treatment of pericarditis: a systematic review. Jama. 2015 Oct 13;314(14):1498-506.
5. Kyriakakis CG, Doubell AF, Mayosi BM, Isaacs A, De Vries E. An approach to the patient with suspected pericardial disease: CME. South African Medical Journal. 2016 Feb 1;106(2):151-5.
6. Seidenberg PH, Haynes J. Pericarditis: diagnosis, management, and return to play. Current sports medicine reports. 2006 Mar; 5:74-9.
7. Blanco CC, Parekh JB. Pericarditis. Pediatrics in Review. 2010: 3(2); 83-84.
8. Troughton RW, Asher CR, Klein AL. Pericarditis. The Lancet. 2004 Feb 28;363(9410):717-27.
9. Hall IP. Purulent pericarditis. Postgraduate Medical Journal. 1989 Jul;65(765):444-8.
10. Mehta A, Mehta M, Jain AC. Constrictive pericarditis. Clinical cardiology. 1999 May;22(5):334-44.
11. Shah AB, Kronzon I. Congenital defects of the pericardium: a review. Eur. Heart J Cardiovasc Imaging. 2015 Aug;16(8):821-7.
12. Goodacre S, Locker T, Morris F, Campbell S. How useful are clinical features in the diagnosis of acute, undifferentiated chest pain? Acad Emerg Med. 2002 Mar;9(3):203-8.
13. Richard W. Troughton, Craig R. Asher, Allan L. Klein. Pericarditis. The Lancet. 2004: 363; 717-727.
14. John H. Haley, A. Jamil Tajik, Gordon K. Danielson, Hartzell V. Schaff, Sharon L. Mulvagh, Jae K. Oh. Transient Constrictive Pericarditis: Causes and Natural History. Journal of American College of Cardiology. 2004: 43(2); 271-275.
15. Stefan C. Bertog, Senthil K. Thambidorai, Kapil Parakh, Paul Schoenhagen, Volkan Ozduran, Penny L. Houghtaling, et al, Constrictive Pericarditis: Etiology and Cause-Specific survival after pericardiectomy. Journal of American College of Cardiology. 2004: 43(8); 1445-1452.
16. Massimo Imazio. Pericarditis: pathophysiology, diagnosis and management. Current Infectious Disease Reports. 2011: 13; 308-316.
17. Imazio M, Spodick DH, Brucato A, Trinchero R, Adler Y. Controversial issues in the management of pericardial diseases. Circulation. 2010 Feb 23;121(7):916-28.
18. Mayosi BM, Ntsekhe M, Volmink JA, Commerford PJ. Interventions for treating tuberculous pericarditis. Cochrane Database Syst Rev. 2002;(4):CD000526.
19. Seidenberg PH, Haynes J. Pericarditis: diagnosis, management, and return to play. Curr Sports Med Rep. 2006 Apr;5(2):74-9.
20. Lotrionte M, Biondi-Zoccai G, Imazio M, Castagno D, Moretti C, Abbate A, Agostoni P, Brucato AL, Di Pasquale P, Raatikka M, Sangiorgi G, Laudito A, Sheiban I, Gaita F. International collaborative systematic review of controlled clinical trials on pharmacologic treatments for acute pericarditis and its recurrences. Am Heart J. 2010 Oct;160(4):662-70.
21. Khandaker MH, Schaff HV, Greason KL, Anavekar NS, Espinosa RE, Hayes SN, Nishimura RA, Oh JK. Pericardiectomy vs medical management in patients with relapsing pericarditis. Mayo Clin Proc. 2012 Nov;87(11):1062-70.