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An Overview of the Pathogenesis, Therapeutic Presentation, Evaluation, Diagnosis, and Therapy of Acute Pericarditis

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Abstract

Pericarditis is a frequent condition with several causes that can manifest in both primary and tertiary care settings. It is identified in 5% of emergency room visits for chest discomfort and 0.1% of all hospital admissions. Even if new diagnostic methods have advanced, Radiation therapy, heart surgery, and percutaneous coronary intervention are among the treatments for idiopathic pericarditis. Procedures have grown to be crucial reasons. Pericarditis often resolves on its own and is benign. For straightforward instances, nonsteroidal anti-inflammatory drugs continue to be the standard of care. Integrating modern imaging techniques makes it easier to accurately identify and treat issues such pericardial effusion or constriction. We do a systematic review in this work covers the causes, symptoms, diagnostic testing, and treatment of acute pericarditis. We provide an overview of the most recent research on cutting-edge and novel treatment approaches.

Keywords: Pericarditis; Pericardial disease; Treatment strategies

Introduction to Pathophysiology

Up to 5% of visits to the emergency room for chest pain without myocardial infarction are caused by acute pericarditis, an inflammation of the pericardium that is identified in around 0.1% of hospital admissions [1]. The differential diagnosis is crucial because acute myocardial infarction has many electrocardiographic characteristics with pericarditis, despite the fact that the two illnesses are treated quite differently. The pericardium, which surrounds the whole myocardium and spreads onto the major arteries, is a double-layered fibroserous sac. Each layer has a thickness of around 1-2 mm. About 15–35mL of the pericardial fluid, a serous fluid, are present between these layers [2]. The inner visceral layer and the outside parietal layer of the pericardium are both affected by an inflammatory process that causes pericarditis. Chronic pericardial inflammation that goes misdiagnosed and untreated can develop to constrictive pericarditis and other consequences including thickening and calcification of the pericardial wall. Pericardial effusion, a fluid buildup inside the pericardial cavity caused by acute pericarditis, is possible. 15% of pericarditis patients may have hemodynamic compromise due to poor filling of the intracardiac chambers during diastole or cardiac tamponade with hemodynamic compromise, which is a life-threatening condition if not identified and treated right once [3].

Etiology

On occasion, it might be challenging to pinpoint the cause of acute pericarditis. Up to 85% of cases of acute pericarditis are classified as having idiopathic origin since the cause is uncertain [4,5]. 90% of the time, the aetiology is assumed to be viral or idiopathic in immunocompetent patients whose symptoms may go away in a couple of days, and no additional workup is required [6].

In viral illnesses, inflammation develops as a result of the virus replicating in the pericardium, which causes a cellular reaction that, in turn, causes inflammation. Numerous viral genomic fragments have the ability to cause inflammation even in the absence of viral replication. Additionally, antibodies to these fragments can persist for years in the myopericardium and may contribute to recurrent pericarditis [7]. These instances are more frequently secondary to coxsackie B viruses or echoviruses and frequently preceded by a recent flu-like illness or gastrointestinal symptoms. The practitioner must, however, include tuberculosis (TB) or neoplasia in the differential diagnosis if tamponade or effusions are seen during examination but not with any indications of inflammation (pain, friction rub) [8].

Bacterial pericarditis is uncommon in Westernized countries but is nevertheless often found in the underdeveloped world. If left untreated, it is always deadly. Mortality still exceeds 40% even with therapy because of complications such tamponade, bacterial toxicity/sepsis, or other infectious problems [9]. Purulent pericarditis is more prone to occur when the prevalence of human immunodeficiency virus (HIV) rises. Pericarditis, which affects up to 20% of HIV/AIDS patients, is the most widespread cardiovascular symptom of acquired immunodeficiency syndrome (AIDS) [10]. Additionally conceivable, particularly in the immunocompromised patient, is TB pericarditis. A sub-acute sickness with fever, effusion, and/or tamponade is the typical presentation. With TB pericarditis, death rates might reach 85%. The prevalence of TB pericarditis is minimal in Western nations, but it can approach 70% in sub-Saharan Africa [11]. Pericarditis may also be linked to neoplasms. Mesothelioma is the most prevalent primary malignant process, although primary tumours are quite uncommon. The most frequent primary lesions are lung, breast, melanoma, lymphoma, and/or leukaemia, and metastatic cancers are 40 times more prevalent [12].

A post-myocardial infarction (MI) condition known as Dressler's syndrome appears weeks to months after a MI or cardiac surgery. It is believed that it is caused by an autoimmune response mediated by antibodies caused by different cardiac antigens. Up to 20% of individuals with renal insufficiency may also experience significant pericardial effusions. In this population, there are two main types

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of pericarditis that have been identified: uremic pericarditis, which affects 6%-10% of patients with advanced renal failure before starting dialysis and a blood urea nitrogen level of.60, and dialysis-associated pericarditis, which affects 13% of patients receiving long-term dialysis [13,14].

Therapeutic presentation

Depending on the underlying cause and the rate at which fluid builds up, acute pericarditis can manifest with a range of signs and symptoms. A pleuritic chest discomfort with a retrosternal location that is often worse by lying flat and relieved by sitting up and leaning forward characterises the standard clinical appearance. Pericardial chest pain frequently spreads to the neck, arms, or sometimes the left shoulder, just like myocardial infarction chest pain does. Given that the phrenic nerve innervates the pericardium, pericarditis-related chest discomfort frequently spreads to both trapezius muscle ridges [2,18,19]. In cases of pericarditis brought on by rheumatoid arthritis, tuberculosis, cancer, uremia, or post-radiation, there may be no chest discomfort. The viral prodrome of fever, nonproductive cough, myalgias, and malaise may also be reported by patients.

Symptoms: 2-4 of the following symptoms should be seen for confirmation: Typical chest pain, pericardial friction rub, Electrocardiography changes, worsening pericardial effusion.

During a physical examination, a pericardial rub a high-pitched scratchy or squeaky sound may be audible on precordium auscultation. When the patient is sitting upright and leaning forward, this is best detected at the left sternal boundary with the stethoscope's diaphragm during expiration [5]. It is believed that the visceral and parietal pericardial surfaces of the heart are in contact and produce friction. Traditionally, the three unique elements of the pericardial rub have been attributed to, respectively, atrial contraction, ventricular contraction, and ventricular relaxation. About half of all cases of the rub are triphasic, while the other half are biphasic and the other third of individuals have monophasic cases [20,21]. The practitioner should exercise caution when attempting to differentiate a pleural rub timed with the respiratory cycle from a pleural rub caused by cardiac friction given the wide range of differential diagnoses in patients with chest discomfort, especially when it has a pleuritic component [22]. Both the appearance and the severity of the illness process vary. If cardiac tamponade is present, one can check for pulsus paradoxus, which is defined as a systolic blood pressure decrease of more than 10 mmHg with inspiration, and one can check for Kussmaul's sign, which is defined as an increase in jugular venous pressure with inspiration, if constrictive physiology is present [23]. Beck's triad, another indication of tamponade, consists of jugular venous distention, hypotension, and muffled heart sounds [24].

Evaluation in lab

Some nonspecific laboratory values, particularly those related to inflammation, may help in the diagnosis in addition to the results of the physical examination. Elevated erythrocyte sedimentation rate (ESR)/C-reactive protein (CRP), leukocytosis, and cardiac biomarkers can all help with diagnosis, prognosis, and aetiology [25]. Some laboratory results can be helpful in figuring out the cause. When pericarditis is caused by TB or a cancer, respectively, pericardial fluid adenosine deaminase and carcinoembryonic antigen might be raised [26]. A rheumatoid panel, which includes antinuclear antibodies and rheumatoid factor, may be helpful if the history is indicative of a rheumatologic origin. Furthermore, an HIV test may be beneficial if the prevalence of AIDS and HIV rises [27].

As was previously mentioned, pericarditis can present in a manner that is comparable to a MI. Given that pericarditis frequently contains cardiac biomarkers, it is crucial to distinguish between the two conditions. Due to the risk of developing hemorrhagic pericardial effusion and tamponade in cases of acute pericarditis, anticoagulation and thrombolytic treatment may be harmful.

According to a research from a single hospital, 40 out of 238 pericarditis patients were administered thrombolytics or were sent to the cardiac catheterization lab on an emergency basis. The significance of a correct diagnosis was highlighted by the fact that only 35% (14) of these individuals who had coronary angiography exhibited any evidence of heart illness, all of which was classified as mild to moderate in character [28].

The diagnosis of acute pericarditis can be made with the use of electrocardiography (ECG). Typically, it shows down-sloping PR segment depressions and widespread ST segment elevations (concave up) in around 80% of patients. The superficial myocardial inflammation is what is causing the ECG abnormalities. Any of these symptoms may be present at the time of presentation. ECG alterations go through four phases over the course of hours to weeks.

Stage 1: All leads save V1 and aVR have diffuse, concave-up ST segment elevations, whereas leads II, aVF, and V4-V6 have down-sloping PR segment depressions, but leads V1 and aVR do not.

Stage 2: T waves flatten and ST and PR segments normalise.

Stage 3: Diffuse T wave inversion.

Stage 4: T waves return to baseline and the alterations are resolved [29,30].

The time course of the development of these ECG abnormalities in 50 individuals with acute pericarditis was documented. After just 0.5 days of symptoms, stage 1 was identified, and at first, only PR segment depressions were noticed. Stage 2 began around 1.5 days after the beginning of symptoms and had both ST alterations and PR segment depressions. 9.1 days after presentation marked stage 3, whereas days 10 to 11 marked resolution or stage 4. Due to the rotation of the heart in an elevated volume of pericardial fluid, electrical alternans, which are described as beat-to-beat oscillating QRS axis observed on ECG, might signal a significant pericardial effusion.

Use of imaging in acute pericarditis- Numerous imaging techniques help the practitioner make a precise diagnosis. The chest X-ray will show an enlarged cardiac silhouette if there is more than 250 mL of fluid in the pericardial area.

Diagnostic tools: Several studies have looked at the value of pericardial biopsy or pericardiocentesis as a diagnostic tool. Emergent pericardiocentesis should be done on individuals who have significant effusions that have compromised their hemodynamics. Pericardiocentesis, when performed for diagnostic reasons, however, only produced a precise diagnosis in 6% of instances. Up to 29% of the time, therapeutic pericardiocentesis carried out in the presence of cardiac tamponade can provide a diagnosis. Results from pericardial biopsy are comparable. When performed for diagnostic purposes, the yield is frequently just 5% as opposed to a yield of 54% in situations when biopsy was performed as part of the therapy process and/or in recurring instances. Overall, only approximately 25% of cases have their aetiology identified.

Therapies or Treatment

Due to a lack of controlled trials, the majority of acute pericarditis

therapy is anecdotal and empirical. There is now just one significant published guideline, the 2004-released European Guidelines. As shown above, the great majority of cases are viral or idiopathic in origin, need no special care. However, in the few cases where a particular aetiology can be determined, the therapy should focus on the underlying mechanism.

NSAIDs (Class I according to the ESC recommendations) are the backbone of treatment, especially in low-risk patients, who are immunocompetent individuals with a suspected viral or idiopathic aetiology. Data on the precise dosage and course of NSAID therapy are scant to nonexistent. It should be highlighted that a strong anti-inflammatory dose must be provided, such as 2-4 g of aspirin per day, 1200–1800 mg of ibuprofen per day, and 75-150 mg of indomethacin per day. It would be a logical decision to continue at higher dosages if the patient has underlying heart disease and is currently taking aspirin for primary or secondary prevention. Ibuprofen, if not currently taking aspirin, may be recommended because to its low incidence of adverse effects, positive effect on coronary blood flow, and wide range of dosages. It should be highlighted that a strong anti-inflammatory dose must be provided, such as 2-4 g of aspirin per day, 1200-1800 mg of ibuprofen per day, and 75-150 mg of indomethacin per day. 38 It would be a logical decision to continue at higher dosages if the patient has underlying heart disease and is currently taking aspirin for primary or secondary prevention. Ibuprofen, if not currently taking aspirin, may be recommended because to its low incidence of adverse effects, positive effect on coronary blood flow, and wide range of dosages. The treatment of acute pericarditis may involve the use of indomethacin. Due to its vasoconstrictory action, indomethacin should not be administered in individuals with known or suspected coronary artery disease. A modest experiment found that administering 25 mg of indomethacin four times per day to individuals receiving dialysis had no overall impact on symptoms or the course of the disease in those patients with renal impairment. In a small study, ketorolac, an intravenous formulation of an NSAID, was administered to patients who had pericarditis related to Dressler's syndrome, idiopathic pericarditis, or post cardiotomy. The study showed fast-acting symptom relief, but it made no mention of the pericarditis's natural course.

Corticosteroids, like NSAIDs, have the potential for adverse reactions of their own, such as problems with glycemic control, cushingoid symptoms, or immunosuppression. The necessity for calcium and vitamin D supplements, as well as the requirement for bisphosphonates when on steroids, are frequently ignored. Patients who received large doses of steroids had more adverse symptoms associated with steroids, as well as an intriguingly increased risk of recurrence. This would suggest that low-dose steroids are preferable, but again, the available data are sparse, and bigger studies are urgently needed. Overall, there is a lack of information about intrapericardial agents, necessitating more research. Other immunomodulating medications including methotrexate, cyclosporine, and azathiopine may also be useful, although their usage in pericarditis is exceedingly uncommon and should be customised for each unique patient.

Currently, it seems that if steroids are required, they should be administered at a low dose of 0.2 to 0.5 mg/kg/day (or higher doses if necessary to control symptoms) for 2-4 weeks until CRP resolves, and then a tapering regimen should be started with the addition of an NSAID or colchicine if tolerated (Table 1).

Table 1: Medicine for treating acute pericarditis.

Drug (duration prior to taper)	Starting dose (dose range)
Aspirin (1-2 weeks	750-1000 mg TID (2-4 g/day)
Ibuprofen (1-2 weeks)	600 mg TID (1600–3200 mg)
Indomethacin (1-2 weeks)	50 mg TID
Prednisone (2 weeks)	75-150 mg
	0.2-0.5 mg/kg/day
	1.0-1.5 mg/kg/day
Colchicineb (3 months for acute pericarditis)	0.5 mg BID
	0.5 mg/day if ,70 kg
TID: 3 times per day	

Conclusion

There are several etiologies for pericarditis, which can cause chest discomfort. Our ability to diagnose pericarditis will advance along with our diagnostic skills for diagnosing chest discomfort. For a lot of years, pericarditis therapy remained unchanged. Colchicine in particular, as well as the current trends of decreasing NSAIDs and other anti-inflammatories, are altering how we manage primary pericarditis.

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Conflict of interest:

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References

1. Tingle LE, Molina D, Calvert CW (2007) Acute pericarditis. Am Fam Physician 76: 1509-1514.
2. Männer J, Pérez-Pomares JM, Macías D, Muñoz-Chápuli R (2001) The origin, formation and developmental significance of the epicardium: a review. Cells Tissues Organs 169: 89-103.
3. Goyle KK, Walling AD (2002) Diagnosing pericarditis. Am Fam Physician 66: 1695-1702.
4. Permanyer-Miralda G, Sagrista-Sauleda J, Soler-Soler J (1985) Primary acute pericardial disease: a prospective series of 231 consecutive patients. Am J Cardiol 56: 623-630.
5. Zayas R, Anguita M, Torres F, Gime D, Bergillos F, et al. (1995) Incidence of specific etiology and role of methods for specific etiologic diagnosis of primary acute pericarditis. Am J Cardiol 75: 378-382.
6. Permanyer-Miralda G (2004) Acute pericardial disease: approach to the aetiologic diagnosis. Heart 90: 252-254.
7. Maisch B, Seferović PM, Ristić AD, Erbel R, Rienmüller R, et al. (2004) Guidelines on the diagnosis and management of pericardial diseases executive summary; The Task Force on the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology. Eur Heart J 25: 587-610.
8. Sagristà-Sauleda J, Mercé J, Permanyer-Miralda G, Soler-Soler J (2000) Clinical clues to the causes of large pericardial effusions. Am J Med 109: 95-101.
9. Keersmaekers T, Elshot SR, Sergeant PT (2022) Primary bacterial pericarditis. Acta Cardiol 57:387-389.
10. Barbaro G, Klatt EC (2002) HIV infection and the cardiovascular system. AIDS Rev 4: 93-103.
11. Mayosi BM, Burgess LJ, Doubell AF (2005) Tuberculous pericarditis. Circulation 112: 3608-3616.
12. Mirvis DM, Goldberger AL (2001) Braunwald's heart disease: a textbook of cardiovascular medicine.
13. De La Serna AR, Soldevila JG, Claramunt VM, De Luna AB (1987) Colchicine for recurrent pericarditis. Lancet 2: 1517.
14. Rostand SG, Rutsky EA (1990) Pericarditis in end-stage renal disease. Cardiol Clin 8: 701-707.
15. Darby SC, Cutter DJ, Boerma M, Constine LS, Fajardo LF, et al. (2010) Radiation-related heart disease: current knowledge and future prospects. Int J Radiat Oncol Biol Phys 76: 656-665.
16. Kumar PP (1980) Pericardial injury from mediastinal irradiation. J Natl Med Assoc 72: 591-594.

17. Imazio M, Spodick DH, Brucato A, Trinchero R, Adler Y (2010) Controversial issues in the management of pericardial diseases. Circulation 121: 916-928.

18. Lange RA, Hillis LD (2004) Clinical practice. Acute pericarditis. N Engl J Med 351: 2195-2202.

19. Spodick DH (2003) Acute pericarditis: current concepts and practice. JAMA 289: 1150-1153.

20. Spodick DH (1975) Pericardial rub. Prospective, multiple observer investigation of pericardial friction in 100 patients. Am J Cardiol 35: 357-362.

21. Spodick DH (1979) PR segment depression, pericardial knock, and pericardial rub in pericarditis. Am Heart J 97: 411.

22. Oakley CM (2000) Myocarditis, pericarditis and other pericardial diseases. Heart 84: 449-454.

23. Bilchick KC, Wise RA (2002) Paradoxical physical findings described by Kussmaul: pulsus paradoxus and Kussmaul's sign. Lancet 359: 1940-1942.

24. Sternbach G (1988) Claude Beck: cardiac compression triads. J Emerg Med 6: 417-419.

25. Imazio M, Trinchero R (2017) Triage and management of acute pericarditis. Int J Cardiol 118: 286-294.

26. Koh KK, Kim EJ, Cho CH, Choi MJ, Cho SK, et al. (1994) Adenosine deaminase and carcinoembryonic antigen in pericardial effusion diagnosis, especially in suspected tuberculous pericarditis. Circulation 89: 2728-2735.

27. Ariyarajah V, Spodick DH (2007) Acute pericarditis: diagnostic cues and common electrocardiographic manifestations. Cardiol Rev 15: 24-30.

28. Salisbury AC, Olalla-Gómez C, Rihal CS, Bell MR, Ting HH, et al. (2009) Frequency and predictors of urgent coronary angiography in patients with acute pericarditis. Mayo Clin Proc 84: 11-15.

29. Baljepally R, Spodick DH (1998) PR-segment deviation as the initial electrocardiographic response in acute pericarditis. Am J Cardiol 81: 1505-1506.

30. Ginzton LE, Laks MM (1982) The differential diagnosis of acute pericarditis from the normal variant: new electrocardiographic criteria. Circulation 65: 1004-1009.