Overview of management procedure of acute pericarditis

Ahmed Hamza Mohammedalhadi, Khawlah Khaled M Nafadi, Abdullah bakr a banani, Musab rashid f alanazi, Ahmed Abdulhamid F Alanazi, Zainab Hassan M Albahrani, Mubarak Mansour M Aldossary, Meshal Munahi D Almutairi, Abdulmajeed Mohammad S Alibrahem

Abstract

Acute pericarditis presents challenging features because there are similarities be- tween other conditions and complications that must be recognized and treated, at times, with urgency. Acute pericarditis is a typical disorder in numerous clinical settings, and may be the very first symptom of an underlying systemic disease ·Epidemiologic research studies are lacking, and the specific incidence and occurrence are unidentified. Acute pericarditis is recorded in about 0.1% of hospitalized patients and 5% of patients admitted to the Emergency situation Department for non-acute myocardial infarction chest pain. An autoptic prevalence from 1 to 6% has been reported, and thus acute pericarditis might be regularly subclinical. A clinical triage is feasible on a clinical basis. Patients with pericarditis can be safely managed on an outpatient basis without a thorough diagnostic evaluation unless the patient has high risk features such as temperature N38 °C, a subacute onset, immunodepression, a history of recent trauma, oral anticoagulant therapy, myopericarditis, a large pericardial effusion, and cardiac tamponade. The reported diagnostic yield of extensive laboratory evaluation and pericardiocentesis is low in the absence of cardiac tamponade or suspected purulent, tuberculous, and neoplastic pericarditis. Invasive procedures should be limited mainly to patients in whom therapeutic intervention is necessary. Comprehensive and systematic application of brand-new techniques for pericardial fluid and tissue analyses might be helpful to establish an etiology-based treatment. Pericardioscopy might boost pericardial sampling efficiency, facilitate direct instillation of treatments into the pericardial space. At present these examinations are not commonly offered or practiced, and their application seems recommended for high danger cases refractory to a full trial of standard medical therapy in well-experienced tertiary recommendation centres.

Introduction

Acute pericarditis is a typical disorder in numerous clinical settings, and may be the very first symptom of an underlying systemic disease [1,2] · Epidemiologic research studies are lacking, and the specific incidence and occurrence are unidentified. Acute pericarditis is recorded in about 0.1% of hospitalized patients and 5% of patients admitted to the Emergency situation Department for non-acute myocardial infarction chest pain [2]. An autoptic prevalence from 1 to 6% has been reported, and thus acute pericarditis might be regularly subclinical [3-5].

The diagnosis of acute pericarditis in a young patient with normal chest pain appears simple, determining the etiology may be rather more challenging [1,4-10] .It has been reported that patients must be admitted to hospital to figure out the etiology, observe for problems, and begin treatment [9] .However, in clinical practice, the etiologic search is typically inconclusive, and the majority of cases have a self-limited and benign course after empiric treatment with a non-steroidal anti-inflammatory drug (NSAID). Deciding on the degree of the diagnostic assessment in the private patient requires a cautious evaluation of the threat-benefit ratio of prepared diagnostic and restorative options, also thinking about the prospective impact of a particular diagnosis on the subsequent therapy. Thus, do we have any clinical tools to select patients at high threat of a specific etiology or complications? In order to offer an evidence-based guide for the clinical management of acute pericarditis we did a thorough Medline search with the MeSH terms "pericarditis", "pericardium" including all documents published in English from January 1970 to Might 2016. Documents with new or essential contents are consisted of in the reference list.

· Etiology

Several possible reasons for acute pericarditis can be listed as the pericardium may be involved in a great deal of systemic disorders or may be unhealthy as a separated process [4,5,8-10] .Acute pericarditis might be subdivided into contagious and non-infectious diseases (Table 1). Non-infectious pericarditis primarily includes autoimmune etiologies (pericardial injury syndromes, connective tissue diseases, and autoreactive forms), neoplastic pericarditis, metabolic disorders, and distressing pericarditis. In clinical practice, with a traditional diagnostic method, viral and idiopathic acute pericarditis is discovered in 80 to 90% of cases in immunocompetent patients from

industrialized countries [5,6,10-13] .The commonest particular etiologies reported in published clinical series were respectively: neoplastic pericarditis (4.7 to 7.0%), tuberculosis (3.9 to 4.7%), autoimmune etiologies (1.7 to 10.2%), and purulent pericarditis (0.3 to 1.0%). New methods of testing and analysis of pericardial fluid and tissue may enhance the etiologic classification of the disease and increase the probability of identifying a specific etiology decreasing idiopathic cases [7-9,14,15] .This approach consists of the substantial use of pericardiocentesis and drainage in order to perform the evaluation of pericardial fluid and tissue, consisting of detection of growth markers, fluorescence triggered cell arranging, polymerase chain reaction and immunohistochemistry [7,8] .Versatile pericardioscopy might allow inspection and targeted epicardial or pericardial biopsy [7,14,15] .This method might increase the yield of biopsy and allow the use of autofluorescence strategies for the photodynamic diagnosis in suspected neoplastic pericarditis [15] .In a series of 260 patients evaluated by this method in a tertiary recommendation center [8,9], the following medical diagnoses were discovered: neoplastic pericarditis (35%), autoreactive pericarditis (23%), uremia (6%), bacterial pericarditis (6%, omitting tuberculosis), tuberculous pericarditis (4%), and idiopathic pericarditis (4%). This survey provides a picked sample from a tertiary referral center, the possible reduction of the so-called "idiopathic" cases is apparent.

On the contrary, the etiology of acute pericarditis is completely different in developing countries, with a high prevalence of specific types related to tuberculosis (for example, 70 to 80% of cases in Sub-Saharan Africa, and even up to more than 90% when pericarditis is related to HIV-infection). The incidence of tuberculous pericarditis is increasing in Africa as a result of the human immunodeficiency infection (HIV) epidemic [16]. In Western countries, tuberculous pericarditis might be found among immigrants from areas with a high frequency of tuberculosis, and HIV-contaminated patients.

On this basis, knowledge of the epidemiologic information is vital for the development of a logical management program for the disease. The different etiology of pericarditis recommends that the diagnostic technique needs to be targeted at the private patient and background.

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Table 1: Etiology of acute pericarditis

Infectious pericarditis:
• Viral (most common: Echovirus and Coxsackievirus (usual), Influenza, EBV, CMV, Varicella, Rubella, Mumps, HBV, HCV, HIV)
Bacterial (most common: tuberculous, other bacterial rare may include Pneumo-, Meningo-, Gonococcosis, Haemophilus, Borreliosis, Chlamydia, Treponema pallidum)
Fungal (rare: Candida, Histoplasma)
Parasitary (very rare: Echinococcus, Toxoplasma, Entamoeba histolytica)
Non-infectious pericarditis: **Autoimmune pericarditis:**
Pericardial injury syndromes (postmyocardial infarction syndrome, postpericardiotomy syndrome, post-traumatic pericarditis)
Pericarditis in systemic autoimmune diseases (more common in systemic lupus erythematosus, rheumatoid arthritis, Sjögren syndrome, systemic sclerosis, systemic vasculitides, Behçet syndrome, and familial Mediterranean fever)

· Presentation and diagnosis

Chest pain is the most typical symptom and is typically unexpected and severe in onset, but it differs greatly in intensity, and can be so serious and crushing, that may be mistaken for ischemic chest pain. Common pericardial pain might be referred to the scapular ridge, and has particular worsenings by motivation, coughing, swallowing, and some changes in posture (supine or left lateral decubitus posture), while it is eliminated by others (leaning forward and upright posture).

The pericardial friction rub is considered the pathognomonic particular physical finding of acute pericarditis [17,18] .It has actually been reported from 33 to 85% of cases according to different reports [6,11,12,17-19]. Pericardial rub existence guarantees the diagnosis however its absence does not exclude it. Pericardial rubs are untouched by respiration, and this feature is useful for the differential diagnosis with pleural rubs.

Electrocardiographic (ECG) modifications are common and usually progress through 4 phases [17,18,20,21]: stage I, scattered ST-segment elevation (normally concave up) and PR- section

depression are taped in the very first hours to days; stage II, normalization of the ST and PR segments; phase III, widespread T-wave inversion; and stage IV, normalization of the T waves . An atrial current of injury, shown by elevation of the PR segment in lead aVR and anxiety of the PR segment in other leads [20], mainly inferior leads, V5 and V6 may be tape-recorded as the early and first ECG problem . A common ECG development is taped in approximately 60% of all cases [6], while atypical development is not uncommon and might mimic an acute coronary syndrome particularly in myopericarditis. Therapy might speed up or alter ECG changes progression. Sustained arrhythmias are unusual in the absence of a considerable myocardial participation or concomitant heart disease [22].

Leucocytosis, raised C-reactive protein, and sedimentation rate prevail aspecific findings. Acute pericarditis may be connected with increases in serum biomarkers for myocardial injury, including modest elevations in the MB fraction of creatine kinase (CK-MB) and serum heart troponin I (cTnI) [23-25]. Moderate increases in cTnI typically take place in the lack of elevations in CK-MB [25]. In an unselected population of patients with acute pericarditis this finding has been reported in 32% of cases [25], but this rate may be as

high as 50 to 70% including just hospitalized patients [23,24] .The increase in serum cTnI in acute pericarditis is short-term, typically fixing within 7 to 10 days after discussion, and roughly related to the degree of myocardial inflammation. Associated features consist of younger age, male gender, ST-segment elevation, pericardial effusion at discussion, and recent beginning [23,25] Unlike acute coronary syndromes, elevations of cTnI do not appear to bring a negative prognosis [25]

Transthoracic echocardiography is recommended [9,26,27] in order to spot pericardial effusion, its hemodynamic value, and to look for concomitant heart disease or paracardiac pathology. Echocardiographic findings are usually non-specific. A pericardial effusion has actually been reported in approximately 60% of cases [6]. A typically used classification of pericardial effusion [5,6,9,25] has been reported by Weitzman et al. [28]: a small effusion is an echo-free space(anterior plus posterior) of less than 10 mm, a moderate effusion is an echo-free pericardial space of 10 to 20 mm, and a severe effusion is an echo-free area N20 mm. Pericardial effusion echo-free spaces are determined at the beginning of the QRS complex in diastole .

Chest radiography should be considered mostly to dismiss problems in the lung fields and mediastinum. In the absence of another cardiovascular disease, cardiomegaly suggests a substantial pericardial effusion of more than 250 ml [9].

Clinical examination with auscultation, ECG, echocardiography, regular blood analyses (consisting of inflammation markers and markers of myocardial lesion), and chest X-ray are thought about necessary in all cases (class I recommendation according to 2004 European Society of Cardiology-ESC standards) [9]

Classical diagnostic requirements consist of pericarditic normal chest pain, pericardial friction rub and widespread ST- sector elevation or PR depressions not previously reported [6,11,25,29] The existence of pericardial effusion helps to confirm the diagnosis [12,30], however its lack does not omit it. When at least 2 of these criteria are present (Table 2), a clinical diagnosis of acute pericarditis is made. Because the same viruses that are responsible for acute pericarditis are likewise associated with myocarditis as etiologic agents, it is not unusual to find some degree of myocardial involvement in patients with acute pericarditis. The terms "myopericarditis" and "perimyocarditis" are used interchangeably or to suggest the dominant kind. Cases, when this involvement can be scientifically obvious, but still pericarditis is primary, are reported as myopericarditis; they represent most of clinically appreciated cases compared to perimyocarditis, where the myocardial element is prominent [21,30,31]. In clinical practice the term "myopericarditis" is typically utilized in both senses. Myocardial involvement is commoner in pediatric patients and young people. Viruses are main reasons for either pericarditis or myocarditis, and older patients might be less vulnerable to infection due to gotten resistance; for instance, after the age 30, approximately 94% of people may have antibodies to one or more Coxsackie B virus serotypes [32].

Table 2: Diagnostic criteria for acute pericarditis and myopericarditis in the clinical setting

Acute pericarditis (at least 2 criteria of 4 should be present) a:
1. Typical chest pain
2. Pericardial friction rub
3. Suggestive ECG changes (typically widespread ST-segment elevation)
4. New or worsening pericardial effusion

Myopericarditis:
1. Definite diagnosis of acute pericarditis, PLUS
2. Suggestive symptoms (dyspnea, palpitations, or chest pain) and ECG abnormalities beyond normal variants, not documented previously (ST/T abnormalities, supraventricular or ventricular tachycardia or frequent ectopy, atrioventricular block), OR focal or diffuse depressed LV function of uncertain age by an imaging study

· Triage and etiologic investigation

Acute viral or idiopathic pericarditis typically follows a brief and benign course after empiric treatment with a NSAID [4-6,9,10,13,17] .As a result, it seems not appropriate to carry out a complete diagnostic evaluation in all patients, due to the fact that there are no particular treatments for viral disease [6,10,13,33] .Lots of physicians confess all new cases of acute pericarditis to hospital, and this has actually likewise been recommended [9]. However this might not be essential in all cases. A patient with easy straightforward acute pericarditis can go through initial evaluation in a very same day health center facility or clinic, while follow-up may be accomplished on an outpatient basis [5,6]. There are no absolute clinical functions that will certainly differentiate in between specific rather than idiopathic pericarditis [11,33,34], a critique of the available literature shows that clinical functions to choose patients at high danger of a specific etiology or problems can be found [5,6] These clinical features may include fever N38 ° C [35-37], subacute beginning (symptoms establishing during a duration of numerous days or weeks) [5,34,38], immunodepression [38,39], injury [35,37,38], oral anticoagulant treatment [35,40], myopericarditis (pericarditis with clinical or serologic proof of myocardial participation) [35,41], extreme pericardial effusion (a diastolic echo-free area of more than 20 mm in width) [11,12,34] and cardiac tamponade [4,14,34,42] .They can be thought about as "clinical poor prognostic predictors" since they are more regularly connected with an increased threat of short-term issues or a high probability of a specific disease [6].

A risk stratification can be carried out at discussion on a clinical basis . Patients without clinical poor prognostic predictors might be considered at low threat and designated to outpatient treatment with a NSAID and gastroprotection without a specific etiologic search.

In many of such patients even a substantial diagnostic examination is most likely to lead to unfavorable etiologic conclusions [6,11,12] .Even viral research studies should not be considered, because the yield is low and management is not altered.

Patients with several "clinical bad predictors" could be considered high threat cases to be confessed to health center for tracking and a specific etiologic search ought to be considered. In one report, 46 of 300 successive patients (15%) with acute pericarditis were at increased threat at discussion and were hospitalized [6] .The remaining 254 patients (85%) were considered to be at low threat. Outpatient aspirin therapy worked in 87%, and none of these patients had serious issues at a mean follow-up of 38 months. The thirty- three low risk patients, who were aspirin resistant, were thought about at moderate to high risk and hospitalized for more assessment. The application of this procedure caused particular diagnosis in 60 out of 300 cases (20.0%) in the unselected group (previous literature data were from 14 to 22% of cases) [11,12], but approximately 36 out of 46 moderate--high danger patients (78.3%), revealing the possible value of patients choice for the etiologic search. Hence, another essential feature of high danger is the lack of action to a NSAID after a minimum of 1 week of therapy. In fact failure to a NSAID indicates the possibility of a specific etiology(Table 3). The lack of response to NSAIDs, a constant or frequent course, and cardiac tamponade at presentation were found to be risk aspects for neoplastic etiology of acute pericardial disease [42].

A program for outpatient treatment of low threat cases is probably not just safe and effective however also cost-efficient minimizing hospitalization rates and management expenses. These data might be valuable in lowering expenses connected with pericarditis, since lots of patients are confessed to a health center for initial examination and treatment rather than managed as outpatient [43] .When an etiologic search is performed, the most typical causes to dismiss are: tuberculous pericarditis, neoplastic pericarditis, and autoimmune pericarditis (Table 1).

The yield of invasive procedures, such as pericardiocentesis and pericardial biopsy, has actually been reported as low when just utilized for a diagnostic function. In the "Barcelona experience" [11,13,33,34] the total diagnostic yield of pericardiocentesis was 19%, but was very various when the treatment was carried out with a diagnostic (5%) or a restorative (29%) indicator. Similar results were found also for pericardial biopsy with a total diagnostic yield of 19%, however it was much

lower when biopsy was performed with diagnostic (4%) instead of with therapeutic (54%) indicator.

Most likely this yield might be higher consisting of a comprehensive and systematic application of brand-new techniques of pericardial fluid and biopsy analyses [7,8,14,15] .Technical advances in clinical instrumentation and introduction of pericardioscopy have improved the diagnostic yield, and pericardioscopic assistance improved pericardial tasting effectiveness, furthermore some authors have emphasized the possible diagnostic supremacy of epicardial biopsies guided by flexible percutaneous pericardioscopy in comparison with parietal pericardial biopsy [14,15]. However the genuine diagnostic yield of these brand-new strategies is unknown in clinical practice, and this diagnostic improvement does not seem to affect substantially the subsequent management in low danger cases. Thus sensible indications to pericardiocentesis must include: 1-. cardiac tamponade (class I suggestion according to 2004 European Society of Cardiology-ESC guidelines), 2-. believed purulent, tuberculous, or neoplastic pericarditis, 3-. persistent symptomatic pericardial effusion in spite of a complete trial of medical treatment.

Pericardioscopy and pericardial biopsy are typically not readily available, and the diagnostic benefits of these strategies probably justify their application just in well-experienced tertiary referral centres and for high threat cases refractory to a complete trial of traditional medical therapy.

Table 3:

Clinical poor prognostic predictors in acute pericarditis (see text for details)
1. Fever > 38 °C
2. Subacute onset
3. Immunodepression
4. Trauma
5. Oral anticoagulant therapy
6. Myopericarditis
7. Severe pericardial effusion

· Treatment

Unfortunately there are few data from randomized trials to direct clinicians in the management of pericardial diseases and optimum treatment length is not well established [4,5,10] .In the treatment

of idiopathic or viral pericarditis, NSAIDs are thought about the essential of treatment (class I suggestion in 2004 ESC guidelines). Ibuprofen might be chosen because of its uncommon side effects, favourable influence on coronary artery blood flow, and large dose variety [9] .Relying on the intensity of pericarditis and private medication response, 300 to 800 mg of ibuprofen every 6 to 8 h might be needed and can be continued for days or weeks as required. Alternative protocols consist of aspirin (for instance 800 mg every 6 to 8 h followed by progressive tapering with decrements of 800 mg each week for a treatment duration of three to 4 weeks) [6,25] .Various representatives might be equally effective at comparable anti-inflammatory doses: for example aspirin (1600 to 3200 mg day-to-day), indomethacin (75 to 150 mg everyday), ibuprofen (1200 to 1800 mg day-to-day), and nimesulide (200 mg everyday). Aspirin might be first option in postmyocardial infarction patients [5,10] .NSAID dosage tapering may be prescribed in an effort to lower the subsequent recurrence rate. Gastroprotection must be advised in every case [6,9,44].

Corticosteroids given in the index attack were discovered to be an independent danger element for recurrences (OR 4.30; 95% CI 1.21 to 15.25; p = 0.024) [45,46], since they could promote viral duplication [5,47,48] .Corticosteroids have been reported as an independent danger aspect for further reoccurrences also in patient with the very first reoccurrence (OR 2.89; 95% CI 1.10 to 8.26; p=0.04) [49] and after 2 or more reoccurrences in a recently published multicentre retrospective analysis (OR 6.68; 95% CI 1.65 to 27.02; p = 0.008) [50] .All these information argue against the use of corticosteroids in the index attack but likewise as first choice drug in persistent pericarditis considering that some cases may have a contagious etiology (reactivation of a previous viral infection, a possible chronic infection, or re-infection) [48,51] .Corticosteroids should be considered only in patients with bad basic condition or in regular crisis unresponsive to NSAID, as a last option. Corticosteroid use is regularly associated with significant side effects. When corticosteroid therapy is required, it is very essential to avoid common mistakes such as to utilize too low dosages or to taper the dose too quickly. It is very important to utilize a high dosage (prednisone 1 to 1.5 mg/kg) for 1 month even if remission appears obvious after a couple of days, and to taper the dose extremely gradually (if possible after C-reactive protein normalization), presenting a NSAID such as aspirin (for example 1.6 g/day till prednisone discontinuation) or ibuprofen toward the end of tapering [48,52] .Throughout tapering colchicine might be added [47] .Tapering need to occur over months. Prednisone needs to be reduced less gradually from initial high dosages (1 mg/ kg/day) to

the important limit of 25 mg than really slowly in decrements as little as 1 to 2.5 mg at intervals of 1 to 4 weeks tailored to the single patient. If symptoms recur during tapering, every effort must be done not to increase again or reinstitute corticosteroids, however, whenever possible, to attempt to control the symptoms with NSAIDs [53], if this is not possible, the last dose that reduced the episode should be considered and kept for 2 to 3 weeks before subsequent tapering.

In the little minority who do not respond combination of different agents must be thought about.

Workout restriction should also be advised and is an essential part of the management method. Numerous patients report intensifying of symptoms after physical exertion. It seems a good idea to limit exertion beyond that required to carry out domestic jobs and carry out sedentary work [10,48].

Colchicine has been successfully utilized to deal with and prevent frequent pericarditis after failure of conventional treatment [54] .On the basis of cumulative anecdotal proof, and observational studies on reoccurring pericarditis, colchicine has been recommended also in the treatment of acute pericarditis (class IIa suggestion in ESC guidelines) [9] .The recommended dosage is 2 mg/day for 1-2 days, followed by a maintenance dosage of 1 mg/day (0.5 mg QUOTE). In a just recently published open-label clinical trial, colchicine (upkeep dose of 0.5 mg QUOTE, reduced to 0.5 mg daily in patients b70 kg) as adjunct to standard treatment substantially reduced the subsequent recurrence rate (actuarial rates at 18 months were respectively 10.7% vs 32.3%, p = 0.004; NNT = 5.0) and symptoms persistence at 72 h (respectively 11.7% vs 36.7%; p = 0.003) in 120 patients with a first episode of acute pericarditis [46] .Since of diarrhea; thus lower doses may be equally efficacious however with a possible lower rate of side impacts, Colchicine was discontinued in 5 patients (8.3%). However caution must be suggested in the prescription of colchicine for acute pericarditis. At present this usage is unlabeled. The more powerful evidence originates from a single open-label randomized study [46] .More proof supports using colchicine in reoccurrences after failure of traditional treatment [49,54] but the accurate percentage of responders is not precisely called properly randomized trials are doing not have [48] .Although at low doses (0.5-1.2 mg/day), colchicine has actually been discovered to be safe even when offered continuously over years [54,55], there are other less common (b1%) possible adverse effects to be considered (bone marrow suppression, hepatotoxicity, and myotoxicity) beyond the well-understood gastrointestinal

negative effects [55-57] .Persistent renal in- sufficiency resulting in increased colchicine levels seems the major danger factor for adverse effects and other possible negative interactions [57] .Colchicine undergoes intensive hepatic metabolic process (CYP 3A4). Drugs (cyclosporine, azole antifungals, ciprofloxacin, clarithromycin, diclofenac, doxycycline, erythromycin, isoniazid, nicardipine, propofol, protease inhibitors, quinidine, and verapamil) that communicate with the cytochrome P450 system may interfere with colchicine increasing the levels/effects of colchicine. Colchicine is likewise a substrate of P-glycoprotein, a transporter associated with the elimination of numerous drugs. Macrolides are inhibitors of P-glyco-protein and cytochrome P450-dependent enzymes and may decrease colchicine excretion. Coadministration of colchicine and macrolides may hinder colchicine removal, resulting in possible drug excess and toxicity especially in the senior and/ or renally compromised [57-60].

At present, it seems reasonable to prevent the coadministration of colchicine and macrolides, in addition to making use of the drug in patients with hepatobiliary dysfunction, severe renal, gastrointestinal disorders, and blood dyscrasias. It is also prudent to lower maintenance/prophylactic dose by 50% in people N70 years, and in patients with impaired kidney function with glomerular filtering rates listed below 50 ml/min. The safety profile of the drug seems to be exceptional to corticosteroids and other immunosuppressive drugs [57], every patient ought to undergo a careful tracking of possible negative effects likewise consisting of blood analyses (transaminases, serum creatinine, creatine kinase, and blood cell count) before beginning the drug, and later at least after 1 month of treatment. Further studies are needed to verify the use of colchicine in acute pericarditis [57].

In patients with an identified cause, specific therapy appropriate to the underlying disorder is suggested, consisting of proper antimicrobial treatment for bacterial pericarditis [16,61].

· Prognosis and prevention

Patients with acute idiopathic or viral pericarditis have an excellent long-term diagnosis. Persistent pericarditis is a tough and typical complication [4,5,9,45,54] .The exact recurrence rate is unidentified, but has been reported 15 to 30% [47,48,54] as well as up to 50% [14] NSAIDs are the pillar of therapy, but colchicine provides the best prophylaxis against recurrences and reduces symptoms throughout

the acute attack [46,49,54] .In some circumstances symptoms might only be managed by corticosteroids, however only in very unusual cases it is essential to resort to other immunosuppressive drugs in reoccurring cases [45], and just weak evidence-based information support their use [62] .Regardless of an excellent life prognosis with extremely rare extreme problems, recurrences may trigger a serious problems of the quality of life. Heart tamponade has been reported from 5 to 28% of cases of acute idiopathic pericarditis [6,11,12], and it is more common in patients with a particular etiology, such as neoplastic, tuberculous, or purulent pericarditis (approximately 68%) [11] .Constrictive pericarditis may occur in about 1% of patients with acute idiopathic pericarditis, and it is more typical in patients with a specific etiology [48] .Tuberculosis has been the most typical cause in the past, while heart surgery and radiation therapy have actually become progressively crucial causes. Patients with a specific etiology are at higher threat of problems, and prognosis is connected to the underlying disease. At follow-up, aspirin resistance was connected with considerable increases in the rates of issues [6,42] .At present, a primary avoidance of reoccurrences is possible restricting using corticosteroid treatment either in recurrent or acute pericarditis [45,46,49,50] ·Growing proof indicates that colchicine might work either for the main [46] or the secondary prevention of reoccurrences [49,50,54].

Table 4

Key points in triage and management of acute pericarditis
1.The diagnostic approach should be targeted at the individual patient and epidemiological background.
2.Although pericarditis may be due to several causes, in clinical practice idiopathic and viral acute pericarditis is found in 80 to 90% of cases in immunocompetent patients from developed countries.
3. The diagnosis is based on clinical criteria, laboratory testing and extensive diagnostic evaluation is not routinely necessary.
4. The reported diagnostic yield of extensive laboratory evaluation and pericardiocentesis is low in the absence of cardiac tamponade or suspected purulent, tuberculous, and neoplastic pericarditis.
5. Acute viral or idiopathic pericarditis typically follows a brief and benign course after empiric treatment with a NSAID.
6. Simple uncomplicated acute pericarditis can undergo initial evaluation in a same day hospital facility or clinic, and follow-up may be accomplished on an outpatient basis.

7. NSAIDs are considered the mainstay of therapy. Different agents may be equally efficacious at equivalent anti-inflammatory doses.

Conclusion

Clinical risk stratification of acute pericarditis may be useful to select patients who should be admitted to hospital, and in whom complete assessment ought to be performed to determine causes that need specific therapy **(Table 4)**. At present double-blind randomized trials are doing not have to assist the management of pericarditis, and therapeutic choices require further examinations.

Restriction of corticosteroids might play a major role in minimizing recurrence rate. NSAIDs are a reasonable first choice, with the possible addition of colchicine for recurrences, before steroid therapy is tried. In patients who have been already provided corticosteroids, every effort must be done to terminate the drug and taper presenting a NSAID and/or colchicine.

Comprehensive and systematic application of brand-new techniques for pericardial fluid and tissue analyses might be helpful to establish an etiology-based treatment. Pericardioscopy might boost pericardial sampling efficiency, facilitate direct instillation of treatments into the pericardial space. At present these examinations are not commonly offered or practiced, and their application seems recommended for high danger cases refractory to a full trial of standard medical therapy in well-experienced tertiary recommendation centres.

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