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REVIEW

Current and emerging strategies for the treatment of acute pericarditis: a systematic review

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Abstract: Pericarditis is a common disorder that has multiple causes and presents in various primary-care and secondary-care settings. It is diagnosed in 0.1% of all hospital admissions and in 5% of emergency room visits for chest pain. Despite the advance of new diagnostic techniques, pericarditis is most commonly idiopathic, and radiation therapy, cardiac surgery, and percutaneous procedures have become important causes. Pericarditis is frequently benign and self-limiting. Nonsteroidal anti-inflammatory agents remain the first-line treatment for uncomplicated cases. Integrated use of new imaging methods facilitates accurate detection and management of complications such as pericardial effusion or constriction. In this article, we perform a systematic review on the etiology, clinical presentation, diagnostic evaluation, and management of acute pericarditis. We summarize current evidence on contemporary and emerging treatment strategies.

Keywords: pericarditis, pericardial disease, treatment strategies

Introduction and pathophysiology

Acute pericarditis is due to inflammation of the pericardium and is diagnosed in approximately 0.1% of hospital admissions, accounting for up to 5% of emergency room visits for chest pain without myocardial infarction.[1] In addition, many of the electrocardiographic features seen in pericarditis are also evident in acute myocardial infarction, whereas treatment of the two conditions differs substantially, making the differential diagnosis of paramount importance.

The pericardium is a double-layered fibroserous sac that covers the entire myocardium and extends onto the great vessels. Each layer is approximately 1–2 mm thick. The space between these layers contains approximately 15–35 mL of serous fluid known as pericardial fluid.[2] Pericarditis is due to an inflammatory process affecting the inner visceral layer and the outer parietal layer of the pericardium. Left undiagnosed and untreated, chronic inflammation of the pericardium can result in complications such as pericardial wall thickening and calcification leading to a constrictive pericarditis (Table 1). Acute pericarditis can lead to fluid accumulation within the pericardial space known as pericardial effusion. In 15% of patients with pericarditis, rapid accumulation of fluid into the pericardial space can result in hemodynamic compromise due to impaired filling of intracardiac chambers during diastole and lead to cardiac tamponade with hemodynamic compromise, which is a life-threatening condition if not recognized and treated promptly.[3]

Etiology

The etiology of acute pericarditis is at times difficult to identify. As many as 85% of acute pericarditis cases are of unknown etiology, labeled as idiopathic origin.[4,5] In

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Table 1 Pericarditis classification scheme[3,4]

Type	Duration	Notes
Acute	<6 weeks	Effusive versus fibrinous
Subacute	6 weeks–6 months	Indolent course
Chronic	>6 months	Effusive versus adhesive versus effusive-adhesive versus constrictive
Recurrent	Intermittent	No symptoms for set time period
	Incessant	Recurrence after discontinuation of therapy

immunocompetent patients where symptoms may resolve in a matter of days, 90% of the time the etiology is thought to be viral or idiopathic, and no further workup is needed.[6]

The cause of inflammation in viral illness is due to the replication of the virus in the pericardium which elicits a cellular response, which in turn leads to inflammation. Even without viral replication, there are a number of viral genomic fragments that can also elicit an inflammatory response. Moreover, antibodies to these fragments can be found in the myopericardium for years and may be an etiology of recurrent pericarditis.[7] These cases are often preceded by a recent flu-like illness or gastrointestinal symptoms and more often are secondary to coxsackie B viruses or echoviruses.

However, if tamponade or effusion is present on examination without signs of inflammation (pain, friction rub) the practitioner must consider tuberculosis (TB) or neoplasia in the differential diagnosis.[8] In Westernized nations, bacterial pericarditis is not common, but it is still often seen in the developing world, and if untreated is 100% fatal. Even with treatment, mortality still approaches 40% due to complications such as tamponade, bacterial toxicity/sepsis, or other infectious complications.[9] As the incidence of human immunodeficiency virus (HIV) increases, the incidence of purulent pericarditis will likely also increase. In fact, pericarditis is the most common cardiovascular manifestation of acquired immunodeficiency syndrome (AIDS), occurring in up to 20% of patients with HIV/AIDS.[10] TB pericarditis is also possible, especially in the immunocompromised patient. The classic presentation is a subacute illness with fever, effusion, and or tamponade. The mortality with TB pericarditis is as high as 85%. In developed countries, the incidence is low, but in sub-Saharan Africa, the incidence of TB pericarditis reaches approximately 70%.[11]

Neoplasms may also be associated with pericarditis. While primary tumors are extremely rare, mesothelioma is the most common primary cancerous process. Metastatic tumors are 40 times more likely, with the common primary lesions being lung, breast, melanoma, lymphoma, and/or leukemia.[12]

Dressler's syndrome is a postmyocardial infarction (MI) finding that develops in weeks to months post-MI or cardiac surgery. It is thought to be due to an autoimmune reaction mediated by antibodies due to various myocardial antigens.[1]

Renal failure may also cause large pericardial effusions in up to 20% of patients. Two basic forms of pericarditis have been described in this population; uremic pericarditis, seen in 6%–10% of patients with advanced renal failure prior to dialysis with a blood urea nitrogen level of >60, and dialysis-associated pericarditis, which occurs in 13% of patients on chronic dialysis.[7,13,14]

Other etiologies include rheumatologic processes, hypothyroidism/myxedema, iatrogenic causes, ie, after open heart procedures (valvular operations > coronary artery bypass grafts) as well as electrophysiology procedures, and radiation therapy for thoracic tumors (Table 2).[15,16]

Clinical presentation

Acute pericarditis can present with a variety of signs and symptoms, which vary depending on the underlying etiology and the rapidity with which fluid accumulates (Table 3). The classical clinical presentation is a pleuritic chest pain, typically retrosternal and positional (exacerbated by lying supine and alleviated by sitting up and learning forward). Similar to chest pain associated with myocardial infarction, pericardial chest pain often radiates to the neck, arms, or even the left shoulder. Given that the pericardium is innervated by the phrenic nerve, the chest pain due to pericarditis most typically radiates to both trapezius muscle ridges.[2,18,19] Chest pain may be absent in rheumatoid pericarditis, or pericarditis due to TB, neoplasm, uremia, and post-radiation. Patients may also complain of a viral prodrome of fever, nonproductive cough, myalgias, and malaise.

Physical exam may reveal a high-pitched scratchy or squeaky sound on the auscultation of the precordium, known as a pericardial rub. This is best identified at the left sternal border with the diaphragm of the stethoscope during expiration with the patient sitting upright and leaning forward.[5] It is thought to be caused by friction between the visceral and parietal pericardial surfaces. Classically, the pericardial rub has three distinct components attributed to atrial contraction, ventricular contraction, and ventricular relaxation, respectively. The rub is triphasic in approximately 50% of cases, biphasic in a third of patients, and monophasic in the remaining.[18,20,21]

Table 2 Etiology of pericarditis

Condition	Cause
Infectious pericarditis (2/3 of cases)	Viral (echovirus, coxsackievirus (most common), influenza, EBV, CMV, adenovirus, varicella, rubella, mumps, HBV, HCV, HIV, parvovirus B19, and human herpes virus 6) Bacterial (tuberculous 4%–5%, *Coxiella burnetii*, pneumococcosis, meningococcosis, gonococcosis, hemophilus, staphylococci, chlamydia, mycoplasma, legionella, leptospira, listeria) Fungal (histoplasma [more likely in immunocompetent patients], aspergillosis, blastomycosis, candida [more likely in immunosuppressed host]) Parasitic (echinococcus, toxoplasma)
Noninfectious pericarditis (1/3 of cases)	
Autoimmune pericarditis (10%)	• Pericardial injury syndromes (post myocardial infarction syndrome, postpericardiotomy syndrome, posttraumatic pericarditis including iatrogenic pericarditis from ablations, catheterizations) • Pericarditis in systemic autoimmune and auto-inflammatory diseases (systemic lupus erythematosus, Sjögren syndrome, rheumatoid arthritis, systemic sclerosis, systemic vasculitides, Behçet's syndrome, sarcoidosis, familial Mediterranean fever) • Autoreactive pericarditis
Neoplastic pericarditis (5%–7%)	• Primary tumors (pericardial mesothelioma) • Secondary metastatic tumors (lung and breast cancer, lymphoma) • Metabolic pericarditis (uremia, myxedema)
Traumatic pericarditis	• Direct injury (penetrating thoracic injury, esophageal perforation, iatrogenic) • Indirect injury (nonpenetrating thoracic injury, radiation injury)
Drug-related pericarditis	• Procainamide, hydralazine, isoniazid, phenytoin, penicillins, doxorubicin, and daunorubicin

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Abbreviations: CMV, cytomegalovirus; EBV, Epstein–Barr virus; HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus.

Given the multitude of differential diagnoses in those with chest pain, especially with a pleuritic component, the practitioner should take care in trying to distinguish a myocardial friction rub from a pleural rub which is timed with the respiratory cycle.[22] As the severity of disease process varies, so does the presentation. If cardiac tamponade is present, one can examine for pulsus paradoxus – defined as a decrease of systolic blood pressure by more than 10 mmHg with inspiration; if constrictive physiology is present, one can evaluate for Kussmaul's sign – defined as an increase of the jugular venous pressure with inspiration.[23] Another sign of tamponade, Beck's triad, consists of jugular venous distention, hypotension, and muffled heart sounds.[24]

Laboratory evaluation

Along with physical exam findings, there are some nonspecific laboratory values that may aid in the diagnosis, primarily those involved with inflammation. Presence of a leukocytosis, elevated erythrocyte sedimentation rate (ESR)/C-reactive protein (CRP), and cardiac biomarkers can all aid not only in the diagnosis but prognosis and etiology as well.[25]

There are some lab values that can be useful for determining the etiology. Pericardial fluid adenosine deaminase and carcinoembryonic antigen can be elevated in the case of tuberculosis- and malignancy-related pericarditis, respectively.[26] If the history is suggestive of a rheumatologic etiology, a rheumatoid panel, including antinuclear antibodies and rheumatoid factor, may be useful. Moreover, as the incidence of AIDS/HIV increases, an HIV screen may be of use.[27]

As discussed above, the presentation of pericarditis can be similar to that of a MI. Given that cardiac biomarkers are often present in pericarditis, it is important to distinguish between the two entities. Anticoagulation and thrombolytic therapy may be detrimental in the case of acute pericarditis due to potential conversion to hemorrhagic pericardial effusion and tamponade. A single center study showed that approximately 20% (40 of 238) of patients with pericarditis are taken emergently to the cardiac catheterization lab or given thrombolytics. However, only 35% (14) of these patients who underwent coronary angiography had any evidence of cardiac disease, all of which was labeled as mild to moderate in nature, highlighting the importance of a proper diagnosis.[28]

Electrocardiographic evaluation

Electrocardiography (ECG) is helpful in the diagnosis of acute pericarditis. Classically, it reveals diffuse ST segment elevations (concave up) and down-sloping PR segment depressions in about 80% of patients. The ECG changes are due to superficial myocardial inflammation.[29] ECG changes evolve in four stages over hours to weeks, and any of these manifestations may be present at the time of presentation:

Table 3 Diagnostic criteria[7,17,22,33] (Two of the four should be present)

Typical chest pain
Pericardial friction rub
Suggestive ECG changes
New or worsening pericardial effusion[a]

Note: [a]This is debatable and may be used to confirm diagnosis, but lack of pericardial effusion does not exclude diagnosis.
Abbreviation: ECG, electrocardiography.

a. Stage 1 – diffuse ST segment elevations that are concave up in all leads except V1 and aVR, with down-sloping PR segment depression in most leads but particularly leads II, aVF, and V4–V6, but not in leads V1 and aVR.
b. Stage 2 – ST and PR segments normalize and T waves flatten.
c. Stage 3 – diffuse T wave inversion.
d. Stage 4 – T waves return to baseline, and resolution of the changes.[30,31]

The time frame of the evolution of these ECG changes was described in 50 patients with acute pericarditis. Stage 1 was noted after only 0.5 days of symptoms, and initially only PR segment depressions were seen. Stage 2 occurred approximately 1.5 days from symptoms onset and showed both ST changes as well as PR segment depressions. Stage 3 occurred 9.1 days from presentation, whereas resolution or stage 4 was noted on days 10–11.[29] Electrical alternans, defined as beat-to-beat oscillating QRS axes seen on ECG, can indicate a large pericardial effusion due to rotation of the heart in an increased amount of pericardial fluid.

Application of imaging in acute pericarditis

Various imaging modalities aid the clinician in accurate diagnosis. If there is greater than 250 mL of fluid in the pericardial space, the chest X-ray will demonstrate an enlarged cardiac silhouette.[3]

The 2004 European Society of Cardiology (ESC) guidelines recommend echocardiography if pericarditis is suspected since the presence of a pericardial effusion can aid in the management and diagnosis.[7] Depending on the size of the effusion, small effusions are denoted as <10 mm of fluid, moderate effusions as 10–20 mm of fluid, and severe effusions when >20 mm of fluid is present.[32] While an echo will demonstrate an effusion in approximately 60% of cases, it is not required for diagnosis.[33]

Diagnostic tools

A number of studies have examined the usefulness of pericardial biopsy or pericardiocentesis to aid in the diagnosis. In patients with large effusions resulting in hemodynamic compromise, emergent pericardiocentesis should be performed. However, when done for diagnostic purposes, pericardiocentesis yielded a specific diagnosis in only 6% of cases. Therapeutic pericardiocentesis performed in the setting of cardiac tamponade can yield a diagnosis up to 29% of the time.[4] Pericardial biopsy has similar results. When done for diagnostic reasons, the yield is often only 5% versus a yield of 54% in cases where biopsy was part of the treatment procedure and or in recurrent cases.[4] Overall, the etiology is determined in only about a quarter of patients.

Clinical course

Current European Guidelines suggest that all patients with newly diagnosed acute pericarditis be admitted for observation.[7] However, the decision to admit these patients has been debated in a number of papers by various authors. Some would suggest that patients with fevers >38°C, those with subacute onset, failure of treatment of nonsteroidal anti-inflammatory drugs (NSAIDs) after 1 week, immunosuppression, trauma, on anticoagulation therapy, with known neoplasm, suspected myopericarditis, severe effusion/tamponade, or hemodynamic instability should be considered high risk and admitted.[17,33,34] Risk factors associated with a poor prognosis include female gender (hazard ratio [HR] of 1.65), large effusion/tamponade (HR 2.51), aspirin or NSAID failure in setting of tamponade (HR 5.5).[35] Some may consider elevated troponins as a risk factor for complication, and while in the setting of acute ECG changes it made lead to a cardiac catheterization, overall the prognostic implication is benign. It should be noted that elevated troponins are more likely associated with myopericarditis, which is considered a high risk factor.[36,37]

Treatment

The treatment of acute pericarditis is largely anecdotal and empirical due to a lack of randomized trials. To date only one major published guideline exists, the European Guidelines published in 2004. As discussed above, the vast majority of cases are idiopathic or viral in nature, and no specific treatment is needed. But, in the few instances where a specific etiology can be identified, the treatment should be geared towards the underlying process.

The mainstay of therapy is NSAIDs (Class I per the ESC guidelines) especially in low-risk patients; with low risk being defined as immunocompetent patients with a presumed viral or idiopathic cause. There are limited to no data on the exact dose and treatment course using

NSAIDs. It should be noted that a high anti-inflammatory dose needs to be prescribed; aspirin of 2–4 g/day, ibuprofen 1200–1800 mg/day, indomethacin of 75–150 mg/day.[38] If the patient has underlying heart disease and is already on an aspirin for primary or secondary prevention, it would be a reasonable choice to continue in higher doses. However, if not already on aspirin, ibuprofen may be the preferred agent due to a low rate of side effects, favorable impact on coronary blood flow, as well as a large dosing range.[7,25]

Indomethacin may be used in the management of acute pericarditis. However, indomethacin should not be used in patients with known or suspected coronary artery disease due to its vasoconstrictory effect.[39] In those patients with renal disease, a small trial demonstrated that 25 mg of indomethacin 4 times daily in patients on dialysis had no overall effect on symptoms or natural history of the disease.[40] Ketorolac, an NSAID with an intravenous formulation, was used in a small study in patients with pericarditis associated with Dressler's syndrome, idiopathic pericarditis, or post cardiotomy and demonstrated symptomatic relief with fast onset, but no comment was made on the natural history of pericarditis.[41]

When using NSAIDs, it is important to consider the medications' side effects including but not limited to platelet inhibition, renal effects, as well as gastrointestinal (GI) upset/bleeding. The American College of Gastroenterology has identified a number of risk factors of GI toxicity related to NSAID use: age >60 years, history of a previous adverse event, high dose NSAIDs, concurrent use of glucocorticoids, or use of concurrent anticoagulants.[42] Hence, a number of the authors of various trials including a NSAID treatment arm have recommend GI protection with 20 mg/day of omeprazole.[33] The length of treatment is debatable. Some would recommend following CRP as an indicator for response to treatment as it can represent the level of inflammation. CRP is recommended instead of ESR given lack of confounding factors and faster changes.[38] Full dose anti-inflammatory NSAIDs should be continued for a total of 7–14 days, and then after CRP normalizes, a taper can be started.

The addition of colchicine has been shown to decrease the duration of symptoms as well as the rate of recurrence of acute pericarditis. A dose of 0.5 mg daily has been recommended by the ESC guidelines. Colchicine is already used in a number of other inflammatory diseases such as gout or serositis associated with familial Mediterranean fever.[43] The drug was first used in 1987 for recurrent pericarditis, and since then a number of retrospective studies have been published to examine the effect of colchicine on pericarditis.[13,44–50] On the basis of these studies, the ESC guidelines have recommended colchicine as Class I for recurrent pericarditis and optional, but probably useful in acute pericarditis (Class IIa). The COlichicine for acute PEricarditis (COPE) trial demonstrated that the addition of colchicine at 0.6 mg twice daily for 3 months to standard therapy with aspirin reduced the recurrence rate from 33% in the aspirin-only group to 11% in the aspirin + colchicine group.[48] Furthermore, there was a longer event-free survival in the colchicine group as well as a faster resolution of symptoms. It would appear that colchicine is a useful adjunct therapy to NSAIDs as well as steroids to prevent recurrence of pericarditis. The COlchicine for REcurrent pericarditis (CORE) trial[49] has confirmed the value of colchicine in the treatment of recurrent pericarditis, demonstrating that the addition of colchicine decreased the recurrence rate compared with conventional treatment in patients with a first episode of recurrent pericarditis.[50] Both trials also highlighted the previous use of corticosteroids as an independent risk factor for higher rate of recurrence. There are a number of ongoing clinical trials also examining the effect of the addition of colchicine to conventional treatment in acute pericarditis.[43,51–54] As with NSAIDs, colchicine also has a number of side effects, including dose-related GI side effects in 10%–15% of patients. Renal insufficiency can raise the levels of colchicine; hence lower doses may be indicated in this patient population.[55]

Corticosteroids are another anti-inflammatory agent that can be used in patients who cannot tolerate NSAIDs or colchicine. However, various experts as well as the European guidelines suggest limiting their use in acute pericarditis, mainly due to a strong concern for increasing the rate of recurrence. Yet, these agents are still administered in 60%–90% of patients in most series.[56] While steroids can often provide fast symptomatic relief, they are often not used correctly or tapered appropriately.[57–59] Furthermore, there are data to suggest steroid use is an independent risk factor for recurrent pericarditis.[17,33,35,55,57] This is likely due to the fact that the majority of cases are viral or idiopathic in nature and corticosteroids are immunosuppressive and affect the body's response to viral illness. One study does suggest benefit from high-dose corticosteroids.[60] In this study, 12 patients with relapse of pericarditis after receiving low-dose steroids were given high doses of prednisone 1–1.5 mg/kg/day for 4 weeks followed by a 2-month taper. Of note, once the taper was started, patients were started on a 5-month course of aspirin at 1.6 g/day during the steroid taper and 0.8 g/day after the taper. All but one patient had no relapse. Initially, it would appear that high-dose steroids show benefit; however, all the patients were on NSAIDs (a Class 1 indication per ESC

guidelines), so it would be difficult to determine the exact effect of the steroids. Hence, it is reasonable to use steroids in patients with rheumatologic causes of pericarditis or, as mentioned previously, to those with NSAID intolerance or failure.[38,57]

There has been one study to specifically look at high- versus low-dose steroids. In this group, 100 patients with recurrent pericarditis were treated with low-dose prednisone 0.2–0.5 mg/kg/day versus 1 mg/kg/day. Each dose was continued for 4 weeks and then tapered. Patients treated with high dose steroids showed a higher rate of steroid related side effects, as well as, interestingly, a higher rate of relapse.[61] This would lead one to believe that low-dose steroids are better, but again the data are limited and there is a specific need for larger trials. Currently it would appear that if steroids are needed, a low-dose regimen of 0.2–0.5 mg/kg/day (or high doses if required to control symptoms) be used for 2–4 weeks until CRP resolves, and then to begin a taper with the addition of an NSAID or colchicine if tolerated (Table 4).[62]

As with NSAIDs, corticosteroids carry their own adverse reaction potential, including issues with glycemic control, cushingoid effects, or immunosuppression. Often overlooked is the need for supplementation of vitamin D and calcium or need for bisphosphonates while on steroids.[17]

Due to its lack of systemic side effects, intrapericardial steroids, triamcinolone in particular, has shown some promise in the treatment of acute pericarditis. Per the ESC 2004 guidelines, intracardiac steroids are a Class IIa indication with B level of evidence. In one study designed to examine the efficacy of intrapericardiac steroids in those with autoreactive effusions, 260 patients underwent extensive workup for pericarditis. Of these 260 patients, 84 underwent intrapericardial instillation of triamcinolone. These patients were divided into two groups; 50 received 600 mg/m^2/24 h, and the other received 300 mg/m^2/24 h. Intrapericardial administration of triamcinolone resulted in symptomatic improvement and prevented effusion recurrence in 92.6% of group 1 (600 mg) versus 86.7% in groups 2 (300 mg) after 3 months, and 86% in group 1 versus 82% in group 2 at 1 year. Moreover, there were no documented treatment-related complications, although the group receiving the higher dose did have a higher rate of transient Cushing's syndrome.[63] In a second study from a registry of 136 patients undergoing pericardiocentesis, 29 patients were selected, 14 with autoimmune pericarditis and 15 with neoplastic effusions as treatment arms. Of the 14 patients with autoimmune pericarditis, 1 g of crystalloid triamcinolone was given and prevented recurrence at 3 months in 13 of the 14 cases, and 12 of the 14 at 1 year. In patients with cancer, 50 mg of cisplatin was used and prevented recurrence in all 15 patients at 3 months and 14 of 15 patients at 6–12 months. Of note, mortality was high in this case series; 47% at 3 months and 80% at 6 months, but it was due to noncardiac tumor progression.[64] There are some data suggesting that intrapericardial cisplatin may be more effective in patients with secondary lung cancer and that thiotepa may be more effective in patients with breast cancer.[7,55] Tetracycline has been used as a sclerosing agent and to prevent fluid reaccumulation in the setting of malignancy. However, there is a high rate of side effects despite an 85% rate of success.[7] Overall, there are limited data on intrapericardial agents and further trials are needed.

While there may be a role for other immunomodulating agents such as methotrexate, cyclosporine, and azathiopine, their use in pericarditis is extremely rare and should be tailored to the rare individualized patient.

The role of pericardiectomy, pericardial window, and other interventional techniques is reserved primarily for those with resistant recurrent cases.[7,17] Limited data exist

Table 4 Medical therapy for acute pericarditis

Drug (duration prior to taper)	Starting dose (dose range)	Tapering every 1–2 weeks after symptom resolution
Aspirin (1–2 weeks)	750–1000 mg TID (2–4 g/day)	750–1000 mg BID then 750–1000 mg/day
Ibuprofen (1–2 weeks)	600 mg TID (1600–3200 mg)	600 mg BID or 400 mg BID then 600 mg qday
Indomethacin (1–2 weeks)	50 mg TID	75–150 mg/day
Prednisone (2 weeks)	75–150 mg	Reduce total dose by 25 mg/day/week
	0.2–0.5 mg/kg/day	If >50 mg, reduce by 10 mg/day every 1–2 weeks
	1.0–1.5 mg/kg/day[a]	If 50–25 mg, reduce 5–10 mg every 1–2 weeks
		If 25–15 mg, reduce 2.5 mg/day every 2–4 weeks
		If <15 mg, reduce 1.0–2.5 mg/day every 2–6 weeks
Colchicine[b] (3 months for acute pericarditis)	0.5 mg BID	Optional for acute cases, consider 2–4 weeks tapering
	0.5 mg/day if <70 kg	

Notes: Doses are all estimated for anti-inflammatory effect. Limited data on tapering and schedule may be changed on an individual basis. [a]High dose versus low dose; likely benefit from low dose with or without adjuvant therapy; [b]Colchicine is used as adjuvant therapy; no data for primary use exists yet. Adapted with permission from Imazio M, Spodick DH, Brucato A, Trinchero R, Adler Y. Controversial issues in the management of pericardial diseases. *Circulation*. 2010;121(7):916–928.

Abbreviations: BID, 2 times per day; TID, 3 times per day; qday; every day.

for pericardiectomy, and actual efficacy is questioned. The current indications should be determined by expert opinion or in those patients presenting with constrictive pericarditis (unless newly diagnosed and hemodynamically stable).

Conclusion

Pericarditis is a cause of chest pain with various etiologies. As our diagnostic abilities of chest pain improve, so too will our diagnosis of pericarditis. The treatment of pericarditis has not changed for a number of years. The current trends of tapering NSAIDs, and various other anti-inflammatories, primarily colchicine, are changing the way we approach treatment of primary pericarditis.

Disclosure

The authors report no conflicts of interest in this work.

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