INTRODUCTION — The pericardium is a fibroelastic sac made up of visceral and parietal layers separated by a (potential) space, the pericardial cavity. In healthy individuals, the pericardial cavity contains 15 to 50 mL of an ultrafiltrate of plasma. Pericardial diseases are relatively common in clinical practice and may have different presentations either as isolated disease or as a manifestation of a systemic disorder. Although the etiology is varied and complex, the pericardium has a relatively non-specific response to these different causes with inflammation of the pericardial layers and possible increased production of pericardial fluid. Chronic inflammation with fibrosis and calcification can lead to a rigid, usually thickened and calcified pericardium, with possible progression to pericardial constriction.

Diseases of the pericardium present clinically in one of several ways [1]:

- Acute and recurrent pericarditis
- Pericardial effusion without major hemodynamic compromise
- Cardiac tamponade
- Constrictive pericarditis
- Effusive-constrictive pericarditis

Acute pericarditis refers to inflammation of the pericardial sac. The term myopericarditis, or perimyocarditis, is used for cases of acute pericarditis that also demonstrate myocardial inflammation. The clinical presentation and diagnostic evaluation for acute pericarditis will be reviewed here. The etiology of pericarditis, treatment and prognosis of acute pericarditis, and other pericardial disease processes are discussed separately. (See "Etiology of pericardial disease" and "Treatment of acute pericarditis" and "Recurrent pericarditis" and "Myopericarditis" and "Cardiac tamponade" and "Constrictive pericarditis" and "Diagnosis and treatment of pericardial effusion".)

EPIDEMIOLOGY — Acute pericarditis is the most common disorder involving the pericardium. Epidemiologic studies are lacking, and the exact incidence and prevalence of acute pericarditis are unknown. However, acute pericarditis is recorded in about 0.1 to 0.2 percent of hospitalized patients and 5 percent of patients admitted to the Emergency Department for non-ischemic chest pain [2,3].

- In an observational study from an urban area in Northern Italy the incidence of acute pericarditis was 27.7 cases per 100,000 persons per year [4].
- In an observational study from Finland that included 670,409 cardiovascular admissions to 29 hospitals across the country over a 9.5-year period, the standardized incidence rate for pericarditis requiring hospitalization was 3.3 cases per 100,000 person-years [3].

Acute pericarditis is a common disorder in several clinical settings, where it may be the first manifestation of an underlying systemic disease or may represent an isolated process (table 1). In developed countries, most cases of acute pericarditis are considered of possible or confirmed viral origin, although the exact etiology of most cases remains undetermined following a traditional diagnostic approach [5-7].

Prior to the widespread availability of antiretroviral therapy to treat infection with the human immunodeficiency virus (HIV), pericardial disease was the most frequent cardiovascular manifestation of the acquired immune deficiency
syndrome (AIDS) [8,9]. However, in developed countries with access to HIV therapy, patients with HIV infection who develop acute pericarditis have an etiologic spectrum very similar to non-HIV infected patients. On the contrary, HIV infection and tuberculosis persist as major causes of acute pericarditis in developing countries. (See "Cardiac and vascular disease in HIV-infected patients", section on 'Pericardial disease'.)

CLINICAL FEATURES — Acute pericarditis can present in a variety of ways, depending on the underlying etiology. Patients with an infectious etiology may present with signs and symptoms of systemic infection such as fever and leukocytosis. Viral etiologies in particular may be preceded by "flu-like" respiratory or gastrointestinal symptoms. Patients with a known autoimmune disorder or malignancy may present with signs or symptoms specific to their underlying disorder.

The major clinical manifestations of acute pericarditis include [5]:

- Chest pain – typically sharp and pleuritic, improved by sitting up and leaning forward
- Pericardial friction rub – a superficial scratchy or squeaking sound best heard with the diaphragm of the stethoscope over the left sternal border
- Electrocardiogram (ECG) changes – new widespread ST elevation or PR depression
- Pericardial effusion

Chest pain — The vast majority of patients with acute pericarditis present with chest pain (>95% of cases) [10]. Chest pain is likely to be present in cases of acute pericarditis caused by infection, but may be minimal or absent in patients with uremic pericarditis or pericarditis associated with a rheumatologic disorder (although in some patients pleuritic chest pain and pericarditis is the initial presentation of systemic lupus erythematosus).

Chest pain that results from acute pericarditis is typically fairly sudden in onset and occurs over the anterior chest. Unlike pain from myocardial ischemia, chest pain due to pericarditis is most often sharp and pleuritic in nature, with exacerbation by inspiration or coughing. One of the most distinctive features is the tendency for a decrease in intensity when the patient sits up and leans forward [5,11]. This position (seated, leaning forward) tends to reduce pressure on the parietal pericardium, particularly with inspiration, and may also allow for splinting of the diaphragm [12].

However, dull, oppressive pain or radiation of the pain to the shoulders (particularly the trapezius ridges) may occur; in such cases it is difficult to distinguish pericarditis from other causes of chest pain [5,11]. The chest pain of pericarditis must always be distinguished from other common and/or life-threatening causes of chest pain such as myocardial ischemia, pulmonary embolism, aortic dissection, gastroesophageal reflux disease, and musculoskeletal pain. (See "Differential diagnosis of chest pain in adults".)

Pericardial friction rub — The presence of a pericardial friction rub on physical examination is highly specific for acute pericarditis (movie 1). Pericardial friction rubs, which occur during the maximal movement of the heart within its pericardial sac, are said to be generated by friction between the two inflamed layers of the pericardium. However, this commonly offered explanation for its mechanism may be an oversimplification as patients with a pericardial effusion may also have an audible friction rub.

The classic friction rub consists of three phases, corresponding to movement of the heart during atrial systole (which is not heard in patients with atrial fibrillation), ventricular systole, and the rapid filling phase of early ventricular diastole. However, some rubs are present only during one (one component) or two phases (two components) of the cardiac cycle [13]. In a review of auscultation and phonocardiography in 100 patients with a pericardial rub, the rub was triphasic in 56 percent of patients in sinus rhythm; overall, biphasic rubs were present in 33 percent and monophasic rubs in 15 percent [13].

Pericardial rubs have a superficial scratchy or squeaking quality that is best heard with the diaphragm of the stethoscope. They may be localized or widespread, but are usually loudest over the left sternal border [13]. The intensity of the rub frequently increases after application of firm pressure with the diaphragm, during suspended respiration, and with the patient leaning forward or resting on elbows and knees (picture 1). This last maneuver is designed to increase contact between visceral and parietal pericardium, but is seldom used in practice since it is cumbersome for the patient.

Friction rubs tend to vary in intensity and can come and go over a period of hours; therefore, the sensitivity for detection of a rub is variable and depends in large part on the frequency of auscultation [11]. Pericardial rubs may be easier to hear in patients without a pericardial effusion, but this finding is not universal and is not well-
documented. In a report of 100 patients with acute pericarditis, a pericardial rub was present in 34 of 40 (85 percent) without an effusion [14]. This prevalence is considerably higher than the 35 percent incidence of friction rubs reported in another series [10].

Suspension of respiration during auscultation permits distinction of a pericardial friction rub from a pleuropericardial or pleural rub. A pleuropericardial rub results from the friction between the inflamed pleura and the parietal pericardium, while a pleural rub is the result of friction between the inflamed visceral and parietal pleura. As such, pleuropericardial and pleural rubs can be heard only during the inspiratory phase of respiration. (See "Auscultation of heart sounds", section on 'Pericardial friction rub and other adventitious sounds'.)

**Electrocardiogram** — Changes in the electrocardiogram (ECG) in patients with acute pericarditis signify inflammation of the epicardium, since the parietal pericardium itself is electrically inert. However, some causes of pericarditis do not result in significant inflammation of the epicardium and, as such, may not alter the ECG. An illustration of this is uremic pericarditis, in which there is prominent fibrin deposition but little or no epicardial inflammation. As a result, the ECG often shows none of the changes associated with pericarditis [15]. (See "Pericarditis in renal failure").

The electrocardiogram (ECG) in acute pericarditis can evolve through as many as four stages of changes [5,11]. However, pericarditis does not always result in significant ECG changes. One series of 300 consecutive patients with acute pericarditis noted typical ECG evolution in 60 percent of cases [10].

The typical progression of ECG changes in patients with acute pericarditis is described below:

● Stage 1, seen in the first hours to days, is characterized by diffuse ST elevation (typically concave up) with reciprocal ST depression in leads aVR and V1 (waveform 1). There is also an atrial current of injury, reflected by elevation of the PR segment in lead aVR and depression of the PR segment in other limb leads and in the left chest leads, primarily V5 and V6. Thus, the PR and ST segments typically change in opposite directions. PR segment deviation, which is highly specific though less sensitive, is frequently overlooked.

The TP segment is recommended as the baseline for comparison when measuring both PR and ST segment changes in acute pericarditis [16].

● Stage 2, typically seen in the first week, is characterized by normalization of the ST and PR segments.

● Stage 3 is characterized by the development of diffuse T wave inversions, generally after the ST segments have become isoelectric. However, this stage is not seen in some patients.

● Stage 4 is represented by normalization of the ECG or indefinite persistence of T wave inversions ("chronic" pericarditis).

The temporal evolution of ECG changes with acute pericarditis is highly variable from one patient to another [16]. Treatment can accelerate or alter ECG progression. The duration of the ECG changes in pericarditis also depends upon its cause and the extent of the associated myocardial damage [17].

Atypical ECG changes are seen in up to 40 percent of patients with acute pericarditis [10]. For example, localized ST-elevation and T-wave inversion occur before ST-segment normalization in a minority of patients with acute pericarditis without myocardial involvement. These changes can simulate ECG changes seen in patients with an acute coronary syndrome. (See 'ECG differentiation from acute myocardial infarction' below and "ECG tutorial: Myocardial ischemia and infarction" and "ECG tutorial: ST and T wave changes").

Sustained arrhythmias are uncommon in acute pericarditis, except in the post-thoracotomy setting. This was illustrated in a study of 100 consecutive patients in which only seven arrhythmias were identified; all were atrial and all occurred in patients with underlying heart disease [18]. In a separate report comparing patients with myopericarditis and simple acute pericarditis, cardiac arrhythmias were also more commonly present in patients with myopericarditis (odds ratio 17.6, 95% confidence interval 5.7 to 54.1) [4]. Thus, the presence of atrial or ventricular arrhythmias is suggestive of concomitant myocarditis or an unrelated cardiac disease.

**ECG differentiation from acute myocardial infarction** — While both acute pericarditis and acute myocardial infarction can present with chest pain and elevations in cardiac biomarkers, the electrocardiographic changes in acute pericarditis differ from those in acute ST elevation MI (STEMI) in several ways [19]. These distinctions assume that the pericarditis does not occur during or soon after an acute MI. (See "Electrocardiogram in the..."
Morphology – The ST segment elevation in acute pericarditis begins at the J point, which represents the junction between the end of the QRS complex (termination of depolarization) and the beginning of the ST segment (onset of ventricular repolarization). The ST segment elevation rarely exceeds 5 mm, and usually retains its normal concavity (waveform 1). In some cases of acute pericarditis, the ST segment rises obliquely in a straight line. Although similar patterns can occur with STEMI, the typical finding in a STEMI patient is convex (dome-shaped) ST elevation (a pattern not characteristic of acute pericarditis) that may be more than 5 mm in height (waveform 2). The basis for these morphologic differences is not known, but is probably related to the greater injury current associated with infarction.

Distribution – ST segment elevations in STEMI are characteristically limited to anatomical groupings of leads that correspond to the localized vascular area of the infarct (anteroseptal and anterior leads V1 to V4; lateral leads I, aVL, V5, V6; inferior leads II, III, aVF) (waveform 2). The pericardium envelops the heart, therefore the ST changes are more generalized and typically are present in most leads (waveform 1). In pericarditis, ST segment elevation in the precordial leads is most commonly seen in V5 and V6, and in decreasing frequency from V4 to V1, while in the limb leads, it is often more evident in leads I and II than in leads III, aVF, and aVL [17].

Reciprocal changes – Acute STEMI is often associated with reciprocal ST segment changes, which are not seen with pericarditis except in leads aVR and V1.

Concurrent ST and T wave changes – ST segment elevation and T wave inversions do not generally occur simultaneously in pericarditis, while they commonly coexist in acute STEMI (waveform 2). Furthermore, the evolution of repolarization abnormalities often takes place more slowly and more asynchronously among affected leads in pericarditis than in STEMI.

Hyperacute T waves – Peaked T waves (>10mm high in precordial leads, >5 mm high in limb leads), also referred to as hyperacute T waves, can be seen in STEMI but are not typical of pericarditis (waveform 3A-B). Rarely, fusion of the ST segment and T wave into a single monophasic wave in pericarditis can mimic the appearance of hyperacute T waves.

Q waves – Pathologic Q waves, which may occur with extensive injury in STEMI, are generally not seen in pericarditis. The abnormal Q waves in MI reflect the loss of positive depolarization voltages because of transmural myocardial necrosis. Pericarditis, on the other hand, generally causes only superficial inflammation. Abnormal Q waves are not seen unless there is concomitant myocarditis or preexisting cardiomyopathy or myocardial infarction.

PR segment – PR elevation in aVR with PR depression in other leads due to a concomitant atrial current of injury is frequently seen in acute pericarditis but rarely seen in acute STEMI.

QT prolongation – Prolongation of the QT interval with regional T wave inversion (in the absence of drug effects or relevant metabolic disorders) favors the diagnosis of myocardial ischemia (or myopericarditis) over pericarditis alone.

ECG differentiation from early repolarization — The early repolarization variant seen on an ECG may be present in as many as 30 percent of young adults and is often confused with acute pericarditis [20]. Early repolarization is characterized by ST elevation of the J point, which represents the junction between the end of the QRS complex (termination of depolarization) and the beginning of the ST segment (onset of ventricular repolarization). As a result, there is elevation of the ST segment itself, which maintains its normal configuration (waveform 4). In early repolarization, ST elevation is most often present in the anterior and lateral chest leads (V3-V6), although other leads can be involved. (See "ECG tutorial: Miscellaneous diagnoses", section on ‘Early repolarization’.)

The following electrocardiographic features can be helpful in distinguishing acute pericarditis from early repolarization:

- ST elevations occur in both the limb and precordial leads in most cases of acute pericarditis (47 of 48 in one study), whereas about one-half of subjects with early repolarization have no ST deviations in the limb leads [21].
- PR deviation and evolution of the ST and T changes strongly favor pericarditis, as neither is seen in early repolarization.
If the ratio of ST elevation to T wave amplitude in lead V6 exceeds 0.24, acute pericarditis is present (positive and negative predictive values are both 100 percent) [22].

Laboratory and imaging findings

Echocardiogram — Echocardiography is often normal in patients with the clinical syndrome of acute pericarditis unless there is an associated pericardial effusion. While the finding of a pericardial effusion in a patient with known or suspected pericarditis supports the diagnosis, the absence of a pericardial effusion or other echocardiographic abnormalities does not exclude it. In one series of 300 consecutive patients with acute pericarditis, pericardial effusion was present in 180 patients (60 percent). In most cases the effusion was small or moderate in size (79 and 10 percent, respectively) without hemodynamic consequences. Cardiac tamponade was present in only 5 percent of patients [10]. (See “Echocardiographic evaluation of the pericardium” and “Diagnosis and treatment of pericardial effusion”.)

Chest x-ray — Chest radiography is typically normal in patients with acute pericarditis. Although patients with a substantial pericardial effusion may exhibit an enlarged cardiac silhouette with clear lung fields (image 1), this finding is uncommon in acute pericarditis since at least 200 mL of pericardial fluid must accumulate before the cardiac silhouette enlarges [2,5]. However, acute pericarditis should be considered in the evaluation of a patient with new and otherwise unexplained cardiomegaly.

Cardiac biomarkers — Acute pericarditis may be associated with increases in serum biomarkers of myocardial injury such as cardiac troponin I or T. In one series of 118 consecutive cases with idiopathic acute pericarditis an elevated level of cardiac troponin I was detected in 38 patients (32 percent) [23]. Such patients should be considered to have myopericarditis. (See ‘Myopericarditis’ below and ”Myopericarditis”, section on ‘Laboratory studies’.)

Signs of inflammation — Since pericarditis is an inflammatory disease, laboratory signs of inflammation are common in patients with acute pericarditis. These include elevations in the white blood cell count, erythrocyte sedimentation rate, and serum C-reactive protein concentration. While elevation in these markers supports the diagnosis, they are neither sensitive nor specific for acute pericarditis. Additionally, in the hyperacute phase of pericarditis, these markers may remain normal and increased levels may be found only on follow-up.

DIAGNOSIS — The diagnosis of acute pericarditis is usually suspected based on a history of characteristic pleuritic chest pain, and confirmed if a pericardial friction rub is present. Pericarditis should also be suspected in a patient with persistent fever and pericardial effusion or new unexplained cardiomegaly. Additional testing, which typically includes blood work, chest radiography, electrocardiography, and echocardiography, can support the diagnosis but is frequently normal or unrevealing. The electrocardiogram is usually the most helpful test in the evaluation of patients with suspected acute pericarditis. Echocardiography is often normal, but can be an essential part of the evaluation if there is evidence of an associated pericardial effusion and/or signs of cardiac tamponade.

Evaluation — For a patient who presents with suspected acute pericarditis, it is our practice to perform the following studies:

- Initial history and physical examination — This evaluation should consider disorders that are known to involve the pericardium, such as prior malignancy, autoimmune disorders, uremia, recent myocardial infarction, and prior cardiac surgery. The examination should pay particular attention to auscultation for a pericardial friction rub and the signs associated with tamponade. (See “Etiology of pericardial disease” and “Pericardial disease associated with malignancy” and “Non-coronary cardiac manifestations of systemic lupus erythematosus in adults”, section on ‘Pericardial disease’ and “Pericarditis in renal failure” and “Pericardial complications of myocardial infarction” and ”Cardiac tamponade”.)
- Initial testing should include:
  - An electrocardiogram in all cases. (See ”Electrocardiogram” above.)
  - Chest radiography in all cases. (See ”Chest x-ray” above.)
  - Complete blood count, troponin level, erythrocyte sedimentation rate, and serum C-reactive protein level. (See ”Cardiac biomarkers” above.)
  - Blood cultures if fever higher than 38°C (100.4°F) or signs of sepsis.
  - Echocardiography should be performed in all cases, with urgent echocardiography if cardiac tamponade is suspected. Even a small effusion can be helpful in confirming the diagnosis of pericarditis,
The absence of an effusion does not exclude the diagnosis [24]. In addition, echocardiography can be particularly helpful if purulent pericarditis is suspected, if there is concern about myocarditis, or if there is radiographic evidence of cardiomegaly, particularly if this is a new finding. (See "Echocardiogram" above and "Echocardiographic evaluation of the pericardium".)

The 2003 American College of Cardiology/American Heart Association/American Society of Echocardiography (ACC/AHA/ASE) guidelines for the clinical application of echocardiography stated that evidence and/or general agreement supported the use of echocardiography for the evaluation of all patients with suspected pericardial disease [25]. Similarly, a 2013 expert consensus statement from the ASE recommends echocardiography for all patients with acute pericarditis [24].

Additional testing may include:

- **Tuberculin skin test** or an interferon-gamma release assay (eg, QuantiFERON TB assay) if not recently performed. The interferon-gamma release assay is most helpful in immunocompromised or HIV positive patients and in regions where tuberculosis is endemic. (See "Diagnosis of pulmonary tuberculosis in HIV-negative patients" and "Tuberculous pericarditis".)

- **Antinuclear antibody (ANA) titer** in selected cases (eg, young women, especially those in whom the history suggests a rheumatologic disorder). Rarely, acute pericarditis is the initial presentation of systemic lupus erythematosus (SLE). It is important to recognize that a positive ANA is a non-specific test. A rheumatology consult should be sought in patients with pericarditis in whom a diagnosis of SLE is being entertained.

- **HIV serology** (see "Cardiac and vascular disease in HIV-infected patients", section on 'Pericardial disease')

- **Computed tomography (CT)** may be useful to confirm the diagnosis and especially evaluate concomitant pleuropulmonary diseases and lymphadenopathies, thus suggesting a possible etiology of pericarditis (ie, TB, lung cancer) [24]. Noncalcified pericardial thickening with pericardial effusion is suggestive of acute pericarditis. Moreover, with the administration of iodinated contrast media, enhancement of the thickened visceral and parietal surfaces of the pericardial sac confirms the presence of active inflammation. Computed tomographic attenuation values can help in the differentiation of exudative fluid (20 to 60 Hounsfield units), as found with purulent pericarditis, and simple transudative fluid (<10 Hounsfield units).

- **Cardiac magnetic resonance imaging** may be performed if the echocardiogram is unrevealing but the diagnosis of acute pericarditis is suspected, especially in patients with ongoing fever, poor response to treatment, or suspicion of hemodynamic compromise [24].

- **Multimodality imaging** is an integral part of modern management for pericarditis and pericardial diseases. Among multimodality imaging tests, echocardiography is most often the first-line test, followed by CMR and/or CT [23].

- **We do not** routinely obtain viral studies, since the yield is low and management is not altered [26].

- **Pericardiocentesis** should be performed for therapeutic purposes in patients with cardiac tamponade. (See "Pericardiocentesis" below and "Treatment of acute pericarditis", section on 'Interventional therapeutic techniques'.)

- **Pericardiocentesis** should be considered for diagnostic purposes in patients suspected of having a malignant or bacterial etiology, or in patients with an effusion refractory to medical therapy. (See "Pericardiocentesis" below.)

**Clinical diagnostic criteria** — Acute pericarditis refers to inflammation of the pericardial sac. The term myopericarditis, or perimyocarditis, is used for cases of acute pericarditis that also demonstrate features consistent with myocardial inflammation.

Because the same viruses that are responsible for acute pericarditis can also cause myocarditis, it is not uncommon to find some degree of myocardial involvement in patients with acute pericarditis. The terms "myopericarditis" and "perimyocarditis" are sometimes used interchangeably or they can be used to indicate the dominant site of involvement. Cases that involve the myocardium in which pericarditis is predominant are reported as myopericarditis; alternatively, the term perimyocarditis is sometimes used when myocardial involvement is most prominent. However, in clinical practice, myopericarditis is more common and this term is often used in both senses. (See "Myopericarditis".)
Acute pericarditis — Acute pericarditis is diagnosed by the presence of at least two of the following criteria (table 2) [5,11,14,26,27]:

- Typical chest pain (sharp and pleuritic, improved by sitting up and leaning forward)
- Pericardial friction rub (a superficial scratchy or squeaking sound best heard with the diaphragm of the stethoscope over the left sternal border) (movie 1)
- Suggestive changes on the electrocardiogram (typically widespread ST segment elevation) ( waveform 1)
- New or worsening pericardial effusion

While echocardiography is often normal, and the absence of a pericardial effusion does not exclude pericarditis, the echocardiogram remains an essential part of the evaluation if there is evidence of an associated pericardial effusion and/or signs of cardiac tamponade.

Myopericarditis — When acute pericarditis is present, myopericarditis can be diagnosed by the detection of one or both of the following in the absence of evidence of another cause [28-31]:

- Elevation in serum cardiac biomarkers, such as cardiac troponin I or T
- New or presumed new focal or global left ventricular systolic dysfunction on imaging studies

A more complete discussion of the diagnosis of myopericarditis is presented separately. (See "Myopericarditis", section on 'Diagnosis'.)

IDENTIFYING THE ETIOLOGY — The yield of the standard diagnostic evaluation to determine the etiology of acute pericarditis is relatively low. This was illustrated in three series that included a total of 784 unselected patients who underwent an extensive evaluation [14,26,32]. A specific diagnosis was established in only 130 patients (17 percent) (table 3). The most commonly confirmed diagnoses were:

- Neoplasia – 5 percent
- Tuberculosis – 4 percent
- Autoimmune etiologies – 5 percent
- Purulent pericarditis – 1 percent

In Western countries, unless there is an apparent medical or surgical condition known to be associated with pericarditis, most cases of acute pericarditis in immunocompetent patients are due to viral infection or are idiopathic (table 1 and table 3) [6,10,27,32-35]. Acute viral or idiopathic pericarditis typically follows a brief and benign course after empiric treatment with antiinflammatory drugs. (See "Treatment of acute pericarditis".)

Because of the relatively benign course associated with the common causes of pericarditis, it is not necessary to search for the etiology in all patients with acute pericarditis. Initial efforts should focus upon excluding a significant effusion or tamponade and the identification of patients in whom a more comprehensive evaluation should be performed to exclude causes that require specific therapy (eg, malignancy, tuberculosis or purulent pericarditis) (table 1) [10]. In addition, among patients at high risk of coronary disease, myocardial ischemia must be ruled out by appropriate studies.

Indications for pericardiocentesis and pericardial biopsy — Studies in patients with acute pericarditis have reported a low yield for diagnostic pericardiocentesis and pericardial biopsy; however, some authors have advocated for a more extensive use of these techniques for diagnostic purposes. The majority of patients with uncomplicated acute pericarditis do not require invasive pericardial procedures. However, some high-risk patients may require pericardiocentesis for both therapeutic and diagnostic purposes (table 4). In addition, while pericardial biopsy is not required to make the diagnosis of acute pericarditis, it may rarely be necessary in an attempt to diagnose a specific etiology. (See "Treatment of acute pericarditis", section on 'Interventional therapeutic techniques'.)

Pericardiocentesis — In patients with a pericardial effusion, pericardiocentesis or surgical drainage can serve both diagnostic and therapeutic purposes. Among patients with acute pericarditis, decisions regarding drainage of the pericardial space are based upon the presence of an associated effusion, its echocardiographic characteristics (eg, size and composition), and clinical significance (eg, causing hemodynamic compromise).

- Patients with symptomatic effusions and evidence of cardiac tamponade should undergo prompt pericardial drainage. (See "Cardiac tamponade".)
When a significant pericardial effusion is present, a diagnostic pericardiocentesis is indicated if a specific etiology is highly suspected, and diagnosis cannot be reached by other means. The investigation is especially indicated when a neoplastic or bacterial etiology is suspected because a definite diagnosis can only be made by identification of the etiologic agent in the pericardial fluid. Fluid samples should be sent for cytology, tumor markers, gram stain, bacterial cultures, and, if tuberculosis is suspected, polymerase chain reaction testing for tuberculosis. (See "Diagnosis and treatment of pericardial effusion" and "Pericardial disease associated with malignancy").

Pericardiocentesis may be considered also for large effusions refractory to medical treatment [36].

Effusions that are small to moderate in size and do not cause hemodynamic compromise (ie, cardiac tamponade) generally do not require drainage, unless a sample of the effusion is necessary for diagnostic purposes. Moreover, pericardiocentesis performed percutaneously has a significantly higher complication rate if the effusion is not large.

A detailed discussion regarding the performance of pericardiocentesis and the treatment of pericardial effusions is presented separately. (See "Diagnosis and treatment of pericardial effusion").

**Pericardial biopsy** — Pericardial biopsy is generally performed as a part of a therapeutic procedure (surgical drainage) in patients with recurrent pericardial effusions and cardiac tamponade after prior pericardiocentesis (therapeutic biopsy), and as a diagnostic procedure in patients with an illness lasting more than three weeks despite treatment without a definite diagnosis. Technical advances in instrumentation with introduction of pericardioscopy, and in contemporary virology and molecular biology have improved the diagnostic value of epicardial/pericardial biopsy. The diagnostic yield of pericardial biopsy is typically higher in patients with pericardial effusion with or without pericarditis than in those who present with apparent acute pericarditis without effusion. Polymerase chain reaction techniques may represent a useful adjunct to conventional laboratory studies in the investigation of pericardial samples, allowing the rapid identification of microorganisms otherwise not easily found [36,37]. Tissue samples should be sent for cytology, tumor markers, gram stain, bacterial cultures, and, if tuberculosis is suspected, polymerase chain reaction testing. (See "Diagnosis and treatment of pericardial effusion", section on 'Pericardial fluid analysis and biopsy'.)

**DETERMINATION OF RISK AND NEED FOR HOSPITALIZATION** — Many clinicians admit all new cases of acute pericarditis to the hospital, but this may not be necessary. A patient with uncomplicated acute pericarditis can undergo initial evaluation in a same day hospital facility or clinic, although outpatient follow-up is required [6,10,32,35]. On the other hand, patients with high-risk features are at increased risk of short-term complications and have a higher likelihood of a specific disease [10,32]. Hospital admission is indicated for high-risk patients in order to initiate appropriate therapy and a thorough etiologic evaluation.

Features of acute pericarditis associated with a higher risk include [10,32]:

- Fever (>38°C [100.4°F]) and leukocytosis
- Evidence suggesting cardiac tamponade
- A large pericardial effusion (ie, an echo-free space of more than 20 mm)
- Immunosuppressed state
- A history of therapy with vitamin K antagonists (eg, warfarin)
- Acute trauma
- Failure to respond within seven days to NSAID therapy
- Elevated cardiac troponin, which suggests myopericarditis

In one report of 300 consecutive patients with acute pericarditis, 15 percent were deemed high risk at presentation and were hospitalized [10]. In the remaining 85 percent of patients who were low risk, outpatient aspirin therapy was effective in 87 percent, and none of these patients had a serious complication (eg, cardiac tamponade) at a mean follow-up of 38 months.

Although chronic use of glucocorticoids should not be considered as a risk factor in a general population of patients with acute pericarditis, they were associated with an increased rate of complications in idiopathic or viral pericarditis [32]. Glucocorticoid therapy given in the index attack may increase the chance of recurrence, probably because of its deleterious effect on viral replication and clearance. (See "Recurrent pericarditis", section on 'Predictors of recurrence'.)
Gender may also predict the likelihood of complications. In a series of 453 consecutive cases of acute pericarditis, women were at increased risk of complications (hazard ratio 1.65, 95% CI 1.08 to 2.52) [32]. A possible explanation of this finding is the higher frequency of autoimmune etiologies (above all connective tissue diseases) in women.

**PROGNOSIS** — Patients with acute idiopathic or viral pericarditis have a good long-term prognosis. Cardiac tamponade rarely occurs in patients with acute idiopathic pericarditis and is more common in patients with a specific underlying etiology such as malignancy, tuberculosis, or purulent pericarditis. Constrictive pericarditis may occur in about 1 percent of patients with acute idiopathic pericarditis, and is also more common in patients with a specific etiology. (See "Constrictive pericarditis".)

Approximately 15 to 30 percent of patients with idiopathic acute pericarditis who are not treated with colchicine develop either recurrent or incessant disease. Immune mechanisms appear to be of primary importance in the majority of cases, and the term "chronic autoreactive" pericarditis has been used. Risk factors for recurrent pericarditis include lack of response to nonsteroidal antiinflammatory drugs, the need for corticosteroid therapy, and inappropriate pericardiectomy or creation of a pericardial window. The pathogenesis, course, and treatment of recurrent pericarditis are discussed separately. (See "Recurrent pericarditis".)

**INFORMATION FOR PATIENTS** — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient information: Pericarditis in adults (The Basics)"
- Beyond the Basics topic (see "Patient information: Pericarditis (Beyond the Basics)"

**SUMMARY AND RECOMMENDATIONS**

- Acute pericarditis (inflammation of the pericardial sac) is the most common disorder of the pericardium and is seen in about 0.1 percent of hospitalized patients and 5 percent of patients admitted to the Emergency Department for non-ischemic chest pain. (See ‘Epidemiology’ above.)
- Idiopathic cases, most of which are probably viral in etiology, are the most common causes of acute pericarditis. Other etiologies of acute pericarditis include any bacterial infections, malignancy, and autoimmune disorders (table 3). The distribution of etiologies varies with geography and type of clinical setting (community hospital versus tertiary referral center). (See ‘Epidemiology’ above.)
- The diagnosis of acute pericarditis is usually suspected based on a history of characteristic pleuritic chest pain, especially when a pericardial friction rub is present. Pericarditis should also be suspected in a patient with persistent fever and pericardial effusion or new unexplained cardiomegaly. (See ‘Clinical features’ above.)
- The evaluation of a patient with suspected acute pericarditis includes blood work (assessing for markers of inflammation or myocardial damage), chest radiography, electrocardiography, and echocardiography. The electrocardiogram (ECG) is often the most helpful test in the evaluation of patients with suspected acute pericarditis. Echocardiography is often normal, but can be an essential part of the evaluation if there is evidence of an associated pericardial effusion and/or signs of cardiac tamponade. (See ‘Diagnosis’ above and ‘Evaluation’ above.)
- Acute pericarditis is diagnosed by the presence of at least two of the following criteria (table 2): (See ‘Diagnosis’ above.)
  - Typical chest pain (sharp and pleuritic, improved by sitting up and leaning forward). (See ‘Chest pain’above.)
  - Pericardial friction rub (a superficial scratchy or squeaking sound best heard with the diaphragm of the stethoscope over the left sternal border) (movie 1). (See ‘Pericardial friction rub’ above.)
• Suggestive changes on the electrocardiogram (typically widespread ST segment elevation) (waveform 1). (See ‘Electrocardiogram’ above.)
• New or worsening pericardial effusion. (See ‘Echocardiogram’ above.)

● Because of the relatively benign course associated with the common causes of pericarditis, it is not necessary to search for the etiology in all patients. Initial efforts should focus upon excluding a significant effusion or tamponade and the identification of patients in whom a more comprehensive evaluation should be performed to exclude causes that require specific therapy (eg, malignancy, tuberculosis or purulent pericarditis). (See ‘Identifying the etiology’ above.)

● A patient with uncomplicated acute pericarditis can undergo initial evaluation in a same-day hospital facility or clinic, although outpatient follow-up is required. Conversely, patients with high-risk features (ie, high fever, large pericardial effusion, cardiac tamponade, failure to respond to empiric antiinflammatory therapy) are at increased risk of short-term complications and have a higher likelihood of a specific disease. Hospital admission is indicated for high-risk patients in order to initiate appropriate therapy and thorough etiologic evaluation. (See ‘Determination of risk and need for hospitalization’ above.)

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REFERENCES

Major causes of pericardial disease

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
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</thead>
<tbody>
<tr>
<td>Idiopathic</td>
<td>In most case series, the majority of patients are not found to have an identifiable cause of pericardial disease. Frequently such cases are presumed to have a viral or autoimmune etiology.</td>
</tr>
<tr>
<td>Infections</td>
<td>Viral - Coxsackievirus, echovirus, adenovirus, EBV, CMV, influenza, varicella, rubella, HIV, hepatitis B, mumps, parvovirus B19, vaccina (smallpox vaccination)</td>
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<td>Bacterial - Staphylococcus, Streptococcus, pneumococcus, Haemophilus, Neisseria (gonorrhoeae or meningitidis), Chlamydia (psittaci or trachomatis), Legionella, tuberculosis, Salmonella, Lyme disease</td>
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<td></td>
<td>Mycoplasma</td>
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<td>Fungal - Histoplasmosis, aspergillosis, blastomycosis, coccidiodomycosis, actinomycosis, nocardia, candida</td>
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<td>Parasitic - Echinococcus, amebiasis, toxoplasmosis</td>
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<td>Infective endocarditis with valve ring abscess</td>
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<td>Radiation</td>
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<td>Neoplasm</td>
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<tr>
<td>Metastatic - Lung or breast cancer, Hodgkin's disease, leukemia, melanoma</td>
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<td>Primary - Rhabdomyosarcoma, teratoma, fibroma, lipoma, leiomyoma, angioma</td>
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<td>Paraneoplastic</td>
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<td>Late postcardiac injury syndrome (Dressler's syndrome), also seen in other settings (eg, post-myocardial infarction and post-cardiac surgery)</td>
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<td>Dissecting aortic aneurysm</td>
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<td>Blunt</td>
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<td>Penetrating</td>
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<td>Iatrogenic - Catheter and pacemaker perforations, cardiopulmonary resuscitation, post-thoracic surgery</td>
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<td>Rheumatic diseases - Including lupus, rheumatoid arthritis, vasculitis, scleroderma, mixed connective disease</td>
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<tr>
<td>Other - Granulomatosis with polyangiitis (Wegener's), polyarteritis nodosa, sarcoidosis, inflammatory bowel disease (Crohn's, ulcerative colitis), Whipple's, giant cell arteritis, Behcet's disease, rheumatic fever</td>
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<tr>
<th>Drugs</th>
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<tr>
<td>Procainamide, isoniazid, or hydralazine as part of drug-induced lupus</td>
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<tr>
<td>Other - Cromolyn sodium, dantrolene, methysergide, anticoagulants, thrombolytics, phenytoin, penicillin, phenylbutazone, doxorubicin</td>
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<th>Metabolic</th>
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<td>Hypothyroidism - Primarily pericardial effusion</td>
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<td>Uremia</td>
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<td>Ovarian hyperstimulation syndrome</td>
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Graphic 67851 Version 6.0

**Cardiac auscultation supine and leaning forward**
Auscultation of the pericardium: To elicit pericardial rubs, the patient is invited to lean forward (A) or rest on elbows and knees (B). Both physical maneuvers increase the contact of visceral and parietal pericardium.

Reproduced from: Heart, Imazio M. Pericardial involvement in systemic inflammatory diseases, 97:1882, Copyright © 2011, with permission from BMJ Publishing Group Ltd.

Electrocardiogram (ECG) in pericarditis

Electrocardiogram in acute pericarditis showing diffuse upsloping ST segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR segment deviation (positive in aVR, negative in most other leads). ST segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR segment changes are due to an atrial
current of injury which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

*Courtesy of Ary Goldberger, MD.*

**Normal ECG**

![Normal ECG](image)

Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°.

**Electrocardiogram (ECG) in an evolving anterior myocardial infarction**

![Electrocardiogram in an evolving anterior myocardial infarction](image)

Electrocardiogram shows findings typical of an evolving Q-wave anterior MI: loss of R waves in leads V1 to V3, ST segment elevations in V2 to V4, and T wave inversions in leads I, aVL, and V2 to V5. Sinus bradycardia (55 beats/min) is present due to concurrent therapy with a beta blocker.

*Courtesy of Ary Goldberger, MD.*

Graphic 81914 Version 3.0

**Normal ECG**
Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Hyperacute (peaked) T waves

Hyperacute T waves are >5 mm in the limb leads, and usually >10 mm in the precordial leads. They have a peaked, symmetric morphology.

Graphic 60464 Version 4.0

Normal ECG

Normal sinus rhythm at a rate of 71 beats/min, a P wave axis of 45°, and a PR interval of 0.15 sec.

Courtesy of Morton Arnsdorf, MD.
**Early repolarization 12 lead ECG**

Early repolarization manifest as inferior J-point slurring and lateral J-point notching, each >1 mm in two contiguous leads.

Graphic 83883 Version 2.0

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**Chest x-ray of a pericardial effusion**

Cardiomegaly due to a massive pericardial effusion. At least 200 mL of pericardial fluid must accumulate before the cardiac silhouette enlarges.

*Courtesy of Massimo Imazio, MD, FESC.*
Graphic 57640 Version 3.0

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**Diagnostic criteria for acute pericarditis and myopericarditis in the clinical setting**
**Acute pericarditis (at least 2 criteria of 4 should be present)**:

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
3. Absence of evidence of any other cause
4. One of the following features: evidence of elevated cardiac enzymes (creatine kinase-MB fraction, or troponin I or T), OR new onset of focal or diffuse depressed LV function by an imaging study, OR abnormal imaging consistent with myocarditis (MRI with gadolinium, gallium-67 scanning, anti-myosin antibody scanning)

**Case definitions for myopericarditis include**:

- **Suspected myopericarditis**: criteria 1 plus 2 and 3
- **Probable myopericarditis**: criteria 1, 2, 3, and 4
- **Confirmed myopericarditis**: histopathologic evidence of myocarditis by endomyocardial biopsy or on autopsy

* Pericardial effusion confirms the clinical diagnosis but its absence does not exclude it. ¶ In clinical practice a confirmed diagnosis would require an endomyocardial biopsy that is not warranted in self-limited cases with predominant pericarditis.

**Acute pericarditis etiology: Data from published clinical studies with unselected populations**

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<thead>
<tr>
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<tbody>
<tr>
<td>Location</td>
<td>Spain</td>
<td>Spain</td>
<td>Italy</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>199 (86.0 percent)</td>
<td>78 (78.0 percent)</td>
<td>377 (83.2 percent)</td>
</tr>
<tr>
<td>Specific etiology</td>
<td>32 (14.0 percent)</td>
<td>22 (22.0 percent)</td>
<td>76 (16.8 percent)</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>13 (5.6 percent)</td>
<td>7 (7.0 percent)</td>
<td>23 (5.1 percent)</td>
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**Indications for invasive workup in acute pericarditis**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>1. Cardiac tamponade</th>
<th>2. Moderate to large effusions refractory to medical therapy and with severe symptoms</th>
<th>3. Suspected bacterial or neoplastic pericarditis</th>
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<tbody>
<tr>
<td><strong>Pericardiocentesis:</strong></td>
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<tr>
<td><strong>Pericardial biopsy and pericardioscopy (targeted biopsy in specialized center):</strong></td>
<td>1. Relapsing cardiac tamponade</td>
<td>2. Suspected bacterial or neoplastic pericarditis</td>
<td>3. Worsening pericarditis (despite medical therapy) without a specific diagnosis</td>
</tr>
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</table>

**Treatment of acute pericarditis**

**Author**
Massimo Imazio, MD, FESC

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Martin M LeWinter, MD

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All topics are updated as new evidence becomes available and our peer review process is complete.

**Literature review current through:** Jun 2015. | **This topic last updated:** Nov 24, 2014.

**INTRODUCTION** — The pericardium is a fibroelastic sac made up of visceral and parietal layers separated by a (potential) space, the pericardial cavity. In healthy individuals, the pericardial cavity contains 15 to 50 mL of an ultrafiltrate of plasma.

Diseases of the pericardium present clinically in one of several ways:

- Acute and recurrent pericarditis
- Pericardial effusion without major hemodynamic compromise
- Cardiac tamponade
- Constrictive pericarditis
- Effusive-constrictive pericarditis

Acute pericarditis refers to inflammation of the pericardial sac. The term myopericarditis, or perimyocarditis, is used for cases of acute pericarditis that also demonstrate myocardial inflammation. The treatment options available for acute pericarditis will be reviewed here. The etiology of pericarditis, clinical presentation and diagnostic evaluation of acute pericarditis, and other pericardial disease processes are discussed separately. (See "Etiology of pericardial disease" and "Clinical presentation and diagnostic evaluation of acute pericarditis" and "Recurrent pericarditis" and "Cardiac tamponade" and "Constrictive pericarditis" and "Diagnosis and treatment of pericardial effusion").

**TREATMENT** — The therapy of acute pericarditis should be targeted as much as possible to the underlying etiology [1-4]. In patients with an identified cause other than viral infection, specific therapy appropriate to the underlying disorder is indicated. However, in developed countries, most cases of acute pericarditis in...
Immunocompetent patients are due to viral infection or are idiopathic. Because of the relatively benign course associated with the common causes of pericarditis (>80 percent of cases), it is not necessary to search for the etiology in all patients. As such, most patients are treated for a presumptive viral cause with nonsteroidal anti-inflammatory drugs (NSAIDS) and colchicine. (See "Pericardial disease associated with malignancy" and "Tuberculous pericarditis" and "Purulent pericarditis").

Most patients with acute pericarditis can be managed effectively with medical therapy alone. However, patients with a large pericardial effusion, a hemodynamically significant pericardial effusion, a suspicion of a bacterial or neoplastic etiology, or evidence of constrictive pericarditis should be evaluated for invasive therapies, such as pericardial drainage and/or pericardiectomy (pericardial window). (See ‘Interventional therapeutic techniques’ below.)

Some clinical features of acute pericarditis impart a higher risk to the patient; as such, patients felt to be at high risk should be hospitalized for additional evaluation and initiation of treatment [5,6]. Features associated with a higher risk include:

- Fever (>38°C [100.4°F]) and leukocytosis
- Evidence suggesting cardiac tamponade
- A large pericardial effusion (ie, an echo-free space of more than 20 mm)
- Immunosuppressed state
- A history of therapy with vitamin K antagonists (eg, warfarin)
- Acute trauma
- Failure to respond within seven days to NSAID therapy
- Elevated cardiac troponin, which suggests myopericarditis

Patients with none of the listed high-risk features can be safely treated on an outpatient basis. A protocol has been proposed for outpatient treatment of those at low risk (algorithm 1) [5]. (See "Clinical presentation and diagnostic evaluation of acute pericarditis", section on 'Determination of risk and need for hospitalization'.)

In acute viral or idiopathic pericarditis, no therapy has been rigorously proven to prevent serious sequelae, such as cardiac tamponade and constrictive pericarditis. Fortunately, however, these complications are rare [7,8]. (See "Constrictive pericarditis" and "Cardiac tamponade",)

Activity restriction — Strenuous physical activity may trigger recurrence of symptoms; therefore, such activity should be avoided until symptom resolution. While there are little if any data to guide recommendations on activity restriction, our experts’ approach to activity restriction is consistent with the position of the European Society of Cardiology in a 2006 position paper [9]:

- Competitive athletes should not participate in competitive sports for at least three months following the resolution of symptoms and normalization of biomarkers, and should be reevaluated by a clinician prior to resuming training and competition.
- Non-competitive athletes should restrict activity until the resolution of symptoms and normalization of biomarkers.

In cases of myopericarditis, we recommend withdrawal from competitive sports for six months and return to play only after normalization of laboratory data (eg, markers of inflammation, ECG, and echocardiogram) (See "Myopericarditis", section on 'Treatment'.)

Nonsteroidal anti-inflammatory drugs — In the treatment of acute pericarditis, the goals of therapy are the relief of pain and resolution of inflammation (and, if present, pericardial effusion). We recommend NSAIDs for all patients without a contraindication, with the duration of treatment based upon the persistence of symptoms, which is usually for two weeks or less. An individualized approach based on symptom control and normalization of C-reactive protein (CRP) has also been proposed, in which CRP is assessed at presentation and then weekly, with anti-inflammatory drugs prescribed until complete resolution of symptoms and normalization of CRP [10]. Long-term data demonstrating that routine measurement of CRP improves outcomes or reduces the risk of recurrent pericarditis are not available.

Based on the results of multiple cohort studies and one randomized study, treatment with NSAIDs alone appears to be effective in approximately 70 to 80 percent of pericarditis cases presumed to be of viral or idiopathic origin.
Primary therapy has been the administration of oral NSAIDs, particularly ibuprofen or aspirin-ketorolac, a parenteral NSAID, is also effective (table 1) [12]. NSAIDs and aspirin function to both reduce inflammation and relieve pain in most patients [5,8,11-13-15]. Despite these benefits, however, there is no evidence that NSAIDs or aspirin alter the natural history of acute pericarditis.

A theoretical concern is that the antiplatelet activity of aspirin or other NSAID might promote the development of a hemorrhagic pericardial effusion. However, such a relationship has never been convincingly established and the risk-benefit ratio seems to favor the use of these drugs.

Failure to respond to aspirin or NSAID therapy within one week (defined as persistence of fever, pericarditic chest pain, a new pericardial effusion, or worsening of general illness) suggests that a cause other than idiopathic or viral pericarditis is present. (See "Clinical presentation and diagnostic evaluation of acute pericarditis", section on 'Identifying the etiology'.)

In a series of 254 patients deemed to be at low risk who were treated with aspirin as outpatients, 98 percent of patients who responded to aspirin were presumed to have idiopathic or viral disease, while 2 percent of the patients who responded to aspirin were subsequently diagnosed with an autoimmune disorder [5]. In contrast, among the patients who did not respond to aspirin after seven days, only 39 percent were deemed idiopathic, while 43 percent were diagnosed with an autoimmune disorder and 18 percent with tuberculous pericarditis. At follow-up, aspirin resistance was associated with significant increases in the rates of recurrent pericarditis (61 versus 10 percent) and constrictive pericarditis (nine versus one percent).

**NSAID regimens** — The 2004 European Society of Cardiology guidelines recommended the use of an NSAID for the treatment of acute pericarditis [16]. Commonly used NSAID regimens include (table 1):

- **Ibuprofen** – Depending on the severity of the pericarditis and individual medication response, ibuprofen three times daily is usually adequate for symptom relief (table 1). Ibuprofen can be continued for days or weeks for recurrent or incessant attacks as needed. NSAID dose tapering may be prescribed in an attempt to reduce the subsequent recurrence rate [5,17]. The 2004 ESC guidelines suggested ibuprofen as the preferred NSAID because of its rare side effects, favorable impact on coronary artery blood flow, and large dose range [16].
- **Aspirin** – Aspirin can be given every six to eight hours (table 1) followed by gradual tapering every two to three days for a treatment period of three to four weeks [5].
- **Indomethacin** – Indomethacin can be administered at a dose of 50 mg three times daily for one to two weeks followed by gradual tapering every two to three days for a treatment period of three to four weeks.

In symptomatic pericarditis occurring within days after an acute myocardial infarction, aspirin is preferred, and the use of an NSAID other than aspirin should be AVOIDED, since anti-inflammatory therapy may impair scar formation [18]. Aspirin may also be the first choice in patients who require concomitant antiplatelet therapy for any reason. With either regimen, gastrointestinal protection should be provided. (See "Pericardial complications of myocardial infarction" and "NSAIDs (including aspirin): Primary prevention of gastroduodenal toxicity" and 'Gastrointestinal protection' below.)

One practical approach is the initial administration of an NSAID at a full dose (ie, "attack dose") every six to eight hours to achieve better symptom control than with a lower dose. The attack dose is maintained empirically for one to two weeks or until complete symptom resolution. Tapering should be considered following the attack dose in an attempt to reduce the subsequent risk of recurrence. Normalization of C-reactive protein may be used to tailor the duration of treatment [10].

**Gastrointestinal protection** — Nonsteroidal anti-inflammatory drugs (NSAIDs) can lead to gastrointestinal toxicity, particularly when used in high doses or for prolonged periods of time. In addition to high doses or prolonged periods of treatment, patient-related factors associated with a higher risk of gastrointestinal toxicity include:

- History of peptic ulcer disease
- Age greater than 65 years
- Concurrent use of aspirin, corticosteroids, or anticoagulants

Patients considered at risk of gastrointestinal toxicity related to NSAID treatment should be treated with NSAIDs for the shortest interval possible and receive concomitant gastroprotective therapy while taking NSAIDs. Proton pump
inhibitors (eg, omeprazole, pantoprazole) are generally preferred for prevention of gastrointestinal toxicity due to their efficacy and favorable safety profile. (See "NSAIDs (including aspirin): Primary prevention of gastroduodenal toxicity").

Concomitant use of antiplatelet and anticoagulation therapies — In patients who require more than one antiplatelet or anticoagulant as therapy for an underlying condition, there is a greater risk of bleeding complications. On occasion, patients with acute pericarditis treated with NSAIDs may also have an indication for an additional antiplatelet or anticoagulant. However, there are no apparent increased risks of hemorrhagic pericardial effusion, cardiac tamponade, or recurrent pericarditis in such patients. (See "Anticoagulation in older adults", section on 'Risk of bleeding'.)

There are no specific contraindications or additional risks of bleeding when antiplatelet therapies are used during acute pericarditis. In this setting aspirin is generally the first choice to treat pericarditis, but doses should be increased to reach anti-inflammatory effects (from 100 to 300 mg to up to 750 to 1000 mg three times per day). (See 'NSAID regimens' above.)

In contrast to antiplatelet therapies, concomitant use of heparin and anticoagulant therapies is often perceived as a possible risk factor for the development of a worsening or hemorrhagic pericardial effusion that may result in cardiac tamponade. Use of anticoagulant therapy has also been considered a possible poor prognostic predictor in the setting of acute pericarditis, but the available evidence does not support this [19].

- A multivariable analysis of nearly 500 consecutive cases of acute pericarditis did not show this to be the case [6].
- In another study of 274 patients with acute pericarditis or myopericarditis, the use of heparin or other anticoagulants was not associated with an increased risk of cardiac tamponade (OR 1.1, 95% CI 0.3 to 3.5) [20].

NSAIDs and aspirin may, however, interfere with the metabolism of vitamin K antagonists (eg, warfarin), thus enhancing the anticoagulant effect of vitamin K antagonists. Consequently, careful monitoring is needed. Additionally, consideration should be given to using alternative anti-inflammatory options such as glucocorticoids that have the potential for fewer bleeding-related drug interactions in patients requiring both anti-inflammatory drugs and chronic anticoagulation therapy. However, the potential benefits of using glucocorticoids to lower the risk of bleeding must be balanced with the potential side effects associated with glucocorticoids. (See "Major side effects of systemic glucocorticoids".)

There are no significant reported interactions between NSAIDs or other antiplatelet therapies and colchicine [21].

Colchicine — The majority of patients have prompt resolution of symptoms without recurrent pericarditis when treated with NSAIDs alone. However, when used as an adjunct to NSAID therapy, colchicine reduces symptoms, decreases the rate of recurrent pericarditis, and is generally well tolerated. As such, we recommend that colchicine be added to NSAIDs in the management of a first episode of acute pericarditis [22] (table 1).

The effect of colchicine in the primary management of acute pericarditis has subsequently been evaluated in two randomized trials:

- In the ICAP trial, a randomized, double-blind study of colchicine versus placebo in addition to standard anti-inflammatory therapy for treatment of a first episode of acute pericarditis (77 percent idiopathic), colchicine added to standard anti-inflammatory therapy significantly reduced the risk of recurrence (17 percent versus 38 percent with anti-inflammatory therapy alone; relative risk reduction 0.56; 95% CI 0.30-0.72) [23]. In addition, colchicine added to anti-inflammatory treatment resulted in significantly better remission rates and fewer hospitalizations compared to anti-inflammatory treatment alone. Colchicine was given without a loading dose colchicine as 0.5 mg twice daily for three months for patients weighing >70 kg or 0.5 mg once daily for patients weighing ≤70 kg. Overall adverse effects and rates of study-drug discontinuation were similar in the two study groups. No serious adverse events were observed.
- In the open label COPE trial of 120 patients with a first episode of acute pericarditis (84 percent idiopathic), the recurrence rate of pericarditis within 18 months was significantly lower in the colchicine plus aspirin group (11 percent versus 32 percent with aspirin alone; number needed to treat to prevent one recurrence equals five). [11].

Two systematic reviews have evaluated the efficacy of colchicine in the treatment of pericarditis:
A 2012 systematic review and meta-analysis (published prior to the ICAP results) included the results of five published trials (795 patients, mean follow-up 13 months) that evaluated the efficacy and safety of colchicine for pericarditis prevention, including three double-blind, randomized trials and two open-label, randomized trials [24]. Colchicine use was associated with a reduced risk of recurrent pericarditis during follow-up (RR = 0.40, 95% CI 0.30-0.54) without a significantly higher risk of adverse events.

A 2014 systematic review and meta-analysis, which included four randomized, double-blind trials (564 patients) of colchicine for both initial and recurrent episodes of pericarditis, reported similar results [25]. Colchicine use was associated with a reduced risk of recurrent pericarditis at 18 months in patients being treated for acute (HR 0.40; 95% CI 0.27-0.61) or recurrent (HR 0.37; 95% CI 0.24-0.58) pericarditis. There was no significant increase in adverse effects related to colchicine therapy [26].

The 2004 ESC guidelines, published before the COPE and ICAP trials and the subsequent meta-analyses, concluded that the weight of evidence supported the efficacy of colchicine (0.5 to 1 mg/day), alone or in combination with NSAIDs, in the treatment of acute pericarditis [16]. The findings in the subsequent trials and meta-analyses, including the relative lack of toxicity, support the addition of colchicine to NSAIDs in acute pericarditis (table 1). Findings of the COPE and ICAP studies are applicable only to adults without elevated levels of aminotransferases, creatinine, or troponin and those without liver disease, myopathy, blood dyscrasias, or inflammatory bowel disease. Pregnant or lactating women were also excluded as well as patients with bacterial or neoplastic pericarditis.

Moreover, colchicine appears to be effective in the prevention of postpericardiotomy syndrome following cardiac surgery. This is discussed in greater detail separately. (See "Post-cardiac injury syndromes").

Colchicine is typically well tolerated. Side effects, most commonly gastrointestinal (eg, diarrhea, nausea, vomiting), are uncommon at low doses (0.5 to 1.2 mg per day), even when given continuously over years. Less common (<1 percent) side effects include bone marrow suppression, hepatotoxicity, and myotoxicity. Chronic renal insufficiency leading to increased colchicine levels appears to be the major risk factor for side effects and other possible negative interactions. In addition, colchicine has drug interactions and altered metabolism in certain patient populations. (See "Treatment of acute gout", section on 'Safety of colchicine'.)

Of note, colchicine is not approved for the prevention of recurrent pericarditis in North America or Europe, and its use as such is off-label. (See "Recurrent pericarditis", section on 'Colchicine'.)

Glucocorticoids — Glucocorticoids should be considered only if acute pericarditis results in symptoms that are clearly refractory to NSAIDs and colchicine, and a specific cause for the pericarditis has been excluded [13]. Corticosteroids may also be used in case of contraindications or failure of aspirin/NSAID, or rarely for specific indications (ie, systemic inflammatory diseases, pregnancy). The number of such patients is quite low as illustrated in the COPE trial and in an observational series of 254 low risk patients in which almost 90 percent of patients responded to aspirin alone within seven days and most of the nonresponders had an autoimmune disease or tuberculosis [5,11].

A number of studies, mostly observational, suggest that glucocorticoid therapy early in the course of the disease is more likely to be associated with recurrent episodes [11,27-29]. However, a concern with observational evidence related to glucocorticoid therapy is that such therapy may be more likely to be used in patients with disease resistant to initial therapy, which would be a predictor of recurrence independent of prior administration of glucocorticoid.

The best data come from the COPE trial of colchicine therapy in which glucocorticoids were given only when aspirin was contraindicated or not tolerated [11]. On multivariate analysis, glucocorticoid use was a significant predictor of recurrence (OR 4.30, 95% CI 1.21 to 15.25). The same effect has been reported for patients with the first recurrence or multiple recurrences and may be due to promotion of viral replication [27,30-32].

A subsequent systematic review evaluated the results of two randomized trials comparing steroid therapy to standard NSAID therapy and one trial of low-dose versus high-dose steroid therapy (with or without other therapy with NSAIDs or colchicine) [33]. The administration of steroids was associated with a trend toward a higher rate of recurrent pericarditis (OR 7.50, 95% CI 0.62 to 90.65).
In addition to concerns about the efficacy of glucocorticoid therapy as initial treatment of acute pericarditis, chronic use of systemic glucocorticoids is associated with a number of potentially significant side effects. (See "Major side effects of systemic glucocorticoids".)

**Approaches to glucocorticoid use** — While NSAIDs and colchicine remain the preferred treatment options for acute pericarditis, a minority of patients will have refractory symptoms requiring treatment with systemic steroid therapy. There are conflicting data, mostly derived from observational studies, regarding the optimal dosing and tapering of steroid therapy when used to treat pericarditis.

**European Society of Cardiology guidelines** — The 2004 European Society of Cardiology (ESC) guidelines recommended that systemic steroid therapy be restricted to patients with the following conditions [16]:

- Patients with symptoms refractory to standard therapy
- Acute pericarditis due to connective tissue disease
- Autoreactive (immune-mediated) pericarditis
- Uremic pericarditis (see "Pericarditis in renal failure")

The 2004 ESC guidelines recommend use of high doses of glucocorticoids (eg, prednisone 1 mg/kg/day) when indicated with rapid tapering to reduce the risk of systemic side effects. However, others have used the lowest steroid dose that provides symptomatic relief [33]. In patients with a coexisting pericardial effusion, intrapericardial steroid administration is an option that limits systemic toxicity [16].

**Our approach to glucocorticoid use** — Our approach to glucocorticoid dosing differs from the 2004 ESC guidelines (table 1). In our experience, rapid tapering of systemic glucocorticoids increases the risk of treatment failure and recurrence. Although high doses of glucocorticoids (eg, prednisone 1 mg/kg/day) have been recommended in the ESC guidelines, use of lower doses (eg, prednisone 0.25 to 0.50 mg/kg/day) may be equally efficacious. These lower doses may be useful in reducing the risk of steroid side effects, which have been reported in up to 25 percent of patients treated with high doses. (See "Major side effects of systemic glucocorticoids".)

We generally add colchicine during glucocorticoid therapy and continue colchicine for several months after glucocorticoid discontinuation (ie, with an overall length of treatment of three months for acute pericarditis, six months in recurrent cases). We introduce aspirin or another NSAID toward the end of tapering or in case of recurrences instead of increasing the dose of the glucocorticoids.

Results from a study of patients with recurrent pericarditis suggest that lower glucocorticoid doses may also be feasible in acute pericarditis, although these populations differ. In an observational study, 100 patients with recurrent pericarditis were treated with glucocorticoids (51 treated with high-dose prednisone 1.0 mg/kg/day and 49 treated with prednisone 0.2 to 0.5 mg/kg/day) [34]. After adjustment for potential confounders only high doses of prednisone were associated with more side effects, recurrences, and hospitalizations (hazard ratio, 3.61; 95% CI 1.96 to 6.63).

We usually begin tapering glucocorticoids at two to four weeks, after resolution of symptoms and/or C-reactive protein normalization. Each decrement in prednisone dose should proceed only if the patient is asymptomatic, particularly for doses lower than 25 mg/day. A proposed tapering scheme follows:

- Daily dose >50 mg – taper 10 mg/day every one to two weeks
- Daily dose 25 to 50 mg – taper 5-10 mg/day every one to two weeks
- Daily dose 15 to 25 mg – taper 2.5 mg/day every two to four weeks
- Daily dose <15 mg – taper 1.25 to 2.5 mg/day every two to six weeks

We introduce aspirin or another NSAID toward the end of tapering or in the case of recurrences, instead of increasing the dose of the corticosteroid. (See "Recurrent pericarditis".)

In a systematic review of published studies on medical therapy for pericarditis, data from three observational studies of steroid treatment showed that steroid use was associated with a trend toward increased risk of recurrent pericarditis (OR 7.50, 95% CI 0.62 to 90.65) [33]. However, low-dose steroids were superior to high-dose steroids for treatment failure or recurrent pericarditis (OR 0.29, 95% CI 0.13 to 0.66), rehospitalization for pericarditis (OR 0.19, 95% CI 0.06 to 0.63), and adverse effects (OR 0.07, 95% CI 0.01 to 0.54).
Interventional therapeutic techniques — Most patients with acute pericarditis can be managed effectively with medical therapy alone. On occasion, however, patients may require invasive therapies for:

● A moderate to large pericardial effusion, particularly if hemodynamically significant and causing cardiac tamponade or symptomatic and refractory to medical therapy
● Suspicion of a neoplastic or bacterial etiology and moderate to large pericardial effusion
● Frequent, highly symptomatic recurrences of acute pericarditis with pericardial effusion
● Evidence of constrictive pericarditis (a late occurrence when present)

Percutaneous and surgical techniques may be considered for such patients.

Pericardial drainage — Prolonged catheter drainage of a pericardial effusion is an effective means of preventing fluid reaccumulation. The mechanism by which this occurs is probably more related to the obliteration of the pericardial space following inflammation provoked by the catheter, rather than fluid drainage itself. Catheter drainage may be required for several days and the catheter should not be removed until drainage is less than 20 to 30 ml/24 hours. (See "Cardiac tamponade" and "Diagnosis and treatment of pericardial effusion", section on 'Treatment'.)

Pericardiotomy, pericardial window and pericardiectomy — Surgical removal of all or part of the pericardium is virtually never required for the treatment of acute pericarditis. However, pericardiectomy may be considered for frequent and highly symptomatic recurrences of pericarditis resistant to medical treatment or recurrent cardiac tamponade [16]. The efficacy of pericardiectomy in the management of recurrent idiopathic pericarditis is unproven and should be considered only in exceptional cases. Other situations in which to consider pericardiectomy include repeated recurrences of pericardial effusions resulting in cardiac tamponade, evidence of serious steroid toxicity limiting further medical treatment, or the late occurrence of constrictive pericarditis. (See "Recurrent pericarditis" and "Cardiac tamponade" and "Constrictive pericarditis".)

Surgical decompression of the pericardium (also known as pericardiotomy, pericardiostomy, and "window" pericardiectomy) can be achieved either by conventional heart surgery or video-assisted thoracoscopy. These techniques may result in a lower incidence of effusion recurrence compared with pericardiocentesis and prolonged catheter drainage. However, surgical experiences are not always concordant, and the efficacy of pericardiectomy remains largely unproven.

Less-invasive options (eg, balloon pericardiotomy) for the management of recurrent symptomatic pericardial effusions are mainly derived from the experience of management of neoplastic pericardial effusions and include prolonged catheter drainage and the creation of the so-called "pericardial window". These techniques, which involve inserting balloon catheters into the pericardial space using a subxiphoid approach under fluoroscopic or echocardiographic guidance, are highly successful in preventing recurrent effusions, especially for patients with a reduced life expectancy since reaccumulation of fluid may occur with longer follow-up. However, stretching of the pericardium is often painful so appropriate analgesia is necessary. (See "Pericardial disease associated with malignancy".)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

● Basics topics (see "Patient information: Pericarditis in adults (The Basics)"
● Beyond the Basics topic (see "Patient information: Pericarditis (Beyond the Basics)"

SUMMARY AND RECOMMENDATIONS
In cases of pericarditis due to an identifiable cause (eg, bacterial infection or malignancy), management is focused upon the underlying disorder and, if necessary, drainage of an associated pericardial effusion. (See ‘Treatment’ above.)

For most patients with acute idiopathic or viral pericarditis, we recommend combination therapy with colchicine plus NSAIDs rather than NSAIDs alone (Grade 1A). This is based upon a reduced rate of recurrent pericarditis and a low incidence of side effects with colchicine (table 1). We generally administer ibuprofen plus colchicine. An acceptable alternative is indomethacin plus colchicine. Because NSAIDs do not alter the natural history of pericarditis, NSAID treatment duration is based upon the persistence of symptoms, which is usually for two weeks or less, while colchicine is generally continued for three months. Individualized treatment length may be based on a weekly assessment of markers of inflammation (ie, C-reactive protein). Intravenous administration of NSAIDs may be useful to control acute and severe symptoms at the beginning of therapy. (See ‘Nonsteroidal anti-inflammatory drugs’ above and ‘Colchicine’ above.)

In patients with acute pericarditis occurring within days following a myocardial infarction (MI), we suggest aspirin plus colchicine (table 1) rather than another NSAID plus colchicine (Grade 2C). This is principally due to the possibility that other NSAIDs may interfere with healing and scar formation. Although the evidence of potential harm from glucocorticoids and NSAIDs other than aspirin is modest, there is no evidence that these medications improve outcomes. For these reasons glucocorticoids and NSAIDs other than aspirin should generally be AVOIDED in patients with acute pericarditis following an acute MI. (See ‘Nonsteroidal anti-inflammatory drugs’ above and ‘Pericardial complications of myocardial infarction’, section on ‘Anti-inflammatory therapy’.)

Patients treated with aspirin or another NSAID should also receive gastrointestinal protection (eg, a proton pump inhibitor). (See ‘Gastrointestinal protection’ above and "NSAIDs (including aspirin): Primary prevention of gastroduodenal toxicity".)

Among patients with acute pericarditis, initial management with systemic glucocorticoid therapy should be restricted to patients with pericarditis due to connective tissue disease, autoreactive (immune-mediated) pericarditis, uremic pericarditis not responding to dialysis, and to patients who have contraindications to NSAID therapy. Glucocorticoid therapy is also used for patients with idiopathic or viral pericarditis that is refractory to combination therapy with NSAIDs and colchicine. (See ‘Glucocorticoids’ above.)

For patients who require glucocorticoid therapy for acute pericarditis, we suggest the use of moderate initial dosing (eg, 0.25 to 0.50 mg/kg/day of prednisone) followed by a slow taper rather than high doses with a rapid taper (table 1) (Grade 2C). (See ‘Our approach to glucocorticoid use’ above.)

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REFERENCES
Initial clinical and echocardiographic evaluation of patients with suspected acute pericarditis

Clinical and echocardiographic evaluation:
Clinical poor prognostic predictors (fever >38°C, subacute onset, immunodipression, trauma, oral anticoagulants, myopericarditis, severe effusion, cardiac tamponade)

- Yes
- No

Admission to hospital
Specific etiologic search
Aspirin trial

Response to out-of-hospital trial of aspirin or other NSAIDs plus colchicine

- Yes
- No

Admission to hospital
Specific etiologic search
Idiopathic acute pericarditis
### Drug therapy in acute and recurrent pericarditis for adult patients

<table>
<thead>
<tr>
<th>Drug</th>
<th>Anti-inflammatory dose</th>
<th>Duration of therapy* (anti-inflammatory dose)</th>
<th>Tapering*</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td><strong>For initial combination treatment of most patients:</strong></td>
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<tr>
<td>Aspirin&lt;sup&gt;3&lt;/sup&gt;</td>
<td>650 to 1000 mg orally three times daily</td>
<td>One to two weeks</td>
<td>Weekly decrease once patient is symptom-free and CRP has normalized</td>
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<td>OR</td>
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<tr>
<td>Ibuprofen&lt;sup&gt;1&lt;/sup&gt;</td>
<td>600 to 800 mg orally three times daily&lt;sup&gt;a&lt;/sup&gt;</td>
<td>One to two weeks</td>
<td>Weekly decrease once patient is symptom-free and CRP has normalized</td>
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<td>OR</td>
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<tr>
<td>Indomethacin&lt;sup&gt;4&lt;/sup&gt;</td>
<td>25 to 50 mg orally three times daily</td>
<td>One to two weeks</td>
<td>Weekly decrease once patient is symptom-free and CRP has normalized</td>
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<tr>
<td>PLUS</td>
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<tr>
<td>Colchicine&lt;sup&gt;5&lt;/sup&gt;</td>
<td>0.5 to 0.6 mg orally two times daily</td>
<td>Three months (acute) Six months or longer (recurrent)</td>
<td>Usually not tapered&lt;sup&gt;f&lt;/sup&gt;</td>
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<tr>
<td><strong>For initial combination therapy of patients following myocardial infarction:</strong></td>
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<td>Aspirin&lt;sup&gt;3&lt;/sup&gt;</td>
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<td>Three months (acute) Six months or longer (recurrent)</td>
<td>Usually not tapered&lt;sup&gt;f&lt;/sup&gt;</td>
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<tr>
<td><strong>For refractory cases or patients with a contraindication to NSAID therapy:</strong></td>
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<tr>
<td>Prednisone</td>
<td>0.25 to 0.5 mg/kg/day</td>
<td>Two weeks (acute) Two to four weeks (recurrent)</td>
<td>Gradual tapering over three months; refer to UpToDate topic review of treatment of acute pericarditis, section on glucocorticoids</td>
</tr>
<tr>
<td>PLUS</td>
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<tr>
<td>Colchicine&lt;sup&gt;5&lt;/sup&gt;</td>
<td>0.5 to 0.6 mg orally two times daily</td>
<td>Three months</td>
<td>Usually not tapered&lt;sup&gt;f&lt;/sup&gt;</td>
</tr>
</tbody>
</table>
CRP: C-reactive protein. * Duration and tapering of therapy (except colchicine) should be tailored according to resolution of symptoms and normalization of markers of inflammation; refer to topic reviews for approach. ¶ Proton pump inhibitor (eg, omeprazole) gastrointestinal protection may be indicated. Δ Some patients may require ibuprofen every six hours (four times daily), in which case the dose should not exceed 600 mg every six hours. ◊ 0.5 mg colchicine is not available in US. It is widely available elsewhere. § Colchicine dose should be reduced to 0.5 to 0.6 mg once daily in patients <70 kg. ¥ The duration of colchicine therapy for recurrent or refractory pericarditis is at least six months. Data from: Lange RA, Hillis LD. Clinical practice. Acute pericarditis. N Engl J Med 2004; 351:2195. Maisch B, Seferovic PM, Ristic AD, et al. Guidelines on the diagnosis and management of pericardial disease: The task force on the diagnosis and management of pericardial disease of the European Society of Cardiology. European Heart Journal 2004; 25:587. Imazio M, Brucato A, Trinchero R, et al. Individualized therapy for pericarditis. Expert Rev Cardiovasc Ther 2009; 7:965.