Pericardial Diseases
Radhika Prabhakar
A 45-year-old woman is evaluated for severe chest pain.

Which of the following conditions is demonstrated on this patient's electrocardiogram?

A. Anteroseptal myocardial infarction
B. Inferior myocardial infarction
C. Pericarditis
D. Posterior myocardial infarction
Educational Objective: Identify electrocardiographic manifestations of pericarditis. Pericarditis is demonstrated on this patient's electrocardiogram. Electrocardiographic changes characteristic of acute pericarditis include diffuse ST-segment elevations and a depressed PR interval, both of which are present in this electrocardiogram. As pericarditis evolves, the electrocardiographic manifestations change and are classified into stages: stage 1 is characterized by diffuse ST-segment elevations; stage 2 is characterized by “pseudonormalization,” in which the ST segments normalize; stage 3 is characterized by diffuse T-wave inversion and possible slightly depressed ST segments; and in stage 4, the electrocardiogram returns to normal.
Definitions & Anatomy

- Pericarditis: Inflammation of the pericardium
- Function of pericardium is to protect the heart and reduce friction between heart and adjacent structures
- Mechanical barrier to infection
- Influences ventricular pressures

Figure 1. A: Anterior view of the anatomy of the pericardium after section of the large vessels at their cardiac origin and removal of the heart. PCR = post caval recess. RPVR = right pulmonary vein recess. LPVR = light pulmonary vein recess. SVC = superior vena cava. ICV = inferior vena cava. B: Cross section of the heart wall and the pericardium, showing the pericardial layers.

The visceral and parietal layers make up serosal pericardium, which lies within the outer fibrous pericardium.
Pericardial Diseases

- Recurrent Pericarditis
- Pericardial Effusion
- Tamponade
- Effusive
- Transient
- Chronic

Complications

- Constrictive Pericarditis
- Acute Pericarditis
Acute Pericarditis

- Acute inflammation of pericardium
- Clinical features: Positional chest pain, characteristic EKG changes and a pericardial friction rub
  - +/- pericardial effusion
- Epicardium can also become involved, leading to myocarditis and elevated troponins
- In developed countries, idiopathic etiology is by far the most common
# Acute Pericarditis

## Causes (Developed World)

<table>
<thead>
<tr>
<th>Infectious (2/3 of cases)</th>
<th>Non-infectious (1/3 of cases)</th>
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<tbody>
<tr>
<td>Viral (e.g. echovirus, coxsackievirus; most viral forms are labeled idiopathic since it is often difficult and technically demanding to reveal an underlying viral infection)</td>
<td>Autoimmune (including systemic autoimmune e.g. RA, SLE) autoinflammatory diseases and pericardial injury syndromes</td>
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<td>Bacterial (tuberculous in 4%-5% of cases)</td>
<td>Neoplastic (commonly metastatic disease e.g. lung, breast, and lymphoma, rarely primary, e.g. pericardial mesothelioma)</td>
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<td>Fungal or parasitic (e.g. Echinococcus, Toxoplasma)</td>
<td>Metabolic (Uremic, myxedema)</td>
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<td>Traumatic (e.g. post-pericardiectomy, blunt trauma)</td>
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<td>Drug related (procainamide, isoniazid, phenytoin, hydralazine, penicillins, anthracyclines)</td>
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Acute Pericarditis
Clinical Presentation

- Sharp retrosternal chest pain
- Improves with sitting, leaning forward, can be pleuritic (worse with inspiration)
- Friction rub heard on exam
Acute Pericarditis

EKG

Diagnostic criteria (2/4 must be met):
1) Characteristic chest pain
   - Retrosternal
   - Worse when supine, upon inspiration, cough
   - Improved with sitting upright
2) Pericardial friction rub
3) Characteristic EKG findings
4) New or worsening pleural effusion
   +/- elevated CRP and pericardial enhancement on cardiac MRI

Stage 1) ST-segment elevation & PR-segment depression in all leads except aVR: lasts hours to days
Stage 2) Pseudonormalization: days to several weeks
Stage 3) T-wave inversion: starts at the end of 2-3 weeks and lasts for several weeks
Stage 4) Return to baseline
Acute Pericarditis
Other diagnostic tools

- 2-D echo
  - Can see small pericardial effusion
- Biochemical markers: Troponin (& other cardiac enzymes), ESR/CRP, WBC
  - Sometimes negative but can be important
  - Troponins may be elevated in progression to myocarditis (should obtain to rule out)
- Chest X-ray
  - Recommended in all pericarditis cases
    - Can see enlarge cardiac silhouette if there is an effusion
    - Can also see e/o associated pathologies (e.g. PNA)
- CT/cMRI
  - Can be obtained with to assess complications of acute pericarditis, or in cases of complex clinical presentations (e.g. trauma-related) or if diagnosis is unclear
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ASA and NSAIDs are first choice
- Add colchicine to idiopathic pericarditis for 3-6 mos
- COPE trial showed reduced recurrence with colchicine + ASA
- Initial dose is for 1-2 weeks and then taper for 1-2 weeks until resolution of symptoms and normalization of CRP
- In post-MI pericarditis ASA is used before NSAIDs
- Steroids reserved for refractory disease
- Treat underlying etiology when possible
- Provide PPI for all patients receiving NSAIDs or ASA
Acute Pericarditis

Treatment

- Outpatient vs Inpatient
  - High risk patients should be hospitalized
    - Pericardial effusion > 2 cm
    - Subacute presentation
    - Febrile
    - Unresponsive to NSAIDS
    - Minor criteria: myopericarditis, traumatic pericarditis, immunodepression, anticoagulant therapy

- All patients should be restricted from physical activity until symptoms resolve and CRP normalizes (if elevated)
Acute Pericarditis

Prognosis

- Prognosticating factors:
  - Overall fairly good prognosis, especially for first occurrence (<~2% of idiopathic acute pericarditis patients have severe complications)

- Poor prognostic factors
  - Presence of effusion/tamponade
  - Recurrence/Failure of NSAIDs
  - Constrictive pericarditis
  - Progression to myo-pericardial inflammatory syndrome is not a bad prognostic factor
A 38-year-old woman is evaluated for a 1-week history of substernal chest pain. Symptoms are aggravated by lying in the supine position or taking a deep breath. The patient is otherwise healthy and takes no medications.

On physical examination, temperature is 37.5 °C (99.5 °F), blood pressure is 112/70 mm Hg, and pulse rate is 84/min. Systolic pressure variation with respiration is 5 mm Hg. The lungs are clear to auscultation. A loud, three-component cardiac rub is heard at the apex. S₁ and S₂ are normal. The remainder of the physical examination is unremarkable.

Laboratory studies are significant for a leukocyte count of 7000/µL (7.0 x 10⁹/L). Echocardiogram shows normal left and right ventricles. A moderately sized circumferential pericardial effusion is noted. The inferior vena cava is normal in size. A Doppler ultrasound shows minimal change in mitral inflow with respiration.

High-dose ibuprofen, a proton pump inhibitor, and colchicine are started.

Which of the following is the most appropriate next step in management?

A Cardiac CT
B Clinical follow-up
C Glucocorticoid administration
D Inpatient monitoring
E Pericardiocentesis
Educational Objective: Manage acute pericarditis on an outpatient basis.

Key Point

- Patients with acute pericarditis who do not have high-risk features (fever, leukocytosis, acute trauma, abnormal cardiac biomarkers, immunocompromise, oral anticoagulant use, large pericardial effusions, or evidence of cardiac tamponade) can be managed medically on an outpatient basis with close clinical follow-up.

- This patient should receive clinical follow-up without hospital admission or further diagnostic testing to monitor her response to therapy, evaluate for possible complications, and assess the timing for tapering her medications. **Slow tapering over 2 to 4 weeks** after initial presentation with improvement in symptoms is usually performed to reduce the risk of recurrent inflammation.

- The vast majority of patients with acute pericarditis, including the patient presented, can be managed medically on an outpatient basis. For a subset of patients, high-risk features of acute pericarditis may be present and warrant hospitalization for treatment and monitoring for possible complications; these include fever, leukocytosis, acute trauma, abnormal cardiac biomarkers, an immunocompromised host, oral anticoagulant use, large pericardial effusions, or evidence of cardiac tamponade.

- CT can be used to show pericardial thickening in patients with acute pericarditis. However, this finding would not change the diagnosis or appropriate management strategy in this patient.

- Medical therapy with anti-inflammatory agents is appropriate for acute pericarditis. However, glucocorticoids are reserved for patients who do not respond to NSAIDs, such as ibuprofen, aspirin, and indomethacin, none of which has been tried yet in this patient. Glucocorticoid therapy may also increase the risk of recurrent pericarditis and should only be considered in highly selected patients with refractory pericarditis.

- **Pericardiocentesis** is indicated only for patients with tamponade or for those in whom the analysis of pericardial fluid can be of assistance in diagnosis and management. Signs of tamponade are not present in this patient whose inferior vena cava is normal in size on echocardiography, whose Doppler ultrasound shows minimal change in mitral inflow with respiration, and whose bedside maneuvers reveal no pulsus paradoxus.
Recurrent Pericarditis

- Recurrence after symptom free period (usually at least 6 weeks)
  - Recurrent pain + at least one sign of inflammation (fever, friction rub, EKG findings, echo showing pericardial effusion, cMRI showing pericardial inflammation, elevated WBC, ESR or CRP)
- Can be difficult complication of acute pericarditis because of imposition on patient’s quality of life
- Incidence has decreased significantly with adjuvant use of colchicine
  - ~17% of idiopathic pericarditis in some studies
  - Overall 20-30% after a single episode of pericarditis

- Work-up for recurrent pericarditis is same as acute. If there is a known etiology for acute episode, then there is no need to look again for etiology.
- However if etiology is unclear then worthwhile to pursue further evaluation, particularly for autoimmune etiologies
Recurrent Pericarditis
Causes (Not in order of frequency!)

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<tr>
<td><strong>3. Inadequate anti-inflammatory therapy</strong></td>
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<td><strong>4. Autoimmune/connective tissue diseases</strong></td>
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<td><strong>5. Post-pericardiectomy</strong></td>
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<td><strong>6. Malignancy</strong></td>
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<td><strong>7. Corticosteroids</strong></td>
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<td><strong>8. Auto-inflammatory disorders;</strong> (e.g. Familial Mediterranean fever (FMF) and TNF receptor associated periodic syndrome (TRAPS))</td>
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## Recurrent Pericarditis Treatment

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- Same medications, longer treatment course
- Taper over days to weeks depending on response
- Definitely add colchicine, for at least 6 months
- Steroids are indicated when a patient has a recurrence unresponsive to NSAIDs+colchicine
- If all above treatments have failed, there have been cases of immunosuppressive agents (azathioprine, IVIG, anakinra)
- Ultimate treatment is pericardiectomy when all medical management has failed
A 68-year-old man is evaluated in the emergency department for a 24-hour history of persistent chest pain. He had a non–ST-elevation myocardial infarction 1 week ago that was managed medically with complete symptom recovery. Yesterday, he developed recurrent chest pain that differs from his previous angina pain. The pain is constant but improved when leaning forward and not associated with other symptoms. Medications are low-dose aspirin, clopidogrel, metoprolol, and atorvastatin.

On physical examination, vital signs are normal. There is no jugular venous distention. The lungs are clear to auscultation. S₁ and S₂ are normal, and there is no S₃ or S₄. A two-component friction rub is present at the left lower sternal border, and a grade 2/6 holosystolic murmur is heard at the apex. The remainder of the physical examination is unremarkable.

Electrocardiogram shows diffuse, concave upward ST-segment elevations and PR-segment depression most prominent in leads V₁ through V₆.

Which of the following is the most appropriate primary treatment?

- **A** High-dose aspirin
- **B** Ibuprofen
- **C** Nitroglycerin
- **D** Prednisone
Educational Objective: Treat a patient with acute pericarditis with high-dose aspirin.

Key Point

Anti-inflammatory therapy with aspirin or other NSAIDs, such as ibuprofen, is indicated in patients with acute pericarditis; when the pericarditis is associated with myocardial infarction, only aspirin should be used because other NSAIDs can impair myocardial healing and increase the risk of mechanical complications.

This patient should receive high-dose aspirin. He has acute pericarditis in the setting of a recent myocardial infarction. The typical chest pain, physical examination findings, and abnormal electrocardiogram (ECG) are all consistent with this diagnosis, especially the findings of concave upward ST-segment elevation and PR-segment depression in all leads, except aVR, on the ECG. Anti-inflammatory therapy with aspirin or other NSAIDs, such as ibuprofen, is indicated in patients with acute pericarditis. In those whose pericarditis is associated with myocardial infarction, such as this patient, only aspirin should be used because ibuprofen and other NSAIDs can impair myocardial healing and increase the risk of mechanical complications. The anti-inflammatory medication should be given in relatively high doses to achieve an anti-inflammatory effect and then tapered slowly over 2 to 4 weeks to reduce the risk of recurrent pericarditis. Colchicine (0.5-1.2 mg/d) also has been shown to be effective as adjunctive therapy to anti-inflammatory agents in patients with acute pericarditis, further reducing the risk of recurrent pericarditis and treatment failure. Colchicine is not recommended for patients with post-infarction pericarditis. Colchicine may be associated with gastrointestinal side effects, liver toxicity, and bone marrow suppression but is generally well tolerated.

Nitroglycerin is an effective therapy for chest pain caused by myocardial ischemia but is not effective for symptoms caused by pericarditis.

Glucocorticoids, such as prednisone, are reserved for patients with contraindications to NSAIDs or those with refractory acute pericarditis, primarily because there is evidence that their use is associated with an increased risk of recurrent pericarditis. As this patient has no apparent contraindication to aspirin use, treatment with glucocorticoids is not indicated.
A 74-year-old man is evaluated 4 months after undergoing uncomplicated bioprosthetic surgical aortic valve replacement. Within the past 2 weeks, he has developed exertional dyspnea, fatigue, and lower extremity edema. Medical history is otherwise unremarkable, and he takes no medications.

On physical examination, vital signs are normal. The estimated central venous pressure is 12 cm H$_2$O, and the jugular venous pulse shows prominent y descents. A pericardial knock is present. Peripheral edema is noted.

An echocardiogram reveals no evidence of pericardial effusion. The aortic and mitral valves are functioning normally. The inferior vena cava is markedly enlarged. A Doppler ultrasound shows expiratory flow reversals in the hepatic veins consistent with constrictive pericarditis.

Which of the following is the most appropriate next step in management?

A. Ibuprofen
B. Invasive cardiac hemodynamic evaluation
C. Pericardiectomy
D. Transesophageal echocardiography
Educational Objective: Treat a patient with potentially transient constrictive pericarditis.

Key Point

In some patients with constrictive pericarditis, the constriction may be transient and either spontaneously resolve or respond to medical therapy, which obviates the need for surgical pericardiectomy.

This patient has evidence of constrictive pericarditis and should be treated with an anti-inflammatory medication, such as a high-dose NSAID or prednisone. Supportive findings are symptoms and signs of right heart failure and congestion, with hemodynamic evidence of constriction on echocardiography. In some patients with constrictive pericarditis, the constriction can be transient and either spontaneously resolve or respond to medical therapy. This subtype of constrictive pericarditis more frequently has idiopathic, viral, or postsurgical causes. Although a minority of patients will have this transient constrictive pericarditis, a trial of medical therapy with an anti-inflammatory medication is reasonable. If medical therapy is successful, then surgical pericardiectomy can be avoided. Anti-inflammatory medication regimens for potentially transient constrictive pericarditis are similar to those for acute pericarditis, with relatively high doses of NSAIDs used (for example, ibuprofen, 800 mg three times daily; indomethacin, 50 mg three times daily; aspirin, 650 mg three times daily), with a slow taper over 2 to 3 weeks.

Cardiac catheterization for hemodynamic assessment of possible constriction is only indicated when diagnostic information cannot be obtained with echocardiography, which is not the case in this patient.

Pericardiectomy is inappropriate before a 2- to 3-month trial of anti-inflammatory medication in this patient. Although pericardiectomy is the definitive treatment for relief of heart failure in patients with constrictive pericarditis, it is a complex, invasive procedure that should not be used in patients with transient constriction.

Transesophageal echocardiography is only indicated when data from other noninvasive imaging studies (such as transthoracic echocardiography) are inconclusive.
Constrictive Pericarditis

- Due to the fibrotic thickening of pericardium, leading to restriction in diastolic filling and equalization of end-diastolic cardiac pressures

- Tuberculous and purulent pericarditis are the highest risk etiologies of constrictive pericarditis (then neoplastic and autoimmune, idiopathic/viral relatively low risk).

- Patients present with signs of right heart failure (jugular venous distension, hepatomegaly, pedal edema)

- Can have symptoms of dyspnea on exertion, decreased exercise tolerance from decreased cardiac output

- Transient, effusive (elevated RAP persists after drainage of pericardial effusion), chronic (protracted course resulting in heart failure and need for surgical pericardiectomy)
Constrictive Pericarditis
Diagnostic Studies

- EKG is non-diagnostic
- CXR also non-diagnostic, can see some pericardial calcification
- Echocardiography is diagnostic test of choice
  - Increased pericardial thickness
  - Restrictive ventricular filling pattern
- CT scan can show pericardial thickening
- Right heart catherization—Gold standard but invasive
  - Use if all other methods fail to yield a diagnosis
  - Disassociation of intrathoracic and intracardiac pressure
  - Equalization of cardiac pressures in end-diastole
Constrictive Pericarditis
Treatment

Treatment:
- Pericardiectomy

Exceptions:
- Unless transient (treat as acute pericarditis)
  - Will self-resolve with standard medical treatment within an average of 8 weeks
  - Trial 2-3 months of anti-inflammatory therapy in hemodynamically stable patients with constrictive pericarditis
  - Very unlikely with radiation-induced
  - Advanced myocardial fibrosis with NYHA class IV sx
  - Poor operative morbidity

Treatment of underlying condition
- Radiation-induced in particular has a poor prognosis
A 52-year-old woman is evaluated for fatigue and lower extremity swelling. One year ago, she had acute idiopathic pericarditis treated with anti-inflammatory medications and colchicine. Symptoms initially improved and her medications were discontinued. However, for the past 3 months, she has had worsening symptoms of exertional fatigue and edema in both legs. She currently takes no medications.

On physical examination, she is afebrile, blood pressure is 130/78 mm Hg, pulse rate is 88/min, and respiration rate is 16/min. Pulsus paradoxus of 15 mm Hg is present. The estimated central venous pressure is 10 cm H$_2$O, and the jugular venous pulse contour shows diminished y descents. The lungs are clear to auscultation. Heart sounds are normal, with no rubs or gallops. Hepatomegaly is present, and peripheral edema is noted in both lower extremities up to the knees.

A 12-lead electrocardiogram is normal. Echocardiogram shows the cardiac chambers to be normal in size and function with a moderate circumferential pericardial effusion. A CT of the heart shows normal pericardial thickness.

A pericardiocentesis fails to resolve the elevated right atrial pressure documented on right heart catheterization.

Which of the following is the most likely diagnosis?

A Cor pulmonale
B Effusive constrictive pericarditis
C Heart failure
D Recurrent acute pericarditis
Educational Objective: Diagnose effusive constrictive pericarditis.

Key Point

Effusive constrictive pericarditis is a clinical entity in which patients who have a pericardial effusion experience persistent symptoms and hemodynamic derangements after relief of the pericardial effusion.

The most likely diagnosis in this patient is effusive constrictive pericarditis. Effusive constrictive pericarditis is a clinical entity in which patients who have a pericardial effusion, with or without cardiac tamponade, experience persistent symptoms and hemodynamic derangements after treatment and relief of the pericardial effusion. In some patients with constrictive pericarditis, pericardial inflammation results in an effusion, which is placed under pressure by the inelastic pericardium. Symptoms of low cardiac output, systemic congestion, and an elevated jugular venous pulse are seen, as in constrictive pericarditis; however, in patients with effusive constrictive pericarditis, a pericardial knock is absent and the y descent of the jugular venous pulse may be less prominent. Additionally, pulsus paradoxus may be present, which is not a typical finding of constrictive pericarditis. This disorder is caused by pericarditis involving the visceral layer of the pericardium. Thickening of the visceral layer of the pericardium can be difficult to detect with CT and other noninvasive imaging, and a high index of clinical suspicion is necessary to establish the diagnosis. In this patient, effusive constrictive pericarditis is suggested by the persistently elevated right atrial pressure following pericardiocentesis.

Similar findings in the jugular venous pulse can occur in patients with cor pulmonale or heart failure. With cor pulmonale, however, evidence of right ventricular dysfunction or chamber enlargement is usually seen on imaging. The clear lungs on auscultation do not support the presence of heart failure.

The presence of a normal electrocardiogram and the absence of the typical symptom of chest pain argue against recurrent acute pericarditis as a diagnosis. Additionally, acute pericarditis cannot explain this patient’s pulsus paradoxus and elevated jugular venous pulse.
Pericardial Effusions

- >50 mL of fluid in pericardial space
- Classifications
  - Time Frame: Acute <1 week, Subacute 1 week-3 months, Chronic > 3mos
  - Size: Small < 10 mm, Moderate 10-20 mm, Large >20 mm
  - Presence of tamponade
  - Composition/type: Hemopericardium, pyopericardium, chylopericardium,
- Exudative (most) vs transudative
  - Use Light’s criteria to differentiate (not as well validated as in pleural effusions)
    - pericardial fluid/serum protein greater than or equal to 0.5
    - pericardial fluid/serum lactate dehydrogenase greater than or equal to 0.6
    - pericardial fluid lactate dehydrogenase greater than or equal to 200 U/L.
  - Transudative is heart failure, hypoalbuminemia, renal insufficiency, pulmonary hypertension
  - Exudative has large differential diagnosis: inflammatory, infectious, malignant, autoimmune
Pericardial Effusion Causes
(similar to causes of pericarditis)

Idiopathic
Infectious
Viral (echovirus, coxsackievirus, adenovirus, HIV/AIDS, and so forth)
Bacterial (Pneumococcus, Staphylococcus, Streptococcus, and so forth)
Fungal (histoplasmosis and coccidiomycosis)
Protozoal (Echinococcus and Toxoplasma)
Inflammatory
Connective tissue disease (systemic lupus erythematosus, rheumatoid arthritis, or scleroderma)
Vasculitis (polyarteritis nodosa, temporal arteritis, or Churg-Strauss disease)
Drug induced (procainamide, hydralazine, or isoniazid)
Postcardiotomy/thoracotomy
Pulmonary Hypertension (Transudative effusion, balanced by high right-sided chamber pressures)
Takatsubo cardiomyopathy

Miscellaneous (sarcoidosis, familial Mediterranean fever, or inflammatory bowel disease)
Postmyocardial infarction (associated with pericarditis)
Early (2-4 days)
Late (Dressler syndrome) (1-8 weeks)
Hemopericardium (after percutaneous cardiac procedures, trauma, free-wall ventricle rupture post-STEMI)
Trauma
Dissecting aortic aneurysm
Iatrogenic (endomyocardial biopsy, post-percutaneous coronary intervention/pacemaker or automated implantable cardioverter-defibrillator placement/ablation-valve repair or replacement/atrial septal defect or ventricular septal defect closure)
Anticoagulants
Malignancy
Miscellaneous
Radiation therapy (earlier than constrictive pericarditis)
Chronic renal failure
Chylopericardium (chest trauma, malignancy, tb, subclavian vein occlusion or thrombosis)
Hyperthyroidism or hypothyroidism
Pericardial Effusions
Clinical Presentation

- Depend on size and (more significantly) rate of accumulation
  - Slowly accumulating effusions (e.g. malignant) can be less symptomatic than rapidly accumulating ones
  - Large effusions carry poor prognosis
- Common symptoms are dyspnea on exertion, orthopnea, palpitations, chest pain/tightness.
- Can be asymptomatic
- Clinical signs:
  - Beck’s triad—muffled heart sounds, jugular venous distension, low blood pressure
  - Pulsus paradoxicus (decrease in SPB >10 mm during inspiration)
Pericardial Effusion Diagnosis

- 2-d Echocardiogram—Test of choice
  - Gives information on effusion’s size and hemodynamic effect
- CT, Cardiac MRI
  - Also very sensitive for presence of effusion
  - Density of fluid can aid in identifying etiology of effusion
  - Can be more precise for localization and quantification and for certain characteristics (pericardial thickening, clots, pericardial cysts)
- EKG
  - Non-diagnostic
  - Sinus tachycardic, decreased voltage, electrical alternans
- Pericardiocentesis, pericardial biopsy
  - Test of last resort
  - Used if for recurrent tamponade, or persistent effusion without defined etiology
  - If pericardial fluid is obtained, detailed studies should be sent out with focus on possible etiologies (e.g. cytology if malignancy is a concern, ADA, acid-fast stain and culture if Tb is possible, etc)
Pericardial Effusion
Treatment

- When NOT associated with tamponade
  - Treat associated etiology
  - E.g. When associated with pericarditis, treat the pericarditis
  - High risk for tamponade: bacterial, tubercular pericarditis, neoplastic, non-chronic moderate-to-large effusion, intrapericardial bleeding

- If there is clinical tamponade
  - Consider pericardiocentesis
    - Percutaenous under echocardiographic guidance (subxiphorid vs. apical approach)
    - May need window for recurrent effusion
  - Can also consider pericardiocentesis for persistent large idiopathic asymptomatic effusion lasting > 3 mos despite therapy
    - Relatively high risk of progressing to tamponade
Pericardial Effusion Treatment

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<th>Action</th>
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<td>Step 1</td>
<td>If tamponade OR suspected bacterial or neoplastic etiology</td>
<td>Pericardiocentesis and workup for underlying etiology</td>
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<td>If Step 1 is negative and pericarditis is the likely etiology</td>
<td>Medical therapy for pericarditis</td>
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<tr>
<td>Step 4</td>
<td>If pericardial effusion is &gt;20 mm and chronic</td>
<td>Consider pericardiocentesis</td>
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Postcardiac Injury Syndromes

Cause of pericarditis
- 1-8 weeks after myocardial infarction-Dressler’s Syndrome
- Thoracic trauma-traumatic pericarditis
- Cardiac surgery-Postpericardiotomy Syndrome
  - 10-40% of cardiac surgeries
  - Usually occurs in the first 3 mos
  - 2/5 criteria: Unexplained fever > 1 week, pleuritic chest pain, pericardial friction rub, pleural effusion with elevated CRP, worsening pericardial effusion
    - Pericardial effusions are very common in the acute period after cardiac surgery
    - Treat with colchicine, +/- NSAIDs, and surgical intervention if necessary
A 68-year-old man is evaluated for new-onset ascites with lower-extremity edema. Symptoms have increased gradually over the past 4 weeks. He has consumed three alcoholic beverages per day for many years. His medical history is notable for coronary artery bypass graft surgery 8 months ago and dyslipidemia. His medications are low-dose aspirin, atorvastatin, and metoprolol.

On physical examination, temperature is 36.8°C (98.2°F), blood pressure is 122/84 mm Hg, pulse rate is 64/min, and respiration rate is 16/min; BMI is 28. Cardiac examination reveals an elevated jugular venous pressure, a normal S₁ and S₂, and no murmurs. Pulmonary examination findings are normal. Abdominal examination reveals hepatomegaly, distention, dullness to percussion over the flanks, and a positive fluid wave. There is 2+ pitting edema of the lower extremities.

Laboratory studies reveal a serum albumin level of 3.5 g/dL (35 g/L). Other studies, including serum alanine aminotransferase and aspartate aminotransferase levels, are normal.

Paracentesis reveals a total nucleated cell count of 120/µL with 30% polymorphonucleocytes. Ascitic fluid albumin level is 2.3 g/dL (23 g/L) and total protein is 3.5 g/dL (35 g/L).

Which of the following is the most likely cause of this patient's ascites?

- A. Alcoholic cirrhosis
- B. Constrictive pericarditis
- C. Nonalcoholic cirrhosis
- D. Tuberculous peritonitis
Educational Objective: Diagnose constrictive pericarditis as a cause of ascites.

Key Point

A serum-ascites albumin gradient (SAAG) of 1.1 g/dL (11 g/L) or greater with an ascitic fluid total protein level of 2.5 g/dL (25 g/L) or greater indicates a cardiac cause of ascites.

The most likely diagnosis is constrictive pericarditis. This patient has undergone previous cardiac surgery, which is a risk factor for constrictive pericarditis. Ascitic fluid analysis should include measurement of albumin and total protein; cell count and bacterial cultures should be checked when infection is suspected. The serum-ascites albumin gradient (SAAG) should be calculated by subtracting the ascitic fluid albumin level from the serum albumin level. The main factors that distinguish a cardiac source for ascites from other sources are a SAAG of 1.1 g/dL (11 g/L) or greater and an ascitic fluid total protein level of 2.5 g/dL (25 g/L) or greater. This patient meets these criteria, making a cardiac cause for his ascites likely. In addition, over 90% of patients with constrictive pericarditis have evidence of jugular venous distention and clear lungs on auscultation. Other less commonly observed findings include Kussmaul sign (rise in jugular pressure on inspiration), paradoxical pulse, and a pericardial knock on cardiac auscultation.

Patients with cirrhosis, portal hypertension, and resultant ascites will also have a SAAG greater than 1.1 g/dL (11 g/L), but the ascitic fluid total protein level will be less than 2.5 g/dL (25 g/L). Therefore, alcoholic and nonalcoholic cirrhosis are not the likely cause of this patient’s ascites.

Tuberculous peritonitis is very uncommon and is associated with a SAAG less than 1.1 g/dL (11 g/L), an ascitic fluid total protein level greater than 3 g/dL (30 g/L), and a lymphocytic predominance in the cell count with differential. Although this patient has a high ascitic fluid total protein level, the SAAG is greater than 1.1 g/dL (11 g/L) and he does not have a predominance of lymphocytes on the ascitic fluid cell count.
Take-Away Points

- Pericardial disease can occur in a wide variety of clinical setting. It may rarely be the presenting symptom of malignancy, connective tissue disease, or an underlying infection like tuberculosis.

- Viral/idiopathic is the most common cause of acute pericarditis in developed countries.

- Aspirin, NSAIDs, colchicine are the mainstay of therapy for pericarditis, whereas corticosteroids are indicated only for refractory cases.

- Colchicine adjuvant therapy has been shown to prevent recurrence.

- Pericardiectomy is a surgical procedure indicated for chronic constrictive pericarditis.

- The most serious complication of a pericardial effusion is tamponade.

- Treatment of a pericardial effusion depends on size, chronicity, hemodynamic stability and cause.
References