



EM CASES SUMMARY

Episode 166 Pericarditis & Cardiac Tamponade

With Drs. Paul Dorian & Walter Himmel

Prepared by Kate Dillon, March 2022

Pericarditis as a diagnosis of exclusion

Pericarditis should be considered a **diagnosis of exclusion**, after ruling out the big chest pain killers (MI, PE, aortic dissection, esophageal rupture, and tension pneumothorax), because it can be easily confused with these more time-sensitive, deadly diagnoses. Avoid premature closure!

The following features are suggestive of pericarditis, but none alone have good enough test characteristics to rule it in or out.

Clinical features of pericarditis

Timing	Persistent chest pain for weeks (this is not typical for ACS, PE, dissection pneumothorax)
Age	Can occur at any age, but in young, otherwise healthy patients , they are more likely to have pericarditis than ACS, PE, or dissections (unless they have specific risk factors for these conditions)
Prodrome	Sometimes preceded by a respiratory or GI prodrome (can be seen in ACS too!)
Characteristics of Pain	Typically, chest pain from pericarditis is: <ul style="list-style-type: none"> • Pleuritic (typically substernal, radiating to back, neck or shoulder) • Central • Sharp • Worse lying supine, better when sitting up and leaning forward Note: this can be difficult for patients to describe!
Exam Features	Fever: can be present (associated with poorer prognosis) Pericardial Friction Rub: Left sternal border, best heard leaning forward at end expiration, triphasic and sounds like sandpaper, remember to apply the stethoscope FIRMLY , or you can mistake its movement for a rub

Pericarditis diagnostic criteria and evaluation

The diagnosis of pericarditis requires 2/4 of the following criteria:

1. Chest pain – typically sharp, pleuritic, positional (>80- 90% of cases)
2. Pericardial rub on auscultation (<1/3rd of cases)
3. New widespread ST elevation or PR depression on ECG (up to 60% of cases)
4. New or worsening pericardial effusion (up to 60% of cases)

Additional supportive findings:

- Elevated inflammatory markers (CRP, ESR, WBC count)
- Evidence of pericardial inflammation on imaging (contrast CT, cardiac MR)

Diagnostic evaluation for pericarditis

- Auscultate for friction rub (excellent specificity/poor sensitivity)
- ECG (see below)
- Ultrasound (see below)
- CXR (enlarged cardiac silhouette if associated with large pericardial effusion)
- WBC + diff – if very elevated consider bacterial cause such as TB and/or alternate diagnosis such as endocarditis
- ESR/CRP – thought to be useful to support diagnosis, risk stratify, assess effectiveness of treatment (serial evaluations) and predict recurrence
- Creatinine to assess for uremia as a cause
- Troponin to help rule out ACS and myopericarditis

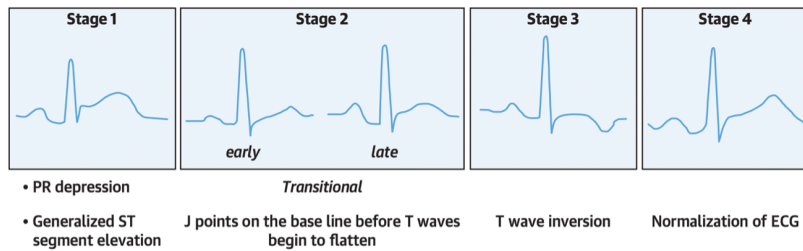
Pearl: CRP is thought to be helpful in pericarditis diagnosis, risk stratification, assessing effectiveness of treatment through serial evaluations and predicting recurrence

ECG in pericarditis – more than just diffuse ST elevation

The 4 stages of ECG changes in pericarditis

It is important to understand that the classic **diffuse ST elevation/PR depression** (stage 1) is only found in 60% of patients with pericarditis as patients may present to medical care at a more advanced stage. The temporal evolution of the 4 changes are highly variable and some patients may skip directly from stage 1 to stage 4 (normalization).

Stage	PR Segment	ST Segment	T-Wave
1 (acute)	Depression, especially in II, aVF, and V4-V6	Elevation, especially in I, V5 and V6; ST amplitude: T-wave amplitude >0.25	N/A
2	Isoelectric or depressed	Returns to isoelectric line	Amplitude decreases, inversion rare
3	Isoelectric or depressed	Isoelectric	T-wave inversion, especially in I, V5 and V6
4	Isoelectric	Isoelectric	Normal



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Pitfall: An important pitfall in ECG interpretation is failing to consider MI in a patient with chest pain and diffuse ST elevation on ECG; diffuse ST elevation can occur in MI.

Pitfall: Some patients evolve directly from Stage 1 to Stage 4 (normalization) before they present to ED, so the ECG may be normal with acute pericarditis. Do not rule out pericarditis based on a normal ECG.

Pearl: Uremic pericarditis does not cause significant inflammation of the epicardium, so the ECG and the inflammatory markers are more likely to be normal.

ECG findings to help differentiate Pericarditis from MI

No single ECG finding is diagnostic for pericarditis; all of the following findings can be seen in patients with cardiac ischemia, underlining the importance of approaching pericarditis as a diagnosis of exclusion.

- Widespread/diffuse **PR depression and/or ST elevation**
- J-point in pericarditis is usually **sharper** compared to a more blurred J point in MI
- ST elevations are more commonly convex shaped in STEMI, while **concave upwards ST elevations** are more typical of pericarditis
- If ST elevation or PR depression is present, there is typically a **preservation of the normal upright T-waves** in pericarditis (note however, that stage 3 is defined by T-wave inversions)
- ST elevation is **rarely > 5mm** in pericarditis
- **ST depressions in V1 and aVR** favor pericarditis

- **aVL ST segment** is typically elevated in pericarditis while aVL ST segment depression is highly specific for inferior MI
- **Comparison of ST elevation in leads II and III** may help differentiate STEMI from pericarditis
 - ST elevation II > ST elevation III favors pericarditis
 - ST elevation III > ST elevation II is highly suspicious for inferior STEMI
- **Spodick's sign** is seen in approximately 80% of patients with acute pericarditis (and in 29% of patients with all stages of pericarditis) and 5% of STEMIs; it is characterized by down-sloping from the T wave to the QRS segments with the terminal PR segment depressed; this is best seen in lead II and the lateral precordial leads.



ECG findings of pericardial effusion that may be associated with pericarditis:

1. Low voltages (also seen in COPD, and patients with obesity)
2. Electrical alternans

Pitfall: There are no ECG findings that are 100% sensitive for specific to differentiate pericarditis from MI underlining the importance of approaching pericarditis as a diagnosis of exclusion

ECG findings to help differentiate pericarditis from early repolarization

Early repolarization is typically a phenomenon of young, healthy, tall athletes. This population overlaps with that of pericarditis.

ECG Feature	Pericarditis	Early Repolarization
ST Elevation	STE is more evenly distributed Degree of STE is less variable May evolve over time	STE typically found in anterior/precordial leads (V2,3,4) Degree of STE can be highly variable Typically, not evolving over a short period of time
J Point	No notched/irregular J-point	Can have a notched/irregular J-point (also called fishhook, best seen in lead 4)
ST/T Wave Ratio	>0.25	<0.25
PR Segment	Depressed diffusely	Normal
T Waves	Prominent amplitude May evolve over time	Normal amplitude Typically, not evolving over a short period of time

Example: Notched J-Point in early repolarization



[Jesse McLaren's ECG Cases post on pericarditis for examples of differentiating pericarditis from MI and early repolarization](#)

PoCUS and pericarditis

Pericardial effusion found on PoCUS *supports* the diagnosis of pericarditis (60% of patients with pericarditis) but is *not diagnostic*.

The presence of a large pericardial effusion can help identify patients with acute pericarditis who are at higher risk for complications. If there are signs of **LV dysfunction**, consider myopericarditis.

To semi-quantitatively describe the size of a pericardial effusion, measure the echo-free space between the pericardial layers at end-diastole.

- **Trivial:** fluid is only seen in systole
- **Small:** <10 mm
- **Moderate:** 10-20 mm
- **Large:** 21-25mm
- **Very Large:** >25mm
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The larger the pericardial effusion, the more likely cardiac tamponade is present; however, cardiac tamponade may occur with small-moderate pericardial effusions.

Troponin – the importance of trending troponins in suspected pericarditis

Troponin may be elevated in a number of chest pain presentations including pericarditis, myocarditis, pulmonary embolism and ACS. The troponin elevation in pericarditis typically remains stable over hours whereas the troponin elevation in MI typically increases over hours in the acute phase. Only a proportion of patients with pericarditis bump their troponin, and usually to a smaller degree compared to patients with myopericarditis and myocarditis.

Inflammatory markers – their utility in pericarditis

CRP elevation can be somewhat useful to support a diagnosis of pericarditis (a CRP >3 has a sensitivity of 80% for pericarditis). An elevated CRP also suggests a higher risk of recurrence and may be helpful serially to assess the clinical course. A very high CRP increases the likelihood of myopericarditis.

Differential diagnosis of pericarditis

Infectious	Viral (common) – enterovirus, herpesvirus, adenovirus, parvovirus B19 Bacterial (mycobacterium tuberculosis is most common) Fungal/Parasitic (very rare)
Autoimmune (common)	Systemic autoimmune/inflammatory diseases – SLE, Sjogren, RA, scleroderma Systemic vasculitis Autoinflammatory diseases (FMF) Sarcoidosis IBD
Metabolic (common)	Uremia Myxedema Anorexia nervosa
Traumatic/Iatrogenic (common)	Early Onset (rare): from direct or indirect thoracic injury, or radiation Delayed Onset (common): post-MI, post-pericardiectomy, post-trauma (including iatrogenic trauma like PCI, pacemaker insertion, ablation)
Neoplastic	Primary tumor (rare): pericardial mesothelioma Secondary mets (common): lung, breast, lymphoma
Drug Related (rare)	Lupus-like syndrome (procainamide, hydralazine) Hypersensitivity with eosinophilia (penicillin)

Cardiac Tamponade – a rare life-threatening complication of pericarditis

Pericardial effusion and cardiac tamponade should be considered in all patients with pericarditis, especially those with a non-viral underlying etiology such as malignancy, TB or severe hypothyroidism.

Pearl: *The presence of a large pericardial effusion and/or cardiac tamponade increases the likelihood of a non-viral cause of pericarditis and should trigger a search for a non-viral cause.*

Cardiac tamponade is a clinical diagnosis, not an imaging one.

While the larger the pericardial effusion, the more likely cardiac tamponade, it is not the size of the effusion alone that matters. Rather, it is the pressure on the right side of the heart that is the keystone of tamponade physiology. Small effusions that collect rapidly can lead to tamponade.

Pitfall: a common pitfall in the diagnosis of cardiac tamponade is ruling out tamponade just because the pericardial effusion is small

Clinical features of cardiac tamponade

5 features occur in the majority of patients with tamponade:

1. Dyspnea (sensitivity 87-89%)
2. Tachycardia (sensitivity, 77%)
3. Pulsus paradoxus (with pericardial effusion) >10mmHg (sensitivity, 82%; LR+ 3.3), <10mm Hg (LR-0.03)
4. Elevated JVP (sensitivity, 76%)
5. Cardiomegaly on chest radiograph (sensitivity, 89%)

Pearl: When the decision to perform a pericardiocentesis needs to be made rapidly, a quick way to assess for pulsus paradoxus is to simply palpate the radial pulse for an inspiratory fall in BP rather than search for a sphygmomanometer (which are sometimes difficult to locate in the ED) and perform the more time-consuming traditional assessment for pulsus paradoxus

PoCUS findings suggestive of cardiac tamponade – scrutinize the right side of the heart and IVC

- A pericardial effusion >25mm (but smaller pericardial effusions can cause tamponade)
- **Diastolic right ventricular collapse** (high specificity)
- **Systolic right atrial collapse** (earliest sign)
- A **plethoric inferior vena cava** with minimal respiratory variation (high sensitivity)
- Exaggerated respiratory cycle changes in mitral and tricuspid valve in-flow velocities as a surrogate for pulsus paradoxus

Pearl: in patients with suspected cardiac tamponade where the diagnosis is not clear, consider a fluid bolus which may make the right heart PoCUS signs and pulsus paradoxus more obvious as well

as increase filling pressure and cardiac output (avoid if severe, pre-existing LV dysfunction)

If the diagnosis of cardiac tamponade is unclear, consider:

1. If the patient is clinically stable, a STAT **echocardiogram done by an ultrasound tech**
2. Placing an **arterial line** for continuous monitoring for pulsus paradoxus
3. Performing a pericardiocentesis which is *both diagnostic and therapeutic*

Pitfall: a common pitfall in the management of cardiac tamponade is delaying pericardiocentesis until the patient becomes hemodynamically unstable

Treatment of pericarditis – don't forget the colchicine!

The main goals of the treatment of pericarditis are

1. To reduce the acute inflammation with NSAIDs and
2. To prevent recurrence, constrictive pericarditis and long term morbidity with colchicine.

Be sure to include colchicine because it has been shown to prevent the most common complication of pericarditis – recurrent pericarditis (as apposed to NSAIDs which have not been shown to alter the natural history of acute pericarditis).

Outpatient management of pericarditis with a presumed idiopathic/viral cause

1. **Restrict strenuous physical activity** which may trigger recurrence of symptoms
2. **Ibuprofen** 600mg TID or **indomethacin** 50mg TID (for acute inflammation)
3. **Colchicine 0.6mg** daily for <70kg, bid for ≥70kg (to help prevent recurrence)
 - Evidence suggests reduction of recurrence rate by up to 50%
 - May also facilitate rapid resolution of symptoms
 - Warn patients of GI upset with colchicine
4. **PPI** for those at high risk of UGIB (especially if using indomethacin, which has a high incidence of gastritis)

Duration of treatment with anti-inflammatories is based on resolution of symptoms and normalization of CRP (in uncomplicated cases, generally 1-2 weeks followed by taper). **Colchicine treatment** will sometimes continue for months. Ensure follow-up within a week and inform them that even if they start to feel better, they should continue medications until follow-up to prevent recurrence.

Corticosteroids – second line therapy; can lead to rebound pericarditis when it is stopped, and is associated with a higher risk for recurrent pericarditis

Treatment for patients with pericarditis due to non-viral causes:

- **Uremia-related Pericarditis** – consider dialysis +/- pericardiocentesis; NSAIDs and colchicine are generally contraindicated in patients with pericarditis who have severe renal impairment
- ### Treatment of patients with pericarditis who are taking anticoagulants/antiplatelet agents
- These patients are at high risk for GI bleed if they require NSAIDs as well as hemorrhagic pericarditis requiring pericardial window; treatment decisions should be made in concert with cardiology/hematology.
- ### Disposition for patients with pericarditis
- Patients who are otherwise healthy with uncomplicated presumed viral pericarditis, who have mild symptoms are generally safe to be discharged home with cardiology follow-up, medications, and instructions not to exercise (this is the majority of patients)
 - Patients with pericarditis from a non-viral cause should be considered for admission and further workup of the etiology as these patients are more likely to develop complications and may require specific time-sensitive treatment
 - **Risk factors for poor prognosis** which should prompt consideration for admission include:
 - Fever (>38C)
 - Failure to respond within 7 days to treatment
 - Large pericardial effusion (>20mm)
 - Oral anticoagulants
 - Myocarditis (elevated troponin)
 - Immunosuppression

Take Home Points for acute pericarditis and cardiac tamponade

- Pericarditis is a **diagnosis of exclusion** after ruling out the big chest pain killers; avoid premature closure!
- No single clinical feature is diagnostic of pericarditis; symptoms over many days, young age, presence of a viral prodrome, sharp pleuritic positional chest pain, presence of a fever, a triphasic pericardial friction rub should all be considered contributory
- **CRP** may be helpful to risk stratify patients and predict recurrence; **a troponin or serial troponins** should be considered to help rule out ACS and myocarditis
- The main pitfall with ECG interpretation is failure to consider MI as a cause of diffuse ST elevation
- There are **4 stages of ECG changes in pericarditis**, and the progression through these stages is variable; patients with pericarditis can have a normal ECG
- There are a number of features on ECG that can help to distinguish between MI and pericarditis and benign early repolarization, but no single feature is diagnostic
- Important pitfalls in the diagnosis and management of cardiac tamponade include delaying pericardiocentesis and assuming there is no tamponade when the size of the effusion is small or moderate
- In time-sensitive situations palpation (rather than using a BP cuff) for pulsus paradoxus is adequate for helping to make the diagnosis of cardiac tamponade rapidly
- Treatment of uncomplicated viral acute pericarditis includes NSAIDs as well as more prolonged treatment with colchicine to prevent recurrence
- Consideration should be given for gastric protection with a PPI for patients receiving high dose NSAIDs
- Patients with uncomplicated, viral pericarditis can usually be safely discharged home with appropriate follow-up, discharge

instructions NSAIDs and colchicine; patients with an uncertain etiology or with risk factors for poor prognosis should be considered for admission for further work up and consideration for pericardial window, dialysis etc.

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