Emergency Department's (ED) Case of the Month is a series of articles discussing topics important to family physicians working in the ED. This department covers selected points to help you avoid pitfalls and improve patient care in the ED. Submissions and feedback can be sent to diagnosis@sta.ca.



All That Glitters is Not Gold

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The emergency department (ED) nurse asks you to see a 61-year-old man who ■ may be a candidate for thrombolysis for an acute myocardial infarction. He has severe chest pain and ST segment elevation in his inferior leads. She hands you the electrocardiogram (ECG) (Figure 1).



Figure 1

Patient Stats

- He complains of severe chest pain for the past four hours, which radiates into his left shoulder. He feels nauseated and short of breath. He states that the pain is worse with deep inspiration.
- The patient tells you he has been feeling "flu-like" for the past two weeks with intermittent low-grade fevers and muscle aches and pains.
- On exam, he appears to be quite uncomfortable and is diaphoretic. Aside from a heart rate of 113 beats per minute, his vital signs are normal.
- · Cardiovascular exam, including heart sounds, are unremarkable. Auscultation of his lungs are clear.
- A portable chest X-ray shows no significant abnormalities.

Ouestions:

- 1. What is the criteria for administering thrombolytics to a patient with acute chest pain?
- 2. What are the contraindications to thrombolysis?
- 3. What is your interpretation of the ECG?
- 4. What is the etiology of this patient's condition?
- 5. How should this patient be treated?

Answers:

1. What are the criteria for administering thrombolytics to a patient with acute chest pain?

Intravenous thrombolytic therapy is clearly indicated for patients presenting within six hours of onset of symptoms who meet the following criteria:

- Have a clinical history of **ischemic** chest pain.
- ST segment elevation of at least 1 mm in two contiguous leads or new left bundle branch block.
- No contraindications to systemic thrombolytic therapy.

Patients receiving thrombolytic therapy six to 12 hours after onset of symptoms derive some survival benefit, but less than patients who present early.

2. What are the contraindications to thrombolysis?

Listed contraindications vary slightly between institutions and associations, however, the commonly listed ones include: The three "A's" of absolute contraindications to thrombolysis: "active (non-menstrual) internal bleed-

Practice pointer

The three "A's" of absolute contraindication to thrombolysis are :

- Active (non-menstrual) internal bleeding
- Aortic dissection
- 3. Acute pericarditis

ing, aortic dissection, and acute pericarditis." Relative contraindications (where the risk of treatment must be weighed against the benefits), include head trauma, other significant trauma or surgery to a non-compressible site within the previous four weeks, ischemic stroke or neurosurgery within six months, previous intracranial bleeding, known cerebral aneurism or neoplasm, severe uncontrolled hypertension (systolic blood pressure greater than 200 mmHg or diastolic blood pressure greater than 110 mmHg), or active peptic ulcer

disease. The use of anticoagulants in therapeutic doses (international normalised rate greater than 2 or 3), known bleeding diathesis, and pregnancy are also included by some authorities as contraindications. Unless you are making these types of decisions frequently, it would be wise to discuss thrombolysis of patients with relative contraindications with a consultant

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before proceeding. Time, of course, is of the essence, because any delay in administration of the thrombolytic increases the risk:benefit ratio of the therapy.

3. What is your interpretation of this ECG?

The ECG shows a sinus tachycardia at a heart rate of 112 beats per minute. There is diffuse upwardly concave ST segment elevation in leads V2-6, II, III and AVF as well as PR segment depression, most prominently seen in lead II. This is classic for acute pericarditis.

Acute pericarditis can be differentiated electrocardiographically from acute myocardial infarction by the diffuse upwardly concave ST segment elevations seen in the former. A further clue pointing towards a

Table 1 ECG Changes During Pericarditis

Characteristic ECG changes occuring throughout the clinical course of pericarditis have been divided into four stages:

Stage 1: Global ST segment elevation as above.

Stage 2: The ST segment returns to baseline and the T wave amplitude decreases.

Stage 3: The T wave inverts in those leads which previously had ST segment elevation.

Stage 4: Resolution of the repolarisation abnormalities.

_{таые 2} Etiology of Pericarditis

Idiopathic Infection	
Viral	Bacterial
Coxsackie B	Tuberculosis
Cytomegalovirus	Staphylococcus
• Echo virus	Mycoplasma
• HIV	Tickettsia
	Fungal
	Parasitic
Immunological Mediated Disease	Trauma Drugs
Postinfectious	Procainamide
Postcardiac injury syndrome	Hydralazine
• e.g. Dressler's syndrome	Methyldopa, warfarin
Autoimmune disorders	Uremia
Rheumatoid arthritis	Radiation
Connective tissue diseases	Neoplasm

diagnosis of pericarditis is the absence of reciprocal ST segment depression typically seen with patients experiacute encing an myocardial infarction (Table 1). Patients with acute pericarditis typically present with a preceeding history of viral symptoms and pleuritic chest pain, although these may be absent.

4. What is the etiology of this patient's condition?

This case is most likely of viral origin (Table 2).

5. How should this patient be treated?

Like any patient in the ED, the ABC's (Airway, Breathing and Circulation) should be initially assessed and stabilised. The most dramatic complica-

Acute pericarditis should be suspected in ECG findings of diffuse upwardly concave ST segment elevations without reciprocal ST depression.

tion of acute pericarditis is cardiac tamponade with resultant hypotension and cardiovascular collapse. Cardiac tamponade is diagnosed clinically or by ultrasound. If cardiac tamponade is suspected in the hemodynamically unstable patient, pericardiocentesis can be performed in an emergency.

Once stablised, treatment is aimed at the underlying etiology. In cases of idiopathic or presumed viral pericarditis, patients can be treated with non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen or indomethacin. If NSAIDs are ineffective, treatment with corticosteroids or colchicine can be considered. Although most patients will recover fully, 15% to 20% will have a recurrence of pericarditis.

In the rare case that bacterial pericarditis is suspected, appropriate antibiotics should be started and referral for consideration of surgical drainage should be made. D_k