Electrocardiographical case. ST elevation: is this an infarct?

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CLINICAL PRESENTATION

A 25-year-old man presented with complaints of chest pain and fever. A 12-lead electrocardiogram (ECG) was performed (Fig. 1). What is the diagnosis?
ECG INTERPRETATION
ECG (Fig. 1) showed concave ST segment elevation in leads I, II, III, aVF as well as V4, V5 and V6. Q waves were not seen and there was PR depression present in leads II, V5 and V6. The PR segment in aVR was elevated. The R waves were well preserved.

DIAGNOSIS
Pericarditis.

CLINICAL COURSE
The patient had a pericardial rub on auscultation. Erythrocyte sedimentation rate was elevated and cardiac enzymes were negative. The patient was admitted to the cardiology unit for observation. He was well when discharged one week later.

DISCUSSION
Pericarditis is a syndrome caused by inflammation of the pericardium. This inflammatory response causes increased vascular permeability, vasodilation and transudation of fluid into the pericardial space. There are many different aetiologies (Fig. 2), the more common causes being idiopathic, viral, tuberculous and neoplastic. Others are listed in Table I (1-6).

Patients typically present with complaints of a sharp central chest pain that is described to be worse with inspiration and recumbency, relieved by sitting up and leaning forwards (7). The pain may radiate to the ridge of the trapezius, shoulder, arm or epigastrium. Non-specific symptoms that have been described include fever, hiccups, hoarseness of voice, palpitations, cough, nausea and vomiting (7).

The pathognomonic finding on clinical examination is a pericardial friction rub that classically corresponds to the three phases of atrial systole, ventricular systole and diastole. This is best heard with the patient in exhalation. However, all three components are heard less than 50% of the time (8).

The ECG is very useful in diagnosing acute pericarditis. Abnormalities have been reported in up to 90% of cases (9). These ECG changes are purported to be due to superficial myocardial inflammation (10). The most sensitive finding has been that of diffuse ST segment elevation, which reflects the abnormal repolarisation as a result of pericardial inflammation. Other conditions must also be considered in the differential diagnosis of pericarditis on ECG (Table II).

The two most common conditions that are confused with that of pericarditis based on ECG findings include that of an acute myocardial infarction and early repolarisation, which is a normal variant on the ECG usually occurring in young black males.
Early repolarisation is distinguished by the absence of symptoms in the patient with failure to evolve through the four stages of ECG changes as first described by Spodick(14). In addition, the ST segments in normal variants are limited to the praecordial leads with elevation of the ST segment in lead V1, an isoelectric ST segment in lead V6, notching of the terminal aspect of the QRS complex and a shift to baseline of the ST segments with exercise(12,15). Ginzton and Laks (16) also noted that a ST/T ratio of >0.25 in lead V6 on ECG discriminated patients with acute pericarditis from normal variants. The various distinguishing features are listed in Table III.

The classical ECG changes of pericarditis have been described into four stages (Table IV), although progression through all four stages occurs in less than 50% of patients.

The pattern of ST segment elevation is important in distinguishing pericarditis from other diseases of the myocardium. Distinct features seen on ECG in patients with pericarditis include:

1. Concave ST segment elevation.
2. Widespread ST segment elevation not corresponding to any specific arterial territory.
3. Absence of reciprocal changes and Q waves.
4. Possible presence of low voltages.

Further investigations include laboratory markers such as C-reactive protein, erythrocyte sedimentation rate and leukocyte count which are usually elevated with pericarditis(7). Some have also reported

Table I: Causes of acute pericarditis.

<table>
<thead>
<tr>
<th>Idiopathic origin</th>
<th>Drugs</th>
<th>Irradiation</th>
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<tbody>
<tr>
<td>Viral infection</td>
<td></td>
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<tr>
<td>Coxsackie virus A and B</td>
<td>Procarbazine</td>
<td></td>
</tr>
<tr>
<td>Echoviruses</td>
<td></td>
<td></td>
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<tr>
<td>Adenoviruses</td>
<td></td>
<td></td>
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<tr>
<td>Human immunodeficiency virus (HIV)</td>
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<td></td>
</tr>
<tr>
<td>Bacterial and purulent infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Streptococcus suis</td>
<td>Ureaemia (dialysis-related)</td>
<td></td>
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<tr>
<td>Mycoplasma pneumoniae</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaerobic bacteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gram-negative species</td>
<td></td>
<td></td>
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<tr>
<td>Fungal infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Histoplasma capsulatum</td>
<td></td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td>Aspergillus species</td>
<td></td>
<td>Sarcoaidosis</td>
</tr>
<tr>
<td>Mycobacterial infection</td>
<td></td>
<td>Scleroderma</td>
</tr>
<tr>
<td>Mycobacterium tuberculosis</td>
<td></td>
<td>Idiopathic thrombocytopenic purpura</td>
</tr>
<tr>
<td>Rickettsial infection</td>
<td></td>
<td>Amyloid</td>
</tr>
<tr>
<td>Parasitic infection</td>
<td></td>
<td>Aortic dissection</td>
</tr>
</tbody>
</table>

Table II. Differential diagnosis of pericarditis on ECG(9,13).

- Transmural myocardial infarction
- Early repolarisation
- Myocarditis
- Pulmonary embolism
- Cerebrovascular accident
- Hyperkalaemia
- Ventricular aneurysm
- Left bundle branch block

Table III. Comparison of ECG changes associated with acute pericarditis, myocardial infarction and early repolarisation(9,13).

<table>
<thead>
<tr>
<th>ECG finding</th>
<th>Acute pericarditis</th>
<th>Myocardial infarction</th>
<th>Early repolarisation</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST-segment shape</td>
<td>Concave upward</td>
<td>Convex upward</td>
<td>Concave upward</td>
</tr>
<tr>
<td>Q waves</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Reciprocal ST-segment changes</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Location of ST-segment elevation</td>
<td>Limb and precordial leads</td>
<td>Area of involved artery</td>
<td>Precordial leads</td>
</tr>
<tr>
<td>ST/T ratio in lead V6</td>
<td>&gt;0.25</td>
<td>NA</td>
<td>&lt;0.25</td>
</tr>
<tr>
<td>Loss of R-wave voltage</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>PR-segment depression</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
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</table>
Table IV. ECG changes in acute pericarditis: the four stages(9,11-13).

<table>
<thead>
<tr>
<th>Stage</th>
<th>Timing</th>
<th>ECG changes</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>First few days (up to two weeks)</td>
<td>• Diffuse concave upward ST segment elevation in leads I, II, aVF and V2 - 6 without reciprocal ST segment depression</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• ST segment elevation is higher in lead II than lead III with ST segment depression in aVR or V1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• PR segment depression best seen in all leads except aVR and V1 (9) indicative of subepicardial atrial injury</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• PR segment elevation seen in aVR is characteristic (14)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Low voltages in leads</td>
</tr>
<tr>
<td>2</td>
<td>Days to weeks</td>
<td>• ST segments return to baseline</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• T wave flattening</td>
</tr>
<tr>
<td>3</td>
<td>Second to third week</td>
<td>• T wave inversion</td>
</tr>
<tr>
<td>4</td>
<td>Weeks to months (up to three months)</td>
<td>• Gradual resolution of T wave inversion(10)</td>
</tr>
</tbody>
</table>

raised Troponin I levels in such patients(19,20). Chest radiographs may be useful in detecting an enlarged cardiac silhouette which could reflect a large pericardial effusion. This would have to be confirmed via echocardiography. Routine pericardiocentesis, pericardial biopsy and pericardecctomy are not routinely performed as they have a low diagnostic yield. These are reserved typically for therapeutic indications or rarely for diagnosis of patients with prolonged illness suspicious of tuberculosis(21).

Idiopathic causes of pericarditis are self-limited and usually resolve within two to six weeks. Potential complications include recurrent pericarditis, cardiac tamponade and constrictive pericarditis. Besides treatment of the underlying aetiology, therapeutic measures involve pain relief and prevention of further inflammation with non-steroidal anti-inflammatory drugs (NSAIDS), bed rest and supportive treatment. Antibiotics are directed toward those with purulent or tuberculous pericarditis. Corticosteroids and immunosuppressant therapy are only reserved for patients whose symptoms are refractory to NSAIDS(7).

REFERENCES
Question 1. Common causes of pericarditis include:
(a) Viral infections such as Coxsackie virus A and B. ☐ ☐
(b) Tuberculosis. ☐ ☐
(c) Neoplastic. ☐ ☐
(d) Idiopathic. ☐ ☐

Question 2. The following statements regarding pericarditis are true:
(a) Pericarditis is characterised by chest pain that is worse on sitting up and leaning forward. ☐ ☐
(b) The pericardial rub is the pathognomonic finding of pericarditis. ☐ ☐
(c) ST segment depression is the most sensitive finding in acute pericarditis. ☐ ☐
(d) 90% of cases of pericarditis have an abnormal ECG. ☐ ☐

Question 3. Differential diagnosis of pericarditis on ECG include:
(a) Non-ST segment elevation myocardial infarction. ☐ ☐
(b) Pulmonary embolism. ☐ ☐
(c) Left bundle branch block. ☐ ☐
(d) Ventricular aneurysm. ☐ ☐

Question 4. The four stages of acute pericarditis include:
(a) Concave upward ST segment elevation. ☐ ☐
(b) Return of ST segments with T wave flattening. ☐ ☐
(c) Peaked T waves. ☐ ☐
(d) Return of T waves to baseline. ☐ ☐

Question 5. Other markers that may be elevated in acute pericarditis include:
(a) C-reactive protein. ☐ ☐
(b) Leukocyte count. ☐ ☐
(c) Troponin I. ☐ ☐
(d) Erythrocyte sedimentation rate. ☐ ☐

Doctor's particulars:
Name in full: _______________________________________________________________________________________
MCR number: ______________________________________ Specialty: ______________________________________
Email address: _______________________________________________________________________________________

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2. Indicate your responses by marking the “True” or “False” box ☑
3. Fill in your professional particulars.
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1. Log on at the SMJ website: URL <http://www.sma.org.sg/cme/smj> and select the appropriate set of questions.
2. Select your answers and provide your name, email address and MCR number. Click on “Submit answers” to submit.


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4. Passing mark is 60%. No mark will be deducted for incorrect answers.
5. The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.