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CME Article

Electrocardiographical case. A young man with chest pain

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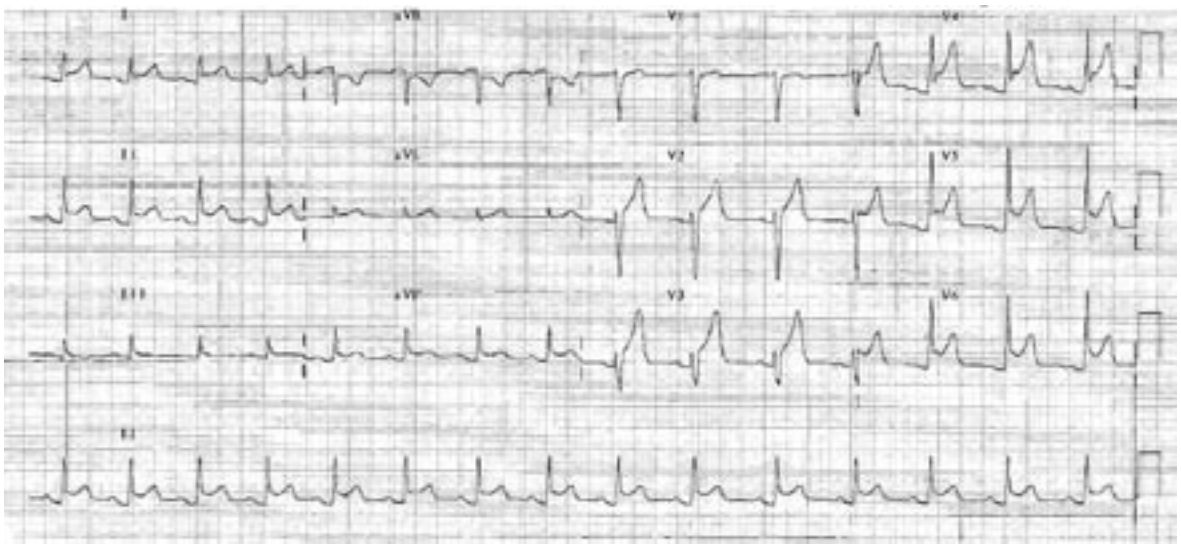

Fig. 1 ECG at initial presentation.

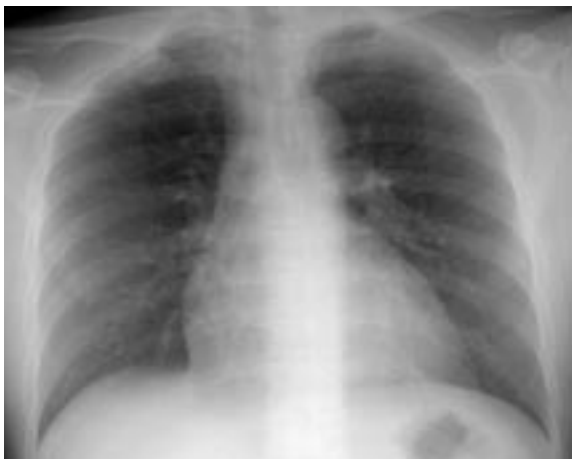

Fig. 2 Frontal chest radiograph at initial presentation.

CLINICAL PRESENTATION

A 31-year-old Chinese man presented to the Emergency Department with complaint of acute chest pain for three days. Other than smoking, he had no other cardiovascular risk factors. Over the past two months, he had similar episodes of chest pain, which was evaluated at a public hospital, with negative results. Physical examination revealed an oral temperature of 37.0 degrees Celsius, pulse rate of 80 beats per minute and blood pressure of 104/67 mmHg. Auscultation revealed normal heart sounds with no murmur or additional sounds. Jugular venous pressure was not elevated. Chest auscultation revealed vesicular breath sounds. Electrocardiography (ECG) (Fig. 1) and chest radiograph (Fig. 2) were performed. What is your diagnosis?

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DIAGNOSIS

Acute pericarditis.

ECG INTERPRETATION

The electrocardiogram shows sinus rhythm, with widespread upward concave ST segment elevations in leads II, III, AVF, V2 to V6, I and AVL. The ST elevation was maximal in lead II rather than lead III. In addition, there was widespread PR segment depression in these leads. Correspondingly, there was PR segment elevation with ST segment depression in AVR. These findings suggest acute pericarditis.

CLINICAL COURSE

Investigations revealed the following results: total white blood cell count of 13.1 X 10^9/L, neutrophils 75.7%, monocyte 11.2%, lymphocytes 9.6%, and haemoglobin 15.8 g/dL. Cardiac enzymes were not elevated. Transthoracic echocardiography revealed a normal left ventricular size and function, with an ejection fraction of 56%, and a small pericardial effusion. No regional wall motion abnormalities or valvular pathology were demonstrated. Serological and immunological studies were negative. Rheumatoid factor was found to be slightly elevated at 11.1 U/mL (<10.3 U/mL). He was started on high dose aspirin 300 mg tds and rofecoxib 25 mg OM. His symptoms resolved with therapy and he was well when discharged. He was reviewed in the outpatient clinic four weeks post-discharge. He did not complain of further symptoms and ECG changes had reverted to normal.

The patient subsequently returned to the Emergency Department two weeks after the outpatient clinic appointment, with recurrence of chest pain similar to the first presentation. Physical examination revealed an oral temperature of 37.2 degrees Celcius, pulse rate of 115 per minute, and blood pressure of 127/77 mmHg. Heart sounds were normal, and no murmur nor additional sounds were heard. Jugular venous pressure was noted to be elevated at 2 cm but the Kussmaul sign and pulsus paradoxus were absent. His ECG (Fig. 3) and chest radiograph (Fig. 4) were repeated. What complication has developed?

DIAGNOSIS

Pericardial effusion.

ECG INTERPRETATION

The electrocardiogram showed sinus tachycardia and when compared with his earlier ECG, the QRS complexes had smaller voltages. There was no electrical alternans suggestive of a large pericardial effusion or tamponade on the ECG. Earlier ECG changes of pericarditis have resolved and there were no residual T wave inversions (stage III change of pericarditis). The subtle ECG findings of reduced QRS voltages together with the chest radiographical findings of cardiomegaly suggest the development of pericardial effusion.

CLINICAL COURSE

An urgent transthoracic echocardiogram showed a large circumferential pericardial effusion (3.5 cm – 3.9 cm), with no echocardiographical features of pericardial tamponade, i.e. absence of early diastolic right ventricle collapse, late diastolic right atrium collapse, plethora of inferior vena cava with blunted respiratory response and abnormal ventricular septal motion. Pericardiocentesis was performed and 800 ml of serous fluid was withdrawn. Analysis of

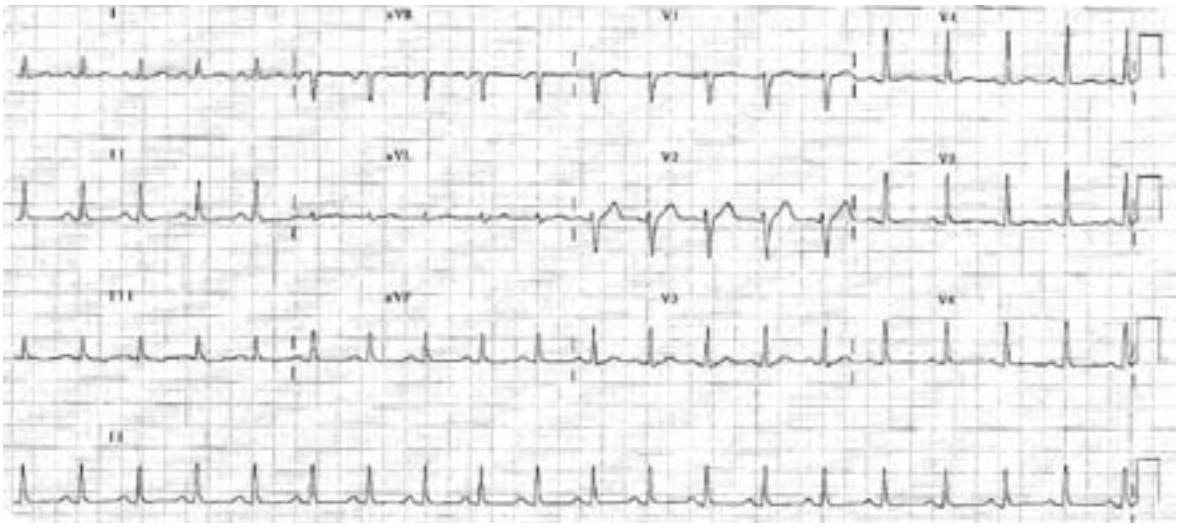

Fig. 3 ECG at second presentation.

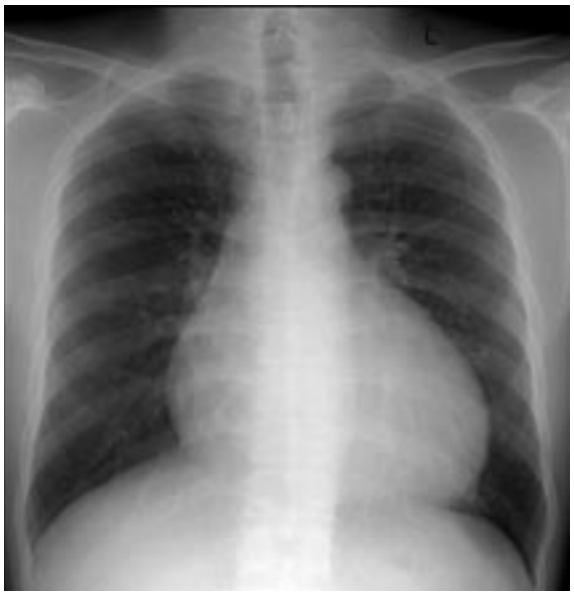

Fig. 4 Chest radiograph at second presentation.

pericardial fluid revealed total protein 66 g/L (serum protein 71 g/L), LDH <20 U/L (serum LDH 209 U/L). Cytological analysis showed some lymphocytes and polymorphs with no malignant cells. Acid-fast bacilli staining and tuberculosis (TB) culture of the fluid were negative. Blood investigations showed ESR of 27 mm/hour, CRP of 49 mg/L, total white blood cell count of 10.2 X 10^9/L, differential count consisting of neutrophils 70%, lymphocytes 10% and eosinophils 13%, and rheumatoid factor of 49 mg/L. Sputum acid fast bacilli and tuberculosis cultures were negative.

He was started on prednisolone 30 mg OM together with empirical anti-TB medication, despite the negative TB cultures. His clinical condition improved with pericardiocentesis and he was well when discharged. Subsequent outpatient follow-up revealed decreasing trends of ESR, CRP and rheumatoid factor, and serial transthoracic echocardiograms revealed no reaccumulation of pericardial effusion and no development of constrictive pericarditis. The patient was clinically asymptomatic during follow-up over the subsequent six months.

DISCUSSION

Classical ECG changes of acute pericarditis include widespread upward concave ST segment elevation and PR segment depression. ECG abnormalities may go through four phases of evolution: diffuse upward concave ST segment elevation and PR segment depression (stage I), normalisation of the ST and PR segments (stage II); widespread T-wave inversions (stage III) and normalisation of T waves (stage IV)[1-4].

Table I. Clinical findings of acute pericarditis and acute myocardial infarction

	Acute pericarditis	AMI
Chest pain		
Quality of chest pain	Sharp stabbing	Heaviness or pressure
Pleuritic pain	Present	Absent unless peri-infarction pericarditis develops
Radiation of pain	Trapezius ridge common.	Shoulders, jaw, neck, arms; not trapezius ridge pain
Posture	Worse on lying, better on leaning forward	No change with posture
Nitrates	No relief	Usually relief
Physical findings		
Pericardial rub	Present	Absent unless peri-infarction pericarditis develops
S3, S4	Absent	May be present
ECG findings		
ST segment elevation	Diffuse elevation; concave; no reciprocal changes	Localised deviation; usually convex; reciprocal changes present
PR segment depression	Frequent	Rare
Abnormal Q waves	None unless previous infarct	Presence in late infarction
T waves	Inverted after ST elevation return to baseline	Inverted when ST segments still elevated
Arrhythmia	Uncommon	Common
Conduction abnormalities	Uncommon	Common
Investigations		
Cardiac enzymes	Normal or elevated	Elevated

Differentiating ECG changes of pericarditis from acute myocardial infarction (AMI), which also show ST segment elevation, is of utmost importance because thrombolysis used for acute myocardial infarction, when used inappropriately in a patient with pericarditis, can lead to disastrous consequences. The clinical and ECG features that could aid in reaching the correct diagnosis are outlined in Table I[5]. Note that the presence of troponin elevation is not a good indicator of AMI as it may be elevated in acute pericarditis as well, especially in young patients and in the male gender. Elevation of troponins in pericarditis, unlike acute coronary syndrome, does not have negative prognostic implications[6].

Causes of pericarditis are myriad. Other than idiopathic pericarditis, the most common

cause is viral infection. Other causes include transmural infarction, other infective agents like tuberculosis, aortic dissection, blunt cardiac trauma, neoplasm, irradiation, uraemia, cardiac surgery, autoimmune diseases and drugs like doxorubicin and hydralazine[7,8]. Evaluation of possible acute pericarditis involves obtaining history suggestive of pericarditis (e.g. characteristics of chest pain), physical examination to detect a pericardial rub, and the presence of classical ECG changes, as discussed above. Further investigations such as chest radiographs, transthoracic echocardiography, serological testing, pericardiocentesis and pericardial biopsy are additional investigations that may be useful to evaluate for specific cause of pericarditis and for detection of complications such as pericardial effusion or cardiac tamponade.

Specific treatment depends on the causative factors. For patients with idiopathic pericarditis, non-steroidal anti-inflammatory agents are the drugs of choice. High-dose aspirin, indomethacin and ibuprofen have been used for this purpose, as in our patient[9]. The use of colchicine has not been tested in randomised controlled trials and is mainly used in recurrent pericarditis not responding to NSAID treatment. Glucocorticoid therapy such as prednisolone should be restricted to recurrent pericarditis not responding to combination NSAID and colchicine therapy, or in suspected or proven TB pericarditis.

ABSTRACT

A 31-year-old Chinese man presented with complaint of acute chest pain. 12-lead electrocardiogram (ECG) showed sinus rhythm, with widespread upward concave ST segment elevations. The ECG changes along with a history of acute chest pain in a young man with minimal coronary risk factors are suggestive of acute pericarditis. He subsequently developed a pericardial effusion. Diagnosis, treatment and complications of acute percarditis are discussed.

Keywords: acute pericarditis, chest pain, electrocardiogram, ST segment elevation.

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REFERENCES

1. Ginzton LE, Laks MM. The differential diagnosis of acute pericarditis from the normal variant: new electrocardiographic criteria. Circulation 1982; 65:1004-9.
2. Spodick DH. Diagnostic electrocardiographic sequences in acute pericarditis: significance of PR segment and PR vector changes. Circulation 1973; 48:575-80.
3. Surawicz B, Lasseter KC. Electrocardiogram in pericarditis. Am J Cardiol 1970; 26:471-4.
4. Wang K, Asinger RW, Marriott HJL. ST segment elevation in conditions other than acute myocardial infarction. N Engl J Med 2003; 349:2128-35.
5. Spodick DH. Acute pericarditis: current concepts and practice. JAMA 2003; 289:1150-3.
6. Imazio M. Demichelis B, Cecchi E, et al. Cardiac troponin I in acute pericarditis. J Am Coll. 2003; 42:2144-8.
7. Zayas R, Anguita M, Torres F, et al. Incidence of specific etiology and role of methods for specific etiologic diagnosis of primary acute pericarditis. Am J Cardiol 1995; 75:378-82.
8. Permanyer-Miralda G, Sagrista-Sauleda J, Soler-Soler J. Primary acute pericardial disease: a prospective series of 231 consecutive patients. Am J Cardiol 1985; 56:623-30.
9. Imazio M, Demichelis B, Parrini I, et al. Day-hospital treatment of acute pericarditis: a management program for outpatient therapy. J Am Coll Cardiol 2004; 43:1042-6.

SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 200605B)

	True	False
Question 1: The following are the ECG features that are characteristic of acute pericarditis:		
(a) PR segment depression.	❑	❑
(b) Widespread ST segment elevation.	❑	❑
(c) Abnormal Q waves.	❑	❑
(d) Convex ST segment elevation.	❑	❑
Question 2: Which of the following statements are true about clinical features characteristic of acute pericarditis?		
(a) Pleuritic chest pain.	❑	❑
(b) Chest pain that changes with posture.	❑	❑
(c) Relief with sublingual nitrates.	❑	❑
(d) Crushing chest tightness.	❑	❑
Question 3: Which of the following statements are true with regard to treatment of acute pericarditis?		
(a) Glucocorticoids are the initial drug of choice.	❑	❑
(b) Recurrent pericarditis can be treated with colchicines.	❑	❑
(c) Predisposing factors such as drugs should be discontinued.	❑	❑
(d) Low-dose aspirin is as effective as high-dose aspirin in the treatment of acute pericarditis.	❑	❑
Question 4: Evaluation for acute pericarditis should include the following in all cases:		
(a) Physical examination.	❑	❑
(b) Careful history-taking.	❑	❑
(c) ECG.	❑	❑
(d) Pericardial biopsy.	❑	❑
Question 5: The following are causes of acute pericarditis:		
(a) Acute myocardial infarction.	❑	❑
(b) Viral infection.	❑	❑
(c) Uraemia.	❑	❑
(d) SLE.	❑	❑

Doctor's particulars:

Name in full:_____

MCR number:_____ Specialty:_____

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Submission instructions:

A. Using this answer form

1. Photocopy this answer form.
2. Indicate your responses by marking the "True" or "False" box ☑
3. Fill in your professional particulars.
4. Post the answer form to the SMJ at 2 College Road, Singapore 169850.

B. Electronic submission

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.

Deadline for submission: (May 2006 SMJ 3B CME programme): 12 noon, 25 June 2006

Results:

1. Answers will be published in the SMJ July 2006 issue.
2. The MCR numbers of successful candidates will be posted online at http://www.sma.org.sg/cme/smj by 20 July 2006.
3. All online submissions will receive an automatic email acknowledgment.
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.