CME Article

Electrocardiographical case. Acute severe chest tightness.

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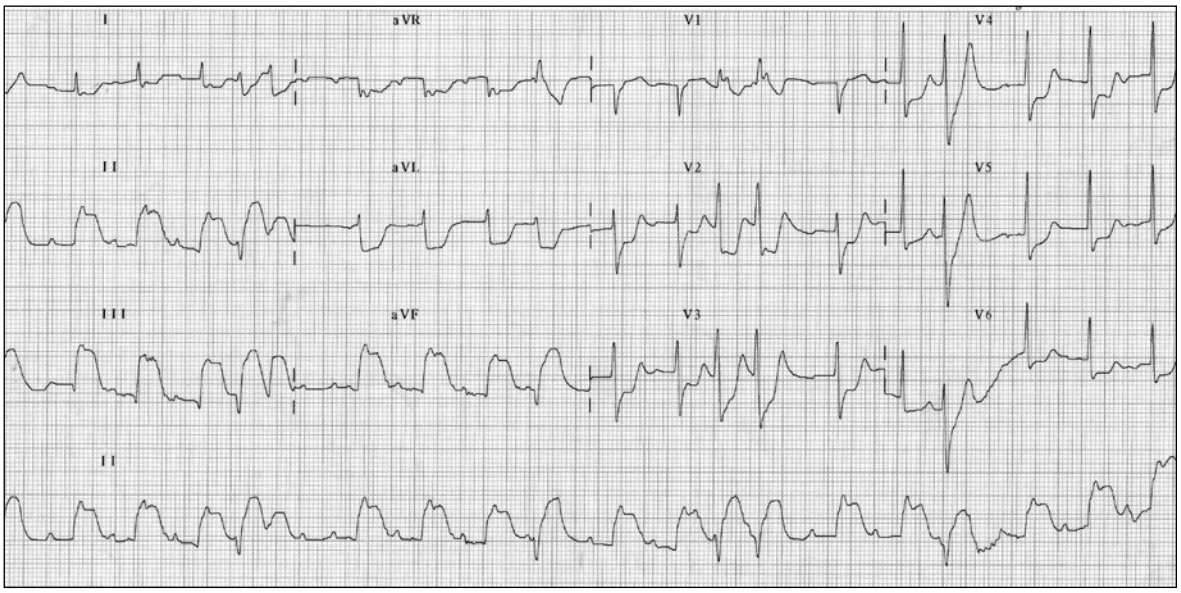

Fig. 1 12-lead ECG.

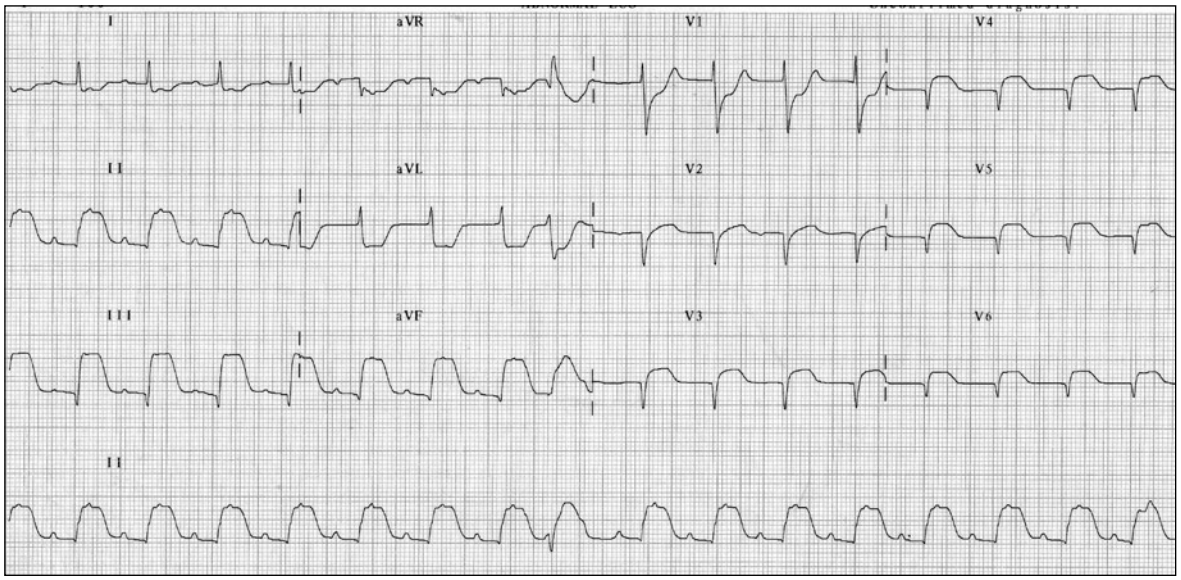

Fig. 2 Right-sided ECG

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CLINICAL PRESENTATION

A 56-year-old man developed sudden onset of severe chest tightness in the morning. His only significant history is that of smoking for the past 20 years. 12-lead electrocardiography (ECG) was done at the emergency department (Fig. 1). Right-sided ECG (Fig. 2) was also performed. What do these show? What is your diagnosis?

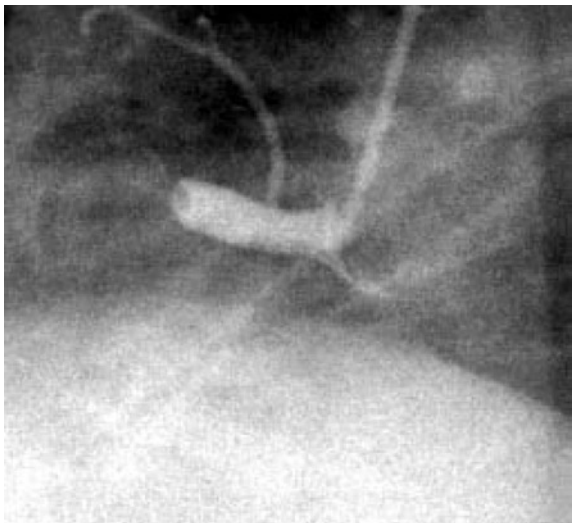

Fig. 3 Coronary angiogram shows an occluded proximal RCA.

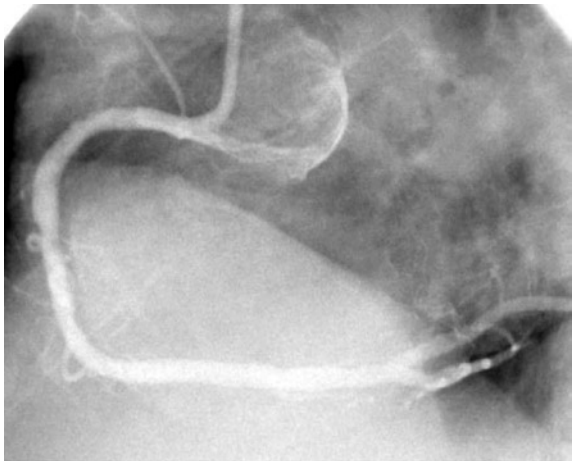

Fig. 4 Coronary angiogram taken post-balloon angioplasty and stenting to a dominant RCA.

DIAGNOSIS

Acute inferior, posterior and right ventricular ST-elevation myocardial infarction.

ECG INTERPRETATION

The 12-lead ECG showed clear ST elevation in the inferior leads II, III, and aVF. There were also deep ST depressions in leads V2 to V6 (Fig. 1). There were frequent ventricular ectopics and ventricular couplets, with variable first and second degree atrio-ventricular (AV) nodal blocks. There was a discordant ST elevation seen in lead V1, suggesting right ventricular infarction as well. Right-sided ECG showed clear ST elevation in RV4-V6, demonstrating right ventricular infarction (Fig. 2). ST elevation of >1 mm in lead RV4 is diagnostic of right ventricular wall infarction.

Fig. 5 Photograph shows containers with the extracted intracoronary thrombi.

CLINICAL COURSE

The patient developed ventricular fibrillation while waiting at the general practitioner's clinic but was fortunately successfully cardioverted by the paramedics who arrived at the clinic when the patient collapsed. Ultimately, for this patient with ST-elevation myocardial infarction, prompt recognition and early reperfusion was crucial. He underwent successful acute percutanous angioplasty and stenting to a proximal right coronary artery (RCA) occlusion (Figs. 3 & 4). Large amounts of intracoronary clots were removed via a thrombectomy device (Fig. 5). An intra-aortic balloon pump was inserted for management of initial associated cardiogenic shock. Recovery was uneventful and he was discharged after seven days. Pre-discharge ECG showed mildly-impaired left ventricular function.

DISCUSSION

The left- and right-sided ECG clearly demonstrates the finding of ST elevation, indicating inferior, posterior and right ventricular infarction. In 90% of individuals, the RCA is dominant, defined as giving rise to the posterior descending artery. Its occlusion gives rise to injury in the basal and middle sectors of the inferior quadrant of the left ventricle, and thus an "inferior" myocardial infraction. The ST elevation would be seen in the three limb leads II, III and aVF. An inferior myocardial infarction can also be due to occlusion of the left circumflex artery (LCX). When the RCA is dominant, the LCX supplies only the left ventricular free wall between the distributions of the left anterior descending artery and the posterior descending arteries. This posterior-lateral quadrant is located away from the positive poles of all the standard 12-lead ECG. Thus, a posterior infarction is indicated by

positive deflections of the QRS complex, distinctly over the precordial leads V1 to V3 and ST depressions in the same leads. This reflects mirror image changes only picked out by additional leads on the posterior thorax[1]. The ECG can also provide additional clues to the culprit vessel.

ST segment elevation in lead III more than lead II with concomitant ST depressions in leads I and aVL, points towards an occlusion in the RCA rather than LCX artery. In contrast, the findings of ST segment elevation in lead II, equal or greater than that of lead III, combined with ST segment depressions in leads V1 to V3 or ST elevation in leads I and aVL, point towards a dominant LCX occlusion[2-4]. A discordant ST segment flattening or elevation in lead V1 compared to the widespread depressions seen in leads V2 onwards is a clue for a concomitant right ventricular (RV) infarction. Of note, lead V1 is actually lead RV2 or a right-sided chest lead. A discordant ST segment flattening or elevation in lead V1 with depressions seen in leads V2 to V3 is a clue for a concomitant RV infarction. The findings of ST elevation in lead V1 and RV4 with an inferior myocardial infarction are indicative of acute proximal RCA occlusion causing right ventricular injury[5,6].

The ECG changes of massive pulmonary embolism can sometimes mimic an inferior RV infarction, especially with the combination of chest pain, hypotension, tachycardia, clear lung fields, raised cardiac enzymes and impaired RV function on echocardiography. However, the absence of ST elevation in RV4, hypoxia and clinical suspicion will help to diagnose pulmonary embolism. Isolated right ventricular infarction is a rare event which typically occurs in conjunction with an inferior-posterior infarction. As the right ventricular branch is supplied proximally, a concomitant right ventricular infarction points to the RCA as the culprit vessel, invariably in the proximal segment. Physical signs for patients with right ventricular infarction arise as a consequence to diminished right-sided filling and compliance. These include hypotension with jugular venous distension and clear lung fields on auscultation secondary to reduced left-sided filling with signs of high right sided filling pressures[7].

The left ventricular infarct size will determine the degree of left heart failure. Most often, a combination of biventricular failure is seen. Other complications that can develop include ventricular septal rupture, acute tricuspid or mitral regurgitation, atrioventricular nodal blocks and ventricular arrhythmias. These complications can cause haemodynamic compromise and need prompt treatment. Right ventricular infarction with inferior involvement giving rise to complications is a marker for bad prognosis[5,7-10]. Mortality rates range from 5.9% to 53% for patients in cardiogenic shock. The ischaemia further predisposes to malignant arrhythmias such as ventricular tachycardia or ventricular fibrillation. Up to one-third of patients do not make it to the hospital after a myocardial infarction as they often present as a sudden cardiac death episode.

AV nodal blocks are also common, with variable first, second or third degree blocks contributing to haemodynamic instability[8]. Pacing is only required, if prolonged bradycardia occurs that causes hypotension as the nodal blockage is often transient, due to the dual supply of the AV node[7,11]. In haemodynamically-unstable patients, management might entail fluid resuscitation with the aid of a Swan-Ganz catheter. The goal is to maintain appropriate left-sided filling with an ideal pulmonary artery wedge pressure. Nitrates, diuretics, beta blockers and calcium channels blockers that decrease preload, contractility and impede AV nodal blockage are used with caution, when hypotension and bradycardia develop. Patients can go into left ventricular failure with a large left ventricular inferior-posterior wall infarction that reduces left ventricular ejection fraction. Intra-aortic balloon pump would then be beneficial[7]. Rarely, patients can go into acute pulmonary oedema from acute mitral regurgitation secondary to posterior papillary muscle dysfunction.

In summary, prompt diagnosis and early reperfusion therapy is crucial in ensuring a favourable outcome for patients with ST elevation myocardial infarction. In patients with RV infarction, the appropriate fluid management with prompt treatment of complications is important to reduce mortality.

ABSTRACT

The early diagnosis of acute myocardial infarction is crucial for the institution of appropriate reperfusion therapy. We describe a 56-year-old man who developed sudden onset of severe chest tightness. Inferior, posterior and right ventricular ST elevation myocardial infarction was diagnosed on electrocardiography (ECG). The ECG interpretation, differential diagnosis and management are discussed.

Keywords: acute myocardial infarction, chest pain, electrocardiography, right ventricular infarction.

Singapore Med J 2006; 47(10):913-917

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

Multiple Choice Questions (Code SMJ 200610B)

	True	False
Question 1: RV infarction is associated with:		
(a) AV nodal blockage.	❑	❑
(b) Ventricular septal rupture.	❑	❑
(c) Tricuspid regurgitation.	❑	❑
(d) Hypoxia.	❑	❑
Question 2: Physical signs of RV infarction include:		
(a) Changes are caused by inhibition of sodium channels.	❑	❑
(b) Clear lung fields on auscultation.	❑	❑
(c) Lower limb oedema.	❑	❑
(d) Early diastolic murmur at the left sternal edge.	❑	❑
Question 3: ECG criteria for RV infarction include:		
(a) ST elevation in RV4.	❑	❑
(b) ST depression in V2 to V4 with tall R waves.	❑	❑
(c) ST elevations in II, III and aVf.	❑	❑
(d) ST elevation in V1.	❑	❑
Question 4: Bad prognostic markers of RV infarction include:		
(a) Cardiogenic shock.	❑	❑
(b) Sinus bradycardia.	❑	❑
(c) Ventricular septal rupture.	❑	❑
(d) Left ventricular failure.	❑	❑
Question 5: ECG pointers to a LCX occlusion versus RCA include:		
(a) ST elevation in lead III more than lead II.	❑	❑
(b) ST elevation in I and aVL.	❑	❑
(c) ST elevation in RV4.	❑	❑
(d) ST elevation in V1.	❑	❑

Doctor's particulars:

Name in full:_____

MCR number:_____ Specialty:_____

Email address:_____

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: (October 2006 SMJ 3B CME programme): 12 noon, 25 November 2006

Results:

1. Answers will be published in the SMJ December 2006 issue.
2. The MCR numbers of successful candidates will be posted online at http://www.sma.org.sg/cme/smj by 15 December 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.