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A Patient With Non-Q Wave Acute Inferior Myocardial Infarction

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Summary

The occurrences of atrioventricular (AV) nodal blockade or right ventricular (RV) infarction in acute inferior myocardial infarction (MI) had been well recognised and signified a proximal right coronary artery (RCA) occlusion. However, they did not point out the extent of the RCA territory involved. We recently managed a patient with incomplete inferior MI in which ECG on presentation already showed the infarct-related RCA as the dominant vessel having proximal occlusion. Various aggressive treatments have been taken subsequently. (HK Pract 1997; 19: 199-202)

Introduction

Acute inferior myocardial infarction (MI) was once believed not to have serious sequela. Recently, new data support the contrary. We recently managed a patient with an acute inferior MI and its manifested complications. New perspectives concerning the disease are discussed.

Case

A 65-years old female non-smoker who had history of hyperlipidaemia presented with sudden onset of severe chest pain to the Accidents and Emergencies Department. ECG showed ST segment elevation (>1 mm) over Leads II, III, and aVF; ST-depression/ T wave inversions over leads V2-V4 and inverted P waves associated with shortened PR interval over various chest and limbs leads (Figure 1). These findings were compatible with extensive acute inferior myocardial infarction (MI) associated with conduction disorder. A right sided ECG was not available. There being no contraindication to thrombolytic therapy, streptokinase 1.5 megaunits was given...
(over one-hour infusion) to this patient 5 hours after the onset of symptoms. It brought about resolution of chest pain and recovery of ST segments (Figure 2). The patient subsequently entered the Cardiac Rehabilitation Programme. On treadmill, she developed signs of cardiac ischaemia with horizontal ST depression over V2-V6 and angina at low workload capacity, which recovered with rest and sublingual nitrate. Cardiac catheterisation showed a critical proximal stenosis in a dominant right coronary artery (Figures 3-4). Left ventriculogram revealed overall good contractility apart from mild inferobasal hypokinesia. Adhoc angioplasty to this lesion was performed. She had an uneventful recovery.

**Discussion**

It is well known that acute inferior MI can be associated with bradycardia and/or AV block. These occurrences may be transient or require temporary pacing, but occasionally a permanent pacemaker will be needed. Conduction disorder did occur in our patient but reverted back to normal sinus conduction when the infarct-related artery (right coronary artery, RCA, in this case) reperfused. In addition, the finding of ST segment elevation of 1 mm or more in lead V4R (right-sided ECG) has a high sensitivity and specificity for detecting right ventricular (RV) infarction, pinpointing the site of occlusion in the proximal RCA. When RV infarction occurs, its stiffness increases, thereby impeding diastolic filling and leads to potential haemodynamic embarrassment. When hypotension or shock occurs, expansion of vascular volume is generally employed as initial therapy. In non-responders, dobutamine or similar inotropic agents may be helpful.

The occurrences of AV nodal blockade or RV infarction in acute inferior MI had been well recognised and signified a proximal RCA occlusion. However, they did not point out the extent of the RCA territory involved (i.e. whether the infarct-related RCA is dominant or not). Certainly, if a proximal occlusion in a dominant RCA
Figure 2: ECG showing reperfusion of acute inferior MI

Figure 3: Right coronary artery in right anterior oblique view showing a critical lesion in its proximal segment (arrow)

Figure 4: Right coronary artery in right anterior oblique view after successful PTCA (arrow)
occurred, more aggressive treatments should be given in order to salvage more myocardium. Wong et al.\(^{6-10}\) reported that the presence of significant precordial ST depression (V1-V3) in acute inferior MI reflects the size of a dominant RCA with large posterolateral and total left ventricular perfusion territory, and is not a benign reciprocal ECG change as previously believed. As exemplified in our case there was significant precordial ST segment depression during the acute event which recovered with reperfusion. Subsequent cardiac catheterisation reviewed a dominant RCA with large posterolateral distribution.

In the thrombolytic era, when successful reperfusion in the infarct related artery occurs, it often results in incomplete MI or Non-Q MI. They merely predict a high one year coronary event rate and therefore necessitate cardiac catheterisation and revascularisation procedure accordingly as performed here.

In summary, acute inferior MIs are not always benign and various clinical and ECG parameters can help identify those high risk cases for more aggressive treatment strategy.

References