

Case Reports

A Case of Myocardial Infarction Showing Extensive Precordial ST Elevation Induced by Second Diagonal Branch Occlusion

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SUMMARY

We observed a case of acute myocardial infarction induced by second diagonal branch occlusion. Electrocardiogram (ECG) on admission showed ST elevation in leads I, aV_L and V₂₋₆. Since emergency coronary angiography disclosed complete occlusion of the second diagonal branch, intra-coronary thrombolysis (ICT) was performed, superselectively. Transient coronary reperfusion was obtained, however, reocclusion occurred after several minutes. Rescue percutaneous transluminal coronary angioplasty (PTCA) was then performed immediately and blood flow was improved to TIMI grade 2. During these processes, the ST-segment on the ECG changed in leads I, aV_L and V₂₋₆ always corresponding to the blood flow of the second diagonal branch.

We have not seen a report hitherto in which occlusion of only the second diagonal branch could be a cause of extensive anterior infarction-like ECG changes. Although the mechanism of ECG changes in this patient cannot be clearly explained by conventional concepts, we report this case because it is considered to be very rare. (Jpn Heart J 35: 81-86, 1994)

Key words: ST elevation Ischemic area ICT PTCA

NON-INVASIVE mapping of the ischemic area in acute myocardial infarction is performed by means of electrocardiography (ECG), echocardiography, myocardial scintigraphy and other techniques. Of these clinical examinations, the ECG can be regarded as an excellent and common method because of its simplicity and sensitivity. Therefore, it is not difficult for a trained cardiologist to determine the ischemic area of a myocardial infarction by ECG findings alone.

Nevertheless, we observed a case of acute myocardial infarction induced by

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occlusion of the second diagonal branch in which it was difficult to diagnose the infarct area because of dissociation between ECG and coronary angiographic findings.

CASE REPORT

A 76-year-old male, admitted for rehabilitation of multiple cerebral infarctions, was sent to our hospital because of acute myocardial infarction. Coronary risk factors such as hypertension, obesity, diabetes mellitus and hyperuricemia were absent. The patient had smoked 30 cigarettes per day for 40 years. On admission, the blood pressure was 134/86 mmHg and the pulse was 62/min and regular. No pathological cardiac murmur was evident and breath sounds were normal. An ECG on admission showed marked ST elevation in leads I, aVL and V₂₋₆ with abnormal Q waves. The echocardiogram revealed local akinesia at the antero-lateral wall of the left ventricle. Chest X-ray did not reveal

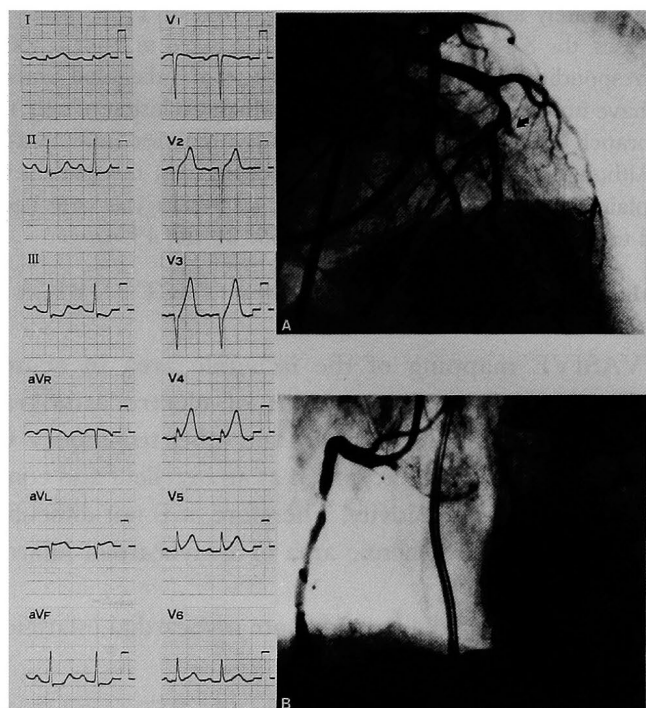


Figure 1. ECG on admission and coronary angiogram in the acute stage. Left panel: Marked ST elevation is observed in leads I, aVL and V₂₋₆. Right panel: Complete occlusion was observed in the second diagonal branch (arrow). No collateral circulation from RCA or LCx to the second diagonal branch is recognized. RCA = right coronary artery; LCx = left circumflex artery.

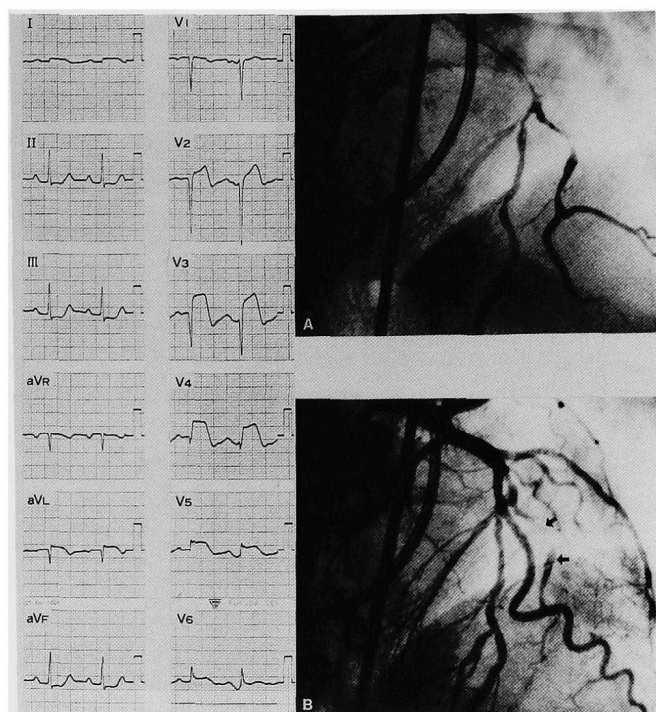


Figure 2. Coronary angiogram and ECG after ICT. Left panel: Re-elevation of ST segment observed in leads I, aVL and V₂₋₆ during ICT. Right panel: Coronary blood flow of the second diagonal branch improved to TIMI grade 2 after superselective ICT (arrow). ICT = intracoronary thrombolysis.

cardiomegaly or pulmonary congestion. Seroenzymological results were within the normal range. However, emergency coronary angiography was performed because severe chest pain persisted. As a result, complete occlusion was observed in the second diagonal branch of the left anterior descending artery and 90% stenosis in the first diagonal branch. However, significant stenosis was observed neither in the left anterior descending artery nor in the left circumflex or right coronary artery (Figure 1). Furthermore, collateral circulation to the second diagonal branch and anatomical abnormalities of the coronary artery were not demonstrated, angiographically. Based on these findings, we diagnosed the second diagonal branch as the infarct-related artery, and performed superselective intra-coronary thrombolysis (ICT) using 3.2 million units of recombinant tissue plasminogen activator. As a result of this treatment, coronary reperfusion to TIMI grade 2 and relief of ST elevation were obtained. After several minutes, however, reocclusion of the same site occurred and ST elevation was again observed in leads I, aVL and V₂₋₆ (Figure 2). Therefore, rescue percutaneous transluminal angioplasty (PTCA) was carried out. During inflation of the balloon,

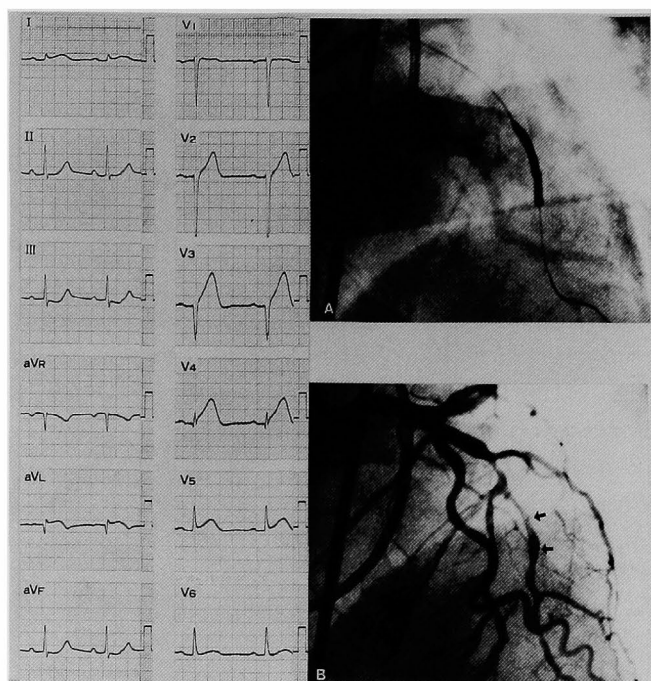


Figure 3. Coronary angiogram and ECG during and after PTCA. Left panel: Improvement of ST segment seen during PTCA. Right panel: Coronary blood flow of the second diagonal branch improved to TIMI grade 3 after PTCA (arrow). PTCA = percutaneous transluminal coronary angioplasty.

re-elevation of the ST-segment was seen in the same ECG leads. After successful PTCA, the blood flow was improved to TIMI grade 3 and apparent improvement of ST elevation was observed with disappearance of chest pain (Figure 3). Peak CPK reached only 858 IU, and acute hemodynamics remained Forrester subset I.

Myocardial scintigram (SPECT) using ^{201}Tl in the chronic stage revealed a decrease of Tl uptake at the antero-lateral wall of the left ventricle, but reverse redistribution was observed in the same region on delayed image, suggesting the maintenance of coronary blood flow in the chronic stage. Coronary angiography in the chronic stage could not be performed because of patient refusal.

DISCUSSION

Generally, when ST elevation or abnormal Q waves are observed in the area including V₂-V₄ of the precordial leads in a patient with acute myocardial infarction, the left anterior descending artery is considered to be the infarct-

related artery. Exceptionally, there are several reports of precordial ST elevation observed in a case of subendocardial infarction of the left circumflex artery as a reciprocal change¹⁾ and in a case of right coronary occlusion including a large right ventricular branch accompanied by right ventricular infarction.^{2,3)} However, no reports referring to infarction of the diagonal branch causing ST elevation in the precordial leads were found. Takatsu et al⁴⁾ who collected 19 cases of diagonal branch infarction reported that ECG changes were positive in leads I, aV_L and V₅₋₆ in all their cases. According to Mager et al,⁵⁾ precordial ST changes such as seen in our case were not evident during PTCA of a diagonal branch.

With respect to the perfusion area of a diagonal branch, Hori and his coworkers⁶⁾ reported that the perfusion area of the first diagonal branch was equivalent to that of the distal anterior descending artery, and pointed out the unexpected extent of the perfusion area of the diagonal branch. However, they did not refer to the relation between ECG findings and localization of the infarction. On the other hand, Norris et al⁷⁾ reported the relation of the extent and degree of the ST elevation at 35 points of the precordial lead to infarct area or its severity. Nevertheless, we could not find any sufficient information in their reports to elucidate the dissociation of the infarct area and ECG findings observed in our case.

Since ST elevation occurred reproducibly in the precordial leads even during rescue PTCA in our case, transient arterial spasm or thrombotic occlusion of the left anterior descending artery could be excluded as a cause. Furthermore, because no pericardial effusion or inflammatory findings were present, pericarditis could also be eliminated as the cause of ST elevation in this case. Cohen and his colleagues⁸⁾ reported experimental findings that ST elevation developed more strongly on the border zone between the normal and ischemic area rather than on the infarct area. Therefore, there is a possibility that the dissociation of ST elevation from the infarct-related artery in our case might have been caused by a similar mechanism.

In any case, even though the mechanism of ECG changes caused by occlusion of the second diagonal branch observed in this patient cannot be definitely explained, we report it as a very rare and interesting case.

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