The Complete Cancellation of Abnormal Q Waves
Due to an Old Anteroseptal Infarction
Following Subsequent Acute Posterior
Myocardial Infarction

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SUMMARY
A rare case of multiple infarction is described. An abnormal Q
wave due to an old anteroseptal infarction was completely masked by a
new contralateral posterior myocardial infarction. Coronary angiogram
revealed multiple stenoses of segments 1 (100%), 7 (99%), 12 (90%) and
13 (99%). Left ventriculography also showed akinesis of segments 2, 3,
4 and 6, and reduced wall motion in segments 1, 5 and 7. These find-
ings, together with electrocardiographic changes from before the present
attack reinforce the above interpretation.

Additional Indexing Words:
Abnormal Q wave  Multiple infarction  Disappearance of Q wave

It is well known that the disappearance or the regression of abnormal Q
waves occurs during follow up of patients with myocardial infarction.
In the case of multiple infarction, the regression of abnormal Q waves due
to previous myocardial infarction may also occur following a new myocardial
infarction involving the contralateral myocardium.

However, it is rather rare to observe such a case in the normal clinical
setting. We describe here a very interesting and rare case of multiple in-
farction with complete disappearance of abnormal Q waves due to an old
anteroseptal infarction following the occurrence of a new posterior myo-
cardial infarction.

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CASE REPORT

A 66-year-old Japanese man had complained of occasional chest discomfort and dyspnea on exertion. On 25 June, 1985, at midnight he experienced sudden onset of severe dyspnea and cold sweating with brownish sputa and left hypochondralgia. Until then, he had not been aware of signs of myocardial infarction such as severe chest pain, cold sweating, etc. However, he had been told that he had ECG changes compatible with an old inferior and anteroseptal myocardial infarction seen initially on an ECG recorded in October, 1984 (Fig. 1B). On admission to a nearby hospital, his ECG (Fig. 1A) showed abnormal Q waves and flat T waves in II, III and aVF, suggestive of an old inferior myocardial infarction and an apparent normalization of the abnormal Q waves due to the old anteroseptal myocardial infarction. In addition, R waves in V₂ and V₃ seemed to be a bit prominent. He was transferred to the CCU of our department (First Department of Internal Medicine, Medical College of Oita). Following a comparison with his previous ECG (Fig. 1B) and enzymological examinations, showing increased serum CPK-MB levels, he was diagnosed as having

![ECG images](image-url)

Fig. 1. Standard 12-lead ECG at the development of dyspnea (A) and that on October 3, 1984 (B). Note the slightly increased R waves in V₂₃₄ (A). The contours of the QRS complex in V₂₋₆ were retouched for clarification. See text for further details.
Fig. 2. Illustration of serial serum enzyme changes. The unfilled triangles represent CPK, the filled triangles LDH, the filled circles white blood cell count and the unfilled circles GOT, respectively.

Fig. 3. Serial ECG changes before and after the present attack. Note the apparent normalization of leads V1 to V3 of the ECG recorded on June 25, 1985.

an acute posterior infarction. Subsequent laboratory findings revealed increased serum CPK-MB levels and successive increases in serum GOT and LDH compatible with an acute myocardial infarction (Fig. 2). However, acute right ventricular overload such as is seen following pulmonary infarction, which might reveal similar ECG changes, should be ruled out to establish the diagnosis. Hence, pulmonary scintigraphy was performed 2 days after admission. The result was not suggestive of acute pulmonary
Fig. 4. Coronary angiogram. Panel A shows the left anterior oblique view of the right coronary arteriogram and panel B shows the left anterior oblique view of the left coronary arteriogram. See text for further details.

Fig. 5. Left ventriculogram. A, end-systole in right anterior oblique view; B, end-diastole in the same view as in A. C, end-systole in the left anterior oblique view; D, end-diastole in the same view.

infarction. Chest X-ray also revealed no sign of pulmonary infarction.

The serial serum enzyme changes and ECG changes are shown in Figs. 2 and 3, respectively. The amplitude of the R waves in leads V1 to V3 were decreased as shown in the figure and small remnant q waves were seen; however the ECGs after the present attack were quite different from those recorded on October 3, 1984.

Cardiac catheterization was performed 2 months later. At this time, the coronary artery showed multiple stenoses (Fig. 4) of segments 1 (100%), 7 (99%), 12 (90%) and 13 (99%). Left ventriculography (Fig. 5) revealed
reduced wall motion in segments 1, 5 and 7 and akinesis in segments 2, 3, 4 and 6.

**Discussion**

During the follow up of patients with myocardial infarction, important changes occur in their ECGs, including the disappearance of abnormal Q waves and normalization of ST-T changes. The disappearance of abnormal Q waves may occur in 5–20% of patients and is attributed to 1) normal evolution, 2) multiple infarction, and 3) intraventricular conduction disturbance. The disappearance of abnormal Q waves is particularly important in cases of multiple infarction, since the development of a new myocardial infarction may not only mask the previous infarction, but also mask the present myocardial infarction itself, as in the case presented above.

In our case, ECG changes due to an old anteroseptal infarction (i.e., Q waves in V1–3) were cancelled by ECG changes attributed to the development of a new posterior infarction (i.e., slightly increased R waves in V1–3). The findings on coronary angiography and left ventriculography support this interpretation.

Similar ECG changes, namely the disappearance of abnormal Q waves following development of a new myocardial infarction have been reported in 2 patients. In neither of these cases was cardiac catheterization performed; however necropsy supported the interpretation. Therefore, our case is the first reported in which diagnosis was confirmed by cardiac catheterization.

With regard to the cause of the disappearance of abnormal Q waves in the precordial leads, another possibility is acute right ventricular overload due to acute pulmonary infarction or pneumothorax. However, in our patient both possibilities were excluded based on the chest X-rays, pulmonary scintigraphy and laboratory examinations done on admission.

Thus, we conclude that the development of a new posterior myocardial infarction completely masked the old anteroseptal myocardial infarction.

**References**

