Annals of Nuclear Medicine Vol. 19, No. 5, 407–409, 2005

Scintigraphic evaluation of regression of abnormal Q waves in myocardial infarction

Tetsuo KONNO, Masami SHIMIZU, Hidekazu INO, Masato YAMAGUCHI, Hidenobu TERAI, Katsuharu UCHIYAMA, Kotaro OE, Tomohito MABUCHI, Tomoya KANEDA and Hiroshi MABUCHI

Molecular Genetics of Cardiovascular Disorders, Division of Cardiovascular Medicine, Graduate School of Medical Science, Kanazawa University

We report regression of the abnormal Q waves of an inferior old myocardial infarction after an additional anterior acute myocardial infarction, and demonstrate the scintigraphic correlation and chronological course of this phenomenon. Scintigraphic findings in the present case here may contribute to an interpretation of regression of abnormal Q waves in myocardial infarction.

Key words: myocardial perfusion scintigraphy, electrocardiography, myocardial infarction

INTRODUCTION

REGRESSION OR DISAPPEARANCE of the abnormal Q waves of old myocardial infarction (OMI) after an additional acute myocardial infarction (AMI) has been described in some previous reports.[1–3] However, no reports have assessed the mechanism of this phenomenon using myocardial perfusion scintigraphy. In the case presented here, we elucidated the pathogenesis of this phenomenon based on the scintigraphic correlation, and also clarified the chronological course in a patient with anterior AMI and a history of inferior OMI.

CASE REPORT

A 68-year-old man with a history of hypercholesterolemia had a first AMI on May 17, 1991. On electrocardiogram (ECG), new abnormal Q waves with ST-T elevations were observed in leads II, III and aV_F. Emergent coronary arteriography showed total occlusion of the proximal right coronary artery (RCA). Percutaneous transluminal coronary angioplasty (PTCA) to the proximal RCA lesion was performed with a 3.0 mm balloon, although only incomplete reperfusion was observed. The patient's plasma creatine kinase level peaked at 2,125 IU/*l* at 30 hours after the onset of chest pain. At the second cardiac catheterization, performed 4 weeks after admission, spontaneous reperfusion of the RCA was observed, although akinesia of the inferior wall was present with a left ventricular ejection fraction (LVEF) of 57%. Abnormal Q waves persisted at a later date while ST-T elevations normalized. On October 6, 2002, the patient was admitted to our hospital after 30 minutes of chest pain unrelieved by sublingual nitroglycerin. In comparison with the ECG taken one month previously, the ECG on admission showed ST-T elevations in leads I, aV_L and from V_1 to V_5 (Fig. 1). Abnormal Q waves in leads II, III and aV_F were observed in both ECGs taken one month previously (Fig. 2A) and on admission (Fig. 2B). Emergent coronary arteriography showed total occlusion of the proximal left anterior descending artery (LAD). The 75% stenotic lesion in the proximal RCA was also observed, which was considered to be the culprit lesion of the inferior OMI. Subsequently, PTCA with a 3.0 mm balloon and implantation of a 3.0 mm stent to the proximal LAD lesion was successfully performed. Four hours after admission, ST-T elevations in the anterior leads and leads I and aV_L decreased. Interestingly, regression of the abnormal Q waves in leads II, III and aV_F was observed (Fig. 2C), which was accompanied by poor R wave progression in the precordial leads. The patient's plasma creatine kinase level peaked at 4,942 IU/*l* at 21 hours after the onset of chest pain. A technetium-99m methoxyisobutylisonitrile scintigram, taken 3 weeks after

Received October 14, 2004, revision accepted January 14, 2005.

For reprint contact: Tetsuo Konno, M.D., Molecular Genetics of Cardiovascular Disorders, Division of Cardiovascular Medicine, Graduate School of Medical Science, Kanazawa University, Takara-machi 13–1, Kanazawa 920–8640, JAPAN.

E-mail: kontetsu@im2.m.kanazawa-u.ac.jp

admission (Fig. 3B), showed an additional defect in the anterior-to-septal wall compared with a thallium-201 scintigram taken in 1991 (Fig. 3A). During hospitalization, the patient had symptoms of congestive heart failure, which required intravenous diuretics and dopamine. At the second cardiac catheterization, performed 4 weeks after admission, restenosis of the proximal LAD was not observed, although hypokinesia of the anterior, septal and inferior walls was present, with a reduced LVEF of 32%. Regression of abnormal Q waves remained evident on the second cardiac catheterization (Fig. 2D).

The follow-up period was uneventful, and regression of the abnormal Q waves has persisted.

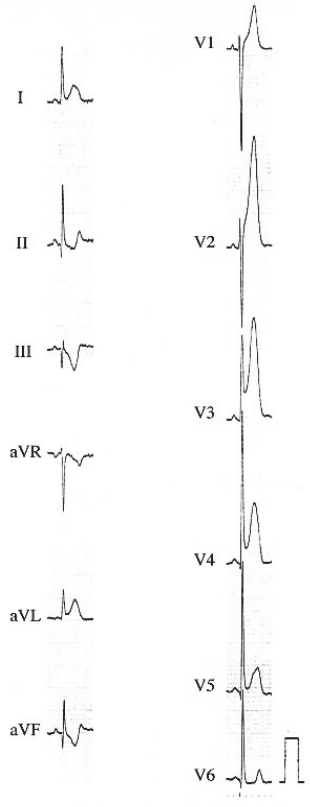


Fig. 1 Electrocardiogram on admission demonstrating ST-T elevations in leads I, aV_L and from V_1 to V_5.

DISCUSSION

In the case presented here, we observed regression of the abnormal Q waves of an inferior OMI after an additional anterior AMI. It may be helpful to begin with a discussion of the pathogenesis of the abnormal Q waves in myocardial infarction to examine this phenomenon.

The area of necrosis becomes electrically silent in the infarcted myocardium.[4] In an inferior myocardial infarction, the balance of the electromotive forces tends to point away from the inferior wall because the opposing electromotive forces of the anterior wall become dominant. Therefore, abnormal Q waves are present in leads II, III and aV_F in inferior myocardial infarction.[5] This suggests that a loss of the electromotive forces of the anterior wall in a subsequent AMI may lead to regression of the abnormal Q waves in the inferior leads.[1]

Myocardial perfusion scintigrams in this case may be useful to understand the above-mentioned mechanisms of this phenomenon visually. Myocardial perfusion scintigrams taken in 1991 and 2002 in this patient indicate that the infarcted areas include the inferior and anterior wall, which are opposing in terms of the electromotive forces. Luca et al. have previously shown the pathologic correlation of disappearance of the abnormal Q waves after superimposed myocardial infarction at necropsy.[1] They postulated that disappearance of the abnormal Q waves

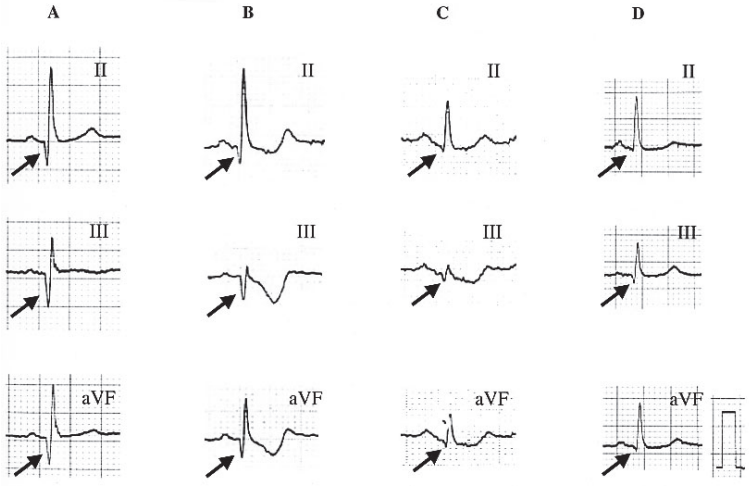
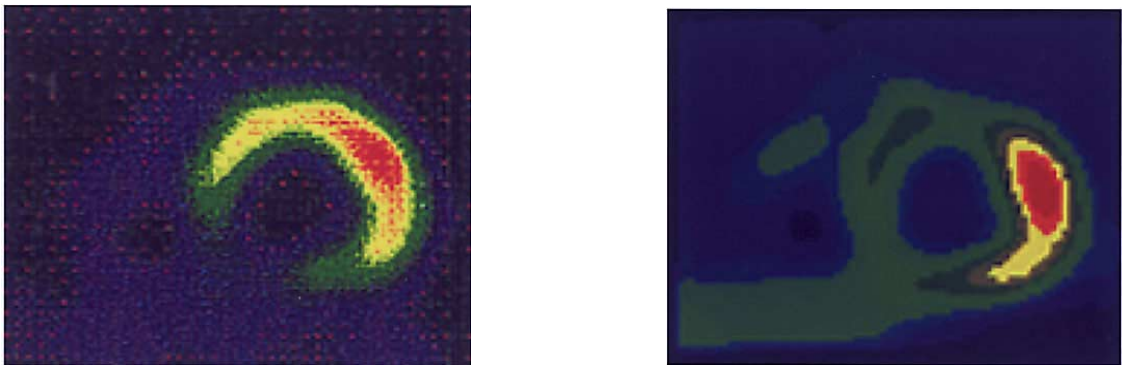


Fig. 2 Electrocardiograms demonstrating chronological course of regression of the abnormal Q waves in leads II, III and aV_F. A: One month before admission, B: On admission, C: Four hours after admission, D: Twenty-eight days after admission.



A B

Fig. 3 A: A thallium-201 scintigram taken in 1991 demonstrating a defect in the inferior wall. B: A technetium-99m methoxyisobutylisonitrile scintigram taken in 2002 demonstrating an additional defect in the anterior-to-septal wall.

might be caused by the loss of an equal amount of electromotive forces between the opposing areas. In our case, the loss of the electromotive forces in the anterior wall may have been slightly less than that in the inferior wall, resulting in the residual small Q waves in the inferior leads. Appearance of conduction disturbances after AMI is also known to cause regression of abnormal Q waves.[3,4] In our patient, we did not observe any conduction disturbance in the clinical course (Figs. 1 and 2). These findings suggest that regression of abnormal Q waves in the inferior leads in our patient was not due to appearance of conduction disturbance, but due to the loss of the electromotive forces in the anterior wall after an additional anterior AMI.

Although there are some previous reports of this phenomenon,[1–3] it is unclear exactly when regression of the abnormal Q waves occurs after an additional AMI. In the present case, we clarified the chronological course of this phenomenon. It is worth noting that this phenomenon was not observed on admission (Fig. 2B), 30 minutes after the onset of chest pain, but was observed 4 hours after admission (Fig. 2C), which was accompanied with poor R wave progression in the precordial leads. These findings suggest that this phenomenon may not occur suddenly after the occlusion of the coronary artery, and may require sufficient necrosis of the myocardium to cause the loss of the electromotive forces in the opposing area. It is possible that the salvaging of the infarcted area may cause reappearance of abnormal Q waves in the opposing leads. In our patient, regression of abnormal Q waves in the inferior leads was present even at 4 weeks after admission (Fig. 2D). A myocardial perfusion scintigram taken 3 weeks after admission demonstrated a massive defect in the anterior wall (Fig. 3B), suggesting that improvement of electromotive forces in the anterior wall may not be sufficient to cause reappearance of abnormal Q waves in the inferior leads. Further studies are necessary to clarify the relationship between the salvaging of the infarcted area and regression of abnormal Q waves in the opposing area.

CONCLUSION

We report regression of the abnormal Q waves of an inferior OMI after an additional anterior AMI. Myocardial perfusion scintigrams were useful to understand the mechanisms of this phenomenon visually. This phenomenon may not occur suddenly after the occlusion of the coronary artery, and may require sufficient necrosis of the myocardium to cause the loss of the electromotive forces in the opposing area. Scintigraphic findings in the present case may contribute to an interpretation of regression of abnormal Q waves in myocardial infarction.

REFERENCES

1. Luca C, Georgescu L, Zorila C, Lazar E. Sudden disappearance and reappearance of the pathologic Q waves after successive myocardial necrosis. *Cardiology* 1981; 67: 103–109.
2. Wang K, Segal M, Ward P. Sudden disappearance of electrocardiographic pattern of anteroseptal myocardial infarction: Result of superimposed acute posterior myocardial infarction. *Chest* 1976; 70: 402–404.
3. Levine HD, Youug E, Williams RA. Electrocardiogram and vectorcardiogram in myocardial infarction. *Circulation* 1972; 45: 457–470.
4. Goldberger AL. In: Myocardial infarction: *Electrocardiographic differential diagnosis*. 4th ed. St. Louis; Mosby, 1991.
5. Chou TC. In: *Electrocardiography in Clinical Practice: Adult and Pediatric*. 4th ed. Philadelphia; WB Saunders, 1996.