【 CASE REPORT 】

Acute Ischemic Mitral Regurgitation Treated by Percutaneous Coronary Intervention after an Accurate Diagnosis on Transesophageal Echocardiography

Ryoichi Miyazaki, Keita Watanabe, Masakazu Kaneko, Sho Nagamine, Nobuhiro Hara,
Tomofumi Nakamura, Yasutoshi Nagata, Toshihiro Nozato and Takashi Ashikaga

Abstract:

An 80-year-old woman with acute posterolateral myocardial infarction, cardiogenic shock, and acute heart failure was admitted to our hospital. Transthoracic echocardiography (TTE) showed dysfunction of the left ventricular inferolateral wall motion and severe mitral valve regurgitation (MR). Emergency coronary angiography revealed triple-vessel stenosis. We performed transesophageal echocardiography in the catheter room to diagnose the cause of MR. Severe tenting of the mitral valve and no rupture of the papillary muscles were revealed. We considered ischemic MR likely to improve with revascularization and performed percutaneous coronary intervention. Subsequently, the patient's circulatory dynamics rapidly stabilized, and MR was significantly improved on follow-up TTE.

Key words: acute myocardial infarction, transesophageal echocardiography, ischemic mitral valve regurgitation, tethering, tenting height, between papillary muscles diameter

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Introduction

Mitral valve regurgitation (MR) after acute myocardial infarction (AMI) has a very poor prognosis and is generally treated by surgery (1, 2). Acute MR caused by papillary muscle and chordae tendon rupture is commonly known. However, if the origin of the MR is related to exterior deviation of the papillary muscles caused by wall motion abnormalities in the region of the culprit coronary artery, it is generally called ischemic MR.

Acute ischemic MR can be improved by revascularization alone (3) if myocardium viability is expected to remain for a short onset-to-reperfusion time or non-total coronary artery occlusion at the first angiogram (4). We herein report a case of AMI with severe acute MR leading to cardiogenic shock that was successfully treated by percutaneous coronary intervention (PCI) following the diagnosis of ischemic MR using transesophageal echocardiography (TEE) in the acute phase.

This paper was approved by institutional ethical commit-

tee of Red Cross Musashino Hospital and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient.

Case Report

An 80-year-old woman who had undergone permanent pacemaker implantation 2 months ago due to intermittent atrioventricular block was admitted to our hospital with chest pain and shock. Her blood pressure and percutaneous oxygen saturation could not be measured. She complained of sustained chest pain, and an electrocardiogram showed ST elevation in leads aVL and aVR, and segment (ST) depression in leads II, III, aVF, and V4-6 (Fig. 1). We diagnosed her with ST-segment elevation posterolateral MI with cardiogenic shock.

Chest X-ray revealed severe lung congestion, and transthoracic echocardiography (TTE) (Vivid S6; GE Healthcare, Tokyo, Japan) revealed a deceased left ventricular inferolateral wall motion and severe MR (Fig. 2A). The meas-
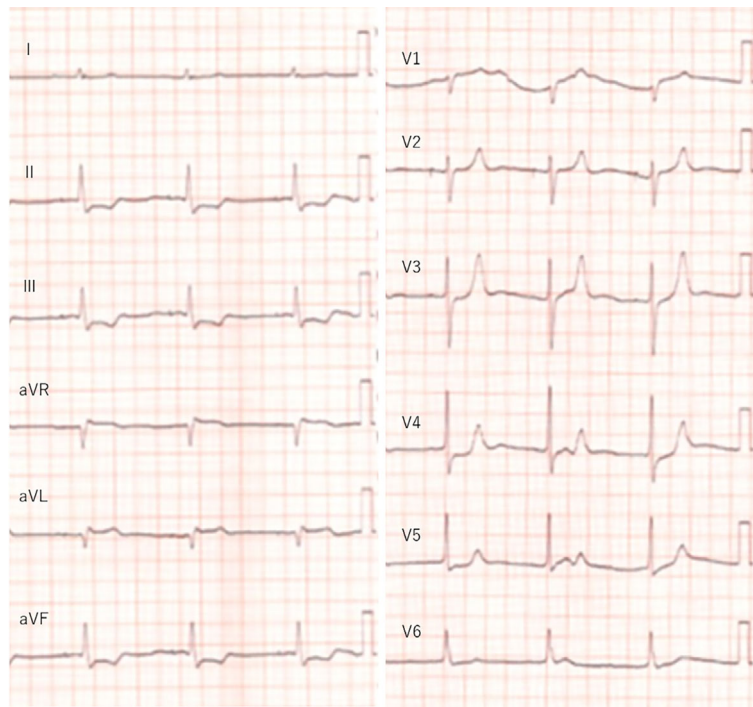
Figure 1. The electrocardiogram obtained in the emergency department. Heart rate: 60 beats per minute, atrial pacing rhythm. ST elevation was observed in leads aVL and aVR, and ST depression was observed in leads II, III, aVF, and V4-6.
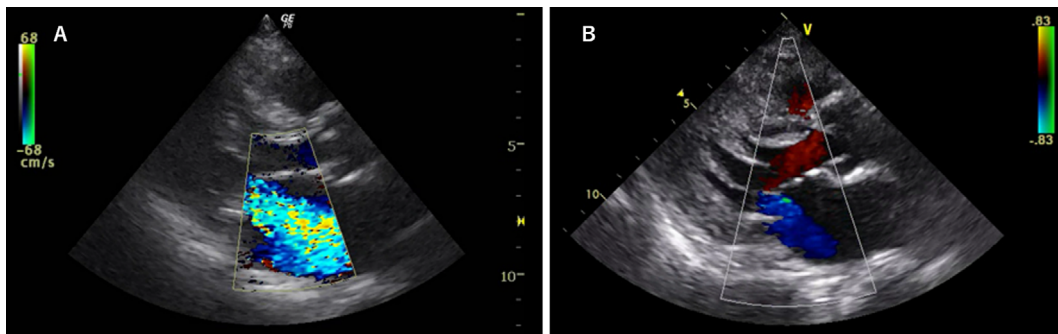

Figure 2. (A) Color doppler imaging by transthoracic echocardiography before percutaneous coronary intervention. Severe mitral valve regurgitation (MR) was detected. (B) Image on the fourth day; MR was significantly improved.

ured values were a left ventricular end diastolic diameter (LVDd) of 42 mm, left ventricular end systolic diameter (LVDs) of 31 mm, and ejection fraction (EF) of 49.8%. We considered the severe MR to be the major cause of the shock. However, the presence of papillary muscle rupture was difficult to ascertain on TTE.

We performed tracheal intubation and started infusion of a vasopressor, but the patient's circulation dynamics did not improve. We performed emergency coronary angiography (CAG) and TEE (F75, Hitachi, Tokyo, Japan) at the same time in order to determine which coronary artery was responsible for the MR. After insertion of the intra-aortic balloon pump (IABP) the patient's circulation dynamics stabilized. CAG revealed triple-vessel stenosis, total occlusion of the distal right coronary artery (RCA) with collateral flow

from the septal branches of the left anterior descending artery (LAD), 90% stenosis of the mid-LAD, 99% stenosis of the first diagonal branch with contrast delay, and 99% stenosis in the left circumflex artery (LCX) (Fig. 3A, B). We determined the culprit lesion to be in the LCX by localization of the dysfunction of the inferolateral wall and ST elevation in aVL on an electrocardiogram.

TEE revealed no papillary muscle or tendon rupture but showed severely tented mitral valve leaflets and severe mitral valve regurgitation due to valve coaptation failure (Fig. 4). The mitral valve leaflet tenting height was 12 mm, indicating that the MR had been caused by tethering due to exterior deviation of the bilateral papillary muscles. To improve the MR, it was necessary to revascularize the coronary blood flow perfusing the bilateral papillary muscle regions,
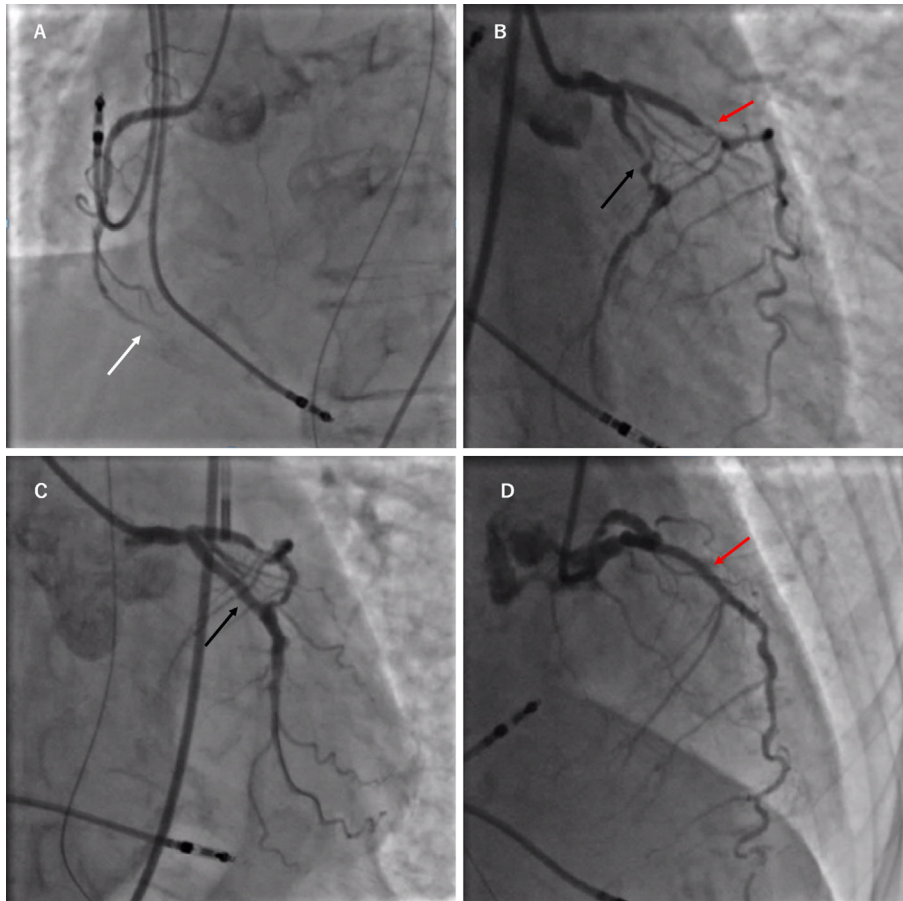
Figure 3. (A) Coronary angiography revealed total occlusion of the distal right coronary artery (white arrow). (B) Note 99% stenosis in the left circumflex artery (black arrow) and 90% stenosis of the mid-left anterior descending artery (red arrow). (C) (D) We performed stent implantation in #11 (black arrow) and #6 (red arrow).
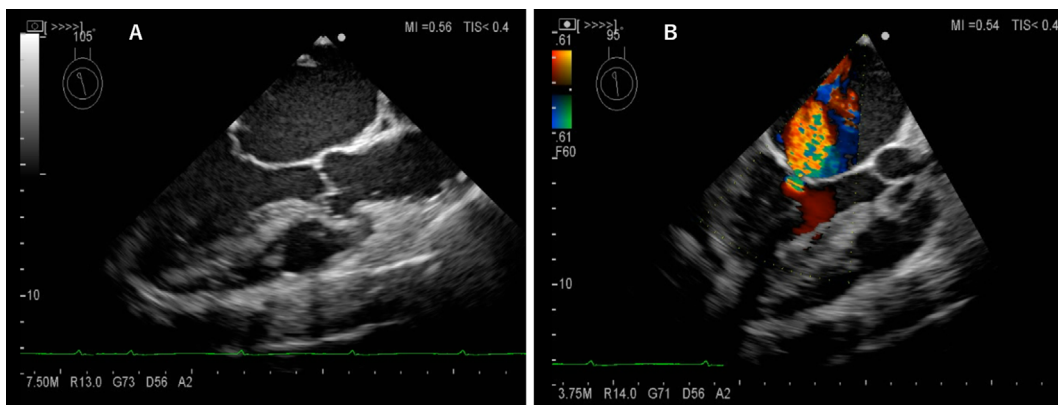

Figure 4. (A) Transesophageal echocardiography showed severely tented mitral valve leaflets. The mitral valve leaflet tenting height was 12 mm. (B) Color doppler showed severe mitral valve regurgitation due to valve coaptation failure.

so we placed stents in the LCX and LAD (Fig. 3C, D).

After PCI, we administered dobutamine hydrochloride, noradrenaline, and diuretics. The patient's circulation dynamics remained stable, and the IABP was able to be withdrawn on the second day, with extubation performed on the third day. The maximum creatine kinase value was 3,853 IU/L, and the creatine kinase-MB level was 204 IU/L.

TTE was performed again on the fourth day, and the MR had significantly improved (Fig. 2B). The LVDd and LVDs had shrunk from 42 to 31 mm and 31 to 21 mm, respectively. The EF had recovered from 49.8% to 63.5%. The mitral valve leaflet tenting height was reduced from 15 to 9 mm (Fig. 5A, B), and the papillary muscle distance (length connecting the central portions of the anterior and posterior
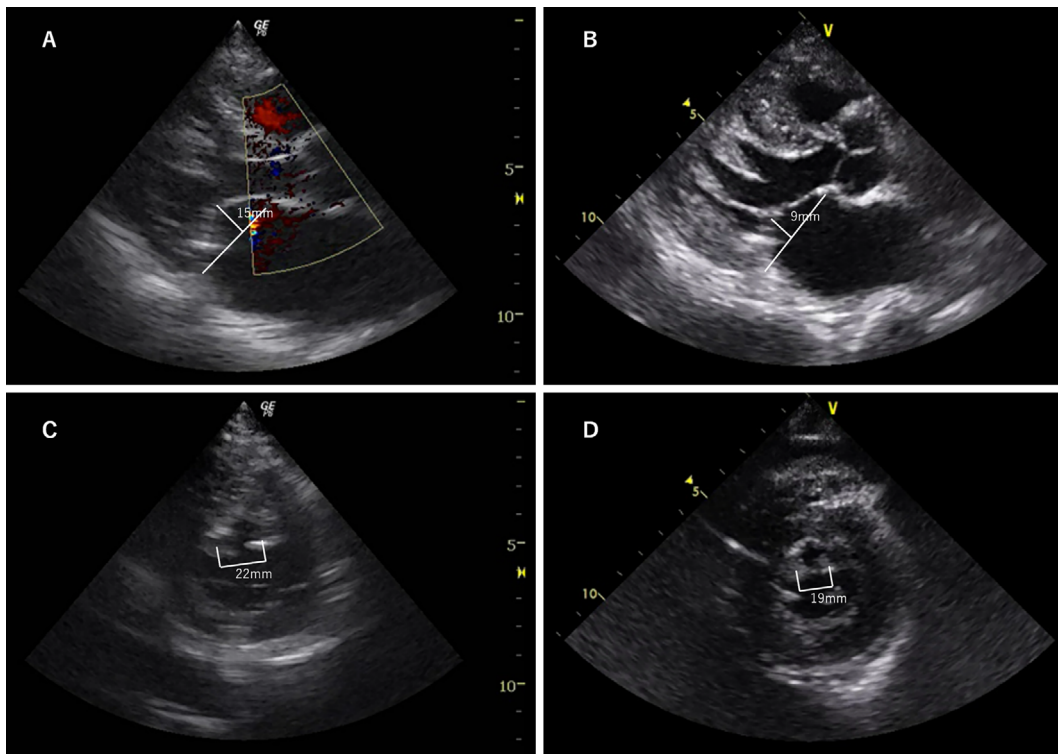
Figure 5. (A) (B) Transthoracic echocardiography showed that the tenting height had decreased from 15 to 9 mm after revascularization. (C) (D) Similarly, the papillary muscle distance at the systolic interval shrank decreased from 22 to 19 mm.

papillary muscles on parasternal short-axis images at the papillary muscle level at the end-systolic interval) had decreased from 22 to 19 mm (Fig. 5C, D). The patient was discharged on the eleventh day and is still alive after two years.

Discussion

MR associated with myocardial infarction is divided into that caused by papillary muscle and chordae tendon rupture and that caused by tethering of leaflets due to lateral excursion of the papillary muscle (2). The anterior papillary muscles are double-dominated by the first diagonal branch (#9) and the obtuse marginal branch (#12), but the posterior papillary muscles are single-dominated by the RCA (#4 AV or #4PD) or LCX (#14 or #15). MR is three times more frequent in cases of posterior papillary muscle rupture than in cases of rupture of the anterior papillary muscle (5). When papillary muscle rupture occurs, it becomes prolapsed and causes flail of the mitral valve leaflet; this is usually easy to evaluate, but the sensitivity fails to exceed 65-85% in TTE due to poor general or inspection conditions (6). Partial rupture does not show a deviation of the papillary muscle head into the left atrium (35%), and TEE has higher diagnostic ability in such instances (7). In the present case, we were able to diagnose acute ischemic MR by TEE in the acute phase.

In general, ischemic MR is a chronic-phase complication that results from left ventricular remodeling, and the mecha-

nism involves a change in the mitral valve complex due to left ventricular expansion with pulling of the leaflets due to deviation of the papillary muscle to the outside (tethering) (8). In cases of inferior wall infarct in particular, although the remodeling range is small, a marked change occurs in the mitral valve complex, resulting in a tendency for ischemic MR to occur (9). Although there have been few reports on acute ischemic MR compared with chronic ischemic MR, the left ventricular diameter, valve tenting area, and leaflet area are small, suggesting that mild valve tethering produces MR. This means that valve adaptation via leaflet remodeling is insufficient (10). Reportedly, even mild MR worsens the short- and long-term prognosis in cases of acute ischemic MR (4).

In the present case, the culprit artery was the LCX, but the diagonal branch had 99% stenosis with contrast delay. This caused a decrease in the wall motion of the anterior papillary muscle area through the onset of acute myocardial infarction (AMI). The decrease in blood pressure caused a decreased blood flow in the RCA receiving collateral circulation from the LAD, simultaneously with a decline in the wall motion of the posterior papillary muscle area. The bilateral papillary muscles simultaneously deviated outward, resulting in severe MR due to high tethering. We needed to revascularize the coronary arteries perfusing the bilateral papillary muscle areas. Myocardium viability was expected to remain because the left ventricular inferolateral wall motion had been reduced but was maintained on TTE findings, and the LCX was a non-occlusive lesion. Furthermore, we

decided to reconstruct the LCX and LAD. Although the RCA directly perfused the posterior papillary muscle area, it was difficult to treat immediately due to the presence of a chronic total occlusion lesion; we therefore revascularized the LAD to increase the collateral circulation to the RCA.

After revascularization, the LVDd, LVDs, tenting height, and papillary muscle distance were rapidly decreased. Furthermore, the MR remarkably improved along with left ventricular reverse remodeling, indicating that the MR had been caused by acute tethering due to lateral excursion of the papillary muscle.

In a recent large study, simultaneous revascularization of major stenosis in non-culprit coronary arteries for AMI accompanied by cardiogenic shock increased the mortality and renal dysfunction (11). However, in the present case, since the cause of shock was MR, we considered it necessary to treat the two stenoses simultaneously in order to improve the MR.

In clinical practice, AMI with severe MR is likely to lead to surgery without the accurate identification of the origin of the MR. However, the prognosis is very poor, even if emergency surgery is performed (1). Although there have been no reports of the identification of the origin of MR by TEE during PCI for ST-segment elevation myocardial infarction, this method should be considered, given the potential for internal treatment to be performed in cases of ischemic MR, as in the present patient.

The predictor of improvement in acute ischemic MR is a short time from the onset to reperfusion and non-total coronary artery occlusion before PCI, whereas the predictor of deterioration is a high maximum creatine kinase-MB value in the acute phase (4). Particularly in cases where the global left ventricular wall motion is maintained and MR seems to be ischemic, early PCI may improve the MR and prognosis.

Conclusion

We were able to treat this case of acute ischemic MR with PCI because an accurate diagnosis had been made with rapid TEE. It is not uncommon for acute MR to accompany AMI, but it is often difficult to obtain a definite diagnosis in AMI-related MR by TTE in the emergency department. TEE is a powerful diagnostic tool for determining the etiology of MR, and if acute ischemic MR can be successfully diagnosed, highly invasive surgery may be avoided in the acute phase. Rapid TEE should be considered in cases where an MR diagnosis is difficult, as it is useful for determining whether surgical or internal treatment is optimal in such cases.

The authors state that they have no Conflict of Interest (COI).

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