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Papillary Muscle Rupture after Acute Myocardial Infarction
- The Importance of Transgastric View of TEE -

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Transesophageal echocardiography was performed to evaluate the exact cause of severe mitral regurgitation in a 64-year-old man presented with hypotension and dyspnea after acute inferior wall myocardial infarction. In mid-esophageal two-and four-chamber view, the ruptured stump of papillary muscle could not be visualized. However, in transgastric two-chamber view, we could clearly visualize the ruptured head of the posteromedial papillary muscle as a separated mass attached by chorda tendinae, as well as the freely mobile stump of the ruptured papillary muscle within the left ventricle. So, the comprehensive transesophageal echocardiography, including transgastric imaging, is always indicated in patients with severe mitral regurgitation after acute myocardial infarction.

Key Words : *Myocardial Infarction, Papillary Muscles, Rupture, Echocardiography, Transesophageal*

INTRODUCTION

Mitral regurgitation (MR) after myocardial infarction is a relatively common complication and its incidence is about 50%. One cause of MR after myocardial infarction is papillary muscle rupture, and this situation usually requires urgent surgical treatment. Since prognosis is favorable with early surgical intervention, early diagnosis of the underlying cause of mitral regurgitation is very important. Transthoracic echocardiography is an excellent tool for detection and estimation of the severity of mitral regurgitation in patients with myocardial infarction, especially those who have a systolic murmur. But transthoracic echocardiography may have limitations for detailed exploration of the subvalvular apparatus of the mitral valve. Multiplane transesophageal echocardiography (TEE) has been used for accurate evaluation of the mechanism of mitral regurgitation in patients with acute myocardial infarction. We experienced a case of severe mitral regurgitation due to papillary muscle rupture that was confirmed only by transgastric view of TEE.

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CASE REPORT

A 64-year-old man with recent inferior wall myocardial infarction was transferred to the emergency department. He had chest pain for 3 days about two weeks previously and underwent coronary angiography at another hospital. The coronary angiography showed total occlusion of mid-RCA and 70% long segment stenosis at mid LAD. PTCA was tried to open RCA but failed.

On admission to our hospital, he was hemodynamically unstable with severe pulmonary congestion, respiratory distress and hypotension requiring vasopressor support. His blood pressure was 90/60 mmHg with infusion of Dobutamine 10 μg/Kg/min, pulse rate was 100/min and respiration rate was 25/min. On physical examination, systolic murmur was audible at the cardiac apex and

coarse breath sounds were audible, with rales and wheezing over both lower lung fields. The electrocardiogram showed sinus tachycardia with the rate of 113/min and Q wave in II, III, aVF that was compatible with old inferior wall myocardial infarction. The chest X-ray showed cardiomegaly with pulmonary congestion.

The transthoracic echocardiogram showed akinesis of inferior wall with moderately reduced left ventricular systolic function with estimated ejection fraction of 40% and moderate to severe mitral regurgitation with anterior mitral leaflet prolapse. The eccentric mitral regurgitant jet was directed to the posterolateral side of the left

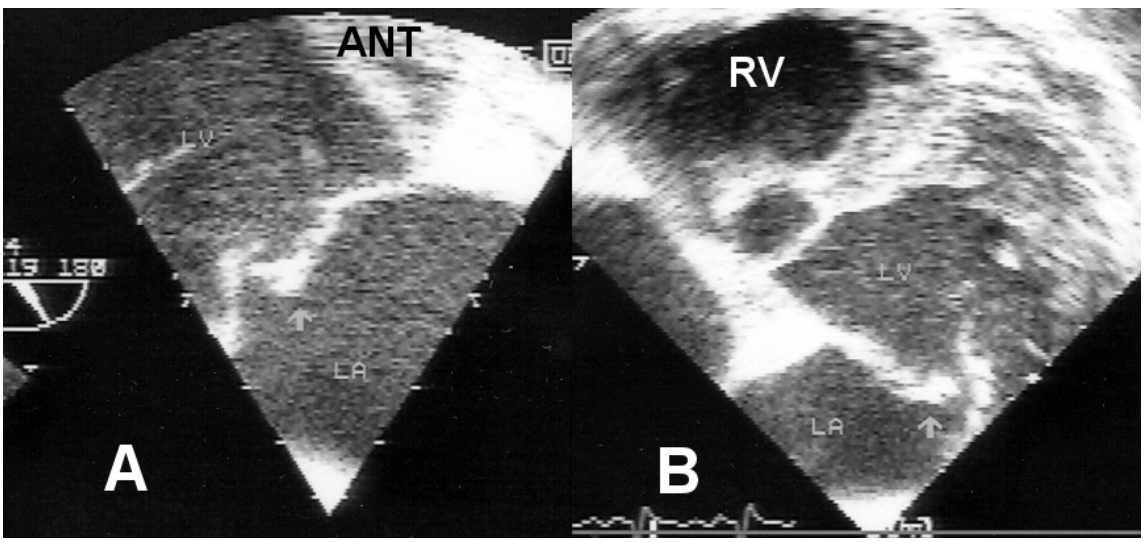

Figure 1. The bang-down mid-esophageal long-axis view (A) and four-chamber view (B) shows anterior mitral valve prolapse (arrow) into the left atrium during systole. (ANT, anterior wall; LV, left ventricle; LA, left atrium; RV, right ventricle)

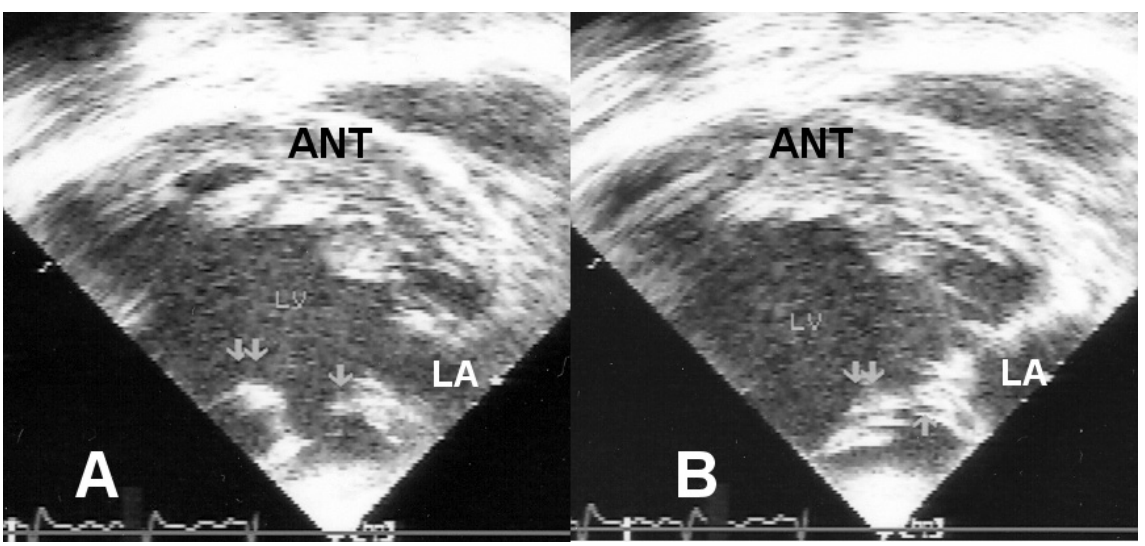

Figure 2. The bang-down transgastric long-axis view clearly shows flail motion of the ruptured stump (double arrows) and the head (arrow) of the posteromedial papillary muscle during diastole (A). During systole, the two ruptured parts approach each other (B). (ANT, anterior wall; LV, left ventricle; LA, left atrium)

atrial chamber in color flow mapping, and pulsed Doppler interrogation of the right upper pulmonary vein demonstrated holosystolic flow reversal suggesting severe mitral regurgitation. The transthoracic echocardiogram could not detect papillary muscle rupture. Initially, he was treated medically with intravenous dobutamine, digitalis, ACE inhibitor and diuretics for 3 days, but the cardiogenic shock and pulmonary edema state continued without improvement. So, he underwent transesophageal echocardiography for further evaluation of mitral regurgitation. In mid-esophageal two-chamber view, grade III/IV mitral regurgitation with anterior mitral leaflet prolapse was identified (Figure 1A). In mid-esophageal four-chamber view, flail motion of anterior mitral valve leaflet was visualized which was highly suspicious of the ruptured chordae tendinae (Figure 1B). In transgastric two-chamber view, we could identify the ruptured head of papillary muscle as a separated mobile mass attached to chordae tendinae of anterior mitral valve leaflet and the stump of the ruptured posteromedial papillary muscle (Figure 2A, 2B). During diastole, the two ruptured parts moved freely within the left ventricular cavity but, during systole, each ruptured end of the papillary muscle parts approaced each other and we could clearly visualize the stump of the ruptured posteromedial papillary muscle within the left ventricle (Figure 2A, 2B). With these findings, he was transferred to cardiac surgery. At the operation field, the ruptured parts of the posteromedial papillary muscle were identified but repair looked impossible, so the valve was removed and mitral valve replacement, as well as LIMA to LAD bypass surgery, was successfully performed. The post-operative course was uneventful and the patient survived.

DISCUSSION

Mitral regurgitation (MR) after myocardial infarction (MI) is a common complication. In the TIMI-II trial, MR was present in 13% and the incidence of MR may be as high as 50% if transient MR is included[1]. It is important to recognize the exact cause of acute MR because papillary muscle rupture needs urgent surgical intervention, while the MR of other causes may improve with medical treatment after thrombolysis or percutaneous coronary intervention. Papillary muscle rupture has been reported in about 1% of patients following acute myocardial infarction, and is most common in the setting of inferior wall infarct due to the occlusion of either RCA or left circumflex artery[2]. The posteromedial papillary muscle is twelve times more likely to rupture than the anterolateral papillary muscle because the blood supply of the two papillary muscles are different. The clinical manifestation of papillary muscle rupture usually includes the development of new systolic murmur and cardiogenic shock 3~7 days after acute myocardial infarction but, in patients with complete papillary muscle rupture or severe left ventricular dysfunction, the murmur may not be audible. In contrast, partial papillary muscle rupture often does not result in severe MR or cardiogenic shock. At right heart catheterization, large v-wave can be recorded in the pulmonary wedge pressure curve in 60% of patients with papillary muscle rupture, but a large v-wave alone is non-diagnostic since it can also be observed in ventricular septal rupture or severe left ventricular dysfunction[3]. So, the echocardiogram has been regarded as a gold standard for the diagnosis of papillary muscle rupture. Transthoracic echocardiogram is useful in the demonstration of severe MR and, if the image is ideal, the diagnosis of papillary muscle rupture is possible[4]. But in some cases, the transthoracic echocardiogram has limitations for detailed exploration of the mitral subvalvular apparatus. And, in critically ill patients, it has other limitations, such as restriction in optimal positioning and limited echocardiographic window, due to mechanical ventilation. Several reports have described the usefulness of transesophageal echocardiography in the diagnosis of papillary muscle rupture. In these cases, the papillary muscle rupture was confirmed only by the use of transesophageal echocardiography[5-7]. Single plane TEE may allow only incomplete evaluation of the subvalvular apparatus and easily miss papillary muscle rupture, so multiplane TEE is more useful[8].

In the current case, despite careful examination, we could not confirm papillary muscle rupture from the mid-esophageal window, although severe MR and a flail anterior mitral valve leaflet did suggest the possibility of chordal rupture, indirectly. But in the transgastric view, the freely mobile ruptured head of the papillary muscle was clearly visualized within the left ventricle, with systolic prolapse into the left atrial cavity. The stump of the ruptured papillary muscle was also visible in this view. Therefore, we suggest that the transgastric window of TEE, especially the two-chamber view with careful clockwise or counter-clockwise rotation, is important for the diagnosis of papillary muscle rupture.

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