Cardiac Rupture Due to Reinfarction in the Early Phase of Apical Myocardial Infarction

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Summary
A 72-year-old woman with hypertension, dyslipidemia, and diabetes mellitus presented to our hospital because of the sudden onset of chest pain. Emergency coronary angiography showed acute occlusion of the distal left anterior descending artery and coronary intervention with a drug-eluting stent was performed. Sudden cardiopulmonary arrest occurred on the sixth day of hospitalization, but coronary angiography showed no remarkable progression of the coronary artery diseases, including the site of stent implantation. An autopsy revealed that the cause of the sudden death was apical free wall rupture. In addition, the different timing of acute and sub-acute infarct findings were observed in the apical wall by histology, which indicated cardiac rupture was due to reinfarction at early phase of apical acute myocardial infarction. Although the rate of mechanical complications, including cardiac rupture, is decreasing in the era of primary coronary intervention, in addition to the well-known risk factors of cardiac rupture, the reinfarction of the culprit myocardial site in the early phase of acute myocardial infarction was considered as a possible risk factor of cardiac rupture.

Key words: Pathology, Recurrent myocardial infarction

The incidence of cardiac rupture after acute myocardial infarction (AMI) has been reduced over the past two decades with the advent of primary coronary intervention and the progress of drug therapy.1,2 However, cardiac rupture remains a fatal complication of AMI and is the second-most common cause of in-hospital mortality in cases of ST-elevation myocardial infarction.3,4 The incidence of cardiac rupture is reported to range from roughly 1% to 3% after AMI, but autopsy research has shown that cardiac rupture is involved in 30% to 65% of in-hospital AMI death.5-7 Although several risk factors have been suggested to be associated with cardiac rupture, the mechanisms involved in this crucial complication are unclear at present.

Case Report
A 72-year-old woman with hypertension, dyslipidemia, and diabetes mellitus presented to our hospital because of the sudden onset of chest pain for 5 hours. She had no history of coronary artery disease, smoking, or chest symptoms. The initial electrocardiogram showed ST elevation in leads V2 to V5 and an abnormal Q wave in leads II and III, as well as an aVf with elevated cardiac enzymes (Figure 1). All vital signs were stable and no manifestation of congestive heart failure was noted. Echocardiography revealed decreased apical wall motion without pericardial effusion. Emergency coronary angiography indicated triple-coronary-vessel disease with a total occlusion of the distal left anterior descending artery (LAD), mid-right coronary artery, and left obtuse marginal branch (Figure 2A-C). Collateral flow served to the distal right coronary artery and obtuse marginal branch but not to the LAD. The distal LAD was considered to be the acute occlusive lesion based on electrocardiogram and echocardiography. Therefore, primary coronary intervention was performed to the distal LAD occlusive lesion. Six hours after the appearance of chest pain, reperfusion of the distal LAD lesion was obtained by balloon angiography and a drug-eluting stent was deployed to the lesion (Figure 2 D-F). The chest pain and ST elevation of the precordial leads rapidly disappeared after reperfusion. Although her general condition, including vital signs and respiratory symptoms, was stable, sudden cardiopulmonary arrest (ventricular fibrillation on monitor) occurred on day 6 of hospitalization. Advanced cardiopulmonary life support was immediately given with extracorporeal membrane oxygenation. Coronary angiography showed no remarkable change in the coronary artery, including the LAD stent-implanted lesion. Echocardiography revealed no peri-
Figure 1. Initial electrocardiogram demonstrating ST elevation in V2-V5 and abnormal Q waves in II, III, and aVf.

Figure 2. A-C: Initial coronary angiography. Total occlusion of the distal left anterior descending artery (white arrowhead) with collateral flow to the right coronary artery (A). Total occlusion of the left obtuse marginal branch (white arrowheads) (B) and mid-right coronary artery (white arrowhead) (C). D-F: Coronary intervention. Reperfusion of the distal left anterior descending artery after thrombus aspiration (D). A drug-eluting stent was deployed to the culprit left anterior descending artery lesion (E). Final angiographic findings (F).
Figure 3. Macroscopic findings of the ruptured heart. Infarct region was confirmed in the inferior wall (A). Acute apical myocardial infarction and free wall rupture; free wall rupture is indicated by the metal stick (B). Histology of infarcted inferior myocardium showing fibrosis tissue without inflammatory cells in hematoxylin and eosin staining (C) and Masson trichrome staining (D). The white round line indicates sub-acute infarct region of the apical wall (E). Histology showing fibroblasts, hemosiderin-laden macrophages, and capillaries at 2 weeks after the onset of AMI; hematoxylin and eosin staining (F). Red zone indicates acute infarct region of the apical wall (G). Histology showing necrotic myocytes and scattered macrophages with early influx of chronic inflammation at 7 days from the onset; hematoxylin and eosin staining (H).
cardial effusion, but she failed to obtain cardiopulmonary resuscitation and ultimately died. We received written informed consent from her family to perform an autopsy to determine the cause of sudden cardiac death.

Gross observations of autopsy indicated cardiac rupture of the left ventricular apical free wall with a large amount of hemorrhage in the intrathoracic space and mediastinum. The infarct region was confirmed to be apical and inferior wall (Figure 3A, B). Histology of the inferior infarct region revealed replaced fibrous tissue without inflammatory cell infiltration, indicating old infarct (Figure 3C, D). Fibroblasts, hemosiderin-laden macrophages, and capillaries were observed at the apical infarct wall, which was considered as healing infarct, approximately 2 weeks from the occurrence of myocardial infarction (Figure 3E, F). However, findings such as necrotic myocytes and scattered macrophages with an early influx of chronic inflammation were observed, indicating acute infarct in the subacute infarct region of the apical wall (Figure 3G, H).

Discussion

This case of cardiac rupture was unusual because acute and sub-acute infarct were heterogeneously observed in the apical wall by autopsy. Moreover, reinfarction in the early phase of apical wall infarction was found to be related to cardiac rupture, which has not been previously reported. According to the histological findings, index apical myocardial infarction occurred 2 weeks before without clinically significant symptoms probably due to a long-standing history of diabetes mellitus and because of intramural infarct. An apical cardiac rupture was identified as the cause of death, but no remarkable pericardial effusion was identified using echocardiography. As injuries of intrathoracic tissues, such as rib fracture or pulmonary damage, are often accompanied by cardiopulmonary life support procedure, especially when the case is a lean, elderly woman, we believe that ruptured blood leaked from the pericardium to the intrathoracic space due to injury of the epicardium caused by chest compression during cardiopulmonary resuscitation.

From a pathophysiological perspective, the infarct region is most vulnerable within 7 to 10 days after AMI and a left ventricular free wall rupture most often occurs later in the first week after AMI.9,10 Various patterns of cardiac rupture have been reported, and endocardial tears and ruptured tracts of the heart are strongly associated with a rapid progression of existing cardiac rupture.11-13 In addition, delayed reperfusion therapy of coronary intervention and a totally occluded LAD with no collaterals are significantly associated with a high incidence of cardiac rupture.14 A pathophysiological assessment by Schuster et al. indicated that inappropriate dilatation and thinning of the infarct region in the early phase of AMI causes increased wall tension, which contributes to intramural stretching and tearing of the myocardial fiber, leading to cardiac rupture.15 In their report, almost all patients with cardiac rupture had dilatation and thinning of the infarct region, and this dilatation and thinning were recognized within days of myocardial infarction. They also mentioned that all cases of cardiac rupture were transmural infarction. A first infarct event and small infarct size are well-known as risk factors of cardiac rupture, because patients with these risk factors tend to be hemodynamically stable and suffer from high left ventricular wall stress. Therefore, transmural infarction and dilatation or thinning of the infarct region in the early phase of AMI play important roles in the occurrence of cardiac rupture. Interestingly, our patient had evidence of multiple infarctions and a large infarct region, which indicate relatively low wall stress of the infarct region, but cardiac rupture still occurred. This can be attributed to the formation of vulnerable heterogeneous necrotic and fibrous myocardium causing structural weakness around the apical wall due to different timing of apical myocardial infarct; first apical infarction was intramural infarct and reinfarction progressed to transmural infarct.

Although the rate of mechanical complications including cardiac rupture is decreasing in the era of primary coronary intervention, the prediction of cardiac rupture after AMI is still difficult. The past retrospective study demonstrated that early phase reinfarction post coronary intervention for AMI had higher short term mortality, but the incidence of cardiac rupture was not reported.60 In this present case, early phase reinfarction was the potential risk factor of cardiac rupture, which may be useful for elucidating the pathophysiological mechanisms of cardiac rupture and managing patients with AMI.

Disclosures

Conflicts of interest: None.

References


