# MYOCARDIAL RUPTURE AFTER ACUTE MYOCARDIAL INFARCTION: A REVIEW

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## Abstract

**Myocardial rupture complications after acute myocardial infarction are infrequent but lethal. They mainly involve rupture of the ventricular free wall, ventricular septum, papillary muscle, or combined. We compare features of different kinds of myocardial ruptures after acute myocardial infarction by reviewing the clinical insights.**

**Keywords:** acute myocardial infarction; left ventricular free wall rupture; papillary muscle rupture; ventricular septal rupture.

## Introduction

Myocardial rupture complications of acute myocardial infarction are infrequent but lethal. They mainly involve the ventricular free wall, interventricular septum, papillary muscle, or combined.[1] They occurred in 2.3% of all cases of acute myocardial infarction, and accounted to 15.7% of hospital mortality.[2] The patient's survival depended on preoperative hemodynamic status, and it was noted that 77% of the patients who developed cardiogenic shock died preoperatively.[3] Women aged 60 to 69 were at the highest risk for rupture. More patients with myocardial rupture had hypertension during hospitalization, persistent pain, and inferior wall myocardial infarction when compared with controls. The majority (95%) of cardiac ruptures occurred within the first six days, 40% within the first 24 hours after the onset of symptoms. Approximately 20% of ruptures were diagnosed as subacute.[2] We compare the features of different kinds of myocardial ruptures after acute myocardial infarction by reviewing the clinical insights.

## Ventricular septal rupture

Ventricular septal rupture complicates approximately 1%-2% of cases of acute myocardial infarction. Such a complication requires urgent surgical treatment because, on medical treatment alone, 60%-70% of patients die within the first 2 weeks.[4] Ventricular septal rupture usually involves the apical septum and frequently occurs with anteroseptal myocardial infarctions.[5] It occurs more commonly in patients with advanced age, hypertension, nonsmokers, anterior infarction, female gender, and lower body mass index.[6] The time of isolated ventricular septal rupture occurred commonly between 2nd and 7th day after the onset of acute myocardial infarction, the length of survival after the onset of rupture varied from less than one day to 60 days, the majority dying within eight days after the onset of rupture.[5]

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Patients often present with pulmonary edema or cardiogenic shock. A new loud, harsh holosystolic murmur and a palpable thrill are usually detected, and may help in differentiating this entity from papillary muscle rupture.[7] Echocardiography is a highly sensitive and specific tool in assessing the presence of ventricular septal rupture and is the diagnostic modality of choice.

Despite optimal treatment, mortality remains 45% in surgically treated patients and 90% in those managed medically.[8] Early repair of posterior ventricular septal rupture following acute myocardial infarction is associated with significant perioperative mortality in comparison with anterior or proximal. Left ventriculotomy might compromise the ventricular geometry and the systolic function, leading to a high risk of surgical injury to the posterior papillary muscle. The friable, recently infarcted myocardium is usually larger at the left ventricular endocardial side, and is threatening for sutures stability. Surgeons have made continuous attempts to minimize either left ventricle remodeling or surgical traumas. da Silva et al.[9] introduced a technique in 1989, which consisted of a transinfarction incision in the left ventricle, placement of a fine Dacron fabric patch that covered all the infarcted septum and closed the ventricular septal rupture, and placement of a second Dacron fabric patch that reinforced the infarcted anterior wall of the heart and supported the buttressed double suture closure of the left ventriculotomy. Postoperative angiography and echocardiography showed good geometry of the left ventricle with no aneurysm formation in all survivors.[10] noted that the left side of the ruptured septum presented a large necrotic area, while the right side appeared less compromised, with necrotic tissue extending barely at the rim of the defect. They proposed that stitches and patch would have durability if applied on the right side. The rupture was repaired by a right ventricular posterior free wall approach to expose the right side of the septum. The edges of the ruptured septum were joined together by a single purse-string pledgetted suture, and a tailored double-folded patch was applied to close both the septal rupture and the right ventriculotomy.[11] proposed right atrium approach into repair of ventricular septal rupture, thereby avoiding direct incision of the ventricle, and reducing postoperative bleeding and impairment of ventricular contractile function. Moreover, the use of fibrin glue has been advocated in the repair of intracardiac structural defects including postinfarction ventricular septal rupture. Immediate intraoperative transesophageal and late follow-up echocardiography documented complete and durable repair of all defects without recurrence.[12] Transcatheter closure of postinfarction ventricular septal rupture was introduced as an alternative that avoids the high risk of surgery, and a lower mortality and morbidity than surgical closure has been achieved.[13]

**Papillary muscle rupture**

The incidence of papillary muscle rupture is 1% of all acute myocardial infarctions and accounts for 5% of the mortality following acute myocardial infarctions.[14] Papillary muscle rupture in myocardial infarction is mostly related to an inferior infarction with the right or the left circumflex coronary arteries being the culprit arteries.[15] It has proved a higher incidence and greater severity of ischemic mitral regurgitation with more severe geometric changes in the mitral valve apparatus and greater displacement of posterior papillary muscle in patients with inferior compared with anterior myocardial infarction.[16] There are two separate papillary muscles: the anterolateral and the posteromedial. The anterolateral papillary muscle is usually fed by a dual blood supply from the left anterior descending artery and by marginal tributaries from the circumflex artery, whereas the posteromedial papillary muscle usually has a single blood supply from the posterior descending artery. Thus, in an acute myocardial infarction, it is the posteromedial papillary muscle that is most vulnerable to infarction, necrosis, and rupture.[17] It has been suggested that cardiogenic shock after papillary muscle repture may result from myocardial infarction, mitral regurgitation, and left ventricular dysfunction due to disconnection of the papillary muscle from the mitral annulus.[18]

Isolated total rupture of a papillary muscle often occurred between the 2nd and 6th day after the onset of the infarction, and the isolated partial rupture of a papillary muscle occurred between the 2nd and the 9th day. Survival after total rupture of a papillary muscle was short with 78% dying within one day after rupture, and the remaining dying within 2-5 days. Among the patients with isolated partial rupture, only 30% died within one day of rupture, others died between three days and seven months.[15] Discovery of a new systolic murmur following infarction suggests rupture of a papillary muscle leading to mitral regurgitation. However, the absence of a new heart murmur after acute myocardial infarction dose not exclude the diagnosis.[19] Echocardiography could show flail chordae and the muscle head itself may be seen to be whirling around in the ventricle with the flail leaflet moving into the atrium in systole; within hours, the muscle head and chordae can become firmly tangled.[17]

Nitroprusside is useful in the treatment of acute mitral valve regurgitation due to papillary muscle rupture because it decreases systemic vascular resistance. Patients with hypotension may tolerate vasodilators after insertion of an intra-aortic balloon pump. Because the prognosis is dismal among medically treated patients, emergency surgery is highly recommended.[2] Repair of mitral valve apparatus has been attempted in patients with partial papillary muscle rupture in the early years, however, without encouraging results.[20] Lately[21] achieved good result in primary mitral plasty of partial instead of complete postinfaction papillary muscle rupture.[20] performed successful repair of complete papillary muscle rupture by reattachment of the papillary muscle to the left ventricular wall. Although experimental studies suggested that rupture of the anterolateral papillary muscle cause a deterioration of left ventricular function, and mitral valve replacement would lead to a further impairment, repair of the ruptured papillary muscle could maintain the left ventricular function, mitral valve replacement still remains the most common procedure in the treatment papillary muscle rupture.[18]

**Left ventricular free wall**

Left ventricular free wall rupture occurs in 4% of patients with acute myocardial infarction[22,23], and is responsible for 5%-24% of post-acute myocardial infarction deaths.[24] The condition occurs up to ten times more often than papillary muscle rupture or interventricular septal rupture.[25] About 30% of the ruptures occur within the first 24 hours postinfarction and 90% occur within the first 14 days. The highest incidence is in the first four days. Left ventricular free wall rupture is more common in women, patients older than 55 with a first acute myocardial infarction, and those with hypertension. Emergent revascularization is associated with a decreased incidence of left ventricular free wall rupture, with primary angioplasty being more effective than thrombolytic therapy.[26] Factors thought to relate to the occurrence of cardiac rupture are those which impose mechanical stress on the ventricular myocardium and impair its structural integrity. Elevation of systolic blood pressure and an increase in end-diastolic volume augment intramyocardial wall stress. The great majority of those with cardiac rupture had hypertension. Anticoagulant therapy, although of uncertain significance in the pathogenesis of rupture, has been associated with a 5-fold increase in cardiac perforation. Another factor of importance is the softness of the infarcted area.[27]

The most common rupture site is the junction of necrotic and normal myocardium in transmural anterior or anterolateral acute myocardial infarction. Rupture is seven times more common in the left ventricle than the right ventricle.[28] classified the morphology of free-wall rupture into three types, which are also relevant to ventricular septal rupture: type I occurs within the first 24 hours, have an abrupt tear in the wall without thinning; type II occurs 1 to 3 days postinfarction, the infarcted myocardium erodes before rupture occurs and is covered by a thrombus; and type III occurs at days 5-10, has marked thinning of the myocardium, secondary formation of an aneurysm, and perforation in the central portion of the aneurysm. Other classifications such as blow-out and ooze rupture have also been used.[29]

A prompt diagnosis and treatment can allow the survival of patients. Sudden onset of chest pain with straining or coughing may herald the onset of myocardial rupture. Acute rupture patients often develop electromechanical dissociation, shock, and sudden death. Other patients may have a more subacute course. They may complain of pain consistent with pericarditis, nausea, and develop hypotension. In a study evaluating 1,457 patients with acute myocardial infarction, 6.2% of patients had free wall

rupture. Approximately one third of these patients presented with a subacute course. Echocardiography demonstrates a defect in the ventricular wall, pericardial effusion, and evidence of pericardial tamponade.[30]

Management of left ventricular free wall rupture hinges on diagnosing and addressing the acute pericardial tamponade. Medical management is rarely successful. An intra-aortic balloon pump can be a temporary measure until the patient can be taken to surgery.[31] Intraaortic balloon pumping support time was 2.3±0.9 days after surgery. Definitive treatment is surgical correction of the defect. The overall surgical mortality rate was 11.8%- 17.6%.[29,32].

Cardiopulmonary bypass is not usually used in repair of left ventricular free wall rupture. The indication of cardiopulmonary bypass was a posterior tear[33], or because of exsanguination, hemodynamic collapse, to locate the tear or to reach graftable vessels.[31] There are four kinds of techniques of repair of left ventricular free wall rupture: infarctectomy and patch reconstruction, direct closure with or without patch covering, simple patch covering anchored by running suture, and a sutureless technique[29] Patients with an oozing type of rupture located on lateral or anterolateral wall can be treated with sutureless techniques often without cardiopulmonary bypass. Patients with a blow out type of rupture would require a sutured approach.[34] In suture techniques, the suture line must be along the nonischemic area and transmural stitches are required, resulting in further deterioration of left ventricular function due to damage to the nonischemic myocardium.[35] The application of a patch covering the area of infarction and anchored to normal myocardium with continuous running sutures. Because the anchoring sutures are placed only in the epicardium and shallow surface of the myocardium, myocardial damage by this technique is minimal.[31] Introduced detailed sutureless technique that we have employed in the present case: an elliptical patch of polytetrafluoroethylene felt was fashioned to be circumferentially 1 cm larger than the macroscopical area of hematoma and muscle necrosis such that the whole perimeter of the patch lies on healthy myocardium and the area of the infarct is covered. The patch was placed over the infarcted area and then soaked with biocompatible glue. After 2 to 3 min the patch is firm and hemostatic.

**Double structural rupture**

A combination of any two the above three types of ruptures is called ventricular double structural rupture, which is a rarer complication after acute myocardial infarction. The report of a double structural rupture can trace back to 1940, when[36] reported a case of muscular septal rupture, with subsequent posterior wall rupture due to thrombotic occlusion of right coronary artery with extensive myocardial infarction. Tanaka et al.[37] observed that double structural rupture occurred in 0.3% of patients with acute myocardial infarction, in 3% of those with free wall rupture, and in 16.1% of those with ventricular septal rupture. Reddy and Roberts[24] noted that the incidence of free wall rupture and ventricular septal rupture was 4%, and that of free wall rupture and papillary muscle rupture was 2%.

Rupture was less frequently in patients with immediate β-blockers. Thrombolytic therapy may increase the frequency of rupture of the left ventricular free wall rupture or septum rupture during acute myocardial infarction. Evidence of recurrent acute infarction (infarct extension) was found in 32% of deaths and occurred with a similar incidence in patients dying of pump failure, arrhythmias, and rupture. Death resulting from rupture occurred earlier after infarction (3.8±3 days) than death resulting from arrhythmias or pump failure. Almost all patients who died of rupture had a single area of infarction as compared to those who died of pump failure, 29% of whom had multiple areas of infarction. Edwards et al. [38] classified myocardial rupture as simple and complex by a study of 53 specimens. Simple: a direct through-and through communication between the ventricle without associated major hemorrhagic tracts or laceration of surrounding tissue. Complex: characterized by the presence of hemorrhagic tracts with myocardial disruption. They noted double structural rupture in 11 cases: 9 had free wall rupture, and 2 had papillary muscle rupture (1 mitral and 1 tricuspid).[39] suggested left ventricular free wall and ventricular septal rupture can be classified as true and junctional: The junctional is the portion of myocardium located directly anterior and posterior to the ventricular septum, but does not contact eother ventricular cavity. Junctional ruptures produce a left-to-right ventricular communication, a left ventricle-to-pericardial communication, or both. True rupture of ventricular septum and free wall involves the myocardial wall actually bordering the ventricular cavity, i.e., outside the junctional area.[37] Identified rupture in eight of their patients, five of them were true rupture, none had junctional, and three were undetermined.[5] Two structural rupture in eight cases, the most common combination being rupture of ventricular septum and left ventricular wall. Inferolateral location of underlying infarction was the common situation in ruptured papillary muscle, while anteroseptal myocardial infarction was more common in rupture of the ventricular septum.[40] Eight cases of ventricular septal rupture, one was associated with both ventricular rupture, and one with posteromedial papillary muscle rupture.[41] A patient with both ventricular septal defect and then cardiac rupture who died within 24 hours of the onset of acute posteroinferior myocardial infarction. At autopsy, a single-vessel disease involving the right coronary artery was found, which hinted that an isolated disease of the right coronary artery can produce unexpected, fatal mechanical complications.[42] A 45-year-old man had a 4-year chest pain, occurred cardiac rupture. At postmortem, an acute large postinfarction of the left ventricle with perforation of the ventricular septum, and intramural dissection of the right ventricle which extended through the right ventricular epicardium near the apex.

Timing of rupture has been studied by several centers.[39] Severe patients with combined left ventricular free wall rupture and ventricular septal rupture during acute myocardial infarction, mean 69 years, the interval from

onset of chest pain to death was 21 hours to 28 days (median 8 days). The interval from onset to a new heart murmur was 15 hours to 6 days (mean 3 days, median 4 days).[43] Two cases and also reviewed three cases of double structural rupture consisting of ventricular septal rupture and subsequent free wall rupture. Time of ventricular septal rupture after acute myocardial infarction was 24 hours to 4 days, and time of free wall rupture from onset of acute myocardial infarction was 8 hours to 21 days.[37] Ten 10 cases of double structural rupture, time from onset to ventricular septal rupture ranged from 1 hour 35 minutes to 13 days, from onset to free wall rupture 25 hours to 21 days, from ventricular septal rupture to free wall rupture 35 minutes to 17 days. Concerning the relation between ventricular septal rupture and free wall rupture, usually free wall rupture occurred after ventricular septal rupture, even after repair of ventricular septal rupture[37,44] However,[45] described 2 cases of sequential left ventricular free wall and ventricular septal ruptures with a rupture interval of 20 days and 2 days, respectively. In some cases, double structural rupture was noticed in autopsy and the sequence of the occurrence of the ruptures was unknown[37]. At least one patient was noticed to have a simultaneous ventricular septal rupture and free wall rupture. The predictors of double structural rupture were estimated as patients' age, first onset of acute myocardial infarction, a history of hypertension, male gender, Killip Class II or above, cardiotonic or thrombolytic agents, delayed reperfusion, increased right ventricular load and reinfarction, etc.[37] The risk factors of the myocardial infarction were extensive transmural, anterior, involving adjacent interventricular septum.[46]

The survival rate of the patients with a double structural rupture was 20%. An urgent operation should be carried out on diagnosis.[37] Cases of successful surgical repair of combined ventricular septal perforation and free wall rupture after acute myocardial infarction have been reported in which Teflon was the material of choice.[46-48] An 80-year-old female had an onset of anterior acute myocardial infarction for two days, was noted a left-to-right shunt at ventricular level by Swan-Ganz catheter. At operation, right ventricular free wall rupture was found. Apical ventricular septal rupture 15×10 mm not adjacent to the right ventricular tear repaired with Teflon, and the right ventricular tear was 11repaired with Teflon pledget. The patient survived.[48] Their techniques in double structural rupture repair, in the management of 4 cm posterior-inferior left ventricular free wall rupture and 1.5×2 cm ventricular septal rupture at the apex side of the infracted free wall. Teflon pledgetted sutures brought together the ventricular septal rupture, a Dacron was placed on the left ventricle side, and free wall rupture was reconstructed with Teflon felt patch. As a result, the patient survived. In summary, myocardial rupture complications after acute myocardial infarction are infrequent. Ventricular septal rupture or free wall rupture occurs after transmural infarction, but papillary muscle rupture may follow subendocardial infarction. Papillary muscle rupture is more common after inferolateral infarction; septal rupture complicates anterior and inferior infarctions with equal frequency; free wall rupture is equally frequent in all segments[49] Almost all the patients with myocardial rupture experienced cardiopulmonary collapse, and required intraaortic balloon pumping support and urgent angioplasty, and were bridged to emergent operation. If a patient's condition could not improve after active medical management of an acute myocardial infarction, a myocardial rupture should be considered. Prompt diagnosis and timely surgical treatment are essential for survival.

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