Advances in the study of Acute Myocardial Infarction Complicated by Cardiac Rupture

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Abstract: *Cardiac rupture is a catastrophic complication after acute myocardial infarction. With the widespread use of primary percutaneous coronary intervention and improved pharmacological treatment, the incidence of cardiac rupture complicating acute myocardial infarction is on the decline, but patient mortality remains high and remains a clinical problem that remains to be addressed. Cardiac rupture is classified according to the location of the rupture into ventricular free wall rupture , ventricular septal rupture and papillary muscle rupture. The aim of this review is to highlight key risk factors, clinical presentations and diagnostic findings of cardiac rupture that may contribute to the early diagnosis of mechanical complications and to review management strategies after cardiac rupture in recent years.*

Keywords: *Acute Myocardial Infarction, Ventricular Free Wall Rupture, Ventricular Septal Rupture, Papillary Muscle Rupture, Risk Factors*

1. Introduction

Cardiac rupture (cardiac rupture, CR) is a catastrophic complication of acute myocardial infarction (AMI). CR is classified according to the location of the rupture into ventricular free wall rupture (FWR), ventricular septal rupture (VSR) and papillary muscle rupture (PMR). With the widespread use of primary percutaneous coronary intervention (pPCI) and the improvement of pharmacological treatment, the incidence of CR complicating AMI has been decreasing, with recent studies reporting a decrease in the incidence of CR to 0.27%-0.91% after ST-Segment Elevation Myocardial Infarction (STEMI)[1,2].However, patient mortality remains high and is still a clinical problem that remains to be addressed. The aim of this review is to highlight key risk factors, clinical presentations and diagnostic findings of cardiac rupture that may contribute to the early diagnosis of mechanical complications and to review management strategies after cardiac rupture in recent years.

2. Rupture of the free wall of the ventricle

The incidence of FWR is 2%-6% in the pre-perfusion era and decreases significantly with the advent of the perfusion era in the Global Registry of Acute Coronary Events (GRACE) [3] ,the incidence of FWR was 0.19%, with an in-hospital mortality rate of 58%[4].The average time to diagnosis of FWR is 2.6 days [5]. According to the Becker typing [6] Type I is characterized by sudden tearing without myocardial thinning, mostly in the acute phase of AMI (<24 h); Type II is characterized by slow tearing with localized myocardial erosion; Type III is characterized by thinning of the damaged myocardium to form a ventricular aneurysm that eventually develops into perforation, usually in the late phase of myocardial infarction (>7 d). Studies of the criminal vessels associated with FWR rupture found that 42% were left anterior descending (LAD), 40% were left circumflex artery (LCX) and 18% were right coronary arteries (RCA) [7]. In another study of FWR, the site of rupture was identified intraoperatively in 35 patients, of whom 15 (42.9%) had ruptures in the lateral wall, 10 (28.6%) in the inferior wall, 6 (17.1%) in the anterior wall and 4 (11.4%) in the posterior wall [5].

Patients with the following factors are more likely to develop FWR: age > 55 years, female, no history of myocardial infarction, complete occlusion of LAD, transmural myocardial necrosis, hypertension, Killip> Class I, persistent ST-segment elevation, pharmacological thrombolysis, use of glucocorticoids and non-steroidal anti-inflammatory drugs [3, 8-10]. In contrast, VFWR is less likely to occur in patients with previous diabetes, peripheral vascular disease, angina attacks, old MI, triple coronary vasculopathy

or congestive heart failure [11, 12]. It is hypothesized that the reduction in cardiac rupture may be due to coronary collateral circulation formation, myocardial ischemic preadaptation, and myocardial scar formation. yuan Fu et al [13].found that women (OR=2.90, 95% CI 1.21-6.9,P<0.05), older patients (OR=7.358, 95% CI 2.31-16.42,P<0.05) were significantly associated with the occurrence of CR. Most studies suggest an increased risk of CR in older and female patients. In an animal study, a higher incidence of CR was observed in older mice (12 months of age) than in younger mice (3 months of age), and pathological examination revealed a stronger inflammatory response and extracellular matrix damage in the infarcted myocardium of older mice [14]. This evidence supports the idea that clinically CR is more likely to occur in older patients. In clinical practice, most female AMI patients are older, so the effect of gender may be at least partially attributable to advanced age. Early recognition of these factors and close observation for early signs of cardiac rupture to achieve early detection and treatment may help to reduce mortality.

Typical symptoms of left ventricular free wall rupture after myocardial infarction are persistent or recurrent chest pain, syncope, and cardiogenic shock [15-17]. Oliva et al [18].reported that more than 80% of people with ventricular rupture presented with at least two of the following symptoms: recurrent chest pain, vomiting, irritability and anxiety. Hemodynamic stability depends on the amount of pericardial hemorrhage, the rate of bleeding [19]. The presence of rapid and massive hemopericardium can lead to sudden electrical-mechanical separation and sudden death [20]. In another single-center retrospective study, over 80% of patients with ventricular rupture presented with pericardial tamponade[5]. The late onset of ventricular rupture may be associated with excessive exertion such as coughing and vomiting. Signs of pericardial tamponade, including dilated jugular veins, paradoxical pulses, hypotension and distant heart sounds, may be seen on examination, while acute pulmonary oedema is relatively uncommon [4, 21].

For cardiac rupture, transthoracic ultrasound (TTE) should be the first choice, with a sensitivity and specificity of 70% and 90% [22]. The TTE should be performed immediately if there is clinical suspicion of a ventricular free wall rupture [11, 23]. Cardiac ultrasound can detect pericardial effusion, intrapericardial blood clots, and right ventricular diastolic collapse [4]. Transesophageal ultrasound (TEE) provides better visualization of cardiac structures and may provide additional diagnostic value in the case of ventricular free wall rupture [24]. If the patient is hemodynamically stable, enhanced cardiac CT or MRI imaging can help to clarify the size and location of the rupture and exclude stable aortic coarctation [25]. If there is no progressive leakage of blood from the ruptured heart, ventriculography may not observe evidence of contrast leakage and thus fail to confirm the diagnosis of cardiac rupture [4]. The above tools are only performed after a failed TTE or TEE.

Pericardial puncture and drainage can rapidly improve the state of hemodynamic deterioration caused by pericardial tamponade, but emergency pericardiocentesis remains controversial in patients with FWR. By reducing cardiac tamponade, it reflexively increases blood pressure, which subsequently leads to an increase in myocardial contractility, thereby accelerating the transformation of a small tear into a full myocardial rupture [26]. Therefore, pericardiocentesis should be used as a temporary means to buy time for cardiac repair surgery. During the preparation for the procedure, it is necessary to maintain satisfactory hemodynamics with positive inotropic drugs, intravenous fluids, intra-aortic balloon counterpulsation (IABP) or extracorporeal membrane pulmonary oxygenation (ECMO) [5, 25]. A conservative strategy of intravenous hydration, IABP and pericardial puncture drainage is preferable in patients where surgery is contraindicated [27]. Subsequent management includes strict bed rest for 1-2 weeks, systolic blood pressure control (< 120 mmHg) and beta-blockers given as soon as tolerated [17, 27].The patient should be given beta-blockers as soon as they are tolerated. It is also important to keep the bowels open and avoid emotional distress. However, it is worth noting that patients on medication have a 90% in-hospital mortality rate [28].

In terms of surgical management, the principles of left ventricular FWR are to reduce pericardial tamponade, close the fissure and prevent re-rupture of the ventricle or the formation of a ventricular pseudoaneurysm. Due to the high mortality rate of FWR, immediate surgical treatment is indicated once the diagnosis or high suspicion of FWR has been made [25]. Preoperative coronary angiography may be considered to assess coronary lesions, but this should depend on the hemodynamic stability of the patient and the urgency of the procedure. The prognosis is significantly improved by intraoperative coronary artery bypass grafting [25]. Postoperative IABP reduces intraventricular pressure and to some extent prevents postoperative cardiac re-rupture [29]. The current operative mortality rate is approximately 24%-50% and the total in-hospital mortality rate is approximately 50%-60% [30].

3. Ventricular septum rupture

The incidence of VSR has fallen to 0.17%-0.21% [1, 2]. Ventricular septal rupture has a high mortality rate, 46% within the first week of conservative treatment and 62%-82% within 2 months [31]. Postoperative mortality is 40% [2]. In the absence of myocardial reperfusion, there are two peak periods for septal rupture, 24 h after myocardial infarction and 3-5 days after myocardial infarction, rarely more than 2 weeks [31]. With thrombolysis and PCI, the median time to VSR is reduced to 24h after AMI [32, 33].

VSRs are classified as simple or complex depending on the form of rupture [31]. Simple in that the perforated channel runs directly through the septum and the perforation is at the same level on both sides. In complex cases, irregular, jagged channels are present in the necrotic myocardium. The upper two thirds of the septum is supplied by the LAD and the lower third by the RCA. However, in some cases, the LAD extends beyond the apical part of the left ventricle to supply the distal inferior wall and inferior septum. Rupture of the septum in anterior wall myocardial infarction often involves the apical region and is simple; in contrast, rupture of the septum in inferior wall myocardial infarction involves the inferior posterior base and usually presents as a complex [34, 35]. Patients with the following risk factors are more likely to develop VSR: female, elderly patients, hypertension, lack of history of angina pectoris and myocardial infarction [32, 33]. Angina and myocardial infarction lead to myocardial ischemic preadaptation and coronary collateral flow formation, which reduces the likelihood of septal rupture [36].

Symptoms of septal rupture include chest pain, dyspnea and symptoms associated with low cardiac output [31]. The patient's hemodynamic stability depends on the left-to-right fractional flow, which depends on the pressure difference between the right and left ventricles and the size of the septal rupture. Septal perforation usually leads to acute pulmonary oedema and right-sided heart failure, and once hemodynamically disturbed, the patient enters decompensation with rapid progression to cardiogenic shock, characterized by oliguria, clammy skin and altered mental status. VSR after AMI usually results in a new all-systolic murmur radiating to the apex and axilla, with palpable tremor in the parasternal. If the patient is in cardiogenic shock, the murmur and tremor are not significant.

When VSR is suspected, TTE or TEE is the diagnostic tool of choice [37, 38]. Characteristic findings of TTE include right ventricular dilatation, pulmonary hypertension, septal perforation and left-to-right flow across the septum [39]. TEE should be considered in cases where there is a high suspicion of VSR and the diagnosis cannot be confirmed by TTE [39]. Due to the widespread use of echocardiography, pulmonary artery catheterization is rarely used. In patients with ventricular septal rupture, oxygen saturation is increased in both the right ventricle and the pulmonary artery as a means of distinguishing increased pulmonary oxygen saturation due to severe mitral regurgitation [40]. Left ventriculography is rarely used to diagnose VSR, and coronary angiography should be performed to assess the coronary arteries if septal repair is being considered in conjunction with revascularization.

Internal treatment should aim to stabilize hemodynamics and buy time for surgical treatment, including respiratory support, IABP, and vasodilators to reduce afterload. Hypotensive patients usually require positive inotropic support. VSR almost always requires surgery and early closure by surgery or intervention improves the prognosis [41]. A recently published review of the Society of Thoracic Surgeons national database (STS database) identified 2876 patients aged 18 years or older who underwent post AMI VSR repair between 1999 and 2010 with an overall operative mortality rate of 42.9%, an elective operative mortality rate of 13.2%, an emergency operative mortality rate of 56.0% and a salvage operative mortality rate of 80.5% [42]. In the GUSTO-I trial, the 30-day mortality rate for non-operative patients was 94% [33]. This suggests that conservative treatment has a high mortality rate. The timing of surgery has been much debated in the past. One study found that surgery after 6 weeks had a longer survival compared to early surgery [43-45]. Patients who underwent surgery within 7 days of presentation had a 54.1% mortality rate, compared to 18.4% if repair was delayed until 7 days later [42]. Papalexopoulou et al [46]. A systematic evaluation of VSR after myocardial infarction found that early surgery was recommended if the VSR was greater than 15 mm with significant shunting resulting in hemodynamic compromise; if the patient was hemodynamically stable, surgery could be delayed up to 3-4 weeks; and if clinical deterioration occurred, urgent surgery should be performed. In summary, surgery is now an effective treatment for VSR, and patients undergoing elective surgery have a higher survival rate and better surgical outcomes. Therefore, individualized and precise treatment needs to be given according to the individual patient. There is controversy over whether to perform both revascularization and septal repair barker et al. [47] reported that incomplete myocardial revascularization was a significant predictor of late mortality after surgical repair of VSR. Therefore, in patients undergoing surgical repair of VSR, simultaneous coronary artery bypass grafting (CABG) is recommended for all stenotic coronary arteries, including those supplying non-infarcted areas. With the

development of interventional techniques, transcatheter ventricular septal occlusion may be an alternative to surgical repair. In a systematic evaluation the authors included 13 studies and found that transcatheter septal occlusion had a success rate of 89%, with 46% of patients having surgery within 2 weeks of detection of VSR and a 30-day mortality rate of 32% [48].The 2018 systematic evaluation comparing medical, transcatheter septal occlusion and surgical treatment of VSR showed that 30-day mortality was significantly higher with medical treatment than with surgery or transcatheter septal occlusion (92% and 61% and 33%, respectively). There was no significant difference in mortality between patients who underwent surgery or transcatheter septal occlusion within 14 days (56% and 54%, respectively), while after 14 days, patients who underwent surgery had a significantly higher mortality rate than those who underwent percutaneous intervention (41% and 16%) [49]. Therefore, for suitable patients, transcatheter septal occlusion at may be an alternative to surgical repair and has shown better results.

4. Papillary muscle rupture

In the PCI era, the incidence of PMR after AMI was 0.26% [2]. PMR most often occurs within 2-7 days of AMI, with a median time of 13 hours [21, 26]. Papillary muscle rupture is more likely to occur in older, hypertensive, single vessel occlusion patients and less frequently in patients with diabetes and previous myocardial infarction [50]. The posterior medial papillary muscle is more likely to rupture than the anterior papillary muscle because the posterior medial papillary muscle is supplied only by the posterior descending artery, whereas the anterior papillary muscle receives a dual blood supply from the LAD and LCX.

The clinical presentation depends on the degree of mitral regurgitation, with severe mitral regurgitation after PMR characterized by acute pulmonary oedema, hypotension and cardiogenic shock. On examination a full systolic murmur can be heard in varying degrees, from severe to no murmur. The murmur may be inaudible due to reduced regurgitant jet volume and rapid equilibration of ventricular and atrial pressures because of left ventricular dysfunction. TTE is the preferred diagnostic modality with a sensitivity of 65-85%; however, when PMR is highly suspected and TTE is not diagnosed, TEE may be required as it is 95%-100% accurate [51, 52].

Medical treatment includes support with oxygen, vasodilators, IABP and positive inotropic drugs. PMR requires urgent surgery, otherwise 90% of patients will die within the first week [53]. When PMR is more complete, surgical repair is often difficult to accomplish because the tissue is very fragile. Segmental prolapse with partial PMR and limited adjacent tissue damage can usually be reliably repaired surgically[54, 55]. Mitral valve surgery for PMR has a very high operative mortality rate; however, this has decreased in recent years due to advances in surgical techniques and concomitant revascularization procedures. The long-term prognosis of postoperative patients is good, with a 5-year survival rate of 60-70% [56, 57]. In a study by Russo et al. [57] found that patients who survived PMR surgery had a similar prognosis to those who did not have PMR. Concomitant coronary revascularization should be performed as it has been shown to improve short and long-term survival in these patients [54, 57].

5. Cardiac rupture prevention

The most effective strategy for preventing CR after AMI is currently pPCI early revascularization. Treatment with ACEI/ARB and beta-blockers is cardioprotective and helps prevent CR [3, 60]. Moderate inflammation after myocardial infarction contributes to the healing of damaged myocardium, whereas excessive inflammatory responses can lead to adverse outcomes. Local inflammation after myocardial infarction is a major feature of the infarcted myocardium, with monocyte, neutrophil and macrophage infiltration as the main alterations. ACEI/ARB prevents cardiac in the acute phase of AMI by blocking the release of monocytes and neutrophils from the spleen, thereby attenuating systemic and regional inflammatory responses rupture [60]. β-blockers mechanism for preventing CR is unclear; one explanation is sympathetic excitation after myocardial infarction, which increases heart rate, raises blood pressure and enhances myocardial contractility, leading to CR. β receptor blockers prevent cardiac rupture after AMI by inhibiting sympathetic excitation. Ivabradine, another heart rate slowing drug (selective and specific inhibition of cardiac pacing If currents), was found in animal studies to prevent cardiac rupture after myocardial infarction by lowering heart rate with Ivabradine (rupture rates of 8% and 26% in the treatment and control groups, respectively, p<0.05), while blood pressure was comparable between the two groups and improved post-MI Survival [61]. Therefore, it may be effective in patients with high heart rates after myocardial infarction who do not adequately tolerate beta-blockers or for whom beta-blockers

are less effective.

6. Conclusion

CR is a serious complication after AMI and should be treated with early revascularization and standardized medical medication. Patients with AMI should be assessed for risk, and patients at high risk should be closely monitored for early diagnosis and timely intervention in the event of CR.

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