Review Article

Post-Myocardial Infarction Left Ventricular Free Wall Rupture: A Review

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Abstract

Left ventricular free wall rupture (LVFWR) is a rare striking complication of acute myocardial infarction [AMI]. It can occur in one of two types: either acute lethal form or a subacute form where a blood clot in rare circumstances seals the defect and results in the formation of a ventricular pseudoaneurysm. A high index of suspicion and close monitoring of patient's symptoms and signs are necessary for diagnosis. Urgent Transthoracic echocardiography (TTE) is the gold standard for definitive diagnosis of AMI complications such as LVFWR. Multi-Detector Computed Tomography (MDCT) is a suitable alternative if the diagnosis is doubtful or to exclude other etiologies of hemopericardium. Cardiac Magnetic Resonance (CMR) is mainly used in stable patients with subacute LVFWR or pseudoaneurysm for more defined tissue characterization. Pericardiocentesis is not a routine procedure and it is done only as an emergency desperate measure in hemodynamically unstable patients while a surgical repair is prepared. Despite the high surgical mortality rates, urgent surgical repair is still the rule for treatment of LVFWR using pericardial patch closure or less frequently infarctectomy with patch placement and ventricular wall reconstruction. Recently sutureless techniques started to play a major role with improved mortality in patients with LVFWR with careful follow-up is required for the risk of recurrent rupture or pseudoaneurysm formation.

Keywords: Left ventricular free wall rupture (LVFWR); Acute myocardial infarction (AMI); Thrombolytic therapy; Pericardiocentesis; Infarctectomy; Sutureless techniques

Introduction

Aim of the review

The aim of this review article is to highlight one of the gravest complications of AMI which is LVFWR and to enlighten physicians about its incidence, types, risk factors, clinical presentations, diagnostic modalities and different treatment options.

Incidence

One of the life-threatening complications of AMI is myocardial rupture which is directly responsible for mortality in 8% of AMI patients. [1] A fatal yet quite rare form of this complication is LVFWR, that takes place in about 2% of cases. [2] but nowadays in the era of percutaneous coronary intervention (PCI), it is less frequently encountered. [3] Still, it is a lethal complication. [4]

Risk factors

The classical risk factors of LVFWR are elderly patients (usually ≥55 years and commonly in-between 65 and 70 years) [5-8], female gender (although no definitive sex bias has been reported but relatively more common in females due to lower reported incidence of AMI in females). [9-12] With higher incidence of arterial hypertension [9,13-15] and no previous anginal attacks (due to lack of collateral circulation), [9,16,17] Also in the first episode of transmural anterior or lateral AMI [9,14-16] without overt heart failure symptoms. [4,7,9,14,16,18] Although urgent reperfusion either by PCI or less frequently used thrombolytic therapies is considered a necessity to reduce the risk of LVFWR but studies showed that late or failed thrombolysis are usually linked to increased rates of LVFWR. [19-21] Also, PCI independently reduces the risk of LVFWR in comparison with thrombolysis. [22] (clinical characteristics of LVFWR patients are summarized in Table 1. In addition to classical risk factors, other triggering factors for LVFWR are the presence of persistent arterial hypertension (⩾ 150 mm Hg) during the 1st 24 hours of the acute infarction while in hospital at rest, and any undue physical effort such as persistent coughing, vomiting, or agitation [10,11,18,23-25] [Table 2].

Killip classification [26] is used for risk stratification of AMI patients into:

- **Killip Class I:** No clinical signs of heart failure
- **Killip Class II:** Mild heart failure
- **Killip Class III:** Acute pulmonary oedema

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- **Killip Class IV:** Cardiogenic shock

Table 1: Clinical characteristics of LVFWR patients.

Age >55 years (67 average)
Female gender
First trans-mural anterior or lateral myocardial infarction
No overt heart failure symptoms (Killip class I or II*)
Persistent ST-segment elevation
Persistent or recurrent chest pain
Sudden or progressive hypotension or sudden electromechanical dissociation

Table 2: Additional factors facilitating LVFWR. [27]

Delayed hospital admission (> 12–24 hours)
Persistent systemic hypertension during first > 10–24 hours
"Unusual" in-hospital physical effort (agitation, repetitive vomiting or coughing, etc)
Extension of myocardial infarction
Expansion of myocardial infarction

Clinical Presentation

LVFWR classically causes manifestations within the 1st day but it can occur up to 1 week after an attack of AMI. [12] The clinical scenario depends on the rate and the amount of the accumulated pericardial bleeding. In most cases of LVFWR sudden hemodynamic collapse is followed by death. In some cases, a blood clot will seal pericardial leaks and form a left ventricular pseudoaneurysm. [27,28] The hallmark of the subacute variant of LVFWR is slow repetitive intermittent bleeding, which occurs in one-third of cases. [29,30] Dissimilar to classic LVFWR patients, the subacute variant patients may endure until emergency surgery is arranged. [12] Several studies have tried to describe the warning signs and symptoms of fatal LVFWR. [10,20,29-31] Prodromal manifestations include persistent chest pain, intractable vomiting, restlessness, also electrocardiogram (ECG) signs as persistent S-T segment elevation, and positive T wave deflection that persists for 72 hours after the onset of chest pain. [10,29] Other classic signs of cardiac tamponade, including pulsus paradoxus and diastolic pressure equalization, may not exist in most of the cases [Table 3]. [30]

Table 3: Types of LVFWR according to time of occurrence or form of presentation. [27]

Time of occurrence	
Early rupture (≤ 48 hours)	Delayed hospital admission
	Persistent pain (> 4–6 hours)
	Acute arterial hypertension
	Frank and persistent ST-segment elevation
	Recurrent chest pain
	Persistent ST segment elevation
Late rupture (> 48 hours)	"Undue" physical exercise
	Infarct extension
	Infarct expansion
Form of presentation	
Acute rupture	Acute tamponade with sudden electromechanical dissociation or severe hypotension
Subacute rupture	Moderate to severe pericardial effusion:
	(A) with tamponade and haemodynamic compromise with modest or progressive hypotension
	(B) without tamponade

Diagnosis

TTE is still the gold standard for the definitive diagnosis of LVFWR. A pericardial effusion or intrapericardial echoes are the usual findings, less commonly a right-sided heart collapse or the actual tear may be seen. TTE offers a 100% sensitivity and 93% specificity for LVFWR diagnosis [Figures 1 and 2]. [30,32,33]

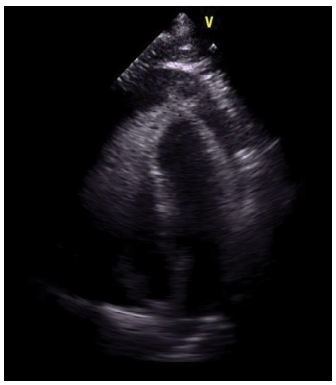

Figure 1: TTE apical 4 chamber view showing a circumferential pericardial effusion with a blood clot at a suspected site of ventricular rupture. [33]

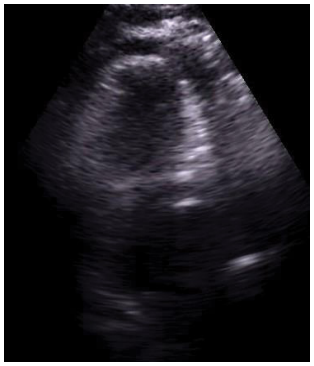

Figure 2: TTE apical 4 chamber view showing apical LVFWR. [33]

MDCT has been shown to be an effective modality for the detection of LVFWR and it may be beneficial to guide the diagnosis especially in cases when the diagnosis is doubtful or to exclude other causes of hemopericardium such as aortic dissection [Figures 3, 4 and 5]. [34,35]

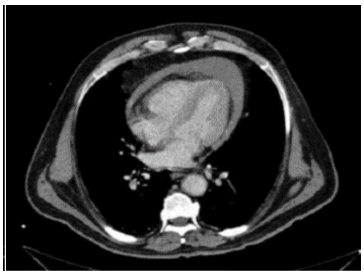

Figure 3: Non-contrast MSCT chest showing area of massive pericardial effusion. [33]

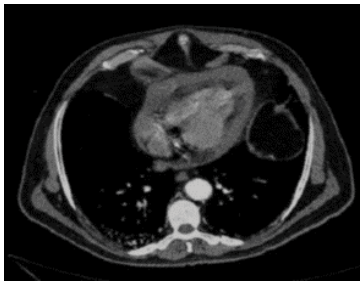

Figure 4: Contrast MSCT chest showing sealed ventricular rupture with a hematoma. [33]

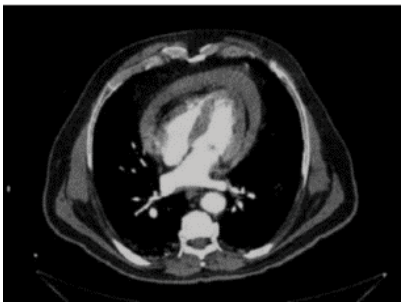

Figure 5: Contrast MSCT chest showing sealed ventricular rupture with a massive pericardial effusion. [33]

CMR is definitely not the modality of choice in acute settings when hemodynamic instability is the case, however, in subacute hemodynamically stable cases, CMR can help to determine the specific anatomical location of LVFWR, ventricular true or pseudoaneurysms, therefore planned surgical intervention can be prepared. In addition, it may delineate areas of ischemic myocardium even in the absence of ECG changes consistent with ischemia also it can show areas of the myocardium at risk of impending rupture [Figure 6 and Table 4]. [36,37]

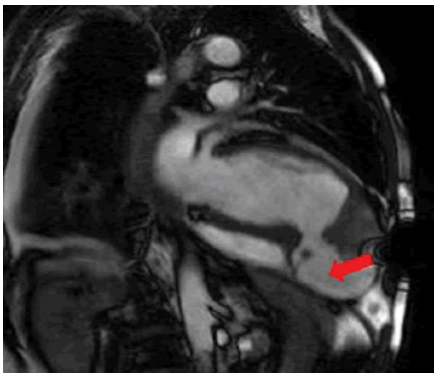

Figure 6: CMR showing left ventricular pseudoaneurysm connected to the left ventricular cavity through a narrow neck. [38]

Table 4: Management of acute and subacute free wall rupture. [27]

Acute phase (sequential course according to response)
Oxygenation/ventilation
Colloid infusion
Dobutamine
Pericardiocentesis if hemodynamically unstable (start with 10–50 ml)
Cardiac massage
Surgical treatment
Maintenance phase
Withdrawal of dobutamine
Blood pressure control with β blockers (systolic 100–120 mm Hg)
Prolonged rest (5–10 days)
Avoidance of physical exercise
Echocardiography (every 2–3 days)

Some articles suggested that conservative medical therapy might be of value in patients with LVFWR and pseudoaneurysm formation especially when hemodynamic stability is rapidly achieved. [38-40]

Rapid fluid infusion and administration of positive inotropic agents are considered the cornerstone of the temporary supportive measures until definitive management is planned. [40]

Emergency pericardiocentesis is not recommended except as a final measure to regain hemodynamic stability if cardiac tamponade occurred while urgent surgery is arranged as it bears the risk of chamber perforation and it may theoretically compromise the effect of a blood clot due to displacement or decompression of the previously contained rupture with further bleeding and accumulation of pericardial effusion. [12,33,40]

Discussion

Intra-aortic balloon pump is a widely used therapy in cases of ventricular septal rupture complicating AMI [41] However, its benefit in LVFWR is yet still debatable except in cases of persistent ischemia and/or LV pump failure. [12,40] Although the highly reported operative mortality which is 40% of cases [42-44] but the ultimate management of LVFWR is emergency surgical repair: the widely accepted surgical option which is associated with a better outcome is LVFWR closure by pericardial patch placement using either biological glue or epicardial sutures. [45,46] Infarctectomy with patch placement and aneursymectomy with ventricular wall reconstruction are less commonly used surgical techniques. [42,45] Pledgeted sutures without infarctectomy, and pericardial, Dacron, Goretex, or Teflon patches adhered with biologic glue or sutures. [30] Recently there is a shift towards the sutureless techniques because of its simplicity, effectiveness and avoidance of friable myocardial tissue. [47]

Sutureless techniques include a fibrin tissue-adhesive collagen fleece TachoSil® (Takeda, Osaka, Japan) combined with bovine pericardial patch anchored by fibrin glue in a case of oozing postinfarction cardiac rupture, [47] Teflon felt (C R Bard Inc, Billerica, MA, USA) attached using BioGlue (Cryolife Inc., Kennesaw, GA, USA) used for an acute LVFWR, [48] gelatin-resorcin-formalin (GRF glue: Cardial, Saint-Etienne, France) applied to a bovine pericardial patch (Impra, Tempe, AZ, USA) [49] and collagen fleece with fibrinogen-based impregnation (Tachocomb; Nycomed Pharma, Linz, Austria). [50] Although sutureless repair is promising in most cases, careful follow-up is mandatory because of the risk of recurrent rupture or ventricular aneurysm or pseudoaneurysm formation at the site of the sutureless repair in some patients. [47,51] Urgent coronary angiography to determine coronary anatomy for a planned coronary artery bypass is permitted in the hemodynamically stable subacute patients [52] Although the combination of surgical repair with coronary artery bypass grafting is most often advised, off-pump repair has been reported. [52] This approach is very beneficial because 80% of patients who had LVFWR are also multi-vessel coronary artery disease patients. [52,53] Some reports claim that operative mortality is 15% while the 5 years postoperative survival rates are 85% after successful surgical repair [Figure 7]. [54,55]

Conclusion

LVFWR is a disastrous complication that is rarely encountered nowadays in the era of PCI. A high index of clinical suspicion together with bedside TTE is necessary for prompt diagnosis. Despite the high surgical risk, surgical repair remains the gold standard for managing this rare condition. Recent advances in surgical techniques such as sutureless techniques are associated with better outcome.

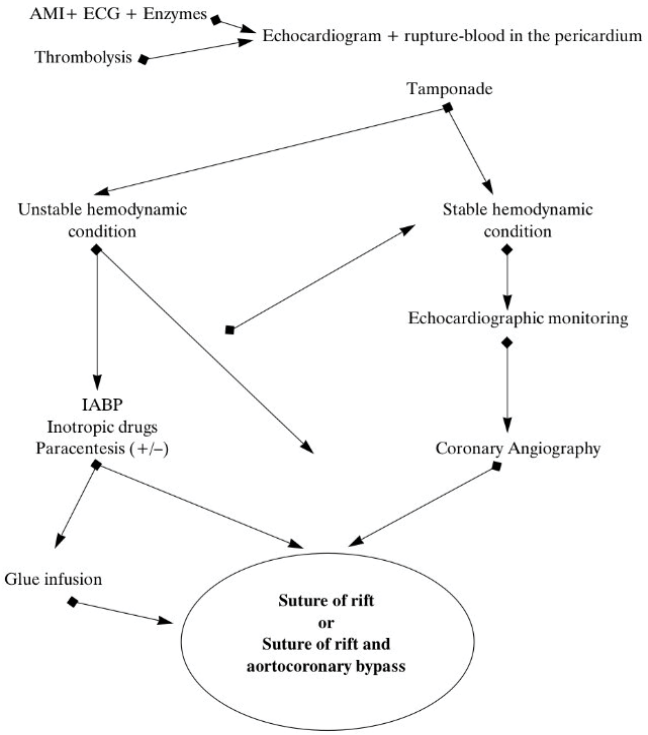

Figure 7: Summarization of LVFWR diagnosis and management. [55]

Conflict of Interest

All authors disclose that there was no conflict of interest.

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