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Sutured and sutureless repair of postinfarction left ventricular free-wall rupture: a systematic review

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Summary

Postinfarction left ventricular free-wall rupture is a potentially catastrophic event. Emergency surgical intervention is almost invariably required, but the most appropriate surgical procedure remains controversial. A systematic review, from 1993 onwards, of all available reports in the literature about patients undergoing sutured or sutureless repair of postinfarction left ventricular free-wall rupture was

performed. Twenty-five studies were selected, with a total of 209 patients analysed. Sutured repair was used in 55.5% of cases, and sutureless repair in the remaining cases. Postoperative in-hospital mortality was 13.8% in the sutured group, while it was 14% in the sutureless group. A trend towards a higher rate of in-hospital rerupture was observed in the sutureless technique. The most common cause of inhospital mortality (44%) was low cardiac output syndrome. In conclusion, sutured and sutureless repair for postinfarction left ventricular free-wall rupture showed comparable in-hospital mortality. However, because of the limited number of patients and the variability of surgical strategies in each reported series, further studies are required to provide more consistent data and lines of evidence.

Keywords: Cardiac rupture • Acute myocardial infarction • Mechanical complication • Cardiac surgery

INTRODUCTION

Left ventricular free-wall rupture (LVFWR) is a potentially fatal complication following acute myocardial infarction (AMI). The incidence of postinfarction LVFWR does not exceed 2% [1, 2]. Notwithstanding, it is estimated that 24%-61% of all deaths due to AMI are a consequence of this complication [1, 3, 4]. Such an event is second only to heart failure and ahead of arrhythmia as a cause of post-AMI in-hospital death [5].

LVFWR is associated with transmural myocardial infarction and usually occurs in the first week after AMI, depending on age, the thrombolytic treatment and admission delay [6–10]. Although AMI-related LVFWR usually proves fatal, some patients with acute or subacute rupture present a window of opportunity for intervention [11]. Under these conditions, prompt diagnosis and surgical treatment are mandatory to salvage the life of the patient. Operative mortality remains, anyhow, as high as 40% [12].

Different surgical techniques have been proposed to treat LVFWR. Initially, direct closure or infarctectomy and closure of the defect with a prosthetic patch have been the standard approaches [13–20]. Recently, the use of epicardial patch secured to the heart with glues or anchoring sutures is increasing in application [11, 21–24].

The optimal surgical treatment for LVFWR, nevertheless, remains still controversial because of the limited number of patients and variability of surgical strategies in each reported series. Particularly, the surgical procedure and results, according to the type of rupture and related surgical repair adopted, remain to be well defined.

In an attempt to clarify this issue, we performed a systematic review of studies reporting in-hospital outcomes of the different surgical approaches (sutured and sutureless technique) used to treat postinfarction LVFWR.

MATERIALS AND METHODS

Definitions

Sutureless repair (STL), or technique, was defined as a repair of the LVFWR using a collagen sponge, or pericardium patch fixed on epicardium with glues, to cover the infarcted myocardium. Sutured repair (ST), or technique, was defined as a repair of LVFWR using sutures to close myocardial tear or to secure a patch on the epicardium. In-hospital mortality was considered any death, regardless of cause, occurring within 30 days after surgery.

Data sources and search strategy

This systematic review was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [25]. PubMed, EMBASE and the Cochrane Central Register of Controlled Trials were searched from January 1993 to August 2018. Search terms were 'myocardial infarction' AND 'ventricular rupture' OR 'cardiac free-wall rupture'. The literature was limited to articles published in English. References of articles were reviewed manually and crosschecked for other relevant reports.

Studies were included if they met all of the following criteria: (i) human study; (ii) studies reporting postoperative outcomes of LVFWR repair; and (iii) studies comparing ST and STL. Moreover, only postinfarction wall ruptures and ruptures of the left ventricle were considered. Exclusion criteria were as follows: (i) animal studies; (ii) studies not reporting the surgical technique used to repair LVFWR; and (iii) studies in which it was not possible to assess the clinical outcomes of each single technique (ST versus STL). Reviews, case reports reporting only 1 or 2 cases and brief communications were not considered. Two independent reviewers (C.C. and M.M.) selected the studies for inclusion, extracted studies, as well as patient characteristics of interest and relevant outcomes, using an appropriate data collection form. Any divergences were resolved by a third reviewer (R.L.). Preoperative demographic data (mean age and clinical presentation), the type of LVFWR (oozing or blow-out), intraoperative use of cardiopulmonary bypass (CPB) and surgical techniques were recorded along with patient outcome (intraoperative death and postoperative in-hospital mortality) and in-hospital complications (rerupture, bleeding requiring reoperation, ventricular pseudoaneurysm formation, acute heart failure and cerebrovascular events). The 2 reviewers independently evaluated each study using the modified Newcastle Ottawa Scale, which is meant to assess the risk of bias when considering case series and case reports for review articles [26].

RESULTS

The PRISMA flow diagram, describing the study selection process along with reasons for exclusion, is presented in Fig. 1. After removal of reports not pertinent to the design of the current review, 25 studies that met explicit inclusion criteria remained, including a total of 209 patients with a mean age of 68.4 years and a predominance of male gender (59.4%). Of those patients, 93 patients (44.5%) underwent STL and 116 (55.5%) patients underwent ST. Most commonly (77.5%, 117/151), LVFWR was of the oozing type. Cardiac arrest was observed before surgery in 17.5% (29/166) of the subjects. Main characteristics of studies and patients are listed in Table 1. The 2 groups (ST and STL) of patients were similar to each other regarding gender, mean age and clinical presentation. From a surgical management standpoint, the majority of patients were operated on CPB (54.9%, 113/206), and concomitant coronary artery bypass grafting (CABG) was performed in 26.8% (56/209) of the patients.



PRISMA Flow Diagram

Figure 1: The PRISMA flow diagram. PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

Intra-aortic balloon pump (IABP) was inserted in 46.7% (71/152) of the cases. Operative data are presented in Table 2. Intraoperative mortality occurred in 1.4% (3/209) of the subjects, while in-hospital mortality was 13.9% (29/209) (Table 3). Postoperatively, rerupture occurred in 3.3% (7/209) of the patients, whereas bleeding requiring reoperation and cerebrovascular events in 2.7% (4/146) of patients and in 6% (10/167) of patients, respectively. Cerebrovascular events included severe neurological injury, vegetative stroke and ischaemic stroke. Ventricular pseudoaneurysm formations were not detected (0/183). Postoperative complications are shown in Table 4. Cause of mortality included low cardiac output syndrome (n = 13), acute kidney injury (n = 1), multiorgan failure (n = 3), rerupture (n = 5), sepsis (n = 2), stroke (n = 2) and inability to wean from CPB (n = 3).

All the selected studies reported either moderate or serious risk of bias. However, given the overall high risk of bias along with the limited number of studies and patient enrolled, all articles were retained for the purpose of this review.

Sutureless repair of left ventricular free-wall rupture

A total of 93 patients were treated with STL. Most commonly (90.2% of the time, 83/92), LVFWR was of oozing type. Cardiac

arrest was observed before surgery in 15.8% (12/76) of the subjects. Only 25.8% (24/93) of the cases were operated on CPB, and concomitant CABG was performed in 21.5% (20/93) of cases. IABP was inserted in 37.7% (20/53) of the patients. In-hospital mortality was 14% (13/93), while no intraoperative death was observed (0/93). Postoperatively, rerupture and cerebrovascular events occurred in 5.4% (5/93 and 5/92 respectively) of the subjects; no bleeding requiring reoperation and ventricular pseudoaneurysms formation were detected (0/84 and 0/90 respectively). Cause of mortality included low cardiac output syndrome (n = 7), rerupture (n = 4), sepsis (n = 1) and stroke (n = 1).

Sutured repair of left ventricular free-wall rupture

A total of 116 patients were treated with ST. Most commonly (57.6%, 34/59), LVFWR was of oozing type. Cardiac arrest was observed before surgery in 18.9% (17/90) of the subjects. The majority of the patients (78%, 89/113) were operated on CPB, and concomitant CABG was performed in 32.8% (38/116) of the cases. In 51.5% (51/99) of the subjects, an IABP was inserted. Intraoperative death was observed in 2.6% (3/116) of the patients, while in-hospital mortality was 13.8% (16/116). Postoperatively, rerupture occurred in 1.7% (2/116) of patients, bleeding requiring reoperation in 6.5% (4/62) of patients and

Table 1: Published studies and patient baseline characteristics

First author [reference]	Year of the study	Number of patients	Mean age (years)	Cardiac arrest ^a (n)	Mean time from AMI to WR (days)	Oozing rupture (n)	Blow-out rupture (n)
Padrò [21]	1993	13	64.6	2	3.8	11	2
Coletti [27]	1995	5	63	0	NA	3	2
Schwarz [28]	1996	5	64.4	0	13.7	3	2
Zeebregts [29]	1997	5	72.2	0	NA	3	2
Nwogu [30]	1998	3	73.3	0	NA	3	0
Imagawa [31]	2000	3	76.7	0	NA	3	0
Kamohara [32]	2000	8	76.3	3	3.5	5	3
Park [33]	2000	4	67	1	NA	3	1
Prêtre [34]	2000	5	68.8	3	4.2	3	2
lemura [35]	2001	17	65.4	NA	NA	14 (7)	3 (0)
Flajsng [36]	2002	24	62.1	0	NA	NA	NA
Lachapelle [22]	2002	6	71.8	4	NA	3	3
Mantovani [37]	2002	17	68	NA	3	NA	NA
Canovas [23]	2003	17	68	0	1.5	17	0
Nappi [38]	2003	7	59.4	3	4.3	NA	NA
Chen [39]	2004	4	62.5	2	NA	NA	NA
Okada [40]	2005	7	66.7	2 (1)	2.8	2 (2)	5 (0)
Leva [41]	2006	9	68	0	5.1	9	0
Fujimatsu [42]	2007	5	76.2	4 (2)	NA	1 (1)	4 (2)
Mishra [43]	2007	6	61	1 (0)	1.5	5 (1)	1 (0)
Carnero-Alcázar [11]	2009	21	74.5	3	2	21	0
Sahibzada [44]	2009	6	57	0	NA	NA	NA
Lee [45]	2013	3	67.3	1 (0)	NA	1 (1)	2 (0)
Raffa [46]	2013	6	74.7	NA	5	4	2
Aoyagi [47]	2014	3	80.7	NA	NA	3	0
Total		209	68.4	29	4.2	117	34

Values in parentheses refer to sutureless group when both techniques are employed in the study.

^aBefore surgery.

AMI: acute myocardial infarction; NA: not available; WR: wall rupture.

Table 2: Operative data

First author	Number of	Sutureless	Sutured	CPB (n)	Concomitant	IABP ^a
[reference]	patients	repair (n)	repair (n)		CABG (n)	(n)
			1			
Padrò [21]	13	13	0	1	0	NA
Coletti [27]	5	0	5	5	0	0
Schwarz [28]	5	0	5	5	3	1
Zeebregts [29]	5	0	5	5	2	4
Nwogu [<mark>30</mark>]	3	0	3	NA	2	2
Imagawa [<mark>31</mark>]	3	3	0	0	0	NA
Kamohara [<mark>32</mark>]	8	0	8	8	0	8
Park [33]	4	0	4	2	2	1
Prêtre [34]	5	0	5	4	3	5
lemura [<mark>35</mark>]	17	7	10	12 (2)	8 (2)	15 (5)
Flajsng [<mark>36</mark>]	24	0	24	11	1	5
Lachapelle [22]	6	6	0	4	3	2
Mantovani [37]	17	1	16	17 (1)	11 (0)	NA
Canovas [23]	17	17	0	0	0	NA
Nappi [<mark>38</mark>]	7	0	7	1	0	5
Chen [39]	4	0	4	4	2	1
Okada [40]	7	2	5	5 (1)	0	5 (2)
Leva [41]	9	9	0	7	4	3
Fujimatsu [42]	5	3	2	5 (3)	1 (0)	3 (1)
Mishra [43]	6	1	5	6 (1)	1 (0)	4 (1)
Carnero-Alcázar [11]	21	21	0	0	11	3
Sahibzada [44]	6	0	6	5	1	0
Lee [45]	3	1	2	3 (1)	1 (0)	2 (1)
Raffa [46]	6	6	0	3	0	NA
Aoyagi [47]	3	3	0	0	0	2
Total	209	93	116	113	56	71

Values in parentheses refer to sutureless group when both techniques are employed in the study.

^aPatients supported postoperatively.

CABG: coronary artery bypass grafting; CPB: cardiopulmonary bypass; IABP: intra-aortic balloon pump; NA: not available.

First author [reference]	Number of patients	Intraoperative mortality (n)	IH mortality (n)	Low cardiac output ^a (n)	Other causes of IH death (<i>n</i>)
Padrò [21]	13	0	0	0	0
Coletti [27]	5	1	1	0	1 ^b
Schwarz [28]	5	0	0	0	0
Zeebregts [29]	5	0	0	0	0
Nwogu [30]	3	0	0	0	0
Imagawa [31]	3	0	0	0	0
Kamohara [32]	8	0	3	2	1 ^c
Park [33]	4	0	0	0	0
Prêtre [34]	5	0	1	0	1 ^d
lemura [35]	17	0	2 (1)	2 (1)	0
Flajsng [<mark>36</mark>]	24	1	1	0	1 ^b
Lachapelle [22]	6	0	1	0	1 ^e
Mantovani [37]	17	0	3 (0)	1 (0)	2 ^{d,f} (0)
Canovas [23]	17	0	4	2	2 ^{c,g}
Nappi [<mark>38</mark>]	7	0	0	0	0
Chen [39]	4	1	2	0	2 ^{b,g}
Okada [40]	7	0	1 (0)	0	1 ^d (0)
Leva [41]	9	0	0	0	0
Fujimatsu [<mark>42</mark>]	5	0	2 (0)	1 (0)	1 ^e (0)
Mishra [43]	6	0	2 (1)	2 (1)	0
Carnero-Alcázar [11]	21	0	4	2	2 ^g
Sahibzada [44]	6	0	0	0	0
Lee [45]	3	0	0	0	0
Raffa [46]	6	0	1	1	0
Aoyagi [47]	3	0	1	0	1 ^g
Total	209	3	29	13	16

Table 3: Operative outcome and causes of IH death

Values in parentheses refer to sutureless group when both techniques are employed in the study.

^bInability to wean from cardiopulmonary bypass.

^cSepsis.

^dMultiorgan failure.

^eStroke.

fAcute kidney injury.

^gRerupture.

IH: in-hospital.

cerebrovascular events in 6.7% (5/75) of patients. In-hospital ventricular pseudoaneurysms formation was not observed (0/93). Cause of mortality included low cardiac output syndrome (n = 6), acute kidney injury (n = 1), multiorgan failure (n = 3), rerupture (n = 1), sepsis (n = 1), stroke (n = 1) and inability to wean from CPB (n = 3).

DISCUSSION

To the best of our knowledge, the current systematic review represents the first attempt to address the difference in in-hospital outcomes between STL and ST to treat postinfarction LVFWR.

LVFWR is a serious and more frequently a fatal mechanical complication of AMI. Although the incidence of postinfarction LVFWR ranges from 2% to 6.2% [15, 48–51], it is estimated that LVFWR is involved in 24%–61% of all deaths due to AMI [1, 3, 4, 15, 50, 52], and it is second only to cardiogenic shock [5, 53].

Most of the ruptures typically occur within the first 7 days following AMI [13, 49, 54], and this correlates with studies of changes in the biomechanical strength of myocardium after acute infarction in animal models [55], but may happen up to 2 weeks from AMI event [56]. Risk factors associated with the development of LFWR include age older than 55 years, female sex, arterial hypertension, no history of previous AMI, totally occluded left anterior descending, transmural infarction and delay of hospitalization [3, 48, 49, 57-59]. Early recognition of these factors in an AMI patient, such as signs of cardiac tamponade, may enhance a timely surgical intervention.

The relationship between the use of thrombolytics and the risk of LVFWR has been controversial. Some studies showed that thrombolysis had no impact on the incidence of rupture [60], while others reported an increased risk [8, 52]. In a meta-analysis, Honan *et al.* [61] demonstrated that thrombolytic therapy early after AMI improves survival and decreases the risk of cardiac rupture. However, late administration of thrombolytics, beyond 14 h of symptom onset, also appears to improve survival, but increases the risk of ventricular rupture. Coronary angioplasty improves mortality and morbidity of AMI, and when compared with thrombolytic therapy and pharmacological treatment, it reduces the risk of LVFWR and its concomitant fatality [62, 63].

Timely diagnosis is key to survival, as LVFWR can be surgically corrected if prompt intervention is performed. Higher degree of suspicion and the widespread availability of cardiac imaging have increased the number of early diagnosis. Clinical presentation depends on the rapidity of bleeding. Patients may present with

^aCause of death.

Tab	le 4:	Postoperative	complicat	ions and	hospital	l stay
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First author [reference]	Number of patients	Rerupture (n)	Bleeding (n)	Pseudoaneurysm (n)	Cerebrovascular events (n)	Mean length of hospital stay (days)
Padrò [21]	13	0	0	0	0	15
Coletti [27]	5	0	1	0	0	16.5
Schwarz [28]	5	0	0	0	0	NA
Zeebregts [29]	5	1	1	0	0	NA
Nwogu [30]	3	0	0	0	0	17
Imagawa [31]	3	0	0	0	0	NA
Kamohara [<mark>32</mark>]	8	0	1	0	0	NA
Park [33]	4	0	0	0	1	28.2
Prêtre [34]	5	0	NA	0	0	NA
lemura [35]	17	0	0	0	0	NA
Flajsng [<mark>36</mark>]	24	0	NA	0	NA	28.6
Lachapelle [22]	6	0	NA	0	1	13.6
Mantovani [37]	17	0	NA	NA	NA	NA
Canovas [23]	17	1	0	0	0	NA
Nappi [<mark>38</mark>]	7	0	0	0	0	NA
Chen [39]	4	1	NA	0	0	NA
Okada [<mark>40</mark>]	7	0	NA	NA	4 (2)	NA
Leva [41]	9	0	0	0	0	8.6
Fujimatsu [<mark>42</mark>]	5	0	0	0	2 (1)	NA
Mishra [<mark>43</mark>]	6	0	0	0	0	9
Carnero-Alcázar [11]	21	2	0	0	0	12
Sahibzada [44]	6	0	0	0	0	NA
Lee [45]	3	0	1 (0)	0	2 (1)	23.5
Raffa [46]	6	1	0	0	0	NA
Aoyagi [47]	3	1	0	0	0	NA
Total	209	7	4	0	10	17.2

Values in parentheses refer to sutureless group when both techniques are employed in the study. NA: not available.

cardiac arrest if bleeding is massive (acute rupture), or hypotension with cardiogenic shock if there is a slow progressive bleeding (subacute rupture) [13, 64].

The most important diagnostic method for LVFWR is transthoracic echocardiography [65]. The presence of pericardial effusion and diastolic compression of the right chambers are the 2 most relevant findings. It is important to keep in mind that pericardial effusion can be present in up to 28% of patients in a post-AMI period in the absence of cardiac rupture. The presence of echogenic masses on the epicardium increases both sensitivity (97%) and specificity (93%) [66]. Magnetic resonance imaging can enhance the diagnosis by identifying the contained ventricular rupture and its anatomical features [67]. The definitive diagnosis is usually made at surgery. Pericardiocentesis prior to surgery, which confirms a haemorrhagic effusion, may further support the diagnosis [68].

Different opinions exist regarding the opportunity to perform cardiac catheterization before surgery [16] or avoiding this investigation in order to 'save time'. Mantovani *et al.* [37], like other investigators [13, 41], demonstrated that LVFWR is not strictly related to a single diseased vessel, and concluded that coronary angiograms should be performed in stable patients as soon as LFWVR is suspected. Indeed, a proper revascularization of the diseased vessels supplying the non-infarcted area has been shown to exert a positive impact on survival and freedom of angina [37].

The treatment of LVFWR is almost exclusively surgical. Recently, a new therapeutic option, namely percutaneous intrapericardial fibrin-glue injection therapy, has been applied to patients with LVFWR in isolated case studies [69, 70]. Terashima *et al.* [71] reported an in-hospital mortality of 25%, thereby suggesting that, at present, such a technique should be only applied to patients with LVFWR and extremely high surgical risk due to poor general condition.

The method of surgical repair of LVFWR has varied over time and is more frequently individualized, depending on the state and location of the rupture. Two anatomopathological entities of LVFWR are described: the oozing type, characterized by a smaller tear or epicardial extravasation, which may be temporarily sealed by a clot or fibrinous pericardial adhesion, and the blow-out type characterized by active bleeding and a macroscopic tear in the epicardium [72]. In our analysis, oozing type rupture was the entity more frequently observed, in almost 80% of the cases, probably due to the fact that oozing rupture usually follows a subacute course, while blow-out ruptures are characterized by cardiac arrest, and death within a few min because of massive haemopericardium and tamponade.

Several different techniques to treat LVFWR have been described in the literature, but all can be referred to 2 different categories: STL and ST, depending on the use or not of sutures to treat the rupture of the ventricular wall. Initially, ST was the only method used. In the conventional approach, infarctectomy is followed by ventricle reconstruction using a prosthetic patch (Teflon[®] or Dacron[®]) or direct closure under CPB [14, 34, 35, 73]. More conservative procedures, such as linear (or direct) closure with horizontal mattress sutures buttressed by Teflon felt, can be also used [35, 74]. These techniques are challenging because the sutures should be tied through fragile myocardial tissue. To avoid these issues, many authors advocate just an on-lay patch (Dacron, Teflon or pericardium) technique, in which a patch is

anchored with a continuous running suture to the epicardium well beyond the ischaemic area [20]. Glue, applied under the patch, is considered by some surgeons [34, 35, 37] as a mandatory complement to this technique, as it increases the compression strength on the myocardial infarction region and prevents blood leakage to the epicardial surface along the suture line.

More recently, with the advent of tissue adhesives, STL have been developed and increasingly used [21]. In this technique, a patch (Dacron, Teflon, or pericardium) is secured to the infarcted myocardium with tissue adhesive (biological glues or synthetic cyanoacrylate monomers). As advocated by Muto *et al.* [75], collagen sponge patches (Tachosil[®] or TachoComb[®]) may be a valid alternative. The application of patches larger than the AMI- or lesion-related area avoids placing sutures through a friable myocardium, preserves left ventricular (LV) geometry and it can be done without CPB [75]. In accordance with this concept, we found that STL was performed on CPB in only one-fourth of cases, while ST was performed on CPB in the majority of cases. The major limitation of using a STL is represented by the fact that the glue is not effective in the presence of active bleeding.

The current review showed that ST and STL can be performed with a comparable (13.8 vs 14%) and relatively low in-hospital mortality, while a trend towards higher rate of in-hospital rerupture was observed in the STL, occurring in almost 5% of the cases when this technique was employed. Although in-hospital mortality appears to be similar using the 2 techniques, the type of procedure should account for the type and extension of the rupture and quality of the involved myocardial tissue. Although STL has also been successfully used for patients with blow-out rupture [22], this condition is probably best approached with ST, provided that sutures may be safely anchored onto myocardial wall around the infarction-related tear [76]. On the other hand, STL can be adopted with excellent results in patients with oozing type-rupture [22, 41]. Interestingly, Padrò et al. [21] reported a 100% survival rate among 13 patients with subacute LVFWR treated with STL. This result may be, in part, attributed to the fact that, in this series, no patient underwent surgery for acute massive ruptures. STL is a simple and fast option in the surgical treatment for LVFWR, but surgeons should be aware that it has a potential risk of rerupture after surgery, and its actual effectiveness in frank blow-out rupture is a matter of controversy.

Preoperative or intraoperative insertion of IABP is advocated by some authors even in the absence of haemodynamic instability [14, 29, 40]. Our analysis revealed that IAPB was inserted in almost half of the patients. Preoperative IABP implantation is advisable in patients with cardiogenic shock in order to achieve temporary haemodynamic stabilization on the way to surgery [38]. In addition, the use of IABP can prevent transition from the oozing to the blow-out type by decreasing LV end-systolic pressure [32]. Postoperatively, IABP, reducing afterload and LV wall stress, can be useful to avoid rerupture or to prevent/reduce other untoward event, such as pseudoaneurysm formation, especially when the STL is performed [35]. Finally, based on the high rate of postoperative events related to low cardiac output syndrome, IABP might be useful to reduce such a complication. Nonetheless, compelling evidence supporting such a concept is still not available.

In the last 2 decades, extracorporeal life support (ECLS) has emerged as a viable circulatory assistance in the presence of refractory cardiogenic shock, or cardiac arrest, for patients who develop LVFWR after AMI [32, 40, 42]. Indeed, ECLS may provide haemodynamic stabilization, playing thus an important role as a rescue/salvage circulatory support, and allowing the transition to surgical repair. Furthermore, ECLS may be useful also in the perioperative phase, particularly in the presence of large AMI with severely depressed LV function, avoiding the development of low cardiac output syndrome, the most common cause of death in these patients and favouring the myocardial recovery. Of note, Formica *et al.* [77] did not observe, in a recent study, any benefit of perioperative ECLS on in-hospital survival in patients with LVFWR. Also, the usefulness of ECLS in the setting of LVFWR repair requires, therefore, further investigation.

Limitations

This systematic review contains all the biases inherent to a systematic review. In particular, the major limitation is the quality of the studies available for the analysis. There were no prospective studies identified and more than half of the reports were case series, with a small number of patients, and including either only one technique or no controls. The inclusion of case reports accounts for a high risk of publication and selection bias. Longterm follow-up was not considered due to limited amount of data. We also acknowledge the lack of some critical information, such as preoperative LV ejection fraction and morbid conditions, which may have impacted the postoperative outcome.

CONCLUSION

LVFWR after AMI is more frequently a dreadful complication with a rather high mortality despite prompt surgical treatment. Early detection of initial signs of LVFWR is crucial for successful management. The definitive and standard treatment is emergency surgical repair. Surgical techniques, ST or STL, can be performed with comparable in-hospital mortality. However, because the existing literature provided limited and low-quality data, further studies are required to provide additional and more consistence data and evidence.

Conflict of interest: Prof. Roberto Lorusso is a Principal Investigator of the PERSIST-AVR Study sponsored by LivaNova (LivaNova, London, UK) and is Consultant for Medtronic (Medtronic, Dublin, Republic of Ireland). The other authors have no conflicts of interest to declare.

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