REVIEW ARTICLE

Post-Myocardial Infarction Left Ventricular Pseudoaneurysm: A Meta-Analysis

Shi-Min Yuan[1*]

[1]*Department of Cardiothoracic Surgery, The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian, Fujian Province, People's Republic of China*

Abstract

Left ventricular pseudoaneurysm (LVPA) is a rare complication of myocardial infarction. The clinical characteristics and treatment of choice in the current era remain to be elaborated. The present article aims to give an overview of post-infarct LVPA and discuss the management strategy and outcomes. The study was based on comprehensive retrieval of literature of the recent 10 years. Myocardial infarctions as the underlying causes of LVPAs were mostly acute myocardial infarctions. The mean time for LVPA formation was 51.7 months after myocardial infarction. Postoperative and follow-up ejection fraction values were significantly higher than preoperative. In the interventional group patients, the oversize ratio of patients in whom devices remained in position was a little larger than that of those with a migrated one, but lack of a statistical significance (1.32±0.25 vs. 1.25±0.21, p=0.707). The mortality rate was significantly higher in the conservative group than in the surgical and interventional groups. Post-infarct LVPAs are curable to surgical aneurysmectomy and left ventricular reconstruction, thereby avoiding unexpected LVPA ruptures and other fatal complications. Elderly patients and patients at a high operative risk may resort to interventional therapy. The conservatively treated patients inevitably carry a considerable risk of death. An oversize ration of >1.3 might be a reference value for preventing device migration in the interventional group patients.

Key Words: *Aneurysm, false, heart ventricles, cardiac surgical procedures.*

INTRODUCTION

Left ventricular pseudoaneurysm (LVPA) is a rare complication of myocardial infarction, cardiovascular surgery, trauma, or infection, with myocardial infarction being the most common etiology [1]. LVPA develops in 0.1% of patients following myocardial infarction [2]. LVPAs can be arising from the free wall, left ventricular outflow tract and mitral-aortic intervalvular fibrosa. The formation time of LVPA with various etiologies varies from 1 day [3] to 25 years [4]. According to the formation time, LVPAs can be divided into acute (onset <2 weeks) and chronic (onset >2 weeks) [5].

There are two types of concurrent LVPAs and true aneurysms of the left ventricle: mixed (conjunct true and false) and false-true superimposed [6]. Moreover, daughter aneurysms arising from the LVPA [6], multiple [7], multi-loculated [5, 8], apical tunnel-shaped [9] and recurrent LVPAs [10] have also been described. The diversity and heterogeneity of the pathological forms of LVPAs make diagnosis difficult and lead to error or delay.

LVPA is formed secondary to an incomplete rupture of the left ventricular wall contained by the pericardium [11]. Davutoglu [5] deliberately narrated the differences between false and true aneurysms: 1) The orifice to cavity ratio was 0.25–0.50 for false, and 0.90–1.0 for true aneurysms; 2) An inferior or posterior location is suggestive of pseudoaneurysm; and 3) The true posterior aneurysm is often associated with extensive infarction and the resultant severe mitral regurgitation, which are seldom seen in false aneurysms.

Post-infarct LVPAs commonly occur in patients with transmural myocardial infarction, where the whole thickness of the myocardium is involved. They are commonly located in the inferior or posterolateral walls [12]. When a pseudoaneurysm develops, serious complications, such as angina, heart failure, arrhythmias, thrombus with embolization and fatal rupture may occur [12]. Moreover, the differential diagnosis between LVPA and true aneurysm of the left ventricle is important, as the former often presents with heart failure and even cardiogenic shock and is more likely to rupture compared to true aneurysm [13]. Thus, the diagnosis and treatment of LVPA are still compelling and challenging. The present article aims to give an overview of post-infarct LVPA and discuss the management strategy and outcomes.

METHODS

Comprehensive retrieval of pertinent literature in the PubMed database, Google Scholar and "Baidu" Scholar was carried out for articles published between 2011 and 2021. The retrieval terms included "myocardial infarction", "post-infarction", "left ventricular pseudoaneurysm", "aneurysmectomy", "left ventricular reconstruction", "percutaneous closure", "cardiopulmonary bypass" and "cardiac surgical procedures". The inclusion criteria were clinical research, case series and case reports of left ventricular pseudoaneurysm following myocardial infarction. The exclusion criteria were

Corresponding author: Shi-Min Yuan, Department of Cardiothoracic Surgery, The First Hospital of Putian, Teaching Hospital, Fujian Medical University, 389 Longdejing Street, Chengxiang District, Putian 351100, Fujian Province, People's Republic of China; Email: shiminyuan@126.com

Received: January 04, 2022; Revised: January 22, 2022; Accepted: January 24, 2022
DOI: https://doi.org/10.37184/lnjpc.2707-3521.4.2

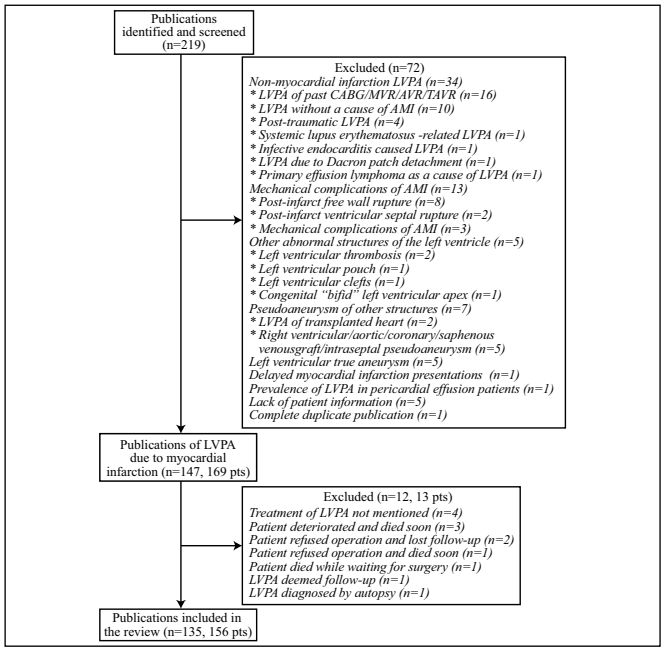

Fig. (1): A flow chart of literature inclusion and exclusion. AMI: acute myocardial infarction; AVR: aortic valve replacement; CABG: coronary artery bypass grafting; LVPA: left ventricular pseudoaneurysm; MVR: mitral valve replacement; pts: patients; SLE: systemic lupus erythematosus; TAVR: Transcatheter aortic valve replacement.

articles describing: LVPA with a cause of other than myocardial infarction (n=34), mechanical complications of acute myocardial infarction (n=13), pseudoaneurysm of other structures than of the left ventricle (n=7), left ventricular true aneurysm (n=5), other abnormal structures of the left ventricle (n=5), delayed myocardial infarction presentations (n=1), the prevalence of LVPA in pericardial effusion patients (n=1), lack of patient information (n=5) and complete duplicate publication (n=1). A total of 72 articles were excluded and 147 articles with 169 patients were retained. Nevertheless, in 13 of the 147 recruited articles, 13 cases of patients with LVPAs were not treated due to patient's sudden death or patient's refusal for surgical treatment, and therefore 12 of these 13 articles were excluded (one of the articles was retained as another case reported in it was eligible for the inclusion criteria). Finally, 135 articles with 156 patients were recruited into this study (**Fig. 1**).

IBM SPSS statistics 22 software was used for the statistical analysis. The measurement data were expressed in mean±standard deviation and compared with independent samples t-test. Categorical variables were compared by Fisher's exact test with continuity correction. $p<0.05$ was regarded statistically significant.

RESULTS

The recruited 135 articles [4, 8, 14–146] included 3 (2.2%) retrospective studies [8, 89, 109], 4 (3.0%) case series [23, 47, 57, 107] and 128 (94.8%) case reports [4, 14–22, 24–46, 48–56, 58–106, 108, 110–146] with a total of 156 patients all of whom were diagnosed with post-infarct LVPA. Gender of two patients was not given. In the remaining 154 patients, there were 114 (74.0%) male and 40 (26.0%) female patients ($\chi^2=71.1$, $p<0.001$) with a male-to-female ratio of 2.9:1. Their mean age was 64.2±11.8 (range, 28–90; median, 64.5) years (n=156).

Table 1: The locations of myocardial infarctions in 120 patients.

Location	*n (%)*
Inferior [15–17, 27, 30, 34, 36, 37, 42–44, 46, 47, 50, 52, 54, 55, 58–60, 62, 64, 66, 69, 71, 72, 75–77, 83, 84, 89, 90, 92, 96, 99, 101, 108, 109, 111, 114, 115, 123, 125, 126, 130, 139, 141]	57 (47.5)
Anterior [14, 24, 25, 29, 41, 47, 48, 56, 65, 67, 74, 93, 100, 105, 106, 112, 120, 127, 131, 133, 135, 138, 140]	23 (19.2)
Lateral [35, 39, 81, 94, 109, 113, 118, 145]	10 (8.3)
Non-ST elevated [26, 28, 86, 102, 107, 143, 144]	7 (5.8)
Inferolateral [21, 33, 61, 98, 128]	5 (4.2)
Inferoposterior/posteroinferior [68, 85, 89]	5 (4.2)
Posterior [63, 87, 89, 119]	4 (3.3)
Anterolateral [31, 45, 129]	3 (2.5)
Anteroinferior (apical)/inferoanterior [18, 32]	2 (1.7)
Anteroseptal [97]	1 (0.8)
Posterolateral [89]	1 (0.8)
Basal inferior & inferolateral [116]	1 (0.8)
Inferior, lateral & posterior [40]	1 (0.8)

Myocardial infarctions as the underlying causes of LVPAs were mostly acute myocardial infarctions, whereas there were 7 (4.5%) subacute and 7 (4.5%) chronic myocardial infarctions. In addition, there were 2 silent myocardial infarctions [80, 146] and 1 unrecognized myocardial infarction [4]. The locations of myocardial infarctions as the underlying causes of LVPAs were shown in Table **1**.

A previous surgical or interventional history was reported in 62 (39.7%) patients: percutaneous coronary intervention with or without stenting was reported in 31 (50%) patients, a previous heart operation (coronary artery bypass grafting, valve replacement/repair, free wall rupture/ventricular septal rupture repair, or left ventricular aneurysmectomy) was performed in 27 (43.5%) patients, and both percutaneous coronary intervention and heart operation in 4 (6.5%) patient. The time interval from myocardial infarction to LVPA formation was 51.7±24.0 (range, 0–276; median, 4)

Table 2: 200 symptoms in 121 symptomatic patients.

Clincial Presentation	*n (%)*
Chest pain [14–16, 21, 23, 28, 29, 34, 37, 40, 46–48, 51, 56, 60, 65, 69, 73, 81, 87, 89, 95, 103, 106, 108, 109, 111, 114, 117, 120, 122, 126, 136–138, 143, 144]	46 (23)
Dyspnea [15, 18, 22–25, 27, 30, 32–34, 42–45, 51, 53, 55, 58, 62, 64, 66, 71, 78, 80, 81, 85, 87–89, 99, 111, 117, 119, 121, 124, 126, 128, 138, 145]	40 (20)
Heart failure [8, 32, 34, 39, 47–49, 54, 68, 79, 80, 82, 84, 87, 99, 100, 109, 116, 123, 125, 130, 135, 138, 141]	34 (17)
Shortness of breath [20, 23, 26, 28, 29, 35, 37, 53, 74, 75, 102, 122, 131, 132]	14 (7)
Shock [15, 16, 38, 45, 49, 53, 60, 72, 81, 136]	10 (5)
Syncope [28, 31, 40, 80, 81, 89, 90, 129, 142]	9 (4.5)
Cough [46, 53, 71, 89]	4 (2)
Orthopnea [20, 55, 137, 119]	4 (2)
Anasarca/edema [53, 54, 91, 132]	4 (2)
Fatigue [20, 42, 88]	3 (1.5)

Clincial Presentation	*n (%)*
Anorexia [34, 91]	2 (1)
Chest discomfort [100, 121]	2 (1)
Nausea [34, 144]	2 (1)
Palpitations [18, 74]	2 (1)
Fatigue/tiredness [34, 53]	2 (1)
Weakness [30, 53]	2 (1)
Weight loss [58, 80]	2 (1)
Abdominal pain [42]	1 (0.5)
Agitated [81]	1 (0.5)
Asthenia [66]	1 (0.5)
Chest distress [25]	1 (0.5)
Confusion [80]	1 (0.5)
Disorientation [117]	1 (0.5)
Epigastric discomfort [112]	1 (0.5)
Epigastric pain [89]	1 (0.5)
Hemiparesis [132]	1 (0.5)
Hypotensive [53]	1 (0.5)
Interscapular pain [78]	1 (0.5)
Lightheadedness [91]	1 (0.5)
New systolic murmur [19]	1 (0.5)
Profuse sweating [81]	1 (0.5)
Shoulder pain [92]	1 (0.5)
Stridorous breathing [104]	1 (0.5)
Transient ischemic attack [132]	1 (0.5)
Verbal aphasia [72]	1 (0.5)

months (n=95).

On current admission, clinical presentations were described for 135 patients: 14 (10.4%) patients were asymptomatic, while 121 (89.6%) patients were symptomatic with a total of 200 symptoms (**Table 2**).

Table 3: The 258 diagnostic modalities for the diagnosis of LVPAs in 139 patients.

Diagnostic Modality	*n (%)*
Transthoracic echocardiography [4, 8, 15, 17–19, 21, 23, 25, 27-39, 41, 42, 44–48, 50–56, 60, 62, 63, 65–68, 70–76, 78–86, 88–92, 95–105, 107–115, 117, 119, 121–129, 131, 133–138, 140–146]	126 (44.2)
Computed tomography [20, 25, 35, 37, 40, 49, 57, 59, 63–65, 72, 76, 77, 79, 80, 87–92, 94, 99, 100, 103–105, 108, 111, 112, 114, 118, 119, 122, 124, 132, 133, 135, 142, 143, 146]	47 (16.5)
Magnetic resonance imaging [4, 17, 20, 28, 30, 35, 36, 39, 40, 46, 48, 53–55, 59, 61, 66, 68, 82, 89, 98, 101, 106, 110, 112, 113, 115, 128, 134, 138, 139, 146]	34 (11.9)
Left ventriculography [14, 21, 22, 31, 43, 62, 68, 70, 90, 95, 102, 109, 118, 121, 128, 143]	25 (8.8)
Transesophogeal echocardiography [26, 29, 45, 68, 109, 124]	18 (6.3)
Chest X-ray [29, 62, 63, 80, 103, 105, 111, 114, 146]	9 (3.2)
Three-dimensional transesophogeal echocardiography [17, 19, 26, 70, 73, 85, 88, 129]	8 (2.8)
Computed tomographic angiography [8, 14–16, 55, 106, 124]	8 (2.8)
Cardiac catheterization [64, 77, 79]	3 (1.1)
Coronary angiography [31, 66, 69]	3 (1.1)
Contrast echocardiography [31, 82]	2 (0.7)
Ultrasonography [49]	1 (0.4)
Myocardial perfusion imaging [112]	1 (0.4)

Table 4: The dimensions of the LVPAs.

Dimension	Mean±SD	Range	Median	n
Longth, mm	71.9±29.4	19-150	70	85
Width, mm	55.5±23.9	5-110	52	70
Hight, mm	47.7±14.4	20-74	47.5	22
Neck, mm	23.7±14.6	3.8-65	21	59
Neck- to - maximum diameter of pseudoaneurysm ratio	0.39±0.22	0.03-1.02	0.36	46

The 258 diagnostic modalities for the diagnosis of LVPAs in 139 patients was shown in Table **3**. It revealed a correct diagnostic rate of 99.2% (256/258). The diagnosis of LVPA was missed by computed tomography in 2 patients [73, 146], with a false-negative rate of 4.3% of computed tomography.

The LVPAs recurred in 6 (6/156, 3.8%) patients: 5 (83.3%) patients recurred once [31, 65, 77, 78, 143] and 1 (16.7%) patient with a ruptured LVPA had twice recurrences and the LVPA ruptured at each recurrence [21]. Moreover, one patient who underwent percutaneous closure of LVPA had twice device migrations [8]. Therefore, there were 166 LVPAs in the 156 patients. The dimensions of the LVPAs were listed in Table **4**.

In 9 (5.4%, 9/166) LVPAs of 9 (9/156, 5.8%) patients, a wide neck rather than a narrow neck was present. The necks of these 9 LVPAs measured 20–65 mm with a neck-to-cavity ratio of 0.54–1.02. However, they were still diagnosed as LVPA rather than a true aneurysm of the left ventricle. In 3 LVPAs of 3 patients, there were 2 orifices for each. In 2 of the above 3 patients, the second orifice communicated with the right ventricle. In another 2 patients, the LVPAs communicated with the right ventricle. Mural thrombus was noted in 44 (26.5%) patients. Pericardial effusions were present in 25 (15.1%) patients. Mitral valve regurgitation was present in 30 (19.2%) patients (mild in 7 (23.3%), mild to moderate in 4 (13.3%) (2 of them had mild aortic regurgitation), moderate in 10 (33.3%) (1 of them had concurrent mild to moderate tricuspid regurgitation), severe in 7 (23.3%), and unspecified degree in 2 (6.7%) patients). Besides, moderate tricuspid regurgitation was noted in 1 patient. In 5 (3.2%) patients, LVPAs ruptured and in one of these patients, LVPA ruptured 3 times. A concomittent true aneurysm was found in 7 (4.5%) patients: 2 (28.6%) were mixed and 5 (71.4%) were superimposed. Other associated conditions were ventricular septal rupture in 11 (7.1%), free wall rupture in 3 (1.9%), papillary muscle rupture in 1 (0.6%), tamponade in 5 (3.2%), infective endocarditis in 2 (1.3%), colon carcinoma in 2 (1.3%), dilatative alcoholic cardiomyopathy in 1 (0.6%) and symmetrical peripheral gangrene in 1 (0.6%) case.

Left ventricular ejection fraction was reported for 62 patients. It was described as normal in 2 patients [97, 143], and preserved in 1 patient [62]. The remaining 59 patients had an ejection fraction of 33.9±11.2 (range, 11-63; median, 32)%. The locations of 98 LVPAs were

Table 5: Locations of 100 LVPAs.

Locations	*n (%)*
Apical [4, 25, 26, 31, 41, 47–49, 66, 78, 104, 106, 127, 129, 131, 135, 140]	17 (17)
Inferior [22, 32, 46, 47, 58, 69, 72, 77, 79, 90, 92, 115, 125]	13 (13)
Lateral [27, 33, 39, 89, 93, 94, 112, 118]	11 (11)
Posterior [23, 30, 70, 87–89, 95, 102, 107, 126]	11 (11)
Inferolateral [20, 40, 55, 82, 83, 101, 111, 114]	8 (8)
Anterior [19, 99, 100, 105, 133, 142]	6 (6)
Anteroapical [8, 53]	5 (5)
Inferoposterior [42, 44, 76, 85, 130]	5 (5)
Anterolateral [74, 80, 124]	3 (3)
Basal inferior [16, 54, 71]	3 (3)
Basal inferolateral [15, 84, 139]	3 (3)
Apicolateral [138, 145]	2 (2)
Inferior, inferolateral [17, 123]	2 (2)
Inferoseptal [36, 96]	2 (2)
Lateral posterior/posterior lateral [122, 136]	2 (2)
Apical, mid-anterolateral, mid-inferolateral [98]	1 (1)
Inferoapical [50]	1 (1)
Infero-basal [21]	1 (1)
Inferolateral, anterolateral [108]	1 (1)
Inferoposterolateral [116]	1 (1)
Lateral, inferior [37]	1 (1)
Mid posterior [23]	1 (1)

described with apical LVPAs being the most common (**Table 5**).

Patients could be divided into 3 groups according to treatment methods: surgical 116 (69.9%) cases, interventional 17 (10.2%) cases and conservative 33 (19.9%) cases. Patient age was much younger in surgical than that of the other two group patients (**Fig. 2**).

Surgical operation of LVPA was performed in 116 cases including 6 recurrences. Twenty (16.9%) cases were operated on an urgent basis. The surgical indications were LVPA enlargement [47], LVPA with a potential to rupture [69, 48, 119], a potential for complications [48], and limited surgical field exposure owing to the previous cardiac operation with videoscopic guidance becoming an only choice [59]. The operational parameters of open surgery were shown in Table **6**.

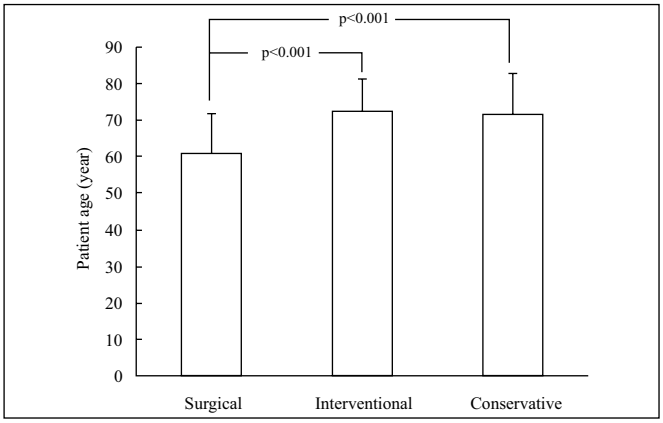

Fig. (2): Patient age was much younger in surgical than that of the other two group patients.

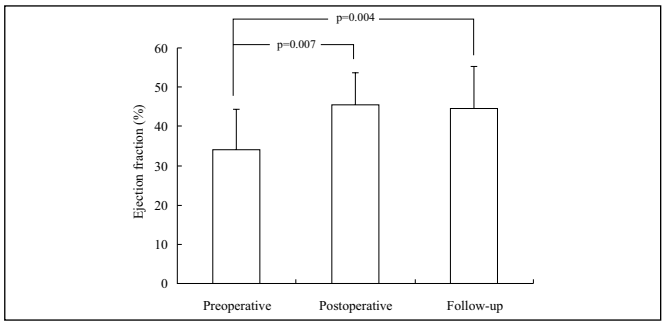

Fig. (3): Postoperative and follow-up ejection fraction values were significantly higher than preoperative value.

Table 6: The operational parameters of open surgery.

Operational Parameter	n (%)/Mean±SD (range; median)
Surgical approach (n=12)	
Sternotomy [22, 45, 53, 57, 68, 74, 86, 91, 127]	8 (66.7)
Thoracotomy [57, 76, 79]	3 (25)
Minithoracotomy (at the right 4th intercostal space for videoscopy) [59]	1 (8.3)
Cardiopulmonary bypass (n=33)	
Standard cardiopulmonary bypass [22, 45, 53, 55, 74, 91, 100, 109, 118, 127]	18 (54.5)
Normothermic cardiopulmonary bypass with on-pump beating-heart [53, 74]	2 (6.1)
Femorofemoral byapss [49, 57, 59, 65, 68, 76, 79, 86, 92, 109]	13 (39.4)
Cardiopulmonary bypass time	
Cardiopulmonary bypass time (min) (n=8)	179.3±91.3 (103–386; 165)
Crossclamp time (min) (n=6)	140.5±73.4 (74–278; 131)
Surgical procedure	
Aneurysmectomy [16, 17, 20, 22, 26, 29, 31, 33, 40, 42–51, 53–55, 57–59, 61–63, 68–71, 74–76, 78, 79, 82, 89, 91, 94, 95, 99, 102, 104, 105, 110, 113, 115, 118, 121–123, 126, 127, 129, 136, 145]	59 (50.9)
Left ventricular reconstruction [16, 17, 20, 21, 23, 26, 28, 29, 31, 33]	96 (82.8)
Coronary artery bypass grafting [35, 36, 38, 40, 42, 43, 45–47, 49–51, 53–55, 57–60, 62–65, 67–69, 71, 73–79, 82, 86, 89, 91, 92, 94, 95, 98, 99, 102, 104–107, 109, 110, 113, 115, 116, 118–123, 126–129, 133, 135, 136, 138, 141–146]	34 (29.3)
Valve operation	10 (8.6)
Mitral valve replacement [44, 50, 58, 59, 91, 109]	7 (70)
Mitral valve annuloplasty [31, 70]	2 (20)
Aortic and mitral valve replacements [109]	1 (10)
Ventricular septal rupture repair [36, 45, 78, 109, 121]	8 (6.9)
Free wall rupture repair [33]	1 (0.9)
Heart transplantation [66]	1 (0.9)
Left ventricular aneurysm resection [31]	1 (0.9)
MAZE procedure [102]	1 (0.9)
Chest wall reconstruction [65]	1 (0.9)
Right ventricular assist device [53]	1 (0.9)
Cardioverter-defibrillator implantation [77]	1 (0.9)

Table 7: Patch repair materials.

Patch Repair Material	n (%)
Bovine pericardium [16, 22, 26, 35, 50, 54, 64, 77, 86, 94, 100, 104, 116, 120, 128, 138]	16 (23.3)
Dacron [33, 46, 68, 69, 70, 76, 78, 95, 105, 107, 109, 118, 141]	14 (20.3)
Patch, unspecified [20, 43, 47, 49, 58, 62, 82, 98, 99, 109, 143]	11 (15.9)
Polytetrafluroethylene (PTFE) [51, 63, 102, 135, 136]	5 (7.2)
Autologous pericardium [109, 126]	3 (4.3)
Gore-Tex [109]	3 (4.3)
Pericardial patch [23, 31, 113]	3 (4.3)
Biological patch [65]	2 (2.9)
Bovine pericardial-Dacron (double) [45, 91]	2 (2.9)
Dacron-equine pericardium [57]	2 (2.9)
2 Bovine pericardia with polyester patch between [40]	1 (1.4)
Bovine-paricardiium (double) [79]	1 (1.4)
Dacron (double) [109]	1 (1.4)
Dacron-bovine pericardium [127]	1 (1.4)
Expanded polytetrafluoroethylene [55]	1 (1.4)
Equine pericardium, Teflon-backed [59]	1 (1.4)
Patch (double) [92]	1 (1.4)
Polyester tube graft tailored patch [110]	1 (1.4)

Techniques for left ventricular reconstruction were described for 82 cases: patch repair in 69 (84.1%) and primary suture in 13 (15.9%) cases. Patch repair materials were listed in Table **7**.

Postoperative and follow-up ejection fraction values were significantly higher than preoperative values (**Fig. 3**).

The surgical patients were on a follow-up of 21.4±47.6 (range, 0.8-288; median 12) months (n=41). Of the total 98 patients with a primary LVPA whose prognoses were indicated, 83 (84.7%) recovered, 6 (6.1%) recurred, and 9 (9.2%) died. The recurrence time of LVPAs was 35.0±51.5 (range, 0.3-120; median, 6) months (n=5).

Interventional therapy was conducted in 17 cases, including 1 patient developing twice device migrations [8] and 1 patient who recurred twice after the initial surgical treatment [21].

The indications for interventional therapy were: high risk of mortality, severe LV dysfunction and heart failure [84], comorbid conditions, previous myocardial infarction with reduced left ventricle function [93], redo operation [93, 97, 130, 131], advanced age [131], patient's refusal of surgical treatment [125] and an EuroScore estimated mortality of 15% [130].

An approach was given for 12 cases: via a retrograde approach in 8 (66.7%) (transfemoral in 5 (one patients with failed retrograde left femoral arterial access and converting to antegrade right femoral vein), transaxillary in 1 [84], retrograde unspecified in 2 [8, 21]), retrograde/transapical in 2 (16.7%) [8], transapical + transseptal in 1 (8.3%) [8] and transapical via a mini anterolateral thoracotomy in 1 (8.3%) patient [130].

In all 18 occluders were used in 17 patients: 11 (61.1%) Amplazter Septal Occluders, 2 (11.1%) Amplazter Muscular VSD Occluders [8, 21], 2 (11.1%) muscular ventricular septal defect occluders in 1 patient for 2 shunts [131], 1 (5.6%) Amplazter ASD [14], 1 (5.6%) RTM muscular VSD device [130] and 1 (5.6%) unspecified Amplazter device [134].

The overall oversize ratio of the devices was 1.28±0.22 (range, 1–1.6; median, 1.4) (n=10). The oversize ratio of the patients in whom devices remained in position was a little larger than that of those with a migrated one, but lack of a statistical significance (1.32±0.25 vs. 1.25±0.21, p=0.707).

They were on a follow-up of 13.3±16.4 (range, 1–60; median 6) months (n=12). Their outcomes were: recovered 14 (82.4%), complicated 2 (11.8%) and died 1 (5.9%).

Conservative treatment of LVPAs was applied in 33 cases. However, 3 of them had an alternative surgical/interventional procedure: surgical free wall rupture repair [143], surgical ventricular septal rupture repair [24] and percutaneous ventricular septal rupture closure (with an Amplatzer Septal Occluder) [15] in 1 patient each. The left ventricular ejection fraction of the conservatively treated patients were 33.8±14.0 (range, 10–63; median, 32.5)% (n=16) before treatment.

The indications for an interventional procedure were advanced age [34, 81, 90], being deemed too high risk, a calcific wall surrounding the pseudoaneurysmatic formation [37], previous cerebral infarction [85], patient decline of surgical treatment, patient's poor prognosis [25], patient's choice [96, 114], severe left ventricular dysfunction and multiple comorbidities [19], small LVPA [24, 143], lower risk of pseudoaneurysm rupture with limited estimated patient survival due to concurrent metastatic colonic cancer [4] and "the 'petrous' consistency which made resection impossible" [108].

The conservatively treated patients were on a follow-up of 15.4±17.2 (range, 1-61; median, 17) months (n=13). The ejection fraction after treatment was not reported in each patient. Outcomes of 7 patients were not described. In the remaining 26 patients, 12 (46.2%) were stable, 2 (7.7%) were improved, 1 (3.8%) was complicated with myocardial infarction, 1 (3.8%) recurred, and 10 (38.5%) died.

In overall, patients were on a follow-up of 18.7±38.8 (range, 0.8-288; median, 8.8) months (n=66). Outcomes of 141 cases were described: 99 (70.2%) recovered, 2 (1.4%) were improved, 3 (2.1%) were complicated, 7 (5.0%) recurred (including a second recurrence), 12 (8.5%) were stable, and 18 (12.8%) died (all were early deaths). In addition, all 8 recurrent cases (including twice chances of device migrations) warranted reinterventions with a reintervention rate of 4.8% (8/166). The mortality rate was significantly higher in the conservative group

than in the surgical and interventional groups (38.5% vs. 9.2% vs. 5.9%, χ^2=15.6, p<0.001).

DISCUSSION

Diagnosis

The diagnosis of LVPA should be considered in patients with poor responses to medical treatment or those with refractory heart failure. A physical examination may reveal to-and-fro heart murmurs [147]. Left ventricular angiography appears promising in revealing posterolateral LVPA; however, it is invasive. Nowadays, echocardiography, cardiac magnetic resonance imaging and computed tomography are alternative noninvasive diagnostic techniques with excellent visualization of LVPA [147]. By echocardiography, the entrance of the LVPA could be clearly visualized [148]. Computed tomography may sometimes not tell false from the true aneurysm, and probably inaccurately determine the location of the origin of LVPA [149]. Magnetic resonance imaging distinguishes among pericardium, thrombus and myocardium, displays disruption of the epicardial fat layer of the LVPAs, but is unable to discriminate mural thrombus from the LVPA walls thus leading to an inaccurate measurement of the size of LVPA [5].

Clinically, true aneurysms are often associated with ventricular tachycardia and (or) heart block and even sudden cardiac death [150], but this is uncommon in cases with an LVPA. Echocardiography may be helpful in the differentiation between true and false aneurysms usually by showing a narrow neck in LVPAs, equivalent neck to the cavity in true aneurysms [151]. Although transthoracic echocardiography is commonly used for the diagnosis of LVPA, transesophageal echocardiography seems superior to it in the evaluation of LVPA [53]. Histopathological examination of the excised ventricular wall helps to confirm the diagnosis [8].

Treatment

LVPAs <3 cm are usually stable [152], and are conservatively managed with acetylsalicylic acid, nitrates, angiotensin-converting enzyme inhibitor and β-blockers [153], and anticoagulants, *etc.* [154]. Patients with an LVPA with an increased size despite regular conservative treatment warrant interventional management [155].

Some authors proposed that due to the high propensity of LVPA rupture, patients with an LVPA are indicated for a surgical repair upon diagnosis is made [156]. In certain cases, LVPA represents a fatal complication of myocardial infarction and it necessitates an urgent operation [53]. However, whether a chronic LVPA needs emergency surgery is not clear [147].

In the surgical treatment of LVPAs, median sternotomy facilitates the establishment of cardiopulmonary bypass and cardioplegic arrest easily and securely. Nevertheless, exposure of a posterolateral LVPA via a median sternotomy seems to be of some difficulties in comparison with a left thoracotomy due to the deep location of LVPA [57]. On a few occasions, an anterior left thoracotomy [10, 157], a right lateral position, or an anterolateral mini-thoracotomy [9] was applied depending on the patient situation.

Prevention of LVPA rupture was the primary goal of repair [8]. Garrido *et al.* [158] emphasized that the surgical technique of choice should follow the principle of protecting the left ventricular geometry. The popular technique of LVPA repair was the endoventricular patch plasty technique (“Dor” procedure) [159]. Mitral regurgitation in cases of posterior LVPAs may be relieved after resection of the LVPA and does not need a valve replacement.

In the past, conservative treatment can be considered in patients who carry a high risk for operation [160]. Nowadays, percutaneous interventional therapy offers an alternative therapeutic possibility for such patients. The size of the devices should be larger [8] or at least equal to the neck of the orifice of LVPA. This suggestion was supported by the present study, where an oversize ratio of ≥1 was found.

Prognosis

Patients with an LVPA have high morbidity and risk of spontaneous rupture and sudden death [161]. Despite the high mortality rates for patients of LVPA without receiving a surgical operation, prolonged survival in some conservative patients was also reported [1]. The rupture rate was reported to occur in 30–45% of the patients [162]. Whereas, the present study demonstrated a much lower rupture rate of only 3.2%. This might be explained by the late formation of LVPAs at a mean of 51.7 months after myocardial infarction. Furthermore, death (9%), hemorrhage (7%) and arrhythmia (6%) were reported to be the more common adverse events of LVPAs. The recurrence rate of LVPA after surgical repair was 5% [1]. Postoperative mortality rates ranged between 7–29% [3]. The recurrence and mortality rates of the present study were similar to what have been reported in the literature. In addition, there was a report describing a self-cured LVPA [163].

This study revealed that the mortality rate of the surgical group patients was 9.2%, which was closer to but a bit better than the reported mortality of surgical patients 12.5% in 2006 [164]. The interventional therapy of LVPAs has not yet been reported in a large number of cases until present, and the overall prognosis of patients cannot be concluded with certainty. But recent reports showed good outcomes in small series of patients [165]. This study also showed that the surgical patients were much younger than the other two group patients. This hinted aged patients carrying high risks of operation resorted to interventional and conservative treatments. The left ventricular ejection fraction improved significantly after surgical aneurysmectomy and during follow-up, thus the operative effect for the treatment of LVPAs was in full proof.

Limitations still exist owing to the pertinent missing data from the reports. The post-infarct left ventricular ejection fractions in the interventional and conservative group patients were not available for comparisons and evaluations. Moreover, the predominance of transmural myocardial infarction, the influence of myocardial infarction on the functions of the papillary muscles and the mitral valve and subsequent treatment of choice of the mitral valve disorders, and the cut-off value of the oversize ratio indicating a possible device migration were not evaluated. These aspects need to be supplemented and assessed on the basis of more perfect data in the future.

CONCLUSION

Post-infarct LVPAs are curable to surgical aneurysmectomy and left ventricular reconstruction, thereby avoiding unexpected ruptures and other fatal complications. Aged and high-risk patients may resort to interventional therapy. The conservatively treated patients inevitably carry a considerable risk of death. An oversize ration of >1.3 might be a reference value for preventing device migration in the interventional group patients.

FUNDING

None.

CONFLICT OF INTEREST

The author declares no conflict of interest.

ACKNOWLEDGEMENTS

Declared none.

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