Percutaneous closure of left ventricular pseudoaneurysm

Shi-Min Yuan

The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian, China

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Abstract

The aim of the present study is to describe the indications, treatment effects, and patient outcomes of percutaneous management of left ventricular pseudoaneurysm (LVPA). The study materials were based on comprehensive literature retrieval since 2004. The mechanisms of LVPA formation can be divided into surgical, percutaneous, and medial disease related. Of the surgical mechanisms, coronary artery bypass grafting prevailed. The formation time was the longest in medical disease-related LVAPs up to 44.4 months. The percutaneous procedures succeeded on the first try in 79 (84.9%) patients, whereas failures were encountered during the percutaneous manoeuvres in 14 (15.1%) patients. Percutaneous closure of LVPA was especially indicated for patients carrying a high surgical risk. The iatrogenic traumas, such as left ventricular venting, should be avoided to prevent this complication. The preliminary cut-off valves of oversize 3.3 mm and oversize ratio 1.6 should be followed for reference for device choice.

Key words: cardiac surgical procedures, left ventricular pseudoaneurysm, transcatheter therapy.

Introduction

Left ventricular pseudoaneurysm (LVPA) is a rare but serious complication of myocardial infarction, cardiac surgery, trauma, and infective endocarditis [1]. Although patients can be asymptomatic, LVPA tends to rupture, leading to cardiac tamponade, shock, and death, and therefore requires an urgent repair [2]. In addition to rupture, LVAPs may have risks of thrombosis and coronary artery compression, and thus closure is recommended [3]. If left untreated, LVPA has a high risk of rupture – up to 45% within the first 3 months of LVPA formation [4]. The treatment of LVPA is challenging. Open surgery is the first-line treatment of choice [5]. However, Sakai et al. [6] reported that 7 patients with LVPA formation after mitral valve replacement were not associated with complication during conservative treatment. Percutaneous management of LVPA has been increasingly used in recent decades [7]. Hitherto there has been no systematic report of percutaneous management of LVPA.

Aim

The present article aims to describe the indications, treatment effects, and patient outcomes of percutaneous management of LVPA.

Material and methods

English-language literature was comprehensively retrieved in the PubMed, Google Scholar, and “Baidu” Scholar databases since 2004 [1–81]. The keywords entered in this search to identify articles were “pseudoaneurysm”, “left ventricle”, “left ventricular outflow tract”, “percutaneous”, and “transcatheter”. The inclusion criteria were clinical research, case series, case report, or proceeding abstracts on percutaneous treatment of LVPA of any aetiology. The exclusion criteria were articles describing the following: non-percutaneous treatment of LVPA (n = 21), pseudoaneurysm of other structures (n = 6), percutaneous closure of other defects (n = 4), and percutaneous treatment of LVPA but lack of patient information (n = 1). In total, 32 articles were excluded, and 66 articles were retained.

The data independently extracted from each study were article types, patient demographics, percutaneous manoeuvres and devices, therapeutic effects, and patient outcomes. Extraction of patient information was conducted carefully from each report. This process was replicated 4 times to avoid omissions and ensure the completeness and reliability of the information.

Statistical analysis

IBM SPSS statistics version 22.0 software was used for the statistical analysis. The quantitative data were expressed as mean ± standard deviation and were compared with independent samples t-test. Categorical variables were compared by χ² or Fisher exact test with continuity correction. P < 0.05 was considered statistically significant.
Results

The 66 recruited articles included 1 (1.5%) retrospective study [22], 4 (6.1%) case series [36, 48, 52, 65], 43 (65.2%) case reports [5, 7, 9–11, 13, 14, 17–21, 24, 25, 27–32, 34, 35, 38–40, 42, 43, 45–47, 49–51, 54, 55, 58, 60, 62, 63, 66–68, 71], 14 (21.2%) medical images [12, 15, 16, 23, 26, 33, 41, 44, 44, 53, 56, 59, 61, 69, 70], and 4 (6.1%) proceeding abstracts/posters [8, 37, 57, 64]. There were a total of 93 patients, including 58 (63.0%) male and 34 (37.0%) female ($\chi^2 = 12.5$, $p = 0.001$), with a male-to-female ratio of 1.7 : 1 (gender of 1 patient was unknown). Six (6.5%) patients were paediatrics [14, 24, 27, 35, 66, 69] and 89 (93.5%) were adults. Their mean age was 63.3 ±20.9 (range: 0.25–90; median: 68.5) years ($n = 78$). No age difference was noted between male and female patients (60.1 ±24.2 years vs. 65.5 ±18.1 years, $p = 0.318$).

All patients were diagnosed as LVPA. In 4 (4.3%) patients the LVPA s for percutaneous closure were recurrent [13, 29, 31, 63], and in the remaining patients the LVPA s were primary.

Table I. Mechanisms of LVPA formation

| Mechanism | N (%) |
|-----------|-------|
| Surgical operation: | |
| CABG ± left ventricular aneurysmectomy/ventricular septal rupture repair [8, 9, 17, 19, 45, 65, 69] | 47 (45.2) |
| Valve operation ± CABG/aorta operation [5, 11, 14, 22, 23, 26, 30, 36, 53, 54, 62, 67] (for infective endocarditis in 4 patients [14, 26, 30, 36]) | 17 (16.3) |
| Redo valve operation ± CABG [18, 33, 36, 49, 56] | 5 (4.8) |
| Apical left ventricular venting/wire perforation in heart operation [12, 20, 21, 48, 50] | 6 (5.8) |
| Aorta operation [36, 38, 52] | 4 (3.8) |
| Redo aorta operation [28, 52] (for infective endocarditis in 1 patient [52]) | 3 (2.9) |
| Free wall rupture repair [13, 29, 68] | 3 (2.9) |
| Ventricular septal rupture repair [10, 46] | 2 (1.9) |
| LVPA resection [31, 63] | 2 (1.9) |
| Congenital heart defect repair (Ross procedure [24], ventricular septal defect (Swiss-cheese) repair [66] and relief of left ventricular outflow tract obstruction with Brom’s technique [35]) | 3 (2.9) |
| Surgical repair of type II endoleaks [47] | 1 (1.0) |
| Postsurgical, unspecified [37] | 1 (1.0) |
| Percutaneous procedure: | |
| Transcatheter aortic valve implant: | |
| Transapical [15, 16, 25, 32, 41, 44, 51, 57] | 8 (72.7) |
| Unspecified [7, 34] | 2 (18.2) |
| Via right subclavian access [43] | 1 (9.1) |
| Transcatheter mitral valve implant: | |
| Transseptal [59] | 1 (50) |
| Transapical [58] | 1 (50) |
| Balloon aortic valvuloplasty [48] | 1 (0.7) |
| Transcatheter closure of ventricular septal defect and patent ductus arteriosus [27] | 1 (0.7) |
| Medical disease: | |
| Myocardial infarction [22, 37, 39, 40, 42, 60, 61, 65, 70, 71] | 12 (70.6) |
| Behcet disease [52, 55] | 3 (17.6) |
| Infective endocarditis [37, 56] | 2 (11.8) |

CABG – coronary artery bypass grafting, LVPA – left ventricular pseudoaneurysm.

In 1 patient, the mechanism of LVPA formation was not described [64]. Four (4/92, 4.3%) patients had 2 mechanisms for LVPA formation [52, 56, 65], while the LVPA formation in 88 (88/92, 95.7%) patients could be contributed to one mechanism. In short, there were in total 96 mechanisms for 92 patients (Table I). The mechanisms can be divided into surgical, percutaneous, and medial disease related. Of the surgical mechanisms, coronary artery bypass grafting prevailed. As for those owing to a valve operation, there were 12 (54.5%) mitral valve replacements, 7 (31.8%) aortic valve replacements, and 3 (13.6%) double valve replacements ($\chi^2 = 7.4$, $p = 0.025$).

The formation time of LVPA was reported for 53 patients. In 1 patient, it was described as “a few weeks” [24]. The mean formation time of the remaining 52 patients was 25.6 ±56.5 (range: 0.07–300; median: 4.5) months. The formation time was the longest in medical disease-related, longer in surgical, and shortest in interventional mechanisms of LVPA, but it did not reach a significant difference (44.4 ±101.7 vs. 29.7 ±56.7 vs. 6.4...
±8.1 months, \( p = \text{NS} \). No difference was found between the formation time among the surgical mechan isms, but a decreasing trend from apical left ventricular venting, to aortic, valvular and coronary operations was found (1.8 ±2.0 vs. 22.4 ±14.5 vs. 41.3 ±80.9 vs. 49.2 ±39.0 months, \( p = \text{NS} \)). The formation time did not differ between patients with a primary surgery and those with a redo operation (29.5 ±60.9 vs. 30.9 ±40.8 months, \( p = 0.960 \)). The formation time of LVPA did not differ between patients with left ventricular venting during open heart surgery and those with transapical transcatheter valve implant (1.8 ±2.0 vs. 3.2 ±3.9 months, \( p = 0.566 \)). The formation time due to apical trauma (both transapical transcatheter valve implant and left ventricular venting) was much shorter than that due to other surgical/interventional traumas, but it did not reach statistical significance (2.8 ±3.5 vs. 29.5 ±54.9 months, \( p = 0.117 \)). The formation time of LVPA due to myocardial infarction was much shorter than due to coronary artery grafting (2.9 ±3.0 vs. 49.2 ±39.0 months, \( p = 0.029 \)).

Clinical presentations of 52 patients were reported. Ten (19.2%) patients were asymptomatic [7, 31, 35, 36, 41, 43, 51, 52], and the remaining 42 (80.8%) patients had 59 symptoms, with chest pain and dyspnoea/shortness of breath being the most common (Table II). The ejection fraction of the patients was 31.6 ±13.9% (range: 16–55%; median: 25%) (\( p = 0.16–55\% \), median: 25%) (\( p = 0.029 \)). The growth speed of LVPA was 15.0 ±2.6 (range: 12–16.5; median: 16.4) mm/week (\( p = 5 \)) [5, 66, 71].

The three-dimensional sizes of the LVPA were 47.7 ±27.4 (range: 7–130; median: 40) mm (\( n = 61 \)), 38.1 ±20.4 (range: 8.6–90; median: 34.5) mm (\( n = 50 \)), and 30.7 ±18.4 (range: 10–90; median: 29.5) mm (\( n = 18 \)). The neck measured 10.0 ±6.8 (range: 2–32; median: 9) mm (\( n = 59 \)), and the neck-to-sac ratio was 0.22 ±0.13 (range: 0.03–0.57; median: 0.18) (\( n = 43 \)).

### Table II. Presenting symptoms

| Symptom                              | N (%) |
|--------------------------------------|-------|
| Chest pain                           | 12 (20.0) |
| Dyspnoea/shortness of breath         | 12 (20.0) |
| Heart failure                        | 10 (16.7) |
| Hemiparesis                          | 9 (15.0) |
| Orthopnoea                           | 2 (3.3) |
| Abdominal pain                       | 1 (1.7) |
| Acute pulmonary oedema               | 1 (1.7) |
| Anorexia                             | 1 (1.7) |
| Altered consciousness                | 1 (1.7) |
| Oedema                               | 1 (1.7) |
| Fatigue                              | 1 (1.7) |
| Fever                                | 1 (1.7) |
| Pulsatile chest wall mass            | 1 (1.7) |
| Stroke                               | 1 (1.7) |
| Syncope                              | 1 (1.7) |
| Tender, pulsatile epigastric mass    | 1 (1.7) |
| Weakness                             | 1 (1.7) |
| Weight loss                          | 1 (1.7) |

### Table III. Diagnostic techniques

| Diagnostic technique                  | N (%) |
|--------------------------------------|-------|
| Transthoracic echocardiography       | 46 (34.3) |
| Computed tomography                  | 29 (21.6) |
| Transoesophageal echocardiography    | 14 (10.4) |
| Magnetic resonance imaging           | 11 (8.2) |
| Left ventricular angiography         | 8 (6.0) |
| Angiography                          | 5 (3.7) |
| Magnetic resonance imaging           | 2 (1.5) |
| Three-dimensional transthoracic echocardiography | 1 (0.7) |
| Contrast fluoroscopic                | 1 (0.7) |
| Computed tomography-guided biopsy   | 1 (0.7) |
| X-ray film                           | 1 (0.7) |

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A mural thrombus inside the LVPA was found in 7 (7.5%) patients [13, 14, 25, 29, 31, 38, 46]. Three (42.9%) of these thrombosed LVPAAs were recurrent [13, 29, 31]. One (1.1%) LVPA was ruptured [13]. The locations of LVPAAs were reported for 69 LVPAAs of 67 patients, and apical LVPA was the most common (Table V).

The indications for percutaneous treatment of LVPAAs were described for 42 patients and previous operations/ sternotomies were a prevailing factor (Table VI).

The percutaneous closure of LVPA was performed on an urgent basis in 3 (3.2%) patients [13, 20, 23]. The percutaneous procedures succeeded on the first try in 72 patients in whom percutaneous manoeuvres twice and thus with one more approach [20]. Of the 8 failures, 5 (62.5%) were retrograde [10, 44, 51, 65, 70] and 3 (37.5%) were antegrade approaches [44, 51].

In 1 patient, the device used was not described. The devices that were used for LVPAA closures included 65 septal occluders, 18 plugs, and 16 groups of embolisation coils. The Amplatzer muscular ventricular septal defect occluder was the most commonly used (Table VIII).

The dimensions of septal devices were divided into 3 groups: moderate (well suited for closure of LVPA neck), small, and large. The size of the devices was much greater in moderate than in small groups ($p = 0.026$). A difference was found between small and large ($p = 0.050$). A difference was found in the oversize of the devices between moderate and small groups. The oversize ratio of

### Table V. The locations of 69 LVPAAs of 67 patients

| Location                        | N (%) |
|---------------------------------|-------|
| Apical [15, 16, 20, 21, 27, 32, 34, 38, 44, 48, 50, 51, 57, 58, 65, 66, 69] | 18 (26.5) |
| Left ventricular outflow tract [11, 18, 24, 26, 28, 33, 36, 52, 56, 62, 63] | 14 (20.6) |
| Anterolateral [22, 25, 31, 43, 49, 60] | 6 (8.8) |
| Paravalve [22]                  | 5 (7.4) |
| Inferolateral [17, 36, 48, 71]  | 4 (5.9) |
| Posteroalateral [9, 13, 36, 64] | 4 (5.9) |
| Lateral [23, 46, 48]            | 3 (4.4) |
| Anterior [14, 35, 42]           | 2 (2.9) |
| Posteroapical [19, 67]          | 2 (2.9) |
| Posteroapical [30, 36]          | 2 (2.9) |
| Anterior [47]                   | 1 (1.5) |
| Anteroapical [22]               | 1 (1.5) |
| Basal inferolateral [39]        | 1 (1.5) |
| Inferior [61]                   | 1 (1.5) |
| Inferior and inferolateral [55] | 1 (1.5) |
| Lateral apical [29]             | 1 (1.5) |
| Posteroinferior [45]            | 1 (1.5) |
| Posteromedial [5]               | 1 (1.5) |

### Table IV. Eighteen associated disorders

| Associated disorder                        | N (%) |
|--------------------------------------------|-------|
| Behcet disease [52, 55]                    | 3 (16.7) |
| True aneurysm [17, 38, 55]                 | 3 (16.7) |
| Extrinsic compression of coronary artery ± pulmonary veins [56, 62] | 2 (11.1) |
| Infective endocarditis (of valve-in-valve S3 [58] and of ventricular septal defect patch [69]) | 2 (11.1) |
| Superior vena cava syndrome [55]          | 1 (5.6) |
| Inferior vena cava syndrome [14]          | 1 (5.6) |
| Cerebral infarct [53]                     | 1 (5.6) |
| Acute myocardial infarction [53]          | 1 (5.6) |
| Coronary artery disease [61]              | 1 (5.6) |
| Infrarenal abdominal aortic aneurysm [67] | 1 (5.6) |
| Loeys-Dietz syndrome [38]                 | 1 (5.6) |
| Small bowel obstruction [16]              | 1 (5.6) |

### Table VI. Indications for percutaneous treatment of LVPAAs in 42 patients

| Indications                                                                 | N (%) |
|----------------------------------------------------------------------------|-------|
| Previous operations/sternotomies [13, 21, 26, 28, 30, 31, 36, 45, 49, 50, 52, 65] | 13 (25.0) |
| High surgical risk [7, 16, 19, 24, 39, 53, 55, 56, 60, 67, 69]                | 11 (21.2) |
| LVPA growing [5, 11, 20, 24, 40, 63, 66, 68, 71]                             | 9 (17.3) |
| Patient’s comorbidities [9, 23, 47, 65]                                      | 4 (7.7) |
| Impending LVPA rupture [15, 50, 65]                                          | 3 (5.8) |
| High risk of operative mortality [10, 30]                                    | 2 (3.8) |
| Despite optimal medical therapy, symptoms of heart failure remained [17]    | 1 (1.9) |
| PA was not enlarged but patient felt chest pain during a 6-month follow-up [25] | 1 (1.9) |
| Expected technical difficulties with surgical repair [20]                   | 1 (1.9) |
| Patient’s advanced age [36]                                                  | 1 (1.9) |
| Preventing further cardioembolic events [29]                                | 1 (1.9) |
| Reducing the risk of aneurysm rupture [29]                                  | 1 (1.9) |
| Previous myocardial infarction with reduced left ventricle function [46]    | 1 (1.9) |
| Pseudoeurysm’s geometry [47]                                                 | 1 (1.9) |
| Recent surgical treatment [48]                                               | 1 (1.9) |
| Thoracotomy for hybrid apical access posing a risk of exsanguination [62]    | 1 (1.9) |

LVPA – left ventricular pseudoaneurysm.
the small devices was much less than that of the moderate devices, but without reaching a significant difference (Figure 1).

Guidance of the percutaneous manoeuvre was described for 44 patients; transoesophageal echocardiography was most commonly used (Table IX).

There were 2 LVPA s in 3 (3.2%) patients [20, 24, 30]. Therefore, there were in total 96 LVPA s in 93 patients. There were 121 wire accesses, with 10 (10.8%, 10/93) patients having 14 (11.6%, 14/121) failed accesses.

The causes of failures were described for 9 chances of 7 patients: tortuosity of the aorta (n = 2) [31, 65], difficulty with crossing the LVPA neck (n = 3) [30, 51], unable to maintain position (n = 1) [51], adequate length, angle, and loop of delivery catheter for stable device deployment (n = 1) [14], delivery sheaths too small (n = 1) [10], and poor visualisation of the exact localisation of the channel to the LVPA at angiography (n = 1) [20].

In 10 patients, 2 or 3 devices were used for one LVPA. In 5 of them, the closures for a single LVPA required more devices [9, 24, 66, 67, 69]. In 5 patients, the requirement of more devices was due to closure failures [11, 30, 37, 44, 63]. In addition, one of the two LVPA s in a patient was not treated and no device was used [30].

There were in total 106 closures for 95 LVPA s (one LVPA was not managed due to small size). Eight patients had closure failures, with one failure in 5 patients [24, 30, 64, 66, 70] and 2 failures in 3 patients [22, 44, 67] (there was an additional open patch repair failure in 1 patient [44]). In addition, surgical open external suture of an LVPA failed in 1 patient [14]. The causes of closure failures were described for 6 patients: device too small or too big (n = 2) [11, 30], problematic design of the device (n = 2) [64, 66], the trabecula over the neck hindering a complete seal (n = 10) [67], and partial/complete extrusion of device as well as pulling the errant coil during the manoeuvres [63]. The time interval between procedures in the same patients (staged, redo after failure, and procedures for closures of true and false aneurysms) was 69.0 ± 74.0 (range: 10–180; median: 30) days (n = 5) [14, 20, 38, 63].

Effects of percutaneous closure for LVPA cavity were reported as follows: complete exclusion of the LVPA (n = 8) [7, 12, 15, 28, 33, 44, 45, 50], complete thrombosis of LVPA with no residual flow (n = 7) [11, 26, 34, 41, 46, 55, 61], contraction of the pseudoaneurysm (n = 1).
partial thrombosis of LVPA (n = 1) [7], no blood communication to LVPA by thrombin injection [51, 37], large LVPA sacs filled with embolisation coils (n = 3) [37], LVPV reduced in diameter [6], and LVPA persistent but not increasing in size and partially thrombosed [39].

Minimal residual flow immediate after closure could be observed in 13 patients [5, 20, 29, 39, 43, 48, 49, 54, 57, 60, 65, 68, 71], and on day 2 in 2 patients [66, 67]. No flow was observed 41 patients 16.7 ±37.8 (range: 0–180; median: 0) days after closure [5, 7, 9, 13, 14, 16–22, 24–29, 31, 33, 38, 41–44, 46–49, 51, 55–58, 60, 61, 65, 66, 68, 70].

Patients’ hospital stay after percutaneous LVPA closure was 5.3 ±6.0 (range: 1–28; median: 3) days (n = 26) [5, 7, 8, 11, 12, 16–18, 21, 25, 27, 28, 31, 34, 36, 38, 40, 41, 45, 46, 48, 49, 51, 56, 58, 60]. Patients were on a follow-up of 12.4 ±19.4 (range: 1–84; median: 6) months (n = 52) [5, 7, 8, 9–15, 17, 18, 20–22, 24–29, 31, 34–39, 42, 43, 46–49, 52, 54–62, 65, 66, 68, 69, 71]. Outcomes were known for 71 patients: 41 (57.7%) recovered [8, 11, 12, 14–18, 20, 21, 24, 25, 28, 34–38, 41, 46–49, 51, 53, 54, 57–60, 62, 63, 65, 66, 68–70], 17 (23.9%) improved [22, 23, 37, 39], and 7 (9.9%) were complicated (including coils lost in the false lumen without clinical sequelae and residual leak [36], access site left femoral artery bleeding [30], aphasia, reversible ischaemic neurologic deficit [48], jaundice, haemolytic anaemia, hyperbilirubinaemia, and acute renal failure [67], mechanical compression of circumflex by occluder [36], and post-procedural pericardial effusion [27]). All complications resolved after proper managements. Six (8.5%) patients died: 2 early deaths [29, 64], 2 late deaths [48, 71], and 2 deaths with time not given [37]. The causes of death were recurrent pulmonary emboli [29], bronchial fistula [37], progressive congestive heart failure [37], unrelated causes [48], multiorgan failure [71], and reason not given [64].

Table IX. Guidance for percutaneous manoeuvres

| Guidance | N (%) |
|----------|-------|
| TEE [5, 7, 9, 10, 15, 17, 25, 28, 33, 41, 46, 60] | 12 (27.3) |
| Fluoroscopy and TEE [13, 16, 21, 24, 29, 43, 51, 54, 55] | 10 (22.7) |
| Fluoroscopy [18, 38, 48, 50, 63, 67, 68, 70] | 8 (18.2) |
| Echocardiographic and fluoroscopy [11, 19, 65] | 3 (6.8) |
| Fluoroscopic and TTE [41, 58, 45] | 3 (6.8) |
| TTE [30, 35, 39] | 3 (6.8) |
| Fluoroscopy and intracardiac echocardiography [42, 56] | 2 (4.5) |
| Computed tomography–fluoroscopy [44] | 1 (2.3) |
| Fluoroscopy and combined TTE/TEE [23] | 1 (2.3) |
| Selective hand angiography [14] | 1 (2.3) |

TEE = transoesophageal echocardiography, TTE = transthoracic echocardiography.

Figure 1. Comparisons of parameters of device choices: A – size, B – oversize; C – oversize ratio between the small-, moderate-, and large-sized devices.
Discussion

LVPA may be iatrogenic (previous cardiothoracic surgery, most commonly mitral valve replacement), traumatic (chest trauma), infective (inflammatory/autoimmune disorders), or because of myocardial infarction [72]. Postoperative LVPA occurs in 0.02–2% of mitral valve operations [73]. Other causes of LVPA include intraoperative venting of the cardiac apex, penetrating trauma, and infection [74]. The pathogenesis of LVPA in Behçet disease is unclear, but there are two hypotheses: a subclinical coronary thrombotic event leading to necrosis of the inferolateral myocardium with subsequent development of LVPA; and focal myocardic processes initially leading to the true aneurysm formation with subsequent sac expansion [55].

Patients with an LVPA can be asymptomatic and can be diagnosed incidentally [75]. Severe patients may present with congestive heart failure, arrhythmias, thrombosis, or cardiac rupture [76].

Echocardiography is the most common modality for the diagnosis of LVPA. Transoesophageal three-dimensional echocardiography improves the diagnostic accuracy. Postinfarction LVPA are diagnosed during cardiac catheterisation by left ventriculography. Computed tomography and magnetic resonance imaging are helpful in differentiation between false and true aneurysms [77].

Chronic, asymptomatic LVPA < 30 mm in diameter could be treated conservatively [78]. Symptomatic patients and those with larger LVPA of impending rupture warrant closure [78]. If an LVPA develops within 2–3 months after myocardial infarction, an urgent operation is warranted owing to the high risk of rupture [79]. Surgical repair is the mainstay of treatment of choice. Surgical techniques include LVPA resection neck closure by primary suture or patch repair. Transcatheter closure of the pseudoaneurysm is done especially in patients deemed to be at high surgical risk [77]. The first percutane- ous LVPA closure was described by Clift et al. in 2004 with the use of a 12 mm Amplatzer septal occluder [19]. Percutaneous closures were achieved with septal occlusion devices, coils, and vascular plugs depending on the anatomy. Multimodal imaging is critical for determining the precise location and relationship of the pseudoaneurysm to the surrounding structures, and it improves the success rate of the percutaneous manoeuvre [77].

As for the percutaneous approaches, the transapical approach provides a shorter course than the transseptal approach for accessing left ventricular outflow tract. Thus, transapical closure is an efficient and safe option for left ventricular outflow tract pseudoaneurysm [33]. The present study revealed that the antegrade transapical approach was the most common irrespective locations of LVPA.

Treatment options include Amplatzer devices, vascular plugs (for moderate- to large-sized pseudoaneurysms with narrow necks), and coil embolisation (for small- to moderate-sized LVPA with narrow necks, and for cases that raise concern about the compressive effects of occluder devices) [54]. Al-Hijji et al. [11] tested different sizes of Amplatz Vascular Plug II devices: the 8-mm device was too small to offer complete seal of the neck, the 12-mm device offered complete seal but it was too large and protruded into the prosthetic aortic valve, whereas the 10-mm device offered a complete seal and no risk of aortic prosthesis obstruction. Moriarty et al. [47] recommended the muscular VSD device oversizing by at least 5 or 6 mm, considering transient neck enlargement and mobility during device placement. The target neck size should be 10–12 mm because the maximal device waist is 18 mm [47]. However, Vascular plugs are usually not recommended for use because they do not have sufficient stability as septal occluders for LVPA closure, and most plugs do not have a sufficient waist [72]. In addition, transapical deployment of an Amplatz septal occluder with the left atrial disk hung in the left ventricle apex and the right atrial disk filling the neck remaining incompletely expanded [48]. Transapical access had been established and the pseudoaneurysm contributed to regurgitant blood flow, and thus an Edwards Sapien XT transcatheter heart valve in valve-in-valve fashion was implanted for closure of the neck instead of an Amplatzer plug [54]. Our patient was eligible for percutaneous closure, but placing an Amplatzer plug was not technically feasible. Placing an Edwards Sapien XT transcatheter valve enabled thrombosis of the LVPA [54].

Untreated LVPA has 30–45% risk of rupture [4], and with medical therapy the mortality is 48% [51]. Even with surgical intervention there is a high mortality rate of up to 35% [80]. The overall hospital mortality of surgical repair of LVPA was 27.3% and mean survival was 61.9 ±4.1 months in the 16 hospital survivors [79]. Moreno et al. [81] reported 1-year and 4-year survival rates of 88.9% and 74.1% in conservatively treated patients, respectively. In this study, percutaneous closure was associated with a mortality rate of 8.5%.

Incomplete patient data was the main shortcoming of the present study. The information of size and oversize of the failed devices merely offered a rough comparison between successful and failed cases. Thus, the cut-off valves of oversize 3.3 mm and of oversize ratio 1.6 preliminarily obtained were references of suitable device choices. Post-procedural ejection fractions were not reported, and thus the assessment of the effect of percutaneous treatment on the left ventricular function was impossible. Total procedural time and fluoroscopy time were described in very few reports. Thus, the complexity of deployments of different devices could not be further evaluated.

Conclusions

Percutaneous closure of LVPA was especially indicated for patients carrying a high surgical risk. Iatrogenic trau-
mas, such as left ventricular venting, should be avoided in order to eliminate this complication. For device choice, the cut-off valves of oversize 3.3 mm and oversize ratio 1.6 should be kept in mind for reference.

Conflict of interest

The author declares no conflict of interest.

References

1. Prakash S, Garg N, Xie GY, Dellsperger KC. Giant left ventricular pseudoneuyma. J Cardiovasc Comput Tomogr 2010; 4: 284-5.
2. Bisoyi S, Dash A, Nayak D, et al. Left ventricular pseudaneu- rysm versus aneurysm a diagnosis dilemma. Ann Cardiac Ana- esth 2016; 19: 169-72.
3. Mohammadi S, Bonnet N, Leprince P, et al. Reoperation for false aneurysm of the ascending aorta after its prosthetic replace- ment: surgical strategy. Ann Thorac Surg 2005; 79: 147-52.
4. Černá D, Veselka J, Vaněk I, et al. Left ventricular pseudoneu- rysm as a complication of myocardial infarction. Cor Vasa 2007; 49: 369-72.
5. Arnaz A, Akansel S, Yalcinbas Y, et al. Transcatheter closure of left ventricular pseudoneuyma after mitral valve replacement. Ann Thorac Surg 2020; 110: e123-5.
6. Sakai K, Nakamura K, Ishizuka N, et al. Echocardiographic find- ings and clinical features of left ventricular pseudoneuyma after mitral valve replacement. Am Heart J 1992; 124: 975-82.
7. Ancona MB, Visco E, Bellini B, et al. Percutaneous exclusion of left ventricular pseudoeurusyma. J Cardiovasc Med 2021; 22: 864-5.
8. Acar Z, Korkmaz L, Parlar AI, et al. PP-156 Percutaneous closure of huge left ventricular pseudoneuyma by atrial septal occlud- er device. Int J Cardiol 2013; 163S1: S81-111.
9. Acharya D, Nagaraj H, Misra VK. Transcatheter closure of left ven- tricular pseudoneuyma. J Invasive Cardiol 2012; 24: E111-4.
10. Afonso Nogueira M, Ferresga A, de Sousa L, et al. Percutaneous closure of a giant left ventricular wall pseudoeurusyma: antero- grade approach with a double snare technique. Rev Port Cardiol 2016; 35: 441.e1-4.
11. Al-Hijji MA, Guerrero M, Rihal CS, Eleid MF. Transapical percutaneous closure of rapidly expanding post-surgical left ventric- ular outflow tract pseudoeurusyma. Catheter Cardiovasc Interv 2019; 94: 859-62.
12. Alkhouli M, Hijazi M, Busu T, et al. Percutaneous closure of left ventricular pseudoneuyma caused by a central venting cannu- la. J Card Surg 2017; 32: 644-5.
13. Alshehri HZ, Momenah TS, AlBaradai A, et al. Successful per- cutaneous closure of post myocardial infarction left ventricular ruptured pseudoeurusyma after failed surgical repair. J Cardiol Cases 2014; 9: 154-7.
14. Baspinar O, Mete A, Davutoglu V. Transcatheter closure of a large left ventricular pseudoeurusyma using an Amplatzer Vascular Plug 4 and stenting of the inferior caval vein in a child. Cardiol Young 2012; 22: 106-9.
15. Boi A, Cocco D, Rossi A, et al. Left ventricular pseudo-aneurysm after transapical valve-in-valve transcatheter aortic valve replacement treated with percutaneous closure. J Cardiovasc Med 2019; 20: 400-2.
16. Bortnick AE, Gordon E, Gutsche J, et al. Percutaneous closure of a left ventricular pseudoeurusyma after Sapien XT transapical transcatheter aortic valve replacement. JACC Cardiovasc Interv 2012; 5: e37-8.
17. Cavalcanti LR, Sá MPBO, Escorel Neto AC, et al. Percutaneous closure of left ventricular pseudoeurusyma in a patient with concomitant true left ventricular aneurysm. J Card Surg 2021; 36: 2113-6.
18. Chatfield AG, Chan Wah Hak YS, White JM, et al. Large left ven- tricular outflow tract pseudoeurusyma closed percutaneously. from a novel apical approach. J Am Coll Cardiol Case Rep 2019; 1: 7-13.
19. Clift P, Thorne S, de Giovanni J. Percutaneous device closure of a pseudoeurusyma of the left ventricular wall. Heart 2004; 90: e62.
20. Cwikel W, Keussen I, Gustafsson R, Mokhtari A. Endovascular treatment of two pseudoeurysm originating from the left ventricle. Cardiovasc Intervent Radiol 2013; 36: 1677-80.
21. Dershowitz L, Wolbinski M, Bapat V, et al. Left ventricular injury – beware the wire. JTCVS Techn 2020; 3: 126-9.
22. Dudy J, Jelinín V, Einhorn BN, et al. Percutaneous closure of left ventricular pseudoeurusyma. Circ Cardiovasc Inter 2011; 4: 322-6.
23. Dudon BK, Yeend RA, Worthley SG. Percutaneous closure of a large peri-prosthetic left ventricular pseudoeurusyma in a high-risk surgical candidate. BMJ Case Rep 2009; 2009: bcr2007128405.
24. Elshershari H, Gossett JG, Hijazi ZM. Percutaneous closure of left ventricular pseudoeurusyma after Ross procedure. Ann Thorac Surg 2009; 85: 634-6.
25. Feldman T, Pearson P, Smart SS. Percutaneous closure of post TAVR LV apical pseudoeurusyma. Catheter Cardiovasc Interv 2016; 88: 479-85.
26. Flores-Umanzor E, Cepas-Guilien PL, Freixa X. Percutaneous closure of a left ventricular outflow tract pseudoeurusyma. Rev Esp Cardiol 2019; 72: 164.
27. Gokalp S, Ugan Atik S, Saltik IL. Percutaneous closure of a left ventricular pseudoeurusyma after transcatheter ventricular septal defect closure. Cardiol Young 2020; 30: 743-5.
28. Graham EM, Bandisode VM, Atz AM, et al. Percutaneous occlusion of a pseudoeurusyma evolving after homograft aortic valve and root replacement with the Amplatzer muscular ventricular septal defect occluder. J Thorac Cardiovasc Surg 2006; 131: 914-6.
29. Harrison W, Ryugko PN, Greaves S, et al. Percutaneous closure of left ventricular free wall rupture with associated false aneu- rysm to prevent cardioembolic stroke. Heart Lung Circ 2008; 17: 250-3.
30. Hnat T, Adlova R, Fiedler J, Veselka J. Percutaneous left ventric- ular pseudoeurusyma closure. Arch Med Sci 2020; 16: 1247-9.
31. Kar B, Ghokal G, Gregoric ID, et al. Percutaneous closure of a left ventricular pseudoeurusyma in a high-risk surgical candidate. Tex Heart Inst J 2012; 39: 680-2.
32. Karimi A, Beaver TM, Fudge JC. Percutaneous transfemoral clo- sure of a pseudoeurusyma at the left ventricular apical access site for transcatheter aortic valve implantation. J Invasive Cardi- ol 2015; 27: 27-9.
33. Katada Y, Ito J, Shibayama K, et al. Transapical transcatheter clo- sure of the pseudoeurusyma in the left ventricular outflow tract after aortic valve replacement. JACC Cardiovasc Interv 2016; 9: e181-3.
34. Kazawa M, Kanazawa H, Shimahara Y, et al. Anesthetic manage- ment of a percutaneous closure of left ventricular pseudoeurusyma. Ann Thorac Surg 2020; 110: e123-5.
35. Khanna S, Dahiya R, Bali A, et al. Percutaneous closure of a left ventricular pseudoaneurysm after homograft aortic valve and root replacement with an Amplatzer muscular ventricular septal defect occluder. Tex Heart Inst J 2012; 39: 680-2.
51. Okuyama K, Chakravarty T, Makkar RR. Percutaneous transapical closure of left ventricular pseudoaneurysm in the left ventricle in a young child. Pediatr Int 2016; 58: 648-51.

50. Negri F, Cernetti C, Favero L, et al. Transcatheter closure of left ventricular pseudoaneurysm using 3D printed heart model. Cardiovasc Anesth 2020; 18: 109-10.

49. Neeraj A, Kumar V, Bisht D, Kumar V. Percutaneous closure of left ventricular pseudoaneurysm using 3D printed heart model for procedure planning: a novel approach. Catheter Cardiovasc Interv 2017; 90: E177-8.

48. Mohamed E, Tellila T, Osaki S, Jacobson K. Percutaneous closure of left ventricle pseudoaneurysm using 3D printed heart model for procedure planning: a novel approach. Catheter Cardiovasc Interv 2017; 90: E227-8.

47. Mishra A, Mondal S, Kapoor L, Bharati S. Percutaneous device closure of ‘Left Ventricular Pseudoneurysm’ following coronary artery bypass graft surgery. IHI Cardiovasc Case Rep 2018; 2: 108-10.

46. Mohamed E, Tellila T, Osaki S, Jacobson K. Percutaneous closure of left ventricle pseudoaneurysm using 3D printed heart model for procedure planning: a novel approach. Catheter Cardiovasc Interv 2019; 94: 874-6.

45. Moriarty J, Harris TJ, Vorobiof G, et al. Direct percutaneous repair of left ventricular pseudoaneurysm via transhilaric deployment of a ventricular septal defect closure device. Tex Heart Inst J 2015; 42: 362-6.

44. Narayan RL, Vaishnava P, Goldman ME, et al. Percutaneous closure of left ventricular pseudoaneurysm. Ann Thorac Surg 2012; 94: e123-5.

43. Neeraj A, Kumar V, Bisht D, Kumar V. Percutaneous closure of a left ventricular pseudoaneurysm: case report with review of cases. IHI Cardiovasc Case Rep 2020; 4: 104-7.

42. Negri F, Cernetti C, Favero L, et al. Transcatheter closure of left ventricular pseudoaneurysm using an Amplatzer Vascular Plug. Cardiovasc Revasc Med 2019; 20: 1013-5.

41. Okuyama K, Chakravarty T, Makkar RR. Percutaneous transapical pseudoaneurysm closure following transcatheter aortic valve replacement. Catheter Cardiovasc Interv 2018; 91: 159-64.

40. Ota H, Morita Y, Saiki Y, Takase K. Coll embolization of left ventricular outflow tract pseudoaneurysms: techniques and 5-year results. Interact Cardiovasc Thorac Surg 2017; 24: 631-3.

39. Pavani M, Scrocco I, Meliga E, De Benedictis M. Percutaneous closure of a left ventricular pseudoaneurysm causing recurrent embolization, with cerebral protection and extracorporeal membrane oxygenation support. JACC Cardiovasc Interv 2018; 11: e7-10.

38. Pehalver J, Shatilla W, Macheda F, Silva G. Transapical implantation of Edwards Sapien XT valve to close a left ventricular outflow tract pseudoaneurysm. Tex Heart Inst J 2019; 46: 157-9.

37. Quimby DL Jr, Ford J, Tanner GJ, et al. Three-dimensional cardiac print assisted percutaneous closure of left ventricular pseudoaneurysm in patient with Behcet's disease. Catheter Cardiovasc Interv 2022; 99: 512-7.

36. Romaguera R, Slack MC, Waksman R, et al. IMAGE CARDIO MED. Percutaneous closure of a left ventricular outflow tract pseudoaneurysm causing extrinsic left coronary artery compression by transseptal approach. Circulation 2010; 121: e20-2.

35. Ruiz-Morales J, Carraquillo S, Ganji M, Soffer D. Inside out: left ventricular aneurysm following transcatheter aortic valve replacement. Chest 2020; 158: A147.

34. Salaun E, Aldebert P, Jaussaud N, et al. Early endocarditis and delayed left ventricular pseudoaneurysm complicating a transapical transcatheter mitral valve-in-valve implantation: percutaneous closure under local anesthesia and echocardiographic guidance. Circ Cardiovasc Interv 2016; 9: e003886.

33. Sawlani N, Berry N, Sobieszczzyk P, et al. Percutaneous closure of a delayed left ventricular pseudoaneurysm following transseptal transcatheter mitral valve replacement. JACC Cardiovasc Interv 2017; 10: 1464-5.

32. Singh A, Kliger C, Ruiz CE. Novel approach for the percutaneous treatment of left ventricular pseudoaneurysms. Catheter Cardiovasc Interv 2015; 85: 1092-6.

31. Smolka G, Peszek-Przybyla E, Sosnowski M, Ochala A. Complete percutaneous obliteration of a post-infarction left ventricular pseudoaneurysm. JACC Cardiovasc Interv 2012; 5: 886-7.

30. Sonawane B, Sivakumar K. A giant left ventricular pseudoaneurysm diagnosed after double-valve replacement. Asian Cardiovasc Thorac Ann 2021; 29: 953-6.

29. Srivastava NT, Hoyer MH. Thromboexclusion of an atypical left ventricular pseudoaneurysm. Catheter Cardiovasc Interv 2015; 85: 274-7.

28. Susam I, Yaylali YT, Tekin I. PP-149 Percutaneous device closure of a left ventricular outflow tract pseudoaneurysm causing recurrent embolization, with cerebral protection and extracorporeal membrane oxygenation support. JACC Cardiovasc Interv 2018; 11: e7-10.

27. Taqatqa AS, Caputo M, Kenny DP, Diab KA. Surgical repair of left ventricular pseudoaneurysm following perventricular device closure of muscular ventricular septal defect. J Card Surg 2016; 31: 697-9.

26. Tsai MT, Tseng CC, Kan CD. Hemolytic hyperbilirubinemia after percutaneous device closure of left ventricular pseudoaneurysm: a disregarded complication. Ann Thorac Surg 2013; 95: 707-9.
68. Vignati G, Bruschi G, Mauri L, et al. Percutaneous device closure of iatrogenic left ventricular wall pseudoaneurysm. Ann Thorac Surg 2009; 88: e31-3.
69. White JM, Lowe BS, Ruygrok PN. A man with 3 lives: long-term follow-up following percutaneous closure of left ventricular pseudoaneurysm neck. JACC Cardiovasc Interv 2015; 8: e77-9.
70. Yudi MB, Love B, Nadir A, et al. Percutaneous closure of left ventricular pseudoaneurysm with septal occluder device and coils: a multimodality imaging approach. JACC Cardiovasc Interv 2017; 10: e159-61.
71. Zhong W, Liu Z, Fan W, et al. Transcatheter closure for the treatment of pseudoventricular aneurysm after acute myocardial infarction: a case report. Ann Transl Med 2020; 8: 1528.
72. Bertog SC, Franke J, Vaskelyte L, et al. Percutaneous closure of aortic, coronary, and ventricular pseudoaneurysms and coronary fistulae. In: Lasala JM, Rogers JH (eds). Interventional Procedures for Adult Structural Heart Disease E-Book: Expert Consult – Online. Elsevier Health Sciences, Philadelphia, PA, USA 2013; 232-41.
73. Yavuz S. eComment. Ventricular pseudoaneurysms in postsurgical cardiac patients. Interact Cardiovasc Thorac Surg 2014; 19: 161-2.
74. Stoddard MF, Dawkins PR, Longaker RA, et al. Transesophageal echocardiography in the detection of left ventricular pseudoaneurysm. Am Heart J 1993; 125: 534-9.
75. Shin MS, Koo BK, Kim HJ, et al. Incidentally found asymptomatic left ventricular apical pseudoaneurysm. J Korean Soc Echocardiogr 2000; 8: 131-3.
76. Adekoya O, Mmagu O, Dittoe N, Ruff T. Medical management of a case of inferior-posterior wall ST elevation myocardial infarction complicated by pseudoaneurysm. Cureus 2020; 12: e7675.
77. Chandrashekar R, Konda MK, Gupta V, Kalavakunta JK. Left ventricular pseudoaneurysm dissecting into the anterior chest wall: a rare case of sudden onset excruciating chest pain. Eur J Case Rep Intern Med 2017; 4: 000518.
78. Prêtre R, Linka A, Jenni R, Turina MI. Surgical treatment of acquired left ventricular pseudoaneurysms. Ann Thorac Surg 2000; 70: 553-7.
79. Fedakar A, Bugra O, Onk A, et al. Repair of left ventricular pseudoaneurysms. Asian Cardiovasc Thorac Ann 2010; 18: 39-43.
80. Eren E, Bozbuga N, Toker ME, et al. Surgical treatment of post-infarction left ventricular pseudoaneurysm: a two-decade experience. Tex Heart Inst J 2007; 34: 47-51.
81. Moreno R, Gordillo E, Zamorano J, et al. Long term outcome of patients with postinfarction left ventricular pseudoaneurysm. Heart 2003; 89: 1144-6.