Left Ventricular Pseudoaneurysm

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ABSTRACT

Objective: To comprehensively present the common features of left ventricular pseudoaneurysms (LVPAs).

Methods: The data source of this article based on a careful collection of the pertinent literature of LVPAs of recent decades.

Results: Most LVPAs develop secondary to acute myocardial infarction and cardiac surgical procedures. Angiography remains the golden standard diagnostic modality. Other diagnostic techniques with excellent visualization include echocardiography, cardiac magnetic resonance imaging and computerized tomography. The additional characteristic findings of LVPAs might be turbulent bidirectional flow and associated mitral regurgitation.

Conclusion: LVPAs often warrant a surgical repair considering the propensity of rupture and subsequent temponade and death. Transluminal interventional therapy is a treatment choice for selected patients.

Keywords: Cardiac surgical procedures, differential diagnosis, left ventricular pseudoaneurysm; myocardial rupture

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INTRODUCTION

Left ventricular pseudoaneurysm (LVPA) is formed secondary to an incomplete rupture of the left ventricular wall contained by the pericardium (1). There are two special types of LVPAs: mixed (conjunct true and false) and false-true superimposed (2). Moreover, daughter aneurysm arising from the LVPA (2), multiple (3), multi-loculated (4, 5), apical tunnel-shaped (6), recurrent LVPAs (7) have been described. LVPAs can be divided into: acute (onset <2 weeks) and chronic (onset >2 weeks) (8). LVPAs can be arising from the free wall, LVOT and mitral-aortic intervalvular fibrosa (MAIVF). The incidence of LVPAs is unknown. Myocardial infarction and previous cardiac surgery accounted for 55% and 33%, respectively, of all causes of LVPAs (9). Frances *et al*. (10) comprehensively described the common features of LVPAs, and recently, some special aspects of LVPAs are instantly presented, indicating the complexity and difficulty in diagnosis and treatment. The aim of this article is to give an overview of LVPAs.

Concept

Morphologically, the LVPA communicates with the left ventricular cavity via a narrow neck (11). Davutoglu (8) 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 resultant severe mitral regurgitation, which is seldom in false aneurysms.

Etiology

LVPAs can occur as a complication of myocardial infarction (MI), cardiac surgery, infective

endocarditis, chest trauma, tuberculosis, rheumatoid arthritis, Kawasaki's disease and Behcet's disease. The most common etiology of LVPAs is myocardial infarction (10). One third of LVPAs resulted from a surgical procedure, most often mitral valve replacement (10). Other causative factors included chest trauma, infections, immune disorders, and multiple factors (Table 1). Congenital ventricular aneurysms are characterized by fibrotic tissue with akinesis or paradoxical systolic motion of the aneurysm sac (51). Subannular pseudoaneurysms were described in African populations, presumably on a congenital or developmental basis. The formation time of LVPA was from 1 day (13) to 25 years (52), and it was shorter in MI patients and longer in cardiac surgical patients.

Clinical manifestations

In some patients, the LVPA was found incidentally by echocardiographic examinations (22). The most common symptoms are chest pain and dyspnea. Congestive heart failure and new murmurs occur in half of the patients (2). LVPA is prone to rupture (53). Sudden death accounted for 3% of the onset symptoms of LVPA (10). Mitral regurgitation may complicate posterior pseudoaneurysm, usually due to distortion or partial rupture of the posteromedial papillary muscle (54).

Diagnosis

The LVPA should be considered in the patients' refractory to the medical treatment or those with unexplained heart failure. A physical examination may reveal to-and-fro heart murmurs (55). Angiography remains the golden standard diagnostic modality with alternative diagnostic techniques with excellent visualization being echocardiography, cardiac magnetic resonance imaging and computerized tomography (55). By echocardiography, the entrance of

the LVPA could be visualized (56). Computed tomography may sometimes not distinguish false from true, probably inaccurately locate the site of origin (57). Magnetic resonance imaging distinguishes among pericardium, thrombus and myocardium, capable of showing disruption of the epicardial fat layer of the LVPAs, but intracavity thrombus may obscure the dimensions of the LVPAs thus leading to an inaccurate measure of the size (8).

Clinically, true aneurysms are often associated with ventricular tachycardia and (or) heart block and even sudden cardiac death (58), but this is uncommon in cases with a LVPA. Echocardiography may be helpful in the differentiation between the true and false aneurysms by showing a narrow neck in pseudoaneurysms, equivalent neck to the sack in true aneurysms (59). Histopathological examination of excised ventricular wall helps to confirm the diagnosis of this disease (9).

Treatment

LVPAs are usually stable under 3 cm in dimension, and if they are detected incidentally, they can be managed conservatively (60). The conservative managements can be pharmacological with acetylsalicylic acid, nitrates, angiotensin-converting enzyme inhibitor and β-blockers (61), and anticoagulants *etc*. (62). Patients with a LVPA with an increased size despite regular conservative treatment warrant interventional management (37).

Some authors proposed that due to the high propensity of LVPA rupture, patients with a LVPA are indicated for a cardiac surgery upon diagnosis is made (63). In certain cases, an emergent operation is needed (5, 7, 15, 16). However, it is uncertain whether a chronic LVPA should be undergone an urgent surgical repair (55).

Median sternotomy is the first-line approach for primary cases of posterolateral

LVPA. Cardiopulmonary bypass and cardioplegic arrest can be easily and securely established. In contrast, exposure of a posterolateral LVPA through a median sternotomy is more difficult in comparison with a left thoracotomy due to the deep location and dense adhesion to the pericardium (64). In a few occasions, anterior left thoracotomy (7, 33), right lateral position (65), or anterolateral mini-thoracotomy (6) was applied.

Prevention of LVPA rupture was the primary goal of repair (9). Garrido *et al*. (17) emphasized to choose a surgical technique according to the policy of not interrupting the normal left ventricular geometry. The popular techniques in LVPA repair was circular patch plasty technique ("Dor" procedure) (66).

Mitral regurgitation in posterior LVPAs may be successfully treated by resection of the pseudoaneurysm and does not always require valve replacement (54).

In the past, conservative treatment can be considered in patients who carry a high risk for operation (67). Nowadays, percutaneous interventional therapy offers an alternative therapeutic possibility for such patients. Size of the devices should be larger (9) or at least equal to the neck of the orifice.

Prognosis

The patients with a LVPA have a high morbidity, and a risk of spontaneous rupture and sudden death (68). Despite the high mortality rates for patients without undergoing a surgical operation, prolonged survival in some conservatively patients were also reported (10). The rupture rate was reported to occur in 30-45% of the patients (69). Death (9%), hemorrhage (7%) and arrhythmia (6%) were reported to be the more common complications. The recurrence rate of LVPA after surgical repair was 5% (10). Postoperative mortality rates

ranged between 7-29% (13). There was a report describing a self-cured LVPA (70).

CONCLUSIONS

Despite survived cases with conservative treatment or self-cured cases, the LVPAs often warrant a surgical repair considering the propensity of rupture and subsequent risks of temponade and death. For very aged and high-risk patients, transluminal interventional therapy is a treatment choice with an increased success rate and reduced postinterventional complications.

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Table: The underlying etiologies of left ventricular pseudoaneurysm formation

Etiology	Description
Myocardial infarction	
Acute	Inferior (12), anterior (13), or lateral wall (14), NSTEMI (15); Due to bridging left anterior descending coronary artery (16); Re-infarction (17).
Remote/healed	Healed (18) Old (19) Postinfarction myocardial rupture (20)
Iatrogenic	
Maneuver	Pericardiocentesis (21); Electrophysiologic study (a mapping catheter insertion for left atrial mapping) (22); Epicardial lead implantation, endomyocardial biopsy, attempted ventricular septal defect repair & apical venting (8); Apical venting (23).
Intervention	Endocardial/epicardial ablation for recurrent ventricular tachycardia (24); Transcatheter low energy DC ablation for WPW syndrome (25); Balloon mitral valvuloplasty (26); Perventricular device closure of muscular ventricular septal defect (27); Transfemoral transcatheter AVR (CoreValve®) (28).
Surgery	Aortic commissurotomy and subaortic muscular resection for LVOTO (29); Partial suture dehiscence after aneurysm resection (8); Subsequent operations: repair of myocardial infarction

	followed by resection of SAS (29);
	Ascending aorta replacement (30);
	Ascending aorta replacement + MVR (mechanical) (31);
	Left ventricular pseuduaneurysmectomy (32);
	Left ventricular true aneurysmectomy (7);
	Apicoaortic bypass (33);
	Mitral valve repair (34);
	Mitral annuloplasty + Maze procedure (35);
	MVR (9, 10);
	AVR (36);
	Transapical aortic valve-invalve implantation (6);
	Transapical transcatheter mitral valve-in-valve implantation (37);
	LVAD implant (38).
Chest trauma	Blunt chest trauma (39);
	Stab wound (penetrating left ventricular injury) (40).
Infection	Infective endocarditis (native valves (41) and prosthetic valve (42));
	Infective pericarditis (43);
	Purulent pericarditis (44);
	Perimyocarditis (45);
	Dengue fever (46);
	Tuberculosis (11);
	Pancarditis + brucellosis (47);
	Disseminated bacterial infection (sepsis, septic shock with septic arthritis) (48).
Combined (infection + heart valve replacement)	Mitral valve IE + *Staphylococcus aureus* pancarditis + MVR (49);
	IE + MVR/AVR/MVR + AVR (50).

Immune disorders	Rheumatoid arthritis, Kawasaki disease (11);
	Behçet disease (11).
Congenital (3, 4)	
Unknown (11, 12)	

AVR: aortic valve replacement; IE: infective endocarditis; LVOTO: left ventricular outflow tract obstruction; MVR: mitral valve replacement; NSTEMI: Non-ST elevation myocardial infarction; SAS: subaortic stenosis.