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Case Report

Spontaneous dissecting aneurysm of the left atrium complicated by cerebral embolism: A report of two cases with review of literature

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ABSTRACT

Left atrial dissection is a very uncommon complication of cardiac surgery and usually causes significant hemodynamic compromise. Little is known about spontaneous dissection of the left atrium. Two patients, one middle-aged man and another elderly woman were evaluated following stroke. Routine trans-thoracic echocardiogram showed vertical division of the left atrium with both chambers communicating with each other through an orifice. Detailed trans-oesophageal echocardiographic study revealed dissection of the left atrium producing an additional false chamber (pseudo-aneurysm) placed posterior to the left atrial appendage and above the postero-lateral aspect of mitral annulus. Spontaneous dissection of the left atrium is extremely rare, and there is no report of cerebral embolism associated with it. Review of literature reveals interesting facets of this rare entity.

1. Introduction

Left atrial (LA) dissection is a rare complication of cardiac surgery, probably related to a contained atrio-ventricular separation allowing pressurized blood to separate the layers of the posterior LA.[1] It has been defined as a false, blood-filled cavity, or lumen from the mitral annular area to the LA free wall or interatrial septum, creating a new chamber with or without communications into the true LA. The most common cause is surgical trauma during mitral valve replacement.[2] It has been also reported to occur following coronary bypass surgery, percutaneous coronary angioplasty, blunt chest trauma, acute myocardial infarction, pulmonary vein cannulation, excision of the LA mass, radiofrequency ablation, repair of the left ventricular pseudo-aneurysm and infective endocarditis etc.[3–12] Presentation may be early or delayed. Spontaneous dissection of the LA has been rarely reported in the literature.[13–20] We herein describe two patients, who presented with stroke and were found to have LA dissection with pseudo-aneurysm formation.

2. Case report-1

A 54-year-old man suffering from type 2 diabetes mellitus and systemic hypertension presented with acute ataxia, nausea and giddiness in February 2014. There was no previous history

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Abbreviations: LA, left atrium; AN, aneurysm; LV, left ventricle; RV, right ventricle; RA, right atrium; AO, aortic root; TEE, trans esophageal echocardiography; LVOT, left ventricular outflow tract.

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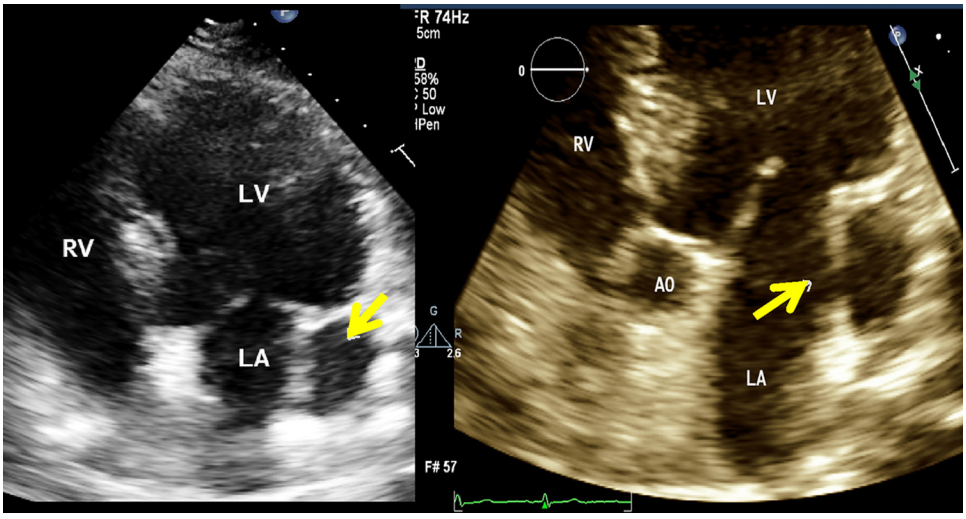

Fig. 1 – Trans-thoracic echocardiographic 4-chamber and 5-chamber views showing an additional chamber within the left atrium just above posterolateral mitral annulus (arrows). Yellow arrow in right image points toward the communicating channel.

of chest trauma, cardiac surgery, external cardiac massage or any prolonged infective state. Physical examination revealed average-built person, regular heart rate of 88 BPM, supine blood pressure 150/80 mmHg, quiet precordium and no evidence of heart failure. Plasma biochemistry was normal with random blood glucose of 158 mg%. A 12-lead electrocardiogram showed sinus rhythm and non-specific ST-T changes. Plain chest skiagram was unremarkable. MRI diffusion imaging showed a fresh infarct in the right cerebellar hemisphere and an old lacunar infarct in the vermis. Detailed trans-thoracic and trans-esophageal echocardiography was performed. Trans-thoracic echocardiogram showed a chamber within the LA just above the posterolateral mitral annulus communicating with the main LA cavity through a 12 mm hole with to-and-fro flow (Fig. 1). Trans-esophageal echocardiographic views confirmed these findings (Figs. 2 and 3, video 1) and showed a flap of variable thickness originating from the left posterior atrio-ventricular junction and spreading up-

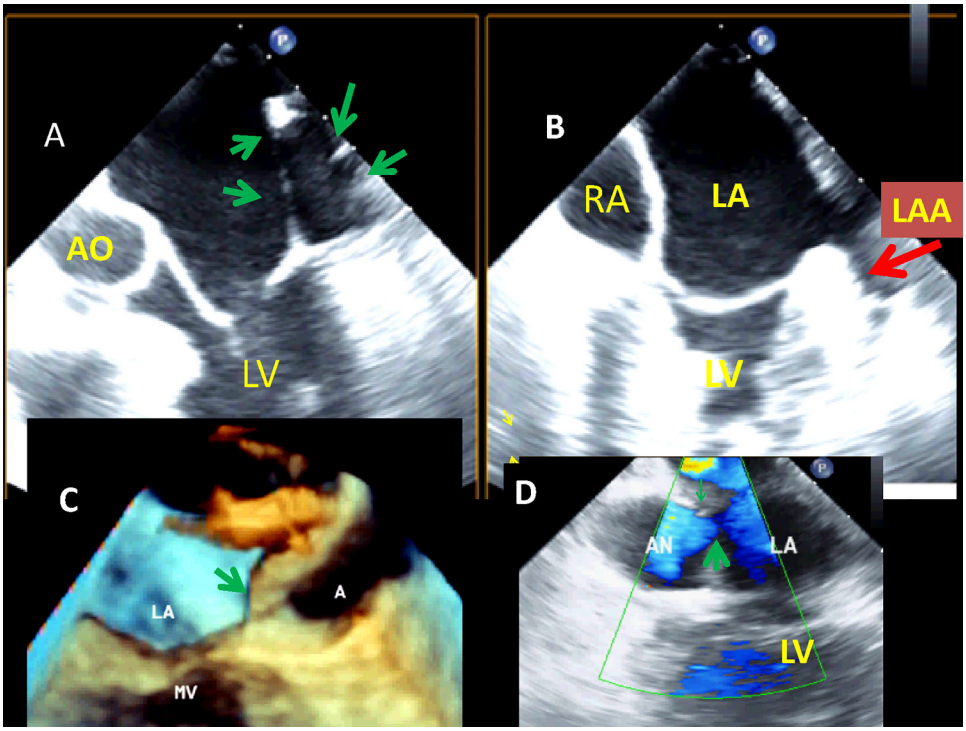

Fig. 2 – Multiple trans-esophageal views showing the pseudo-aneurysm (A, green arrows), separate left atrial appendage (red arrow) and communication between the true left atrial chamber and the false chamber on color flow mapping (D).

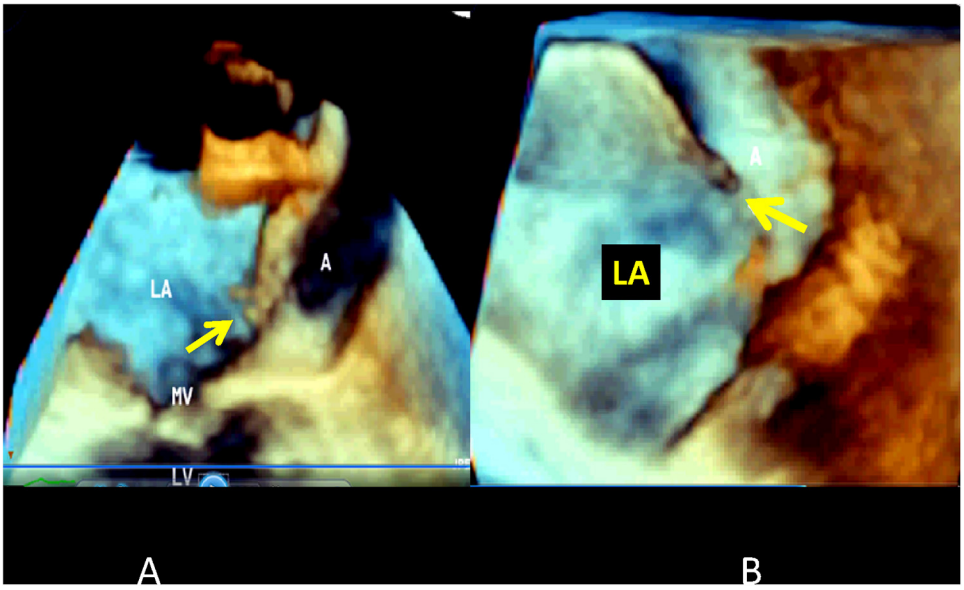

Fig. 3 – Multiplanar 3D TEE views showing the dissection flap in two different views (long axis and short axis). Yellow arrow points at the communicating orifice. A is the false chamber. MV – mitral valve.

wards posteriorly and superiorly upto inter-atrial septum and a definite communicating orifice by color flow mapping. Pulmonary venous drainage was normal, and there was mild mitral regurgitation. No thrombus was seen in any part of the LA.

In view of no significant abnormalities on echocardiography, this stable patient has been managed with non-operative approach with anti-platelets, anticoagulants, control of hypertension, and diabetes mellitus. Follow-up at one year revealed no change in clinical condition and same sized dissecting LA aneurysm without any thrombus and no other significant abnormalities on echocardiography.

3. Case report-2

A 72-year elderly lady was admitted with heart failure in two weeks following right middle cerebral artery stroke. She denied history of chest trauma, interventional procedure, and cardiopulmonary resuscitation. Physical examination showed a frail elderly woman, irregularly irregular heart rate of 88 BPM, blood pressure of 130/90 mmHg, distended jugular veins, pitting pedal edema, non-tender hepatomegaly, basal rales, apical third heart sound and a pan-systolic murmur. A 12-lead electrocardiogram revealed atrial fibrillation, incomplete right bundle branch, and generalized T wave inversions. The plain chest skiagram showed enlarged cardiac silhouette, right-sided pleural effusion, pulmonary venous congestion, and prominent LA shadow. Serum creatinine was 2.7 mg% (eGFR 28), hemoglobin 9.8 g%, plasma albumin 2.8 g%, and neutrophilic leukocytosis (12,800 cmm^{-1}, polymorphs 86%) was observed. Transthoracic echocardiographic examination showed enlarged and distended inferior vena cava, right atrium, and the right ventricle, multiple atrial septal defects with left-to-right flow, severe tricuspid regurgitation and pulmonary regurgitation and an estimated pulmonary pressure of 85/40 mmHg. There was moderate mitral regurgitation and the left ventricular ejection fraction was 0.50.

The LA was split into two vertical chambers by a flap in apical 4-chamber views starting from the lateral edge of the mitral annulus and reaching upto the left upper pulmonary vein (Fig. 4, video 2). Communicating orifice was present just above the mitral annulus and showed two-and-fro flow. In apical 5-chamber view, the false aneurysm appeared like a cystic mass within the LA with clear separation from the LA appendage and the left pulmonary veins.

Trans-esophageal echocardiography revealed a cystic mass adjacent to but separate from the LA appendage; with communication in the true LA just below the orifice of the left upper pulmonary vein and (Figs. 5 and 6). In addition, two atrial septal defects were seen. Irregular echogenic material was seen in the false chamber which could be thrombotic material.

In view of patient's reluctance, further evaluation including magnetic resonance imaging and surgery was not contemplated. The patient has been managed with non-operative approach including decongestive therapy, anti-platelets and anticoagulants.

4. Discussion

Left atrial (LA) dissection is a rare complication and is defined as a gap from the mitral or tricuspid annular area to the interatrial septum or LA atrial wall. One of the mechanisms of this entity is likely to be partial atrio-ventricular separation causing dissection of the layers of the posterior LA. True incidence, etiology, pathophysiology, clinical course, and management are poorly understood. It can be caused by any cardiac interventions, including both surgical and catheter-

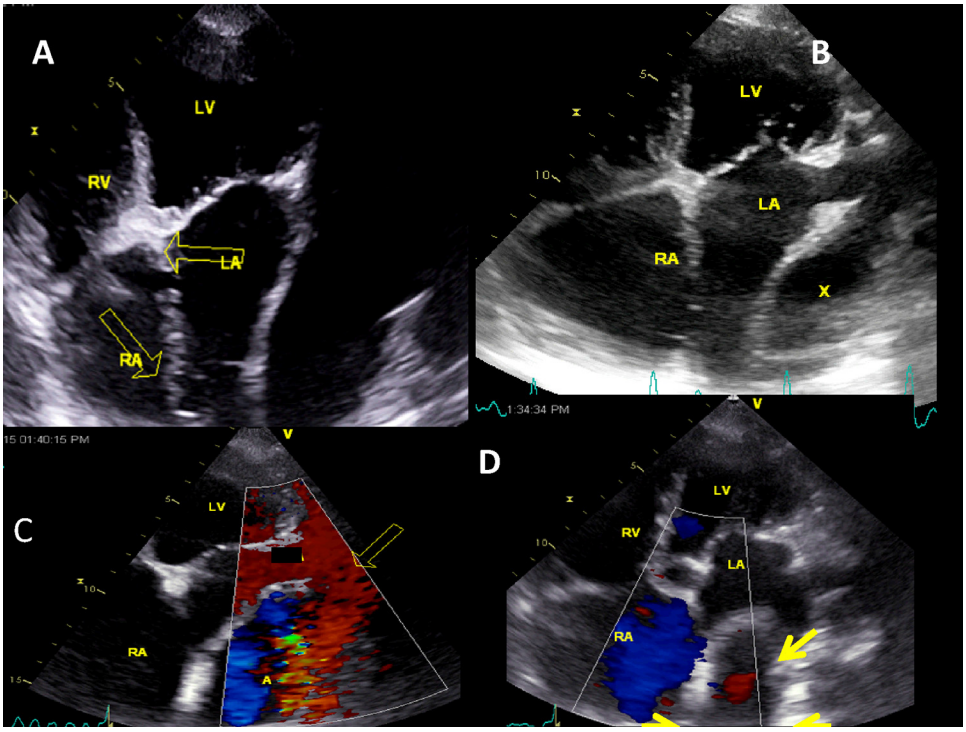

Fig. 4 – (Case #2): Multiple apical trans-thoracic echocardiographic views showing vertically split left atrium in 4-chamber views (X is the false chamber). In 5-chamber view, the false chamber or pseudo-aneurysm appears like a cystic mass (yellow solid arrows). (B, C) Communication between the two chambers situated above the posterior and lateral edge of the mitral annulus.

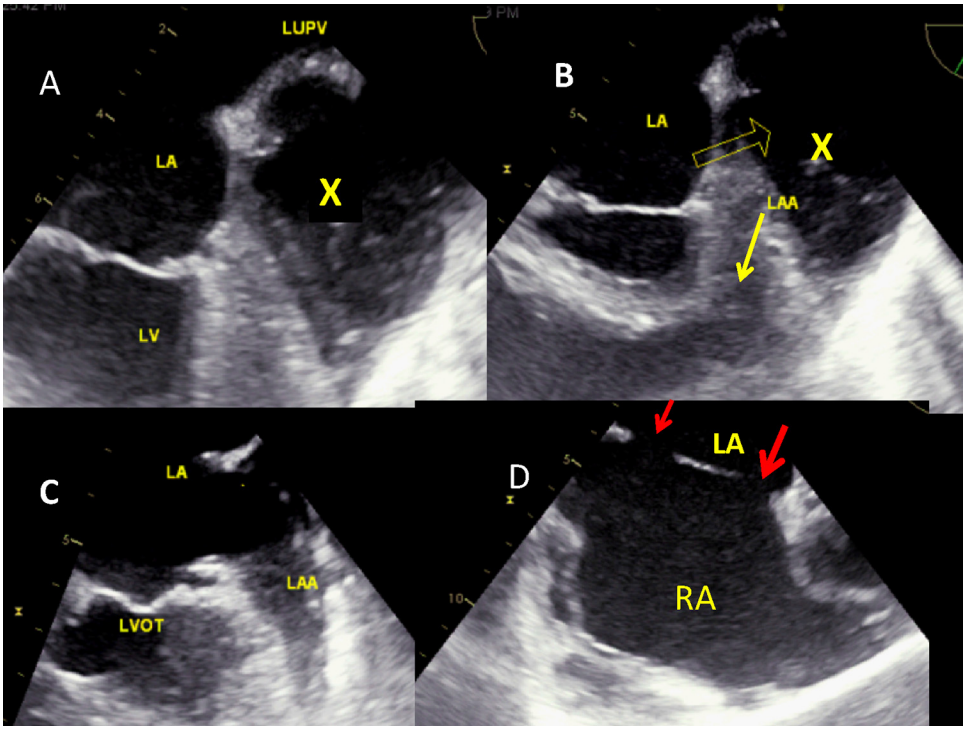

Fig. 5 – Multiple 2D TEE views showing a cystic mass with some echogenic structure in the lower part (X) lateral to the left atrial appendage (C). (D) Two secundum atrial septal defects.

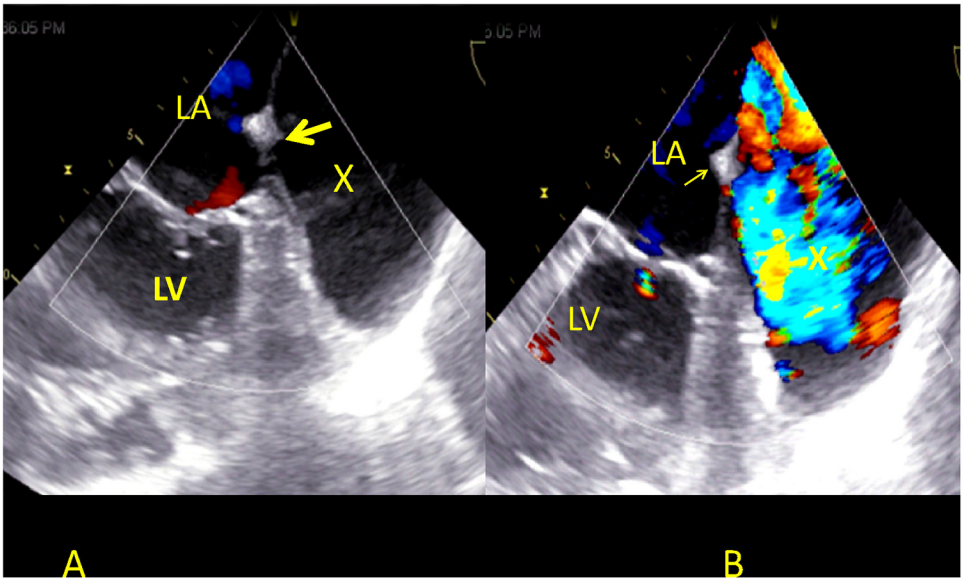

Fig. 6 – Trans-esophageal two-chamber view showing the false chamber (X) communicating with the true chamber (yellow arrow). (B) The false chamber with low velocity swirling flow.

based that involve manipulation of the LA. LA dissection is related to mitral valve surgery as well as coronary artery disease, arrhythmia, trauma, tumor, and spontaneous occurrence.[2–20] Reported incidence rate following mitral valve surgery is 0.16%.[1] In mitral valve surgery, aggressive debridement of the posterior annulus or subvalvular apparatus, oversizing of the prosthesis, inappropriate suturing or intense traction applied on the annulus might all be contributory as a cause of laceration of the annulus or bleeding from the AV groove. LA dissection most frequently occurs along the posterior wall of the left atrium. Unlike the anterior leaflet, the attachment of the posterior leaflet to the annulus is primarily muscular with a little or no fibrous tissue. In addition, the attachment of the anterior leaflet is tougher and has fewer calcifications than that of the posterior leaflet.[1] The AV groove also provides a naturally weakened transitional area vulnerable to over-distension and forceful manipulation.[1] It is postulated that this anatomical difference accounts for the tendency of occurrence of LA dissection on the posterior wall in patients undergoing mitral valve surgery Aside from mitral procedures, aortic valve replacement,[5] coronary artery bypass grafting,[6] left ventricular aneurysmectomy,[7] pulmonary vein cannulation[8] and cardiac mass excision[9] have been associated with LA dissection. There have been non-cardiac surgical aetiologies described in the literature, including myocardial infarction, percutaneous coronary intervention[11]], radiofrequency ablation,[12] infective endocarditis,[20] and blunt cardiac trauma.[21–22] LA dissection due to radiofrequency ablation may be caused by creation of an endocardial flap of the LA wall due to a stiff wire manipulation.

The dissection may form a large cavity between the endocardium and epicardium of the LA, causing obliteration of the LA cavity and resultant hemodynamic compromise due to the mass effect, obstruction to mitral valve inflow, pulmonary artery hypertension, mitral insufficiency, right heart failure, and LA thrombus. These complications almost always require immediate surgical intervention. In our second case, hemodynamic deterioration and heart failure occurred due to mass effect in the LA, which raised pulmonary venous pressure and possibly increased left-to-right shunt across the atrial septal defects. However, it may be asymptomatic with stable hemodynamics with no mass effect or mitral insufficiency as in our first case and recognized with the routine use of TEE for a variety of conditions and improvement in the accuracy of echocardiographic interpretation. As the clinical course of LA dissection varies from a self-limited stable disease (without mass effect, mitral valve obstruction or insufficiency) managed conservatively (24%) to a devastating dissection with catastrophic outcomes (76%), it suggests two distinct entities, although a clear separation into either category is often difficult.[1]

In review of literature, 9 cases of spontaneous LA dissection without any intra-cardiac manipulations have been reported.[4,13–20] Some of these spontaneous cases were attributed to underlying pathology including 3 cases of amyloid light-chain amyloidosis,[15,16,19] 2 cases of severe mitral annular calcification,[4,6] one case of infective endocarditis[20] and three were truly spontaneous without any known etiology. None of our cases had mitral annular calcification or endocarditis. Relation of stroke to LA dissection is not clear although thrombus can form in the false chamber or at the site of communication. Stroke has not been described as a presenting feature in previous reports. In our second case, thrombo-embolism may have been related to atrial fibrillation rather than LA dissection.

Although there are no definitive diagnostic criteria, the TEE findings can be vital, which include: (1) a gap from the mitral annular area to the LA wall or atrial septum, (2) the false chamber appears as an echolucent area causing or not causing partial obliteration of the atrial cavity, (3) mitral and tricuspid

regurgitation, (4) mitral valve periprosthetic leak, and (5) pulmonary venous obstruction. The efficacy and diagnostic value of transthoracic echocardiography, computed tomography scan and magnetic resonance imaging may be limited. An entry of dissection or communication between the inflow and the LA on TEE, even in the presence of expanding dissection, is often difficult to visualize. The echocardiographic findings of LA dissection are distinct but may resemble a left atrial mass, aneurysmal dilatation of the coronary sinus os, cyst and loculated cardiac tamponade, especially when the dissection is focal and containing clots. Computed tomography and contrast-enhanced magnetic resonance imaging may show an oval or oblong mass not dissociable from the posterior wall without significant enhancement after contrast injection. However, it is a challenging entity to diagnose even when the best imaging techniques are applied. Only surgical exploration, sometimes, can clarify the nature of the mass. Serial imaging follow-up studies of non-operatively managed cases have shown resolution of the dissected cavity, suggesting a remodeling process of the LA wall (6). We did not observe resolution of false chamber in our two cases.

Conflicts of interest

The authors have none to declare.

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