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Case Report

An unusual echocardiographic presentation of left atrial dissection following cardiopulmonary cerebral resuscitation

Chia-Ling Lee ^{a, b, *}, Yi-Tso Cheng ^c, Ming-Chon Hsiung ^d, Chen-Fuh Lam ^b^a Institution of Pharmacology and Toxicology, Tzu Chi University, Hualien, Taiwan^b Department of Anesthesiology, Buddhist Tzu Chi General Hospital, Hualien, Taiwan^c Department of Thoracic and Cardiovascular Surgery, Buddhist Tzu Chi General Hospital, Hualien, Taiwan^d Division of Cardiology, Cheng Hsin General Hospital, Taipei, Taiwan

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ABSTRACT

Left atrial dissection (LatD) is a relatively rare perioperative complication during mitral valve repair. Here, we report a 64-year-old man who developed LatD after cardiopulmonary resuscitation was needed due to an extensive myocardial infarction. Aggressive cardiac massage resulted in cardiac rupture and massive bloody pleura effusion. Intraoperative three-dimensional transesophageal echocardiography revealed posterior-medial papillary muscle ruptured and separation of the endocardium from the left atrial myocardium. We speculate that this is the first report in the literature of LatD after cardiopulmonary cerebral resuscitation.

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1. Introduction

Left atrial dissection (LatD) is a rare diagnosis that is characterized by the creation of a false chamber distinct from the true left atrium. LatD is mostly related to complications after mitral valve repair surgery, as well as when there is cardiac trauma and percutaneous intervention affecting the heart [1]. Transesophageal echocardiography (TEE) is the mandatory tool for evaluating cardiac structure and function during cardiac surgery. For patients with a massive myocardial infarction (MI), myocardial rupture is a predictable complication that can be diagnosed by cardiac tamponade. We report a patient who was shock 2 days after primary percutaneous coronary intervention (PCI). After cardiopulmonary cerebral resuscitation (CPCR) and secondary PCI, he was finally diagnosed with myocardial rupture by the presence of massive left side bloody pleura effusion. An accidental finding of LatD by three-dimensional (3D) TEE was also noted during the surgery. The patient did not survive after the myocardial repair surgery. In this

study we analyze cause and effect in relation to this patient and review the etiologies of LatD and myocardial rupture.

2. Case Report

This 64-year-old man was a heavy smoker with a medical history of dyslipidemia and was diagnosed with acute MI at his local hospital. After primary treatment with aspirin, clopidogrel, morphine, and heparinization, he was transferred to our medical center for primary PCI. Cardiac catheterization indicated total occlusion of the left anterior descending artery (LAD) and 80% stenosis of the right coronary artery. Two drug eluting stents were placed in the LAD under appropriate venodilation therapy and heparinization. Unfortunately, 2 days after the procedure, the patient was found to have suffered loss of consciousness and had developed pulseless electric activity on the electrocardiogram. CPCR was performed immediately and transthoracic echocardiography revealed no evidence of cardiac tamponade. Urgent extracorporeal membrane oxygenation was started together with the installation of an intra-aortic balloon pump. After successful CPCR and mechanical cardiopulmonary support, the patient was readmitted to the cardiac catheterization room for coronary angiography, which showed a patent LAD but a severely stenotic right coronary artery. At the same time, the extracorporeal membrane

Conflict of interest: none.

* Corresponding author. Department of Anesthesiology, Buddhist Tzu Chi General Hospital, 707, Section 3, Chung-Yang Road, Hualien, Taiwan. Tel.: +886 38561825x6238; fax: +886 38560977.

E-mail address: brainynanna@gmail.com (C.-L. Lee).

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oxygenation had become dysfunctional and the flow rate had decreased. Two-dimensional TEE revealed collapse of the right heart chambers and a newly developed massive left pleural effusion; the latter was found to be bloody by tapping. An urgent sternotomy was performed due to a high suspicion of cardiac rupture.

Following sternotomy, two ruptured sites in the anterior wall of the left ventricle (LV) were identified and the perforations were closed by direct cardiac suture. A TEE equipped with a 3D probe (Philips, Eindhoven, The Netherlands) was inserted and severe mitral regurgitation (MR) with rupture of the posterior medial papillary muscle was seen. Furthermore, an endocardial flap that had separated from myocardium was also noted; this had originated from the posterior mitral leaflet and extended to the posterior aspect of the left atrium (Fig. 1). LatD was then carried out (Fig. 2). Due to severely impaired systolic function, despite the administration of inotropic agents, the patient passed away after cardiac repair.

3. Discussion

Our patient had an MI that led to myocardial rupture and LatD. The most common etiologies of myocardial rupture include recent MI, cardiac trauma, endocarditis and cardiac tumors [2]. Two types of LV free wall rupture have been reported based on perioperative findings, these are the blow-out and the oozing types [3]. Our patient had extensive myocardial injury involving ruptures of the LV anterior wall and papillary muscle. The fact that cardiac tamponade was not recognized at the beginning of the CPR might have been due to the slow oozing of blood from the LV wall; furthermore, this might then have been exacerbated after massive fluid resuscitation and aggressive chest compression. Aggressive chest compression could also have contributed to pericardium rupture into the pleural space. Therefore, the absence of pericardial effusion during cardiac resuscitation may not necessarily exclude the development of cardiac rupture.

The patient was accidentally noted by 3D TEE to have LatD accompanied by severe MR secondary to the rupture of the posterior–medial papillary muscle. Although chordae or papillary muscle rupture with acute MR is not a rare complication of MI and usually requires emergency surgical intervention [4], post-MI-related LatD has been rarely reported. Dissection of the left atrial endocardium is more commonly diagnosed as a relatively rare complication after mitral valve surgery. Other causes of LatD reported in the literature include valvular endocarditis, left atrium

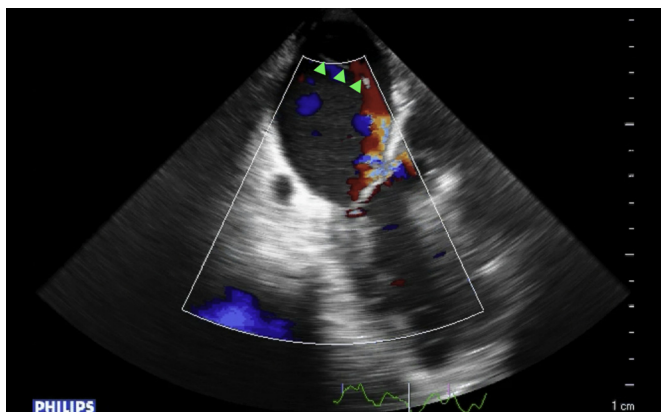


Fig. 1. Two-dimensional color Doppler of the transesophageal echocardiography image. Severe mitral regurgitation was noted along with a left atrial dissection flap. Green triangles: left atrial dissection flap.

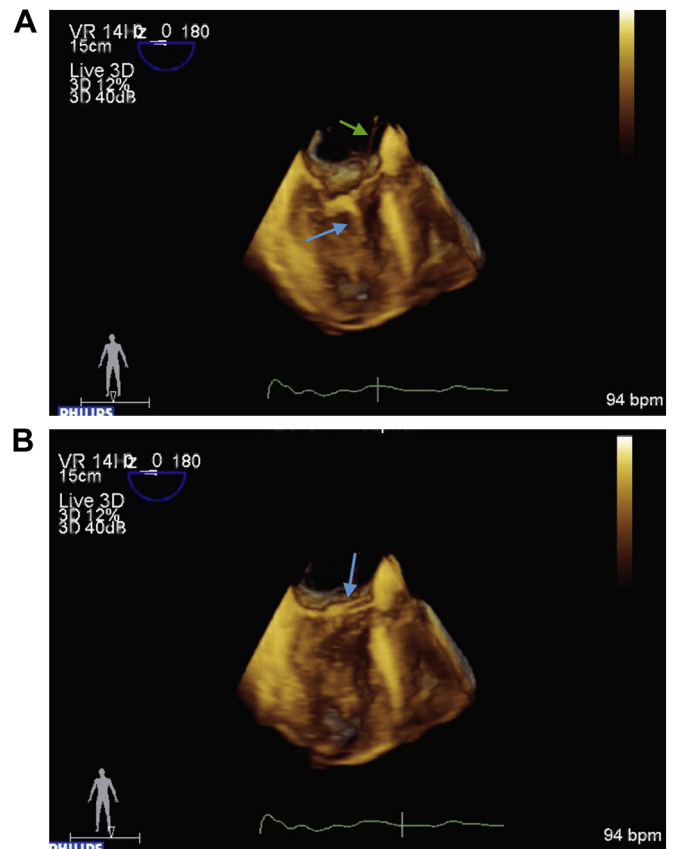


Fig. 2. Live three-dimensional transesophageal echocardiography images. (A) reveals the diastolic phase of the left atrium and left ventricle, a ruptured papillary muscle (blue arrow) and dissection of the left atrium (green arrow). (B) Reveals flail of the anterior mitral leaflet together with rupture of the papillary muscle (blue arrow) during the systolic phase.

hematoma after excision of a LA myxoma, radiofrequency ablation of atrial tachycardia and cardiac trauma [5–8]. The possible pathogenesis of LatD may be atrioventricular junction injury resulting in the separation of the endocardium from the myocardium within the left atrium along the posterior mitral annulus, thereby causing dissection or hematoma in the left atrium [9]. According to expert opinion, the development of intraoperative LatD does not always require left atrial reconstruction. Close observation of the patient's hemodynamics and further echocardiography or cardiac magnetic resonance follow-up are, however, necessary [4]. The indication for surgical intervention are considered to be the severity of the blood flow disruption from the pulmonary veins through the mitral valve [1,5].

In our opinion, the application of excessive transmural pressure on the left atrial wall during cardiac massage generated high fluid shear force along the endocardium of the posterior left atrium and this resulted in separation of the endocardium from myocardium. Nevertheless LatD was an accidental echocardiographic finding during the aggressive cardiac resuscitation of our patient, and rupture of the cardiac free wall associated with poor systolic function was the most likely cause of death.

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