CASE REPORT
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Late Simultaneous Presentation of Left Ventricular Pseudoaneurysm and Tricuspid Regurgitation after Blunt Chest Trauma

Ho-Ki Min[1], Do Kyun Kang[1], Hee Jae Jun[1], Youn-Ho Hwang[1], Sang-Hoon Seol[2], Kyubok Jin[2], Jong Woon Song[3], and Cheol Kyu Oh[4]

Departments of [1]Thoracic and Cardiovascular Surgery, [2]Internal Medicine, [3]Radiology, and [4]Urology, Haeundae Paik Hospital, Inje University College of Medicine, Busan, Korea

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Address for Correspondence:
Ho-Ki Min, MD
Department of Thoracic and Cardiovascular Surgery, Haeundae Paik Hospital, Inje University College of Medicine, 875 Haeundae-ro, Haeundae-gu, Busan 612-030, Korea
Tel: +82.51-797-3135, Fax: +82.51-797-3135
E-mail: minhoki@naver.com

A 32-yr-old man developed progressive exertional dyspnea 4 yr after blunt chest trauma due to an automobile accident. Two-dimensional echocardiography and computed-tomographic coronary angiography demonstrated a large pseudoaneurysm of the left ventricle and severe tricuspid regurgitation. The patient underwent successful surgical exclusion of the pseudoaneurysm by endoaneurysmal patch closure and repair of the tricuspid valve regurgitation. To the best of our knowledge, this is the first case of these 2 different pathologies presenting late simultaneously after blunt chest trauma and successful surgical repairs in the published literature.

Key Words: Wounds and Injuries; Tricuspid Valve Insufficiency; Aneurysm; False

INTRODUCTION

Blunt chest trauma may result in various cardiac defects including tricuspid valve injury, mitral valve avulsion, ventricular septal rupture, pseudoaneurysm, and true aneurysm (1). We report a case of a left ventricular pseudoaneurysm (LVP) and severe tricuspid regurgitation (TR) presenting coincidentally 4 yr after a blunt chest injury. Herein we describe successful surgical repair of the tricuspid valve and exclusion of the pseudoaneurysm by endoaneurysmal patch closure between the aneurysm and the left ventricular cavity.

CASE DESCRIPTION

A 32-yr-old man visited our out-patient-department with paroxysmal coughing and dyspnea on exertion in July 2011. He had suffered from multiple traumas due to an automobile accident 4 yr earlier in a foreign country. At that time, he underwent hepatic resection for liver injury and thoracostomy for hemothorax. Blood pressure was 120/80 mmHg, heart rate was 90 beats per min, and other vital signs were normal. On auscultation, systolic murmurs were noted on the right parasternal border, which increased by inspiration. Chest roentgenogram showed an enlarged heart and a circular mass-like lesion overlapped with the cardiac silhouette. Two-dimensional echocardiography revealed a large pseudoaneurysm arising from the inferobasal surface of the left ventricle with a neck measuring 18 mm and the sac 87 mm at its widest diameter, and severe tricuspid valve regurgitation due to the prolapsed anterior leaflet. The right ventricle was dilated with a normal systolic function and the left ventricular function was preserved (Fig. 1).

Transesophageal echocardiography confirmed these impressions. The coronary CT angiography showed normal coronary arteries and the presence of a large left ventricular pseudoaneurysm (Fig. 2).

The operation was performed using cardiopulmonary bypass and cardioplegic arrest. The pericardium was free of adhesions. The aneurysm was located close to the medial side of the left circumflex artery and near to the cephalic side of the coronary sinus. The aneurysm was entered and organized thrombi were removed. Additionally, we approached to the left atrium using transseptal approach and identified the opening of the aneurysm and the relation between the aneurysmal neck and subvalvular apparatus via the mitral valve. The neck of the aneurysm was identified on each side of the aneurysm, both externally and internally (Fig. 3). Then, a patch of Dacron was su-

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tured at the base of the pseudoaneurysm by multiple pledget-buttressed 4-0 Prolene (Ethicon, Somerville, NJ, USA) and reinforced by a single running suture of 4-0 Prolene, to close the communication between the left ventricle and the pseudoaneurysm. The external wall of the pseudoaneurysm was resected and closed with a double row of 4-0 Prolene by placing two wide strips of Teflon felt on each side. After a saline test, the prolapsed anterior leaflet of the tricuspid valve was confirmed near to the annulus due to the rupture of chordae and the annulus was dilated. The anterior leaflet was fused to the septal leaflet adjacent to the annulus using a simple 5-0 Prolene suture and was stabilized with an Edwards MC^3 annuloplasty ring (Edwards Life-Sciences, Irvine, CA, USA). The patient was easily weaned off bypass, and transesophageal echocardiography showed no residual communication, the achievement of a complete repair of the aneurysmal cavity, and a successful repair of the valve without detectable regurgitation.

The postoperative course was uneventful, and the patient was discharged to home on the seventh postoperative day. Transthoracic echocardiography confirmed that the left ventricle had a normal size, normal shape, good systolic function,

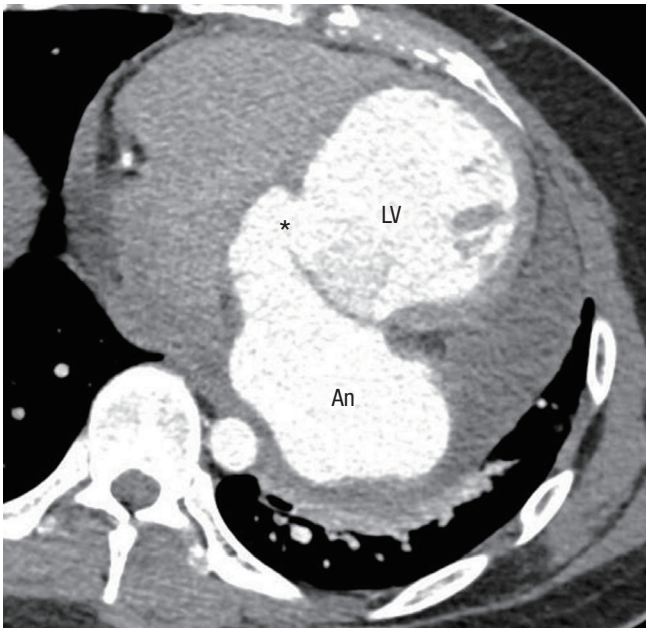

Fig. 2. Enhanced chest CT scan with axial image shows the pseudoaneurysm (An) of the left ventricle (LV) at the level of the mitral valve, which is communicating via the small opening (*).

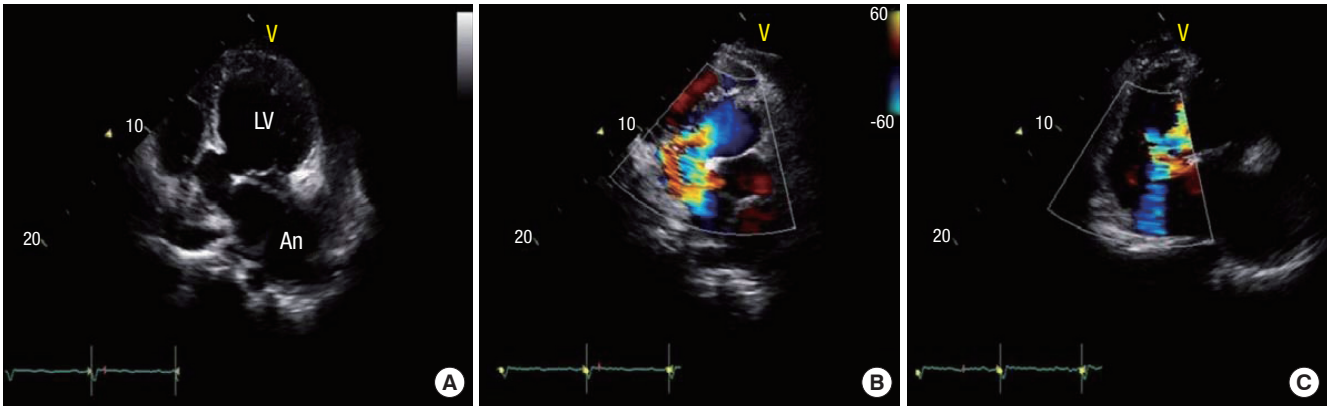

Fig. 1. Transthoracic echocardiographic images from the apical four-chamber view. (**A**) The pseudoaneurysm (An) is clearly demonstrated, (**B**) with aberrant flow through the basal left ventricular (LV) communication on color flow mapping, and (**C**) the modified four-chamber view with color flow mapping reveals severe regurgitation of the tricuspid valve with a prolapsed anterior leaflet.

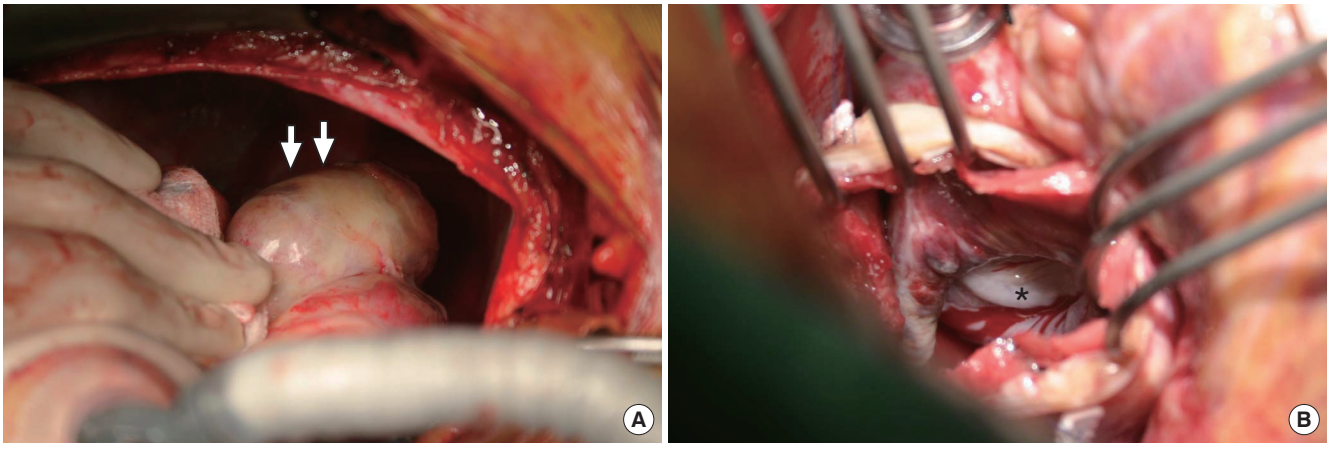

Fig. 3. These photographs show an intra-operative view (arrows) (**A**) and an opened neck (*) of the pseudoaneurysm (**B**).

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trivial TR, and there was no evidence of pseudoaneurysm recurrence before discharge. Clinical follow-up 4 month later showed a healthy young man with improved exercise capacity.

DISCUSSION

Blunt chest trauma may result in various cardiac defects including tricuspid valve injury, mitral valve avulsion, ventricular septal rupture, pseudoaneurysm, and true aneurysm (1).

Traumatic LVP is an uncommon complication of chest trauma. The possible mechanisms of pseudoaneurysm formation after chest injury include a contusion of the myocardial wall, a coronary artery lesion leading to ischemic necrosis, and an intramyocardial dissecting hematoma (2-4).

The clinical course of an untreated LVP is relatively poor (5). They have a high tendency for rupture and sudden death, unlike true ventricular aneurysms, due to their comparatively thinner wall containing the hematoma (6). A "steal" of blood into the aneurysm during systole reduces cardiac output and causes dyspnea and lethargy, which our patient suffered.

Regarding the surgical technique, surgical repair of post-traumatic LVP presents several options (6). With large defects near the base of the heart, a patch closure at the neck of the aneurysm may be preferable to avoid excessive traction on the myocardium or distortion of the coronary artery and coronary sinus. A patch closure is probably more often recommended to preserve the correct shape and function of the left ventricle. For a small defect with the absence of myocardial disease, direct primary suture repair may be an option. In our case, we used a patch closure with excision and oversewing the sac because the defect size was as large as 20 mm and the lesion was adjacent to the left circumflex artery and the coronary sinus.

Post-traumatic TR is a rare occurrence, but due to its anterior location, it remains the most frequently reported valve injury following blunt chest trauma (7, 8). Major mechanisms for traumatic tricuspid valve injury include rupture of the papillary muscles, chordal disruption, free rupture of the leaflets or complete destruction of the valve (7, 8). The surgical strategy should be adapted individually to the patient's specific pathology. Besides the general consensus to repair the valve whenever feasible, various approaches and techniques for effective surgical treatment have been described. In terms of repair, tricuspid annuloplasty is mandatory because the annulus is dilated in most cases. In our case, we used a fusion of the antero-septal commissure followed by the implantation of an annuloplasty ring to reduce and stabilize the annular size.

In this report, the LVP combined with severe TR with the prolapsed anterior leaflet was noted coincidentally 4 yr after blunt chest trauma. To the best of our knowledge, this is the first case of these 2 different pathologies presenting late simultaneously after blunt chest trauma and successful surgical repairs in the published literature.

In conclusion, we report an unusual case of a post-traumatic LVP combined with TR presenting late simultaneously. Clinicians must be aware of this rare, but important pathology, and that it may present late after blunt chest trauma. Repair is targeted safely and effectively with an appropriate technique according to specific pathologies.

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