Case Report

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Left Atrial Appendage Aneurysm: A Case Report

좌심방 부속기 동맥류: 증례 보고

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Left atrial appendage aneurysm (LAAA) is a rare heart anomaly caused by congenital dysplasia of the pectinate muscle or by an acquired pathological condition of the mitral valve or cardiac muscle. It is often incidentally discovered during chest CT or echocardiography as an abnormal dilatation of the LAA. LAAA is associated with life-threatening complications and most patients require surgical treatment. Therefore, it is important to evaluate associated complications as well as precise diagnoses. This report presents the case of a surgically confirmed LAAA in a 53-year-old female. We also discuss the pathophysiology of LAAA and significant findings related to mortality that can be detected on CT and MRI.

Index terms Left Atrial Appendage; Left Atrial Appendage Aneurysm; Computed Tomography, X-Ray; Magnetic Resonance Imaging

INTRODUCTION

Left atrial appendage aneurysm (LAAA) is a rare heart anomaly. It is mostly asymptomatic and often be diagnosed incidentally on echocardiography or CT. However, some patients show nonspecific cardiovascular symptoms, tachyarrhythmia and thromboembolism. These are associated with cardiovascular morbidity and mortality, therefore early and accurate diagnosis is important (1, 2). In addition, radiologic findings that induce disturbance of heart and its surrounding structures, or thromboembolism should be considered together (2). In this

case report, we introduce a case diagnosed as LAAA with surgical excision and briefly review the literature.

CASE REPORT

A 53-year-old female has visited the hospital with dyspnea and mild palpitation that had worsened two days ago. She had no other underlying disease. Laboratory findings and physical examination were within normal limits.

On posteroanterior chest radiographs, heart was enlarged, left upper cardiac border was bulging in a round shape, and cardiac apex was uplifted (Fig. 1A).

On enhanced chest CT, left atrial appendage (LAA) was diffusely dilated and its size was approximately 24 mm, 49 mm, and 71 mm in the orifice diameter, body width, and length of the LAA, respectively. There was no filling defect thought to be thrombus and no extrinsic compression of left ventricle (LV), left coronary arteries and cardiac veins (Fig. 1B).

MRI also showed diffused dilated LAA with smooth inner and outer wall. It was located within pericardium (Fig. 1C), and the size was increased in systolic phase and decreased in diastolic phase. There was no significant mass effect such as LV outflow obstruction or compression of left atrium (LA) and LV chambers. Subtle contrast enhancement of aneurysm wall was seen on late delayed T1 weighted image, and it was thought to be some fibrosis of LAAA wall (Fig. 1D). But thrombus was not detected in LAAA on MR image. High signal intensity within LAAA on T1 and T2 weighted turbo spin-echo black blood image was noted and it was considered slow mixing blood (Fig. 1E), and it was also observed on cine image. Cardiac function was normal with no accompanying cardiac anomaly and no perfusion defect presenting myocardial ischemia. These were consistent with uncomplicated LAAA.

The patient underwent excision of LAAA via video-assisted thoracoscopic surgery. LAAA was confirmed in pathology with diffuse fibrosis and a small thrombus (Fig. 1F).

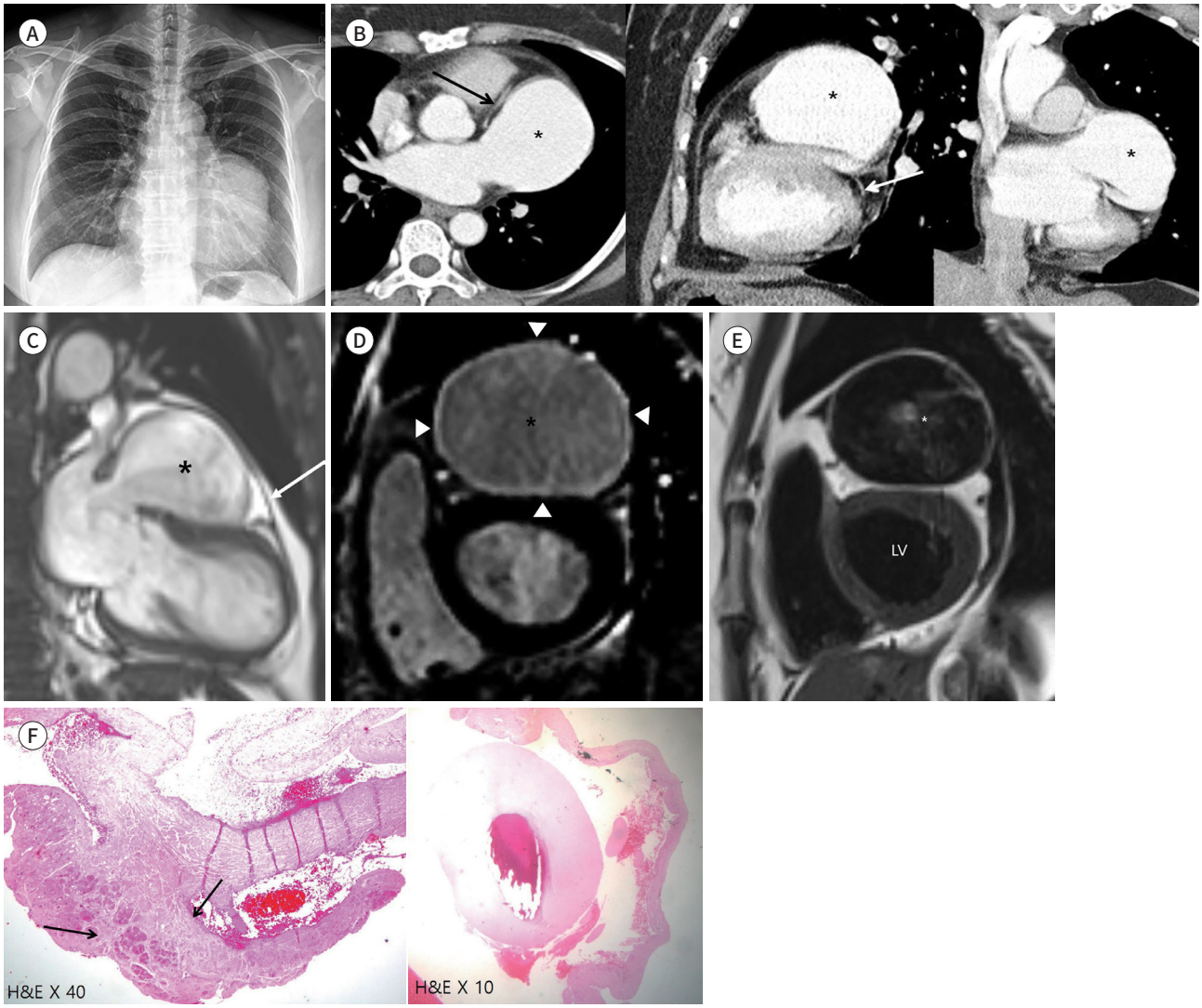
This retrospective case report was prepared according to the ethical principles in the Declaration of Helsinki.

DISCUSSION

LAAA is one of the left atrial aneurysms, and it occurs mainly in the LA appendage, but can occur anywhere in the LA wall (1). LAAA was first published in 1960 by Dimond et al. (3), there were about 150 cases described in the literature. There is no exact cutoff value for size that defines LAAA, but in a quantitative study, LAAA can be defined as an orifice diameter, body width, and length of the LAA greater than 27 mm, 48 mm, and 67.5 mm, respectively (4). LAAA can be classified as congenital or acquired, and congenital type is more common. Congenital LAAA is caused by dysplasia of pectinate muscles and other related atrial muscle bands while acquired LAAA is due to left atrial enlargement resulting from mitral valve disease or myocardial pathologic conditions such as mitral stenosis, mitral regurgitation, syphilitic myocarditis, and tuberculosis (2). Corresponding to the above, the common histopathologic finding of LAAA is fibrosis of endocardium or myocardium, and myocardial hypertrophy and fatty infiltration of myocardial fibers may be seen in some cases (5). LAAA can also be

classified as intrapericardial and extrapericardial depending on the location. The intrapericardial type is caused by the weakening of the left atrial wall and/or appendage. The extrapericardial type is associated with pericardial defect, which can be herniate and can progress to aneurysmal dilatation. The extrapericardial type is known to have a better prognosis (2).

Fig. 1. A 53-year-old female with a left atrium appendage aneurysm.
A. Chest posteroanterior shows cardiomegaly with a bulging contour of the left upper cadiac border and uplifted cardiac apex.
B. Axial, sagittal, and coronal images of enhanced chest CT depict diffuse enlargement of the LAA (24 mm × 49 mm × 71 mm, orifice diameter, body width, and length of the LAAA) with no filling defect or extrinsic compression of the LV and the surrounding major vessels (black arrow in the axial image: left anterior descending coronary artery, white arrow in the sagittal image: cardiac vein, *: LAAA).
C. Two-chamber cine MRI shows diffuse enlargement of the LAA with an intact pericardium (arrow). It reveals a slow mixing flow in real-time (*: LAAA).
D. Delayed (18 minutes) T1 weighted image gadolinium-enhanced sagittal image reveals continuous mural enhancement (arrowheads) without pericardial enhancement (*: LAAA).
E. T2 turbo spin-echo black blood contrast short-axis image shows heterogeneous high signal intensity in LAAA, which suggests low-velocity blood flow (*: LAAA).
F. Histologic specimen shows fibrosis (arrows) of the myocardium of LAAA (right), and a small thrombus within LAAA (left).
H&E = hematoxylin and eosin, LAAA = left atrial appendage aneurysm, LV = left ventricle



LAAA occurs in all ages, the mean age of presentation is 31 ± 20 years (fetus at 28 weeks to 88 years) (1). Clinical manifestation ranges from asymptomatic to palpitation, dyspnea and chest pain. Physical examination is normal to cardiac murmur and irregular pulse. These clinical manifestations are come from accompanying heart anomaly or complication of LAAA (6). The most common accompanying abnormalities are atrial tachyarrhythmia and thrombus. Firstly, with LAAA there is enlargement of the atrium, this provides a substrate in which an electrical signal is triggered or reentrant, causing atrial tachyarrhythmia (6). Secondly, LAA, a small pouch with a lot of trabeculation, provides a favorable environment for clots forming. The bigger the pouch, the more likely it is to develop a blood clot. If there is atrial fibrillation, the myocardium does not contract normally, resulting in blood stagnation and higher blood clotting. The resultant thrombus can induce thromboembolism which is the most common life-threatening complication of LAA. Third, as an aneurysm grows, it can cause mass effects. Most commonly it compresses LV and left coronary artery resulting in cardiac dysfunction and myocardial ischemia (1). Rarely, it presses the left recurrent laryngeal nerve causing chronic cough and irritation of the left phrenic nerve, causing hiccups (7, 8). Fourth, LAAA causes volume overload to LA. This induces slow LA myocardium remodeling, and subsequent left atrial diastolic and LV dysfunction. Although it is very rare, a LA rupture can cause cardiac tamponade (6).

Transthoracic echocardiography (TTE) is considered the primary diagnostic tool. However, it is limited by low sensitivity and its invasive nature. Aryal et al. (5) reported that the sensitivity of TTE is 45%, and LAAA on TTE is easily misinterpreted as a pericardial cyst, cavity or pseudoaneurysm of coronary artery and LV (1). On the other hand, it is easy to monitor the blood flow of the aneurysm, and the pathogenic condition of the LA and mitral valve can be easily identified using color doppler. Although invasive, transesophageal echocardiography (TEE) is superior to thrombus detection in LAAA.

CT is an effective diagnostic tool that not only helps to identify the location, morphology, and size of the aneurysm but also the relationship with the surrounding structure, and offer the benefit of objective tool than TEE. LAA is a finger shaped protrusion from the LA and is divided into an orifice, body, and apex. It is located above the LV and below the main pulmonary artery and forms the left border of the heart. It has an axis parallel to left pulmonary vein, driving upper wall of LV and forming a tip towards the pulmonary trunk. Left circumflex artery and great cardiac vein travel below the LAA, and left phrenic nerve passes through the posterolateral side of LAA (6). The size and shape of the LAAA should be described on the CT image, as its large size and irregular shape may be associated with thrombus formation. The positional relationship between aneurysm and main blood vessels such as the left circumflex coronary artery, pulmonary veins and left phrenic nerve should be also examined. These surrounding structures can be compressed by LAAA and may be damaged during surgical excision. The location of the pulmonary veins near the LAAA orifice and the accompanying cardiac anomalies also should inform whether surgical treatment may be affected. Multiplanar reconstruction and volume rendering can provide better anatomical description. In addition, when there is a delayed phase image of contrast enhanced CT, the detection of thrombus has a very high positive predictive value of 92%. However, it must be distinguished from filling defects due to stagnant blood flow.

MRI has excellent anatomic resolution owing to its superior soft tissue contrast and functional information of the myocardium. On T1 weighted images, intra- or extra-pericardial LAAA is distinguished by observing defect of the pericardium or pericardial fat. Cine MRI images enable observation of the blood mixing pattern inside the aneurysm and the dynamic mass effect of the aneurysm during the cardiac cycle. Delayed contrast enhancement on T1-weighted image can be suggested LAAA wall fibrosis (9). Slow blood flow in LAAA can be detected by internal hyperintensity on spin echo black blood contrast image (10). Fibrosis of the LAAA wall is can be associated with impaired LAAA conduction and reduced contractility, and may increase the risk of thromboembolism, along with stagnant blood flow in the LAAA cavity (6, 9). Therefore, it should be included in the evaluation of LAAA. However, artifacts in patients with arrhythmia can lower the image quality.

Surgical intervention is recommended for the management of LAAA (1, 5). But if the patient is asymptomatic and does not have a thrombus, anticoagulation therapy with close monitoring should be considered. The points to be evaluated in the postoperative image varies depending on surgical method. Above all, it is important to determine whether there is left atrial deformity or residual aneurysm.

In conclusion, LAAA is a rare but clinically significant disease. For accurate diagnosis and prevention of complications, radiologists should understand the pathophysiology and accompanying complications of LAAA.

Author Contributions

Conceptualization, C.Y.J., K.J.S.; data curation, C.Y.J., K.J.S.; investigation, all authors; methodology, C.Y.J., K.J.S.; project administration, K.J.S.; resources, all authors; supervision, K.J.S.; validation, C.Y.J., K.J.S.; visualization, C.Y.J., K.J.S.; writing—original draft, C.Y.J.; and writing—review & editing, K.J.S.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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좌심방 부속기 동맥류: 증례 보고

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좌심방 부속기 동맥류는 심장 빗살근의 선천성 이형성이나 승모판 또는 심근의 후천적 병변에 의해 생길 수 있는 매우 드문 심장 질환이다. 주로 흉부 CT나 심초음파에서 좌심방이 비정상적 팽창되어 우연히 발견되며, 환자의 대부분은 무증상이지만 생명을 위협하는 합병증과 관련이 있어 외과적 치료를 필요로 한다. 그러므로 정확한 진단과 함께 이에 동반된 합병증을 찾아내는 것은 중요하다. 본 증례 보고에서는 수술적 치료로 확진 된 53세 여성의 좌심방 부속기 동맥류를 소개하고 병태생리학을 바탕으로 진단과 함께 놓치지 말아야 할 합병증에 대한 CT 및 MRI 검사의 소견을 소개하고자 한다.

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