

Case Report

Echocardiographic diagnosis of giant coronary aneurysm: a case report

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Abstract: Giant coronary artery aneurysms are uncommon and may be congenital or acquired. Few cases of Giant coronary artery aneurysms have been reported in the literature. We describe a case of giant coronary artery aneurysm located in the pericardial cavity of a 61-year-old female. The patient presented with recurrent palpitations and shortness of breath after physical activity. Transthoracic echocardiographic revealed a 10.3 cm × 9.2 cm spherical mixed echo mass in the left ventricular wall within the pericardial cavity. A surgical resection was performed with an excellent outcome and no abnormal signs after 1-year follow-up.

Keywords: Echocardiography, coronary aneurysm

Introduction

Coronary aneurysm refers to focal or diffuse dilation of coronary arteries, which is caused by a variety of etiologies. Coronary dilatation exceeds the diameter of normal adjacent artery segments by 1.5-fold. A giant coronary aneurysm is defined as coronary dilatation that exceeds the diameter of normal adjacent segments by four-fold [1]. The incidence of coronary aneurysm was very low, but it has been diagnosed with increasing frequency given the popularity of coronary angiography. The incidence is approximately from 0.02% to 0.03% in the normal population and from 1.4% to 4.9% in individuals with coronary angiography. However, giant coronary aneurysm is relatively rare [2]. Although greater than 50% of coronary aneurysms are suspected to be associated with atherosclerosis, the exact etiology is still not completely understood. Interestingly, a common histopathological finding is damage to the medial layer artery in aneurysmal dilated coronary segments [3]. In this work, we report a rare case of adult giant coronary aneurysm that was successfully removed by surgery.

Case report

A 61-year-old female was admitted to the hospital due to a 2-year history of recurrent palpita-

tions and shortness of breath after physical activity that was exacerbated for 1 month. On physical examination, her heart rate was 71 beats/min with normal heart rhythm. Her blood pressure was 106/62 mmHg (1 mmHg = 0.133 kPa). Distension of the jugular vein and cyanotic lips were not observed. Her chest was clear to auscultation bilaterally. Her precordium appeared normal. The point of maximal impulse was enlarged with visible heaving. Palpable thrill was noted on the apical area. No pericardial rubs were present. The electrocardiogram (ECG) revealed sinus rhythm with ST-T changes in some leads and low QRS voltage in left chest leads. The apical four-chamber view of preoperative transthoracic echocardiography revealed pericardial fluid sonolucent area and a 10.3 cm × 9.2 cm spherical mixed echo mass (predominate with solid area) in the left ventricular wall within the pericardial cavity. The left ventricular wall was significantly displaced medially due to compression of the mass. A 0.25-cm wide sonolucent tubular structure was noted on the surface of the mass, and its opening is located in the cystic area of the mass. Color Doppler flow imaging (CDFI) revealed no blood flow signals in the solid area of the mass, but an arterial blood flow spectrum was noted in the sonolucent tubular structure. The blood flow velocity was 1.6 m/s. A multicolored jet

Giant coronary aneurysm

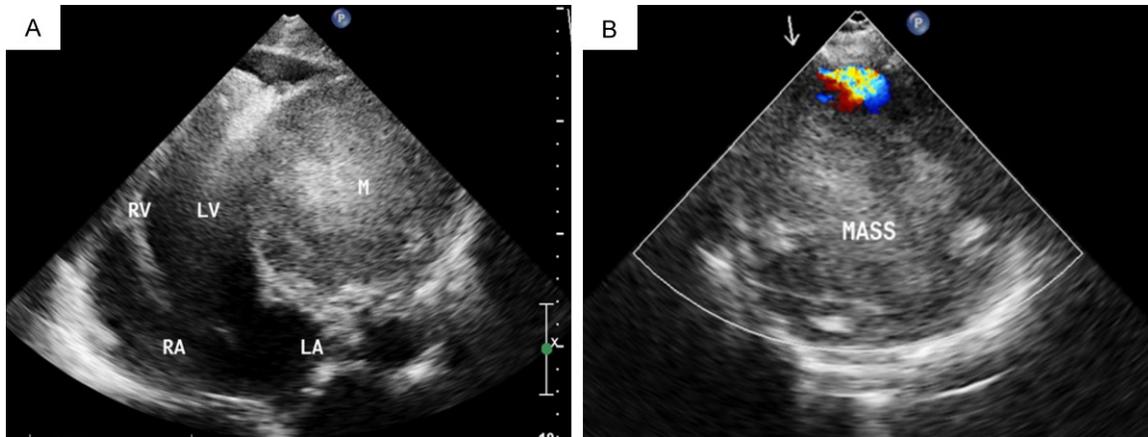


Figure 1. Echocardiographic findings. A. A solid mass is located in the lateral wall of the left ventricle within the pericardial cavity, compressing the lateral wall of the left ventricle; B. Color Doppler Flow Imaging (CDFI) revealing a multicolor jet flow at the opening of the tubular structure. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; M, mass.

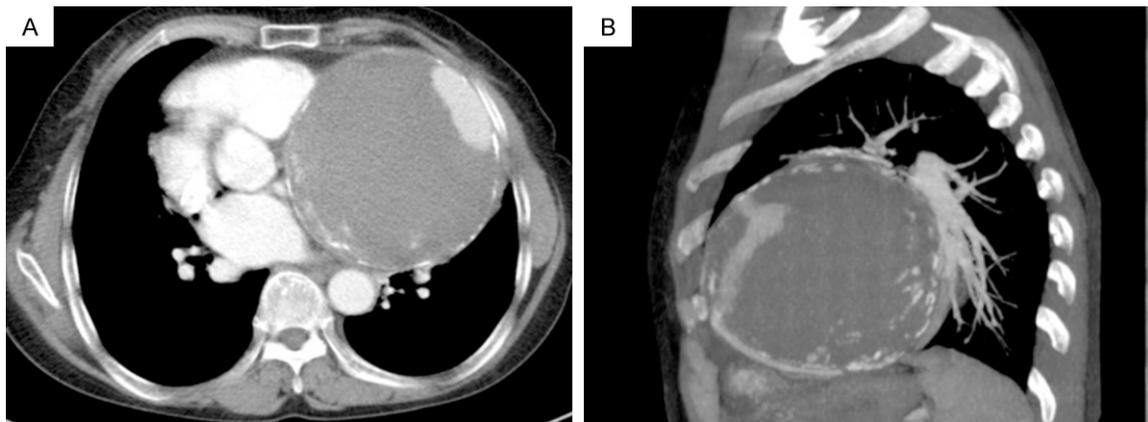


Figure 2. Enhanced CT scan findings. A. Axial images revealing a nearly rounded, low-density mass in the left pericardium; patchy-like calcifications are noted at its periphery; vascular enhancement with contrast is seen within the mass; B. Sagittal images revealing irregular tumor-like dilation in the distal left coronary artery; the proximal left coronary artery is apparently compressed and shifted.

flow was observed during systole at the opening of the tubular structure. The flow rate was faster at approximately 1.8 m/s (**Figure 1A, 1B**). The ultrasound diagnoses are as follows: giant coronary aneurysms with thrombosis and pericardial effusion. Chest plain and enhanced CT scan revealed a nearly round, low-density, well-defined, 11.3 cm × 10.2 cm mass in the left ventricular wall in the pericardial cavity, and multiple patchy-like calcifications were noted at its periphery. Enhanced CT scan revealed a strip-patch-like enhanced area within the mass. A thicker “blood vessel shadow” that connects to the left coronary artery was observed in the enhanced area (**Figure 2A, 2B**). The signs of a

small pericardial effusion and compression of all atriums and ventricles were noted. No enlarged mediastinal lymph nodes were present. CT diagnoses are as follows: distal left coronary aneurysm and mild pericardial effusion. Coronary angiography revealed that the left coronary artery opening was extremely enlarged, and a giant aneurysm had formed in its distal end. The right coronary artery opening was normal. The diagnosis of giant left coronary aneurysm was confirmed.

Surgical findings revealed no significant enlargement and deformity of the heart. Mild-moderate pericardial effusion was present in

Giant coronary aneurysm

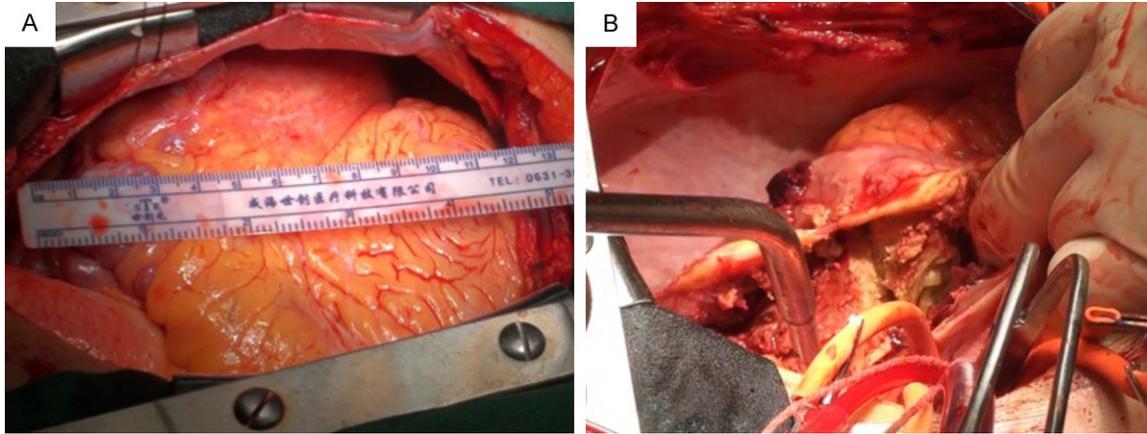


Figure 3. Intraoperative findings. A. A round mass is palpable in the left pericardial cavity; B. A large number of dark-reddish thrombi are noted while incising the mass.

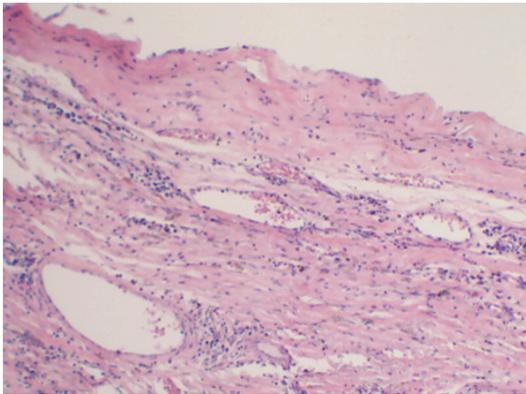


Figure 4. Pathological diagnosis confirms coronary artery aneurysms and thrombosis (H & E staining, $\times 100$).

the pericardial cavity. A round mass measuring 11 cm \times 9 cm \times 8 cm was observed in the left side of the heart and firmly attached to the left ventricular wall. The mass is tough and unmovable with no sign of fluctuation. The mass had a smooth surface and pericardial adhesions and could be completely separated from the pericardium by blunt dissection. When the mass was incised, the solid component predominates, i.e., many dark-reddish thrombi with partial organization. The mass wall was 0.5 cm thick (Figure 3A, 3B). A vascular opening that is 0.2 cm in the inner diameter was observed in the anterior-inner wall of the mass, from which cardioplegic solution drained at the time of aortic antegrade perfusion. Further exploration revealed that the opening leads to the left circumflex coronary artery. No communicating branch with other blood vessels was observed.

Under general anesthesia and cardiopulmonary bypass, giant coronary aneurysm resection and coronary artery bypass grafting were performed.

Gross pathological examination revealed a 9 cm \times 7 cm coronary aneurysm wall and a 6 cm \times 5 cm coronary thrombus (reddish and grayish tissue debris) that exhibited a tough cutting section sent out for pathological examination. Microscopic pathological examination reveals an intima defect of the middle segment of left coronary artery and the thrombus attached to coronary aneurysm wall that contained a large number of scattered lymphocytes infiltrates. Increased fibrous tissue with collagen formation between smooth muscle bundles and focal calcification was identified (Figure 4). The pathological diagnosis was coronary aneurysms with thrombosis.

Discussion

Coronary artery aneurysm refers to focal or diffuse dilation (spherical, sac-like and fusiform shape) of coronary arteries caused by a variety of etiologies. Coronary dilatation exceeds the diameter of normal adjacent segments by 1.5-fold. Giant coronary aneurysm is diagnosed when the coronary dilatation is four-fold increased compared with the diameter of normal adjacent segments. The incidence is approximately from 0.02% to 0.03% in the normal population and 1.4% to 4.9% in individuals with coronary angiography. However, giant coronary aneurysm is relatively rare [2]. Coronary aneurysm typically occurs in the proximal and

Giant coronary aneurysm

middle right coronary arteries or secondarily in the left anterior descending artery and the left circumflex artery. The condition rarely occurs in left main branch and other small branches [4].

The major cause of coronary aneurysm is atherosclerosis followed by congenital coronary artery abnormalities, Kawasaki disease, and coronary artery fistula. The underlying pathogenesis remains unclear. Currently, it is believed that coronary elastic fiber (medial layer muscle) damage caused by atherosclerotic lesions is responsible for aneurysm formation. The thin and weak arterial wall bulges outward under blood pressure and results in a spherical coronary or cystic dilatation [5]. Thrombosis is always present in coronary aneurysms, but rupture of the aneurysm is not common. Histopathology reveals increased endometrial fibrosis in the aneurysm wall, loss of the elastic lamina between the intima and media layer, accumulation of a large number of myxoid matrix in the media layer, and the formation of multiple small dissections [6]. The etiology of coronary aneurysms differs geographically. In Europe and North America, the cause of coronary aneurysms is coronary atherosclerosis, congenital heart disease, and Kawasaki disease which accounts for 50%, 17%, and 10% of its occurrence, respectively. In Asia, especially Japan, 50% to 60% of coronary aneurysms are caused by Kawasaki disease [7, 8].

In this case, the main differential diagnoses for cystic-solid mass in left ventricular lateral wall within the pericardial cavity complicated by pericardial effusion include the following: (1) Pericardial cyst is the most common pericardial cystic mass that has single cavity, ranging in size from 2 to 16 cm in diameter. It is typically located in the anteroinferior right or right-superior mediastinum. Echocardiography reveals that the cyst is located outside the heart margin and attaches to the pericardium. The cyst wall is smooth and a strong echo dense area can be observed if calcification present. A fluid sonolucent area can be observed within the cyst; (2) Ventricular aneurysm is a complication of myocardial infarction. In this case, it is most likely located in the anteromedial region of the ventricle close to the apex. (3) Pericardial teratoma includes inner, middle and outer layers of ectodermal tissue. Ultrasound imaging can detect strong echo caused by tooth or calcification [9].

Clinical symptoms of coronary aneurysms manifest as non-specific. Angina pectoris or myocardial infarction may be present. In a dilated aneurysm, vascular intimal pathological changes and local hemodynamic abnormalities lead to thrombosis, which causes myocardial infarction. Damage to intimal cells and an abundance of smooth muscle cells can cause vasospasm at the junction of the proximal and distal aneurysm with the normal coronary artery. Coronary angiography is the mainstream method for diagnosis and can determine the location and size of aneurysm and distal coronary artery blood supply. Common complications of giant coronary aneurysm include thrombosis and thromboembolism, which may cause secondary acute ischemic heart disease and even death. Its prognosis is poor. To prevent complications, surgery is the preferred method of treatment [10]. In this case report, coronary aneurysm resection plus coronary artery bypass were performed at the time of diagnosis because the giant mass and thrombus compress the left ventricle and compromise the ejection function of the left ventricle. Postoperative echocardiography revealed no myocardial ischemia. No abnormal signs were reported after 3 months of follow-up. Although coronary angiography is the most valuable tool for the diagnosis of coronary aneurysms, echocardiography is also of great significance in understanding the internal structure of the aneurysm, detecting dilation of the heart chambers, and assessing cardiac function. Therefore, multiple imaging examinations are conducive to the comprehensive diagnosis of coronary aneurysm, and its complications and can be helpful in selecting a surgical approach.

Disclosure of conflict of interest

None.

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Giant coronary aneurysm

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