



CASE REPORT

Giant left anterior descending artery aneurysm resulting in sudden death



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Abstract Coronary artery aneurysm is a rare congenital or vascular inflammation-based anomaly for which the clinical course and optimal timing of treatment remain unclear. Here, we report a case of sudden death caused by a giant coronary artery aneurysm of the left anterior descending artery that presented with chest pain. This case suggests that urgent interventional or surgical repair is needed when a large coronary aneurysm presents with acute ischemic symptoms.

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1. Introduction

A coronary artery aneurysm (CAA) is defined as a dilation of a coronary artery segment exceeding the diameter of normal adjacent segments by 1.5 times.¹ CAAs have a 1.5% to 5% incidence² and most commonly involve the right coronary artery (RCA), followed by the left anterior descending (LAD) and left circumflex (LCX) coronary arteries.³ Giant CAAs (generally defined as >4 times the

normal diameter or >8 mm in internal diameter) have the greatest risk of thrombosis and myocardial infarction as well as a slight risk of rupture.^{4,5} Here, we present a case of giant CAA complicated with acute myocardial infarction (AMI) that resulted in sudden death.

2. Case Presentation

A 46-year-old man was admitted with a 5-day history of chest pain and dyspnea on exertion. His risk factors for coronary artery disease (CAD) included untreated hypertension, obesity (body mass index, 26.9 kg/m²), and current smoking (30 pack-years). There was no personal or family history of connective tissue or infectious disease or of trauma. Upon examination, his blood pressure was 110/70 mmHg, and his heart rate was 104 beats per minute. An electrocardiogram (ECG) showed sinus tachycardia with T-

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wave inversion on V₁₋₃ and mild ST-segment depression on V₄₋₆ (Fig. 1A). No murmur was heard on a precordial auscultation. Blood tests revealed elevated levels of troponin I (0.054 ng/mL), brain natriuretic peptide (331 pg/mL), and glycosylated hemoglobin (8.8%); however, both inflammatory marker and white blood cell levels were normal. A chest roentgenogram suggested no abnormalities except for cardiomegaly (Fig. 1B). A bedside 2D echocardiogram showed hypokinesia of the LAD territory, left ventricular systolic dysfunction (ejection fraction, 35%), and mild compression of the left ventricular anterior wall by a vague cystic mass (Fig. 2). A cardiac computed tomography scan showed a huge cystic mass originating from the LAD ostium, which compromised the right ventricular outflow track (Fig. 3). The patient underwent coronary angiography (CAG), which revealed a large (8 × 8 cm) round aneurysm originating from the proximal segment of the LAD, with minor distal flow originating from the aneurysm (Fig. 4). Irregular ectasia was noted in the proximal segments of the LCX and RCA. The patient was transferred to the intensive care unit in preparation for surgery. Ten hours later, the patient experienced recurrent chest pain with ST-segment elevation in V₁₋₆ and, later, ventricular tachycardia on ECG. The patient died despite resuscitation efforts.

3. Discussion

The primary factor in CAA formation is the presence of abnormalities (erosion, ulceration and hemorrhage) in the vessel wall media layer, possibly as extension of intimal atherosclerosis or inflammation.³ It has been suggested that chronic overstimulation of the production of nitric oxide, an endothelium-derived relaxation factor, might lead to an imbalance between its beneficial and detrimental effects on coronary dilatation.² Although CAAs are most commonly atherosclerotic in origin, they may also be congenital⁶ or secondary to: mucocutaneous lymph node syndrome

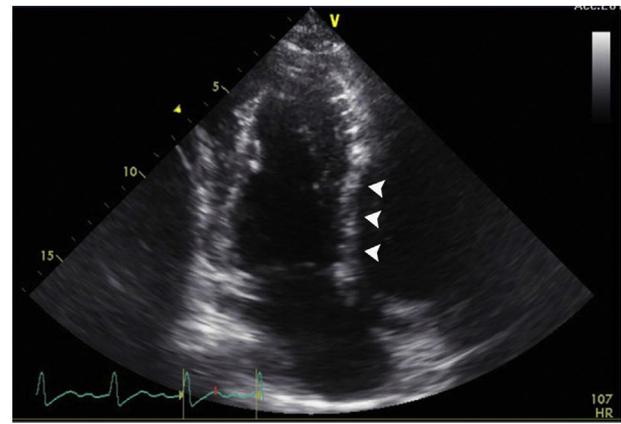


Figure 2 Transthoracic echocardiogram. Apical 2-chamber view at time of hospital admission revealed hypokinesia of anterior wall which was compressed by a very large cystic mass (arrows).

(Kawasaki disease),⁷ connective tissue disease, arteritis, mycotic emboli, coronary artery bypass graft surgery or trauma.^{3,8} Therefore, when available, clinical history and serological tests could facilitate diagnosis. The precise factor underlying CAA development in the patient reported here was unknown; however, atherosclerosis and its associated risk factors (hypertension, undiagnosed diabetes, obesity, and smoking) appear to have played a role.

CAA has varying clinical presentations. The majority of patients are asymptomatic and often identified incidentally during coronary angiography. However, patients often present with ischemic symptoms, such as angina or myocardial infarction,⁹ and with supraventricular or malignant ventricular tachyarrhythmia.¹⁰ Ischemic symptoms may be associated with atherosclerosis, or with thrombosis and distal embolization of coronary arteries near the aneurysm. The presence of dilated coronary segments

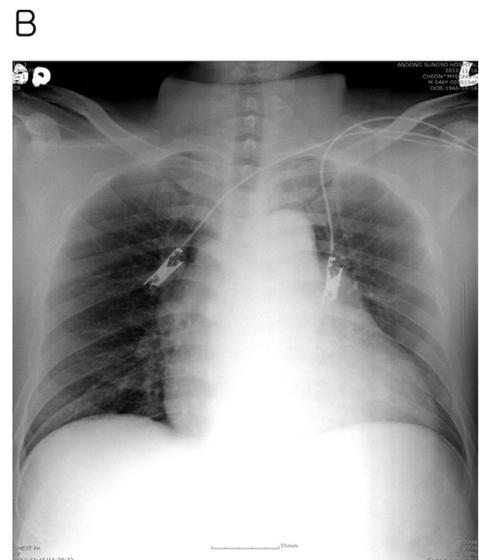
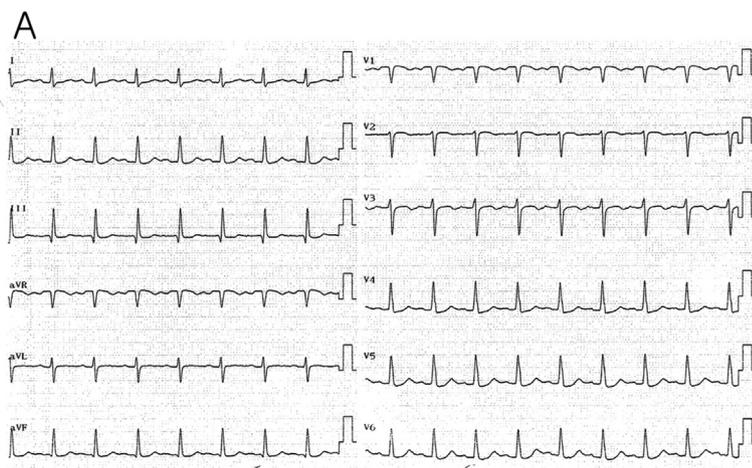


Figure 1 (A) Electrocardiogram showing sinus tachycardia with T-wave inversion on V₁₋₃ and mild ST-segment depression on V₄₋₆. (B) Chest roentgenogram shows cardiomegaly with mild pulmonary congestion (Figure 1B).

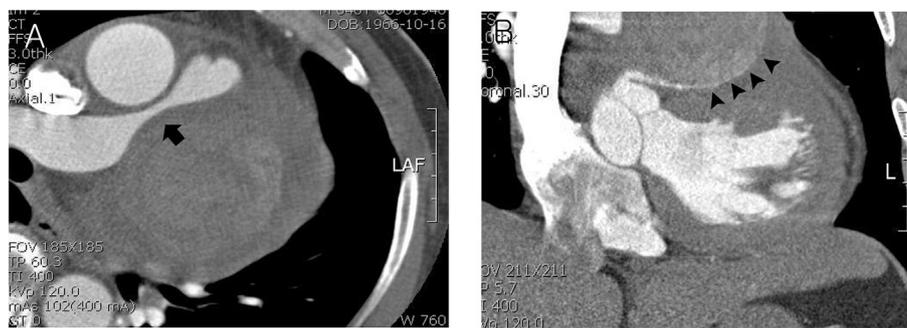


Figure 3 Cardiac computed tomography showing: (A) right ventricular outflow track compromised by aneurismal mass (arrow); and (B) linear and faint staining of the inferior wall of the aneurysm (arrowheads).



Figure 4 Left coronary angiogram revealed a very large (8 × 8 cm) round aneurysm (arrowheads) originating from the proximal segment of the LAD. LAD flow beyond the aneurysm was faint and it was not seen in static images.

likely induces alterations in blood flow and stasis of coronary blood flow, which may predispose patients to ischemic symptoms.¹¹ If the CAA is large enough, it might compress adjacent structures with associated symptoms,³ as exemplified by a reported case of a giant CAA causing superior vena cava syndrome and congestive heart failure through right atrial compression.¹² The patient in this report initially presented with ischemic symptoms, namely, chest pain and dyspnea, which may have been caused by the mass of aneurysm itself and LAD flow disturbance due to aneurysm.

The natural pathology of CAA is not well understood. The main complications are thrombosis and distal embolization, myocardial ischemia or infarction,⁹ calcification, dissection, vasospasm, fistulization¹³ and, very rarely, rupture.¹⁴ In an autopsy study by Daoud et al., thrombus was present in 70% of aneurysms.⁸ Although the exact incidence of distal embolization is unknown, aneurysm of more proximal segments is likely to have a more turbulent flow within CAA and thus is at a higher risk of thromboembolism in the adjoining coronary arteries and systemic circulation.¹⁵ The patient in this report suffered an ST-segment elevation MI and ventricular tachycardia leading to sudden death. Although the cause of these complications is unclear, they

might be related to thrombosis after CAG or, albeit rare and unpredictable, coronary aneurysmal rupture.

Because of the rarity of CAA, its management is not yet well established. Although simple observation may be sufficient for small, asymptomatic aneurysms, surgery should be considered for larger, symptomatic cases.¹⁶ Surgery is strongly recommended if the diameter of the CAA exceeds three to four times the original vessel diameter, even in asymptomatic patients, because of the increased likelihood of complications such as progressive enlargement, thrombosis and rupture.¹⁷ Other studies recommend surgical treatment for all aneurysms over 3 cm.¹⁸ Therefore, in the case reported here, where the patient had a very large aneurysm (8 × 8 cm) and ischemic chest pain, immediate surgical correction appeared to be a reasonable therapeutic strategy.

In conclusion, the present case of a very large aneurysm originating from the LAD and resulting in AMI, ventricular tachycardia and sudden cardiac death, illustrates that very large aneurysms warrant urgent surgical treatment, especially after studies using contrast agents, because of their risk of thrombosis and/or embolization.

Conflict of interest

There is no potential conflict of interest relevant to this article to report.

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