

Case Report

Giant Right Coronary Artery Aneurysm in an Adult Male Patient with Non-ST Myocardial Infarction

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Key words: Giant coronary aneurysm, diagnosis, etiology, treatment, complications.

The combination of a giant coronary aneurysm with multiple coronary aneurysms in adults is an extremely rare entity—especially in atherosclerotic patients, since it is most commonly associated with Kawasaki disease in children. We report an interesting case of a 59-year-old male patient with multiple atherosclerotic aneurysms of the left coronary system and a giant aneurysm of the right coronary artery. The patient was admitted to our hospital because of a non-ST myocardial infarction. Diagnosis was established by echocardiography, computed tomography angiogram, and coronary angiography. In view of the clinical symptoms and the extent of the giant right coronary aneurysm, with the associated risk of rupture, the patient was successfully treated with urgent surgical intervention.

We also present a review of the current literature on this anomaly and a statistical analysis of all atherosclerotic giant coronary artery aneurysms previously reported.

Manuscript received:
September 29, 2010;
Accepted:
January 15, 2011.

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Coronary artery aneurysm (CAA) is defined as a coronary dilation that exceeds the diameter of normal adjacent segments or 1.5 times the diameter of the patient's largest coronary vessel.¹ On rare occasions, a CAA grows large enough to be called giant. Although a precise definition of the term "giant" CAA is still lacking,² it generally refers to a dilatation that exceeds the reference vessel diameter by four times, or has a diameter exceeding 8 mm.³ Giant CAAs are usually associated with Kawasaki's disease in infants and children.⁴

CAAs are rare, with an estimated incidence of 0.3-5% among patients undergoing coronary angiography.¹ Giant CAAs are even less common in atherosclerotic cases, with an incidence of 0.02%.¹ Moreover, the combination of a giant CAA with multiple fusiform CAAs of the remaining coronary arteries is even rarer.

We describe an interesting case of a 59-year-old patient who had an athero-

sclerotic giant CAA in the right coronary artery (RCA) combined with multiple CAAs in the left circumflex (LCX) and left anterior descending (LAD) coronary arteries. In addition, we present a statistical analysis of all atherosclerotic giant CAAs previously reported and we review the literature, focusing on the location, etiology, diagnosis, management and prognosis of giant CAAs.

Case presentation

A 59-year-old Caucasian male was referred to our hospital for a new onset of chest pain at rest radiating to the back and left arm, lasting for more than 20 minutes. His familial history was unremarkable; however, he had a medical history of an anterior wall myocardial infarction associated with all major risk factors (arterial hypertension, non insulin-dependent diabetes mellitus, family history of coronary artery disease, obesity and smoking). On

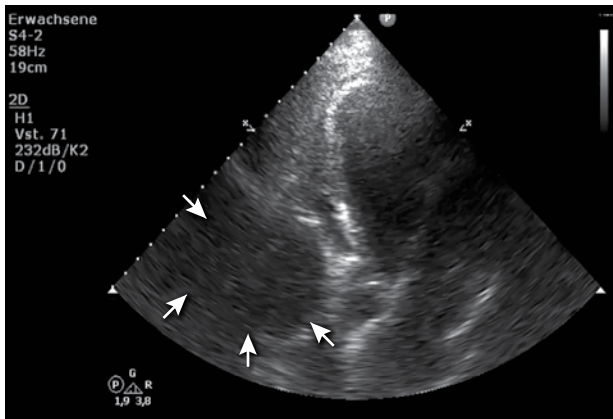


Figure 1. Apical four-chamber view shows a large (~50 mm diameter), smooth, echodense, cystic mobile mass close to the posterolateral wall of the right atrium.

admission he was hemodynamically stable. He had a blood pressure of 170/90 mmHg, without significant difference between the right and left arms, a heart rate of 90 /min, and normal peripheral pulse status. Further physical examination showed height 180 cm, body weight 89 kg, and temperature 36.6 °C, without any pathological findings or neurological deficits. The electrocardiogram was consistent with inferior ischemia (ST-depression in leads II, III, and aVF). Routine laboratory investigation showed positive troponin and maximal creatine kinase of 1697 U/l. The two-dimensional echocardiogram revealed a moderate reduction in systolic left ventricular (LV) function, apical wall akinesia, as well as inferior and posterolateral wall hypokinesia. Echocardiography also showed a large (~50 mm diameter) smooth cystic mobile mass

close to the posterolateral wall of the right atrium (Figure 1). Computed tomography (CT) angiography revealed a giant aneurysm of the proximal RCA (~60 mm in diameter) associated with a partly thrombotic aneurysmatic periphery (Figure 2A,B). According to ventriculography, systolic LV function was moderately reduced, with a calculated ejection fraction of 40% due to the inferior wall akinesia and the aneurysm of the anterior wall. Coronary angiography confirmed the findings of the CT angiography, with an aneurysmatic dilatation of the proximal LAD artery until the mid-segment (second diagonal branch), aneurysmatic dilatation of the LCX until the first marginal branch (Figure 3A), and a giant CAA in the proximal segment of the RCA (Figure 3B). The proximal RCA opened into a large (60 mm diameter) spherical cavity that filled with contrast medium in a swirling fashion with slow opacification, without a shunt or fistula.

In view of these findings, in combination with the clinical symptoms, the patient was treated with urgent surgery. Under conditions of general anesthesia initially, the left sided radial artery was harvested in skeletonized fashion and a segment of the right sided great saphenous vein was harvested in typical manner. After median sternotomy the left internal thoracic artery was prepared in a typical fashion, also using the skeletonized technique. After incision of the pericardium, the giant CAA of the RCA was easily identified (Figure 4A). Extracorporeal circulation was initiated via cannulation of the ascending aorta, and the superior and inferior *vena cava*. Cardiac ischemia was induced by ante- and retrograde infusion of cold blood cardioplegia. First, the segments

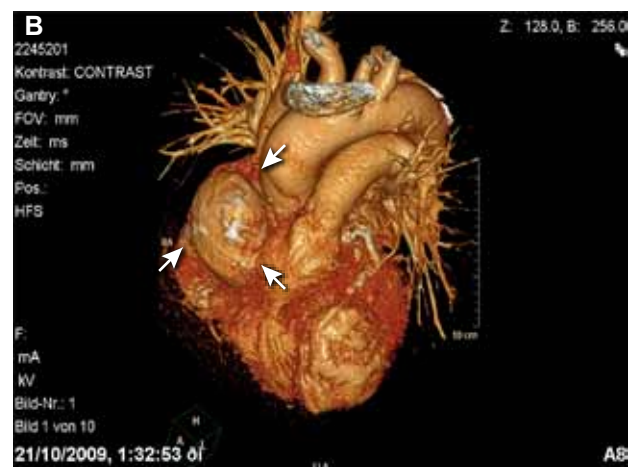
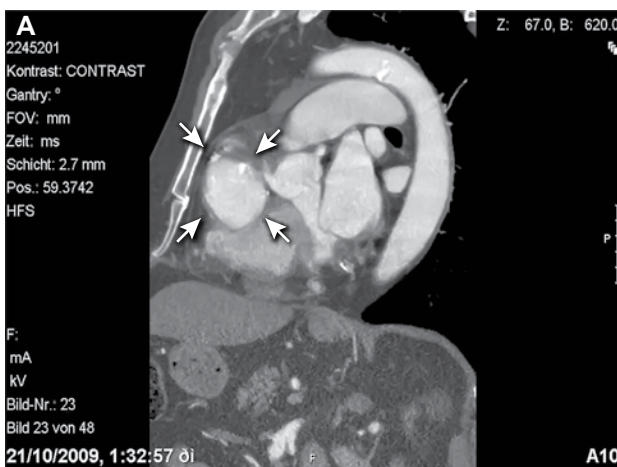


Figure 2. A. Computed tomography (CT) angiogram before surgery revealed a giant coronary aneurysm (CAA) 60 mm in diameter. B. CT three-dimensional image reconstruction showing the giant CAA originating from the right coronary artery.

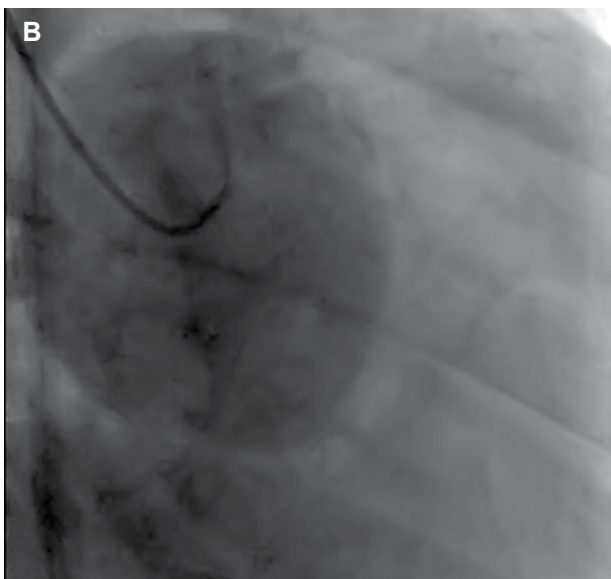
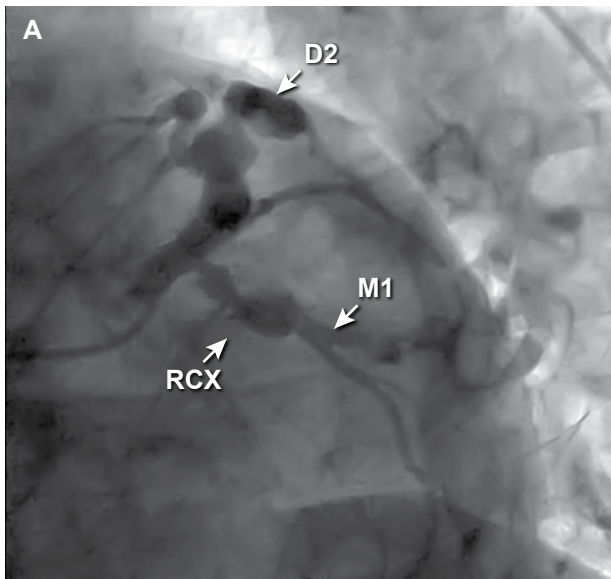


Figure 3. A. Left coronary angiogram in right anterior oblique and caudal view reveals an aneurysmatic dilatation of the proximal left anterior descending artery until the middle segment, and an aneurysmatic dilatation of the proximal circumflex artery until the first marginal branch. B. Right coronary angiogram in left anterior oblique view shows the proximal right coronary artery opening into a giant spherical aneurysmatic cavity.

of the giant CAA were incised in a stepwise fashion and all intraluminal thrombus and atheroma were removed. The ostium of the RCA (Figure 4B), as well as the outflow to the adjacent normally dimensioned segments of the RCA, were identified and occluded by sutures. The redundant wall of the CAA was resected and closed. The LV was opened via the aneurysm's anterior wall and the margins of the LV

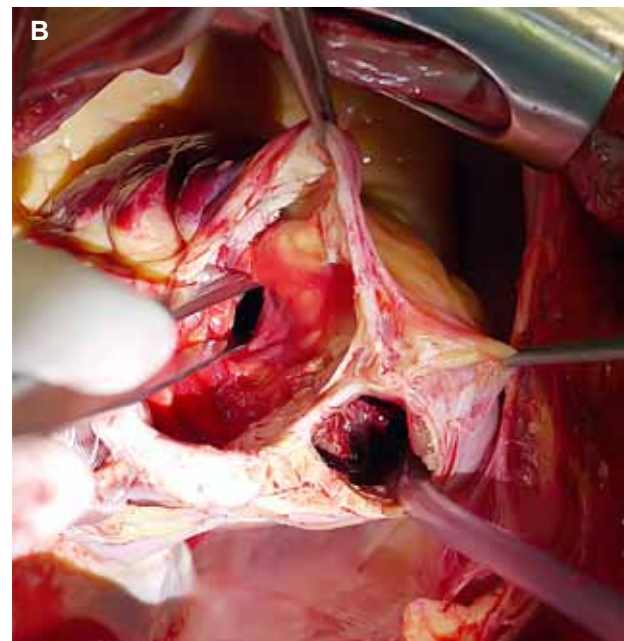
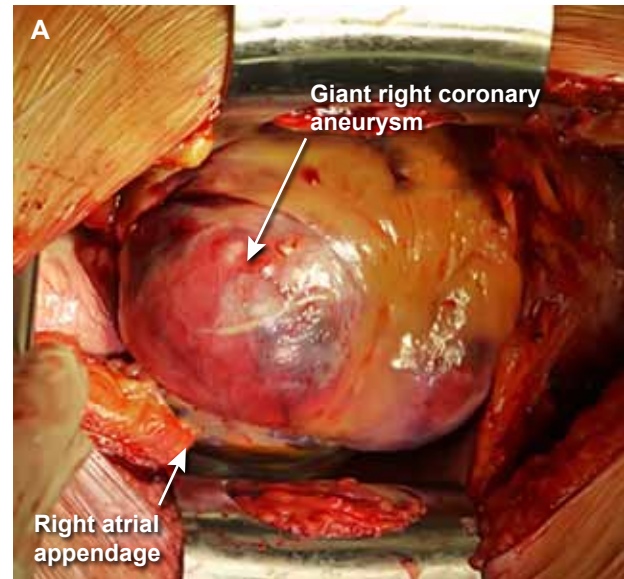


Figure 4. A. Surgeon's view of intraoperative findings after incision of the pericardium. B. Anatomy of the coronary aneurysm with forceps indicating the ostium of the right coronary artery and the suction tip inside the second aneurysmatic segment.

aneurysm were defined. LV geometry was restored with a Dacron patch using Dor's endoventriculoplasty. Revascularization of the coronary arteries was then performed with a single saphenous vein graft to the right posterior descending artery. A sequential radial artery graft was used for bypass of the obtuse marginal branch of the LCX and the right posterolateral branch. The left internal mammary artery was used as a bypass graft for the LAD. Saphenous vein

and radial artery grafts were implanted into the ascending aorta.

The patient was successfully weaned from cardiopulmonary bypass support with a low dose of catecholamines and implantation of an intra-aortic balloon pump. His further course in the intensive care unit was prolonged due to the development of a heparin-associated thrombocytopenia and a respiratory infection that required tracheostomy. After a complete and subsequently uneventful recovery the patient was discharged to a rehabilitation facility in a good state of health. He has been seen several times in our clinic since then and continues to do well, without any clinical symptoms and with normal exercise capacity.

Discussion

The first pathologic description of a coronary artery aneurysm (CAA) was reported in 1761,⁵ while the first clinical case was reported in 1812.⁶ CAA is defined as a localized dilatation when it exceeds the diameter of an adjacent normal segment by 50%. According to their morphology, CAAs can be classified as fusiform or saccular.⁷ They are termed giant if their diameter exceeds the reference vessel diameter by more than four times or if they are more than 8 mm in diameter.³ However, a precise definition of giant CAAs has not yet been established. In fact, the reported diameter of giant CAAs in adults varies from 50-150 mm and only a few cases have been described in the literature.¹

Incidence – location

CAA occurs in 1.5-5% of patient undergoing coronary angiography.^{1,8} Giant CAAs are even rarer, with an incidence of 0.02%.¹ CAAs are more common in men and in most cases affect only a single coronary artery.¹ The majority of giant CAAs involve the RCA adjacent to the right atrium.⁹ Previous reports have shown that CAAs occur at a rate of 68% in the RCA, 60% in the LAD, 50% in the LCX, and 0.1% in the left main artery,^{10,11} indicating that the adjacent area of atrial tissue appears to be a weak point for the formation and enlargement of a CAA.¹² Our analysis of the giant CAAs of atherosclerotic origin that have been reported in the literature (PubMed) showed a location rate of 52.2% in the RCA, 19.4% in the LAD, 7.4% in the LCX, 13.4% in the left main, and 4.5% in both the RCA and LAD (Figure 5A). Inter-

estingly, according to our analysis, the combination of an atherosclerotic giant CAA in the RCA with CAAs in the LCX and LAD coronary arteries is extremely rare, with an incidence of 6.6% (Figure 5B).

Etiology

Giant CAAs can be acquired (atherosclerotic and non-atherosclerotic) or congenital in origin.¹² Congenital CAAs are usually large and occur in young patients (17%).¹² A possible histopathologic connection may exist between giant CAAs and congenital coronary artery fistulas (CAF), since the former occur in approximately 6% of patients with CAF.¹³ Moreover, congenital CAA may predispose to secondary atherosclerotic changes, which in turn would increase the risk of aneurismal dilatation.¹⁴

Vasculitis can cause giant CAAs without atherosclerosis. This is most obvious in Kawasaki disease,

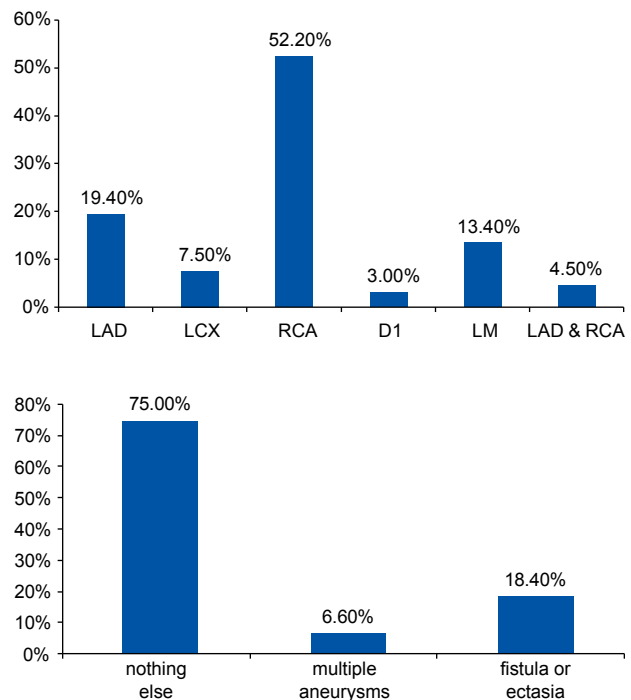


Figure 5. A. Analysis of the distribution of the origin of atherosclerotic giant coronary artery aneurysms (CAAs) in published cases in PubMed. The majority of giant CAAs were detected in the right coronary artery. B. The vast majority of patients with atherosclerotic giant CAAs did not have any other aneurysm/fistula/ectasia in other coronary vessels. Interestingly, our analysis showed that the combination of a giant CAA with multiple CAAs in other coronary arteries occurs only in 6.6% of the cases reported in PubMed. LAD – left anterior descending; LCX – left circumflex; RCA – right coronary artery; D1 – first diagonal branch; LM – left main.

where it has been shown that the incidence of giant CAAs was 4-5%,⁴ while in Japan and China it seems to be the most significant causative factor.¹⁵⁻¹⁷ Pathological examinations have revealed that vasculitis causes hyaline degeneration and thickening of the intima due to fibrocellular proliferation, inflammatory obliterative endarteritis of the *vasa vasorum*, disappearance of the *elastica interna*, fibrosis and calcification within the muscular wall, weakening and thus predisposing the arterial wall to aneurysm formation.¹⁸

Connective tissue disorders such as systemic lupus erythematosus (SLE) can also be a cause of non atherosclerotic giant CAAs. In SLE patients the incidence of CAAs is 0.15-4.9% of the cases undergoing coronary angiography, while the incidence of giant CAA is even rarer, since only two cases have been reported in the literature.¹⁹ Interestingly, in SLE the CAAs may develop during the long inactive stage, emphasizing the need for screening of coronary lesions in the management of such patients.²⁰ Other non-atherosclerotic causative factors of giant CAA can be infections (such as narcotic emboli, syphilis, Lyme disease and Epstein-Barr virus infection), trauma, drug abuse (such as cocaine), and iatrogenic.¹

However, in adults the most common cause of giant CAA is atherosclerosis, accounting for 50% of the reported cases in Europe and North America.¹ According to our analysis, the majority of cases occur between the ages of 60 and 80 years (31.6% of the patients were aged 60-70 years, 36.8% 70-80 years, and the rest <60 years). Interestingly, CAA formation has been associated with spontaneous coronary artery dissection, especially in patients with familial hypercholesterolemia.²¹ In case of atherosclerotic CAAs, histological examinations have revealed hyalinization and lipid deposition of the intima, intramural hemorrhage, and inflammatory reactions consistent with arteriosclerosis. The atherosclerotic process affects both the endothelium, forming luminal stenoses or occlusions, and the media and adventitia, resulting in arterial remodeling, dilatation and thus CAA formation.^{22,23} These findings suggest that aneurysmal coronary disease is not a distinct entity but a variant of coronary atherosclerosis, although CAAs are considered to be a systemic vascular wall abnormality.²³ With regard to the etiology in our case, the relatively advanced age of our patient, his multiple risk factors, and the histopathological examination suggest atherosclerosis as the most probable cause of the CAA.

Clinical presentation – complications

Clinically, the majority of CAAs are asymptomatic; however, up to one third of them may present with angina, myocardial infarction, sudden cardiac death and congestive heart failure. The most important predictor of myocardial infarction is the aneurysmal size.^{4,24,25} A patient with a giant CAA in the LAD has been described who presented with recurrent profuse hemoptysis most probably due to compression of the bronchial tree.²⁶ Recently, a rare case was described of a combination of giant CAA and pulmonary artery fistula associated with progressive atherosclerosis in a patient who had chronic renal disease and was under hemodialysis.²⁷ In addition, some reports associated coronary artery fistula with giant CAAs, while others correlated them with ectasias or other aneurysms.²⁸ Interestingly, the above mentioned correlations may be age-related, since our analysis revealed that older patients were more likely to have a combination of a giant CAA with fistula, ectasia, or multiple aneurysms: 45.2% of patients were aged 60-70 years and 39.9% 70-80 years, compared to only 14.9% aged less than 60 years ($p=0.096$).

In general, CAAs are small, thick-walled structures that have a relative low risk of rupture but are usually associated with myocardial ischemia.²⁹ However, giant CAAs may present as a mediastinal, intracardiac mass, or as superior *vena cava* syndrome in addition to ischemic symptoms, and are associated with a high risk of rupture.³⁰ The risk of rupture increases with age (especially >40 years), angina, congestive heart failure, infectious endocarditis or distal embolization.³¹ Furthermore, giant CAAs may mimic extracardiac pathologies (such as aneurysm of the ascending aorta or pulmonary trunk, cardiac tumor, pericardial tumor, thymoma or mediastinal masses).³² In general, large CAAs may fill with mural thrombus, creating diagnostic problems.

Diagnosis

Detection of giant CAAs can be achieved by noninvasive and invasive methods, such as echocardiography, CT, magnetic resonance imaging (MRI), and coronary angiography. Cardiac catheterization remains the gold standard tool, providing information about the size, shape, location, and coexisting anomalies such as coronary artery disease, and is also helpful for planning the strategy of surgical resection.³³ Limitations of the technique are that it is invasive, expensive

and that the true size of the CAAs may be underestimated if they contain substantial thrombus.³⁴

CT angiography is a noninvasive, fast, and relatively cheap technique for the diagnosis of CAAs that is available in most centers. Cardiac CT enables high quality 2- and 3-D reconstructions that are valuable in the delineation of the topographical anatomy of CAA by displaying the spatial relationship of the aneurysms, large vessels and the heart. Three-phase CT arteriography has also been reported as helpful in confirming large CAAs. This technique shows homogeneous and similar densities of a mass and cardiac chambers in the unenhanced and equilibrium phases, and turbulent enhancement in the arterial phase.³⁵ In addition, CT provides information regarding the extent of luminal blood flow. Also, CT appears particularly useful during follow-up imaging after CAA exclusion with polytetrafluoroethylene-covered stents.³⁶ Thus, CT is a noninvasive alternative to catheter coronary angiography in the diagnosis and long-term follow up of patients with giant CAAs. However, it may have limitations in delineating the distal part of the coronary arteries, in demonstrating clots or thrombus inside the vessels, and in simulating a large CAA as an inhomogeneous mass because of the blood turbulence within it.²⁹

MRI is an alternative noninvasive cross-sectional technique for the diagnosis and evaluation of CAAs, obviating the large radiation dose associated with CT.³⁷ However, MRI is not available in all medical centers, has inferior spatial resolution compared to CT, and does not show the typical linear peripheral calcifications of the CAA, which are important for the correct diagnosis. Finally, intravascular ultrasound provides detailed, high-quality images that can be valuable in distinguishing CAA from coronary artery ectasia, as well as true CAAs from pseudoaneurysms.³⁸ However, even with the above mentioned powerful imaging techniques, in certain cases the differentiate diagnosis of a giant CAA from a cardiac mass can not easily be made until surgery.³⁹

Prognosis - treatment

The natural history and prognosis of giant CAAs remain obscure; however, the overall 5-year survival is reported to be 71%.^{7,33} Given their rarity and the non-availability of controlled trials, there is no optimal management strategy for patients with a giant CAA. Depending on the symptoms, etiology and associated lesions, medical treatment, stent implanta-

tion, or surgical exclusion of the CAA using a resection or ligation technique have been described.^{1,40} It is unknown whether size alone is an indication for surgical intervention. However, for small aneurysms with no symptoms, conservative treatment consisting of anticoagulant and antiplatelet drugs aims mainly to prevent thromboembolic complications. It is unclear whether this treatment influences the prognosis among patients with CAAs in relation to patients with coronary atherosclerosis. However, there is one reported case of a giant CAA that was treated successfully with aggressive medical management, without symptoms or further cardiac events and complications.⁴¹ Thus, the combination of anticoagulant and antiplatelet drugs has acceptably high cardiac-event-free survival in patients with giant CAA, though it has a certain risk of hemorrhagic complications.

However, in case of giant CAAs, especially when symptoms are present, data suggest surgery as the preferred treatment in order to achieve complete exclusion and resection of the aneurysmal sac, and also to manage associated conditions.⁴¹ Various surgical strategies have been adopted, such as reconstruction, resection, and isolation with concomitant coronary artery bypass. In most cases, surgical management requires a median sternotomy, the use of extracorporeal circulation, and coronary artery bypass. In cases of giant CAAs combined with fistula, closure of the fistula is also mandatory.⁴²

Recent data support the notion that percutaneous intervention may be an alternative to surgical treatment, especially in patients with high predicted perioperative risk.²⁸ Various percutaneous methods have been used, depending upon the underlying anatomical properties of the aneurysm and coronary circulation.⁴³ Recently, a case of giant mycotic CAA following infective endocarditis was successfully treated with intravenous antibiotics and a covered stent, highlighting a new treatment modality in critically ill patients.⁴⁴

Conclusions

Giant CAA is a rare entity in atherosclerotic patients, especially when it is associated with multiple CAAs. Moreover, its clinical presentation varies and is often combined with other cardiac anomalies. Therefore, giant CAA should be considered in the differential diagnosis of other conditions mimicking similar symptoms. This rare anomaly and the associated lesions require the proper use of imaging and invasive

technology for exact diagnosis and successful management. Surgical management needs to be planned carefully and requires appropriate techniques for a better outcome with fewer complications. The use of an interventional approach can be a good alternative, especially in patients who are at high surgical risk.

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