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Case Reports

Giant Aneurysm of Left Main Coronary Artery in a Patient With Prior Operation for 4-Valve Endocarditis

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Abstract

Left main coronary artery aneurysm is an unusual complication of infective endocarditis. Although this type of aneurysm is often asymptomatic, rupture and thrombus formation that result in myocardial infarction are known complications; therefore, prompt recognition and surgical intervention are warranted. This report describes a patient who presented with a giant left main coronary artery aneurysm 3.5 years after being treated for 4-valve endocarditis. The management and technical aspects of this challenging case are discussed here.

Keywords: Mycotic aneurysm; endocarditis; coronary aneurysm

Introduction

Aneurysms of the left main coronary artery (LMCA) account for approximately 0.1% of patients who undergo routine angiography in large angiographic series.[1] The most common cause is atherosclerosis; other etiologies include congenital malformations, Kawasaki disease, trauma, dissection, cocaine use, complications of instrumentation, vasculitis, syphilis, and bacterial and fungal infections. Confusion has surrounded the term mycotic since Osler[2] used it to describe a mushroom-shaped infected aneurysm in a patient with bacterial endocarditis. The term infected aneurysm will be used here to avoid further confusion. Coronary aneurysms are an unusual but known complication of infective endocarditis, occurring in less than 0.5% of all infective endocarditis cases.[3] The gold standard for diagnosis is coronary angiography, although the use of computed tomography (CT) with cardiac gating and angiography is increasing. Putative mechanisms for aneurysm formation in the setting of active infection include embolic occlusion of coronary vasa vasorum, direct bacterial invasion of the arterial wall, and injury from deposition of immune complexes in the arterial wall.[4] Coronary artery aneurysm (CAA) in the setting of infective endocarditis was first reported in 1812 by Bougon,[5] who made the diagnosis post mortem after spontaneous rupture. Crook and colleagues[6] reported the first antemortem diagnosis in 1973. Since then, there have been fewer than 50 reports of infected CAAs in the literature, of which 6 involved the LMCA, including 3 patients who survived.[7] This report describes a fourth.

Case Report

In November 2012, a 51-year-old man with a known history of untreated perimembranous ventricular septal defect was admitted to the hospital with acute, decompensated, diastolic, congestive heart failure; acute kidney injury; liver failure; and sepsis. His blood cultures were positive for *Enterococcus faecalis*, and an echocardiogram demonstrated vegetations involving all 4 cardiac valves with preserved biventricular function. After optimization on the medical service, he was brought to the operating room, where he underwent debridement of all infected and nonviable tissue, replacement of the pulmonic valve with a bioprosthesis, replacement of the mitral and aortic valves with mechanical prostheses, repair of the tricuspid valve with an annuloplasty ring, patch closure of the ventricular septal defect, and resection of an infected aneurysm of the noncoronary sinus with bovine pericardial reconstruction. An anterior pericardiectomy was also performed for chronic inflammatory pericarditis. There was no gross evidence of coronary disease. Although he required hemodialysis for the first postoperative week, his kidney function eventually recovered,

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and he was discharged on intravenous antibiotics that were continued for 2 months.

The patient returned to work and was asymptomatic until March 2016, when he presented with acute-onset chest pain. Elevated cardiac markers prompted a coronary angiogram, which demonstrated a 3-cm aneurysm of the LMCA (Fig. 1), with a Thrombolysis in Myocardial Infarction score 2 flow down the left circumflex and left anterior descending arteries. The right coronary artery was unremarkable, with a Thrombolysis in Myocardial Infarction score 3 flow.

Cardiac CT imaging with contrast demonstrated enlargement of the orifice of the LMCA (6.3 mm) and a 3-cm aneurysm involving the distal LMCA (Fig. 2A). The aneurysm contained mural thrombus with a wall thickness of 5 mm. A repeat echocardiogram showed preserved left ventricular ejection fraction of 50% with no evidence of endocarditis. Blood cultures showed no growth, and the international normalized ratio was 2.5. A heparin infusion was started in the setting of 2 mechanical valves, mural thrombus in the aneurysm, and distal myocardial ischemia. The patient's case was presented at a multidisciplinary conference, where surgical intervention was the consensus for treatment. To assist with planning, 3-dimensional (3D) reconstruction was performed using CT multiplanar reconstruction, followed by a 3D print of the anatomy (Fig. 2B and 2C).

Through a redo median sternotomy, the patient was placed on cardiopulmonary bypass with central cannulation after the left internal mammary and a segment of greater saphenous vein were procured. Under moderate hypothermia (28 °C), the main pulmonary artery was dissected free from the aorta and transected at its bifurcation, allowing for exposure of the LMCA and

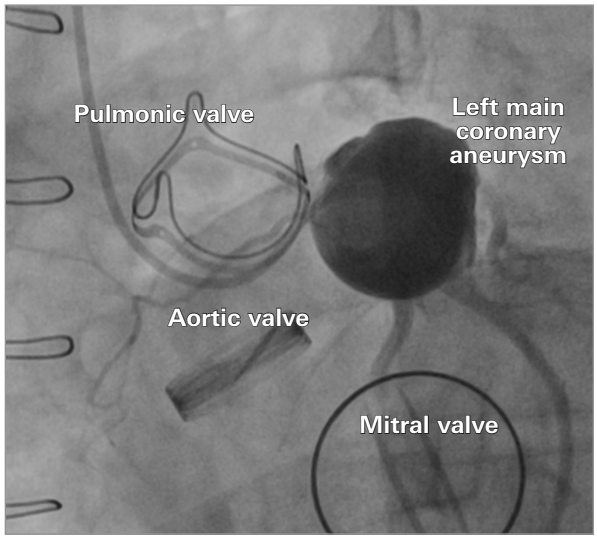


Fig. 1 Coronary angiogram image demonstrates a large left main coronary artery aneurysm along with mechanical aortic and mitral valves and a bioprosthetic pulmonary valve.

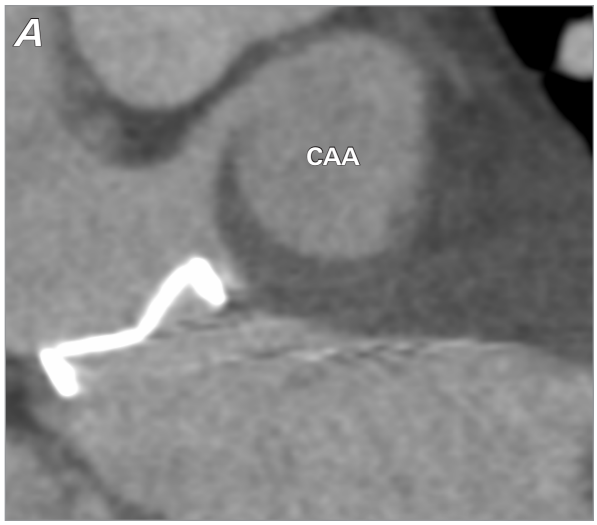
Abbreviations and Acronyms

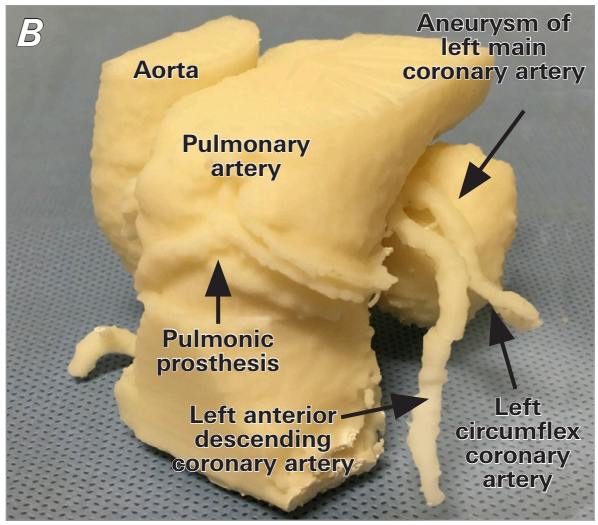
3D	3-dimensional
CAA	coronary artery aneurysm
LAD	left anterior descending artery
LMCA	left main coronary artery

the aneurysm. The heart was arrested using retrograde cardioplegia and topical ice slurry. The anterior wall of the LMCA aneurysm was incised, and the thrombus was evacuated from the aneurysm sac. It was difficult to visualize the orifices of the left anterior descending and left circumflex coronary arteries because the ostia were compressed by the thick aneurysm wall, and the vessels were stretched apart from each other. A 1.5-mm coronary artery probe was useful in identifying the ostia and marking the course of the coronaries and the site for graft anastomoses. A saphenous vein graft was anastomosed to the left circumflex artery in the atrioventricular groove, and the left internal mammary artery was grafted to the mid–left anterior descending coronary artery. An additional vein graft to the diagonal coronary artery was also placed, in the event that the left internal mammary artery conduit developed vasospasm postoperatively. After the 3 distal anastomoses were completed, the orifices of the left main, left anterior descending, and left circumflex arteries were oversewn at the junction of the aneurysmal sac, excluding the aneurysm from coronary circulation. The proximal anastomoses for the vein grafts were then performed, and after deairing maneuvers, the aortic cross clamp was removed. Then, while still on cardiopulmonary bypass, the main pulmonary artery continuity was reconstructed by using a bovine pericardial patch to minimize tension on the repair. After weaning from cardiopulmonary bypass, intraoperative echocardiography confirmed good myocardial and valvular function. Anticoagulation was resumed postoperatively. Eight days later, the patient was discharged home on warfarin and aspirin. He made an uneventful recovery, and on last follow-up in January 2021, he was back at work and symptom free.

Discussion

Although the mechanism and evolution of this patient's disease process are speculative, the original infection in 2012 likely contributed to inflammation that extended to the LMCA, resulting in gradual aneurysmal expansion in the subsequent 40 months before his second presentation. Because the patient had no atherosclerotic coronary artery disease, the chest pain of his presentation was likely the result of compromised coronary blood flow caused by compression from the aneurysm or distal embolization of clot. It is interesting, although





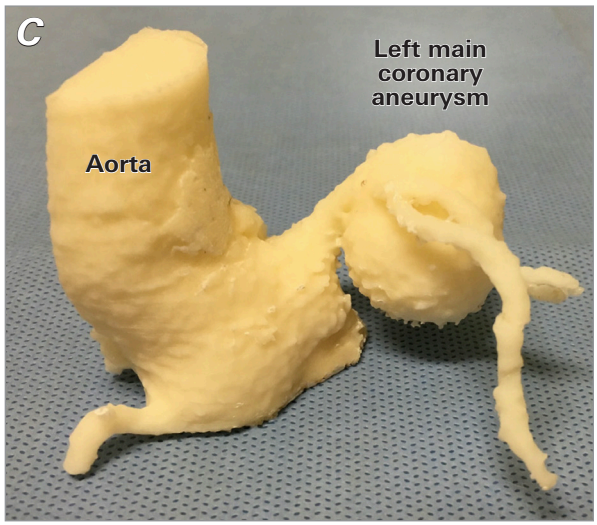


Fig. 2 A) Computed tomography image shows the left main CAA and ascending aorta. **B**) Three-dimensional print of the anatomy shows the relationship between the pulmonary artery, the pulmonic valve, the aorta, and the coronary aneurysm. **C**) The pulmonary artery and pulmonic valve are removed to expose the aorta and aneurysm.

CAA, coronary artery aneurysm.

likely coincidental, that there has been only 1 other reported case of CAA resulting from *E faecalis* infection and that the LMCA was also the implicated vessel in that case.[8]

Because the natural history of CAAs is largely unknown and randomized trials comparing treatment strategies are lacking, the current indications for intervention and management recommendations are based on small case series and anecdotal evidence. Although some small, asymptomatic CAAs have been successfully managed medically,[9] other studies have shown a higher incidence of complications with medical management.[10] The role of antiplatelet or anticoagulation therapies for incidentally discovered coronary aneurysms remains a matter of debate; some studies support the practice,[11] whereas others suggest it may not be necessary.[12] Percutaneous options for coronary aneurysms include stenting and coil embolization, although outcome data for these strategies are also limited. The majority of published reports involve patients presenting with acute cardiac events and document higher procedural complication rates than with percutaneous coronary interventions on nonaneurysmal vessels, including stent thrombosis, distal embolization, and myocardial infarction, during intermediate-term follow-up.[13] Recommendations generally include thrombectomy with possible intracoronary thrombolytics.[9] The indication for percutaneous therapies in the absence of symptoms is somewhat murky because there are even fewer published outcome data. Saccular aneurysms and small pseudoaneurysms can be treated effectively with stent exclusion. For giant aneurysms (defined variously in the literature with the lowest threshold being >20 mm) and those involving the LMCA, surgery is generally considered the first-line therapy.[9]

Aneurysms that result as complications of infective endocarditis are more prone to rupture than are those resulting from atherosclerotic disease, and thrombosis related to stasis of blood flow can occur despite adequate systemic anticoagulation, as occurred in the patient in this case.[14] Although previously reported,[14] the role of catheter-based intervention in the management of giant CAAs can be complicated by its potential to dislodge thrombi and cause myocardial damage. The placement of a custom covered stent in the LMCA and inside a potentially infected aneurysm runs the risk of infecting the stent and would present challenges to the preservation of flow to the displaced and compressed left anterior

descending and left circumflex coronary arteries. Therefore, percutaneous approaches in this setting are best reserved for patients who are deemed to be poor operative candidates. The best-proven treatment of giant CAAs regardless of etiology is surgical exploration, incision for evacuation of hematoma or drainage of infection, isolation of the aneurysm, and restoration of distal coronary flow with appropriate grafting. Exclusion of the CAA by ligation of inflow and outflow branches is necessary to control bleeding, prevent further thrombus embolization, and avoid competitive flow that can cause the grafts to fail. This operative approach is similar to the management of a previously described, similarly located giant CAA,[15] although it is believed this is the first to attempt this exposure with a prosthesis in the pulmonic position.

The surgical approach to CAAs is based on preoperative 3D imaging that defines the relationship of the other major structures. In this case, transection of the main pulmonary artery (despite the pulmonic prosthesis) provided direct exposure to the giant CAA. Previous pericardiectomy, extensive infection, and a mechanical prosthesis in the mitral position made it difficult to expose the distal coronary artery branches. Probing the left circumflex (Fig. 3) through its tiny, compressed orifice within the aneurysm allowed for localization and grafting of the left circumflex in the atrioventricular groove, avoiding the need to lift the heart—a maneuver that could be hazardous in the setting of a rigid mitral prosthesis. The initial reconstruction of the main pulmonary artery with end-to-end anastomosis resulted in significant bleeding related to friable, thin arterial wall under some tension in the redo setting where pulmonary artery branches could not be mobilized. A bovine pericardial patch to extend the posterior wall of the main pulmonary artery allowed for a tensionless repair with good hemostasis. In a patient with no prior cardiac surgery or infection, aortotomy can be a more direct approach to exclusion of a LMCA aneurysm. Aortotomy allows for patch closure of the LMCA origin.

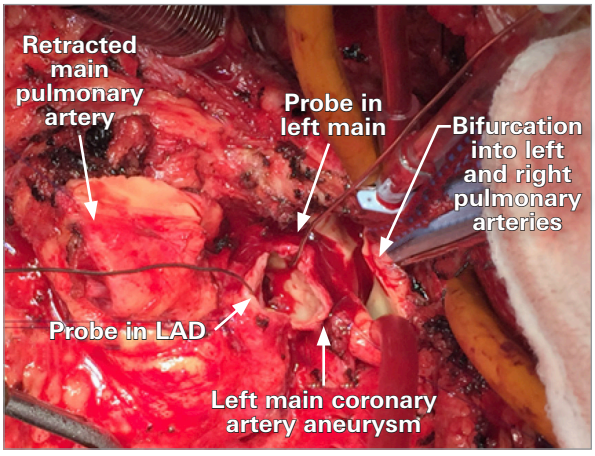


Fig. 3 Image shows the intraoperative anatomic landmarks.

LAD, left anterior descending artery.

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