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INCIDENTAL FINDING OF A GIANT CARDIAC MASS

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Abstract

Coronary artery fistula (CAF) is a rare anomalous connection between a coronary artery and another coronary artery, major vessel, or cardiac chamber. Prevalence of CAF is reportedly 1% to 2% in patients who undergo coronary angiography.[1] One of the most common complications of CAF is formation of a coronary artery aneurysm (CAA). A study conducted by Said and colleagues in 1995 found that CAA formation was present in 26% of patients who had proven CAF by way of angiography.[2] Although a precise definition of the term "giant" CAA is still lacking, it generally refers to a dilatation that exceeds the reference vessel diameter by four times.[3] We report an interesting case of a 38-year-old patient who was incidentally found to have a presumed large right ventricular aneurysm, which after an open-heart surgery was identified as a CAF with formation of an unruptured giant CAA.

Case Presentation

A 38-year-old Caucasian male with no past medical history presented to a local community hospital with a chief complaint of right-sided nonexertional flank pain that started abruptly. Pain was described as sharp, 8 out of 10 in severity, and radiating to the back. He denied shortness of breath, hematuria, or a history of nephrolithiasis. The patient had no past medical history, had never been hospitalized, and had no prior medication use. His family history was noncontributory. He was a former Navy Seal officer. The patient admitted to a tobacco addiction with a 15-pack/year history of smoking.

On physical exam his blood pressure was 125/80, heart rate was 75 beats per minute, temperature was 38.5°C, and he was oxygenating at 98% on room air. He appeared to be in no acute distress. Cardiac auscultation revealed a nondisplaced point of maximal impulse, regular rate and rhythm, and normal S1 and S2 without murmur, rub, or gallops. His jugular vein was not distended. Examination of his lungs and abdomen were normal. Significant costovertebral angle tenderness near the right flank was noted. Laboratory workup that included blood count, basic metabolic panel, and urinalysis revealed no abnormalities. Electrocardiogram showed normal sinus rhythm at 70 beats per minute, normal axis, and normal intervals with no ischemic changes. A computerized tomography (CT) scan of abdomen and pelvis revealed a millimeter-sized calcification in the left ureter. In addition, there was an incidental finding of focal bulging at the left ventricular apex with slightly narrowed neck (Figure 1).

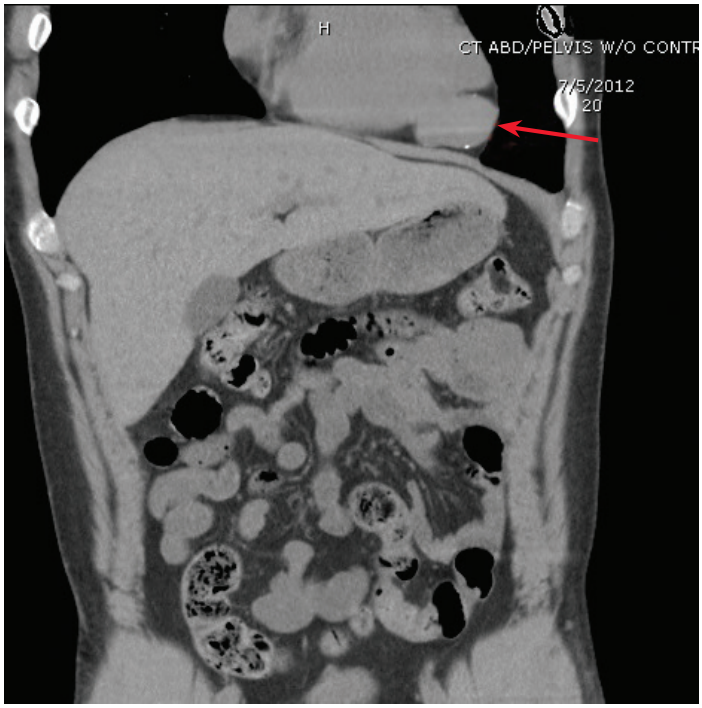
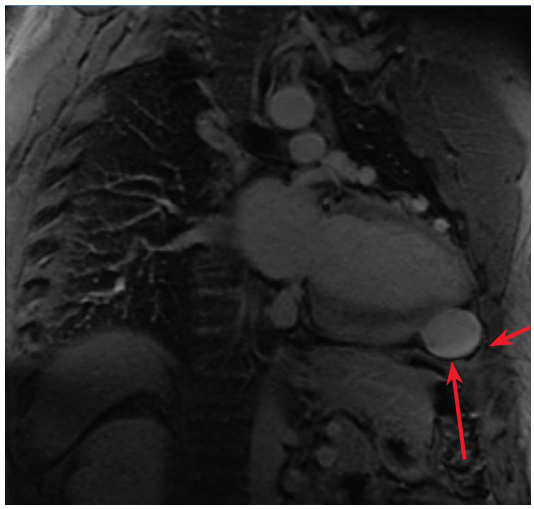


Figure 1. An incidental finding of focal bulging at the left ventricular apex with slightly narrowed neck.



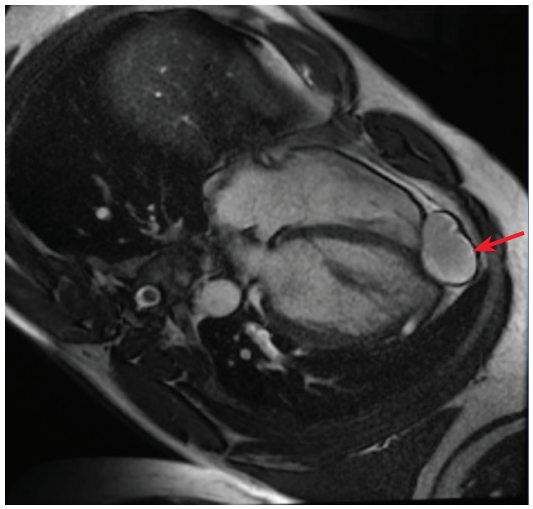
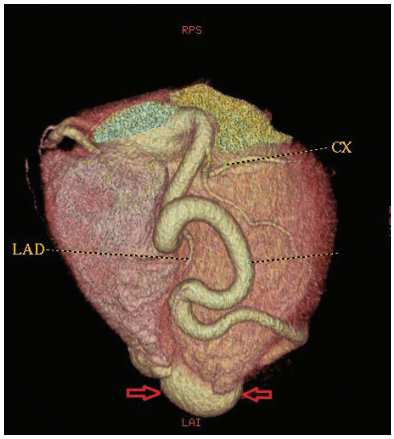
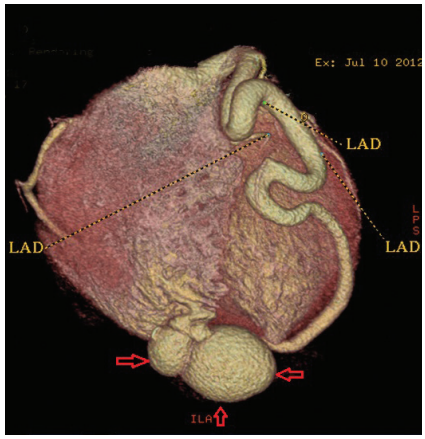


Figure 2. Cardiac magnetic resonance imaging coronal view reveals a 4.5 x 3.2 x 3.0 cm ovoid structure adjacent to the right ventricle. Cross-sectional transverse view shows a well-circumscribed bulging structure at the cardiac apex.





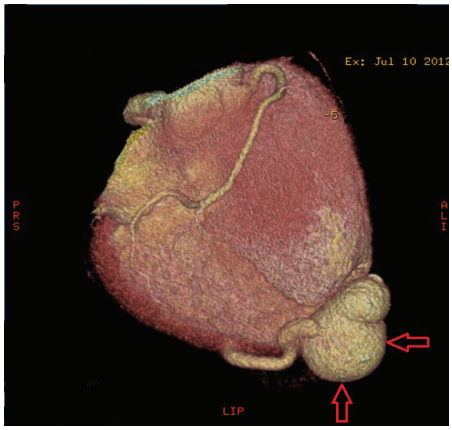


Figure 3. The left anterior descending artery is dilated as it travels down to the ventricular apex, where it become a saccular aneurysm.

In view of the latter finding, the patient was transferred to New York Presbyterian/Weill Cornell Medical Center for further medical management. The patient had follow-up with cardiac magnetic resonance imaging (MRI) to evaluate the ventricular mass (Figure 2), revealing an ovoid structure adjacent to the right ventricle with approximate dimensions of 4.5 x 3.2 x 3.0 cm. No evidence of myocardial scarring or inflammation was identified. The structure appeared to have a pulsatile mobility pattern, and fistulous communication with the ventricles could not be excluded

Suggested diagnostic considerations included right-sided ventricular aneurysm, right-sided ventricular pseudoaneurysm, coronary aneurysm, arteriovenous malformation, coronary artery fistula, and pericardial cyst. Subsequent follow-up with coronary CT angiography revealed proximal left anterior descending (LAD) coronary artery dilatation of 10 mm. The LAD remained dilated as it traveled down its predicted course to the ventricular apex, where it became a saccular aneurysm measuring 4.9 x 3.5 cm (Figure 3).

Workup for the atherosclerotic and inflammatory etiologies were considered to rule out any active disease process. Total cholesterol was 210, HDL was 50, LDL was 136, and antinuclear antibody, C-reactive protein, and erythrocyte sedimentation rate were all normal.

Treatment Course

The patient was referred to the Texas Heart Institute, where surgical intervention to repair the giant CAA was recommended. The patient underwent open-heart surgery via total cardiopulmonary bypass, which revealed a 4-cm by 3.5-cm thin-walled aneurysm located at the cardiac apex as expected based on presurgical imaging. Interestingly, it was noted that two vessels (the distal end of the left anterior descending artery and a distal branch of the right coronary artery) were in communication with each other, forming a CAF as well as the origin of the aneurysm. No communication was identified with either of the ventricular chambers. The CAF was closed and the entire aneurysm was excised. The patient had no perioperative or postoperative complications.

Surgical Specimen Pathology

The tissue was a gray-white to yellow-tan, rubbery, wrinkled fragment of fibromembranous soft tissue measuring 4.0 x 3.5 x 0.6 cm in aggregate. Microscopic findings were characterized by attenuated blood vessel wall with degenerative changes, fatty infiltration, and focal thin-wall calcification with degenerative changes (Figure 4).

Discussion

Most CAFs are congenital, but other causes may include atherosclerosis, focal inflammation, and trauma (e.g., cardiac catheterization). In patients older than 20 years of age with known CAF, there is up to a 35% chance of fistula-related complications.[4]

Clinically, the majority of CAFs are asymptomatic. The natural history of CAF is variable, with long periods of stability in some patients or complications with either sudden onset or gradual progression in others. The most important clinical predictor of myocardial infarction is associated with the size of aneurysm.[5] Affected coronary arteries consist of fragile smooth muscle with constant exposure to blood flow at arterial pressures, which over time leads to atherosclerotic plaque deposition and aneurysmal changes. As an aneurysmal lesion progresses, the vessel loses width, elasticity, and strength.[6] With aneurysm enlargement, complications such as heart failure, myocardial infarction, opening (rupture) of the aneurysm/fistula, and coronary steal syndrome may ensue.[6]

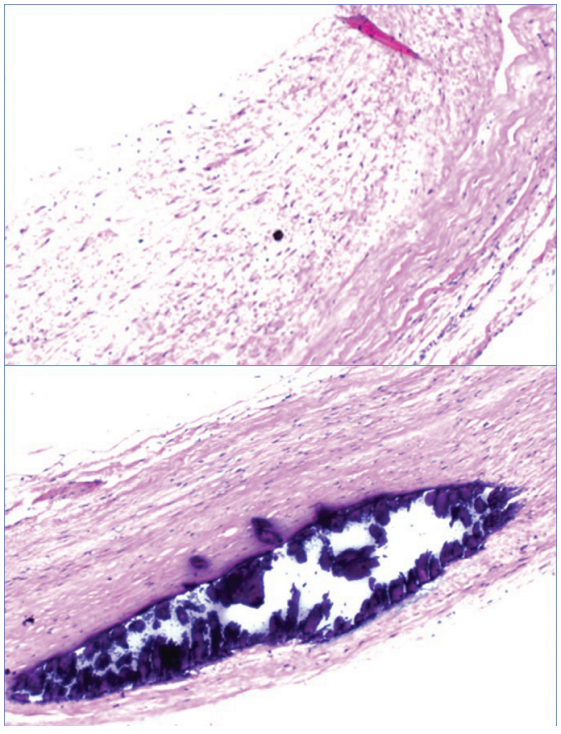


Figure 4. Microscopic findings show attenuated blood vessel wall with degenerative changes, fatty infiltration, and focal thin-wall calcification with degenerative changes

Given the rarity and unavailability of controlled trials, no evidence-based guidelines exist for the optimal management of giant coronary artery aneurysms. Lowe and colleagues recommend that all patients with coronary artery fistula be considered for surgical correction, especially in cases of giant aneurysms and progressive enlargement of CAF.[7]

Conclusion

This case report demonstrates a very rare and unique finding of coronary artery fistula complicated with giant coronary artery aneurysm formation in an otherwise young, healthy man. Coronary artery aneurysm may be found incidentally on imaging, and timely surgical referral and management may be crucial in preventing significant morbidity and mortality of the patient.

Conflict of Interest Disclosure: The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

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Keywords: incidental finding, giant coronary aneurysm, coronary artery fistula, healthy

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