Case Report

Acquired Left Coronary Artery Fistula Draining to the Right Ventricle after Myocardial Infarction

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Abstract

Coronary arteriography performed in a 42-year-old male patient showed communications from the left anterior descending artery to the right ventricle one month after a myocardial infarction. The area of communication did not correspond to the site of the infarction as established by electrocardiogram and previous angiography. Exactly how the fistula developed in our case remains unknown. Angiogenesis also suggests that myocardial infarction can lead to spontaneous formation of these coronary anomalies.

Keywords: Coronary artery fistula, myocardial infarction, right ventricle

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Introduction

Coronary artery fistula (CAF) is a seldomly encountered anomaly that involves abnormal communication between one of the coronary arteries and a cardiac chamber or a vessel lying adjacent to the heart. CAF may be congenital or acquired. Acquired CAF has been reported rarely. It may occur due to various reasons such as acute myocardial infarction, endomyocardial biopsy, coronary angioplasty, and thoracic trauma. Most coronary fistulas are asymptomatic. Symptoms depend on the size of the fistula and on the receiving chamber. In the majority of the cases, it is either the right ventricle (45%), the right atrium (25%), the pulmonary artery (15% – 20%), or – less frequently – the coronary sinus (7%) that constitutes the receiving chamber.[1,2] The anatomic location is generally hibernating or even ischemic myocardium. However, in our case, the fistula was not related to the culprit lesion, and thus, this is the first case report of left CAF draining to the right ventricle after myocardial infarction.

Case Report

The patient, a 42-year-old male, was presented to our hospital with typical chest pain that had started one hour before. The only known risk factor was smoking. An electrocardiogram showed ST elevation in inferior leads (II, III, aVF) and ST depression in V_{1-3} (Figure 1). Laboratory tests revealed a high level of creatine kinase muscle-brain fraction (CK-MB), amounting to 42 U/L (normal range, 0-24). Troponin (Tn)-I was normal (0.2 ng/mL; normal range, 0-1). Physical examination revealed a low blood pressure (90/60 mmHg) and there were no murmurs. Chest X-ray examination showed a cardiac silhouette at the upper limits of normal size, and there was no evidence of congestive heart failure. The patient was taken to the catheter laboratory in the first hour of his chest pain for early revascularization. The percutaneous transluminal coronary angioplasty and coronary stent implantation were made in the 100% thrombotic lesion between the circumflex (Cx) and obtuse marginal 2 (Cx-OM2) coronary arteries in the bifurcation region. The left anterior descending (LAD) artery was normal and no invasive procedure was performed (Figure 2). After the intracoronary stent replacement, ST elevation decreased and the patient's chest pain recovered. However, as the lesion at the Cx-OM2 bifurcation could not be fully opened due to intracoronary thrombus, intravenous tirofiban infusion was started. The patient was discharged after one week with optimal anti-ischemic and anti-coagulant treatment including warfarin. On the echocardiography one week after discharge, we established ejection fraction as 57%, with minimally hypokinetic mid inferior and posterobasal segments, 1-2/4 mitral regurgitation, and probable papillary muscle dysfunction. The patient was called for a follow-up coronary angiography. In his electrocardiogram, there were pathologic Q waves in inferior leads. Cardiac catheterization was performed. Injection of the left coronary artery showed no lesion in the main coronary artery. There was a 20% narrowing of the stent and a 60% narrowing in the proximal Cx-OM2. A 50% narrowing and hazy appearance were present at the proximal LAD, and a portion of the fistula was seen from the distal part of the LAD to the right ventricle (Figure 3). The right coronary artery was normal and dominant. One month later, a fractional flow reserve for assessment of the LAD was performed, and the lesion was detected as insignificant. The patient has been under follow-up at the polyclinic for two years without any problems.

Discussion

Coronary artery fistula (CAF) is a very rare anomaly. It was first described by Krause in 1865, and the first surgical treatment was done by Bjork and Crafoord in 1947. About 50% of the patients with CAF do not reveal any symptoms, and it is also possible for some of the CAFs to disappear on their own during childhood.[3] Many acquired fistulas have been described as a consequence of acute myocardial infarction or heart surgery or following trans-

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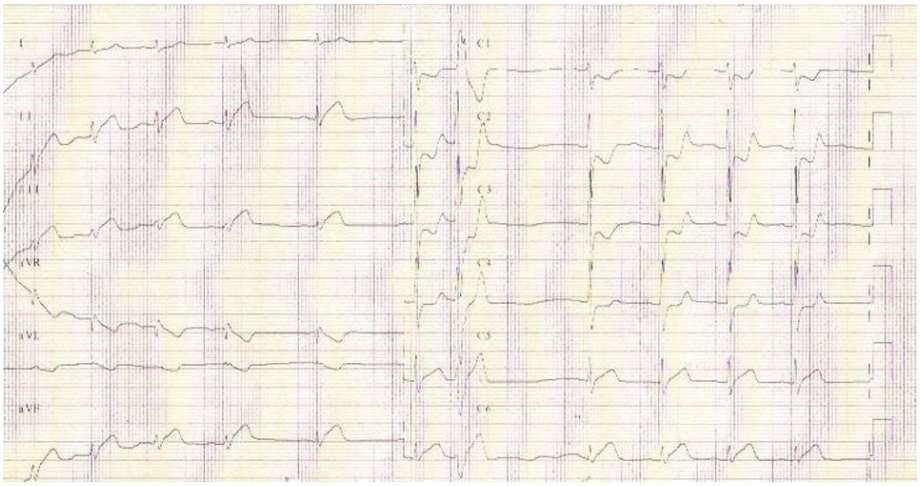

Figure 1. An electrocardiogram showed inferoposterior acute myocardial infarction with ST elevation in inferior leads (II, III, aVF) and ST depression in V_{1-3}.

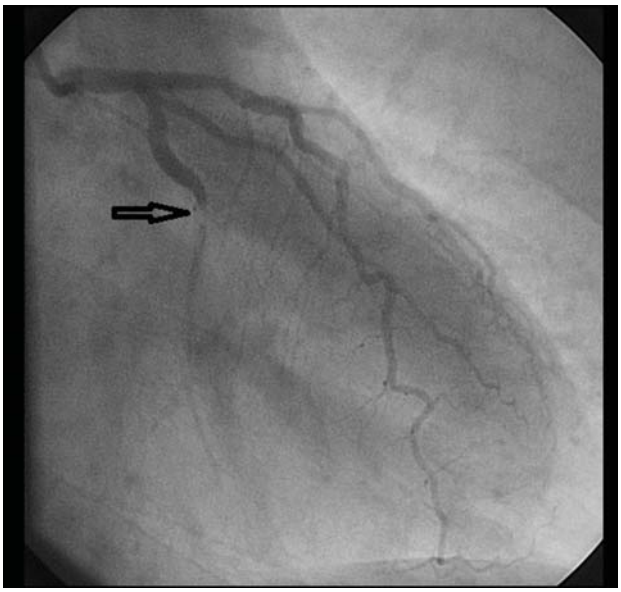

Figure 2. A total (100%) occlusion in the circumflex coronary artery (arrow) and normal left anterior descending artery are seen in the right anterior oblique section.

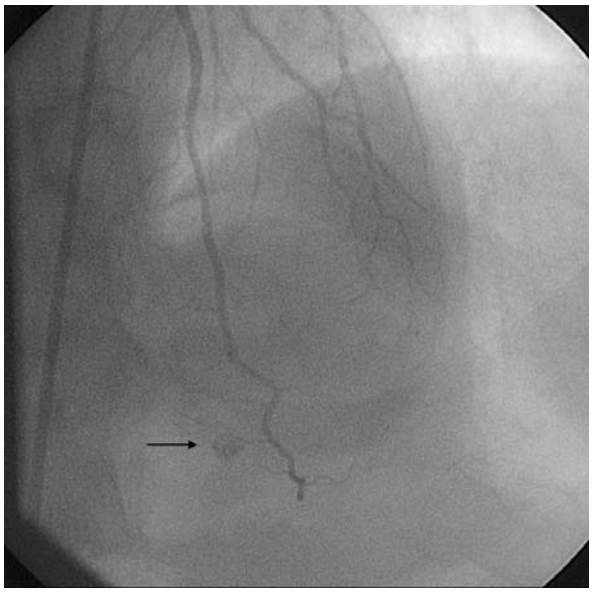

Figure 3. The coronary fistula is seen in the left cranial position (arrow).

catheter interventions.[4–6] In more than 90% of the cases, the fistula drains into venous structures. The site from which fistulas most frequently originate (in about half of the cases) is the right coronary artery. However, in a recently published series (by Tirilomis, et al.), the site of origin for the majority of the fistulas was the proximal LAD artery.[7] The most frequent complications are ischemia through myocardial "steal", thromboembolism, heart failure, rupture, endocarditis, or endarteritis. Whether adult patients are symptomatic or not depends on the size and location of the CAF; most of them do not reveal any symptoms at all. Symptomatic adult patients may have symptoms of dyspnea, fatigue, and angina. These symptoms might be due to the concomitant presence of underlying cardiac disease.[7–9]

The indications for treatment of CAFs include the presence of a large or increasing left-to-right shunt, left ventricular volume overload, myocardial ischemia, left ventricular dysfunction, and congestive cardiac failure, and for prevention of endocarditis/endarteritis.

The treatment options for CAFs include surgery or catheter closure. Surgery involves internal closure of the fistula within the receiving chamber or vessel whenever feasible; however, when the fistula is associated with a large aneurysm of the feeding artery, it may need to be ligated from within the aneurysm.[10] Surgery is not risk-free, and is associated with a low morbidity and mortality rate, ranging from 0 – 6%;[11,12] myocardial infarction may occur in less than 5% of the cases, and there is a risk of fistula recurrence.[13]

Catheter closure of fistulas is now considered to be an effective and safe alternative to surgery.[14,15] The aim of catheter closure is to occlude the fistula artery as distally and as close to its termination point as possible, so as to avoid any possibility of occluding branches to the normal myocardium. If, however, embolization is done too distally, and if there is no significant stenosis within the vessel, the embolization device could migrate beyond the fistula into the pulmonary circulation. Whichever technique of catheter closure is used, the occlusion should be at a precise point. Different types of embolization materials can be used, such as detachable balloons, stainless steel coils, or platinum micro-coils.[16–18]

Conclusion

As our patient was asymptomatic and hemodynamically stable and the fistula was small, we did not plan any treatment for the CAF. Collateral development in coronary arteries is a natural process after ischemia of the heart. This may be one of the mechanisms of acquired CAF development. However, the culprit lesion was the Cx-OM2 branch involving blood supply of the high lateral wall of the left ventricle, and the acquired CAF presented at the apex of the left ventricle. The theory of this acquired CAF is the progress of collaterals after myocardial infarction and the fistula constituted within this new angiogenesis in another site.

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