Myocardial ischemia due to a coronary-to-pulmonary artery fistula proximal to an intermediate stenosis in the left anterior descending coronary artery: percutaneous closure by means of angioplasty and stent grafting

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This case report refers to a 46-year-old patient with symptomatic stable angina and myocardial ischemia documented at ^{99m}Tc-MIBI SPECT scintigraphy. Coronary angiography revealed a coronary artery fistula (CAF) and a non-significant stenosis of the left anterior descending coronary artery (LAD). We performed coronary angioplasty and stenting of the LAD and closure of the CAF using a covered stent. After the procedure the patient was asymptomatic and a myocardial scintigraphy, performed 6 months later, confirmed the absence of myocardial ischemia. This suggests that a CAF arising before a coronary stenosis may contribute to the genesis of myocardial ischemia, perhaps by giving rise to a steal phenomenon.

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Introduction

Congenital coronary artery fistulae (CAF) are rare anomalies (0.3-0.4% of all congenital heart defects) and consist of a communication between a coronary artery and a cardiac chamber, a great artery, or the superior vena cava[1,2]. Coronary-to-pulmonary artery fistula is the most common type of fistula which is incidentally found in adulthood. The majority of these fistulae arise from the left anterior descending (LAD) or the right coronary artery while the circumflex coronary artery is rarely involved. Such fistulae may be single, i.e. arising from a single coronary artery, or bilateral, i.e. originating both from the left and right coronary arteries[3], or multiple, i.e. involving all the three coronary vessels. They drain more often to the pulmonary artery or its branches. The incidence is higher in the male sex. Generally, during the early decades CAF do not produce symptoms, but in later decades they are associated with an increased risk of complications, including cardiac failure, infective endocarditis, atrial fibrillation, rupture, myocardial ischemia, and angina[4]. Indeed, CAF, through several mechanisms such as the absence of capillarization or the steal phenomenon, may alter the coronary blood flow and determine myocardial ischemia further aggravated by the altered coronary artery properties when aneurysms or vascular channels develop[5,6].

Case report

A 46-year-old male was referred for coronary angiography in the setting of typical exercise angina and myocardial ischemia documented at ^{99m}Tc-MIBI SPECT scintigraphy. The patient had a past history of smoking and presented with hyperlipidemia and hypertension; 5 years before admission he had had a non-Q wave myocardial infarction. He remained asymptomatic on antianginal medications until 3 months before admission, when he started to complain of angina on moderate exercise. For this reason, he was submitted to myocardial scintigraphy that documented a completely reversible, moderate to severe perfusion defect of the anterolateral wall at the basal and mid segments, and a non-reversible, mild to

moderate perfusion defect at the inferoapical segment (Fig. 1). Baseline ECG was normal and no significant ST-segment changes were noted throughout the test; the patient achieved the first minute of the V step of the Bruce protocol with a peak exercise rate-pressure product of 32 800 mmHg×b/min. Coronary angiography performed during admission showed double vessel disease involving the LAD and the right coronary artery. At quantitative coronary angiography (QCA), a long, eccentric lesion causing a 46% stenosis of the internal luminal diameter was identified in the middle segment of the LAD, just after the origin of a minor first diagonal branch and a first septal branch. In the proximal segment of the LAD, before the origin of these two minor branches, a coronary-to-pulmonary artery fistula was present. The circumflex artery showed no significant stenosis (Fig. 2). A short, eccentric, non-calcified lesion causing a 41% stenosis of the internal luminal diameter was identified in the middle segment of the right coronary artery at QCA. Right and left heart catheterization excluded other abnormal shunts and confirmed normal pulmonary pressures and resistance. The calculated pulmonary-to-systemic flow ratio was 1.5/1.0. Two-dimensional echocardiography showed moderate inferoseptal hypokinesis. Surgical closure and bypass grafting were not considered because it was held that the patient's atherosclerotic coronary disease was amenable to percutaneous coronary intervention. Therefore, on the basis of the evidence of myocardial ischemia in the anterolateral region, the authors decided to treat both the LAD stenosis and the fistula by a percutaneous approach during the same procedure. Before the procedure, an intravenous bolus of heparin was administered (100 U/kg of body weight) to achieve an activated clotting time > 300 s. The left coronary artery was engaged with a 7F XB4SH guiding catheter (Cordis, Miami, FL, USA). Then, the middle LAD stenosis and CAF were assessed at intracoronary ultrasound (ICUS); a 3F-electronic monorail ICUS probe (Clear View Ultra, Boston Scientific Europe, Lisbon, Portugal) was placed distally to the LAD stenosis over a standard floppy 0.014'' Balance Middle Weight (ACS Hi-Torque, Guidant, Diegem, Belgium) guide wire and a video-loop was recorded with an automatic 0.5 mm/s pullback device. The reference maximum lumen diameter of the middle LAD was 3.3 mm and the vessel lumen area was 11.2 mm^2. The minimum vessel diameter of the plaque-containing segment was 2.5 mm and the minimum lumen area was 6.4 mm^2. This resulted in a calculated lumen stenosis of 43% (Fig. 3). The origin of the fistula was identified, at ICUS, in the LAD wall and had an ostial diameter of 1.9 mm. Following ICUS, a 3.0/32 mm balloon expandable bare metal stent was used to treat the mid LAD lesion, and a 3.5/16 mm balloon expandable polytetrafluoroethylene-covered stent (Jostent, Jomed International AB, Helsingborg, Sweden), was implant-

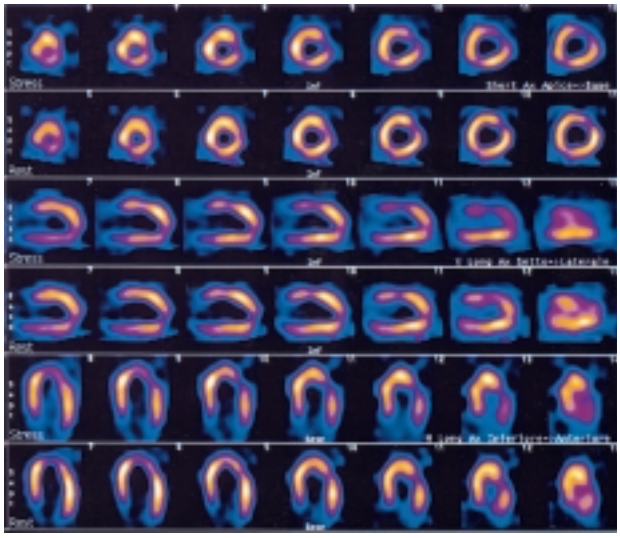

Figure 1. *^{99m}Tc-MIBI SPECT myocardial scintigraphy at stress and at rest before the percutaneous coronary intervention and fistula closure.*

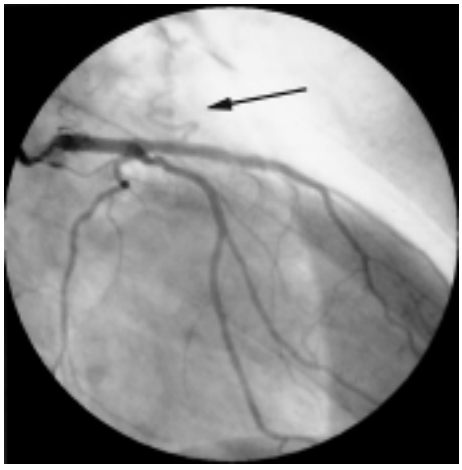

Figure 2. *30^0 right anterior oblique and 0^0 caudal projections of the left anterior descending and circumflex coronary arteries; the* black arrow *indicates the coronary artery fistula.*

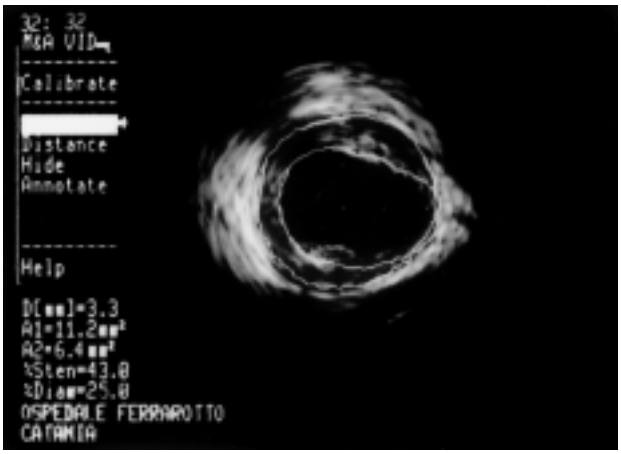

Figure 3. *Intracoronary ultrasound examination at the site of the maximal mid left anterior descending coronary artery stenosis.*

ed in the proximal segment of the LAD to cover the origin of the fistula. Both stents were deployed at high inflation pressures (16 atm). A final angiogram showed complete exclusion of the fistula, disappearance of both the first diagonal and septal branches and no residual LAD stenosis (Fig. 4). The pre- and post-percutaneous coronary intervention ECG were compared and non-significant changes were noted; there was no significant increase in the creatine kinase-MB levels. The patient was discharged on antianginal medications including ticlopidine 250 mg bid for 4 weeks, in addition to long-term aspirin. The patient's clinical conditions improved and at follow-up he was found to be totally asymptomatic. Six months after the revascularization procedure, a myocardial scintigraphy was performed, with no evidence of reversible perfusion defects and only a non-reversible mild to moderate perfusion defect at the inferoapical segment (Fig. 5). The exercise test was stopped at the first minute of the IV step of the Bruce protocol, with a peak exercise rate-pressure product of 34 100 mmHg×b/min. Throughout the test, no significant ST-segment changes were observed.

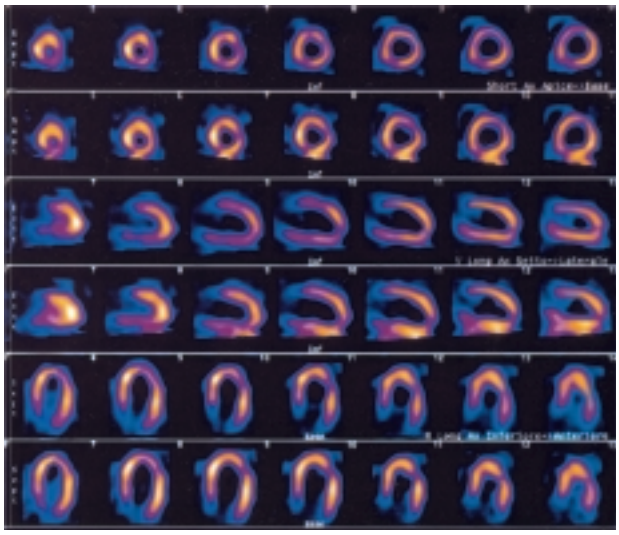

Figure 5. *^{99m}Tc-MIBI SPECT myocardial scintigraphy at stress and at rest after the percutaneous coronary intervention and fistula closure.*

Discussion

Several authors have reported that CAF are frequently associated with myocardial ischemia[7,8]. The increased blood flow over the systemic-to-pulmonary artery fistula may reduce the distal intracoronary diastolic pressure and produce ischemia by a coronary steal phenomenon[9]. The association between coronary congenital anomalies and atherosclerotic obstructions of the coronary arteries has been reported[2]. We know that coronary arterial atherosclerosis affects patients with congenital fistulae in the same way as normal humans[10], and that the severity of myocardial ischemia increases when the coronary steal phenomenon is associated with atherosclerotic involvement of the same vessel[11]. Furthermore, there is a recognized relationship between myocardial ischemia and infarction in the presence of CAF even when significant atherosclerotic coronary artery stenosis is lacking[12].

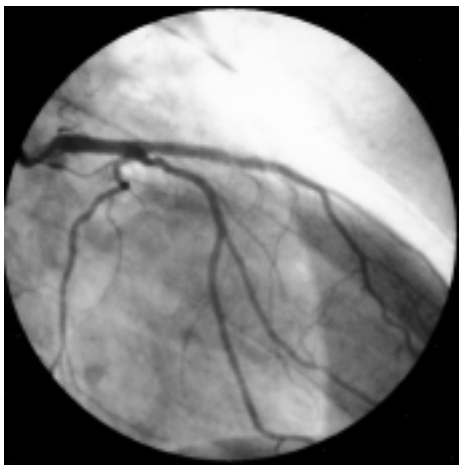

Figure 4. *30^0 right anterior oblique and 0^0 caudal projection left coronary angiograms after the percutaneous coronary intervention and fistula closure.*

The natural history of CAF includes spontaneous closure. Various authors suggest percutaneous embolization for the closure of symptomatic and asymptomatic small CAF during childhood when the anatomical features are favorable[13]. Cardiopulmonary bypass is reserved for patients in whom percutaneous treatment of the CAF is not possible, or for those with coexisting significant coronary stenosis. Transcatheter embolization of CAF has been performed in the past using Gianturco coils, detachable balloons, polyvinyl alcohol foam, and double umbrella devices. The complications associated with these transcatheter methods included transient T-wave inversion at ECG associated with a small increase in creatine kinase levels, embolization of the coil to the pulmonary artery and transient arrhythmias[14]. All these techniques have been designed to occlude the fistula. In the present case the authors decided to treat the CAF percutaneously using a covered stent, the Jostent; this is a two-stent device with a pericardial layer between the stents; it creates an artificial vessel wall that allows exclusion of aneurysms or vessel perforation. The same therapeutic strategy has been described by other authors[15-18]. A limitation of stent graft implantation is the stent-dependent occlusion of side branches arising from the same target segment. This anatomical configuration is a concern for the LAD, which often gives rise to important septal branches or diagonal vessels. However, in this case no major branches were seen to arise at the site of the fistula.

In this patient, with three risk factors for coronary artery disease, coronary angiography showed the coexistence of a congenital anomaly and atherosclerotic dis-

ease in the same vessel. In this setting, the presence of the fistula may have significantly contributed to the genesis of myocardial ischemia. Therefore it would have been appropriate, before any intervention, to independently assess the hemodynamic significance of both the CAF and the lesion. The coronary pressure-derived fractional flow reserve (FFR) was thought to be appropriate for the assessment of the functional significance of the CAF; however, as the fistula arose just at the proximal edge of the stenosis, measurement of FFR would have been affected by the presence of the fistula itself[19]. Thus, we decided to treat both the lesion and the CAF during the same procedure. However, implantation of both a covered stent and a long bare metal stent which resulted in a 48 mm stent length could have increased the risk of restenosis. This is a recognized complication. Unfortunately, no drug-eluting stent with a diameter of 3.5 mm was available at the time of the procedure.

An alternative approach could have been first to exclude the fistula with a covered stent and then to measure the functional significance of the stenosis by means of FFR or myocardial scintigraphy. However, this approach could have resulted in some complications such as: plaque shift onto the lesion, vessel dissection, and the inability to recross the covered stent with a long stent in case it was necessary to treat the mid LAD lesion.

This case report highlights the feasibility of a percutaneous coronary procedure to close a CAF using a covered stent. As suggested by the significant reversible perfusion defects found at myocardial scintigraphy, the presence of a CAF associated with non-significant atherosclerotic disease in the same vessel is likely to have significantly contributed to the genesis of myocardial ischemia probably by inducing a steal phenomenon.

References

1. Imperadore F, Moschini E, Volta G, et al. Congenital arterio-venous coronary fistula associated with patent ductus arteriosus: description of a clinical case and review of the literature. Cardiologia 1997; 42: 525-8.
2. Rigatelli G, Rigatelli G. Coronary artery anomalies: what we know and what we have to learn. A proposal for a new clinical classification. Ital Heart J 2003; 4: 305-10.
3. Baim DS, Kline H, Silverman JF. Bilateral coronary artery-pulmonary artery fistulas. Report of five cases and review of the literature. Circulation 1982; 65: 810-5.
4. Reydi JF, Anjos RT, Qureshi SA, et al. Transcatheter embolization in the treatment of coronary artery fistulae. J Am Coll Cardiol 1991; 18: 187-92.
5. Rangel-Abundis A, Munoz-Castellanos L, Marin G, et al. Morphofunctional correlation in congenital anomalies of the coronary arteries. I. Coronary artery fistulae. Arch Inst Cardiol Mex 1994; 64: 161-74.
6. Chang SM, Matangi MF. Unstable angina with coronary to pulmonary artery fistulae. Can J Cardiol 1987; 3: 220-2.
7. Kiuchi K, Nejima J, Kikuchi A, et al. Left coronary artery-left ventricular fistula with acute myocardial infarction, representing the coronary steal phenomenon: a case report. J Cardiol 1999; 34: 279-84.
8. Wolf A, Rockson SG. Myocardial infarction due to multiple coronary-cameral fistulae: two case reports and review of the literature. Cathet Cardiovasc Diagn 1998; 43: 179-83.
9. Theman TE, Crosby DR. Coronary artery steal secondary to coronary arterio-venous fistula. Can J Surg 1981; 24: 231-6.
10. Rangel A, Chavez E, Badui E, et al. Case report of association of congenital coronary fistulae with coronary atherosclerosis. Rev Invest Clin 1995; 47: 481-6.
11. Mukai H, Minemawari Y, Hanawa N, et al. Coronary stenosis and steal phenomenon in coronary-pulmonary fistula-assessment with stress thallium tomography after coronary angioplasty and fistulectomy. Jpn Circ J 1993; 57: 1021-6
12. McLellan BA, Pelikan PC. Myocardial infarction due to multiple coronary-ventricular fistulae. Cathet Cardiovasc Diagn 1989; 16: 247-9.
13. Aggoun Y, Bonhoeffer P, Sidi D, et al. Congenital coronary-cardiac fistula in children. Effects of surgical occlusion and percutaneous embolization. Arch Mal Coeur Vaiss 1997; 90: 605-9.
14. Mavroudis C, Backer CL, Rocchini AP, et al. Coronary artery fistulae in infants and children: a surgical review and discussion of coil embolization. Ann Thorac Surg 1997; 63: 1235-42.
15. Dorros G, Thota V, Ramireddy K, Joseph G. Catheter-based techniques for closure of coronary fistulae. Catheter Cardiovasc Interv 1999; 46: 143-50.
16. Atmaca Y, Altin T, Ozdol C, et al. Coronary-pulmonary artery fistula associated with right heart failure: successful closure of fistula with a graft stent. Angiology 2002; 53: 613-6.
17. Mullasari AS, Umesan CV, Kumar KJ. Transcatheter closure of coronary artery to pulmonary artery fistula using covered stents. Heart 2002; 87: 60.
18. Balanescu S, Sangiorgi G, Medda M. Successful concomitant treatment of a coronary to pulmonary artery fistula and a left anterior descending artery stenosis using a single covered stent graft: a case report and literature review. J Interv Cardiol 2002; 15: 209-13.
19. Baim DS, Grossman W. Evaluation of myocardial blood flow and metabolism. In: Baim DS, Grossman W, eds. Cardiac catheterization, angiography and intervention. 6th edition. Philadelphia, PA: Lippincott Williams & Wilkins, 2000: 393-421.