Dual Coronary Artery Fistulas as a Cause of Unstable Angina

To the Editor,

Coronary artery fistulas are a rare congenital coronary anomaly. Although often small and diagnosed by chance, they can cause a wide range of symptoms associated with myocardial ischemia, including sudden death.1

We present the case of a 63-year-old woman with cardiovascular risk factors of overweight, smoking, and high blood pressure. For several years, following moderate effort the patient had reported symptoms clearly indicating angina although these had always been labeled as of musculoskeletal origin. She came to the Emergency Room following a more prolonged episode than normal that occurred while resting and after a family argument. The electrocardiogram showed no changes, and serial measurements of cardiac enzymes and baseline echocardiography were normal. Exercise testing performed 1 day later was inconclusive, with mild clinical symptoms of angina at the start of Bruce protocol stage 3, without reaching maximum heart rate (MHR). Leads V5-V6 did show a slight but nonsignificant tendency towards ST-segment depression. Given these results, the patient underwent dobutamine stress echocardiography. On reaching 135 bpm, she presented the same symptoms of angina accompanied by ST-segment depression in precordial leads V4-V5, I, and aVL, as well as akinesia in all apical and mid-inferior segments. The test was interrupted and analytical tests 6 h later revealed a 0.25 ng/ml troponin T peak.

![Figure 1](https://www.revespcardiol.org/) A: dual coronary artery fistula in left anterior descending artery draining towards the right ventricle. B and D: closure of both fistula paths by coil implantation. C: fistula in right descending coronary artery also draining into the right ventricle.
Coronary angiography showed the absence of significant atherosclerotic lesions. From the left anterior descending artery proximal and mid segments, a dual coronary artery fistula drained into the right ventricle (Fig. 1A). The underdeveloped right descending coronary artery presented another coronary artery fistula draining into the right ventricle (Fig. 1C). Both fistula paths were occluded by deploying 10 coils via radial access (Figs. 1B and D). Final results were good with no complications.

During her check-up after 8 weeks, the patient reported a clear improvement in functional class, having presented no further symptoms of angina despite receiving no anti-angina drugs other than 100 mg losartan/day for high blood pressure control for more than 6 years. She underwent control exercise testing, completing the Bruce protocol stage 3 and reaching 96% of MHR with neither electrical nor clinical changes. During a second stress echocardiography test, she reached MHR in the maximum stage of 40 μg/kg/min without presenting electrical or echocardiographic changes.

A congenital coronary artery fistula is an abnormal communication between an epicardial coronary artery and a cardiac chamber or vascular structure located near the heart: 40% to the right ventricle, 25% right atrium, 15% to 20% pulmonary artery and 7% coronary sinus.1 It is found in approximately 0.10% to 0.15% of coronary angiographies and most frequently affects the right descending coronary artery (60%).1–3 It is usually found by chance with no identifiable associated symptoms, but symptoms do appear when the shunt is significant, fundamentally variable degrees of ischemia and of heart failure.2

Dual coronary artery fistulas affecting both coronary territories are extremely rare (5% of the total) and more frequently present phenomena of secondary coronary ischemia than of significant left-right shunt with phenomena of “coronary steal”.4 The percutaneous approach and coil deployment—which is less invasive and involves a shorter hospital stay—is the current method of choice.2 In our case, it permitted symptom control with proven absence of inducible ischemia in the follow-up. Surgical closure is currently reserved for large multiple fistulas, which are generally diagnosed in childhood,2 when different technical considerations need to be borne in mind.3

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Sandwich Stenting to Treat an Ostial Left Main Narrowing Following Transcatheter Aortic Valve Implantation

Implantación de stent «en sandwich» para tratar una estenosis del ostium de la principal izquierda tras implantación de válvula aórtica percutánea

To the Editor,

Transcatheter aortic valve implantation (TAVI) is becoming a feasible and effective therapeutic option in patients with severe symptomatic aortic stenosis at high risk for surgical aortic valve replacement (AVR). Despite being less invasive than conventional surgical AVR, TAVI is still associated with periprocedural complications including acute coronary obstruction.1

An 86-year-old woman with severe symptomatic aortic stenosis (peak gradient 100 mmHg, mean gradient 68 mmHg) presented to our institution with dyspnea (NYHA III) and rest angina (CCS 4) despite medical treatment. The patient was deemed to have a high surgical risk for AVR on the basis of age, general frailty, and the presence of a porcelain aorta, and was referred for transfemoral TAVI.

The procedure was performed as previously described.2 Following implantation of a 23-mm Sapien XT (Edwards Life-sciences, Irvine, California) aortic bioprosthesis (Fig. 1A, video), the patient experienced acute hypotension with a blood pressure of 85/60 mmHg (Fig. 1B). Echocardiography showed severe left ventricular dysfunction without cardiac tamponade. Aortography excluded severe aortic regurgitation, aortic dissection, and aortic root rupture but left main coronary artery (LMCA) ostial obstruction was suspected (Fig. 1C), especially as the patient developed ST-segment depression in the precordial leads (Fig. 1D). The LMCA was engaged with a guiding catheter and angiography confirmed severe ostial narrowing, most probably due to displacement and obstruction by the calcific native valve leaflets. A coronary wire was advanced through the lesion (Fig. 2A) and a 3.5 × 12 mm Promus Element stent (Boston Scientific, Natick, Massachusetts) was implanted at the LMCA ostium. Subsequent coronary angiography revealed incomplete stent expansion, most likely due to acute stent recoil (Fig. 2B). Post-dilatation with a 3.5 × 12 mm noncompliant balloon showed good expansion of the balloon (Fig. 2C) but fluoroscopy again demonstrated dynamic recoil of the ostial part of the stent (Fig. 2D). We concluded that the stent did not have sufficient radial strength to push the bulky leaflet away from the ostium. Thus we decided to implant a 3.5 × 9 mm cobalt-chromium bare-metal stent within the first stent (“sandwich stenting”) to increase the radial strength of the scaffold without doubling the drug dose (Fig. 2E). Following

1220 Letters to the Editor / Rev Esp Cardiol. 2011;64(12):1215–1230