Evolutionary Recanalization of Spontaneous Coronary Artery Dissection
Insights From a Multimodality Imaging Approach

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A 50-year-old man, a former smoker, presented with an ST-segment elevation anterior myocardial infarction that was treated with thrombolysis in another center. Because of the absence of reperfusion criteria, he was referred to our hospital for a rescue coronary intervention. A transthoracic echocardiogram showed that the overall left-ventricle systolic function was preserved but with apical akinesia. Coronary angiography revealed an occlusive lesion (100%) in the mid-left anterior descending coronary artery distal to a segment with a tapering caliber (Figure 1A). At that time, the patient was asymptomatic, and, therefore, a conservative management was decided, including dual antiplatelet therapy and full anticoagulation. Biomarkers included elevated creatine kinase (peak: 926 U/L; n<190), creatine kinase-MB peak (peak: 36.4 ng/mL; n<5), and troponin I peak (peak: 15.90 ng/mL; n<0.2). A new coronary angiogram performed 7 days later revealed a complete recanalization of the left anterior descending coronary artery but with a very diffuse narrowing of its mid-segment (that persisted after nitroglycerin administration) with an abrupt recovery of vessel caliber near the apex (Figure 1B). Optical coherence tomography accurately visualized the affected coronary wall along the entire vessel length unraveling the presence of a long intramural hematoma. The thickness and circumferential distribution of the intimo-medial membrane was accurately depicted (Figure 2). The residual lumen size was preserved (3.2 mm²), and an entry tear was not visualized. Ten days after admission, multislice computed tomography was performed, showing a very mild, diffuse, lumen narrowing, with a subtle wall thickening at the target segment (Figure 1C). The patient was discharged after an uneventful hospitalization. At discharge, medications included 1-year dual antiplatelet therapy (aspirin/clopidogrel), β-adrenergic blockers, angiotensin-converting enzyme inhibitors, and statins. After a 6-month follow-up, the patient remained asymptomatic, and the echocardiogram displayed a complete recovery of the previous apical segmental motion abnormalities.

Spontaneous coronary artery dissection (SCAD) is an infrequent cause of acute coronary syndrome, typically affecting young otherwise healthy women.1 Dissection of the coronary intima or media is a hallmark finding, and hematoma formation deeper within the vessel wall is often present. It remains unclear whether dissection or hematoma is the primary event, but both may cause luminal stenosis and occlusion.1 Pressure-driven expansion of the false lumen induces axial propagation of the disease and true lumen compression, resulting in myocardial ischemia.2 However, angiography is sometimes unable to visualize the coronary wall, so its diagnostic accuracy for SCAD is often limited.3 However, intramural hematomas should be suspected in patients with otherwise completely smooth vessels (typically with curly or even corkscrew appearance) at segments showing a diffuse lumen narrowing that causes straightening of the vessel or a characteristic broken-line appearance.2 Conversely, optical coherence tomography provides unique insights on most relevant morphological features of the condition, including entry tear, intimo-medial flap, double-lumen morphology, intramural hematoma, and associated thrombus.3,4 Because coronary computed tomography angiography provides a direct view of the coronary plaque composition and wall thickness, it may be useful in the diagnosis and assessment of this pathology.

In this case, we observed an interesting short-term spontaneous recanalization after conservative management in an SCAD setting. The treatment of SCAD remains controversial, but probably the conservative choice is the best option in stable patients. Computed tomography angiography as a noninvasive method typically shows the presence of a double lumen. However, despite progress with this diagnostic method, it has some limitations in the accurate visualization when the segment involved is more distal (smaller diameter). Thus, the definitive diagnosis of SCAD, because of its prognostic implications, may require invasive intracoronary imaging techniques, such as intravascular ultrasound or mainly optical coherence tomography.

Disclosures

None.

References


From Interventional Cardiology (B.C.D., I.J.N.-G., R.H., C.C., P.J.Q., J.E., C.M., A.F.O.) and Cardiovascular Imaging (J.J.G.d.D.), Cardiovascular Institute, Clinical Hospital San Carlos, Madrid; and Cardiology Department, Hospital de la Princesa, Madrid, Spain (F.A.).

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**Figure 1.** Coronary angiography and multislice coronary angiotomography in spontaneous coronary artery dissection. **A**, Coronary angiography showing an occlusive lesion in the medium left anterior descending coronary artery (arrow). **B**, Left anterior descending coronary artery open with significant reduction in medium size and recovery distal diameter (arrow). **C**, Multislice coronary angiotomography showing double lumen in medium left anterior descending coronary artery (arrow).

**Figure 2.** Coronary angiography and optical coherence tomography in spontaneous coronary artery dissection. **A**, Left anterior descending coronary artery open with significant reduction in medium size and recovery distal diameter (arrow). **B**, An image of trabecular intramural hematoma dissection was demonstrated in the mid and distal segments of the vessel (arrow).
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