Searching for the Culprit Vessel in Acute Myocardial Infarction Beyond Angiography
Role of Cardiac Magnetic Resonance

Irene Méndez, MD; Eduardo Pozo, MD; Amparo Benedicto, MD; Maria José Olivera, MD; Alfonso Ascensión, MD; Luis Jesús Jiménez-Borreguero, MD; Fernando Alfonso, MD, PhD

An 80-year-old hypertensive man with previous percutaneous coronary interventions with stent implantation on the right and left circumflex coronary arteries was referred to our center because of ST-segment elevation anterior myocardial infarction. At admission the ECG showed a right bundle-branch block and ST-segment elevation in V1 through V4, III, and aVF. Akinesis in the middle and distal interventricular septum with preserved left ventricular systolic function was showed by transthoracic echocardiography. No images suggestive of aortic dissection were visualized. Emergent coronary angiography revealed a moderate lesion in the mid-left anterior descending coronary artery with good anterograde coronary flow (Figure 1A). The stents on the right and left circumflex coronary arteries showed no significant stenoses and a normal coronary flow. Although the patient reported persistent chest pain and the ECG remained unchanged, no coronary revascularization was attempted as the culprit lesion could not be identified. Clinical outcome was characterized by an important rise in cardiac biomarkers that showed an early peak (high-sensitivity troponin T 2624 ng/mL [normal value, 0–14 ng/mL]; creatin-kinase 1107 IU/L [normal value, 7–177 IU/L], and MB fraction 123 IU/L). To further ascertain the cause of the myocardial necrosis, a cardiac magnetic resonance was performed. Cine imaging confirmed the presence of akinesis in the mid- apical anterior septum with a left ventricular ejection fraction of 56% and persistence of akinesis in the myocardial necrosis, a cardiac magnetic resonance was instrumental in confirming the confined septal infarction (Figure 2A). Early gadolinium enhancement sequences detected a transmural perfusion defect in the middle and apical anterior septal segments that corresponded with a transmural necrosis on late gadolinium enhancement in the territory of a septal coronary artery (Figure 2B and 2C). All of these findings were consistent with a large acute myocardial infarction of a major septal branch.

A new coronary angiography demonstrated a subocclusive thrombus in the proximal segment of a large septal branch with thrombolysis in myocardial infarction flow grade 2, not previously visualized probably because of its ostial occlusion (Figure 1B). Optical coherence tomography imaging of this septal branch revealed a large atherosclerotic plaque associated with a nearly occlusive red thrombus (Figure 3). Selective thromboaspiration of the septal branch improved the angiographic image and achieved a thrombolysis in myocardial infarction flow grade 3. Histology of the thrombus was performed, showing a tissue composed by fibrin, erythrocytes, and inflammatory cells (Figure 4), characteristic findings of a red thrombus. Two months after the septal myocardial infarction, an echocardiogram was made, demonstrating a left ventricular ejection fraction of 56% and persistence of akinesis in the mid-apical anterior septum.

The inability to detect a culprit lesion during emergent coronary angiography in patients presenting with the diagnosis of acute myocardial infarction is not uncommon, representing up to 14% of the cases. This scenario may be explained either by diseases that mimic an acute coronary syndrome (myocarditis or Tako-Tsubo cardiomyopathy) or by infrequent presentations of acute ischemic events that entail a difficult angiographic diagnosis (vasospasm, lysis of an embolus or thrombus, and ostial occlusion of branch vessels). In this clinical setting, cardiac magnetic resonance has an important role in allowing the differentiation between ischemic and nonischemic etiology, as well as providing a specific diagnosis in the latter, and thus relevant prognostic information.

This unique case of isolated ostial septal occlusion highlights the fact that the apparent absence of significant stenosis in coronary angiography does not necessary reflect a nonischemic cause and suggests that further diagnostic efforts should be pursued in these challenging patients. In our case, cardiac magnetic resonance was instrumental in confirming the confined septal infarction, whereas optical coherence tomography unraveled the underlying substrate on the culprit septal artery. The use of optical coherence tomography and thromboaspiration has not been reported previously on a septal branch, although the size of the culprit septal branch in our patient justified these procedures. In patients with acute myocardial infarction and elusive...
culprit vessels, cardiac magnetic resonance should be considered as a key test to obtain a definitive diagnosis.

**Disclosures**

None.

**References**


**Figure 1.**

**A**, Coronary angiogram of the left coronary artery in a right anterior oblique (30°) projection with cranial (20°) angulation. A long, borderline lesion is visualized in the mid-left anterior descending coronary artery (LAD) that showed a normal (thrombolysis in myocardial infarction; TIMI 3) anterograde coronary flow. D1 and D2 indicate first and second diagonal branches. **B**, Coronary angiogram 4 days later. A large major septal branch is now readily visualized with a clear intraluminal filling defect (arrow) at its proximal segment.

**Figure 2.**

Cardiac magnetic resonance images. **A**, T2-weighted imaging demonstrating (arrow) a hyperintense signal in the septal myocardium suggestive of myocardial edema. **B**, Early gadolinium enhancement showing a transmural perfusion defect in the septum. **C**, Late gadolinium enhancement depicting the transmural necrosis.
Figure 3. Optical coherence tomography images of the reperfused large septal branch. A, Healthy vessel wall (characteristic 3-layer vessel wall morphology) with a protruding red thrombus (T; arrow) inducing major dorsal shadowing. B, Red thrombus causing severe lumen narrowing. C, Most proximal aspect of the red thrombus in a segment with a large fibrotic plaque but a widely preserved lumen. D, Longitudinal reconstruction of the septal branch disclosing the corresponding thrombus location (small arrows). LAD indicates left anterior descending coronary artery; and Prox, Proximal. *Wire artifact.

Figure 4. Micrograph showing red thrombus composed of erythrocytes and inflammatory cells entrapped by fibrin network (hematoxylin-eosin, ×40 magnification).
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