A 46-year-old woman without a history of cardiovascular disease and no coronary risk factor was urgently admitted to our hospital with rest crushing chest pain, which had started 3 hours before. The ECG demonstrated minimal anterior ST-segment elevation in leads V1 to V4 (Figure 1). In the first sample cardiac troponin T was 0.08 ng/mL. Coronary angiography was urgently performed and demonstrated a 50% smooth stenosis in the proximal left anterior descending (LAD) coronary (Figure 2) with a faint dye staining just proximal to the first diagonal branch. Because initial differential diagnosis included variant angina, intracoronary ergonovine (32 μg) was given. Diffuse LAD coronary vasoconstriction, more severe on the LAD coronary ostium, was seen. Because there was still no clear indication of a culprit lesion, intravascular ultrasound (IVUS) examination was performed, which surprisingly showed an intramural hematoma starting from the mid-LAD up to its ostium, with mild lumen compromise, and the presence of nonobstructive atheroma (Figure 2). Echo-free material, consistent with contrast medium or saline, was seen mixing with echogenic blood within the hematoma (Figure 3), indirectly indicating that the hematoma was the result of a spontaneous coronary dissection (SCAD), with an occult entry flap. Stenting was considered, but because of the stable hemodynamic condition, medical management with aspirin, clopidrogel, nitrates, and β-blockers was chosen. After 12 hours, repeat ECG demonstrated deep, negative T waves in the anterior leads. (Figure 1). The cardiac troponin T peak was 0.2 ng/dL at 24 hours. Angiography and IVUS were repeated at 48 hours and showed persisting intramural hematoma, although with reduced lumen compression (Figure 4). Because of the absence of what we considered to be “high-risk” IVUS features, the patient was discharged home on medical therapy. At 3 months, IVUS control showed an almost complete disappearance of intramural hematoma, and echocardiography was normal (Figure 5). One-year clinical follow-up was uneventful.

SCAD is an uncommon cause of acute coronary syndrome and sudden death. Its etiology and pathogenesis have not been fully elucidated, and prognosis and treatment are not clearly defined. Connective tissue disease, immune system alterations, pregnancy, oral contraceptive use, intense physical activity, and illicit drug consumption are known to be involved in SCAD etiology, and yet in a relevant percentage of SCAD, as in our case, none of these conditions is overtly present.

The initial presentation is frequently an acute coronary syndrome, and SCAD should be taken into consideration in patients without known cardiovascular risk factors presenting with acute coronary syndrome. Because there is lack of consensus concerning SCAD treatment, management decisions are generally made on a case-by-case basis, integrating the clinical scenario with angiographic assessment of dissection location and the extent and degree of flow compromise. Treatment options include medical therapy, percutaneous intervention, and, in extreme cases, surgical revascularization that is reserved to left main or multivessel dissections. Coronary stenting (drug-eluting stents have been used) offers a rapid revascularization option because it can seal off intimal tears and/or tamponade further hemorrhage. However, stenting remains limited by the possible longitudinal extension of the dissection and by the subsequent risk of in-stent restenosis in a generally young and otherwise healthy patient. In our case, in consideration of the stable hemodynamics and of the absence of “high-risk” features (side branch or ostial involvement, tendency to grow) at 48-hour IVUS control, we chose a conservative strategy. IVUS is uniquely useful to differentiate atherosclerotic disease from SCAD in cases with ambiguous coronary angiography and to determine the morphology and the extent of dissection. Moreover, if stenting is chosen as a treatment, IVUS is invaluable in confirming correct guide wire placement before stenting (risk of false-lumen stenting), stent apposition, symmetry and expansion, and especially to rule out dissection extension. IVUS, however, may not have the resolution power to show small entry flaps (as it is likely in our case), as opposed to optimal coherence tomography, which is in turn limited by reduced depth penetration and has not yet been tested in this setting.

An association between intense coronary vasospasm and intramural hematoma formation has been reported, but in our case the possibility of an iatrogenic effect is highly unlikely because the patient presented with ischemic symptoms and signs before intracoronary ergonovine, and these symptoms and signs did not reappear after drug administration.
In conclusion, SCAD should be considered in any young adult who presents with an acute coronary syndrome without a history of cardiovascular disease, and IVUS imaging should be considered extremely useful (or perhaps mandatory) in the diagnosis and treatment of SCAD.

Disclosures
None.

References


Keywords: spontaneous coronary artery dissection coronary disease acute coronary syndrome IVUS
Figure 2. Coronary angiography and intravascular ultrasound at baseline. Coronary angiography demonstrated a 50% smooth stenosis in the proximal LAD coronary artery. Intravascular ultrasound examination showed an intramural hematoma starting from the mid-LAD up to the LAD ostium, with lumen compromise (minimal lumen area $\approx 5.2 \text{ mm}^2$ at ostium) but without side branches or left circumflex compromise. Nonobstructive atheroma is present.

Figure 3. IVUS features indicating that the intramural hematoma derives from a spontaneous coronary dissection. In these 2 IVUS slices taken at approximately 5-mm distance, echo-free space compatible with contrast/saline (A, arrow) can be seen mixing with echogenic stagnant blood (B, arrow). This confirms a communication between lumen and intramural hematoma, probably smaller than the resolution power of IVUS.
Figure 4. Coronary angiography and IVUS at 2 days after the first ones. Two days after the first ones, angiography and IVUS were repeated and showed the persistence of the intramural hematoma, although with reduced lumen compression (minimal lumen area >6 mm² at the LAD coronary ostium).

Figure 5. Coronary angiography and IVUS at 3 months. At 3 months, IVUS control showed the almost complete disappearance of intramural hematoma.
Intravascular Ultrasound–Documented Healing of Spontaneous Coronary Artery Dissection

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