An Uncommon Myocardial Infarction:
Spontaneous Coronary Artery Dissection

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Background

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS), occurring in 0.1-1.1% of angiographic cases (1).

Case Presentation

A 43-year-old previously healthy woman presented with acute chest pain and diaphoresis with otherwise benign exam and vitals. An electrocardiogram (EKG) showed an inferior STEMI with ST elevations in leads II, III, aVF, with initial Troponin I of 137. She was given aspirin, plavix, and heparin. Catheterization and intravascular ultrasound (IVUS) revealed a false lumen and spontaneous acute dissection of a dominant right coronary artery (RCA), then emergently stented. Her post-catheterization EKG showed ST elevation resolution. She was started on metoprolol post-procedure to reduce wall stress, and dual anticoagulation with aspirin, beta blockers, and heparin. Initial EKG showing ST elevations in leads II, III, aVF, V5, and V6, with some reciprocal ST deprevations.

Workup

Figure 1: Coronary angiograms in the RAO view focused on the right coronary artery (RCA). Left: angiogram showing spontaneous coronary artery dissection (SCAD) of a dominant mid-RCA. Right: angiogram after percutaneous coronary intervention (PCI) with overlapping stents that sealed the inlet and outlet of the dissection from the ostium to the mid-RCA.

Figure 2: Intravascular ultrasound (IVUS) showing a false lumen, congruent with a type 2 SCAD.

Figure 3: Associated conditions, inciting factors, and angiographic diagnosis of SCAD.

Discussion

SCAD results from non-traumatic, non-iatrogenic separation of the coronary arterial wall, found more commonly in the younger, female population (2). Types include intimal tear with a subsequent false lumen within the medial layer, but more commonly an intramural coronary hematoma between media and intima layers. Although the underlying mechanism is not completely understood, it is proposed that an intimal wall tear results in intramural bleeding and subsequent dissection (2). Others propose that underlying vasculatures such as tortuosity or FMD predispose the coronary arteries to fragility and increased dissection risk (2,4). It is estimated that 1 in 16,000 pregnancies are complicated by acute myocardial infarction (MI) with about 1 in 4 due to SCAD, likely due to increased hemodynamic stress during pregnancy and hormonal imbalances (5,6). Newer studies have shown increasing prevalence in postmenopausal women (7). SCAD can also be seen in connective tissue diseases like Marfan’s and Ehlers-Danlos, in which medial degeneration may lead to wall weakness (7). The most common presenting symptom was chest pain (10) and usually accompanied by troponin elevation (11). Diagnosis is achieved via angiography, with the most commonly affected vessel being the left anterior descending artery (LAD) (12). Management is usually conservative with medications such as aspirin, beta blockers, and short-term clopidogrel (12), although revascularization is recommended if concerned for ischemia or hemodynamic instability (13) Post-SCAD, patients are recommended for cardiac rehabilitation (13). As SCAD may be the initial manifestation of an underlying systemic arthropathy, as in our patient, arterial imaging with CTA or MRA is recommended (13).

Conclusion

Consider SCAD in younger women presenting with chest pain without significant cardiac history. There lacks standard practices for revascularization versus medical management, and results in patient-specific decisions. All patients with SCAD should undergo cardiac rehabilitation and advanced arterial imaging to determine other underlying arterial abnormalities.