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Spontaneous coronary artery dissection: Practical considerations in management

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Over recent years, spontaneous coronary artery dissection (SCAD) has gained a substantial reputation as a specific form of coronary artery disease mostly in young females [1–3]. Pathologically, it is defined as a separation between intima and media layers in epicardial coronary arteries (mostly left anterior descending [LAD] artery) in the absence of traumatic and iatrogenic triggers [1–3]. On invasive coronary angiogram (CAG), this phenomenon might present with a variety of patterns including characteristic dissection flap (type-1), intramural hematoma (type-2 and 3) and total occlusion (type-4) [1]. In their recently published expert opinion paper, Kadziela et al. [1] have presented a didactic overview of current information on SCAD and its management. In this context, we would like to put a further emphasis on certain aspects of SCAD management based on our perspectives and experiences:

First, we agree with the authors that management of SCAD with invasive strategies (mostly percutaneous coronary interventions [PCIs]) should be the preferred option in the setting of high-risk features including hemodynamic instability, malignant arrhythmias, and persistent ischemia [1, 2]. However, an existing high-risk anatomy (for instance; SCAD involving proximal LAD), unlike other high-risk features, might not be regarded as an indication for urgent management. Accordingly, a ‘deferred PCI’ strategy (planned a few days or a week
later following admission) might arise as a viable option particularly in the setting of challenging type-1 SCAD (spiral or long dissection, etc.) involving anatomically high-risk coronary segments. This delayed strategy might significantly enhance the success of PCI, and significantly diminishes complication rates due to the partially regressed false lumen at the time of deferred intervention. Importantly, strict control of blood pressure and heart rate along with the administration of glycoprotein IIb/IIIa inhibitors, heparin and dual antiplatelet therapy (DAPT) generally prevent potential complications (SCAD thrombosis or propagation) [1, 2] until the time of deferred PCI. However, SCAD involving the left main coronary artery (LMCA), even if clinically silent, should be regarded as an exception that requires urgent intervention due to its life-threatening risks [1, 2].

Second, SCAD might occasionally extend to proximal aorta during coronary interventions [4] or spontaneously. Of note, absence of atherosclerosis generally facilitates retrograde SCAD propagation [2] potentially leading to extensive aortic involvement. Importantly, patients with SCAD involving LMCA or ostial right coronary artery (RCA) should be particularly evaluated in terms of co-existing aortic dissection with further imaging modalities (computed tomography [CT], etc.). In this context, extensive involvement of aorta and/or aortic dilatation strongly favor urgent aortic repair and coronary artery bypass grafting rather than PCI [4]. More alarmingly, use of glycoprotein IIb/IIIa inhibitors or thrombolytic therapy in SCAD patients with a missed aortic dissection might result in catastrophic complications (including aortic rupture). Taken together, a high index of suspicion for aortic involvement is mandatory in the setting of SCAD particularly involving LMCA or ostial RCA.

Third, preferential use of cutting balloons might be regarded as a routine strategy in the setting of type-2 and type-3 SCADs [5]. Technically, cutting balloons potentially allow drainage of intramural hematoma [1] into the true lumen (through creation of intimal microfenestrations), and generally obviates the need for subsequent stent implantation with life-time complication risks in this young population [5]. This is of paramount importance particularly in the setting of type-2 SCAD that requires long or multiple stent implantations.

Finally, severe degrees of myocardial wall motion abnormalities (including akinesia, etc.) might potentially be attributable to post-ischemic myocardial stunning [3] that might persist for variable durations even after complete recovery of SCAD, yet eventually vanishes in time. Therefore, assessment of myocardial viability with nuclear imaging, etc. might differentiate between myocardial necrosis and reversible stunning in severely affected myocardial segments associated with SCAD, and might help guide the subsequent management strategies (including decision-making for cardiac device implantation).
More interestingly, takotsubo syndrome (TTS) might potentially co-exist with SCAD mostly due to the common trigger of these entities including severe stressors [3]. Moreover, this co-existence might have prognostic and therapeutic implications (attributable to more severe adrenergic discharge, etc.) [3]. For instance; SCAD in association with TTS might be particularly prone to vascular complications including dissection propagation, and hence; warrants early intervention even in the absence of high-risk features [3].

In summary, SCAD management might be regarded as a multi-faceted phenomenon. Notably, meticulous evaluation of clinical details might significantly impact the management and prognosis of patients with SCAD.

REFERENCES


