Spontaneous coronary artery dissection: practical considerations in management

Kenan Yalta[1], Gokay Taylan[1], Tulin Yalta[2], Ertan Yetkın[3]

[1]Trakya University, Cardiology Department, Edirne, Turkey
[2]Trakya University, Pathology Department, Edirne, Turkey
[3]Derindere Hospital, Cardiology Department, Istanbul, Turkey

Correspondence to:
Kenan Yalta, MD, Trakya University, School of Medicine, Cardiology Department, Balkan Yerleşkesi, 22030, Edirne, Turkey, phone: +90 505 657 9856, e-mail: kyalta@gmail.com

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TO THE EDITOR

Over recent years, spontaneous coronary artery dissection (SCAD) has gained a substantial reputation as a specific form of coronary artery disease affecting mostly young females [1–3]. Pathologically, it is defined as a separation between the intima and media layers in epicardial coronary arteries (mostly the 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 a total occlusion (type-4) [1]. In their recently published expert opinion paper, Kądziela et al. [1] have presented a didactic overview of current information on SCAD and its management. In this context, we would like to place more 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 case of high-risk features, including hemodynamic instability, malignant arrhythmias, and persistent ischemia [1, 2]. However, the existing high-risk anatomy features (for instance, SCAD involving the 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 an admission) might arise as a viable option, particularly in the case 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 it 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 the proximal aorta during coronary interventions [4] or spontaneously. Notably, the absence of atherosclerosis generally facilitates retrograde SCAD propagation [2], potentially leading to an extensive aortic involvement. Importantly, patients with SCAD involving the LMCA or the osteal 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, the use of glycoprotein IIb/IIIa inhibitors or thrombolytic therapy in SCAD patients with a missed aortic dissection might result in catastrophic complications (including an aortic rupture). Taken together, a high index of suspicion for aortic involvement is mandatory in the setting of SCAD, particularly involving the LMCA or the osteal RCA.

Third, preferential use of cutting balloons might be regarded as a routine strategy in the case of type-2 and type-3 SCADs [5]. Technically,

cutting balloons potentially allow drainage of intramural hematoma [1] into the true lumen (through the creation of intimal microfenestrations), and generally obviate the need for subsequent stent implantation with lifetime complication risks in this young population [5]. This is of paramount importance particularly in the case 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 and eventually vanish in time. Therefore, an assessment of myocardial viability with nuclear imaging, etc. might differentiate between myocardial necrosis and reversible stunning in severely affected myocardial segments associated with SCAD. It might help guide 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 it warrants an 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.

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