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EDITORIAL COMMENT

Management of Iatrogenic Coronary Artery Dissections



Failing to Prepare Is Preparing to Fail*

David L. Fischman, MD, Alec Vishnevsky, MD

Catheter-induced coronary artery dissection (CICAD) is an infrequent but potentially catastrophic complication of coronary angiography and percutaneous coronary intervention (PCI). Distinct and clinically separate from spontaneous coronary artery dissection, CICAD is characterized by mechanical disruption of the endothelial cell layer of a coronary artery followed by extravasation of blood into subendothelial tissue planes. Depending on coronary artery perfusion pressures and the integrity of the external elastic lamina, the dissection can remain localized or propagate extensively in an antegrade or retrograde manner. In the era of balloon angioplasty, the National Heart, Lung, and Blood Institute (NHLBI) developed a classification system for intimal tears occurring during angioplasty that has since been adopted to characterize iatrogenic dissections. The spectrum of angiographic appearance of the various dissection morphologies described ranges from simple radiolucency during angiography (type A) to total occlusion of the coronary artery without antegrade flow (type F) (1).

The clinical incidence of CICAD has been estimated to be <0.1%, although the true incidence is likely underreported (2,3). In 1 retrospective 10-year cohort study of 56,968 patients undergoing coronary angiography, catheter dissections occurred more frequently in the right coronary artery (50%) than the

left main artery (45%), with the majority of these caused by guiding catheters. Most were managed with stenting (82%) or conservative therapy (12%), with only 6% requiring surgery (4). The long-term prognosis was favorable, with 94% surviving to discharge. In a registry of 17,225 patients undergoing PCI, CICAD occurred in 185 (1.1%) patients, which gradually decreased in the 10-year study timeframe despite the fact that the complexity of the disease increased (5). The most important determinant of clinical outcome in this study was antegrade perfusion. In those with impaired perfusion, in-hospital adverse events were more than 4-fold higher compared to those without CICAD.

CICAD occurs more frequently during PCI because of the use of larger-caliber catheters and manipulation of coronary guidewires and other interventional equipment within the coronary artery. Additional risk factors for CICAD include female sex, left main atherosclerosis, use of Amplatz-shaped catheters, complex lesions, and deep seating of the catheter within the artery (2). In general, the 2 most important factors determining prognosis after CICAD include the size of the compromised vessel and the integrity of antegrade flow (5,6). Minor dissections may not require treatment, but those that are long or result in >50% stenosis with impaired Thrombosis In Myocardial Infarction flow in a vessel of >2.5 mm are considered severe and should be treated (6).

Integral in the management of significant dissections is obtaining wire access to the true lumen during diagnostic angiography and maintaining wire access across the dissection entry site during PCI. Crossing or recrossing the dissected segment can be accomplished with a soft workhorse guidewire with a low tip load (6,7). Such wires provide enhanced tactile and visual feedback that increase chance of successfully finding the true lumen. It is imperative

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to avoid creating new dissection planes or causing acute vessel occlusion when attempting to gain wire access to the true lumen. In this context, intravascular ultrasound can visualize the dissection entry point, confirm the presence of the wire in the true lumen, and provide useful information regarding dissection length and vessel diameter that can guide stent selection (8). Other strategies to confirm wire location in the true lumen include use of optical coherence tomography and injection of contrast through a microcatheter or over-the-wire balloon. If antegrade wiring cannot be readily accomplished, other techniques to gain access to the true lumen have been described, including retrograde crossing of collateral channels, subintimal re-entry, and limited antegrade subintimal tracking (7).

Although there is limited evidence in published reports guiding stenting strategies in CICAD, a typical approach is to stent from the dissection entry to an area of normal vessel, thereby sealing the dissection and compressing any existing intramural hematoma. If the dissection is extensive and the target vessel is tortuous or calcified, it could be difficult to deliver a sufficiently long stent. Furthermore, if the distal vessel is not readily visualized because of a compressive intramural hematoma, it could be difficult to determine the appropriate stent length. One strategy to reduce the hematoma to provide adequate visualization of the distal vessel is the use of a cutting balloon at the site of the hematoma, although this carries the risk of further propagation. Before the advent of stenting, acute vessel reocclusion rates with various types of dissections ranged from 3.1% for type B dissections to as high as 67% for type F dissections and 37% overall (1). That this rate has decreased to <2% in the modern stent era is a testament to the importance of deploying stents to prevent subsequent acute vessel occlusion (5).

In this issue of *JACC: Case Reports*, Sumiyoshi et al. (9) highlight an important strategy for the management of an occlusive dissection and present a novel application of microcatheters to reduce an obstructive hematoma. The authors describe the successful use of a 2.3-F microcatheter (Transit-II, Johnson and Johnson, Bridgewater, New Jersey) to aspirate blood from the subintimal space to allow for visualization of the distal vessel. In this case, a stent was deployed at low pressure (8 atm) to seal the proximal dissection entry site, and the microcatheter was used to aspirate the residual intimal hematoma and establish

Thrombolysis In Myocardial Infarction flow grade 3. The stent was then post-dilated successfully to high pressure. Sumiyoshi et al. thus illustrate a technique to effectively re-establish distal flow in dissections complicated by severe compressive hematomas, minimizing the need for extensive stenting beyond the original dissection site.

The most important aspect of CICAD, however, is in its prevention, and the key to prevention is understanding modifiable risk factors and the use of prophylactic measures to mitigate the risk. A meticulous catheterization technique should be used. This includes utilizing appropriately sized and shaped catheters without deep intubation of the vessel, avoidance of noncoaxial alignment of the catheter, and not injecting if the pressure waveform is dampened or at an atherosclerotic plaque. Some risk factors, such as presentation of acute myocardial infarction or presence of a complex lesion, are not modifiable. In either case, awareness of risk and prompt recognition of the presence of a dissection will increase the speed at which definitive therapy can be delivered. In this context, it is imperative to distinguish CICAD from other look-alikes on angiography. These include contrast streaming, wire- or guide-mediated straightening of the vessel, overlapping of a radio-opaque portion of a coronary guidewire, and the presence of a small branch overlying a major artery (2). A true dissection will appear as a persistent intraluminal smooth flap or extraluminal linear or spiral extravasation of contrast media with or without diminished antegrade flow.

In summary, CICAD is a rare but important complication of coronary angiography and PCI. The current report by Sumiyoshi et al. (9) provides a novel strategy to consider while managing CICAD. Careful attention to preventative techniques and knowledge of effective treatment strategies will prepare operators well and facilitate successful outcomes in this potentially life-threatening complication.

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