Spontaneous Coronary Artery Dissection: A Call for Consensus and Research Advancement*

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Recognition of spontaneous coronary artery dissection (SCAD) as an important cause of acute myocardial infarction (AMI), particularly in younger women without traditional atherosclerotic risk factors, is increasing. The number of publications listed in PubMed using the keywords “spontaneous coronary artery dissection” has increased from 13 in 2000 to 129 in 2018. In 2018, the American Heart Association (AHA) and the European Society of Cardiology (ESC) separately released expert consensus statements on SCAD (1,2), and in 2019, the results of a large multicenter, prospective registry of SCAD patients were reported (3). Despite the recent gains in knowledge regarding the presentation, associated vascular disorders, and natural history of SCAD, both the AHA and ESC SCAD documents (1,2) list the study of optimal initial revascularization strategies in AMI due to SCAD as an urgent research priority.

In this issue of the Journal, Lobo et al. (4) sought to understand the role of urgent revascularization in patients with ST-segment elevation myocardial infarction secondary to SCAD (STEMI-SCAD) and to determine differences in outcomes compared with patients who receive revascularization in ST-segment elevation myocardial infarction secondary to atherosclerosis (STEMI-ATH). Accessing databases from 2 large STEMI centers, which collected data for all STEMI activations over a span of 14 years (2003 to 2017), a cohort of 4,245 STEMI-ATH and 53 STEMI-SCAD patients was created. SCAD was the cause of STEMI in 1% of this population but 19% in women ≤50 years of age with STEMI. In line with previous reports, STEMI-SCAD patients were more commonly female, younger, and with fewer atherosclerotic risk factors. Additionally, despite higher rates of left main and left atrial descending artery involvement, higher rates of cardiogenic shock, and longer lengths of stenting required for revascularization, Thrombolysis In Myocardial Infarction flow grade 3 was restored in 91% of STEMI-SCAD patients who underwent percutaneous coronary

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intervention (PCI). When compared with age- and sex-matched STEMI-ATH patients, STEMI-SCAD patients had higher 3-year survival rates (98% vs. 72%) with no STEMI-SCAD deaths occurring after hospital discharge.

Cardiogenic shock was present in 10 (19%) STEMI-SCAD patients: 7 had left main involvement, 3 required coronary artery bypass grafting, and 4 required mechanical support. Four of the patients with left main involvement had pregnancy-associated spontaneous coronary artery dissection (P-SCAD), which is consistent with prior work demonstrating higher rates of STEMI presentation, left main and multivessel SCAD, and lower ejection fraction among P-SCAD (5). These findings support the notion that some SCAD patients, including those with P-SCAD, are at risk for severe clinical courses and represent a subset of patients for whom emergent invasive strategies may be critical despite the increased procedural risk. Such risk includes potential downstream dilemmas. For instance, at 1-year follow-up in the Lobo et al. (4) cohort, 9% of patients treated with stents had in-stent restenosis, and 40% of these were recurrent episodes of restenosis. One patient presented in follow-up with stent thrombosis of the left main and left anterior descending stents ascribed to stent malapposition likely following hematoma resorption. Although these challenges do not negate the role for revascularization when deemed necessary, they illustrate the unique aspects of SCAD to be considered during acute and future management strategies.

The reported PCI success rate of 91% is on the high end of previously published estimates ranging from 29.1% (3) to 92.3% (6), an uncomfortably large range for what would seem to be a fundamental question: “did the intervention work?” Potential causes for the variability in reported success rates likely reflect patient-, disease-, and intervention-related factors; however, some variability is also because we are not “speaking the same language.” For instance, Saw et al. (3) recently reported a PCI success rate of 29.1% in 106 SCAD patients undergoing PCI. However, 40.8% of these were classified as “partial success.” When comparing definitions for PCI success, the “partial success” definition used by Saw et al. (3) fits within the Lobo et al. (4) definition of PCI “success,” such that the comparable PCI success rates from the Saw study would be 70% instead of 29%. The variability in reported PCI success rates emphasizes the importance of further terminology refinement for the study of SCAD in general. For example, Lobo et al. (4) state that the current SCAD classification scheme (7) does not encompass all angiographic findings observed in STEMI-SCAD. However, other classification schemes have been proposed (8,9), and the ESC SCAD document (2) includes a “type IV” SCAD, which is used to describe total occlusion of a distal vessel. Consensus on terminology will be paramount to advancing SCAD research. Although the AHA (1) and ESC (2) SCAD documents laid the foundation, it is evident that more work is necessary.

Lobo et al. (4) have taken a systematic approach at identifying consecutive cases of STEMI due to SCAD, and this type of methodology overcomes 1 major limitation of other published registry data: referral bias toward those who survive their initial event and are well enough to be enrolled in outpatient registries. The approach taken increases the likelihood of capturing the more severe presentations and adverse acute outcomes from STEMI-SCAD. However, this study suffers from the same major limitation that plagues several of the published comparative, retrospective SCAD studies that have come before it—the lack of systematic
review of all AMI coronary angiograms for the presence of SCAD. As evidenced by the
temporal data demonstrating increased prevalence of STEMI-SCAD over time (0.2% from
2003 to 2007 vs. 1.5% from 2013 to 2017), the recognition of the angiographic appearance
of SCAD has increased. The increased angiographic recognition of SCAD is due to several
factors: the use of intracoronary imaging, such as intravascular ultrasound and optical
coherence tomography that can more clearly differentiate type 2 SCAD (intramural
hematoma) from atherosclerosis; a common classification system for SCAD allowing for
enhanced communication and education (7) among health care providers; recognition of
coronary artery tortuosity (10), which may prompt a closer review of the angiogram for
subtle SCAD findings; and increased awareness of SCAD as a cause of AMI in general.
Retrospective review of 14 years of coronary angiograms is expensive, time-consuming, and
impractical, but the methodology to determine the prevalence of AMI from SCAD employed
by this paper and others before it likely significantly underestimates the burden of SCAD as
a cause of myocardial infarction.

Underestimation of disease prevalence carries with it a cascade of potential downstream
effects, including delayed development of adequate treatments and resources allotted for
disease prevention. For this reason, systematic, large-scale, prospective methodologies must
be adopted to adequately describe the population prevalence of SCAD. One relatively
obvious means for doing this is to incorporate SCAD as a cause of AMI, along with SCAD-
related angiographic variables, into existing national outcomes-based cardiovascular patient
registries, such as the CathPCI Registry contained within the National Cardiovascular Data
Registry (11). Although more granular data about patient characteristics and coprevalent
vascular diseases can be well-delineated from smaller SCAD-specific registries, national
databases where SCAD is a recognized cause of AMI will be necessary to adequately
describe the burden of disease.

Finally, although the work by Lobo et al. (4) includes a large number of STEMI patients,
only 53 of these patients were STEMI-SCAD patients. Because SCAD is still considered to
be uncommon, larger-scale, multicenter efforts must be supported to recruit an adequate
number of patients at a pace that will meaningfully change practice and improve outcomes.
Eventually, and hopefully soon, SCAD research must transition from retrospective reviews
and patient registries to clinical treatment trials.

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