LETTERS TO THE EDITOR

Fibromuscular Dysplasia and Spontaneous Coronary Artery Dissection

Coincidental Association or Causality?

We read with great interest the elegant study by Saw et al. (1) that carefully described predisposing conditions in a large Canadian series of 50 patients with spontaneous coronary artery dissection (SCAD). Notably, fibromuscular dysplasia (FMD) in noncoronary territories was found in 86% of patients. Due to the exhaustive screening for associated conditions and the potential clinical implications of these results, additional information on the study findings would be of major interest. First, in this series, revascularization was only needed in 18% of patients and no patient died during hospitalization. This excellent clinical outcome with a conservative approach corroborates our previous experience (2) and suggests that a systematic watchful waiting strategy is reasonable for “stabilized” patients with SCAD (2). A conservative strategy is further supported by the evidence of dissection healing at late follow-up (2) that was also confirmed in the present study (1). This issue is important as revascularization in these patients may be challenging and associated with suboptimal results (2–4). In this regard, information on whether some SCAD patients were excluded from the study and additional data on major events during late follow-up would be highly reassuring. Second, in this series, post-partum SCAD was only found in 1 patient and associated systemic inflammatory conditions were also anecdotal. This is in contradistinction with some previous reports, but in agreement with our experience where associated inflammatory/immunologic diseases were rarely found despite a systematic screening (2). Finally, the most provocative finding of the study is the striking (nearly universal) association with FMD in large arteries. This association, initially described by these investigators, has also been confirmed by other groups (4,5). Interestingly, the presence of FMD may actually orientate toward a diagnosis of SCAD in patients with “ambiguous” coronary angiograms (6). In the study by Saw et al. (1), data on renal, iliac, and cerebrovascular involvement was detailed and it was also specified that 76% of patients had renal artery catheter angiography. Although the superior diagnostic accuracy of angiography over noninvasive imaging techniques for the identification of FMD was suggested, additional data on the yield of the different techniques (angiography vs. others) in the diverse vascular territories would be of upmost importance and helpful to shape future research efforts. We concur with the investigators’ proposal that the striking prevalence of FMD in their patients with SCAD suggest causation. This persuasive hypothesis should be further investigated to unravel the elusive predisposing underlying pathologic substrate of this challenging clinical entity (7).

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Reply

We thank Dr. Alfonso and colleagues for their kind letter in response to our paper (1). We agree that revascularization for patients with spontaneous coronary artery dissection (SCAD) is challenging and often terminates with suboptimal results, and thus we have adhered to a conservative approach unless patients have ongoing or recurrent ischemia. Indeed, repeat angiography in a subset of patients treated conservatively invariably demonstrated spontaneous healing of dissections (1). Our cohort included all nonatherosclerotic SCAD cases identified and evaluated in our center (no patient was excluded). We recently reported good long-term outcomes in our expanded cohort of 86 SCAD patients at the 2013 American College of Cardiology meeting, despite our low revascularization rate (2). Our results, together with your reported cohort of 45 SCAD patients in Spain (3), confirm that the natural history of SCAD is such that the vast majority heals spontaneously. Thus, clinicians should avoid intervention based purely upon the “oculostenotic reflex” in the absence of ischemia.