

ACAOS Is Better Revealed by Intravascular Ultrasonography than by Computed Tomographic Angiography

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In their case report,¹ Yildiz and co-authors present a typical example of anomalous origin of a coronary artery from the opposite sinus of Valsalva (ACAOS). This abnormality can affect either the right coronary artery (R-ACAOS) or the left coronary artery (L-ACAOS).² Our understanding of this entity is finally reaching maturity. In 2007, ACAOS was redefined as an anomaly that involves a variably located coronary artery ostium but always has an intramural course inside the aortic wall, with some degree of accompanying stenosis.³ The mechanism of stenosis and the clinical consequences are related to entrapment of the proximal coronary artery inside the aortic tunica media, with resulting hypoplasia and lateral compression.³

Anomalous origin of a coronary artery can also occur without an intramural course. In such cases, the course can be preaortic (in front of the pulmonary infundibulum), intraseptal (infundibular, inside the upper ventricular septum), or retroaortic. In these cases of alternative ectopic origination, the proximal ectopic segment of the involved coronary artery is usually not stenotic.² This characteristic has been clarified through the use of systematic, in vivo intravascular ultrasonography (IVUS).

In ACAOS, the passage of a coronary artery inside the aortic wall becomes complicated by dynamic structural change (lateral compression). The compression is worse during systole and tachycardia, because the systolic time interval lengthens. As the results of catheter coronary angiography (CCA) and coronary computed tomographic angiography (CCTA) show, the intramural course in typical ACAOS is accompanied by a preaortic passage of the ectopic artery in front of the anterior commissure of the aortic valve, at the level of the sinotubular junction.⁴ In the hands of expert investigators, CCA and CCTA are reliable techniques for diagnosing ACAOS; however, they cannot clarify the location (intramurality) of the proximal coronary trunk or quantify its severity of stenosis.⁴ Conversely, IVUS always shows the intramural course and the accompanying stenosis. In the case of L-ACAOS described by Yildiz and colleagues,¹ Figure 2A cannot be interpreted as evidence that the course is intramural versus “between the aorta and pulmonary artery.” However, the image does, indeed, show lateral compression of the crossing left coronary artery (LCA) at the aortopulmonary septum. The longitudinal diameter of the artery is about 3 times the transverse diameter, and stenosis is most likely present. In comparison with the distal reference cross-section (the authors’ Fig. 2B), Figure 2A suggests a stenosis that involves 50% to 75% of the cross-sectional area during diastole.

By means of its in vivo, high-definition, moving images, only IVUS can clearly quantify the severity of stenosis at the LCA proximal trunk, even though elaboration of the images might be delicate and definite cutoff values for recommending intervention have not yet been determined.²

Although IVUS is not a simple, routine procedure in the catheterization laboratory, its performance during coronary angiography, especially in patients with L-ACAOS, is becoming integral to the evaluation of ACAOS at expert centers.⁵ As Yildiz and co-authors recognize, IVUS is necessary to improve our understanding of the pathophysiology of ACAOS and to determine criteria that can guide clinicians in handling each presentation of these difficult cases.

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