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CAN WE TALK? THE RESIDUAL QUESTIONS ABOUT SURGERY FOR CORONARY ARTERY ANOMALIES

To the Editor:

This letter refers to an important recent review by one of the most active surgical centers for treating coronary artery anomalies (CAAs),[1] which our Center believes calls for an open discussion from an alternative adult-cardiology source. The comprehensive and welcome report by Bonilla-Ramirez and colleagues,[1] which describes their experience with 71 consecutive pediatric patients during 2012-2019, is illuminating in stating their current rationale and practices. Our group would like to add essential but missing important aspects of related current investigations by supplying alternative viewpoints and diagnostic techniques.[2,3]

GENERAL ISSUES

The authors describe their experience with all the CAAs of origin that they corrected surgically. Their implication seems to be that any kind of anomaly of the right and left coronary arteries are part of the same anatomic, functional, prognostic, and surgical entity, whereas we believe that their features (especially mortality) are substantially different and must be reported and discussed separately by specific anomaly type. We now know that approximately 1,300,000 people are born with certain, specific kinds of anomalies (anomalies of origin and of intramural course), and the full plan of action must account for this common denominator (intramural course with dynamic lateral compression and variable stenosis). Initially, adequate screening is generally necessary in high-risk populations like athletes.[4,5]

Incidentally, we and many other authors use currently a more refined and expressive nomenclature for this pathology than the generic term "anomalous aortic origin of a coronary artery." We believe that any CAA can be described as anomalous right or left (R- or L-) coronary artery origin from the opposite or improper sinus (ACAOS) with the addition of a term describing its course from the ectopic origin to the dependent territory (intramural [IM] is the one the authors apparently included; the others are prepulmonary, intraseptal, and retroaortic), which individually identifies the implied mechanism of possible dysfunction. The authors[1] do not even report the incidence of R-ACAOS-IM and L-ACAOS-IM cases separately, with their specific features, which is basic identifying information for anatomy, function, and prognosis.

DIAGNOSIS

The preoperative diagnostic evaluation of ACAOS should identify not only its general type (qualitative) but also the individual case's quantitative stenotic severity. Echocardiography, computed tomography angiography (CTA), and coronary catheter angiography cannot assess stenosis severity reliably with respect to the distal refence vessel, which is the fundamental reason to surgically treat ACAOS cases. The only precise methods for quantitative evaluation are intravascular ultrasonography (IVUS) and optical coherence tomography, which are not used routinely by many pediatric cardiology centers, even those that specialize in CAA surgery.

Both symptoms and stress testing are neither precise nor sensitive. Magnetic resonance angiography and CTA are adequate for qualitative diagnosis of ACAOS but not for quantitative study of severity in individual cases. In particular, CTA is limited to end-diastole, when the severity of stenosis is lowest during the cardiac cycle, in these dynamic IM entities; also, exercise increases the severity substantially by increasing cardiac output, stroke volume, and systolic time (leading to maximal functional stenosis, which varies also with aortic root elasticity). Dobutamine testing has not been approved in cases of ACAOS-IM, especially in the absence of volume expansion and exertional tachycardia, as it is in the saline–atropine–dobutamine test[2,3] (sometimes performed in the catheterization laboratory, under IVUS monitoring). Also, adenosine stress testing (a study of vasodilatory capacity at rest) is not useful for diagnosing ACAOS-IM severity, as it does not substantially increase cardiac output, blood pressure, or heart rate to simulate exercise conditions. By IVUS imaging, stenosis severity at rest in cases of ACAOS-IM (initially qualitatively diagnosed by CTA) varies between 20% and 90% (or 30%-100% with saline–atropine–dobutamine testing, in systole). Such diagnostic evidence is an essential indication for surgical intervention.

TREATMENT

The authors' experience[1] apparently depends frequently on surgical findings, which unfortunately cannot be used to establish cross-sectional stenosis objectively (which requires the distal reference cross-sectional area) to serve

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as a valid parameter for surgery. As a consequence, for example, 37% of initial unroofing cases were eventually changed to ostial reimplantation. Especially in cases of R-ACAOS-IM in older patients, IVUS-guided stent angioplasty (not mentioned in the article by Bonilla-Ramirez and colleagues, but widely reported) is probably a much simpler, safer, and more reliable solution. Also, during the IVUS-monitored procedure, the results are confirmed immediately after stenting. Obviously, the utility of stent treatment must be evaluated in prospective, controlled studies at coordinated and dedicated centers of excellence. Late restenosis when using drug-eluting stents is less in R-ACAOS-IM than in coronary atherosclerotic disease and is not accompanied by late lateral stent compression, as reported experience of our 50 cases suggests.[2]

I hope these short notes, proposed in the spirit of information-sharing and professional collaboration, can promote a more logical and effective treatment paradigm to treat ACAOS-IM.

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