Cardiac Computed Tomography to Visualize Typical Variants of the Right-Sided Origin of the Left Main Coronary Artery

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LETTERS TO THE EDITOR

Impact of Additional Ballooning on Plaque Prolapse After Stent Implantation in Patients With Acute Myocardial Infarction

With great interest, we read the article by Hong et al. (1), in which they examined the incidence, predictors, and outcome of plaque prolapse after stent implantation in acute myocardial infarction. We have reported the incidence, diagnostic characteristics, and clinical features of plaque prolapse in 303 patients undergoing nonemergent stent implantation (2). In a subgroup analysis, prolapse areas did not differ significantly after additional ballooning from those immediately after stent implantation. It would be of great help if the authors would provide intravascular ultrasound data before and after additional ballooning to clarify the behavior of plaque prolapse after stent implantation in acute myocardial infarction.

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Please note: Dr. Fitzgerald has received a research grant and is on advisory boards for Cordis Johnson & Johnson.

REFERENCES


REPLY

In our study (1), we did not perform additional ballooning for the plaque prolapse (PP) lesions routinely. Instead, we performed additional ballooning when we observed stent underexpansion after stent implantation. Among a total of 85 PP lesions, additional ballooning was performed in 30 lesions. The PP areas did not differ significantly after additional ballooning from those immediately after stent implantation (maximum PP areas, 0.8 ± 0.6 mm² vs. 0.7 ± 0.5 mm², p = 0.5, and PP volumes, 2.6 ± 2.0 mm³ vs. 2.3 ± 1.7 mm³, p = 0.4, respectively). However, when we compared 30 PP lesions with additional ballooning with 55 PP lesions without additional ballooning, there was a trend that cardiac enzymes were increased more significantly in the additional ballooning group compared with the nonadditional ballooning group (Δcreatinine kinase-myocardial band, +14.5 ± 39.3 U/l vs. +10.5 ± 28.9 U/l, p = 0.092, and Δcardiac troponin I +19.8 ± 63.4 ng/ml vs. +14.5 ± 46.3 ng/ml, p = 0.088, respectively). The incidence of stent thrombosis was not significantly different between the additional ballooning group and the nonadditional ballooning group during 1-month clinical follow-up (1 of 30 [3.3%] vs. 1 of 55 [1.8%], p = 0.8). Therefore, our results suggest that it is not necessary to perform additional ballooning to reduce PP areas when we observe PP after stent implantation in patients with acute myocardial infarction because there are no differences in PP areas and the risk of stent thrombosis, with a strong trend toward more significant myonecrosis in PP lesions with additional ballooning compared with PP lesions without additional ballooning.

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Astonishingly, Ropers et al. (1) begin their excellent imaging vignette describing various right-sided origins of the left main coronary artery with the statement that anomalous right coronary arteries (ARCA) emerging from the left sinus, unlike anomalous left coronary arteries arising from the right sinus, are considered harmless, despite the fact that their own second reference (2) documents 4 of 27 sudden deaths (confirmed at autopsy by Basso et al. [2]) because of ARCA from the left sinus.

Moreover, there are numerous reports that ARCAs originating from the left sinus are uncommon but may lead to sudden

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death due to a variety of reasons, such as a slit-like ostial opening, acute take off at the origin, an intramural course within the aortic wall leading to mechanical/dynamic compression, interarterial course between the aorta and pulmonary artery and, in older patients, atherosclerotic stenosis (3–7). The management of symptomatic and hemodynamically significant ARCA arising from the left sinus is still undefined (8,9).

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REFERENCES


REPLY

We very much welcome the response by Dr. Natarajan to our report (1) concerning the computed tomography (CT) appearance of anomalous left main coronary arteries. He voices his surprise that we did not classify anomalous left-sided right coronary arteries as harmful. Indeed, there is a remarkable divergence of opinions concerning the relevance of a left-sided origin of the right coronary artery with subsequent passage between the aorta and pulmonary artery. In an excellent review article concerning this issue, Gersony (2) provides a thorough review of the available data. Because anomalous right coronary arteries are substantially more frequent than left coronary arteries with an anomalous origin, many large series failed to demonstrate an association of anomalous right coronary arteries with sudden death (in a study with 6.3 million individuals followed, there were 21 exercise-related sudden deaths in patients with an anomalous left coronary artery but not a single one associated with the much more prevalent situation of an anomalous right coronary artery [3]), and a recent report describes the frequent occurrence of ischemia after surgical repair of previously asymptomatic anomalous right coronary arteries (4), he makes a convincing argument toward the benign nature of anomalous right coronary arteries for which he does not recommend any treatment, as long as the patients are asymptomatic. Symptomatic patients with signs of ischemia clearly require a different approach, and we should probably have made this distinction more clearly in our brief article.

We welcome this discussion, because coronary CT angiography frequently reveals coronary variants. In all cases, the potential associated dangers need to be weighed against the risk of treatment: first, do no harm. Complications after treatment of coronary anomalies are not zero, and it is imperative that we do not recommend therapies that cause more problems than the entity they are intended to cure.

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REFERENCES

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