

Reply to the Letter to the Editor: "Ambiguity of Medina 0.0.1 Bifurcation Disease Without Intravascular Imaging"

To the Editor,

We thank the authors¹ for their interest in our study² and for their thoughtful comments.

First, we fully agree that intravascular imaging provides valuable information in the assessment of bifurcation lesions, particularly in Medina 0.0.1 disease. As the authors correctly emphasized, angiography may underestimate lesion extent because of vessel overlap, foreshortening, eccentric plaque distribution, and the presence of subclinical plaque extension into the main vessel. For this reason, the absence of intravascular imaging was explicitly acknowledged as one of the major limitations of our study. However, our investigation was designed to reflect real-world practice in a retrospective cohort and to evaluate the procedural safety and feasibility of a DCB-only strategy rather than to establish definitive anatomical characterization. Therefore, our conclusions were intentionally limited to safety and feasibility and should be interpreted within this context.

Second, regarding ostial LCX lesions, we acknowledge the possibility of plaque extension from the left main coronary artery into the LCX. Nevertheless, all angiograms were reviewed by experienced interventional cardiologists, and lesions were classified according to angiographic Medina criteria. Furthermore, the primary objective of the present study was not to determine plaque distribution but to evaluate clinical and procedural outcomes following DCB treatment in lesions considered suitable for a DCB strategy in routine clinical practice. We agree that future studies incorporating systematic IVUS or OCT assessment would provide a more accurate understanding of plaque extension and lesion morphology.

Third, regarding lesion preparation, all lesions underwent mandatory predilatation before DCB application. Drug-coated balloon treatment was performed only when accepted angiographic criteria were achieved, including TIMI 3 flow, residual stenosis $\leq 30\%$, and absence of severe dissection. Because of the retrospective nature of the study and the relatively small sample size, detailed data regarding the use of adjunctive lesion preparation devices, such as cutting balloons, scoring balloons, or atherectomy systems, were not consistently available and therefore were not included in the analysis. We agree that these devices may be particularly valuable in selected calcified ostial lesions and require further investigation in future prospective studies.

In conclusion, we appreciate the authors' comments, which highlight important considerations regarding lesion assessment and preparation in Medina 0.0.1 bifurcation disease. We believe our findings should be viewed as hypothesis-generating and supportive of further prospective studies utilizing routine intravascular imaging and detailed lesion characterization.

LETTER TO THE EDITOR REPLY

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Available Online Date: August 3, 2026

Cite this article as: Akın H, Bilge Ö, Işık F, et al. Reply to the Letter to the Editor: "Ambiguity of Medina 0.0.1 Bifurcation Disease Without Intravascular Imaging." *Anatol J Cardiol*. 2026;XX(X):X-X.

DOI: 10.14744/AnatolJCardiol.2026.6760



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Declaration of Interests: The authors have no conflict of interest to declare.

Funding: The authors declared that this study has received no financial support.

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