Correspondence

Letter by Bourantas et al Regarding Article, “Nonculprit Plaques in Patients With Acute Coronary Syndromes Have More Vulnerable Features Compared With Those With Non–Acute Coronary Syndromes: A 3-Vessel Optical Coherence Tomography Study”

To the Editor:

We have read with great interest the report by Kato et al1 on the comparison of nonculprit plaques in patients with and without acute coronary syndromes with regards to their prevalence and optical coherence tomography (OCT) characteristics. The authors for the first time demonstrated that the nonculprit lesions of the acute coronary syndrome patients had similar prevalence and distribution, but exhibited differences in the composition of plaque by OCT. However, in order to better understand their conclusions on the prevalence and distribution, the authors should report the length of the pullback and the localization of the imaged segments. There are 2 pieces of information that makes us think that the imaged length is limited: one is the use of balloon occlusion and second is the fact that the maximum length of the OCT pullback is 55 mm. In particular, the use of proximal balloon occlusion is a critical issue as this is positioned at the proximal segment of the coronary artery, which is also the most common location of vulnerable plaques. Stent implantation in the culprit lesion further shortened the imaged length. Thus, we wonder how much the reported data reveals the real prevalence of vulnerable plaques. The definition of plaque is not in line with the consensus report.2 The authors considered plaque to be those segments with 30% diameter stenosis compared to a reference segment (the latter is not defined in the report). Why did the authors use 30% and not any other cut-off? On these plaques, the cap thickness was measured, but the reproducibility numbers from measurements are not provided. This information is of utmost importance since in the consensus document it is mentioned that it is challenging to measure the fibrous cap.3

Plaque characterization using OCT is complex, panel A of Figure 1 is labeled as a thin-capped lipid-rich plaque, but we are not certain whether the arrows are pointing is a thin cap or an artifact called “tangential signal dropout.”5

Although the increased amount of macrophages reported in the acute coronary syndrome group is in agreement with the results derived by other imaging techniques, a significant pitfall of the current analysis is the methodology implemented to assess the presence of macrophages. In contrast to previous studies, which used a semi-automated methodology to quantify macrophages accumulation in the fibrous cap, in this report the identification of macrophages was based on visual inspection, a fact that is likely to introduce bias leading to erroneous estimations. The definition of microchannels in this analysis is under question. In histology reports, the microvessels have specific dimensions (50–300 μm), whereas previous OCT studies characterized microvessels only when these were detected in consecutive frames.3,5 These characteristics were not taken into account in the current analysis. Further, the lack of association between plaque characteristics and the amount of microchannels should be also attributed to the poor penetration of OCT in the lipid tissue, which does not allow visualization of the tunica media and the base of the plaque where neovascularure is prominent in unstable lesions.

Disclosures

None.

References


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