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Are Microcalcification and Hemosiderin Really Limitations of OCT in Detection of TCFA?

With great interest, we read the recent work by Fujii et al. (1) published in iJACC reporting the diagnostic accuracy of optical coherence tomography (OCT) and grayscale intravascular ultrasound for the detection of coronary thin cap fibroatheroma (TCFA) with histologic validation in human autopsy hearts. In this publication, Fujii et al. (1) documented a limited capability of OCT and intravascular ultrasound for TCFA identification when each imaging device was solely utilized. The authors attributed the low accuracy of OCT in TCFA detection to 4 reasons: presence of foam cell macrophage, microcalcification, hemosiderin accumulation, and organizing thrombus inclusive of fibrin. However, we have concerns about whether or not this finding is undoubtedly acceptable.

Firstly, as the authors admitted in the limitation section, there might be issues in co-registering OCT images to histology. From our experience of comparison between histology and OCT, the morphologies of coronary atheroma are divergent even with a minor deviation. Without serial histologic sectioning (by micron-level deeper cuts), it is difficult to achieve complete co-registration. Next, our previous report demonstrated that macrophage and fibrin accumulation exhibited resemblance to TCFA by signal analysis on OCT (2), therefore we agree that presence of macrophage and fibrin thrombus are the primary causes of misdiagnosis of TCFA by OCT (OCT-derived “pseudo” TCFA). However, it was doubtful that OCT could detect microcalcification and hemosiderin because they are subtle materials beyond the resolution limitation of OCT. It is often observed that apoptotic macrophage is involved in the formation of microcalcification (3). We therefore presumed that a cluster of foamy macrophages with or without adjacent microcalcification (calcified apoptotic macrophage) might cause signal interference on OCT, that is, the living macrophages, not microcalcification per se, show similar appearance to TCFA. One needs to do specific macrophage staining before a conclusion can be drawn regarding microcalcification, and signals for calcium should be of low signal intensity. Further, it is known that hemosiderin is frequently seen in the region of intraplaque hemorrhage as a result of phagocytosis and breakdown of hemoglobin by macrophages (hemoglobin-stimulated macrophages) (4). We speculated that the phagocytic macrophages or intraplaque hemorrhage, not hemosiderin per se, showed resemblance to TCFA on OCT. Hemosiderin particles are small at the level of 2 to 6 μm and therefore beyond the level of resolution of OCT. Nevertheless, clusters of hemosiderin may be visible, but without serial sectioning and staining for CD68 it is impossible to determine whether one is seeing hemosiderin within macrophages. The image of OCT the authors show does not match the location of hemosiderin in the histologic section shown; especially when the authors presented only 2 histologic sections showing presence of iron.

OCT has become a standard imaging device to support our daily percutaneous coronary intervention procedure because of its great ability for the recognition of detailed structures and qualitative components of the plaque, however, the observers need to interpret OCT with caution until histologic validation is further established by serial sectioning and the availability of multiple samples with similar findings.

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