segments, of which 72 (21%) were positive on LGE; the median number of inflammatory segments was 3 per patient. On CT, all patients also presented with positive LIE, with a median number of inflamed segments of 3 per patient. The iodine concentration and tissue density in nonenhanced segments were 17.4 ± 3.5 μg/cm^3 and 49 ± 17 HU, respectively. They increased to 28.9 ± 4.9 μg/cm^3 and 78 ± 19 HU in enhanced segments.

In patient-based analysis, the sensitivity of spectral CT was 100%. On a segment-to-segment comparison, the sensitivity of spectral CT was 82% (59 of 72 positive segments), its specificity was 99% (264 of 268 negative segments), its positive predictive value was 94% (59 of 63 segments), and its negative predictive value was 95% (264 of 277 segments). Finally, the overall accuracy of spectral CT was 95% (59 + 264)/340 compared with CMR. The interobserver variability was excellent, with a kappa value of 0.84.

The duration of the CMR examination was 41 ± 5 min, including 10 ± 2 min for LGE imaging and only 15 ± 4 min for CT examination, including 3 ± 1 min for LIE imaging. The median radiation dose due to spectral acquisition was 1.2 mSv (range 1.1 to 1.3), which is 4 times less than the dose received when undergoing coronary CT angiography.

In this proof-of-concept study, spectral CT thus appears to be a valid alternative to CMR for the evaluation of inflamed myocardium in acute myocarditis. Acute myocarditis may be easily misclassified as ACS. These results provide new perspectives because CMR remains of limited availability in some centers. The large availability of CT combined with the possibility to rule out ACS with coronary CT angiography during the same examination, makes it promising for refining acute myocarditis imaging.

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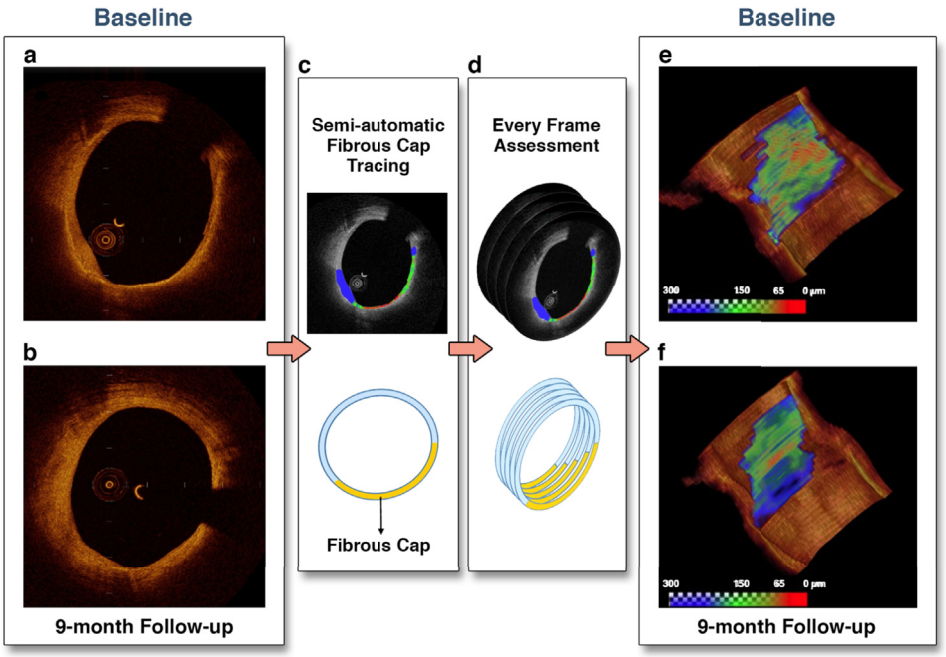
Fate of Nonculprit Plaques in Patients With STEMI Undergoing Primary PCI Followed by Statin Therapy

A Serial Optical Coherence Tomography Analysis From the OCTAVIA Study

In patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (p-PCI), the temporal effect of statin therapy on nonculprit lipid-rich plaques (NCLPs) is unclear. To better characterize the morphological changes of NCLP according to the intensity of statin therapy, we used serial optical coherence tomography (OCT) and a validated software that permitted semiautomated, comprehensive fibrous cap (FC) quantification at each point of its luminal boundary, with higher accuracy compared with individual measurement of arbitrary points.

This is a pre-specified analysis of the OCTAVIA (Optical Coherence Tomography Assessment of Gender Diversity in Primary Angioplasty) trial, a prospective multicenter study that evaluated the

FIGURE 1 OCT Semiautomated Method for Measuring Serial Changes of the FC and Analyzed Data



Optical coherence tomography (OCT) cross sections display the increased fibrous cap (FC) thickness from **(a)** baseline to **(b)** 9 months. **(c)** The semiautomated assessment of FC enables the quantification of its thickness in multiple points. **(d)** As FC measurements are performed at every frame, the measurement of FC surface area and volume can be obtained. The color-coded map (**red**: cap thickness <65 μm, **green**: 65 μm ≤ cap thickness <150 μm, and **blue**: 150 μm ≤ cap thickness <300 μm) demonstrates the differences in thickness and surface area of the entire FC and the changes of each individual component at fixed FC cutoff values between **(e)** baseline and **(f)** 9 months.

mechanisms of atherothrombosis in STEMI patients who underwent p-PCI (1). By protocol, patients underwent OCT of the infarct-related artery at baseline and at 9 months. Patients with OCT scans including the proximal and/or distal segments of implanted stents were included in the present analysis. Patients who at 9 months were on a high-dose statin regimen (defined as 80 mg/day [or 40 mg/day] of atorvastatin or 40 mg/day [or 20 mg/day] of rosuvastatin) were grouped into a high-intensity statin group, and those on <40 mg/day of atorvastatin, <20 mg/day of rosuvastatin, or different statins were grouped into a low-intensity statin group. Qualitative plaque assessment was performed by dividing each cross section into 4 quadrants and labeling each quadrant according to its most prevalent component, as follows: normal, fibrous plaque, calcified plaque, or lipid plaque. Lipid-rich plaque was defined as lipid that occupied ≥2 adjacent quadrants of the cross-sectional area, and thin-cap fibroatheroma (TCFA) was defined as lipid-rich plaque with a FC of ≤65 μm. Only nonculprit segments >10 mm in length with a FC thickness <300 μm were included in this study. Quantitative metrics of a FC covering NCLP were obtained using a semiautomated method (**Figure 1**) (2), and included FC thickness measured at all points on the FC boundary in all frames covering the lesion, FC surface area calculated as the product of the frame interval and arc length of the FC summed over the involved frames, and FC volume determined from cross-sectional FC surface areas in individual frames using the Simpson rule. Fractional FC surface areas were also measured based on threshold values (e.g., cap thickness <65 μm, 65 μm ≤ cap thickness <150 μm, 150 μm ≤ cap thickness <300 μm).

Overall, 108 of 140 patients in OCTAVIA fulfilled the entry criteria for the present analysis. At baseline, there were 72 NCLPs (66.7%). Of the 72 NCLPs, 33 (45.8%) were TCFAs. At follow-up, 72 patients (67%) were on a high-intensity statin regimen compared with 78 patients at baseline. Significantly greater changes in total and low-density lipoprotein

cholesterol were observed in the high-intensity statin group (−1.1 ± 1.1 mmol/l [interquartile range (IQR): −44 ± 44 mg/dl] vs. −0.4 ± 1.4 mmol/l [IQR: −17 ± 53 mg/dl]; p = 0.006, and −1.2 ± 1.0 mmol/l [IQR: −45 ± 40 mg/dl] vs. −0.6 ± 1.1 mmol/l [IQR: −24 ± 41 mg/dl]; p = 0.02, respectively). Compared with baseline, there was a decrease at follow-up in the proportion of lipid plaque components (11.9% [IQR: 5.0% to 22.9%] to 8.2% [IQR: 2.5% to 16.5%; p < 0.001), length of lipid plaque (3.4 mm [IQR: 2.2 to 4.9 mm] to 2.4 mm [IQR: 1.4 to 3.8 mm]; p < 0.001), maximum lipid arc angle (134.5° [IQR: 104.0° to 190.0°] to 94.0° [IQR: 79.0° to 128.3°]; p < 0.001), FC surface area (7.4 mm^2 [IQR: 4.0 to 13.7 mm^2] to 3.8 mm^2 [IQR: 2.1 to 7.6 mm^2]; p < 0.001) and FC volume (1.4 mm^3 [IQR: 0.7 to 3.0 mm^3] to 0.8 mm^3 [IQR: 0.5 to 1.7 mm^3]; p = 0.001), whereas the minimum FC thickness was significantly increased (66.0 μm [IQR: 51.0 to 83.3 μm] to 91.5 μm [IQR: 66.0 to 122.0 μm]; p < 0.001). The proportion of TCFA decreased significantly from baseline to follow-up in the high-intensity statin group (26.4% [n = 19] vs. 9.7% [n = 7]; p = 0.002) compared with the lower-intensity group (38.9% [n = 14] vs. 25% [n = 9]; p = 0.180). In addition, the proportion of TCFA in high-intensity statin group was significantly smaller than in lower-intensity group at follow-up (9.7% [n = 7] vs. 25% [n = 9]; p < 0.001). In 20 of 33 cases (61%), TCFAs observed at baseline were converted into non-TCFAs (i.e., FC >65 μm). FC surface area <65 μm further increased in 3% of NCLPs located distally to the implanted stent, compared with 16% of NCLPs located proximally. Patients were divided into 2 groups based on the statin intensity at 9 months. Status of statin intensity was changed in 12 of 108 (11%) patients during follow-up, and this should be acknowledged as a study limitation.

In STEMI patients undergoing p-PCI, most of the untreated TCFAs along the infarct-related artery vary into a more stable phenotype after 9 months of statin therapy, particularly when higher doses are used.

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