A "Stable" Coronary Plaque Rupture Documented by Repeated OCT Studies

A 78-year-old man with stable coronary artery disease and a history of percutaneous coronary stenting in the mid segment of the left anterior descending (LAD) artery for unstable angina was admitted to San Giovanni Addolorata Hospital. Coronary angiography and frequency domain-optical coherence tomography (FD-OCT) revealed a new significant stenosis distal to the stented segment (Fig. 1A). FD-OCT identified a ruptured plaque in the proximal LAD that was not associated with thrombus (1) (Figs. 1B and 1C). Because the minimum lumen area at the ruptured plaque site was large (7.75 mm²), the lesion was not stented. The patient was discharged with double antiplatelet drugs and a full dose of statin.

Six months later, the patient experienced exertional chest pain. Coronary angiography and FD-OCT showed severe intra-stent restenosis (Fig. 1D). The ruptured plaque in the proximal LAD, observed in the previous study, was again imaged by FD-OCT and showed the same morphologic features identified 6 months earlier (rupture without thrombus) and unchanged minimum lumen area (7.75 mm²) (Figs. 1E and 1F). To further investigate the features of the ruptured plaque, an offline OCT tissue property software (2,3) was applied, identifying a low OCT-derived macrophage density of the fibrous cap at both the first OCT (Fig. 1G) and the second OCT (6 months later) (Fig. 1H).

The presence of asymptomatic plaque rupture is a well-known concept deriving from intravascular technique (4) and postmortem

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**Figure 1. Angiographic and OCT Findings at Baseline and 6 Months Later**

Coronary angiographic of the left anterior descending (LAD) coronary artery and frequency domain-optical coherence tomography (FD-OCT) of the ruptured plaque at the first assessment (A, B, C) and second assessment (D, E, F). (A) Angiography shows a de novo coronary stenosis (red arrow) located distally compared with the previous stented segment (blue line). (B) FD-OCT cross-section shows the mid portion of the ruptured plaque (asterisk) characterized by a smooth floor and ruptured fibrous cap (arrows). Arrowheads indicate side branches. (C) FD-OCT cross-section shows the distal portion of the ruptured plaque (asterisk) characterized by a smooth floor and thin fibrous cap (arrow). (D) Angiography shows severe intra-stent restenosis (red arrow) involving the overlapped stent segment. (E) FD-OCT cross-section shows the mid portion of the ruptured plaque 6 months after the first assessment. The ruptured plaque maintains the same morphological aspects. The ruptured plaque (asterisk) is characterized by a smooth floor and ruptured fibrous cap (arrows). Arrowheads indicate side branches. (F) FD-OCT cross-section shows the distal portion of the ruptured plaque (asterisk) 6 months later characterized by a smooth floor and thin fibrous cap (arrow).
studies (5). The plaque-healing process can lead to the progression of coronary narrowing mainly because of the incorporation of thrombus remnant (5).

Our observation extends these concepts, suggesting that in the absence of additional pathogenetic components of coronary instability, ruptured plaques may remain stable over a long period of time on double antiplatelet therapy. We documented an obvious coronary plaque rupture that maintained the same morphologic features and luminal area over a 6-month period without developing thrombus or progressive stenosis or acute coronary syndrome despite a deep wall defect and a thin fibrous cap (average thickness of 60 μm) (Figs. 1I and 1J). However, the relatively short follow-up period (6 months) does not allow us to speculate on the long-term stability of disrupted plaque. At the 6-month assessment, the plaque showed a higher signal attenuation, likely suggesting a greater lipid content. These aspects, together with minimal luminal area, may affect the long-term plaque stability.

The absence of obvious inflammation associated with double antiplatelet therapy and statins may account for the stability of the dramatic lesion morphology observed in our study.

Luca Di Vito, MD, PhD, Francesco Prati, MD,* Eloisa Arbustini, MD, Filippo Crea, MD, Attilio Maseri, MD

*Interventional Cardiology, San Giovanni Addolorata Hospital, Via dell’Amba Aradam 8, 00184 Rome, Italy. E-mail: fprati@hsangiovanni.roma.it.

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REFERENCES


