Plaque Stabilization by Proprotein Convertase Subtilisin/Kexin Type 9 Inhibitor in a Patient With Familial Hypercholesterolemia Undergoing Percutaneous Coronary Intervention

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A 78-year-old man with familial hypercholesterolemia underwent percutaneous coronary intervention for a left anterior descending artery stenosis. Given that low-density lipoprotein cholesterol (LDL-C) was >110 mg/dL even under strong statin therapy, a proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor (evolocumab; 140 mg every 2 weeks) was used, and LDL-C was maintained at <30 mg/dL. At baseline and at 12-month follow-up, a non-culprit lesion in the left circumflex artery was observed on intravascular ultrasound (IVUS), which was seen to have unchanged lumen volume (from 7.3 to 7.4 mm³/mm), and slightly reduced vessel and plaque volumes (from 14.8 to 14.1 mm³/mm; from 7.5 to 6.7 mm³/mm, respectively). Lipid component on integrated backscatter-IVUS was decreased (from 55.8% to 47.6%), while other components were increased (calcification: from 1.5% to 2.9%; dense fibrosis: from 5.2% to 7.4%; fibrosis: from 37.5% to 42.1%; **Figure**).

PCSK9 inhibitors can drastically reduce LDL-C, and lead to atherosclerotic plaque regression.¹ As observed in the present case, PCSK9 inhibitors may induce plaque stabilization as well as plaque regression, which possibly contributes to the reduction of cardiovascular events.²

Figure. Gray-scale and integrated-backscatter intravascular ultrasound analysis of a non-culprit lesion in the left circumflex artery at (A–E) baseline and (F–J) 12-month follow-up. LA, lumen area; PA, plaque area; VA, vessel area.

Disclosures

The authors declare no conflicts of interest.

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