6108

Efficacy of the PCSK9 inhibitor for lipid-rich coronary plaque reduction: a near-infrared spectroscopy analysis

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Background: Recently, some studies have highlighted proprotein convertase subtilisin-kexin type 9 (PCSK9) inhibitors produce incremental low-density lipoprotein cholesterol (LDL-C) lowering effect. However, it is unknown whether the lipid composition of plaque changes is associated with serum LDL-C reduction due to PCSK9 inhibitors administration.

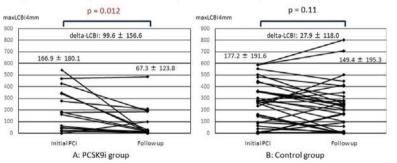
Purpose: The purpose of this study was to determine the effects of PCSK9 inhibitor (PCSK9i) on coronary plaque component in patients with a history of coronary artery disease (CAD) assessed by near-infrared spectroscopy intravascular ultrasound (NIRS-IVUS).

Methods: A total of 67 non-culprit coronary segments were identified in 34 patients. These lesions were analyzed utilizing NIRS-IVUS at baseline and follow-up coronary angiography (CAG). The subjects were divided into two groups according to lipid-lowering treatment; administration of PCSK9i group (PCSK9i: 19 segments, 9 patients) and traditional statin treatment group (Control: 48 segments, 25 patients). The change of lipid-rich plaque distribution between baseline and follow-up NIRS-IVUS was defined as the change of maximal lipid core burden index (LCBI) score for each of the 4-mm longitudinal segments (maxLCBI4mm).

Results: Mean duration from baseline to follow-up CAG was 239.4 \pm 52.4 days in the PCSK9i group and 341.0 \pm 84.1 days in the Control group (p<0.001). Despite the higher total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) in the PCSK9i group at baseline (206.6 \pm 40.9 mg/dl vs. 168.5 \pm 37.1 mg/dl, 131.5 \pm 35.4 mg/dl vs. 100.0 \pm 29.5 mg/dl; respectively, p<0.001 for both), the PCSK9i group was significantly lower TC and LDL-C at the follow-up (111.5 \pm 23.5 mg/dl vs. 157.4 \pm 27.8 mg/dl, 40.8 \pm 15.7 mg/dl vs. 86.2 \pm 19.6 mg/dl; respectively, p<0.001 for both). Furthermore, the PCSK9i group induced greater regression of maxL-CBI4mm than that of Control group (99.6 \pm 156.6 vs. 27.9 \pm 118.0, p=0.046) (Figure).

Conclusion: Compared with traditional statin therapy, PCSK9i treatment resulted in a greater decrease in lipid component in non-culprit coronary plaques. Therefore, PCSK9i may be useful option in preventing from adverse coronary events for the patients with CAD.





p = 0.046 comparison between groups of treatment

Figure 1