IMPACT OF OUT-STENT PLAQUE VOLUME ON IN-STENT INTIMAL HYPERPLASIA: RESULTS FROM SERIAL VOLUMETRIC ANALYSIS WITH HIGH-GAIN INTRAVASCULAR ULTRASOUND

i2 Poster Contributions
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Background: It has been shown that reference coronary plaques unrelated to percutaneous coronary intervention (PCI) can be reduced by aggressive LDL-C lowering therapy. However, few data exists regarding the clinical impact of the plaque behind the stent struts, which we named “out-stent plaque volume (OSPV)” after LDL-C lowering therapy. Thus we aimed to determine the clinical impact of OSPV in clinical settings.

Methods: Volumetric intravascular ultrasound (IVUS) analyses using high-gain technique were prospectively performed before and after 18 months follow-up period for 42 patients undergoing IVUS guided PCI on optimal medical therapy. Out-stent plaque area was defined as external elastic membrane (EEM) area minus stent-strut area of all through the stents. Intimal hyperplasia was defined as stent-strut area minus lumen area of all through the stents. Reference plaque area was defined as EEM minus lumen area of the plaque, which is irrelevant to the PCI procedure. The primary endpoints were the %change in OSPV, %volume of intimal hyperplasia, and %change in reference plaque volume (RPV).

Results: Thirty patients completed this study (drug-eluting stent:DES=14, bare-metal stent:BMS=16). OSPV significantly increased from 177.3±100.8 to 190.7±111.1mm3 or by 9.3% (p<0.05), although RPV substantially unchanged from 39.9±18.9 to 39.5±20.7mm3 under the condition where LDL-cholesterol levels moderately decreased from 121.2±48.0 to 103.3±48.9mg/dl, yielding significant correlation between them (r=0.43, p<0.05). Interestingly, the change in OSPV in DES group was significantly lower than that in BMS group (2.7±1.2% vs 14.0±11.0 %, p<0.05) in consequence of the results that the change in OSPV significantly correlated not with the change in LDL-C level, but with the increase in-stent intimal hyperplasia (r=0.536, p<0.05).

Conclusion: These results demonstrate that the progression of intimal hyperplasia correlates with that of OSPV independent of LDL-C level. A manipulation targeting OSPV is required to fully control in-stent restenosis.