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Five-Year Clinical Outcomes of the OLIVUS-Ex (Impact of OLmesartan on progression of coronary atherosclerosis: Evaluation by IntraVascular UltraSound Extension) Trial

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Background: The OLIVUS trial, using volumetric IVUS, reported a positive role in achieving a potentially lower rate of coronary atheroma progression through the administration of Olmesartan, an angiotension-II receptor blocker (ARB), for stable angina pectoris (SAP) patients requiring percutaneous coronary intervention (PCI). However, the benefits between ARB administration on long-term clinical outcomes and serial atheroma changes by IVUS remain unclear. Thus, we examined the 5-year clinical outcomes from OLIVUS according to treatment strategy with Olmesartan.
Methods: In the OLIVUS trial, serial volumetric IVUS examinations (baseline and 14 months) were performed in 247 patients with SAP. When patients underwent PCI for culprit lesions, IVUS was performed in their non-culprit vessels. Patients were randomly assigned to receive 20-40mg of Olmesartan or control, and treated with a combination of β-blockers, calcium channel blockers, diuretics, glycemic control agents and/or statins per physician's guidance. Five-year clinical outcomes and annual progression rate of atherosclerosis, assessed by IVUS (mean lengths 43mm), were compared with major adverse cardio- and cerebrovascular events (MACCE).
Results: Cumulative event-free survival was significantly higher in the Olmesartan group than in the control group (p=0.04; log-rank test). By adjusting for validated prognosticators, Olmesartan administration was identified as a good predictor of MACCE (HR 0.73, 95%CI 0.49-0.88; p=0.04). On the other hand, patients with adverse events (n=38) had larger annual atheroma progression than the rest of the population (22.4±17.9% vs. 2.4±14.1%, P<0.001).

	Control	Olmesartan	p-value
n=	121	126	
14-months follow-up (%)			
Plaque Volume Change	5.4 ± 15.5	0.6 ± 12.9	0.016
Percent Change in Plaque Volume	3.6 ± 9.5	−0.7 ± 8.4	0.038
In-stent Restenosis	10.7	7.9	0.42
5-Years Follow-up (%)			
Composite MACCE	20.4	10.4	0.034
Cardio-, cerebral Death	2.2	1.2	0.75
Non-fatal Stroke	1.7	0.3	0.08
Myocardial Infarction	1.6	1.6	0.89
Unstable/Progressive Angina	12.4	6.5	0.10
(Culprit Related/De novo Lesions)	(5.4/6.8)	(3.4/3.1)	
Deteriation of Heart/Renal Failure	2.5	0.8	0.30

Conclusions: Olmesartan therapy appears to confer improved long-term clinical outcomes. Atheroma volume changes, assessed by IVUS, seem to be a reliable surrogate for future MACCE in this study cohort.

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Effect of statin pretreatment on the morphology of coronary culprit plaques in patients with stable angina pectoris –An intravascular ultrasound and optical coherence tomography study–

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Background: Prior studies have shown that statin may stabilize atheromatous plaques by increasing fibrous-cap thickness. The aim of this study was to examine statin pretreatment on plaque vulnerability assessed by intravascular ultrasound (IVUS) and optical coherence tomography (OCT) in patients with stable angina pectoris (SAP).
Methods: Culprit plaques in 110 patients with SAP were interrogated by both IVUS and OCT before percutaneous coronary intervention (PCI). Volumetric analyses were performed for external elastic membrane (EEM), lumen, and plaque plus media at 1-mm intervals for 11 IVUS images per patient. The thinnest part of the fibrous cap was measured by OCT.
Results: In patients with statin pretreatment, low density lipoprotein-cholesterol (LDL-C) level (n = 73) was lower than those without statin pretreatment (n = 37) (80mg/dl vs. 113mg/dl, P<0.01). Patients with statin pretreatment had smaller EEM volume, plaque plus media volume, and plaque burden than those without (107 mm^3 vs. 129 mm^3, P<0.05, 65 mm^3 vs. 91 mm^3, P<0.01, and 62% vs. 71%, p < 0.01, respectively). By OCT, Patients with statin pretreatment had a lower incidence of lipid rich plaque than those without (43% vs. 68%, P=0.02). Statin pretreatment was associated with thicker fibrous cap thickness (115μm vs 90μm, P=0.03) and fewer incidence of thin-cap fibroatheroma (TCFA) (6% vs. 22%, p = 0.02). Multivariate logistic regression analysis identified statin pretreatment as a negative determinant of lipid rich plaque and TCFA independent of age, gender, LDL-C, and high-sensitivity C-reactive protein (odds ratio 0.36; 95% CI 0.16−0.83, p = 0.02 and odds ratio 0.21; 95% CI 0.06−0.75, p = 0.02, respectively).
Conclusions: In patients with SAP, lack of statin pretreatment was associated with larger plaque volume and more vulnerable plaque morphology independent of LDL-C levels.

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Coronary Evaginations Are Caused By Positive Vessel Remodeling And Are Nearly Absent Following Implantation Of Newer-Generation Drug-Eluting Stents: An Optical Coherence Tomography and Intravascular Ultrasound Study

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Background: Angiographic ectasias and aneurysms in stented segments have been associated with a risk of late stent thrombosis. Using optical coherence tomography (OCT) at follow-up, some stented segments show coronary evaginations reminiscent of ectasias. The occurrence, predictors and mechanisms of evaginations following drug-eluting stent (DES) implantation are unknown.
Methods: Evaginations were defined as outward bulges in the luminal contour between struts. They were considered major evaginations (ME) when present in ≥3 consecutive frames, with a depth ≥10% of the stent diameter. A total of 228 patients who had sirolimus (SES)-, paclitaxel-, biolimus-, everolimus (EES)-, or zotarolimus (ZES)-eluting stents implanted in 254 lesions, were analysed after 1, 2 or 5 years; and serial assessment using OCT and intravascular ultrasound (IVUS) was performed post intervention and after 1 year in 42 patients.
Results: ME occurred frequently at all time points in SES (~26%) and were rarely seen in EES (3%) and ZES (2%, p=0.003). SES implantation was the strongest independent predictor of ME (adjusted OR [95% CI]: 10.3 [1.3-85.5, p=0.01). Malapposed and uncovered struts were more common in lesions with vs. without ME (77% vs. 25%, p<0.001, and 95% vs. 20%, p<0.001, respectively). Post-intervention intra-stent dissection and protrusion of the vessel wall into the lumen were associated with an increased risk of evagination at follow-up (OR [95% CI]: 2.9 [1.8-4.9], p<0.001 and 3.3 [1.6-6.9], p=0.001, respectively). In paired IVUS analyses, ME showed a larger increase in the external elastic membrane area (20% area change) compared with lesions without ME (5% area change, p<0.001).
Conclusions: OCT-detected coronary evaginations are a morphological footprint of early-generation SES and are nearly absent in newer-generation DES. Evaginations appear to be related to vessel injury at baseline, and are mainly caused by positive vessel remodeling.

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Relation between peak high sensitivity CRP levels before coronary angiography and culprit lesion morphology in non-ST-segment elevation acute coronary syndrome -An optical coherence tomography study-

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Background: C-reactive protein (CRP) levels sometimes elevate during admission without any inflammatory symptom after onset of acute coronary syndrome, although its clinical significance remains unknown. In this study, we investigated the relation between peak high sensitivity CRP (hs-CRP) levels and culprit lesion morphology by optical coherence tomography study (OCT) in the acute phase of non-ST-segment elevation acute coronary syndrome (NSTEACS).
Methods: Culprit plaques in 100 patients with NSTEACS, who received elective percutaneous coronary intervention (PCI), were interrogated by OCT before PCI. The blood samples were obtained from all patients 0, 3, and 6 hours after admission and every day until coronary angiography. Patients were divided into high peak hs-CRP group (peak hs-CRP ≥50 mg/l) and low peak hs-CRP group (peak hs-CRP <50 mg/l) on the basis of highest hs-CRP levels before coronary angiography.
Results: Patients in high peak hs-CRP group (N=23) had higher Troponin I levels than those in the low peak hs-CRP group (N=77) (0.17ng/ml vs. 0.07ng/ml, P=0.02). We observed significantly more plaque rupture (78% vs. 47%, P<0.01), thrombus (96% vs.