

Lack of statin therapy is associated with plaque instability in non-culprit non-ischemic lesions of diabetic patients – data from the COMBINE OCT-FFR studyB. Berta¹, T. Roleder², R.S. Hermanides¹, A.J.J. Ijsselmuiden³, F. Alfonso⁴, F. Kauer⁵, J. Escaned⁶, G. De Luca⁷, W. Kennedy⁸, W. Wojakowski⁹, E. Kedhi¹⁰¹Isala Hospital, Zwolle, Netherlands (The); ²Regional Specialist Hospital, Research and Development Center, Wrocław, Poland; ³Amphia Hospital, Breda, Netherlands (The); ⁴University Hospital De La Princesa, Madrid, Spain; ⁵Albert Schweitzer Hospital, Dordrecht, Netherlands (The); ⁶Hospital Clinic San Carlos, Madrid, Spain; ⁷University of Eastern Piedmont, Novara, Italy; ⁸Beaumont Hospital, Dublin, Ireland; ⁹Medical University of Silesia, Department of Cardiology, SHS, Katowice, Poland; ¹⁰St-Jan Hospital, Cardiology Department, Brugge, Belgium**Funding Acknowledgement:** Type of funding source: Private grant(s) and/or Sponsorship. Main funding source(s): The trial is founded from the Department of Cardiology Zwolle Heart-centrum with support from a non-restricted grant from St Jude Medical (now Abbott)**Background:** Effect of statin therapy on coronary plaque stabilisation and reducing adverse cardiovascular events is well known both in primary and secondary prevention. Nevertheless, there is a paucity of data presenting the impact of statins on plaque morphology as assessed by optical coherence tomography (OCT).**Purpose:** The goal of this analysis was to evaluate the plaque morphology using OCT within non-culprit, non-ischaeamic coronary lesions in diabetes mellitus (DM) patients with or without statin pre-treatment.**Methods:** All patients of the COMBINE (FFR-OCT) trial underwent fractional flow reserve (FFR) measurement followed by OCT in FFR negative lesions. OCT recorded the presence of thin-cap fibroatheroma (TCFA), plaque rupture (PR), plaque erosion (PE) and calcified nodule (CN).**Results:** From the 391 patients, 82 (21%) had no statin at baseline. OCT was performed in 463 lesions of which 96 lesions assessed in statin naive and 367 lesions in statin treated group. The median angiographic diame-ter stenosis was 50% and the median FFR value was 0.88 in both groups ($p=0.953$ and $p=0.448$, respectively). Myocardial infarction (MI) at presentation was 16.6% and did not differ between groups ($p=0.380$). Patients without statin pre-treatment were characterized by lower rate of known hypercholesterolemia (47.6% vs. 63.0%; $p=0.011$), male gender (52.4% vs. 65.7%; $p=0.027$), active smokers (8.5% vs. 22.3%; $p=0.004$) and previous MI (22.0% vs. 35.3%; $p=0.022$) as compared to patients with statin pre-treatment, respectively. The results of the qualitative OCT findings see in Table 1.**Conclusions:** Non-ischemic lesions of DM patients without statin pre-treatment showed more vulnerable and instable plaque features like wider lipid arc, thinner fibrotic cap and a higher prevalence of lipid-rich plaque, TCFA and PR suggesting a stabilizing effect of statins on non-ischemic atherosclerotic lesions.

Table 1

	Without statin pre-treatment (n=96)	With statin pre-treatment (n=367)	All lesions (n=463)	p
Fibroatheroma	74 (77.1)	295 (80.4)	369 (79.7)	0.474
Calcified plaque	84 (87.5)	317 (86.4)	401 (86.6)	0.773
Calcium arc (°)	147 [89–231]	138 [81–234]	143 [83–234]	0.789
Lipid-rich plaque	77 (80.2)	242 (65.9)	319 (68.9)	0.007
Lipid arc (°)	226 [176–272]	180 [129–245]	193 [138–251]	<0.001
Cap thickness (µm)	93 [61–159]	118 [65–200]	112 [64–187]	0.008
Thin-cap fibroatheroma	32 (33.3)	63 (17.2)	95 (20.5)	<0.001
Plaque rupture	16 (16.7)	35 (9.5)	51 (11.0)	0.047

Values are n (%) or median [first quartile–third quartile].