Effect of high-intensity statin therapy on atherosclerosis in non-infarct related coronary arteries: a serial intravascular ultrasonography study

IBIS-4 (Integrated Biomarkers and Imaging Study)

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□ I have the following potential conflicts of interest to report:

□ Research contracts

□ Consulting

Employment in industry

□ Stockholder of a healthcare company

□ Owner of a healthcare company

□ Other(s):

I do not have any potential conflict of interest

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BACKGROUND

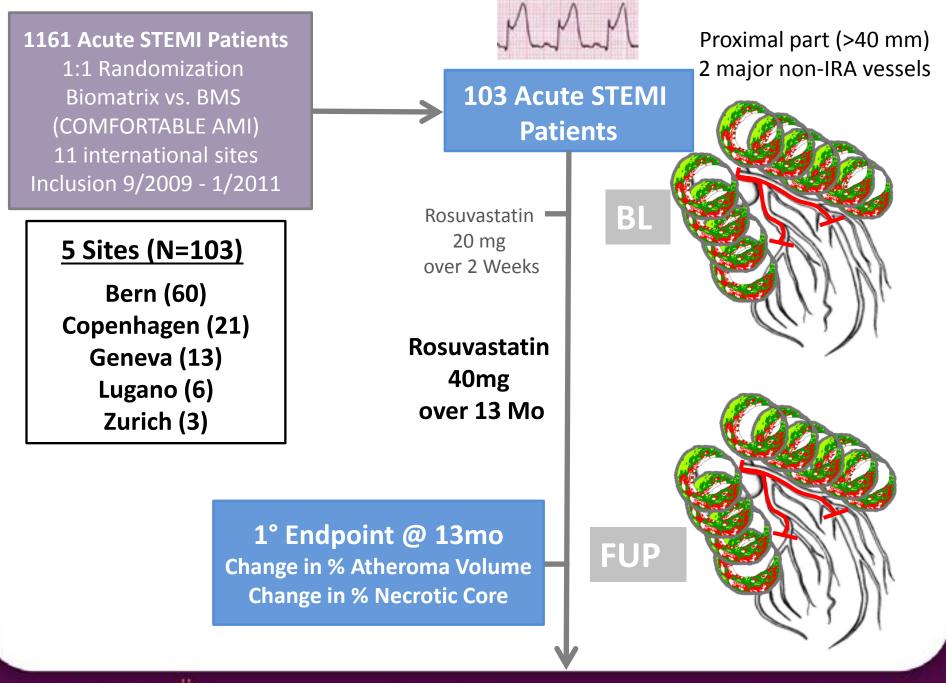
- Statins potently reduce cardiovascular events and IVUS studies have shown that high intensity statin therapy results in atheroma regression in stable CAD patients.
- Acute STEMI remain have not been included in IVUS regression studies despite their higher risk for recurrent events and high frequency of vulnerable plaques typically extending beyond the culprit lesion.
- Plaque phenotype is relevant in the pathogenesis of future events. Therefore, it is of interest to study changes in plaque <u>composition</u> in response to highintensity statin therapy.

Hypothesis

Coronary <u>atherosclerosis regression</u> can be achieved by the highest dose of rosuvastatin therapy (40 mg daily) in the proximal segments of non-infarct related arteries of STEMI patients within 13 months.

Similarly, a reduction of <u>RF-IVUS defined necrotic core</u> and a decrease in the frequency of high risk plaque (thin cap fibroatheromas) can be achieved.



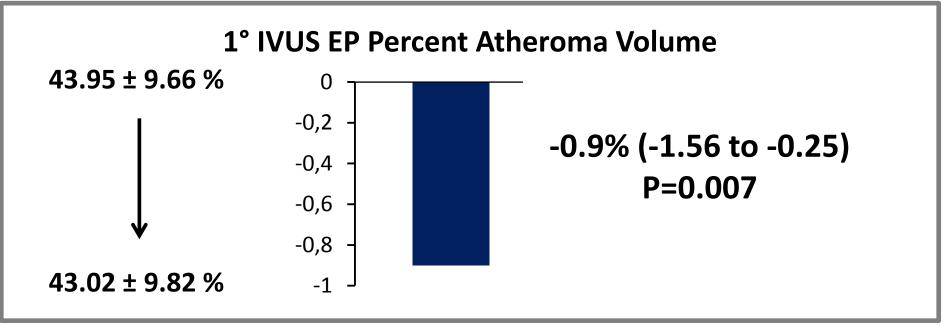


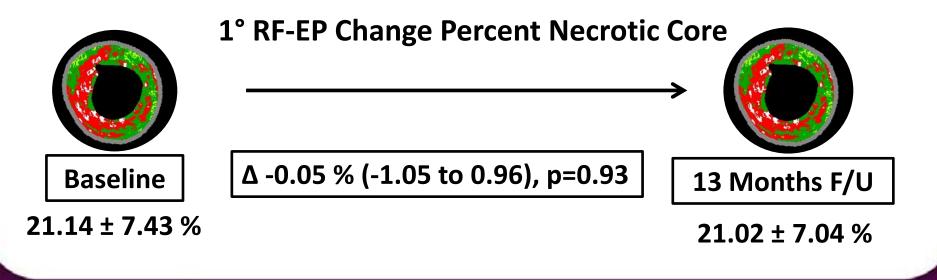
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PRIMARY IVUS AND RF-IVUS ENDPOINT





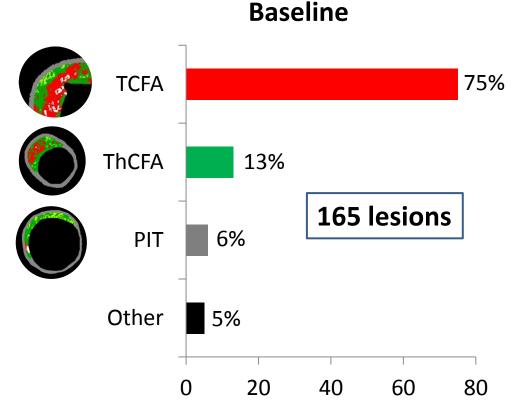
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RF-IVUS LESION PHENOTYPE ANALYSIS

82 serially assessed patients with 146 analysed vessels



Other: fibrocalcific, fibrotic 1 lesion was not present at BL but at FUP



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CONCLUSIONS

- The proximal segments of non-IRA of STEMI patients feature a high atherosclerotic plaque burden with the majority of lesions characterized as thin-cap fibroatheromas.
- High-intensity statin therapy throughout 13 months is associated with a significant reduction of coronary atherosclerosis.
- High-intensity statin therapy did not change the proportion of necrotic core and plaque phenotypes.

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