High-dose rosuvastatin shrinks coronary plaque in heart attack patients

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One year of treatment with the highest dose of the cholesterol-lowering drug rosuvastatin can shrink plaque inside the arteries of patients who have had a certain type of heart attack known as ST-segment elevation myocardial infarction (STEMI), according to a new study presented today at ESC Congress 2014.

Although STEMI patients undergo a revascularisation procedure to unblock the "culprit" artery that caused their heart attack, they remain at increased risk for similar events due to plaque formation in other untreated coronary arteries.

The IBIS-4 study, which was published simultaneously in the *European Heart Journal* is the first to use ultrasound imaging inside coronary arteries both at the time of heart attack and after 13 months of treatment to show the benefit of high-dose statin therapy on plaque burden, said study investigator Lorenz Räber, MD an interventional cardiologist from Bern University Hospital in Bern, Switzerland.

The study was an investigator-initiated trial performed at five sites in Europe (University Hospitals of Bern (CH), Copenhagen (DK), Geneva (CH) and Zurich (CH) and Cardiocentro Lugano (CH)) with support from the Swiss National Science Foundation as well as a stent manufacturer (Biosensors SA) and an ultrasound imaging company (Volcano) but without support from a pharmaceutical cholesterol-lowering manufacturer.

"Previous work has shown that high-dose rosuvastatin can reduce plaque size in stable patients, but until now this has not been specifically investigated in arteries of patients with acute heart attacks, a setting known to harbour additional high risk plaques that can be the source for future cardiovascular events," said Dr. Räber. "Additionally, our study is the first to use intracoronary ultrasound to assess the actual plaque composition and the plaque phenotype, and to observe how both respond to treatment."

IBIS-4 included 103 acute heart attack patients who were first successfully treated to unblock the culprit vessel.

Subjects then underwent imaging, both at the start of the study and then after 13 months of high-intensity rosuvastatin treatment, to assess the drug's impact on their non-culprit arteries.

Rosuvastatin was given at a dose of 40 mg daily. After 13 months, ultrasonography showed that 85% of patients had regression of plaque in at least one artery, and 56% had regression in both.

Overall, intracoronary plaque volume was reduced by a mean of -0.9% (p=0.007), with a mean change of the total atheroma volume of -13.7 mm3 (p=0.006). Although the reduction in plaque volume was independent from cholesterol levels at baseline, it was directly related to the extent of cholesterol reduction at 13 months.

An analysis of the plaque tissue composition using radiofrequency intravascular ultrasonography showed no changes in high risk tissue (necrotic core) and no reduction in "high risk plaques".

As expected, rosuvastatin also had beneficial effects on cholesterol levels. Low-density lipoprotein (LDL) cholesterol ("bad cholesterol") decreased from a median of 3.29 mmol/L at baseline to 1.89 mmol/L (p