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Abstract 45: Dual Antiplatelet Therapy to Inhibit Myocardial Injury in Patients With High-risk Coronary Artery Plaque: A Randomised Controlled Trial

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Introduction: High-risk coronary atherosclerotic plaque is associated with higher plasma troponin concentrations suggesting ongoing myocardial injury that may be a target for dual antiplatelet therapy. To determine whether ticagrelor reduces high-sensitivity troponin I concentrations in patients with established coronary artery disease and high-risk coronary plaque.

Methods: In a randomized double-blind placebo-controlled trial, patients with multivessel coronary artery disease underwent coronary 18F-fluoride positron emission tomography-computed tomography and measurement of high-sensitivity cardiac troponin I and were randomized (1:1) to ticagrelor 90 mg twice daily or matched placebo. The primary endpoint was troponin concentration at 30 days in patients with increased coronary 18F-fluoride uptake.

Results: In total, 202 patients were randomized and 191 met the pre-specified criteria for inclusion in the primary analysis. Patients with increased coronary 18F-fluoride uptake (n = 120/191) had higher baseline cardiac troponin I concentrations (3.8 ± 2.9 *versus* 2.5 ± 2.6ng/L, p = 0.004). At 30 days, ticagrelor markedly reduced adenosine diphosphate-stimulated platelet activation (P-selectin expression: ratio of geometric mean fluorescence, 0.06; $p < 0.001$) but had no effect on plasma troponin concentrations in patients with coronary 18F-fluoride uptake (ratio of geometric means for ticagrelor *versus* placebo, 1.11, [95% confidence interval 0.90 to 1.36], p = 0.32). At 1 year, ticagrelor had no effect on troponin concentrations in patients with increased coronary 18F-fluoride uptake (ratio of geometric means, 0.86, [95% confidence interval 0.63 to 1.17], p = 0.33).

Conclusions: Dual antiplatelet therapy with ticagrelor does not reduce plasma troponin concentrations in patients with high-risk coronary plaque, suggesting that subclinical plaque thrombosis does not contribute to ongoing myocardial injury in this setting.

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