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dual antiplatelet therapy to inhibit myocardial injury in patients with high-risk coronary artery plaque: a randomised controlled trial

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Topic(s):
Positron Emission Tomography (PET)

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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.

Purpose

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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<table>
<thead>
<tr>
<th></th>
<th>Adjusted Geometric Mean (GSE)</th>
<th>Ratio of Geometric Means (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac troponin I, ng/L (18F-fluoride activity)</td>
<td>3.8 (1.1)</td>
<td>3.4 (1.1)</td>
<td>1.11 (0.90 to 1.36)</td>
</tr>
<tr>
<td>Cardiac Troponin I, ng/L (No 18F-fluoride activity)</td>
<td>2.4 (1.1)</td>
<td>2.3 (1.1)</td>
<td>1.02 (0.80 to 1.31)</td>
</tr>
</tbody>
</table>

Estimates are back transformed estimates from analysis of log transformed values at 30 days adjusting for age, sex and log transformed baseline troponin. Ratio of geometric means is Ticagrelor divided by Placebo. GSE, geometric standard error.