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Troponin in the chronic coronary syndrome

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Background: Troponin (hs-cTnI) is a powerful predictor of adverse outcomes in acute coronary syndromes. Emerging evidence suggests that it could also have an important role in risk stratification in the chronic coronary syndrome. However, the mechanism of troponin release in patients with stable coronary disease remains unclear, and it is first necessary to establish whether concentrations associate with myocardial ischaemia and are modified by revascularisation.

Methods: Patients with severe single-vessel CAD were randomized 1:1 to PCI (n=105) or placebo procedure (n=95). Troponin was measured before and after cardiopulmonary exercise testing (CPET) at baseline and 6-weeks after randomisation.

Results: Following CPET, exercise time strongly predicted the exercise-induced hs-cTnI change (OR 2.90, p<0.0001) but there was no association between ischemia (p=0.114) or ischaemic-symptom severity (p=0.201). In patients randomised to revascularisation, resting hs-cTnI were not altered by PCI (OR 0.78, p=0.375). A strong association persisted between exercise-induced hs-cTnI change and exercise time (OR 3.40, p<0.0001), however there was little evidence that revascularisation reduced exercise-induced hs-cTnI increment compared to placebo (OR 1.42, p=0.251).

Conclusion: There was little evidence that ischemia or successful revascularisation influenced hs- cTnI at rest or after exercise. Exercise intensity was instead the most important predictor of hs- cTnI