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New cardiac troponin T assay in stable coronary artery disease

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Cardiac troponins (Tp) T and I are components of the contractile apparatus of cardiomyocytes and are the preferred markers of myocardial necrosis in patients with acute coronary syndromes. Among these patients, a strong association between elevated Tp levels and recurrent ischemic events has been already established. It has been shown that even very small elevations in Tp (greater than 0.01 μg per liter) are associated with an increased risk of an adverse outcome in patients with acute coronary syndromes.

In most patients with stable coronary artery disease (SCAD), cardiac TpT levels are below the limit of detection for the conventional assay. The distribution of very low circulating TpT levels, as well as their association with cardiovascular events, in such patients is unknown. A highly sensitive assay for cardiac TpT has recently been developed, permitting measurement of concentrations that are lower by a factor of 10 than those measurable with conventional assays.

This study involved 3679 patients who had been included in the Prevention of Events with Angiotensin Converting Enzyme Inhibition (PEACE) Trial, with SCAD and preserved left ventricular systolic function. All included patients were free of heart failure at baseline, and none had been hospitalized for unstable angina within the 3 months preceding trial entry. Patients were followed every 6 months, for a median of 5.2 years. It had been used a new, high-sensitivity assay to determine the concentration of cardiac TpT in plasma. The lower limit of detection of this highly sensitive assay was 0.001 μg per liter. The detection limit of the older troponin T assay is 0.01 μg per liter. The outcomes examined in this analysis included cardiovascular death, fatal and nonfatal heart failure, and fatal and nonfatal acute myocardial infarction.

Of the 3679 participants, 3630 had TpT levels that would be expected to be undetectable by the conventional assay (i.e., <0.01 μg per liter, which corresponds to <0.03 μg per liter with the new assay). In this subgroup, 117 cardiovascular deaths occurred. The risk estimate for these 3630 patients was similar to that for the entire cohort ($P<0.001$), suggesting that the newer troponin T assay provides powerful prognostic information in patients who would have undetectable levels with the conventional assay. The risk of heart failure associated with increasing quartiles of TpT levels was also strong ($P<0.001$). TpT levels were higher in men than in women ($P<0.001$). There was no association between TpT levels as measured with the

highly sensitive assay and the incidence of myocardial infarction (P=0.11). The presumed mechanisms responsible for the release of very low levels of cardiac troponin T in patients with SCAD could be transient, clinically silent ischemic episodes and small-vessel occlusions, inflammatory processes, cardiomyocyte apoptosis, reduced renal clearance, and increased myocardial strain due to pressure or volume overload.

In summary, very low levels of troponin T were detectable in the great majority of patients with SCAD. Levels well below the limit of detection of previous assays and below the 99th percentile in apparently healthy blood donors were strongly associated with the incidence of cardiovascular death and heart failure, but not with myocardial infarction in these patients.

Comment on a paper:
Torbjorn O, James A de Lemos, Marc SS et al – A Sensitive Cardiac Troponin T Assay in Stable Coronary Artery Disease. PEACE Trial Investigators. *NEJM* 2009, November 25; 10:1056