

## **Impact of the implementation of a sensitive troponin assay on the frequency of inducible myocardial ischemia and outcomes in patients referred to a chest pain unit**

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**Purpose:** Sensitive troponin assays allow an earlier and more accurate rule out of myocardial infarction (MI). Our aim was to assess the impact of the implementation of a sensitive cardiac troponin I (s-cTnI) assay on the frequency of detection of inducible myocardial ischemia and on the risk of subsequent cardiac events in patients with suspected acute coronary syndrome and negative troponin results.

**Methods:** We evaluated 1939 patients with acute chest pain suggestive of ischemic origin, nondiagnostic electrocardiograms (ECGs) and negative serial troponin levels who were referred to our chest pain unit and underwent exercise echocardiography. We compared the frequency of exercise-induced echocardiographic and electrocardiographic myocardial ischemia before (n=1144) and after (n=795) the implementation of a s-cTnI assay in our hospital, which lowered the threshold of detection of myocardial necrosis from 0.20 ng/mL (previous generation assay) to 0.06 ng/mL. We also compared the cumulative 6-month rate of cardiac death, MI and coronary revascularization between two subgroups of patients admitted to our unit within one year before (n=344) and one year after (n=363) the implementation of the s-cTnI assay.

**Results:** The percentage of patients with exercise-induced echocardiographic myocardial ischemia was significantly lower in the s-cTnI assay group (21.8%) than in the previous generation test group (30.3%,  $p < 0.001$ ). In the subset of 1523 patients with interpretable baseline ECGs, there was also a lower probability of exercise-induced ischemic ECG changes in the s-cTnI test group (16%) than in the previous generation assay group (21.8%,  $p = 0.004$ ). After multivariate adjustment, patients with negative serial s-cTnI tests had a significantly lower risk of echocardiographic (OR 0.67, 90% CI 0.54-0.85,  $p = 0.001$ ) and electrocardiographic (OR 0.73, 95% CI 0.55-0.96,  $p = 0.02$ ) ischemia compared with those with negative results of the previous generation assay. The rate of cardiac death, MI or coronary revascularization at 6 months was 14.9% in the subgroup of patients with a

negative s-cTnI test as compared with 17.4% in the subgroup evaluated before the implementation of this assay ( $p=0.35$ ).

Conclusions: In patients with acute chest pain, nondiagnostic ECGs and negative troponin levels, the implementation of a s-cTnI assay was associated with a significant reduction in the probability of inducible myocardial ischemia. Nonetheless, we did not observe a significant decrease in the rate of cardiac death, MI or coronary revascularization at 6 months.