# **Abstract: P383**

## High-sensitivity troponin T is predictive of adverse outcome after acute ischemic stroke

### Authors:

M. Furtner<sup>1</sup>, A. Hammerer-Lercher<sup>2</sup>, S. Kiechl<sup>1</sup>, J. Mair<sup>3</sup>, <sup>1</sup>Innsbruck Medical University, Department of Neurology - Innsbruck - Austria, <sup>2</sup>Innsbruck Medical University, Central Institute for Medical Laboratory Diagnostics - Innsbruck - Austria, <sup>3</sup>Innsbruck Medical University, Department of Internal Medicine III, Cardiology - Innsbruck - Austria,

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**Purpose:** High-sensitivity cardiac troponin T (hs-cTnT) is used for risk stratification in cardiac diseases. Data on cTnTs and ischemic stroke are conflicting due to low analytical sensitivity. We investigated hs-cTnT as a predictor for stroke outcome.

**Methods:** 517 consecutive stroke patients screened. Data included onset time, severity scales on admission and discharge[NIH Stroke Scale (NIHSS), modified Rankin Scale (mRS), Barthel Index (BI)], and various clinical/laboratory parameters. At day 90, mRS and BI were assessed by telephone interview. Exclusions: transient ischemic attacks, bleedings, undetermined onset, no hs-cTnT<24 h, estimated glomerular filtration rate (eGFR)<15 ml/min, MI during stay or 4 weeks prior, no follow-up. Final sample: 340 patients.

Definitions for adverse outcome: death; mRS>2; BI<90; mRS>3 and/or BI<60 combined.

hs-cTnT was divided into 5 groups: <5 ng/L; quartiles of elevated hs-cTnT (Q1 5.09-8.0; Q2 8.29-12.6; Q3 12.76-27.62, Q4 27.7-221 ng/L). hs-cTnT vs. outcomes and other parameters, and odds ratios (ORs) for outcomes vs. hs-cTnT were calculated using multivariate logistic regression. P for trend was calculated from group medians.

Results: Median age, 68 years (range 18-96); 208 men. 117 (52%) had hs-cTnT levels<5ng/L. Median hs-cTnT (Q1 to Q4): 6.70, 10.30, 18.65, and 43.0 ng/L. Patients with higher hs-cTnT were older, more often male, and more severely ill. There was a significant rise of NIHSS and adverse outcomes over quartiles. eGFR, Hb, and CRP were more pathological in higher quartiles. Stroke etiology was more likely cardiac, and heart conditions (atrial fibrillation, coronary heart disease) more prevalent in Q3/Q4. Associations between hs-cTnT and outcomes remained significant after adjustment for heart conditions. hs-cTnT was independently associated with all adverse outcomes (p for trend<0.001). NIHSS and age were the only other independent predictors.

Conclusions: hs-cTnT is independently predictive of adverse stroke outcomes after 90 days. Associations with definitions of outcome were robust. Categorization of hs-cTnT into quartiles showed a dose-effect relation, with significantly higher ORs in the more elevated groups. Multivariate adjustment for preexisting cardiac conditions did not alter significance. Hence, hs-cTnT elevation may indicate cerebral impairment translating into cardiac damage rather than vice versa. Stress-induced patchy myocytolysis caused by centrally mediated vegetative fluctuations has been implicated. hs-cTnT is a functionally meaningful predictor of clinical course; pathophysiological connections, however, still need to be elucidated.