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Use of high-sensitivity cardiac troponin I (hs-cTnI) for secondary prevention in high-risk patients suffering from stable coronary artery disease

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Background: There are several tools for primary prevention (e.g. Framingham, ESC) that can be used to predict mortality risk in healthy individuals. However, only a few scores have been validated to predict outcome in patients with cardiovascular disease. One of these instruments is the REACH (REduction of Atherothrombosis for Continued Health) score. The ESC guideline for stable coronary artery disease (CAD) places a clear emphasis on carrying out risk stratification before using invasive treatment. Recent studies have revealed a prognostic value of serum hs-cTnI in patients with stable CAD.

Purpose: The aim of this study was to evaluate the prognostic information provided by hs-cTnI in stable high-risk CAD patients.

Methods: Between 2011 and 2014, consecutive stable patients with suspected CAD undergoing coronary angiography were included in the study. Data from a 4-year follow-up was obtained; the study endpoint was defined as all-cause mortality. Serum hs-cTnI was measured before angiography using a high-sensitivity assay.

Results: A total of 3,742 patients were included, of whom 2,274 (60.1%) had confirmed CAD. Patients with an estimated annual mortality rate above 3% using the REACH score were defined as having high risk (n=996 in the low-risk group, n=1,278 in the high-risk cohort). Patients with higher risk were more often male (81.5% vs. 69.2%, $p < 0.001$), were older (mean age 73.2 ± 8.1 y vs. 63 ± 9.4 y), and had more cardiovascular risk factors

(diabetes mellitus (DM) 43.5% vs. 13.7%, $p < 0.001$; arterial hypertension 90.8% vs. 86%, $p < 0.001$). Median hs-cTnI was elevated in high-risk patients (6.9 ng/L [IQR 1–3: 3.8–14.8 ng/L] vs. 3 ng/L [IQR 1–3: 1.7–5.9 ng/L]; $p < 0.001$). A total of 298 patients (23.3%) died in the high-risk group compared with 74 patients (7.4%) in the low-risk group. Log(hs-cTnI) was found to be a risk factor based on regression analysis including age, gender, DM, arterial hypertension and the REACH score (OR 2.02 [95% CI 1.61–2.54]). The area under the ROC of hs-cTnI for predicting all-cause mortality was 0.69 (95% CI 0.66–0.72) for hs-cTnI and 0.72 (95% CI 0.69–0.72) for the REACH score. There was a correlation between hs-cTnI and the REACH score (Spearman correlation 0.458; $p < 0.001$). In patients at low risk, the best cut-off for hs-cTnI was 3 ng/L, and for high-risk patients 8.25 ng/L was the best threshold value. Using low REACH score and low hs-cTnI levels, it was possible to identify patients at very low risk with a mortality rate below 3.4% in a follow-up of 48 months. It was also feasible to determine patients at very high risk in the group of patients who were already at high risk using the hs-cTnI cut-off (mortality 15.2% vs. 33.7%).

Conclusion: Hs-cTnI was found to be an independent risk factor in low as well as high-risk patients. Hs-cTnI levels correlate with the REACH risk score. Moreover, it was possible to separate patients at very high and very low risk by combining REACH score and hs-cTnI.

