Abstract: P1730

Serial high-sensitivity troponin I measurements to discriminate type 2 from type 1 myocardial infarction

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Topic(s):
Acute Coronary Syndromes: Biomarkers

Citation:
European Heart Journal (2019) 40 (Supplement), 1028

Background: Acute myocardial infarction (MI) is associated with high morbidity and mortality. A robust differentiation between type 1 and type 2 MI (T1/T2MI) has prognostic and therapeutic implications. We investigated whether serial high-sensitivity cardiac troponin I measurements could reliably discriminate T1MI from T2MI in patients presenting with a non-ST elevation myocardial infarction (NSTEMI).

Methods: We used data from a prospective acute coronary syndrome biomarker registry of patients with suspected MI that presented at or were transferred to one of two study centres. Here, we analysed an unselected group of 265 NSTEMI patients (67.2% males). Blood was drawn on admission and after 3 hours. High-sensitivity troponin I (hs-cTnI) was measured in frozen samples by a technician blinded to patient characteristics. T1MI or T2MI was defined as the gold-standard study diagnosis by two independent cardiologists based on all available data according to the Third Universal Definition of MI.

Results: A diagnosis of T2MI was made in 55 patients (20.8%) in the NSTEMI cohort. T2MI patients did not differ from T1MI patients regarding age, gender, traditional risk factors, or percentage of those with a history of coronary artery disease. Median baseline hs-cTnI levels were higher in T1MI (436.25; IQR 63.7–1918.8 ng/L) than in T2MI patients (48.4; IQR 11.7–305.9 ng/L; p<0.001). Absolute change in hs-cTnI concentration between 0 and 3 h was greater in T1MI than in T2MI patients with Dhs-cTnI 93.6 ng/L (IQR 13.5–815.3 ng/L) vs. 20.4 ng/L (IQR 2.5–106.5 ng/L) (p<0.001). hs-cTnI yielded an area under the receiver operator characteristics (AUROC) curve for identifying T2MI at baseline of 0.71 (IQR 0.64–0.79) and after 3 h of 0.7 (IQR 0.61–0.78). Dhs-cTnI was associated with an AUROC of 0.68 (IQR 0.6–0.76). Regarding a rule-out approach, Youden-optimized cut-offs for hs-cTnI at baseline as well as for the absolute change in hs-cTnI concentration were calculated (186.5 ng/L; 154.4 ng/L). Use of these two criteria yielded a sensitivity of 89% (78–96%) and a negative predictive value of 95% (89–98%) to exclude T2MI. 49 of 55 T2MI patients would have been ruled out using this algorithm.

Conclusion: Our data show that hs-cTnI concentrations differ between patients presenting with T1 and T2MI. The concentration of hs-cTnI and its change over time has the potential to rule out T2MI and therefore to identify patients who might benefit from an early invasive management. The differentiation between T1MI and T2MI by using hs-cTnI is nevertheless challenging, and further research on specific algorithms is needed.