intracoronary p-Selectin and activated PMA [r^2=0.5; p=0.03, r^2=0.9; p=0.0008 respectively]. PMP correlated positively with intracoronary p-selectin and activated PMA [r^2=0.7; p=0.01 and r^2=0.6; p=0.02 respectively] but they also correlated with TF+ PMA [r^2=0.7; p=0.01]. CD62P+ PMP was strongly positively related with intracoronary p-Selectin [r^2=0.9; p=0.0001]. CD105+ EMP also correlated positively with intracoronary p-selectin, activated PMA and TF+ PMA [r^2=0.7; p=0.007, r^2=0.6; p=0.01 and r^2=0.5; p=0.04 for intracoronary p-selectin, activated PMA and TF+ PMA respectively]. CD 62E+ EMP positively correlated with intracoronary p-selectin, activated PMA and TF+ PMA [r^2=0.6; p=0.02, r^2=0.6; p=0.01 and r^2=0.5; p=0.03 respectively].

Conclusions Markers of platelet activation at the site of the culprit lesion during ST elevation ACS correlated strongly not only with total MPs and PMPs but also with endothelial derived MPs. The interaction between activated platelets and monocytes with endothelial cells and the subsequent formation of PMP and EMP during ACS highlights the importance of the downstream effect of activated platelets, monocytes and endothelial cells via MP formation and their contribution in the pathophysiology of ACS.

128 IMPLICATIONS OF LOWERING THE THRESHOLD OF CARDIAC TROPONIN IN THE DIAGNOSIS OF MYOCARDIAL INFARCTION

doi:10.1136/heartjnl-2012-301877b.128

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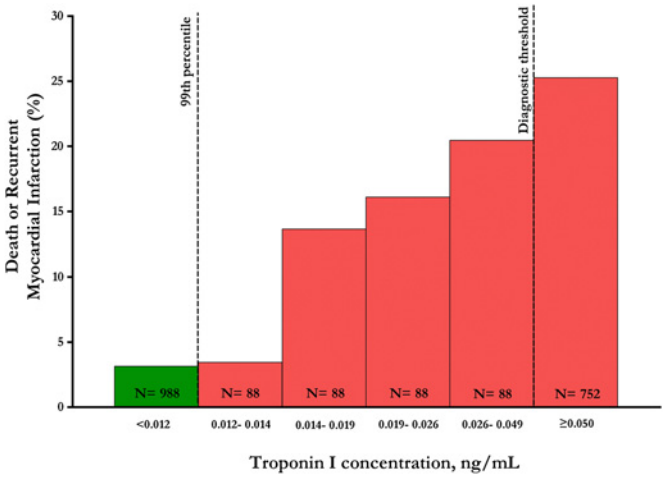
Introduction The Universal Definition recommends the 99 percentile of cardiac troponin as the diagnostic threshold for myocardial infarction (MI) in patients with suspected acute coronary syndrome if the assay achieves a coefficient of variation (CV) <10%. However, due to imprecision in contemporary assays and concern of over diagnosing myocardial infarction, diagnostic thresholds are currently set at higher concentrations where the assay can achieve CV ≤10%. The aim of this study was to assess the relationship between plasma troponin I concentrations, assay precision and clinical outcomes in patients with suspected acute coronary syndrome.

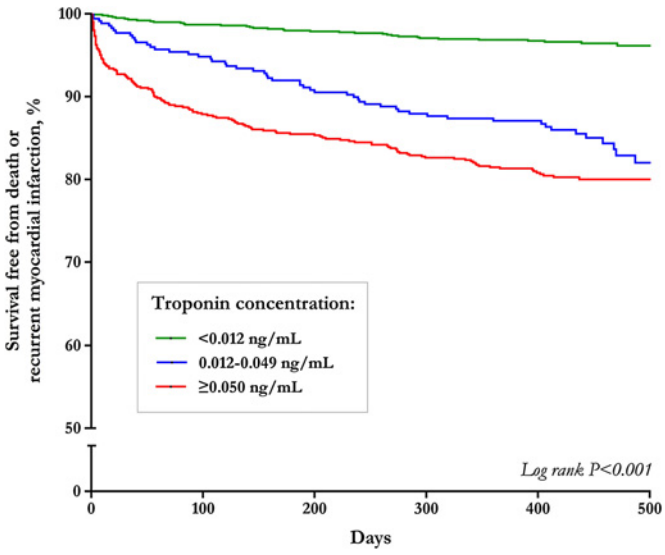
Methods Using a contemporary sensitive troponin I assay, consecutive patients admitted with suspected acute coronary syndrome (n=2092) were stratified according to the 99th percentile (0.012 ng/ml; CV 20.8%) and current diagnostic threshold (0.05 ng/ml; CV 7.2%) into three groups: <0.012 ng/ml, 0.012–0.049 ng/ml and ≥0.05 ng/ml. Event-free survival (recurrent myocardial infarction or death) at 1 year was compared between patients grouped by troponin I concentration.

Results Plasma troponin concentrations were <0.012 ng/ml in 988 patients (47%), 0.012–0.049 ng/ml in 352 patients (17%) and ≥0.05 ng/ml in 752 patients (36%). At 1 year, patients with troponin concentrations 0.012–0.049 ng/ml were more likely to be dead or readmitted with recurrent myocardial infarction compared to those with troponin concentrations <0.012 ng/ml (13% vs 3%; OR 4.8, 95% CI 3.0 to 7.7; p<0.001). Compared to troponin ≥0.050 ng/ml, patients with troponin 0.012–0.049 ng/ml had a higher risk profile but were less likely to be diagnosed with, or investigated and treated for, acute coronary syndrome.

Conclusions Lowering the diagnostic threshold to the 99th percentile and accepting greater assay imprecision would identify those at high-risk of recurrent MI and death, but increase the diagnosis of MI by 46%. It remains to be established whether reclassifying and treating these patients as MI would improve outcome.



Abstract 128 Figure 1



Abstract 128 Figure 2

129 THE PROGNOSTIC; VALUE OF A 7-WEEK HIGH SENSITIVITY TROPONIN T LEVEL AFTER AN ACUTE CORONARY SYNDROME

doi:10.1136/heartjnl-2012-301877b.129

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Background High sensitivity Troponin T (HS-TnT) is a well established diagnostic and prognostic tool in acute coronary syndrome (ACS). However, its role in the convalescence phase after an ACS is unknown. Our first aim was to assess the prognostic utility of a single HS-TnT level at 7-week post ACS. Second, we evaluated whether any serial changes in HS-TnT between the index admission and 7 weeks post ACS had any link with prognosis. Third, we assessed whether the prognostic utility of HS-TnT is independent of various echocardiographic abnormalities.

Methods We measured HS-TnT levels in 326 consecutive patients at 7 weeks after an ACS event. The composite end point of death from any cause or acute myocardial infarction (AMI) was evaluated over a median duration of 30 months.

Heart: first published as 10.1136/heartjnl-2012-301877b.128 on 16 May 2012. Downloaded from http://heart.bmj.com/ on April 19, 2024 by guest. Protected by copyright.