69 **THE RELATIONSHIP BETWEEN TROPONIN LEVEL AND MORTALITY IN AN UNSELECTED POPULATION OF OVER 250,000 PATIENTS WITH SUSPECTED ACUTE CORONARY SYNDROME (NIHR HEALTH INFORMATICS COLLABORATIVE TROP-RISK STUDY)**

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Background Current evidence suggests a direct relationship between the magnitude of troponin elevation and mortality, albeit over a limited range of troponin levels, and clinicians generally work under the impression that higher troponins signify higher mortality in all age groups. The objective of our study was to use big data to determine the relationship between the full spectrum of troponin level and mortality in patients in whom troponin testing has been performed for clinical purposes.

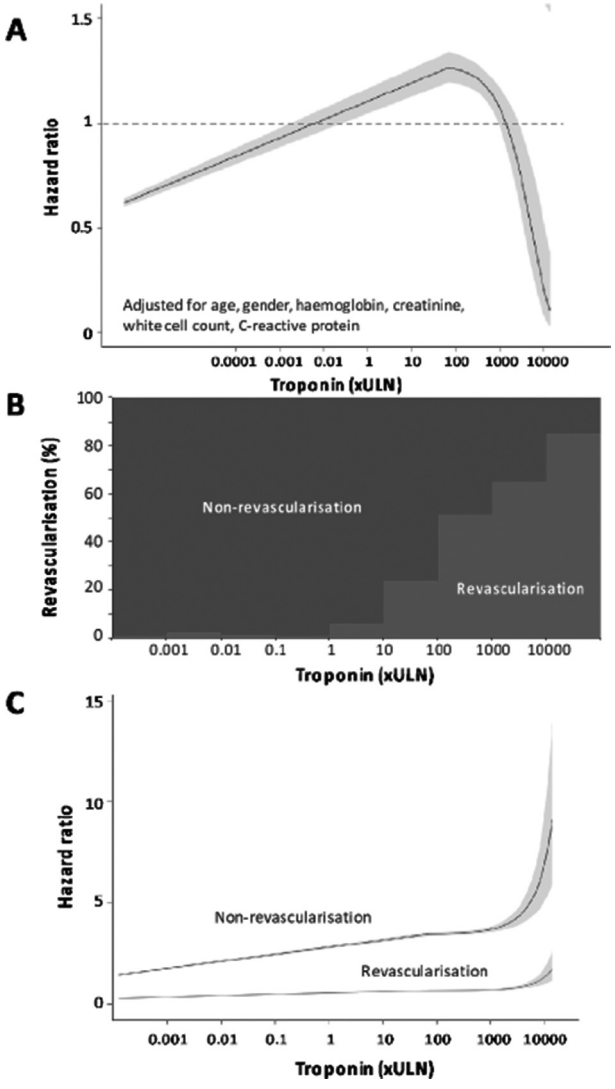
Methods As part of the National Institute for Health Research Health Informatics Collaborative project, all troponin values measured during the study period (2010 to 2017) were assembled from five cardiovascular centres. Troponin concentrations were standardised as a multiple of each laboratory's 99th-percentile of the upper limit of normal (ULN). All patients were followed up until death or censoring on 1st April 2017. To model the relation between peak troponin level and all-cause mortality we used restricted cubic spline Cox regression analysis. Splines were adjusted for patient age, gender, haemoglobin, creatinine, white cell count and C-reactive protein.

Results 257,948 patients underwent troponin assessment. During a median follow-up of 1,198 (IQR, 514–1,866) days, there were 55,850 (21.7%) deaths. Using multivariable-adjusted restricted cubic spline Cox regression analysis, an inverted-U shaped relationship was observed between peak troponin level and mortality in all patients (figure 1A). Among patients who were admitted to hospital, the recorded diagnostic code was acute coronary syndrome (ACS) in 14,468 patients and non-ACS in 120,049 patients.

The revascularisation rate within 3 months was 61.0% (n=8,820) in ACS versus 4.0% (n=4,793) in non-ACS patients. There was a very different rate of revascularisation across the spectrum of troponin. The rate was only 1.4% for troponins below 1 xULN, and 6.1% between 1 and 10 xULN. Beyond 10 xULN, rate of revascularisation rose rapidly to over 85% for greater than 10,000 xULN (figure 1B). Stratifying patients by revascularisation, the restricted cubic spline Cox regression curve showed a progressive increase in mortality within both the revascularised and non-revascularised strata, even to very high peak troponin levels (figure 1C). Overall, revascularisation was associated with lower hazard ratios across all troponin levels. A similar pattern was seen when patients were stratified by the presence or absence of ACS diagnosis.

Conclusion An elevated troponin, even slightly above the ULN should be taken seriously. The inverted-U shaped mortality relationship with troponin occurred because patients with the highest troponin formed a different clinical subgroup who underwent different clinical management with a high revascularisation rate. These data on troponin level and mortality may help to inform clinical practice decisions and guide future risk stratification algorithms for patients with elevated troponin.

Conflict of Interest No conflicts of interest



Abstract 69 Figure 1 A) Adjusted association between troponin level and the hazard ratio for all-cause mortality using restricted cubic splines; B) Proportion of patients undergoing coronary revascularisartion according to troponin level; C) Association between troponin level and the hazard ratio for all-cause mortality using restricted cubic splines stratified by revascularisation status.
The shaded region shows the 95% confidence interval