164 ASSOCIATION BETWEEN AGE, TROPONIN LEVEL AND MORTALITY IN PATIENTS PRESENTING TO HOSPITAL WITH ACUTE PULMONARY EMBOLISM (NIHR HEALTH INFORMATICS COLLABORATIVE TROP-PE STUDY)

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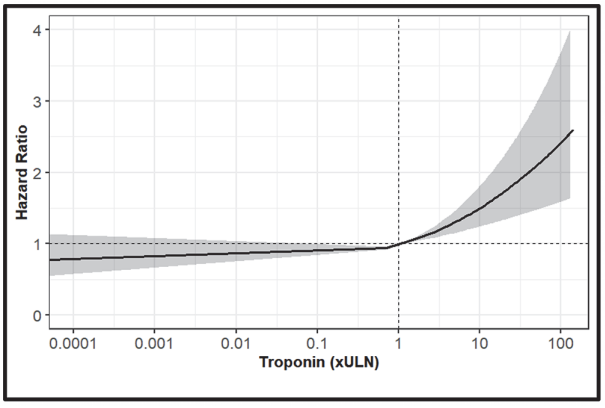
Background A positive cardiac troponin (cTn) is an independent predictor of short-term mortality in individuals presenting with acute pulmonary embolism (PE). However, there is limited evidence regarding the impact age has on the association between cTn levels and mortality in patients with PE. The aim of our study was to investigate the relationship between cTn level, age, and all-cause mortality, in hospitalised patients with a PE.

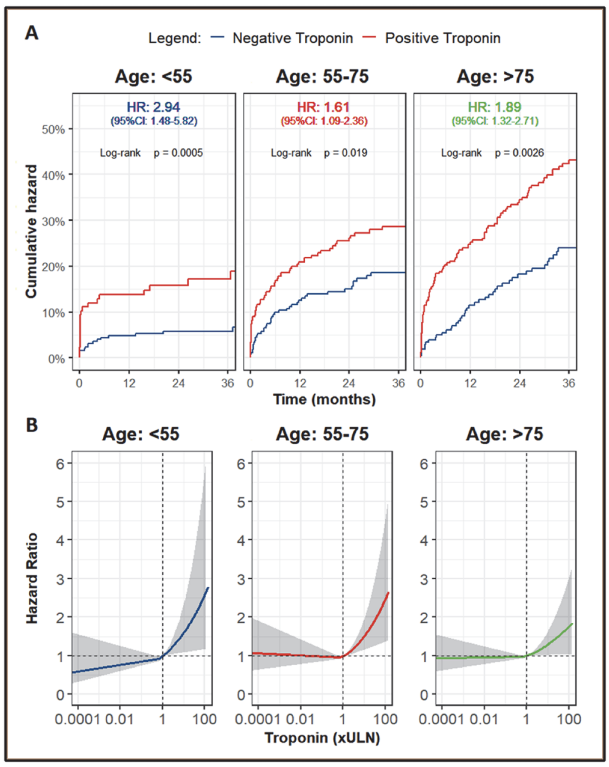
Methods A retrospective cohort study using the National Institute for Health Research Health Informatics Collaborative Cardiovascular dataset of all consecutive patients who had a troponin measured at five hospitals (Imperial, University College London, Oxford, King's and Guy's and St Thomas') between 2010 and 2017. Patients admitted to hospital with a primary diagnosis of PE with at least one cTn measurement were included. Survival analyses were performed using multivariate Cox-Regression analyses. The peak cTn level (highest level measured), standardised to the upper limit of normal (ULN), was used for all analyses.Results1,477 patients with at least one cTn measurement and a diagnosis of PE were included. During a median follow-up of 34.8 months, there were 290 (19.6%) deaths. Elevated cTn (>1xULN) was associated with mortality with a hazard ratio (HR) of 3.29 (95% confidence interval [CI] 1.95–5.53) for 30-day mortality and 2.12 (95% CI 1.63–2.75) for 3-year mortality. Higher cTn levels were progressively associated with a higher mortality risk, reaching a maximum HR of 2.59 (95% CI 1.64–4.09) at 141xULN (Figure 1). Younger patients (<55 years), compared with those aged over 55, had the highest 3-year HR associated with a positive cTn of 2.94 (95% CI 1.48–5.82) despite having the lowest troponin levels (mean 7.01xULN) on admission (Figure 2).

Conclusion Elevated cTn, at all ages, is associated with an increased mortality risk in patients presenting with PE, with increasing cTn levels conferring a progressively worse long-term prognosis. Elevated cTn, no matter how small, needs to be taken seriously, particularly in young patients with an acute PE.

Conflict of Interest No conflicts of interest



Abstract 164 Figure 1 Restricted Cubic Spline demonstrating relationship between standardised peak troponin level and hazard ratio



Abstract 164 Figure 2 Mortality risk associated with troponin status and continuous troponin level stratified by age

165 ASSOCIATION BETWEEN TROPONIN AND MORTALITY IN ACUTE STROKE (NIHR HEALTH INFORMATICS COLLABORATIVE TROP-STROKE STUDY)

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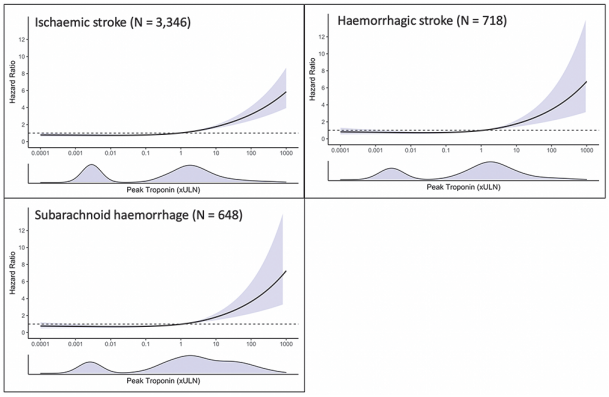
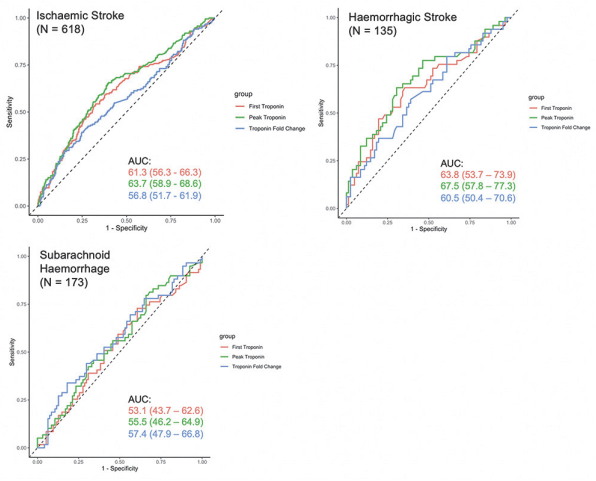


Abstract 165 Figure 1 Adjusted restricted cubic splines for the association between peak troponin and all-cause mortality at 6 months in patients with: A) ischemic stroke, B) haemorrhagic stroke, or C) subarachnoid haemorrhage, with corresponding density plots



Abstract 165 Figure 2 Time-dependent Receiver Operator Characteristic (ROC) curves for first troponin (red line), peak troponin (green line) and troponin fold change (blue line) for ischaemic stroke, haemorrhagic stroke and subarachnoid haemorrhage

Introduction Acute stroke accounts for significant morbidity and mortality globally. The role of troponin for risk stratification in stroke is unclear. The aims of this study were to assess the relationship between peak troponin and mortality in patients with ischemic stroke, haemorrhagic stroke, or subarachnoid haemorrhage and to compare this with the predictive value of first troponin or dynamic troponin change.
Methods A retrospective cohort study was carried out using the National Institute for Health Research Health Informatics Collaborative Cardiovascular dataset of all consecutive patients who had a troponin measured at five hospitals (Imperial, University College London, Oxford, King's and Guy's and St Thomas') between 2010 and 2017. Patients with at least one troponin measurement and a primary diagnosis of ischaemic stroke, haemorrhagic stroke or subarachnoid haemorrhage during a hospital admission were included. The main exposure variables were first and peak troponin, and dynamic troponin change, and the main outcome was all-cause mortality. Results were analysed using multivariable adjusted restricted cubic spline Cox regression. Receiver Operator Characteristic (ROC) curves were generated to assess the predictive value of each exposure variable.Results4,712 patients were included in the analysis (ischaemic stroke: 3,346; haemorrhagic stroke: 718; subarachnoid haemorrhage: 648). Peak troponin was above the upper limit of normal in 47.4% of ischaemic stroke patients, 52.8% of haemorrhagic stroke patients, and 57.1% of subarachnoid haemorrhage patients. Patients with elevated peak troponin were older and had more cardiovascular risk factors.A direct positive relationship was seen between peak troponin level and mortality hazard ratio in all three types of stroke (Figure 1). This relationship was consistent when considering dynamic troponin fold change for ischaemic or haemorrhagic stroke. For all three types of stroke, there was no added predictive value of peak troponin or dynamic troponin change over first troponin in predicting mortality (Figure 2).
Conclusions A positive peak troponin and positive first admission troponin are associated with increased mortality in patients presenting with ischaemic stroke, haemorrhagic stroke, and subarachnoid haemorrhage, while dynamic troponin change is associated with increased mortality only in patients with ischaemic stroke. Overall, serial troponin measurements may not improve mortality prediction beyond a single measurement. These findings may have implications for risk stratification of patients with acute stroke syndromes.
Conflict of Interest No conflicts of interest

166 IMPACT OF SEX ON THE INCIDENCE OF ATRIAL FIBRILLATION IN THE COMMUNITY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction Atrial fibrillation (AF) is the most common cardiac arrhythmia and is associated with significant morbidity and mortality. By 2060, it is estimated that 18 million adults over 55 years will have AF in Europe, and significant healthcare costs are projected to accompany this substantial increase in prevalence. Sex plays a major role in its pathogenesis, but conflicting results have been reported on whether the risk of new-onset AF differs between men and women. This systematic review and meta-analysis of epidemiological studies aims to compare the incidence of new-onset AF between men and women in the community setting.
Methods We searched PubMed Medline and Ovid Embase for longitudinal studies from their inception. We selected studies if they presented sex-specific incidence estimates of new-onset AF for participants without a prior history of AF recruited from the general population. We assessed the risk of bias from methodological quality using the Newcastle Ottawa Scale (NOS). We pooled data using a random-effects model with inverse-variance weighting approach, and carried out subgroup analyses to explore heterogeneity based on age and the sampling population. Risk ratio (RR) and 95% confidence interval (CI) were generated to compare the incidence of new-onset AF between men and women.
Results A total of 54 prospective cohort studies met the inclusion criteria and were systematically reviewed. Of these, 45 with data on a total of 10,530,623 participants (40% women) were included in the meta-analysis. Pooled analysis demonstrated that men had a statistically significant higher risk of developing new-onset AF than women (RR 1.53; 95% CI