172 ABSTRACT WITHDRAWN

173 PROGNOSTIC SIGNIFICANCE OF TROPONIN IN PATIENTS WITH MALIGNANCY (NIHR HEALTH INFORMATICS COLLABORATIVE TROP-MALIGNANCY STUDY)

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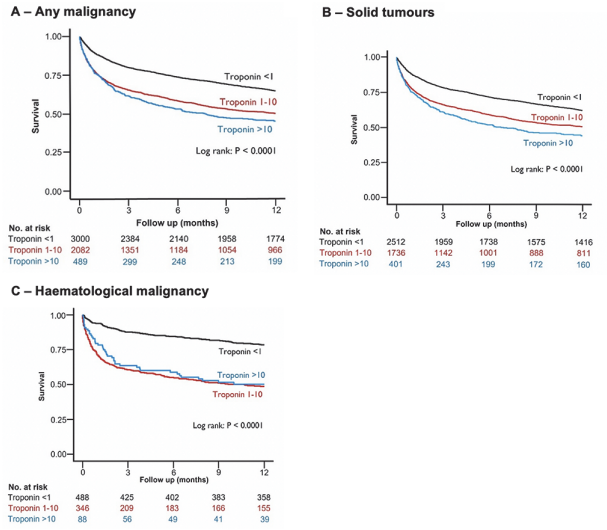
Background Cardiac troponin is commonly raised in patients with malignancy and may aid clinicians in risk prediction. The prognostic significance of raised troponin in these patients with known malignancies remains unclear. We sought to investigate the relation between troponin and mortality in a large, well characterised cohort of patients undergoing cardiac troponin testing with a concomitant malignancy. MethodsA 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 a primary inpatient diagnosis of malignancy who had at least one cTn measurement during their hospital stay were identified. Patients were classified into solid tumour or haematological malignancy subgroups. Survival analyses were performed using multivariate Cox-Regression analyses and Kaplan-Meier plots. The peak cTn level (highest level measured), standardised to the upper limit of normal (ULN), was used for all analyses.Results5571 patients undergoing troponin testing had a primary diagnosis of malignancy and comprised of twenty-one different cancer types. 4649 patients were diagnosed with solid tumours and 922 patients were diagnosed with haematological malignancies. Patients with raised troponin had a higher burden of cardiovascular comorbidities compared to patients with a troponin level below the ULN. The median follow-up in the cohort



Abstract 173 Figure 1 One-year Kaplan-Meier survival curves for different troponin levels in patients with (A) any malignancy, (B) solid tumours or (C) haematological malignancy

was 14 months (interquartile range 2–39 months). At 1-year follow-up, 2495 (42%) of patients died.Figure 1 shows Kaplan-Meier plots for patients stratified by troponin level. Patients with a troponin level >1xULN had a higher risk of death compared to patients with a troponin level <1xULN (Figure 1A). A similar trend was shown in cancer subtypes (Figure 1B-C). Raised troponin was an independent predictor of mortality in all patients with malignancy (adjusted hazard ratio 1.66, 95% confidence interval [CI] 1.52–1.81), in solid tumours (adjusted hazard ratio 1.63, 95% CI 1.48–1.81) and in haematological malignancy (adjusted hazard ratio 1.75, 95% CI 1.44 to 2.13) when compared to troponin level below the ULN.

Conclusion Raised troponin level was associated with increased mortality in patients with malignancy regardless of cancer subtype. Troponin may be more widely useful in the risk stratification of patients with cancer. Although the appropriate management of patients in response to raised troponin in the absence of acute coronary syndrome is not clear, stratification of clinical risk of mortality can be helpful in general decision making.

Conflict of Interest No conflicts of interest

174 CT CORONARY ANGIOGRAPHY SIGNIFICANTLY CHANGES TREATMENT TARGETS VERSUS CORONARY ARTERY CALCIUM SCORING IN HIGH-RISK DYSLIPIDAEMIA PATIENTS

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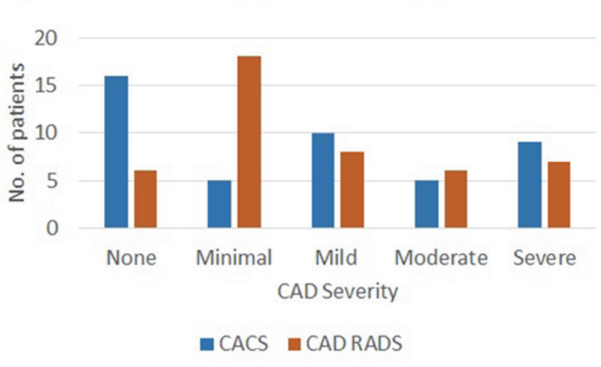
Introduction Dyslipidaemia accelerates atherosclerosis. Patients with genetic dyslipidaemias, Familial Hypercholesterolaemia

(FH) being the most common, are at heightened risk of premature cardiovascular events. However, this risk is heterogeneous within identical genotype diseases, and modifiable with treatment. Coronary imaging identifies subclinical atherosclerosis, personalises risk stratification and treatment targets. Coronary artery calcium scoring (CACS) is first-line for primary prevention. However, calcification is a late-stage process in CAD pathogenesis and the CACS has low specificity in young patients with severe FH. CT coronary angiography (CTCA) may identify non-calcific CAD and high risk plaque (HRP) features unseen with CACS. This study aimed to quantify the impact of CTCA vs traditional CACS on clinical management in real-world asymptomatic Lipid Clinic patients.

Methods A retrospective single-centre review of asymptomatic Lipid Clinic electronic patient records with both CACS and CTCA from May 2019 to December 2020. A vignette was compiled for each patient providing all relevant clinical data. CACS was recorded as Agastston score and CTCA as the Coronary Artery Disease - Reporting and Data System (CAD RADS) grading of anatomical stenosis with a modifier for HRP features.Findings were compiled into an anonymised online survey which Consultant Biochemists from across the UK were invited to complete. Data was revealed in a stepwise fashion to the participating clinician: (i) vignette only, (ii) CACS, and (iii) CAD RADS. Clinicians were asked their lipid target and management after each data-point was unblinded. Background information on CACS and CTCA result interpretation was provided prior to participation. Statistical analysis was performed using SPSS v.21 and significance was defined as two-tailed $p<0.05$.

Results 45 asymptomatic patients (55±9 years, 49% female) were included. 7 Consultant Biochemists from 6 institutions (4 [67%] tertiary/teaching hospitals and 2 [33%] district general hospitals) participated.CACS and CAD RADS assessment of disease burden is presented in Figure 1, with CTCA re-classifying CAD severity vs CACS in 28/45 (62%) patientsLipid targets were altered significantly more frequently with CTCA vs CACS (19% vs 12%; $\chi 2$ 57.0, $p<0.005$), even after CACS result available (Figure 2). The LDL target selected was altered by CACS in 12%, and in a further 19% when CAD RADS result was unblinded, which was statistically significant ($\chi 2$ 57.0, $p<0.005$). This finding was consistent across FH and non-FH patients. Increasing CACS and CAD RADS severity were significantly associated with change in lipid target ($\chi 2$ 54.2, $p<0.001$; $\chi 2$ 27, $p<0.001$), the latter even after a high CACS result was available, as did presence of HRP ($\chi 2$ 9.3, $p=0.002$).



Abstract 174 Figure 1 CAD severity breakdown by CACS vs CTCA

	CTCA		
CACS	n = 315	Target change	No change
	Target change	38 (12%)	0 (0%)
	No change	49 (16%)	228 (72%)

Abstract 174 Figure 2 Correlation table assessing impact of CACS vs CTCA on change in lipid target

Conclusion In high-risk asymptomatic dyslipidaemia, CTCA alters treatment targets beyond CACS by demonstrating higher CAD severity burden and HRP. This may differentiate high risk and very high risk patients in an important population.

Conflict of Interest Nil

175 UPSTREAM INVESTIGATION OF STABLE CHEST PAIN IN PATIENTS UNDERGOING INVASIVE CORONARY ANGIOGRAPHY: CONCORDANCE WITH NICE GUIDELINES AND LIKELIHOOD OF REVASCULARISATION

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Introduction The 2016 National Institute for Health and Care Excellence (NICE) CG95 guideline for investigation of patients with stable chest pain without known coronary artery disease (CAD) recommends CT coronary angiography (CTCA) for first-line investigation. For those with indeterminate CAD on CTCA, a non-invasive functional test is recommended, using myocardial perfusion scintigraphy, dobutamine stress echocardiography, stress MRI or CT-Fractional Flow Reserve (CT-FFR). Invasive diagnostic coronary angiography (iCA) is considered a 3rd line test for stable chest pain assessment in the Evidence Based Interventions Guidance from The Academy of Medical Royal Colleges. We assessed concordance with CG95 in patients attending for iCA and likelihood of subsequent revascularisation according to upstream investigation.

Methods All patients undergoing iCA at a UK tertiary referral cardiac centre between 1 March 2021 and 28 May 2021 were identified. Patients presenting with acute coronary syndromes, pre-existing CAD (defined as previous PCI or coronary artery bypass graft (CABG)), and those for coronary assessment prior to non-coronary cardiac surgery were excluded as outside the CG95 guideline scope.Hospital electronic records were accessed using patient NHS number. All tests and events in the 24 months prior to iCA were recorded. An extended workup time was used to allow for