Results RT-PCR found SGLT1 mRNA is expressed in whole myocardium and in individual cardiac chambers. SGLT1 expression was not detected in isolated cardiomyocyte but it is detected in the non-cardiomyocyte population. Cardiomyocytes were found to express mRNA SGLT1 if incubated overnight. RNAscope detected SGLT1 mRNA within intact myocardium: not in the cardiomyocyte, but rather in a perivascular distribution. Importantly, hyperglycaemia (22mmol) at reperfusion increased infarct size (51.80 ± 3.52% vs 40.80 ± 2.89%; p-value: 0.026) compared to normoglycaemia, and the mixed SGLT inhibitor, Phlorizin, significantly attenuated infarct size (from 64.7±4.2%to 36.6±5.8%; p-value<0.01) when given at reperfusion.

Conclusion We have shown that SGLT1 is present in the myocardium, but not expressed in cardiomyocytes. The cell type is yet to be determined, but the distribution of SGLT1 is perivascular. Hyperglycaemia appears augment myocardial infarction and inhibition of SGLT1&2 attenuates this increase. We suspect SGLT1 may plays a role in exacerbating the injurious effect of glucotoxicity during ischemia-reperfusion.

Conflict of Interest No

21 HIGH-SENSITIVITY CARDIAC TROPONIN CONCENTRATIONS AT PRESENTATION IN PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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Background The widespread adoption of high-sensitivity cardiac troponin testing has encouraged the use of pathways to accelerate the rule-out and rule-in myocardial infarction in the Emergency Department. These pathways are not recommended for

patients with ST-segment elevation, but there is a risk they may be applied incorrectly given that interpretation of the

electrocardiogram is subjective, dependent on experience, and signs may be masked in those with posterior myocardial infarction.

Methods Consecutive patients with suspected acute coronary syndrome were enrolled in a stepped-wedge cluster randomized

controlled trial across ten hospitals in Scotland. The index diagnosis was adjudicated two clinicians independently in all patients

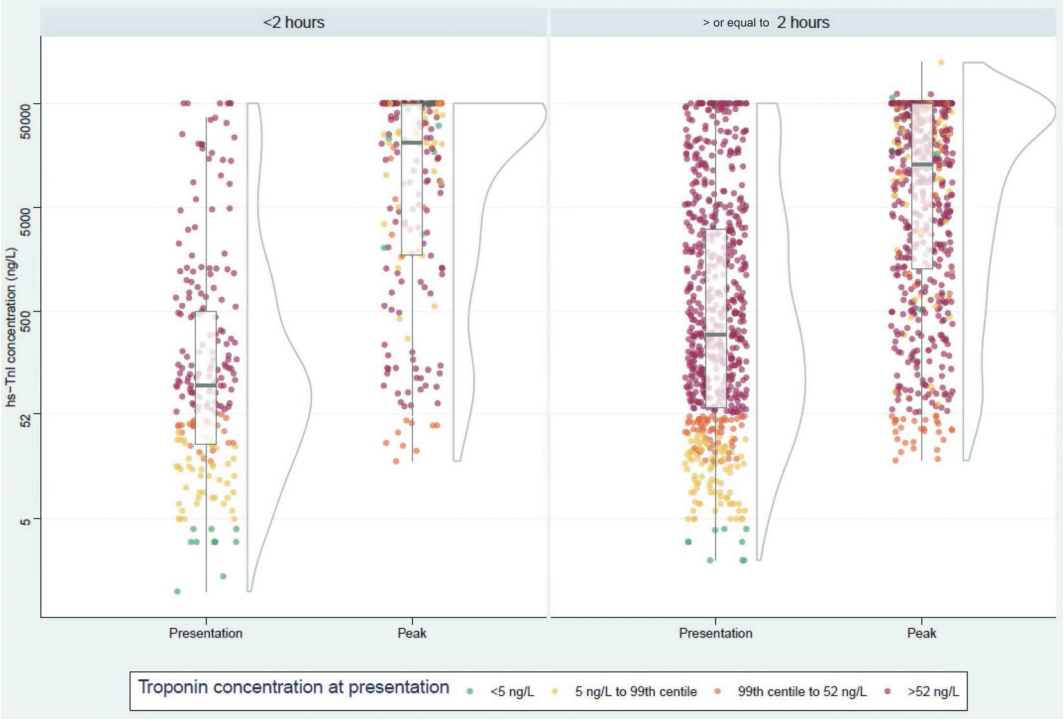
with high-sensitivity cardiac troponin I concentrations above the sex-specific 99th centile on serial testing and abnormalities on

the electrocardiogram recorded. The proportion of patients with ST-segment elevation myocardial infarction and concentrations

below the rule-out threshold (<5 ng/L), 99th centile (<16 ng/L and <34 ng/L for women and men) and rule-in threshold (<52 ng/L)

at presentation were determined. Secondary analysis determined the effect of symptom duration, and culprit vessel location, on troponin concentrations.

Results In total, we enrolled 48,282 consecutive patients with suspected myocardial infarction were enrolled, with 925 having an index diagnosis of STEMI. The majority (83.5%, 772/925) of patients had a troponin concentration above the 99th-centile on presentation. The median troponin concentration on presentation was 196 ng/L [46.0, 21611.], with 2.2% (20/925) and 14.4% (133/925) under the 5ng/L rule-out threshold, and <99th-centile respectively. Relying on a rule-in threshold of 52ng/L would miss more than 1 in 4 patients (26.8%, 248/925) with STEMI. Patients



Abstract 21 Figure 1

presenting soon after symptom onset had significantly lower troponin concentrations (<2 hours, median 96.0ng/L [26.0, 494.0] vs ≥2 hours, median 294.ng/L [59.0, 3042.0], p<0.001).

Discussion In patients with suspected acute coronary syndrome who have a final diagnosis of ST-segment elevation myocardial

infarction, high-sensitivity cardiac troponin concentrations are below the rule-out and rule-in threshold at presentation in more than 1 in 50

and 1 in 4 patients, respectively. Clinicians should not rely on cardiac troponin concentrations to guide initial treatment decisions

in patients with possible ST-segment elevation myocardial infarction.

Conflict of Interest None

22 DISTRIBUTION OF HIGH SENSITIVITY TROPONIN TAKEN WITHOUT CONVENTIONAL CLINICAL INDICATIONS IN CRITICAL CARE PATIENTS AND ITS ASSOCIATION WITH CRITICAL CARE MORTALITY

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Background High sensitivity troponin (hs-cTn) concentrations above the manufacturer's upper limit of normal (ULN) are frequently seen outside the context of MI, particularly in critical care patients. The potential value of hs-cTn as a biomarker for prognosis in CC patients has never before been systematically explored.

Aims 1. To describe both the distribution of hs-cTn in critical care and 2. the association of this distribution with clinical outcomes, including in hospital mortality.

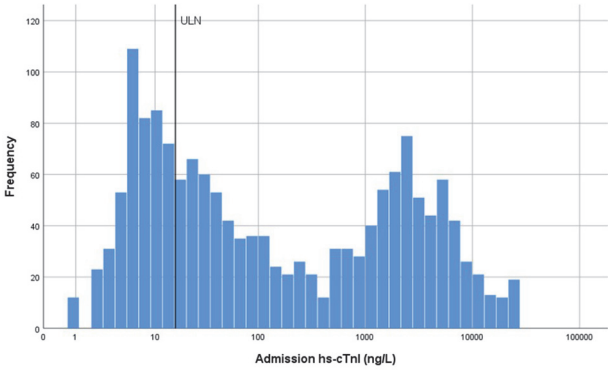
Methods Consecutive patients admitted to the three adult CC units (cardiothoracic (CCCU), general (GCCU), neuroscience (NCCU)) over a six month period had hs-cTnI tests performed serially throughout the admission, regardless of whether the supervising team had identified a clinical indication. Except when requested for clinical reasons, the results were nested and not revealed to patients or clinicians. The association between hs-cTnI concentration and clinical outcome was evaluated.

Results After excluding patients diagnosed with a type 1 MI, there were 1,563 patients remaining in the study cohort (CCCU 530, GCCU 750, NCCU 283). The median hs-cTnI was 77ng/L (IQR 11-1932ng/L), with 1081 (69.2%) patients above the manufacturer-provided ULN. Overall there was a bimodal distribution; GCCU and NCCU were positively skewed and CCCU negatively skewed (figure 1). Concentrations above the ULN were associated with increasing age, comorbidity, markers of illness severity and need for organ support (table 1). On multivariate analysis the degree by which the admission hs-cTnI concentration was above the ULN remained an independent predictor of critical care mortality (figure 2), but not length of stay for patients in NCCU and GCCU. Furthermore, the addition of the degree by which the hs-cTnI concentration was above the ULN to the Acute Physiology and Chronic Health Evaluation (APACHE) II score improved the area under the curve for critical care mortality (from 0.828 to 0.846 (p<0.001)). Peak hs-cTnI concentration and the degree of hs-cTnI change did not provide additional prognostic information compared with the admission hs-cTnI alone.

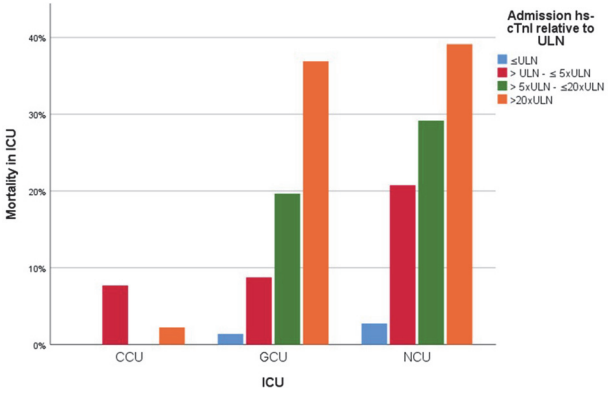
Conclusion Admission hs-cTnI concentration, taken regardless of a clinical indication, is an independent predictor of ICU mortality and provides additional discriminative ability to the APACHE II score alone. This assay may represent a novel prognostic biomarker on admission in critical care settings.

Abstract 22 Table 1 Admission characteristics, need for support, length of stay and death in NCCU/GCCU comparing patients with hs-cTnI above and below the ULN

	Admission hs-cTnI ≤ ULN (total 472)	Admission hs-cTnI > ULN (total 560)	p-value
Admission sequential organ failure assessment score	3 (1 – 6)	6 (4 -9)	<0.001
APACHE II score^	10 (6 – 16)	17 (10 – 22)	<0.001
Need for I &V	182 (38.6)	288 (51.4)	<0.001
Need for filter	11 (2.3)	60 (10.7)	<0.001
Need for vasopressor	146 (30.9)	302 (53.9)	<0.001
Length of stay	2 (1 – 3)	3 (1 – 6)	<0.001
Death in critical care	9 (1.9)	109 (19.5)	<0.001



Abstract 22 Figure 1 Distribution of hs-cTnI concentration across all CC environmental (log10 scale for hs-cTnI concentration)



Abstract 22 Figure 2 Mortality by hs-cTnI concentration relative to the ULN across the three CC environments