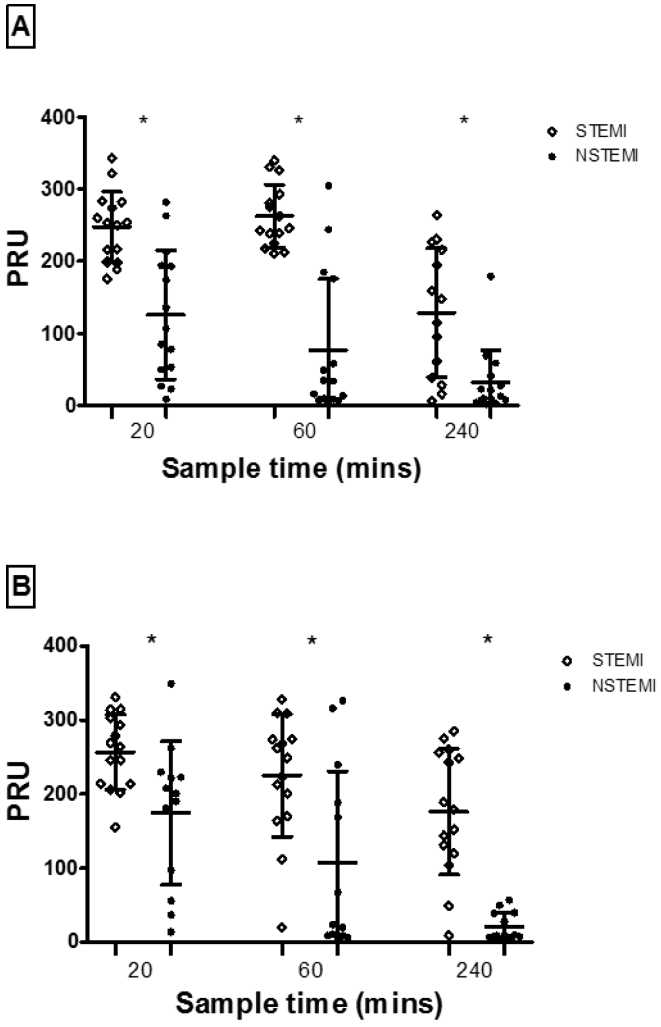

Abstract 109 Figure 1 The degree of inhibition of platelet reactivity (expressed as PRU) over time following the administration of prasugrel (A) and ticagrelor (B) in STEMI and NSTEMI patients. PRU = P2Y12 reactivity units

difference in the effect of ticagrelor vs prasugrel over time in either STEMI or NSTEMI/UA.

Conclusion Prasugrel and ticagrelor in the context of STEMI do not provide adequate P2Y12 inhibition at reperfusion and the first hour post loading when compared to patients with NSTEMI/UA.

Abstract 109 Table 1 Baseline characteristics

Characteristic	STEMI (N=30)	NSTEMI (N=28)	P-value
Age (yrs)	59.94 ± 12.68	61.61 ± 11	0.595
Female	7	3	0.301
Diabetes Mellitus	6	12	0.089
Hypertension	14	13	1
Current Smoker	7	4	0.508
Ex-Smoker	12	11	1
Hyperlipidaemia	8	16	0.032
Familial History of CAD	17	15	1

CAD = Coronary Artery Disease

110 THE PROTHROMBOTIC RISK OF PATIENTS WITH TYPE 2 DIABETES IN STABLE AND UNSTABLE CORONARY ARTERY DISEASE

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Aims/hypothesis Clot properties are altered in acute coronary syndromes (ACS). However, data on clot properties and the impact of concomitant disease and medication in patients with diabetes in ACS are incomplete. Therefore, the present study investigates clot parameters in stable and unstable coronary artery disease (SCAD and UCAD respectively).

Methods Hundred-eighty patients were included in a consecutive manner based on their diabetes and CAD status between March 2012 and December 2014. Clot properties were determined by a turbidimetric assay in 90 controls (noCAD N=39; SCAD N=29;, UCAD N=22) and 90 patients with diabetes (noCAD N=21; SCAD N=41; UCAD N=28).

Results Clot structure was not affected by CAD status. However, clot lysis time was significantly increased in UCAD compared to SCAD and absence of CAD in control patients (1414 ± 703, 915 ± 461 and 1069 ± 414 respectively; p = 0.003). In contrast, in patients with DM clot lysis time did not differ between UCAD, SCAD and absence of CAD (1260 ± 649, 1304 ± 658 and 1318 ± 675 respectively; p = 0.947). Interestingly, clot lysis time in diabetes patients without CAD was comparable to UCAD control patients (p = 0.654). In an adjusted multiple regression model clot lysis time was significantly predicted by PAI-1 (p = 0.023), CRP (p = 0.042) and presence of UCAD (NSTEMI p = 0.010, STEMI p = 0.002) in control patients. Strikingly, in diabetes patients solely PAI-1 (p = 0.004) predicted clot lysis time.

Conclusions Unstable coronary artery disease leads to an increase in clot lysis time in control patients. In contrast, clot lysis time in patients with diabetes is not affected by UCAD. Strikingly, clot lysis time in diabetes patients without CAD is comparable to control patients with UCAD indicating their increased prothrombotic risk already present in a stable situation.

111 THE DEGREE AND TIME COURSE OF PLATELET INHIBITION FOLLOWING THE ADMINISTRATION OF ORAL ANTIPLATELET AGENTS IN PATIENTS PRESENTING WITH ST ELEVATION MI (STEMI)

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Introduction Oral P2Y12 inhibitors have proven clinical efficacy in a variety of cardiological settings. The degree and time course of platelet inhibition using common P2Y12 inhibitors during the acute phase of a myocardial infarction is an under explored area. We aimed to determine the effect of clopidogrel, prasugrel and ticagrelor in the first four hours following loading in patients admitted with STEMI undergoing primary percutaneous coronary intervention (PPCI).

Abstract 111 Table 1 Baseline Characteristics

Characteristic	Clopidogrel (N=13)	Prasugrel (N=15)	Ticagrelor (N=15)	P-value
Age (yrs)	78À9.7	56À12.95	67.3À11.59	<0.001
Female	6	3	4	0.302
Diabetes Mellitus	0	4	2	0.127
Hypertension	6	7	7	1
Current Smoker	2	3	4	0.761
Ex Smoker	4	8	4	0.271
Hyperlipidaemia	4	5	3	0.691
Familial History of CAD	6	9	8	0.765

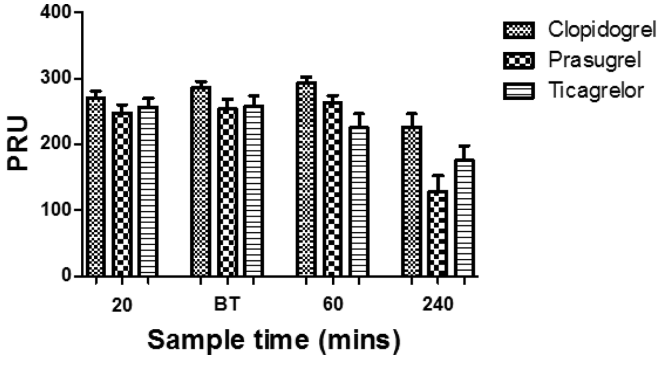
CAD = Coronary Artery Disease

Methods A single centre non-randomised study in patients presenting with STEMI. All patients gave informed consent. Enrolled patients were administered a loading dose of aspirin 300mg and then either clopidogrel 600mg, prasugrel 60mg or ticagrelor 180mg prior to percutaneous coronary intervention. Platelet reactivity was measured using a Verify Now assay at 20 minutes post loading, first balloon inflation, 1 and 4 hours post loading. Results are expressed a P2Y12 reaction units (PRU). PRU $\geq$ 208 indicates poor antiplatelet response. The effect of the three drugs over time were assessed using 2 way ANOVA. $P < 0.05$ was taken as a significant result.

Results A total of 43 STEMI patients were recruited to the study. Refer to Table 1 for baseline characteristics. PRU results for the 3 drugs at 20 minutes, balloon time, 60 minutes and 240 minutes were determined. Mean dose to balloon time was 26.8 + 12.7. At balloon time, 20 minutes and 60 minutes none of the agents achieved a PRU < 208. There was a significant difference over time between clopidogrel and ticagrelor ($P = 0.04$), clopidogrel and prasugrel $P < 0.0001$ and no statistical difference was found when ticagrelor was compared with prasugrel over time (Figure 1).

Conclusion Clopidogrel in the context of STEMI does not provide adequate platelet inhibition as evidenced by PRU > 208 at all data collection time points, Both prasugrel and ticagrelor are superior to clopidogrel resulting in a significant reduction in the degree of platelet reactivity.

Although Prasugrel and ticagrelor are comparable in terms of inhibition of platelet activity at 20 minutes, balloon inflation, 1 hour and 4 hours, they still do not achieve the desired levels of platelet inhibition necessary during PPCI as demonstrated by PRU > 208. It is possible that fast acting intravenous antiplatelet agents may have a role to play in achieving adequate platelet inhibition in the context of PPCI.



Abstract 111 Figure 1 The degree and time course of platelet inhibition (expressed as PRU) over time following the administration of clopidogrel, prasugrel or ticagrelor in STEMI patients. PRU = P2Y12 Reactivity Units

112 HIGH-SENSITIVITY CARDIAC TROPONIN, PRE-TEST PROBABILITY AND THE DIAGNOSIS OF MYOCARDIAL INFARCTION: A PROSPECTIVE COHORT STUDY

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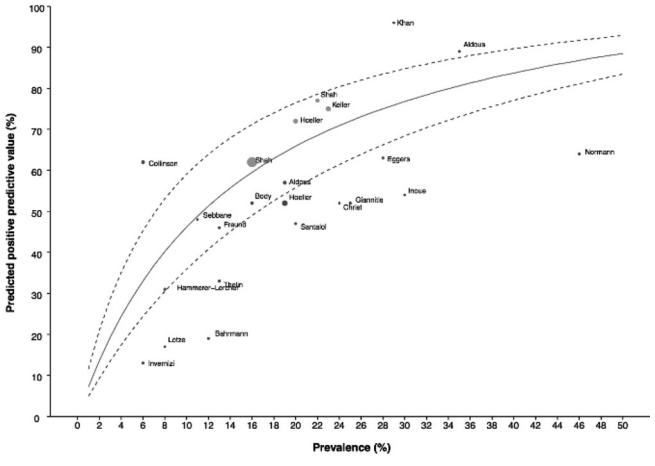
Background Cardiac troponin is integral to the diagnosis of myocardial infarction, but is elevated in many patients without acute coronary syndrome. We evaluate the impact of a clinical assessment of pre-test probability on the positive predictive value (PPV) of cardiac troponin for a diagnosis of myocardial infarction.

Methods In 1,054 consecutive patients undergoing blood sampling in the Emergency Department, cardiac troponin I was measured in excess serum using a high-sensitivity assay. Patients were classified as type 1 or type 2 myocardial infarction, or myocardial injury, and the prevalence determined in the entire cohort and in those where testing was requested by the attending physician. We determine the predicted PPV for type 1 myocardial infarction across different pre-test probabilities.

Results Cardiac troponin was requested in 136 patients identifying 15 (11.3%) with type 1 myocardial infarction. Testing everyone identified 2 further patients with type 1 (17/1,054 [1.7%]), but 127 (12%) patients with type 2 myocardial infarction or myocardial injury. In patients selected for testing the PPV for type 1 myocardial infarction was 50.0% (95% confidence interval [CI] 32.8%-67.2%), falling to 13.6% (95% CI 8.0%-19.7%) when measured across the cohort. The PPV was heavily influenced by the pre-test probability (Figure 1). Irrespective of diagnosis, cardiac troponin was a independent predictor of death (hazard ratio 1.26 per 2-fold increase, 95% CI 1.06 to 1.49) (Figure 2).

Conclusions Cardiac troponin concentrations are elevated in one in eight patients in the Emergency Department. If testing



Abstract 112 Figure 1