

## 216 DOES AORTIC STENOSIS EFFECT PLATELET CLOSURE TIME AND VON WILLEBRAND FACTOR ACTIVITY?

<sup>1</sup>Ferrah Choudhary\*, <sup>2</sup>Richard Kirby, <sup>2</sup>Christina Peter, <sup>2</sup>Robert Henderson. <sup>1</sup>Nottingham City Hospital; <sup>2</sup>Nottingham University Hospitals NHS Trust; \*Presenting Author

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**Background** Abnormal platelet function is associated with increased bleeding risk. Limited evidence suggests that platelet dysfunction is a cause of bleeding in patients with aortic stenosis (AS).

**Method** 40 patients with degenerative AS underwent detailed haematological assessment. Platelet closure time (PCT) in seconds, was measured from a citrated blood sample by a PFA 100 benchtop analyser. In the presence of standardized flow conditions and collagen and ADP agonists, high shear rates are created, leading to aggregation of platelets and subsequent occlusion at the aperture site. The PCT is recorded as the time taken for the aperture occlusion to develop. Mean platelet volume (MPV) and platelet count (as per the impedance method) were measured from an EDTA sample by a XE2100 analyser. Von Willebrand factor activity was assessed with collagen binding assay (vWF:CB). Each patient completed a questionnaire to document use of antiplatelet therapy (with aspirin/clopidogrel) and bleeding events (using the ISTH bleeding questionnaire).

**Results** Of the 40 patients, 8 had mild AS, 23 moderate AS and 9 severe AS. The average age was 74.9 years (range 39–97) and 47.5% were male. All patients had a normal MPV. A low platelet count was noted in 3 patients and all had an associated prolonged PCT. VWF activity was abnormal in 60% (n = 24: 5 mild AS, 12 moderate, 7 severe) but there was no correlation with AS severity.

Overall, 25 patients (62.5%) had a prolonged PCT, including 5 patients (1 mild AS, 1 moderate AS and 3 severe AS) with significant bleeding events (2 patients with gastrointestinal bleeding and 3 with epistaxis). All bleeding events occurred within 3 months of the haematological assessment and in 2 cases were ongoing when the patient completed the questionnaire. Only 1 patient with normal PCT had significant bleeding. A higher PCT was associated with more severe AS (p = 0.002, Table 1) and with the use of antiplatelet therapy (p = 0.043). Patients without antiplatelet therapy (n = 18) had a mean PCT of 126.5 (SD 36.94) vs patients on antiplatelet therapy, mean PCT of 174.5 (SD 82.15). There was no statistically significant relationship between PCT and vWF activity (p = 0.16).

Abstract 216 Table 1

Severity of AS	Mild	Moderate	Severe
Number of Patients	8	23	9
Presence of Anaemia (% and range)	25% (109–118 g/L)	39.1% (90–126 g/L)	33% (75–127 g/L)
Platelet Closure Time in seconds (mean and SD)	163.75 SD 72.84	109.5 SD=25.13	215.44 SD 81.80
Prolonged Closure Times (%)	50%	52.2%	100%
Antiplatelet Use in Patients with Prolonged CT (%)	50%	41.7%	77.8%
Abnormal vWF:CB assay (%)	50%	63.6%	55.6%

\*Reference ranges: Normal Hb levels in women = 115–165 g/L and in men = 130–180 g/L. PFA closure times with collagen/ADP = 71–106 s

**Conclusion** This study demonstrates that PCT is associated with AS severity but this association may be partly explained by an excess of antiplatelet therapy use in patients with severe AS. In this small sample there was no association between platelet closure time and abnormal vWF activity. Ongoing studies will explore these findings in greater detail.

## 217 DIFFERENCES IN BLOOD BIOMARKER COMPOSITION BETWEEN PAROXYSMAL AF AND SINUS RHYTHM PATIENTS, WITHOUT HEART FAILURE

<sup>1</sup>Samantha Tull\*, <sup>2</sup>Elton Dudink, <sup>3</sup>Bob Weijs, <sup>4</sup>Syeda Nashitha Kabir, <sup>4</sup>Larissa Fabritz, <sup>5</sup>Harry J Crijns, <sup>4</sup>Paulus Kirchhof. <sup>1</sup>University of Birmingham; <sup>2</sup>MUMC, Department of Cardiology; <sup>3</sup>Maastricht University Medical Center; <sup>4</sup>Institute of Cardiovascular Sciences, University of Birmingham; <sup>5</sup>Maastricht University Medical Center Department of Cardiology, Cardiovascular Research Institute; \*Presenting Author

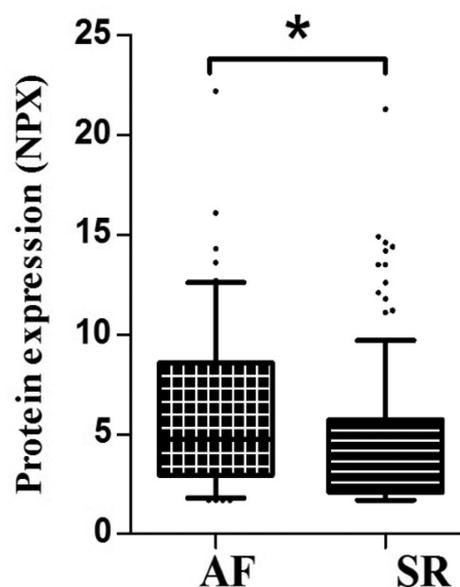
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**Introduction** Atrial fibrillation (AF) affects 2% of the population and is associated with cardiovascular disease and increased stroke and mortality rates. The myocardium releases proteins (SCF, VEGF-D and BNP). Detection of such markers in blood could be used to differentiate specific types of AF, or to guide screening for silent, undiagnosed AF.

**Aim** To identify plasma proteins discerning between patients with and without AF.

**Methods** We studied blood from consecutive patients undergoing CT coronary angiography at Maastricht Medical center. We only enrolled patients without a history of stroke, hypertension, diabetes or heart failure. Using unique DNA-coupled paired antibodies and qPCR, we simultaneously analysed 92 plasma proteins. CCL21 protein was measured by ELISA (n = 240). Stats: Mann-Whitney test, mean normalized protein expression (NPX) +/-SEM.

**Results** 176 patients (paroxysmal AF=50, sinus rhythm (SR) controls=126) were analysed. Mean age was 54 years in both groups. N-terminus terminal fragment proB-type natriuretic peptide



Box and whisker graph with median (line), interquartile range (box) and 10-90% (whiskers)

Abstract 217 Figure 1 Plasma NT-pro-BNP

(NT-proBNP) protein was higher in patients with than in those without AF ( $6.23 \pm 0.62$  vs  $4.72 \pm 0.33$ ,  $p = 0.012$ ,  $n = 176$ ). We also performed an exploratory analysis only in patients without signs of coronary artery disease on CTCA (AF=27, SR=80). In this subgroup, NT-proBNP ( $6.478 \pm 0.8442$  vs  $4.554 \pm 0.4122$ ,  $p = 0.023^*$ ), BNP ( $1.111 \pm 0.083$  vs  $0.9713 \pm 0.029$ ,  $p = 0.0175^*$ ), Stem Cell Factor (SCF,  $162.8 \pm 7.860$  vs  $140.4 \pm 5.127$   $p = 0.0097^{**}$ ) and VEGF-D ( $47.44 \pm 2.708$  vs  $41.38 \pm 1.694$   $p = 0.0389^*$ ) were higher in the 27 patients with AF.

**Conclusion** While NT-proBNP is mostly known as a marker for heart failure, NT-proBNP appears as a potential blood marker for AF in patients without history of stroke, hypertension, diabetes or heart failure. Further validation of these initial, hypothesis-generating results seems warranted.

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#### THE RELATIONSHIP BETWEEN CAROTID ARTERY INTIMAL-MEDIAL THICKNESS AND LEFT VENTRICULAR FUNCTION BY SPECKLE TRACKING ECHOCARDIOGRAPHY IN PATIENTS WITH CORONARY ARTERY DISEASE

Hanan Radwan, Tamer Mostafa\*, Abdelhakem Seleem, Ekhlas Hussein. *Faculty of Medicine Zagazig University Hospital; \*Presenting Author*

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**Background and aim of the work** Since cardiovascular diseases are associated with high mortality and generally undiagnosed before the onset of clinical findings, there is a need for a reliable tool for early diagnosis. Carotid intima-media thickness (CIMT) is a non-invasive marker of coronary artery disease (CAD) and is widely used in practice as an inexpensive, reliable method. Left ventricular (LV) function can be accurately assessed by 2D speckle-tracking strain echocardiography (2D-STE). In our study, we aimed to investigate the relationship of CIMT and LV function assessed by 2D-STE in patients with stable (CAD) and the ability of 2D-STE and CIMT to predict significant CAD.

**Methods** Cross sectional study included 40 patients with history suspected stable angina pectoris, normal LV ejection fraction. All patients were examined by 2D-STE, carotid ultrasound, and coronary angiography (CA). 2D-STE was performed in the 3 apical projections. Peak regional longitudinal systolic strain was measured in 17 myocardial segments and averaged to provide global longitudinal peak systolic strain (GLS). LVGLS results were compared with CA findings in a receiver operating curve (ROC) to determine the cut-off for normal and abnormal strain values. The calculated optimal strain value was compared to mean CIMT measurements. The patients were divided into two groups according to the result of the CA: group 1 (29 patients) with significant coronary lesion  $> 70\%$ , and group 2 (11 patients) having at least one lesion more than  $50\%$  within the main branches of the coronary arteries.

**Results** GLS was significantly lower in patients with CAD+ (group 1) compared to patients without CAD- (group 2) [ $-11.86 \pm 2.89\%$  versus  $-18.65 \pm 0.79\%$ ]  $P < 0.001$ . ROC curve between GLS and CA showed cut-off value for LVGLS was less than  $-15.6\%$  for prediction of significant CAD with AUC = 0.878; 95% CI 0.78–0.96  $p = 0.00$ . The diagnostic performance of GLS for detecting severity of CAD was [sensitivity 93.1%, specificity 81.8% and accuracy 90%]. The mean

CIMT was  $1.49 \pm 0.35$  mm in group 1, vs  $0.75 \pm 0.3$  mm in group (2) ( $p = 0.000$ ). ROC curve between mean CIMT and CA showed cutoff value for mean CIMT was  $> 1.1$  mm for prediction of significant CAD with AUC= 0.871 (95% CI 0.79–0.97,  $p = 0.00$ ). There was a significant, nearly linear correlation between IMT and GLS and advancing CAD ( $p = 0.00$ ), as there was incremental significant increase in CIMT and decrease of GLS with increasing number of coronary vessels involved. Further analyses showed that GLS was highly significant negatively correlated with mean CIMT.

**Conclusion** GLS assessed by 2DSTE at rest was predictor of significant CAD; So 2DSE seems capable of identifying high-risk patients. Increased carotid IMT values were associated with decreased LV function assessed by 2D strain measurements and the presence and severity of CAD. So these findings support the use of carotid IMT measurements as marker of subclinical LV dysfunction and to predict risk and severity of coronary heart disease.

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#### HIGHLY SELECTIVE TROPONIN T (HSTNT) AND HEART-TYPE FATTY ACID-BINDING PROTEIN (H-FABP) AS MARKERS OF TYPE 4A MYOCARDIAL INFARCTION AND ADVERSE EVENTS IN ELECTIVE PERCUTANEOUS CORONARY INTERVENTION (PCI)

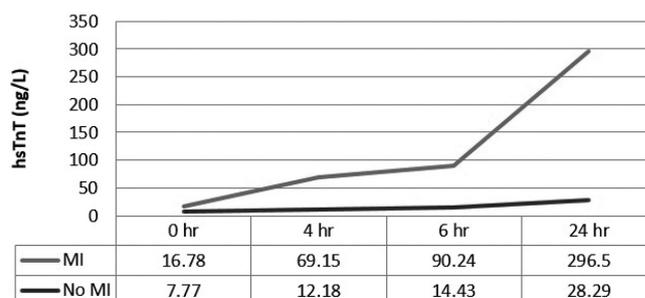
Michael Connolly\*, James Shand, Michelle Kinnin, Ian Menown, David Mc Eneaney. *Craigavon Area Hospital; \*Presenting Author*

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**Introduction** Heart-type Fatty Acid-Binding Protein (H-FABP) may be useful for early diagnosis of ACS1,2 and has been associated with increased cardiovascular events. Type 4a procedural myocardial infarction (MI) may occur after percutaneous coronary intervention (PCI).<sup>3</sup> Little is known about the use of early biomarkers as predictors of cardiovascular events following elective PCI.

**Methods** We prospectively evaluated highly sensitive troponin T (hsTnT), H-FABP, troponin I (TnI), creatine kinase MB type (CKMB), myoglobin, glycogen phosphorylase BB (GPBB) and carbonic anhydrase III (CAIII) at 0, 4, 6 and 24 h following elective PCI. Baseline demographic and cardiac risk factors were recorded. The primary endpoint was type 4a MI, diagnosed as a rise of  $>5 \times 99^{\text{th}}$  upper reference limit (URL) of 14 ng/L (i.e. rise of  $>70$  ng/L) at 6 h if hsTnT was normal at baseline or  $> 20\%$  from 0 to 6 hrs if hsTnT was  $>14$  ng/L at baseline.<sup>3</sup> Patients were followed up at 1 year to assess for

#### Median hsTnT release in type 4a MI and no type 4a MI



**Abstract 219 Figure 1** hsTnT release between type 4a MI ( $n = 37$ ) and no type 4a MI ( $n = 172$ )