Abstracts

17 A CASE-CONTROL STUDY WITH COMPUTATIONAL MODELLING OF ACUTE LEFT VENTRICULAR DYSFUNCTION

H Gao, 1,2,3 K Mangion, 2,3 D Carrick, 2 X Luo, 2,3 C Berry. 1 School of Mathematics and Statistics, University of Glasgow, UK; 2 Institute of Cardiovascular and Medical Sciences, University of Glasgow, UK; 3 Golden Jubilee National Hospital, Clydebank, UK

Introduction The pathophysiology of acute left ventricular dysfunction after acute ST-elevation myocardial infarction (STEMI) is incompletely understood. In this study, we carried out a case-control study of left ventricular (LV) biomechanical behaviour in vivo using an immersed boundary-finite element (IB/FE) method.

Methods Cardiac magnetic resonance images were acquired from six healthy volunteers (the healthy group) and six patients with acute LV dysfunction post-acute STEMI (the MI group) with no-reflow, defined as an acute reduction in myocardial blood flow despite a patent epicardial coronary artery. The two groups were age matched. The passive and active myocardial parameters were determined from the measured in vivo data (LV cavity volume and strain) to match the LV dynamics in diastole and systole.

Results Student’s t-test was used for significance test. Our results show that even though the average measured peak cuff-pressure in the MI group is much lower than the healthy group (122 ± 21 vs. 150 ± 20 mmHg, p < 0.05), the peak systolic active tension in the MI group is higher (68.9 ± 6.8 vs. 64.7 ± 8.4 kPa, p = 0.15), therefore the required active tension per mmHg increase is significantly higher in the MI group (0.57 ± 0.05 vs. 0.43 ± 0.05 kPa/mmHg, p < 0.01), which may suggest that the viable myocardium in the MI group is working harder to compensate for the loss of contraction even with much lower blood pressure. Compared to the healthy group, the MI group also has a larger end-diastolic volume (143 ± 23 vs. 122 ± 16 mL, p = 0.09), and a lower ejection fraction (39 ± 3% vs. 53 ± 2%, p < 0.01).

Conclusions In summary, we have shown for the first time that, compared with controls, patients with recent ST-elevation myocardial infarction exhibit increased active tension generation in combination with predictable changes in end-diastolic volume and ejection fraction. Active tension generation is a novel biomechanical index, and its clinical and prognostic significance merits further study.

Funding Beatson Oncology Centre Fund

18 CINE-DERIVED STRAIN USING THE GLASGOWHEART METHOD

K Mangion, 1 H Gao, 1 A Radjenovic, 1 X Luo, 1 C Haig, 1 C Berry. 1 Beatson Oncology Centre Fund; 2 Beatson Oncology Centre Fund

Background The GlasgowHeart method is designed to overcome some limitations of currently available feature-tracking methods by incorporating all of the myocardial tissues using an in-house developed intensity-based b-spline deformable registration method. The aim of this pilot study was to ensure that peak circumferential strain (Ecc) estimation is feasible and reproducible with minimal intra- and inter-observer variability.

Methods 20 healthy volunteers underwent 1.5T CMR twice, < 2 days apart. Mid-LV cine sequences, were analysed with the Glasgowheart software (Figure 1). Two observers independently analysed 40 short axis slices for inter-observer variability. One observer reanalysed the 40 short axis slices. Pearson correlation and Bland-Altman analysis were used.

Results 20 participants were used in the analysis (mean age ± SD 49.5 years (17.2) 50% male). Ecc measured on the first set of MRIs by the two observers was highly correlated (R = 0.915, p < 0.001) and in excellent agreement (mean difference = 0.01; 95% LoA: −0.01, 0.02). Repeated image analysis also disclosed a high degree of association in paired measurements of Ecc that was strongly correlated (R = 0.915, p < 0.001) and in excellent agreement (mean = 0.00; 95% LoA: −0.01, 0.02). Ecc measured in the second set of MRIs by 2 observers was well correlated (R = 0.937, p < 0.001) and in excellent agreement (mean = 0.00; 95% LoA: −0.016 and 0.021). The repeated image analysis at follow-up yielded Ecc that was well correlated (R = 0.942, p < 0.001) and in excellent agreement (mean = 0.00; 95% LoA: −0.009 and 0.009). There was no difference between the average global Ecc at different time points (p > 0.05).

Abstract 16 Figure 1 Boxplots of global myocardial perfusion index values by time point