Scientific poster presentations

P1 ^{99M}TC-SINGLE DOMAIN PD-L1 ANTIBODY (NM01): A NOVEL IMAGING BIOMARKER FOR NON-INVASIVE MYOCARDIAL PD-L1 EXPRESSION

[1,2]Muhummad Sohaib Nazir, [3]Daniel Hughes, [4]Hong Hoi Ting, [4]Gitasha Chand, [5]Damion Bailey, [5]Victoria Gibson, [5]Kathryn Adamson, [5]Jessica Johnson, [2,6]Alexander R Lyon, [3]Gary JR Cook. *[1]Department of Cardiovascular Imaging, School of Biomedical Engineering and Imaging Sciences, King's College London, UK; [2]Royal Brompton Hospital, Guy's and St Thomas Hospital NHS Trust, London, UK; [3]Department of Cancer Imaging, School of Biomedical Engineering and Imaging Sciences, King's College London, UK; [4]NanoMab Technology Ltd, UK; [5]Department of Nuclear Medicine, Guy's and St Thomas' NHS Foundation Trust, London, UK; [6]National Heart and Lung Institute, Imperial College London, London, UK*

10.1136/heartjnl-2022-BSCI.6

Objective Immune checkpoint inhibitors have revolutionized the care of cancer patients and involve blockade of the PD-L1 receptor. Myocardial PD-L1 expression has been implicated in mice models in myocarditis and autoimmune dilated cardiomyopathy. PD-L1 myocardial expression may have a potential role in immune checkpoint inhibitor myocarditis. We aimed to determine the non-invasive assessment of myocardial PD-L1 expression using 99mTc-single domain PD-L1 antibody (NM01) SPECT-CT.

Methods Cancer patients (n=10) underwent two SPECT-CT scans, at baseline and following 9 weeks cancer therapy. Patients were administered with a novel radiotracer, 99mTc single domain PD-L1 antibody (NM-01), prior to imaging. Baseline and 9-week left ventricular to blood pool ratios (LVmax:BP) and right ventricular to blood pool ratios (RVmax:BP) were measured. Measurements were repeated following a three-month period.

Results Myocardial PD-L1 expression was evident in all patients. Mean LVmax:BP values were 2.9±0.63 at baseline vs 2.60±0.82 at 9-week, (p=0.37). Mean RVmax:BP were 1.74 ±0.28 at baseline vs 1.68±0.51 at 9-week, (p=0.71). There was excellent reliability of measurements with intraclass correlation coefficient of 0.99 (95% confidence interval 0.94–0.99, p<0.001). The mean bias of measurements was 0.08±0.23 with 95% limits of agreement -0.38 to 0.54. There were no major adverse cardiovascular events or myocarditis during follow up.

Conclusion We demonstrate the first in man study for a novel quantitative imaging biomarker for the assessment of PD-L1 expression of the heart. This finding will now facilitate future studies to allow us to determine the role of PD-L1 in a range of cardiac disease such as myocarditis and cardiomyopathies.

P2 BSCI/BSTI CORONARY ARTERY CALCIFICATION GUIDELINES CHANGE MANAGEMENT AND PREDICT PROGNOSIS REGARDLESS OF AGE

[1,2]John Graby, [3]Jimena Soto-Hernaez, [1]David Murphy, [1]James LAF Oldman, [4]Pia FP Charters, [4]Tim A Burnett, [1]Thuvaarahan Thanaraaj, [3]Adam Barrishi, [5]Benjamin J Masterman, [1]Ali Khavandi, [2,4]Jonathan CL Rodrigues. *[1]Department of Cardiology, Royal United Hospitals Bath NHS Foundation Trust, Combe Park, Bath, UK; [2]Department for Health, University of Bath, Claverton Down, Bath, UK; [3]School of Medicine, University of Bristol, Tyndall Avenue, Bristol, UK; [4]Department of Radiology, Royal United Hospital, Combe Park, Bath, UK; [5]Yeovil District Hospital NHS Foundation Trust, Higher Kingston, Yeovil, UK*

10.1136/heartjnl-2022-BSCI.7

Objective BSCI/BSTI guidelines recommend reporting coronary artery calcification (CAC) on all CTCHEST regardless of indication. This study assessed CAC prevalence, prognosis and potential clinical impact of its reporting in routine CTCHEST.

Methods Single-centre retrospective analysis of consecutive CTCHEST (January-December 2015) for 200 patients per age group (<40, 40–49, 50–59, 60–69, 70–79, 80–89, ≥90). CTs were re-reviewed for CAC presence and severity, excluded if prior coronary intervention. Comorbidities, statin prescription and clinical outcomes (myocardial infarction [MI], stroke and all-cause mortality) were recorded. Impact of reporting CAC was assessed against pre-existing statin indication/prescription.

Results 1344 were included (mean age 63±20 years, 56% female). Inter- and intra-observer variability for CAC presence (ICC 0.95, p<0.001; ICC 1.0, p<0.001) and severity (ICC 0.92, p<0.001; ICC 1.0, p<0.001) was excellent. CAC was observed in 728/1344 (54%), more frequently in males (p<0.001) and rising age (p<0.001). Severity increased with age (p<0.001). A high proportion of patients in all age groups with CAC had no prior statin indication/prescription (42% of 80–89 to 100% of <40). 'Number needed to report' to potentially impact management (all age groups) was 2. 691 (51%) patients died (median follow-up 73 months). CAC presence was associated with risk of MI, stroke and all-cause mortality (p<0.001). After adjusting for confounders, severe calcification predicted risk of all-cause mortality (HR 1.6 [1.1–2.2], p=0.01).

Conclusion Grading of CAC was reproducible, and though prevalence rose with age, prognostic and treatment implications were maintained in all ages. Reporting CAC provides a simple opportunity to risk-stratify patients for cardiovascular risk optimisation.

P3 NOVEL INSIGHTS INTO DIMINISHED CARDIAC RESERVE IN HYPERTROPHIC CARDIOMYOPATHY FROM 4D FLOW CMR COMPONENT ANALYSIS

[1]Z Ashkir, [2]AJ Lewandowski, [3]CJ Carlhäll, [1,4]E Wicks, [1]S Johnson, [5]A Hess, [1]M Mahmod, [1]S Myerson, [3]T Ebbers, [6]H Watkins, [1]S Neubauer, [1]B Raman. *[1]Oxford Centre for Clinical Magnetic Resonance Imaging (OCMR), University of Oxford, Oxford, UK; [2]Oxford Cardiovascular Clinical Research Facility (CCRF), University of Oxford, Oxford, UK; [3]Division of Diagnostics and Specialist Medicine, Department of Health, Medicine and Caring Sciences, Linköping University, Linköping, Sweden; [4]Inherited Cardiovascular Conditions (ICC) Service, Oxford University Hospitals NHS Foundation Trust and the University of Oxford, Level 6, West Wing, John Radcliffe Hospital, Headley Way, Headington, Oxford, UK; [5]Nuffield Department of Clinical Neurosciences (NDCN), University of Oxford, Oxford, UK; [6]Division of Cardiovascular Medicine, Radcliffe Department of Medicine, University of Oxford, Oxford, UK*

10.1136/heartjnl-2022-BSCI.8

Objective Ventricular flow component analysis using 4D-flow cardiac magnetic resonance (4D-flow CMR) is a novel approach permitting comprehensive haemodynamic evaluation. Abnormal patterns of flow component distribution have been described in dilated and ischaemic cardiomyopathies, and have been linked to functional limitation. This study aimed to characterise ventricular flow component changes in hypertrophic cardiomyopathy (HCM) and assess their relationship with phenotypic severity and predicted sudden cardiac death (SCD) risk.

Methods Fifty-one participants (37 non-obstructive HCM and 14 age, sex & BMI-matched controls) underwent 4D-flow CMR. Left ventricular (LV) end-diastolic volume was separated into four components: direct flow (blood transiting the ventricle within one cycle), retained inflow (blood entering the ventricle and retained for one cycle), delayed ejection flow (retained ventricular blood ejected during systole), and residual volume (ventricular blood retained for over two cycles).

Results HCM patients demonstrated greater direct flow compared to controls (47.5±9% vs 39.4±6%, p=0.003), and reduction in other components. In contrast to controls, HCM exhibited a paradoxical reduction in stroke volume (r=-0.31) with increasing direct flow suggesting diminished cardiac reserve. This direct flow component proportion correlated with LV mass index (r=0.38), end-diastolic volume index (r=-0.42), and SCD risk (r=0.38). Neither LV ejection fraction, nor stroke volume correlated with markers of phenotypic severity.

Conclusion HCM possesses a distinctive pattern of flow component distribution typified by direct flow-stroke volume decoupling, and in keeping with a diminished cardiac reserve. The correlation of direct flow proportion with phenotypic severity and SCD risk highlights its potential as a novel and sensitive haemodynamic measure of cardiovascular risk in patients.

P4 HIV ASSOCIATED CARDIOVASCULAR DISEASE BASED ON ADVANCED CARDIAC IMAGING: A SYSTEMATIC REVIEW AND META-ANALYSIS

[1,2]Jonathan Hudson, [3,4]Edith D Majonga, [3,5]Rashida A Ferrand, [6]Pablo Perel, [6,7]Shirjel R Alam, [6,8]Anoop SV Shah. *[1]Kings College London BHF Centre, School of Cardiovascular and Metabolic Medicine and Sciences, London, UK; [2]Department of Cardiology, Epsom and St. Helier University Hospitals Trust, UK; [3]Biomedical Research and Training Institute, Harare, Zimbabwe; [4]Department of Medical Physics and Imaging Sciences, Faculty of Medicine and Health Sciences, University of Zimbabwe, Zimbabwe; [5]Department of Clinical Research, London School of Hygiene and Tropical Medicine, London, UK; [6]Department of Non-communicable Disease, London School of Hygiene and Tropical Medicine, London, UK; [7]Department of Cardiology, North Bristol NHS trust, Bristol, UK; [8]Department of Cardiology, Imperial College NHS Trust, London, UK*

10.1136/heartjnl-2022-BSCI.9

Objective To systematically review and meta-analyse data from advanced cardiovascular imaging studies evaluating computed tomography coronary angiography (CTCA), positron emission tomography (PET), and cardiac magnetic resonance (CMR), in people living with HIV (PLHIV) compared to uninfected individuals.

Methods Three databases were searched for studies investigating the association between cardiovascular pathology and HIV using CTCA, CMR and PET in PLHIV from inception to February 11th 2022. Primary outcomes moderate to severe (>50%) coronary stenosis (CTCA), vascular and myocardial target-to-background ratio (PET), late gadolinium enhancement prevalence (CMR). Prevalence and risk ratios (RR) (comparing PLHIV to uninfected individuals) were pooled for using a random effects model.

Results Forty-five studies including 5218 PLHIV (mean age 48.5 years) and 2414 uninfected individuals (mean age 49.1 years) met the inclusion criteria. Sixteen studies (n=5107 participants) evaluated CTCA, 10 (n=681) vascular PET, 3 (n=146) both CTCA and vascular PET, and 16 (n=1698) CMR. No studies originated from low-income countries. The prevalence of moderate/severe coronary disease in 17.3% in PLHIV and 13.8% in controls (RR 1.33, 95%CI 0.96–1.82, I2= 62%). The prevalence of myocardial fibrosis was 47.5% in PLHIV and 31.7% in controls (RR 2.34, 95% confidence interval [CI] 1.34–4.08, I2=88%). PET studies indicated that PLHIV have an increase in vascular inflammation however these findings are derived from populations with well controlled HIV in middle age.

Conclusion Significant associations were observed between HIV and risk of myocardial fibrosis but not moderate to severe coronary disease. These findings were derived largely from populations in regions of low HIV endemicity.

P5 EPICARDIAL ADIPOSE TISSUE VOLUME AND CHARACTERISTICS ARE ASSOCIATED WITH STAGE B HEART FAILURE IN TYPE 2 DIABETES

[1]Sarah L Ayton, [1]Jian L Yeo, [1]Gaurav S Gulsin, [1]Abhishek Dattani, [1]Emer M Brady, [1]J Ranjit Arnold, [1]Matthew PM Graham-Brown, [1]Anvesha Singh, [2]Damini Dey, [1]Gerry P McCann, [1]Alastair J Moss. *[1]Department of Cardiovascular Sciences, University of Leicester and the NIHR Leicester Biomedical Research Centre, Glenfield Hospital, Leicester, UK; [2]Biomedical Imaging Research Institute, Cedars-Sinai Medical Center, Los Angeles, CA, USA*

10.1136/heartjnl-2022-BSCI.10

Objective To assess whether epicardial adipose tissue (EAT) is associated with early cardiac dysfunction in type 2 diabetes (T2D).

Methods Prospective case-control study. Participants with T2D and no known cardiovascular disease underwent comprehensive multimodality phenotyping. CT derived EAT volume was measured using a deep learning method and indexed to body surface area. Total EAT was quantified using CT adipose tissue attenuation range of -30 to -190 Hounsfield Units (HU) and low attenuation EAT as -90 to -190 HU. Left ventricular (LV) volumes, strain and diastolic function were assessed using cardiac MRI and echocardiography.

Results Two hundred and fifteen T2Ds (median age 63 years, 60% male) and 39 controls (median age 59 years, 62% male) were included. T2Ds had higher LV mass/volume ratio (median 0.89 (0.82, 0.99) vs 0.79 (0.75, 0.89)), reduced global longitudinal strain (GLS; mean 16.13±2.33% vs 17.18 ±2.16%) and worse diastolic function (lower circumferential peak early diastolic strain rate and average E/e'). Total and low attenuation indexed EAT volumes were 1.6-fold and 2-fold higher, respectively, in subjects with T2D. After adjustment for age, sex, ethnicity, systolic blood pressure and waist/hip ratio, low attenuation indexed EAT volume was independently associated with LV mass/volume ratio (ß= 0.002, p= 0.03) and GLS (ß= -0.03, p= 0.03) and total indexed EAT volume with GLS (ß= -0.02, p 0.02), but neither were independently associated with indices of diastolic dysfunction in T2D.

Conclusion EAT volumes are higher in T2D, and excess EAT accumulation is independently associated with early markers of cardiac dysfunction.