Abstract 156 Figure 2 Home page and pages for data entry of patient demographics and measured parameters allowing for automated calculation of Z-score online and on the EchoCalc app for use on android/iPhone. The data can be stored as a ".pdr document for emailing or storage on most browsers

Methods 417 (297 Arab, 120 black) male paediatric (11-18 years) athletes, were evaluated by 2D echocardiography as per the as per British Society of Echocardiography guidelines, and biological age (by radiological X-ray) assessment. Z scores involved allometric ($a*[BSA]^{((b+c*\text{chronological age}))}$) modelling for measures of LV structure.

Results Residual linear regression, revealed normalised measures of LV to be independent of BSA, race, chronological and biological age. Z-scores for an athlete with a specific BSA and/or chronological age can be calculated 1) visually in the nomograms provided (figure 1) 2) automatically online/on the EchoCalc app (Figure 2), or 3) manually from by using the specified a, b, and c mean predicted parameters and d, e, and f RSD for that parameter of LV structure from table 1: $z = (\text{obs} - (a*[BSA]^{((b+c*\text{chronological age}))})) / (d + (e*BSA) + (f*\text{chronological age}))$

Conclusion For the first time, we present BSA, race, chronological, and biological age independent nomograms and Z scores for LV, LA and Aortic size to BSA in male Arab and black paediatric athletes. Nomograms are provided to assist in the tracking of the paediatric athlete necessitating annual follow-up and online/app calculation to facilitate use in day-to-day practice (figures 1 and 2). In clinical context, we propose a new tool for differential diagnosis of physiological remodeling to an indicator of cardiac pathology in the cardiac screening of the paediatric athlete.

Conflict of Interest None

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18F-SODIUM FLUORIDE POSITRON EMISSION TOMOGRAPHY, AORTIC DISEASE ACTIVITY AND ISCHAEMIC STROKE RISK

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Background Arterial 18F-sodium fluoride (18F-NaF) activity on positron emission tomography (PET) is a marker of active microcalcification and atherosclerosis. Coronary 18F-NaF activity (CMA) predicts coronary artery disease progression and subsequent myocardial infarction. We aimed to investigate whether aortic 18F-NaF activity (AMA) predicts thoracic aortic atherosclerotic disease progression and subsequent ischaemic stroke in patients with established cardiovascular disease.

Methods In a post-hoc observational cohort study, we evaluated AMA in patients with stable coronary artery disease (n=239) or aortic stenosis (n=158) who had undergone thoracic 18F-NaF PET and computed tomography (CT). We assessed the associations between AMA and progression of thoracic aortic calcification on follow up CT and subsequent ischaemic stroke or myocardial infarction.

Results In 141 patients with repeat CT imaging at 12.7 ± 2.7 months, AMA correlated with progression of thoracic aortic calcium scores ($r=0.21$, $p=0.011$). In 397 patients, 16 had an ischaemic stroke and 25 had a myocardial infarction after 4.7 ± 1.6 years. After adjusting for clinical risk factors, CMA and calcium scoring, AMA was associated with stroke (hazard ratio, 1.71 [95% confidence interval 1.00-2.90], $p=0.048$). AMA was superior to clinical risk and calcium scores in identifying patients with stroke (c-statistic 0.76 versus 0.58 versus 0.63 respectively, $p<0.05$). Survival analysis demonstrated that AMA was associated with ischaemic stroke ($p<0.001$) but not myocardial infarction ($p=0.45$), whereas CMA was associated with myocardial infarction ($p<0.001$) but not stroke ($p=0.39$).

Conclusions In patients with established cardiovascular disease, AMA is associated with progression of aortic atherosclerosis and future ischaemic stroke. Arterial 18F-NaF identifies localised areas of atherosclerotic disease activity that relate to regional atherothrombotic events.

Conflict of Interest none to declare

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NON-INVASIVE NEAR-INFRARED FLOURESCENCE IMAGING OF MALODIALDEHYDE-OXIDISED LOW-DENSITY LIPOPROTEIN IN ATHEROSCLEROSIS

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Introduction Atherosclerosis is an insidious disease where the accumulation of low-density lipoprotein in the arterial walls generates plaques, which may rupture leading to an untimely death or loss of quality of life. A key barrier for the treatment of atherosclerosis is identifying and predicting those plaques which are most vulnerable to rupture. This project aimed to bridge this diagnostic gap by using monoclonal antibodies for targeted imaging of malondialdehyde-low density lipoprotein (MDA-LDL), a component of vulnerable plaques.

Method A humanised monoclonal antibody (LO1-huFab) was expressed in human embryonic kidney cells (HEK)-293 cells with an unpaired sulphhydryl group for site-specific maleimide conjugation. The purified protein was site-specifically conjugated to a near-infrared fluorophore (LO1-huFab-750) and one nanomole injected in LDL receptor-deficient (LDLR^{-/-}) and wild-type (C57/BL6) mouse model. Mice were additionally co-injected with fluorophores targeting matrix metalloproteinases (MMPsense) and integrin $\alpha 5 \beta 3$ (IntegriSense). Non-invasive fluorescence molecular tomography imaging was

performed four hours post-injection with the amount of the three different tracers in the aortic arch quantified.

Results LO1-huFab demonstrated *in vivo* targeting in LDLR^{-/-} mouse model with the ability to distinguish animals with and without disease (63.16 ± 12.64 pmol vs 23.92 ± 4.35 pmol, $p=0.045$, $n=4$ per group). Multiplexing with MMPsense and IntegriSense revealed LO1-huFab-750 to be the first probe to distinguish between disease states. Ex vivo fluorescence reflectance imaging validated the focal signal of LO1-huFab-750 observed in the aortic root and abdominal aorta. Rapid clearance of LO1-huFab-750 through the glomerular filtration

Abstract 158 Figure 1 Representative images of FMT-CT cotocalisation of huFab, IntegriSense and MMPsense. A) huFab (blue), IntegriSense (green) and MMPsense (red) signals overlaid in a diseased LDLR^{-/-} mouse fed a high fat diet for 30 weeks. Red circles indicate the region of interest utilised for quantification of signal. Red arrows indicate the aorta. B) huFab, IntegriSense and MMPsense signals in a LDLR^{-/-} mouse fed a high fat diet for 30 weeks. C) huFab, IntegriSense and MMPsense signals in a C57/BL6 (WT) mouse on a chow diet. $N=4$ for all groups. Panels arranged in coronal, axial and sagittal order (left, centre, right, respectively)

Abstract 158 Figure 2 Quantification of FMT signal of LO1-huFab, IntegriSense and MMPsense in LDLR^{-/-} mouse fed a high fat diet for 30 weeks and age-matched C57/BL6 (WT) mice. $N=4$ for all groups. Values are mean $\pm$ SEM. Statistical significance calculated using unpaired T-test with Welch's correction

system was evident through ex vivo quantification of fluorescence in the kidneys.

Conclusion Site-specific near-infrared labeling of antibodies allowed for non-invasive imaging of atherosclerosis demonstrating its potential in identifying critical components of vulnerable plaques. These promising results have prompted the conjugation of LO1 antibody fragments with nanoparticles for targeted therapeutics of atherosclerosis.

Conflict of Interest N/A

159 MYOCARDIAL FIBROSIS IS ASSOCIATED WITH REDUCED CORONARY FLOW VELOCITY RESERVE IN END-STAGE RENAL DISEASE

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Background Uraemic cardiomyopathy is present in >70% of patients with end-stage renal disease (ESRD). Myocardial fibrosis is a hallmark of uraemic cardiomyopathy and is associated with an increased risk of heart failure and sudden cardiac death. Coronary microvascular dysfunction (CMD) is common in ESRD and is an adverse prognostic marker that may contribute to this increased mortality. In hypertrophic cardiomyopathy (HCM), a condition that shares phenotypical similarities with uraemic cardiomyopathy including hypertrophy, cellular disarray and fibrosis, a negative correlation exists between myocardial fibrosis and CMD, raising the possibility that fibrosis is the result of microvascular ischaemia. This study aimed to determine whether a similar relationship exists in ESRD.

Methods 15 patients with ESRD underwent transthoracic echocardiography, coronary flow velocity reserve (CFVR) assessment by Doppler echocardiography and 3Tesla cardiac magnetic resonance imaging. Subjects with known coronary artery disease, moderate/severe valvular heart disease, diabetes