Abstract 155 Table 2 Univariable and multivariable analysis for the prediction of myocardial infarction

Univariable		Multivariable [#]		
HR (95% CI)	P value	HR (95% CI)	P value	
Total plaque burden*	1.44 (1.15-1.18)	<0.0001	1.33 (0.97-1.82)	0.0720
NCP burden*	1.41 (1.14-1.75)	<0.0001	1.30 (0.96-1.75)	0.0883
LAP burden*	1.87 (1.36-2.57)	<0.0001	1.80 (1.16-2.80)	0.009
CP burden*	1.70 (1.26-2.12)	<0.0001	1.55 (0.92-2.63)	0.1021
PCAT-RCA [§]	1.55 (1.08-2.22)	0.0171	1.47 (1.02 – 2.12)	0.0382
Cardiovascular risk score	1.03 (1-1.05)	0.0463	-	-
Agatston calcium score*	1.2 (1.1-1.3)	<0.0001	-	-
Obstructive disease	3.02 (1.6-5.8)	<0.0001	-	-

[#] Multivariable analysis includes the individual quantitative plaque measure, Agatston calcium score, obstructive disease and cardiovascular risk score. Full model results are presented in Table 1 in the Data Supplement.

* Per doubling.

[§] Per 1 standard deviation increment in PCAT attenuation.

CI, confidence interval; CP, calcified plaque; LAP, low attenuation plaque; HR, hazard ratio; PCAT-RCA, pericoronary adipose tissue attenuation around the right coronary artery.

a myocardial infarction (HR 2.45, 95% CI 1.23 to 4.80; $p=0.001$). Patients with low-attenuation plaque burden (greater than 4%) were nearly 5 times more likely to suffer a myocardial infarction (HR 4.87, 95% CI 2.03 to 11.78, $p<0.0001$). When the two metrics were combined, patients with both low-attenuation plaque burden $>4\%$ and PCAT-RCA ≥ -70.5 HU were at the greatest risk of myocardial infarction (HR 11.7, 95% CI 3.3 to 40.9, $p<0.0001$), followed by those with low-attenuation plaque burden $>4\%$ and

PCAT-RCA < -70.5 (HR 5.1, 95% CI 1.5 to 17.7, $p<0.0001$; figure 1). In ROC analysis, low attenuation plaque burden was a stronger predictor of future fatal or non-fatal myocardial infarction than PCAT-RCA (area-under-the-curve, 0.71 (95% CI 0.62-0.81) to 0.75 (95% CI 0.65-0.8) ($\Delta AUC=0.04$; $p=0.01$).

Conclusion CT coronary angiography defined PCAT attenuation and low-attenuation plaque have marked and additive predictive value for the risk of fatal or non-fatal myocardial infarction.

Conflict of Interest Nil

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A NEW TOOL TO AID THE DIFFERENTIAL DIAGNOSIS OF PHYSIOLOGICAL REMODELLING FROM CARDIAC PATHOLOGY WHEN ASSESSING LEFT VENTRICLE AND LEFT ATRIAL STRUCTURE IN MALE ARAB AND BLACK PAEDIATRIC ATHLETES

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Background Physiological remodelling of the paediatric athletes' heart can overlap with phenotypic expression of cardiomyopathies, which increase the risk of sudden cardiac death. This overlap may be compounded by BSA, race, chronological and biological age. We established nomograms and Z-scores for clinical evaluation of left ventricular (LV) and atrial (LA) structure in Arab and black paediatric athletes, accounting for the impact of BSA, race, chronological and biological age.

Abstract 156 Table 1 Models, Predicted Mean and Regressed Standard Deviation Parameters for Measurements of Left Ventricle, Atrial and Aortic Root Size in the Male Arab and Black Paediatric Athlete

Parameter	Predicted Mean Parameters			Regressed SD Parameters		
	a	b	a	b	a	b
LVIDd, mm	40.469	0.228	0.008	2.276	0.250	0.047
IVSd, mm	6.055	-0.020	0.031	1.100	0.062	-0.008
PWTd, mm	5.891	0.214	0.021	0.562	0.194	0.005
LV Vol D, mm	66.236	0.20	0.046	-10.763	13.239	0.465
LVM, g	68.499	0.396	0.049	1.230	8.960	0.429
LAD, mm	24.094	0.533	-0.005	3.950	1.035	-0.143
LA Vol, ml	22.538	0.948	0.006	-4.254	4.719	0.326

Abstract 156 Figure 1 Scatter plots of: A, Left Ventricle Internal Diameter (LVIDd); B, LV Mass to BSA in 297 Arab (white dots) and 120 black athletes (black dots), with predicted Z boundaries. Solid blue line, Z=0; dashed blue line, Z=1 and -1; solid red line, Z=2 and -2

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