Abstract 154 Table 2 Changes in CMR findings with empagliflozintreatment

Variable	Baseline (n=18)	Follow up (n=18)	P value
LV end diastolic volume (ml)	164 ± 50	149 ± 38	0.02
LV end diastolic volume index (ml/m^2)	86 ± 27	79 ± 21	0.02
LV end systolic volume (ml)	83 ± 45	65 ± 39	0.001
LV end systolic volume index (ml/m^2)	44 ± 25	35 ± 21	0.001
LV stroke volume (ml)	81 ± 20	84 ± 16	0.39
LV ejection fraction (%)	52 ± 13	59 ± 15	0.001
LV mass (g)	119 ± 33	109 ± 29	0.06
LV mass index (g/m^2)	61 ± 15	56 ± 12	0.07
LV mass to LV end diastolic volume (g/ml)	0.76 ± 0.27	0.75 ± 0.18	0.6
RV end diastolic volume (ml)	151 ± 34	155 ± 32	0.5
RV end diastolic volume index (ml/m^2)	79 ± 19	83 ± 24	0.3
RV end systolic volume (ml)	71 ± 25	76 ± 27	0.4
RV end systolic volume index (ml/m^2)	38 ± 15	41 ± 22	0.4
RV stroke volume (ml)	78 ± 11	80 ± 12	0.7
RV ejection fraction (%)	53 ± 9	53 ± 11	0.9
Peak circumferential strain (%)	-17.4 ± 4.1	-18.8 ± 4.9	0.3
Global longitudinal strain (%)	-10.2 ± 2.9	-13.1 ± 4.1	0.01
Peak diastolic circumferential strain rate (1/s)	1 ± 0.2	1.1 ± 0.3	0.4
Peak diastolic longitudinal strain rate (1/s)	0.7 ± 0.2	0.7 ± 0.2	0.9
LA maximum volume (ml)	63 ± 2	57 ± 17	0.2
LA ejection fraction (%)	48 ± 13	48 ± 15	0.2
Mean T1 (ms)	1285 ± 104	1310 ± 42	0.6
Extra-cellular volume (%)	24 ± 3	25 ± 3	0.1
Cell volume (ml/m^2)	92 ± 30	84 ± 27	0.04
MBF rest (ml/g/min)	0.62 ± 0.3	0.66 ± 0.3	0.4
MBF stress (ml/g/min)	1.6 ± 0.5	1.4 ± 0.3	0.08
MPRI	2.9 ± 1.5	2.3 ± 0.8	0.06

Values are means (SD) ormedian (IQR) for continuous variables and number (%) for categorical variables. LV indicates left ventricular; ml, milliliter; ml/m^2, milliliters per square meter of body surface area; g, gram; g/m^2, gram per square meter of body surface area; LVEF, left ventricular ejection fraction; RV, right ventricular; LA, left atrium; MBF, myocardial blood flow; ms, milliseconds; MPRI, myocardial perfusion index.

pro hormone B-type natriuretic peptide (NT-proBNP) levels were measured. **(Table 1)** Ten controls with no diabetes underwent an identical 31P-MRS and CMR protocol on a single visit.

Results When compared to controls, patients with T2D showed: lower myocardial energetics (1.52±0.40 vs 2.20±0.5, p=0.0005), lower stress myocardial blood flow (1.60±0.50 vs 2.10±0.50, p=0.02) and lower left ventricular ejection fraction (52±13% vs 63±4%, p=0.01). Treatment with empagliflozin led to significant improvements in myocardial energetics (PCr/ATP: 1.52 to 1.76, p=0.009). This was accompanied by a relative 13% improvement in left ventricular ejection fraction (p=0.001), 3% improvement in global longitudinal strain (p=0.01), 61% reduction in NTproBNP (p=0.05), and 9% reduction in myocardial cell volume (p=0.04). No significant change in myocardial blood flow or diastolic strain was detected **(table 1)**.

Conclusions Empagliflizon improves myocardial energetics and function, reduces myocardial cellular volume, and reduces NT-proBNP levels in patients with T2D **(figure 1)**. This study, by using the myocardial phosphocreatine to ATP ratio to monitor the early energetic response of the heart to treatment, and CMR to monitor structural and functional changes, provides significant novel insights into potential mechanisms by which empagliflozin exerts its beneficial cardiovascular effects.

Conflict of Interest NONE

155 PERICORONARY ADIPOSE TISSUE ATTENUATION, LOW ATTENUATION PLAQUE BURDEN AND 5-YEAR RISK OF MYOCARDIAL INFARCTION

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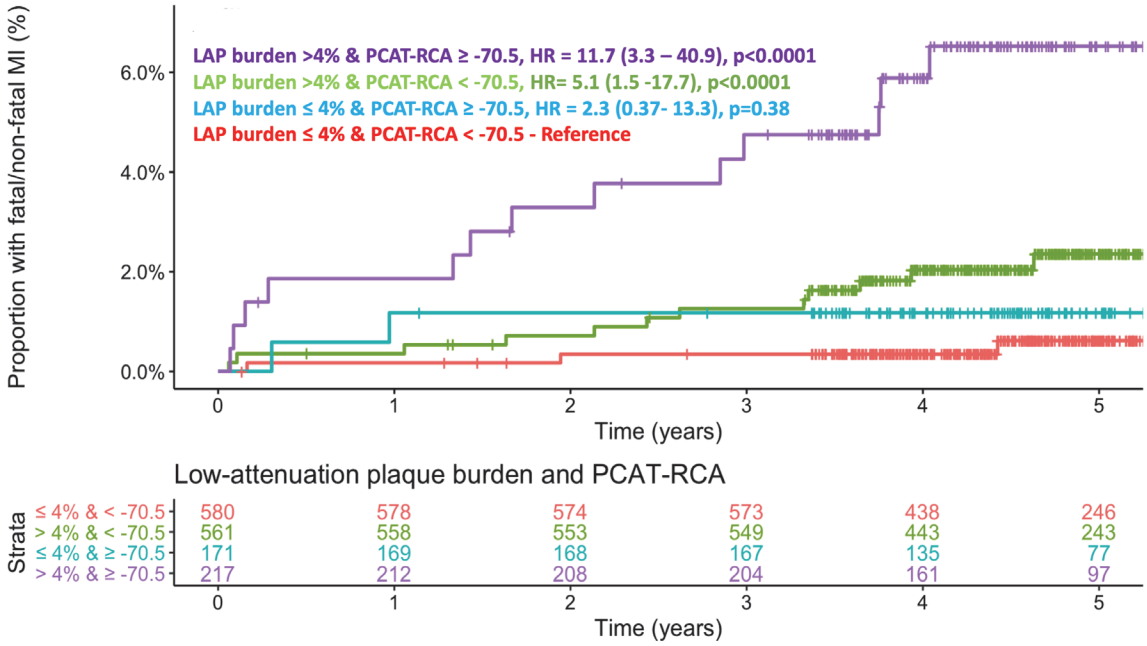
Introduction Pericoronary adipose tissue (PCAT) attenuation has emerged as a surrogate marker of pericoronary inflammation. To date, no studies have compared the impact of pericoronary adipose tissue(PCAT) attenuation and quantitative plaque burden on cardiac outcomes. We aimed to establish the relative merits of these approaches to risk prediction and hypothesised that the combination of PCAT attenuation and quantitative plaque burden measures could provide additive and improved prediction of myocardial infarction in patients with stable chest pain.

Methods In a post-hoc analysis of a randomized controlled trial, we investigated the association between the future risk of fatal or non-fatal myocardial infarction and PCAT attenuation measured from CT coronary angiography using multivariable Cox regression models including plaque burden, obstructive coronary disease and cardiac risk score

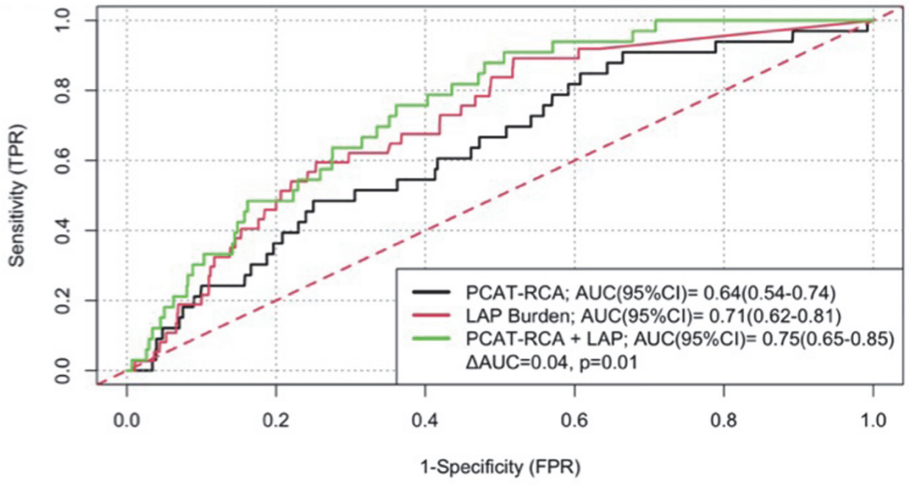
Abstract 155 Table 1 PCAT attenuation and quantitative plaque burden inpatients with and without subsequent myocardial infarction

	No event	Event	p value
Plaque burden (%)	38.9 [0 to 49.2]	50.1 [43.0 to 53.8]	<0.001
Noncalcified plaque burden (%)	35.4 [0 to 45.3]	42 [37.39 to 48.95]	<0.001
Low-attenuation plaque burden (%)	4.1 [0 to 6.7]	7.4 [4.8 to 9.1]	<0.001
Calcified plaque burden (%)	0.39 [0 to 2.69]	3.18 [0.9 to 7.97]	<0.001
PCAT-RCA (HU)	-76.0 ± 7.8	-72.5 ± 8.3	0.009
PCAT-LAD (HU)	-77.0 ± 7.8	-76.2 ± 8.6	0.5432
PCAT-LCx (HU)	-73.3 ± 7.7	-71.6 ± 7.3	0.3345
Agatston CACS (Agatston Units)	19 [0 to 218]	283 [59 to 1041]	<0.001
Obstructive disease	416 (25%)	19 (51%)	<0.001
Cardiovascular risk score (%)	18 ± 11	22 ± 12	0.039

Median [interquartile interval], mean ± standarddeviation or number (%).
CACS, coronaryartery calcium score; HU, Hounsfield units; PCAT, Pericoronary adipose tissueattenuation.



Abstract 155 Figure 1



Abstract 155 Figure 2

(incorporating age, sex, diabetes, smoking, hypertension, hyperlipidaemia and family history of cardiovascular disease).

Results In 1697 evaluable participants (mean age 58±10 years), there were 37 myocardial infarctions after a median follow-up of 4.7 [interquartile interval, 4.0-5.7] years. Median low-attenuation plaque burden was 4.20[0-6.86] % and mean PCAT -76±8 Hounsfield units (HU). PCAT-RCA attenuation was higher in patients who suffered a myocardial infarction (-72.5±8.3 HU versus -76.5± 7.8 HU, p=0.0063), but there was no difference in PCAT-LAD (-76.3±8.6 HU vs -77.0±7.8 HU, p=0.54) or PCAT-LCx(-71.6±7.3 HU vs -73.3±7.7 HU, p=0.33). Patients sustaining a myocardial infarction also had higher total, non-calcified, low-attenuation and calcified plaque burden, higher Agatston calcium score, higher cardiovascular risk score and increased presence of obstructive disease on CCTA (table 1). On univariable analysis, PCAT-RCA attenuation was a predictor of myocardial infarction (HR 1.55, 95% CI 1.08 to 2.22, p=0.017), but PCAT-LAD or PCAT-LCx were not. Univariable analysis also identified the burden of non-calcified, low-attenuation and calcified plaque as well as Agatston coronary calcium score, presence of obstructive coronary artery disease and cardiovascular risk score were predictors of myocardial infarction (table 2). Male sex, age and body-mass index were not predictors of future myocardial infarction (p=0.06, p=0.86 and 0.26 respectively). In multivariable analysis, only the low-attenuation plaque burden (HR 1.80, 95% CI 1.16 to 2.81, p=0.011, per doubling) and PCAT-RCA (HR 1.47 95%1.02 to 2.13, p=0.040, per standard deviation increment) remained predictors of myocardial infarction (table 2). Based on the Youden's index of the ROC curves, the optimal cut-off of the right coronary artery PCAT attenuation was –70.5 HU for the primary endpoint of fatal or non-fatal myocardial infarction. Patients with PCAT-RCA above ≥–70.5 HU were nearly 2.5 times more likely to suffer

Abstract 155 Table 2 Univariable and multivariableanalysis 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	-	-

Multivariableanalysis includes the individual quantitative plaque measure, Agatston calciumscore, obstructive disease and cardiovascular risk score. Full model resultsare presented in Table i in the Data Supplement.
* Per doubling.
§ Per 1 standard deviation increment in PCAT attenuation.
CI, confidence interval; CP, calcifiedplaque; LAP, low attenuation plaque; HR, hazard ratio; PCAT-RCA, pericoronaryadipose 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) (ΔAUC=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

156 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 PAEDAITRIC ATHLETES

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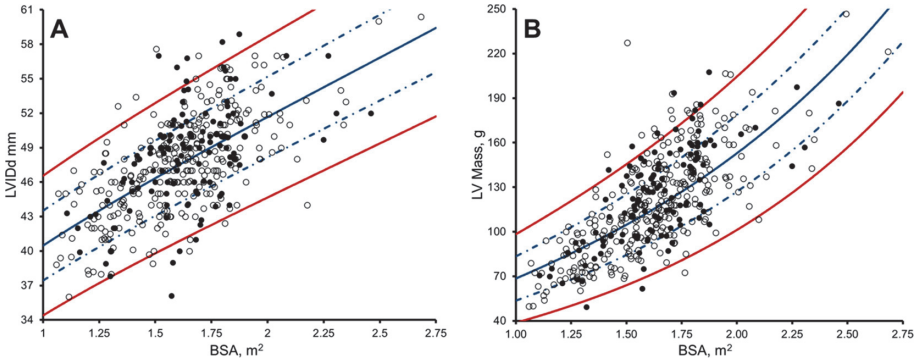
Background Physiological remodelling of the paediatric athletes' heart can overlap with phenotypic expression of cardiomyopathies, which increase the risk of sudden cardiac death. This overlap may be compounded by BSA, race, chronological and biological age. We established nomograms and Z-scores for 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

	Predicted Mean Parameters			Regressed SD Parameters		
Parameter	*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