

Abstract 214 Figure 2 Overall survival Free of Secondary Outcomes of Stroke, TIA and MI (Expressed in percentage). Patient outcomes compared from patients assessed within the Stroke-Cardiology-MDT (October 22 to December 23) compared to prior usual care without Stroke-Cardiology-MDT (December 2019-December 2021)

existing healthcare resources. Our study shows preliminary evidence supporting the SC-MDT implementation to improve clinical outcomes including reduction of recurrent stroke, TIA or MI.

Conflict of Interest Nil to declare

215 LONGITUDINAL STUDY OF PLAQUE MORPHOLOGY IN MASTERS ATHLETES

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Introduction Cross-sectional studies of Master's athletes with a low atherosclerotic risk profile have identified a greater prevalence of high coronary artery calcium (CAC) scores, coronary plaque and luminal stenoses than matched controls. Athletes exhibited predominantly calcified plaque which is purportedly more stable and less prone to rupture.

We sought to assess the progression of coronary disease amongst a cohort of male athletes and controls.

Methods 72 ostensibly fit male Master's-athletes and 24 matched controls of similar age without cardiovascular risk factors at baseline underwent evaluation with health questionnaire, cardiopulmonary exercise testing (CPET) and CT

coronary angiography (CTCA) with CAC score at baseline and 8-year follow-up. Athletes were required to continue running or cycling at least 10km or 30km/week respectively.

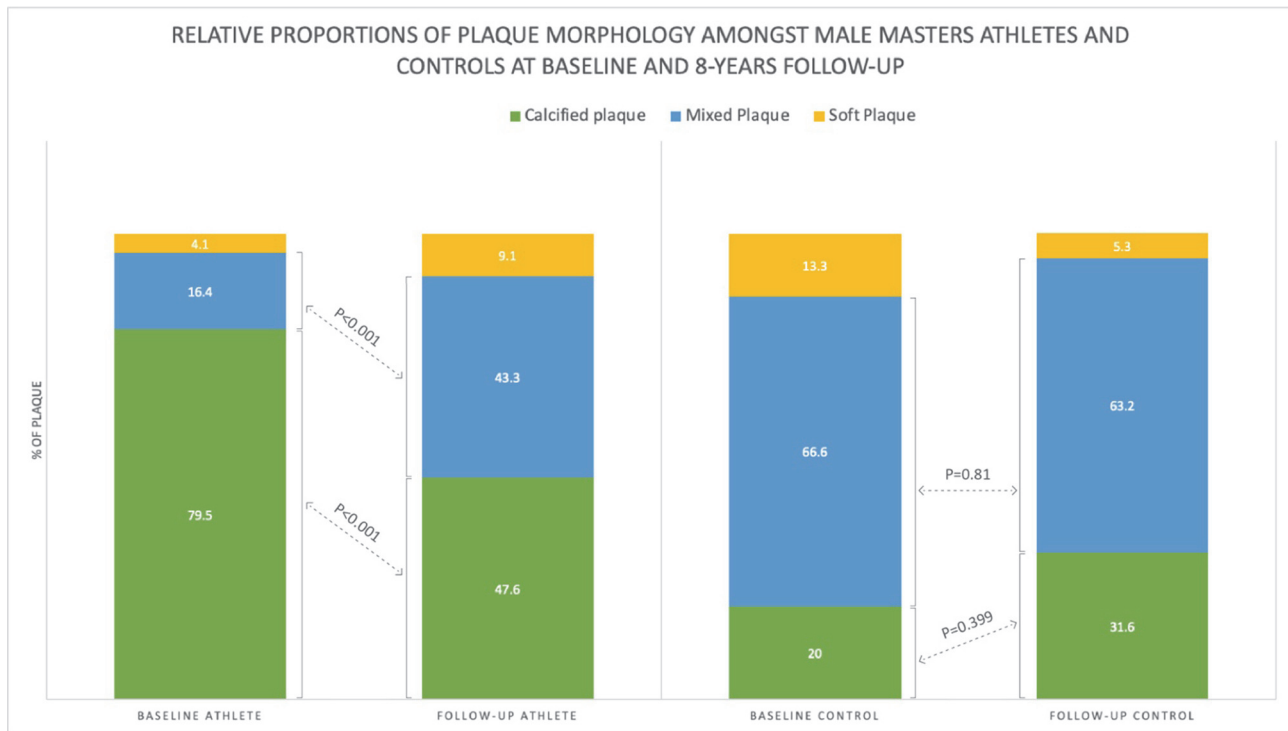
CTCA was interpreted in accordance with established guidelines assessing luminal stenosis severity and plaque morphology (calcified, mixed or non-calcified). CAC was calculated using the Agatston method and adjusted for age and sex. A 3-vessel plaque score was calculated based on the co-existing presence of any plaque in the LAD, LCX or RCA irrespective of severity.

Results Athletes remained well matched with respect to cardiovascular risk factors at follow-up, weighed significantly less, demonstrated higher HDL cholesterol, and achieved significantly greater CPET metrics in regard to maximum workload, peak VO₂ and % predicted peak VO₂ ($p < 0.001$). Only athletes demonstrated a peak systolic blood pressure exceeding 220 mmHg.

There were no significant differences between athletes and controls with respect to CAC scores, proximal or 3-vessel plaque at baseline or follow-up. Albeit only athletes demonstrated CACS ≥ 300 and ≥ 400 AU and luminal stenoses $> 50\%$ at baseline.

At baseline amongst athletes, 79.5% of plaques were calcified versus 20% in controls ($p < 0.001$). The dominant plaque morphology in controls was mixed plaque (66.7%).

At follow-up, athletes demonstrated a significant change in plaque composition with calcific and mixed plaque now accounting for 47.6% and 43.3% of plaques respectively ($p < 0.001$). There were no significant differences in plaque



Abstract 215 Figure 1

Abstract 215 Table 1

	Athlete (n=72)	Control (n=24)	P-value
Age at f/u	63 ± 8	61 ± 8	0.327
Height (cm)	178	179	0.426
Weight (Kg)	74.6 ± 9.3	83.7 ± 12.7	<0.001
BSA	1.92 ± 0.13	2.05 ± 0.17	<0.001
Caucasian%	95	79	0.026
Weekly Exercise (Mins)	436	156	<0.001
Hba1c	36.06	35.65	0.578
Total Cholesterol	1.0	5.0 ± 1.2	0.59
HDL	1.7 ± 0.4	1.4 ± 0.6	0.011
Creatinine	81 ± 15	84 ± 13	0.449
Antiplatelet	4 (5)	1 (4)	0.791
Anticoagulation	8	3	0.853
Statin	10 (14)	3 (12.5)	0.87
Antihypertensive	10 (14)	3 (12.5)	0.87
Max METS	11.7 ± 1.9	8.6 ± 1.4	<0.001
Max Watts	266 ± 52	207 ± 48	<0.001
Max BP (S)	191 ± 21	184 ± 19.9	0.156
CPET BP >220 mmHg	11 (15)	0	0.046
VO2 Max	41.0 ± 6.7	30.0 ± 4.9	<0.001
VO2% Pred	145 ± 19	112 ± 14	<0.001

composition amongst controls, who continued to demonstrate significantly more mixed-morphology plaque than athletes (63.2% vs 43.3%, $p=0.027$).

Discussion Plaque composition amongst athletes' changes with increasing age from a predominantly calcified plaque morphology at baseline to a similar proportion of both calcified and mixed plaque at follow-up. By contrast, the relative

Abstract 215 Table 2

	Follow-Up CTCA			Baseline CTCA		
	Athlete	Control	P-value	Athlete	Control	P-value
CAC Zero	26 (37)	12 (50)	0.228	38 (53.2)	15 (63)	0.407
CAC ≥10	43 (59.7)	10 (42)	0.123	29 (40.2)	5 (20.8)	0.085
CAC ≥100	23 (31.9)	4 (17)	0.149	11 (15.3)	1 (4.2)	0.154
CAC ≥300	16 (22.2)	3 (13)	0.301	5 (6.9)	0	0.185
CAC ≥400	12 (16.7)	1 (4)	0.121	2 (2.8)	0	0.409
CAC ≥50th	22 (30.6)	6 (25)	0.604	18 (25)	4 (16.7)	0.4
CAC ≥70th	14 (19.4)	3 (13)	0.44	11 (15.2)	3 (12.5)	0.738
3V-Plaque	14 (19)	2 (8)	0.206	5 (7)	1	0.626
≥1 Plaque	47 (65)	11 (46)	0.916	35 (50)	8 (35)	0.204
Proximal Plaque	44 (94)	10 (91)	0.137	25 (35.7)	5	0.214
Stenosis ≥50%	10 (14)	3 (13)	0.566	4 (5.7)	0	0.241
Stenosis ≥70%	2 (3)	2 (8)	0.238	0	0	n/a
Plaque morphology						
Calcified plaque	78 (47.6)	12 (31.6)	0.074	58 (79.5)	3 (20)	<0.001
Mixed Plaque	71 (43.3)	24 (63.2)	0.027	12 (16.4)	10 (66.6)	<0.001
Soft Plaque	15 (9.1)	2 (5.3)	0.44	3 (4.1)	2 (13.3)	0.16

proportions of plaques amongst control subjects remained stable, largely attributable to mixed plaque. Exercise may induce plaque stabilising effects which is progressively offset by age. Further longitudinal studies are required to understand cardiovascular event rates relative to plaque composition in athletes.

Conflict of Interest None