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Myocardial Blood Flow Reserve is Impaired in Patients with Aortic Valve Calcification and Unobstructed Epicardial Coronary Arteries



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Introduction: Although coronary atherosclerosis is associated with calcific aortic valve disease (CAVD), it is not known whether they share pathophysiological mechanisms in early disease.

Aims: To investigate the relationship between myocardial blood flow reserve (MBFR) - a measure of coronary microcirculatory function, and early CAVD. We also determined whether this relationship was independent of coronary artery disease (CAD) and hs-CRP, a marker of systemic inflammation.

Methods & Results: 183 patients with chest pain and unobstructed coronary arteries were recruited. AVCS, coronary total plaque length (TPL), and coronary calcium score were quantified from multislice CT. MBFR was assessed using vasodilator myocardial contrast echocardiography. Inflammatory markers were obtained from venous sampling.

Mean age was 59.8 years, with 52.5% being male. The mean AVCS was 68AU (SD 258). Mean TPL was 15.6mm, and median coronary calcification score was 43.5AU. The mean MBFR was 2.20 (SD 0.52). Mean hs-CRP was 2.52mg/L (SD 3.86), with 59% participants having normal hs-CRP levels. Multivariate linear regression modelling incorporating demographics, coronary plaque characteristics, MBFR, and inflammatory markers, demonstrated that age (β =+0.05, CI:+0.02,+0.08, P=0.007), hs-CRP (β=+0.09, CI:+0.02,+0.16, P=0.010) and presence of diabetes (β =+1.03, CI:+0.08,+1.98, P=0.033), were positively associated with AVCS. In contrast, MBFR (β=-0.87, CI:-1.44,-0.30, P=0.003), body mass index (β=-0.11, CI:-0.21,-0.01, P=0.033), and LDL cholesterol (β=-0.32, CI:-0.61,-0.03, P=0.029) were negatively associated with AVCS. TPL and coronary calcium score became insignificant when included in multivariate analysis.

Conclusions: MBFR is an independent predictor of aortic valve calcification and this effect is independent of the presence of CAD and also systemic inflammation.

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New Insights Into the Dramatic Changes in Transpulmonary Haemodynamics Using Eplar with Exercise in Ironman Endurance Athletes at Extremely High Workload (>20mets) Compared to the Non-Athlete Population

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Introduction: Ironman athletes can develop right ventricular dysfunction predisposing them to sudden cardiac death. In this novel stress echocardiography study, we sought to characterise ironman athletes' transpulmonary haemodynamics at extreme workloads (>20METs) compared with a non-athlete population using the novel echocardiographic parameter, ePLAR ("echocardiographic Pulmonary to Left Atrial Ratio" = TRVmax/mitralE/e')

Method: 19 athletes (age 38.3 ± 10 years; 84% male) underwent treadmill stress echo including haemodynamic data (TRVmax, E/e' ratio and ePLAR) at rest and within 60 seconds of exercise cessation. Athletes were compared to 181 non-athletes (age 66 ± 10 years)

Results: The athletes achieved 20.7 ± 1.6 METs vs. 9.9 ± 3.2 METs in non-athletes. No statistical difference was seen in resting TRVmax (2.4 ± 0.17 m/s vs. 2.4 ± 0.3 m/s) and mitral E velocities (0.82 ± 0.14 vs. 0.74 ± 0.21) between athletes and non-athletes. However, resting e' velocities were significantly higher in athletes (0.11 ± 0.02 m/s vs. 0.08 ± 0.03 m/s, p«<0.0001), resulting in statistically different ePLAR values between the two groups (0.33 ± 0.06 m/s vs. 0.27 ± 0.09 m/s, p<0.01). At peak exercise, there was a substantial increase in TRVmax in athletes (3.81 ± 0.54 m/s vs. 3.19 ± 0.49 m/s, p«<0.0001) resulting in significant rise in ePLAR (0.47 ± 0.13 m/s vs. 0.34 ± 0.11 , p«<0.0001) consistent with a marked increase in transpulmonary gradient

Conclusion: RVSP and transpulmonary gradient (estimated by ePLAR) increase with exercise and are significantly exaggerated in ironman athletes compared to the non-athlete population suggesting a possible physiologic mechanism for RV systolic dysfunction in the long-term endurance athletes.

	Pre-exercise		Post-exercise	
	Non athlete	Athlete	Non athlete	Athlete
TR Vmax (m/s)	2.4±0.30	2.4±0.17 (ns)	3.19±0.49	3.81±0.54 (***)
Mitral E (m/s)	0.74±0.21	0.82±0.14 (ns)	1.1±0.59	1.33±0.18 (ns)
e' (m/s)	0.08±0.03	0.11±0.02 (***)	0.12±0.59	0.16±0.03(ns)
E/e' ratio	9.94±4.13	7.52±1.27 (*)	10.147±3.9	8.53±1.97 (ns)
ePLAR (m/s)	0.27±0.09	0.33±0.06 (*)	0.34±0.11	0.47±0.13(***)
*** significant at p<0.0001, ** significant at p<0.001, *significant at p<0.01, ns = not significant				

http://dx.doi.org/10.1016/j.hlc.2016.06.566

