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Echocardiography and strain analysis in Malaysian elite athletes versus young healthy adults

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ABSTRACT

Background: Athletes have changes that can mimic pathological cardiomyopathy.
Methods: Echocardiographic study of 50 male, female athletes (MA, FA) and non-athletes (MNA, FNA) age 18 to 30 years. These athletes participate in sports with predominantly endurance component. All participants exhibit no known medical illnesses or symptoms.
Results: MA have thicker wall (IVSd) than MNA. No MA have IVSd > 1.2 cm and no FA have IVSd > 1.0 cm. Left ventricle internal dimension (LVIDd), left ventricle end diastolic volume index (LVEDVi) is bigger in athletes. None have LVIDd > 5.8 cm. Right ventricle fractional area change (FAC) is lower in athletes. (MA vs MNA, $p = 0.013$, FA vs FNA, $p = 0.025$). Athletes have higher septal and lateral e' (Septal e'; MA 13.57 ± 2.66 cm/s vs MNA 11.46 ± 2.93 cm/s, $p < 0.001$, Lateral e'; MA 17.17 ± 3.07 cm/s vs MNA 14.82 ± 3.14 cm/s, $p < 0.001$), (Septal e'; FA 13.46 ± 2.32 cm/s vs FNA 12.16 ± 2.05 cm/s, $p = 0.04$, Lateral e'; FA 16.92 ± 2.97 cm/s vs FNA 15.44 ± 2.29 cm/s, $p = 0.006$).No difference in Global longitudinal (GLS), Right ventricle free wall (RVFWS) and Global circumferential strain (GCS). Left atrial reservoir (LArS) and left atrial booster strain (LAbS) is smaller in athletes. (LArS, MA 44.12 ± 9.55% vs MNA 52.95 ± 11.17%, $p < 0.001$ LArS, FA 48.07 ± 10.06% vs FNA 53.64 ± 8.99%, $p = 0.004$), (LAbS, MA 11.59 ± 5.13% vs MNA 17.35 ± 5.27%, $p < 0.001$ LAbS FA 11.77 ± 4.65% vs FNA 15.30 ± 4.19%, $p < 0.001$).
Conclusion: Malaysian athletes have thicker wall and bigger left ventricle than controls. No athletes have IVSd > 1.2 cm and/or LVIDd > 5.8 cm. There is no difference in GLS, RVFWS and GCS but athletes have smaller LArS and LAbS.

1. Introduction

The adaptation of athlete's heart continues to perplex clinicians since it was recognized at the end of 19th century through chest percussion of cross-country skiers [1]. Athletes can have bigger and thicker left ventricle that mimic dilated and thick wall cardiomyopathy [1]. The upper limits for these adaptations have been studied mainly in the Caucasian and Black athletes who are generally taller with bigger body surface area with scarce data from Asian countries. It is not known whether the threshold can also be applied to these populations [1–6]. These adaptations are not limited to the left side of the heart. The right heart also enlarges and in some circumstances differentiation from pathological process is difficult [7]. Another importance advances in the field of echocardiography is speckle tracking strain imaging that can detect cardiac dysfunction earlier with greater sensitivity [8].The aim of this study are (1) Ascertained remodeling characteristics in Malaysian

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athletes and non-athletes, compared to those in western population such as the upper limit of wall thickness, left ventricle dimension and also geometry (2) Echocardiographic and strain parameters in relatively less studied female athletes and non-athletes and finally (3) Comprehensive strain analysis including Global Longitudinal Strain (GLS), Right ventricle Free Wall Strain (RVFWS) and left ventricle circumferential strain (GCS) in athletes vs non-athletes.

2. Materials and methods

This is a prospective study done in collaboration between the National Heart Institute (IJN) and National Sport Institute of Malaysia (ISN). We recruited 50 elite male athletes (MA) versus 50 young healthy male non-athletes (MNA) and 50 elite female athletes (FA) versus 50 young healthy female non-athletes (FNA) in our study. All the study subjects have no medical illnesses and no symptoms. Their age must be between 18 and 30 years. Our athletes must have participated in national competition within 6 months of recruitment and the type of sport is predominantly endurance in nature. The training must be at least 4 days in a week with not<5 h per day. The demographic, blood pressure (BP), body surface area (BSA), heart rate (HR), weight and body mass index (BMI) are listed in Table 1 and the specific sports involved are listed in Table 2. Any participants with known medical illnesses, symptoms, or at imaging found to have obvious congenital cardiac abnormalities are excluded from the study. 2D- echocardiography and strain analysis are done in IJN by senior cardiac technologist and analyzed by experienced echocardiologist.

2.1. Echocardiographic data

Echocardiogram was done using Philips (Epiq CVX) machine. Ejection fraction (EF) was measured using biplane Simpson's technique. The left ventricle (LV) linear dimensions were measured at parasternal long axis (PLAX) view (Interventricular septum wall thickness in diastole (IVSd), posterior wall thickness in diastole (PWTd), left ventricle internal dimension in diastole (LVIDd), Interventricular septum wall thickness in systole (IVSs), posterior wall thickness in systole (PWTs) and left ventricle internal dimension in systole (LVIDs)).Relative wall thickness (RWT), left ventricle mass index (LVMi), Left ventricle end diastolic volume index (LVEDVi) and left ventricle end systolic volume index (LVESVi) were also analyzed. Zoom view at the PLAX was used to measure left ventricle outflow tract diameter (LVOTd) at mid-systole from blood-tissue interface to blood-tissue interface. For annulus, sinus of Valsalva (SOV), sino-tubular junction (STJ) and ascending aorta (ASCAo) the measurement was done at end-diastole. For the right ventricle (RV), RV focused view was utilized. Measurements was performed for right ventricle fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE), right ventricle systolic tissue velocity (RV S'), right ventricle basal dimension (RVBd), right ventricle mid dimension (RVMd), right ventricle longitudinal dimension (RVLd), myocardial performance index using blood doppler (Blood Tei) and using tissue doppler (Tissue Tei). Measurement of proximal right ventricular outflow tract diameter (pRVOT) and distal right ventricular outflow tract diameter (dRVOT) was done at aortic-pulmonary echocardiographic view (Ao-PA). Measurement of both left atrial volume index (LAVI) and right atrial volume index (RAVI) were done at apical-4 (A4C) and apical-2 chamber (A2C) views using area-length method. For diastology parameters measurement of tissue doppler was done at basal infero-septal (Early diastolic septal velocity (Septal e'), late diastolic septal velocity (Septal a') and systolic septal velocity (Septal s')) and at basal antero-lateral wall, Early diastolic lateral velocity (Lateral e'), late diastolic lateral velocity (Lateral a') and systolic lateral velocity (Lateral S') was done. Pulse wave doppler at the tip of mitral valve level in A4C was utilized to measure late diastolic mitral inflow duration (MVAdur) early diastolic mitral inflow velocity (E) and late diastolic mitral inflow velocity (A), and calculation of the E/A ratio and average E/e' ratio (E/((Septal e' + Lateral e')/2) was also done. Measurement of deceleration time (DT), isovolumetric relaxation time (IVRT) was performed and pulse wave (PW) doppler was positioned in the pulmonary vein to get systolic velocity (S), diastolic velocity (D) and pulmonary vein atrial reversal duration (PVAdur). We then calculate the value of PVAdur minus MVAdur (PVAdur-MVAdur). Finally, the stroke volume index (SVi) and cardiac index (CI) was calculated for all the participants.

Table 2
Athletes chosen are national level athletes that have competed within 6 months of the time of scanning and the sport is predominantly endurance in nature.

Sports Male		Sports Female	
Football	14	Netball	5
Ice Hockey	5	Hockey	5
Badminton	4	Cyclist	9
Swimming	4	Long distance	10
Squash	4	Rugby	7
Long distance	8	Squash	2
Cycling	4	Swimming	8
Hockey	7	Football	4
Total	50	Total	50

2.2. Strain analysis

Speckle tracking strain (endomyocardial strain) analysis was done using Tom Tec arena software also by Philips retrospectively by using A4C view, apical 3 chamber (A3C) view and A2C view for global longitudinal strain (GLS), Apical 4 chamber view for left atrial reservoir strain (LAr-S), left atrial conduit strain (LAc-S) and left atrial booster strain (LAb-S). For right ventricle free wall strain (RVFW-S) and right ventricle global longitudinal strain (RVGLS), right ventricle focused apical 4 chamber views was utilized. Finally, global circumferential strain (GCS) was done using short axis view at the mitral level (base), at the papillary muscles level (mid) and apical level (apex). All the strains are auto-strain where the software automatically traced the endocardial border and the echocardiographer can then do adjustments to get perfect tracing. Radial strain is not included in this study, as our institution's licensed software does not offer this feature. Additionally, our access to the software is limited due to cost constraints.

Table 1
There are 50 subjects in each category with age between 18 and 30 years old and no medical illnesses.

	Male athletes		Male non-athletes		Female athletes		Female non-athletes		MA vs MNA	FA vs FNA	MA vs FA	MNA vs FNA
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p1	p2	p3	p4
Age (years)	21.37	3.60	28.02	4.04	21.18	2.94	28.52	2.97	<0.001	<0.001	0.389	0.482
Weight (kg)	68.43	8.78	77.06	16.35	60.98	15.01	62.6	13.3	0.002	0.062	<0.001	<0.001
Height (cm)	172.02	5.05	170.48	5.96	160.04	17.14	156.5	5.55	0.078	<0.001	<0.001	<0.001
BMI (kg/m^2)	23.17	2.77	26.43	4.93	22.32	2.42	25.49	4.94	<0.001	<0.001	0.108	0.340
BSA (m^2)	1.81	0.12	1.9	0.22	1.63	0.1	1.64	0.19	0.005	0.540	<0.001	<0.001
HR (beats/min)	60.66	10.99	78	12.77	68.92	11.63	80.44	10.2	<0.001	<0.001	<0.001	0.302
Systolic BP (mmHg)	122.44	9.73	133.42	15.54	112.29	8.67	119.32	12.67	<0.001	0.002	<0.001	<0.001
Diastolic BP (mmHg)	75.58	8.28	85.32	9.74	72.1	9.52	77.44	10.57	<0.001	0.014	0.078	<0.001

2.3. Statistical analysis

The categorical variables were presented as percentage and the continuous variables were presented in terms of mean and standard deviations. Repeated measures ANOVA were used to compare differences between groups at different times points. P values < 0.05 were considered statistically significant. Statistical analysis was performed using SPSS ver. 27.0 (SPSS, Chicago, IL, USA).

3. Results

There are 50 subjects in each category. The mean age for MA is 21.17 ± 3.41 years old and MNA is 23.24 ± 2.8 years old. The mean age for FA is 21.24 ± 2.63 years old and FNA is 24.42 ± 3.17 years old. The mean heart rate (HR) for MA is 60.7 ± 11.0 beats/min and for MNA is 78.0 ± 12.8 beats/min. The mean HR for FA is 68.9 ± 11.6 beats/min and for FNA is 80.4 ± 10.2 beats/min. MA have significantly lower bodyweight than MNA (68.43 kg vs 77.06 kg, $p = 0.002$), while there is no statistical difference between FA vs FNA (60.98 kg vs 62.6 kg, $p = 0.062$) (Table 1). The types of sports practiced are listed in Table 2.

There is no statistical difference in EF between MA and MNA (60.5 ± 4.1% vs 61.6 ± 3.8%, $p = 0.142$). However, FA have lower EF than FNA (61.6 ± 4.0% vs 63.5 ± 2.8%, $p = 0.006$). MA have significantly bigger IVSd (Fig. 1), PWTd, LVIDd, LVEDVi and LVESVi than MNA (IVSd; MA 0.95 ± 0.12 cm vs MNA 0.86 ± 0.15 cm, $p = 0.003$, PWTd; MA 0.91 ± 0.11 cm vs MNA 0.83 ± 0.12 cm, $p < 0.001$, LVIDd; MA 5.05 ± 0.36 cm vs MNA 4.66 ± 0.4 cm, $p < 0.001$, LVEDVi; MA 67.36 ± 14.75mls/m^2 vs MNA 46.16 ± 8.31mls/m^2, $p < 0.001$, LVESVi; MA 27.84 ± 8.14mls/m^2 vs MNA 18.22 ± 5.01mls/m^2, $p < 0.001$). There is no statistical difference in IVSd (Fig. 2) between FA vs FNA (0.77 ± 0.11 cm vs 0.73 ± 0.1 cm, $p = 0.11$), however like MA, FA have significantly bigger PWTd, LVIDd, LVEDVi and LVESVi than FNA (PWTD; FA 0.80 ± 0.31 cm vs FNA 0.70 ± 0.12 cm, $p = 0.035$, LVIDd; FA 4.56 ± 0.32 cm vs FNA 4.39 ± 0.33 cm, $p = 0.009$, LVEDVi; FA 57.25 ± 11.55mls/m^2 vs FNA 43.55 ± 8.88mls/m^2, $p < 0.001$, LVESVi; FA 24.03 ± 7.55mls/m^2 vs FNA 16.30 ± 4.67mls/m^2, $p < 0.001$). Both MA and FA have higher LVMI than their counterparts (LVMI; MA 93.53 ± 15.85 g/m^2 vs MNA 69.11 ± 12.87 g/m^2, $p < 0.001$ and FA 71.57 ± 28.21 g/m^2 vs FNA 57.66 ± 8.87 g/m^2, $p = 0.002$). The LVOTd and annulus is bigger for MA vs MNA and not significant for FA vs FNA (LVOTd; MA 2.23 ± 0.14 cm vs MNA 2.12 ± 0.16 cm, $p < 0.001$, annulus; MA 2.20 ± 0.14 cm vs MNA 2.12 ± 0.16 cm, $p = 0.007$, LVOTd; FA 1.95 ± 0.13 cm vs FNA 1.93 ± 0.15 cm, $p = 0.606$, annulus; FA 1.92 ± 0.13 cm vs FNA 1.89 ± 0.16 cm, $p = 0.315$). On the other hand, FA have significantly smaller SOV and STJ diameter versus FNA whereas these are not significant for MA vs MNA. (SOV; FA 2.53 ± 0.18 cm vs FNA 2.63 ± 0.24 cm, $p = 0.025$, STJ; FA 2.13 ± 0.19 cm vs FNA 2.30 ± 0.27 cm, $p < 0.001$, SOV; MA 3.01 ± 0.26 cm vs 3.03 ± 0.34 cm, $p = 0.694$, STJ; MA 2.40 ± 0.24 cm vs MNA 2.50 ± 0.30 cm, $p = 0.073$) (Table 3).

For the right heart parameters, RVFAC is smaller for MA and FA versus their counterparts. However, for both TAPSE and RVS' there is no differences between athletes and non-athletes. (RVFAC; MA 42.8 ± 7.6% vs MNA 47.2 ± 10.0%, $p = 0.013$, RVFAC; FA 45.52 ± 6.3% vs FNA 48.68 ± 7.4%, $p = 0.025$). Right ventricle dimensions are significantly bigger for MA and FA versus their counterparts. (RVBd; MA 3.92 ± 0.32 cm vs MNA 3.23 ± 0.39 cm, $p < 0.001$, RVBd; FA 3.52 ± 0.34 cm vs FNA 3.03 ± 0.37 cm, $p < 0.001$, RVMd; MA 3.26 ± 0.31 cm vs MNA 2.79 ± 0.41 cm, $p < 0.001$, RVMd; FA 3.00 ± 0.38 cm vs 2.52 ± 0.11 cm, $p < 0.001$, RVLd; MA 7.47 ± 0.98 cm vs MNA 6.29 ± 0.83 cm, $p < 0.001$, RVLd; FA 6.45 ± 0.76 cm vs FNA 5.66 ± 0.70 cm, $p < 0.001$). RAVI is significantly bigger in both MA vs MNA and FA vs FNA (RAVI; MA 25.21 ± 8.7mls/m^2 vs MNA 16.89 ± 4.29mls/m^2, $p < 0.001$, RAVI; FA 19.40 ± 5.32mls/m^2 vs FNA 14.66 ± 3.92mls/m^2, $p < 0.001$) (Table 3).

MA and FA have significantly higher septal and lateral e' but significantly lower septal and lateral a' compare to MNA and FNA (Septal e'; MA 13.57 ± 2.66 cm/s vs MNA 11.46 ± 2.93 cm/s, $p < 0.001$, Septal e'; FA 13.46 ± 2.32 cm/s vs FNA 12.16 ± 2.05 cm/s, $p = 0.04$, Lateral e'; MA 17.17 ± 3.07 cm/s vs MNA 14.82 ± 3.14 cm/s, $p < 0.001$, Lateral e'; FA 16.92 ± 2.97 cm/s vs FNA 15.44 ± 2.29 cm/s, $p = 0.006$, Septal a'; MA 6.55 ± 1.56 cm/s vs MNA 8.96 ± 1.69 cm/s, $p < 0.001$, Septal a'; FA 6.70 ± 1.08 cm/s vs FNA 7.91 ± 1.28 cm/s, $p < 0.001$, Lateral a'; MA 6.71 ± 1.67 cm/s vs MNA 8.71 ± 2.15 cm/s, $p < 0.001$, Lateral a'; FA 7.12 ± 1.7 cm/s vs FNA 8.17 ± 1.72 cm/s, $p = 0.003$). There are no differences in E velocity between athletes and non-athletes, but A velocities are lower for athletes compare to their same sex counterpart. Therefore, the E/A ratio are also higher for MA and FA versus their non-athletes counterparts (A; MA 35.84 ± 8.47 cm/s vs MNA 49.77 ± 11.54 cm/s, $p < 0.001$, A; FA 42.33 ± 14.11 cm/s vs FNA 49.26 ± 11.35 cm/s, $p = 0.009$, E/A; MA 2.26 ± 0.66 vs MNA 1.69 ±

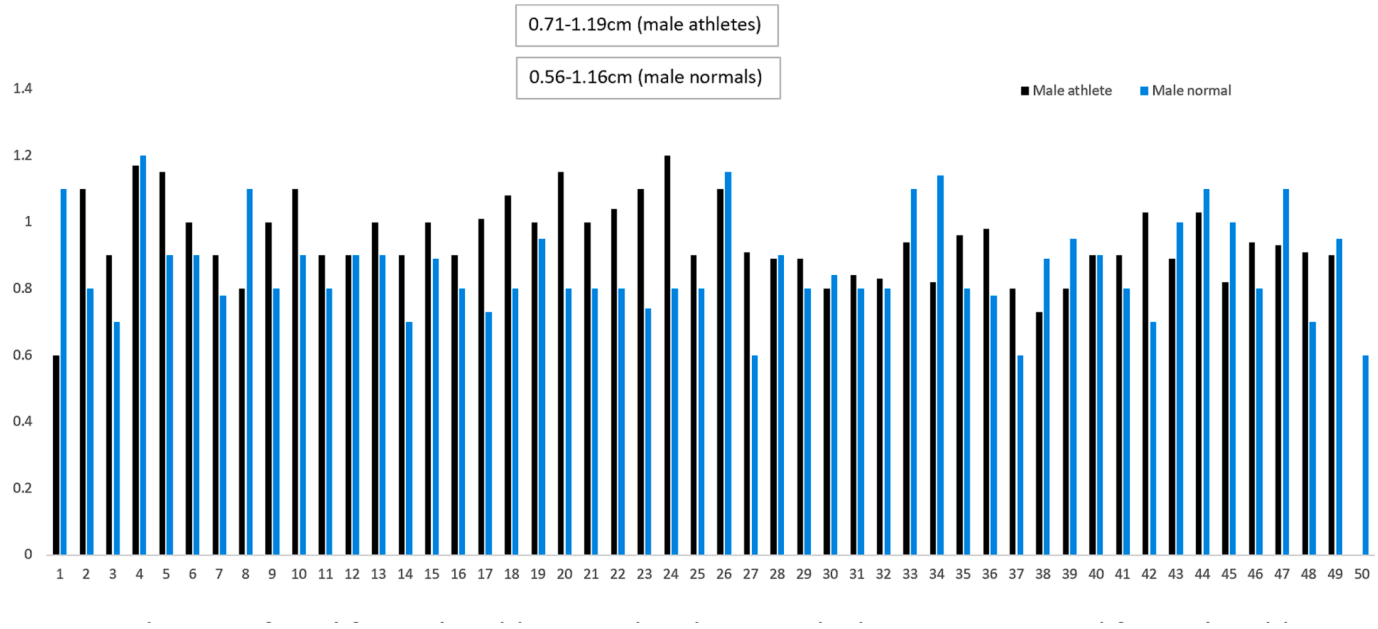


Fig. 1. Distribution of IVSd for male athletes and male normal. The maximum IVSd for male athletes is 1.2 cm.

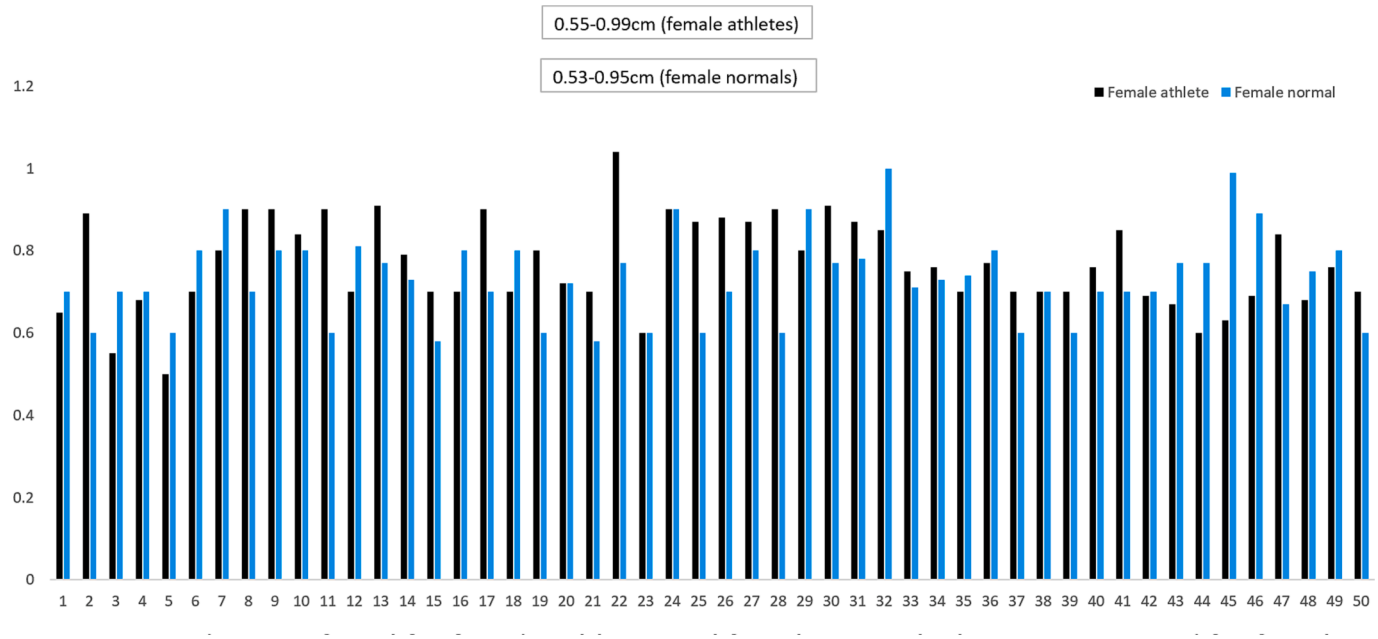


Fig. 2. Distribution of IVSd for female athletes and female normal. The maximum IVSd for female athletes is 1.04 cm.

Table 3
Left and right heart echocardiographic parameters. Athletes have significant enlargement of all cardiac chambers compare to non-athletes (p1 = MA vs MNA, p2 = FA vs FNA, p3 = MA vs FA and p4 = MNA vs FNA).

	Male athletes		Male non-athletes		Female athletes		Female non-athletes					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p1	p2	p3	p4
EF (%)	60.5	4.05	61.6	3.8	61.6	3.95	63.5	2.75	0.142	0.006	0.179	0.007
IVSd (cm)	0.95	0.12	0.86	0.15	0.77	0.11	0.73	0.1	0.003	0.11	<0.001	<0.001
PWTd (cm)	0.91	0.11	0.83	0.12	0.8	0.31	0.7	0.12	<0.001	0.035	0.021	<0.001
LVIDd (cm)	5.05	0.36	4.66	0.4	4.56	0.32	4.39	0.33	<0.001	0.009	<0.001	<0.001
IVSs (cm)	1.27	0.17	1.24	0.17	1.07	0.14	1.07	0.14	0.473	0.943	<0.001	<0.001
PWTs (cm)	1.42	0.16	1.35	0.2	1.25	0.17	1.13	0.2	0.086	0.095	<0.001	<0.001
LVIDs (cm)	3.4	0.39	2.88	0.39	2.94	0.29	2.7	0.32	<0.001	<0.001	<0.001	0.01
LVEDVi (mls/m^2)	67.36	14.75	46.16	8.31	57.25	11.55	43.55	8.88	<0.001	<0.001	<0.001	0.133
LVESVi (mls/m^2)	27.84	8.14	18.22	5.01	24.03	7.55	16.3	4.67	<0.001	<0.001	0.018	0.051
LVMI (g/m^2)	93.53	15.85	69.11	12.87	71.57	28.21	57.66	8.87	<0.001	0.002	<0.001	<0.001
RWT	0.36	0.05	0.36	0.06	0.35	0.13	0.32	0.06	0.705	0.131	0.546	0.003
SVi (mls/m^2)	42.24	9.98	34.81	6.72	40.34	8.08	36.43	4.98	<0.001	0.005	0.301	0.173
CI (L/mins/m^2)	2.82	2.9	2.44	0.5	3.74	7.66	2.59	0.5	0.37	0.297	0.433	0.136
LVOTd (cm)	2.23	0.14	2.12	0.16	1.95	0.13	1.93	0.15	<0.001	0.606	<0.001	<0.001
Annulus	2.2	0.14	2.12	0.16	1.92	0.13	1.89	0.16	0.007	0.315	<0.001	<0.001
SOV (cm)	3.01	0.26	3.03	0.34	2.53	0.18	2.63	0.24	0.694	0.025	<0.001	<0.001
STJ (cm)	2.4	0.24	2.5	0.303	2.13	0.19	2.3	0.27	0.073	<0.001	<0.001	<0.001
ASCAo (cm)	2.54	0.24	2.53	0.312	2.25	0.63	2.38	0.28	0.83	0.022	<0.001	0.012
RVFAC (%)	42.75	7.56	47.22	9.96	45.52	6.29	48.68	7.43	0.013	0.025	0.052	0.41
TAPSE (cm)	2.31	0.36	2.29	0.31	2.34	0.33	2.32	0.26	0.788	0.655	0.626	0.66
RV S' (cm/s)	13.31	1.5	13.22	1.84	12.71	1.97	13.16	1.48	0.801	0.196	0.089	0.848
RVBd (cm)	3.92	0.32	3.23	0.389	3.52	0.34	3.03	0.37	<0.001	<0.001	<0.001	0.012
RVMd (cm)	3.26	0.31	2.79	0.41	3	0.38	2.52	0.11	<0.001	<0.001	<0.001	0.002
RVLd (cm)	7.47	0.98	6.29	0.83	6.45	0.76	5.66	0.7	<0.001	<0.001	<0.001	<0.001
pRVOT (cm)	2.78	0.49	2.55	0.47	2.54	0.35	2.44	0.42	0.016	0.198	0.005	0.209
dRVOT (cm)	2.35	0.3	2.23	0.39	2.15	0.3	2.11	3.27	0.091	0.482	0.001	0.08
RAVI (mls/m^2)	25.21	8.7	16.89	4.29	19.4	5.32	14.66	3.92	<0.001	<0.001	<0.001	0.008
Blood Tei	0.21	0.09	0.19	0.13	0.18	0.13	0.19	0.09	0.529	0.577	0.196	0.9
Tissue Tei	0.41	0.12	0.37	0.07	0.34	0.06	0.32	0.08	0.029	0.136	<0.001	0.002

0.51, $p < 0.001$, E/A; FA 2.33 ± 0.82 vs FNA 1.89 ± 0.50, $p = 0.002$). The Average E/e' for MA is significantly lower than MNA but not significant for FA versus FNA. LAVI is significantly bigger for both MA vs MNA and FA vs FNA (Average E/e'; MA 5.11 ± 1.03 vs MNA 6.21 ± 1.39, $p < 0.001$, Average E/e'; FA 6.25 ± 2.33 vs FNA 6.5 ± 1.11, $p = 0.49$, LAVI; MA 31.09 ± 9.76mls/m^2 vs MNA 22.70 ± 5.52mls/m^2, $p < 0.001$, LAVI; FA 28.00 ± 6.16mls/m^2 vs FNA 23.18 ± 5.16mls/m^2, $p < 0.001$) (Table 4).

Athletes have significantly lower LArS and LAbS than non-athletes (LArS; MA 44.12 ± 9.55% vs MNA 52.95 ± 11.17%, $p < 0.001$, LArS; FA 48.07 ± 10.06% vs FNA 53.64 ± 8.99%, $p = 0.004$, LAbS; MA 11.59 ± 5.13% vs MNA 17.35 ± 5.27%, $p < 0.001$, LAbS; FA 11.77 ± 4.65% vs 15.3 ± 4.19%, $p < 0.001$). There are no significant differences between athletes and non-athletes in GLS, RVFWS, RVGLS and GCS (Table 5, Fig. 5).

Despite higher left ventricle mass index, most athletes have normal

Table 4
Diastology parameters. Athletes have significantly higher septal e', lateral e' and LAVI compared to non-athletes. (p1 = MA vs MNA, p2 = FA vs FNA, p3 = MA vs FA and p4 = MNA vs FNA).

	Male athletes		Male normal		Female athletes		Female normal					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p1	p2	p3	p4
Septal e' (cm/s)	13.57	2.66	11.46	2.93	13.46	2.32	12.16	2.05	<0.001	0.04	0.818	0.17
Septal a'(cm/s)	6.55	1.56	8.96	1.69	6.7	1.08	7.91	1.28	<0.001	<0.001	0.593	<0.001
Septal s'(cm/s)	8.11	1.37	8.36	1.24	7.41	1.22	7.7	1.28	0.348	0.265	0.009	0.01
Lateral e'(cm/s)	17.17	3.07	14.82	3.14	16.92	2.97	15.44	2.29	<0.001	0.006	0.686	0.264
Lateral a'(cm/s)	6.71	1.67	8.71	2.15	7.12	1.7	8.17	1.72	<0.001	0.003	0.231	0.166
Lateral s'(cm/s)	8.93	2.5	8.81	1.88	8.74	1.81	8.88	1.93	0.792	0.713	0.669	0.86
E velocity(cm/s)	77.62	16.36	80.41	16.3	91.38	20.1	89.05	16.44	0.397	0.527	<0.001	0.01
A velocity(cm/s)	35.84	8.47	49.77	11.54	42.33	14.11	49.26	11.35	<0.001	0.009	0.007	0.822
E/A	2.26	0.66	1.69	0.51	2.33	0.82	1.89	0.5	<0.001	0.002	0.643	0.047
Average E/e'	5.11	1.03	6.21	1.39	6.25	2.33	6.5	1.11	<0.001	0.49	0.002	0.247
DT (ms)	178.8	41.2	178.74	36.15	179.2	35.6	167.98	33.08	0.994	0.107	0.962	0.126
IVRT (ms)	76.61	12.91	70.96	13.1	65.89	11.06	64.03	8.3	0.034	0.343	<0.001	0.002
S/D	0.91	0.27	1.05	0.26	0.98	0.31	1.23	0.32	0.012	<0.001	0.277	0.002
PVAdur-MVAdur (ms)	9.67	25.4	−0.02	19.55	19.7	17.63	5.43	19.24	0.037	<0.001	0.026	0.083
LAVI (mls/m^2)	31.09	9.76	22.7	5.52	28	6.16	23.18	5.16	<0.001	<0.001	0.064	0.653

Table 5
Athletes have significantly lower LArS and LAbS than non-athletes. There are no significant differences in RVFWS, RVFWS, RVGLS and GCS between athletes and non-athletes. (p1 = MA vs MNA, p2 = FA vs FNA, p3 = MA vs FA and p4 = MNA vs FNA).

	Male athletes		Male normal		Female athletes		Female normal					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p1	p2	p3	p4
GLS (%)	19.96	2.37	19.23	2.75	21.91	2.54	21.59	2.50	0.16	0.53	<0.001	<0.001
LArS (%)	44.12	9.55	52.95	11.17	48.07	10.06	53.64	8.99	<0.001	0.004	0.048	0.73
LAcS (%)	32.53	6.45	35.87	9.05	36.3	8.30	38.34	8.52	0.038	0.232	0.013	0.166
LAbS (%)	11.59	5.13	17.35	5.27	11.77	4.65	15.3	4.19	<0.001	<0.001	0.85	0.03
RVFWS (%)	23.97	4.40	25.2	5.85	26.52	3.71	26.97	4.44	0.24	0.586	0.002	0.095
RVGLS (%)	21.04	3.34	21.65	4.21	22.85	3.08	23.49	3.54	0.43	0.34	0.006	0.02
GCS Avg (%)	21.03	2.77	20.9	4.04	22.75	2.39	22.45	3.43	0.85	0.62	0.001	0.042

left ventricle geometry (80% of MA and 96% of FA). The second commonest left ventricle geometry for MA and FA is eccentric hypertrophy (10% and 4% respectively). 18 (36%) male athletes have LAVI > 34mls/m^2 and 8 (16%) female athletes have LAVI > 34mls/m^2 (Fig. 3, Fig. 4). 22% of MA have RAVI > 32mls/m^2 and 10% of FA have RAVI > 27mls/m^2. 34% MA have LVEDVi > 74mls/m^2 and 40% of FA have LVEDVi > 61mls/m^2. 64% of both MA and FA have E/A > 2.0. None of the athletes have LVIDd > 5.8 cm and only 6% of the FA have LVIDd > 5.2 cm. Higher proportion of MA have adequate tricuspid regurgitation (TR) doppler to measure peak TR velocity and gradient vs MNA (40% vs 16%) while the proportion are the same for FA vs FNA (34% vs 34%). None of the participants have peak TR velocity > 2.8 m/s or inferior vena cava (ivc) > 2.1 cm and non-collapsible. (Table 6).

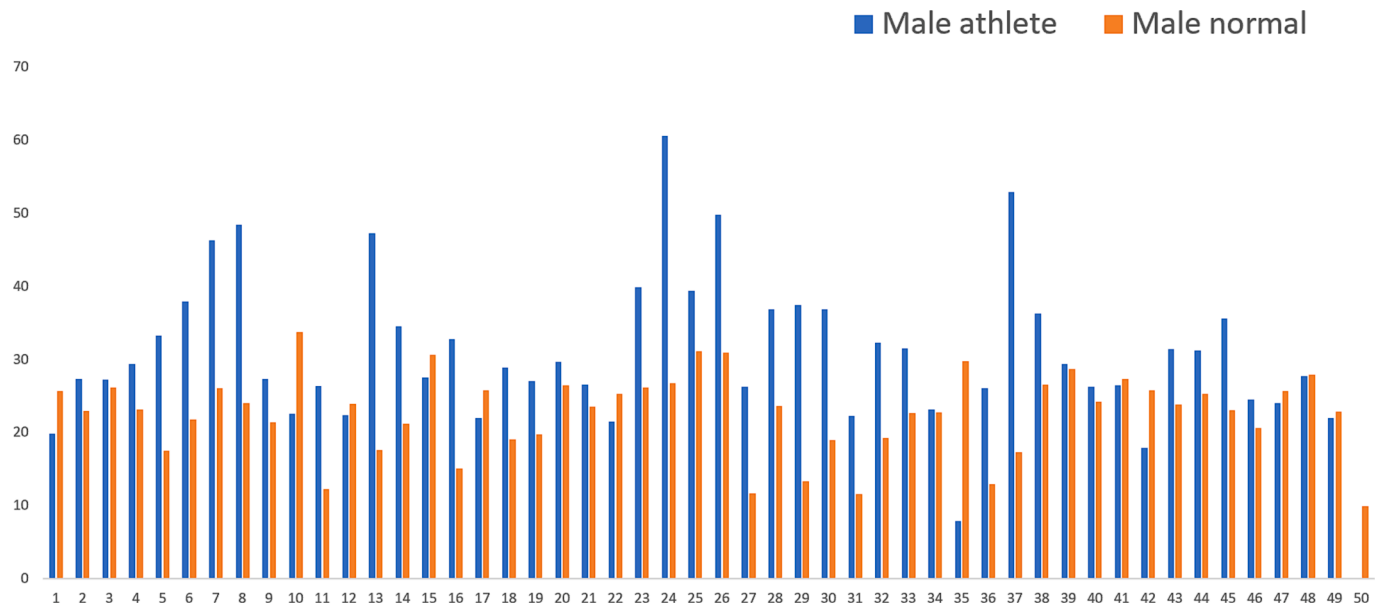


Fig. 3. Male athletes have significantly higher LAVI than male non-athletes. 18 (36%) male athletes have LAVI > 34mls/m^2 and none of the non-athletes have LAVI > 34mls/m^2.

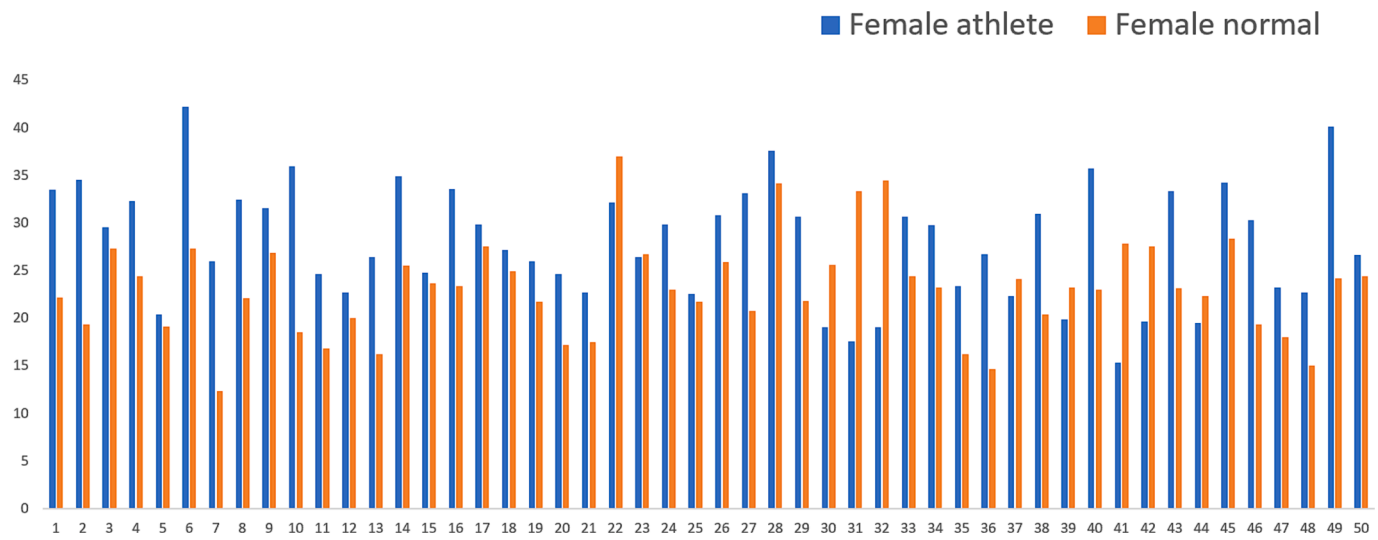
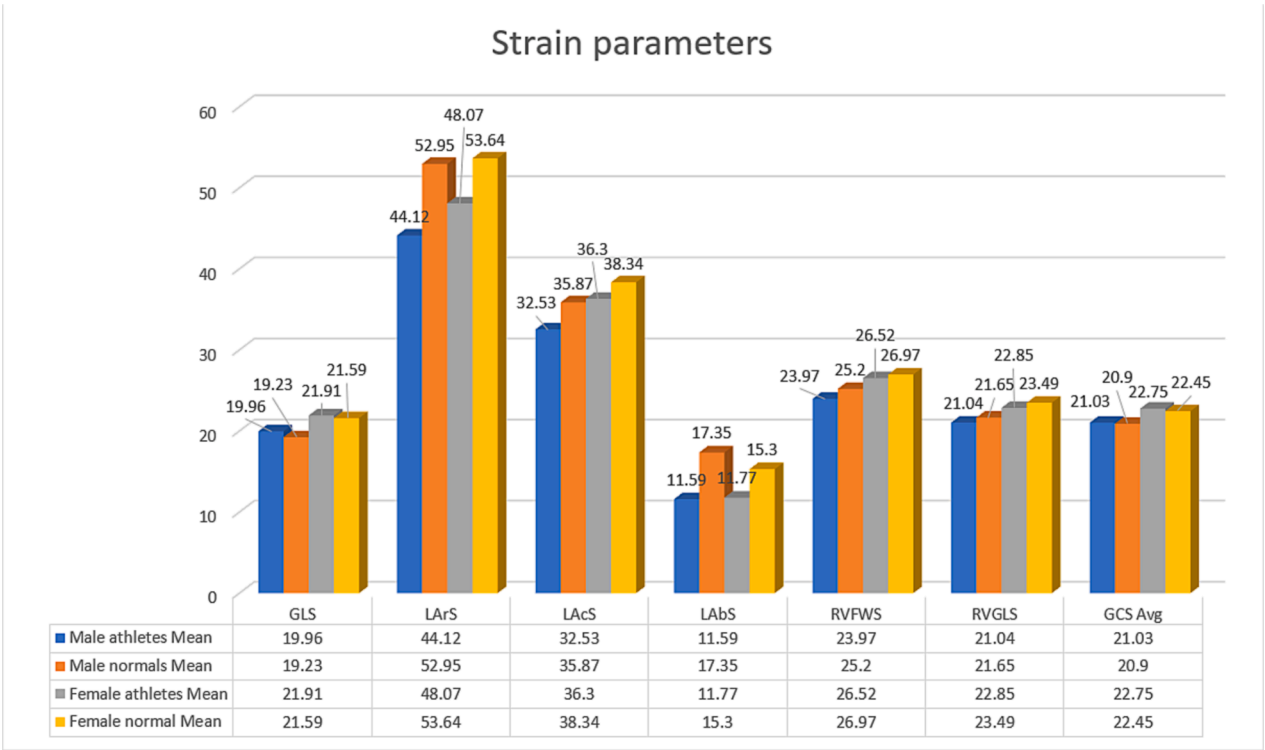


Fig. 4. Female athletes have significantly higher LAVI than female non-athletes. 8 (16%) of female athletes have LAVI > 34mls/m^2 and 3 (6%) of female non-athletes have LAVI > 34mls/m^2.



	GLS	LArS	LAcS	LAbS	RVFWS	RVGLS	GCS Avg
Male athletes Mean	19.96	44.12	32.53	11.59	23.97	21.04	21.03
Male normals Mean	19.23	52.95	35.87	17.35	25.2	21.65	20.9
Female athletes Mean	21.91	48.07	36.3	11.77	26.52	22.85	22.75
Female normal Mean	21.59	53.64	38.34	15.3	26.97	23.49	22.45

Fig. 5. Strain analysis. GLS, RVFWS, RVGLS AND GCS are not statistically difference between athletes and non-athletes. LArS and LAbS are significantly lower in athletes compare to nonathletes. GLS, RVGLS and GCS are higher in female than in male.

4. Discussion

This study involved sports that have predominantly endurance training. Endurance exercise cause cardiac chambers enlargement which generate large stroke volume alongside reduction in peripheral vascular resistance [1,2,4]. These adaptations happen in respond to predominantly volume loaded left ventricle in order to normalized wall stress [1]. The left ventricle dilatation and increase in wall thickness in athletes can leads to erroneous diagnosis of dilated cardiomyopathy, thick wall cardiomyopathy or even arrhythmogenic right ventricle cardiomyopathy (ARVD) [1–4,7]. Furthermore, hypertrophic cardiomyopathy (HCM) and ARVD are among the commonest cause of sudden cardiac death, and this will influence whether athletes can continue to compete [9]. Most study in athletes' heart are done in western Caucasian and Black athletes who generally are taller, with bigger body surface area [1–4,7]. Therefore, this study sought to investigate the extend of athlete heart adaptation in Malaysia. In addition, we also incorporated relatively novel strain analysis including left atrial, right atrial, and circumferential strain.

Most study shows that athletes have normal EF [1,3,10]. This study also shows that our athletes have EF within the normal range, even though EF for FA is lower than FNA (61.6% vs 63.5%, p = 0.006) due to

Table 6
Proportion of athletes with selected echocardiographic parameters. (p1 = MA vs MNA, p2 = FA vs FNA, p3 = MA vs FA and p4 = MNA vs FNA).

	Male athletes		Male normal		Female athletes		Female normal	
	Number	%	Number	%	Number	%	Number	%
Normal geometry	40	80	45	90	48	96	46	92
Eccentric hypertrophy	5	10	0	0	2	4	0	0
Concentric hypertrophy	1	2	0	0	0	0	0	0
Concentric remodelling	4	8	5	10	0	0	4	8
LAVI > 34mls/m2	18	36	0	0	8	16	3	6
RAVI > 32mls/m2 for male, >27mls/m2 for female	11	22	0	0	5	10	0	0
LVEDVi > 74mls/m2 for male, >61mls/m2 for female	17	34	0	0	20	40	2	4
LVESVi > 31mls/m2 for male, > 24mls/m2 for female	16	32	1	2	20	40	3	6
E/A > 2.0	32	64	8	16	32	64	21	42
Adequate TR doppler	20	40	8	16	17	34	17	34
TR v max > 2.8 m/s	0	0	0	0	0	0	0	0
IVC > 2.1 cm and < 50% collapsible	0	0	0	0	1	2	0	0
LVIDd > 5.8 cm for male, >5.2 cm for female	0	0	0	0	3	6	0	0

more pronounced increased in LV volumes in FA. Similar to previous studies [1,3,4,10], our athletes have bigger left ventricle dimensions and volumes. In a study of 1309 elite athletes by Pelliccia et al, 14% of the athletes have LVIDd > 6.0 cm [10]. Unlike western counterparts none of our athletes have LVIDd > 6.0 cm. In fact, none of our athletes have LVIDd > 5.8 cm and LVIDd > 5.8 cm in our population therefore might be suspicious for pathological processes. Another finding that often cause dilemma to clinicians is the increase in wall thickness in athletes. This is usually accompanied by dilated left ventricle in normal athletes' heart adaptation [3]. Abnormal wall thickness with small LV cavity on the other hand is suggestive of pathological processes [1,4]. How much wall thickness is abnormal and cannot be attributed to athletes' heart? Another earlier study by Pellicia et al shows that it is very rare for athletes to have IVSd > 12 mm which is only identified in 16 out of 947 (1.7%) athletes [3]. The thickest wall up to 16 mm is found in male black or African American basketball players and none of female athletes have thickness>12 mm [3]. In this study none of our athletes have IVSd > 12 mm suggesting that this might be the value above which thick wall or hypertensive cardiomyopathy should be suspected. Even though the left ventricle mass is higher in athletes and previous studies indicating that athletes commonly have eccentric hypertrophy, vast majority of athletes in our study have normal left ventricle geometry (80% of MA and 96% of FA) and only 10% of MA and 4% of FA have eccentric hypertrophy [1].

Athletes heart have what is term "balanced-ventricle". The right ventricle tracks the left ventricle dilatation as what goes out from the left ventricle must also come from the right ventricle in the absence of shunt [7]. Like other studies, our study shows that the right ventricle dimensions (basal, mid, longitudinal and outflow tract) are bigger in athletes versus non-athletes [11]. Importantly however, the dimensions do not exceed normal values and involves all part of the right ventricle. Interestingly, in our study FAC is smaller in athletes but both TAPSE and S' are similar for athletes and non-athletes. This is because FAC reflects volume which is higher in athletes due to increase in stroke volumes, and therefore athletes need less fraction of this volume to pump out same cardiac output as non-athletes. On the other hand, TAPSE and S' reflects longitudinal function which is preserved in athletes consistent with normal RVFWS and RVGLS findings in this study. Other points to differentiate ARVC is the absence of regional wall motion abnormalities or aneurysm in athletes' heart [7].

One of the largest studies on diastolic function in athletes shows similar E velocity but significantly decreased A velocity and significantly lower doppler tissue septal e' in athletes [12]. Another earlier study on the other hand shows no difference in septal e' between athletes and controls [8].In fact, low left ventricle tissue doppler indicates sick myocardium and points against normal athletes' adaptation. In our study, left ventricle early relaxation is significantly better in athletes, with higher septal e' and lateral e' for both MA and FA and lower E/e' for MA. The E/A ratio is also higher in athletes due to smaller a velocity. This is important because E/A > 2 is common in athletes (64% of MA and FA in this study) and erroneous application of diastology can mistakenly call athletes to have restrictive or Grade 3 diastolic dysfunction. Left atrial enlargement is also a common adaptation in athletes' heart and have close association with LV cavity enlargement [13–15]. In our study, LAVI is much higher in athletes and significant minority of athletes have value > 34mls/m^2 (36% MA and 16% FA in this study) and therefore this value in isolation should not be used to indicate diastolic dysfunction in well trained athletes.

GLS is a more specific and sensitive index of left ventricle function [16]. This is because subendocardial is the first layer to be involved in many cardiac diseases [16].Almost all patients with hypertrophic cardiomyopathy with preserved ejection fraction have reduction in average GLS [17,18]. This is also true for other thick wall cardiomyopathy like cardiac amyloidosis and Andersons Fabry disease [17,18]. GLS values for athletes versus non-athletes in literatures are mixed with some studies showing lower values for athletes [18,19] and other studies showing no difference [17,20,21]. Importantly however, all these studies shows that the GLS for athletes is still within the normal range [17–21]. Therefore, abnormal GLS is against normal athletes' adaptation with value < -12% suggested as definitely abnormal value [16]. In our study there are no differences in GLS values between athletes and non-athletes. 2 standard deviation lower limit for MA is −15.22% and 2 standard deviation lower limit for FA is −16.83%.

Right ventricle strain is an important tool to detect dysfunction as it is relatively easy to do and can overcome other 2-dimensional limit of RV quantitation. Studies done before this also show mixed result for the values of RV strain with some shows higher value in athletes [22] and some show lower values [23]. Lower RV GLS is postulated due to lower basal RV deformation needed to generate the same stroke volume [23]. In this study there is no difference in RVFWS, RVGLS and GCS between athletes and non-athletes and all the values are within normal range.

Interestingly the LArS and LAbS are statistically smaller in athletes versus non-athletes. This is counterintuitive but there are suggestions that even though resting LAbS and LArS are smaller in athletes at rest, during exercise these strains is augmented higher than non-athletes suggesting extra cardiac reserves in athletes [24].Our study is similar to a *meta*-analysis published in 2019 showing significant reduction of LArS and LAbS in athletes. An intriguing observation lies in the similarity of LA strain patterns (reduction in LArS and LAbS) between HCM patients and athletes. In such cases, GLS can serve as a useful parameter, as it tends to be consistently low in HCM patients while remaining within the normal range for athletes. Additionally, as mentioned earlier, the role of exercise becomes significant, as it leads to improvement in athletes but diminished effects in HCM patients.

4.1. Study limitation

Our study is limited by resources to enroll higher number of athletes. Because of this we are unable to strictly recruit similar types of sports between the female and male athletes. Because of the same limitation even though all the participants are between 18 and 30 years of age, we are unable to strictly controlled the age difference between groups. It is important to note that the findings of this study cannot be extrapolated to older age groups, specifically Masters athletes. The scope of this research is limited to a specific age range and demographic group, and therefore, caution must be exercised when applying the results to other populations.

5. Conclusions

Malaysian elite athletes have bigger left and right ventricle, thicker wall but this occurs to a smaller extent compared to western athletes. The LArS and LAbS is smaller in athletes but there are no differences in GLS, RVFWS, RVGLS and GCS.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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