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Myocardial Performance in Elite Athletes: The Role of Homocysteine, Iron, and Lipids

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Background: The myocardial performance index (MPI) is a comprehensive measure of global systolic and diastolic function of the ventricle, and it has an inverse correlation with maximal oxygen consumption. In this study, the potential association between left ventricle MPI and biochemical biomarkers (including iron, homocysteine, and lipids) in elite athletes was investigated.

Material/Methods: This cross-sectional observational study consisted of 80 young male elite soccer and basketball players (age: 18–34 years) examined for a seasonal medical check-up. Cardiological examinations and transthoracic echocardiography of these athletes were performed and blood samples were analyzed according to standard laboratory protocols. Tissue Doppler recording was acquired from the mitral annulus using apical 4-chamber view and then the tissue Doppler-derived MPI was computed.

Results: Athletes were separated into 2 groups based on MPI values (MPI ≤0.40 and MPI >0.40), and baseline demographic, clinical, and biochemical variables of the study participants were compared between these 2 groups. Serum triglyceride, high-density lipoprotein, total cholesterol, homocysteine levels, and iron parameters did not significantly differ between groups, while low-density lipoprotein level was significantly lower in the MPI ≤0.40 group (103.8±26.0 mg/dl *vs.* 116.8±30.2 mg/dl; p=0.043). Correlation analysis and multivariate linear regression analysis demonstrated a significant association between low-density lipoprotein and MPI.

Conclusions: In this study, various biochemical markers were evaluated for possible association with left ventricle MPI as a surrogate of cardiac performance. Among these biomarkers, only low-density lipoprotein was significantly associated with MPI in elite athletes.

MeSH Keywords: **Athletes • Homocysteine • Iron • Lipoproteins, LDL • Myocardium**

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Background

Beneficial effects of regular moderate exercise on general health status and cardiovascular functions are widely accepted. Current data suggest that cardiorespiratory fitness, lipid profiles, blood pressure, and weight control are all positively influenced by regular moderate bouts of exercise [1–3]. On the other hand, morphological, functional, and electrical changes may be induced by intense exercise in which physiological or pathological conditions may not be easily differentiated. In addition to the frequently mentioned sudden cardiac death risk, long and intensive endurance exercises may result in cardiac fatigue associated with ventricular systolic and diastolic dysfunction [4–6]. Moreover, cardiac performance is one of the major factors affecting exercise capacity and, hence, athletic performance [**7**], and defining prospective echocardiographic and metabolic markers of cardiac dysfunction or cardiac fitness may be beneficial for the maintenance of athletic performance and athlete's health status as well. Sole evaluation of ejection fraction for the systolic function may not adequately reflect the subtle changes in an athlete's heart, and additional evaluation of diastolic functions may be necessary, as the diastolic dysfunction occurs before systolic dysfunction in both ischemic and non-ischemic models [8,9].

In this context, the myocardial performance index (MPI) is a comprehensive measure of global systolic and diastolic function of the ventricle, of which lower values represent better cardiac performance [10,11]. It is also a useful parameter for early detection of left ventricle dysfunction, even in patients with normal systolic functions such as critical coronary disease or occult cardiac toxicity secondary to chemotherapy [12,13]. MPI is relatively independent of loading conditions, heart rate, and ventricular geometry, which may be substantially altered in an athlete's heart. Furthermore, MPI shows an inverse correlation with maximum oxygen consumption, which is one of the most widely used fitness measures of human performance [14]. All these properties of MPI suggest the potential value of this highly reproducible index in the estimation of athletic cardiac fitness and fatigue status.

The objective of this study was to investigate the potential association between left ventricle MPI as a surrogate of cardiac performance and biochemical markers related to physical fitness, nutrient deficiency, overtraining, cardiovascular risk, inflammation, and metabolism (including iron, homocysteine, and lipids) in elite athletes.

Material and Methods

Design and study population

This cross-sectional observational study included 80 male soccer and basketball players (age: 18–34 years) who were examined at Acıbadem Ankara Hospital during a seasonal medical checkup between the years 2016 and 2017. All soccer and basketball players were professional licensed members of a national (3 Premier Football League teams and one Basketball League teams) league team and actively attending the regular training programs. Cardiological examinations and transthoracic echocardiography imaging of these athletes were performed by a single cardiologist. Blood samples were analyzed according to standard laboratory protocols. Athletes with a personal history of hypertension, coronary artery disease, diabetes mellitus, hypertrophic cardiomyopathy, syncope, or electrocardiographic or echocardiographic high-risk features of sudden cardiac death were excluded. None of the participating athletes had acute metabolic disease, any form of medication use, or poor echocardiographic imaging windows, enabling proper evaluation. Acıbadem University Medical Ethics Committee approved the study (ATADEK approval no. 2017/12).

Biochemical analysis

Venous blood samples were collected at 08–10 a.m. following 12 h of fasting and were promptly analyzed. Complete blood count was performed using an automated hematology analyzer (Sysmex XT 2000i, SYSMEX Corp, Hyogo, Japan). Blood glucose, electrolytes (Sodium, Chlorine, Potassium, Calcium, Magnesium) iron parameters (Serum iron, Total iron binding capacity, Transferrin saturation, Unsaturated iron binding capacity), Urea, Uric acid, Serum creatinine, lipid parameters (Triglycerides, Total Cholesterol, High-density lipoprotein, Low-density lipoprotein), Alanine Aminotransferase, Aspartate Aminotransferase, Gamma Glutamyl Transferase, Alkaline Phosphatase, Lactate Dehydrogenase, C-Reactive Protein, Total protein, Albumin, Homocysteine, Thyroid-Stimulating Hormone, and Vitamin B12 levels were measured with an automated analyzer (Roche Integra 400, Roche Diagnostics, Switzerland) using commercially available kits.

Echocardiography

Transthoracic echocardiography was performed in accordance with current practice guidelines [15] using the Vivid 7 pro system (GE Vingmed, Horten, Norway) with a 2.0–3.5-MHz transducer by a single experienced cardiologist. Left ventricular internal dimensions were measured during end-diastole and end-systole in the parasternal long-axis view just below the level of the mitral valve leaflet tips, avoiding oblique measurements. Left ventricle ejection fraction was calculated according

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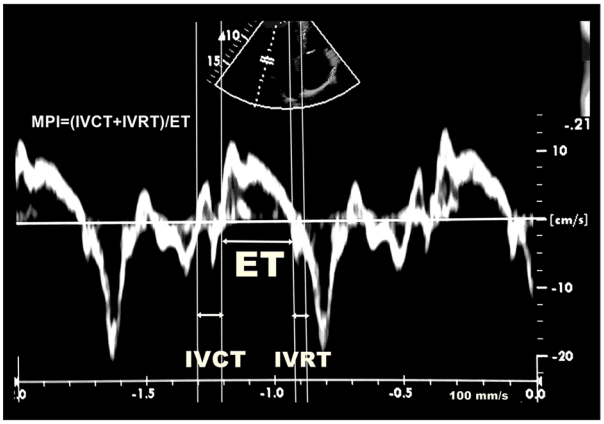

Figure 1. Calculation of tissue Doppler-derived left ventricular myocardial performance index. IVCT – isovolumic contraction time; ET – ejection time; IVRT – isovolumic relaxation time.

to the biplane method of disks. The end-diastolic interventricular septal thickness and end-diastolic left ventricle posterior wall thickness were evaluated at end of diastole. The left atrium was measured as the internal dimension of anteroposterior diameter in the parasternal long-axis view perpendicular to the aortic root long axis. Linear measurement of the right ventricle was performed as proximal right ventricle outflow diameter measured from the anterior right ventricle wall to the interventricular septal-aortic junction in parasternal long-axis view. The pulsed-wave Doppler evaluation of the mitral inflow velocity was acquired from apical 4-chamber views by placing the sample volume between the tips of the mitral leaflets. Peak velocities of early (E) and late (A) velocities were obtained from mitral inflow velocity curve, and the ratio of early to late peak velocities (E/A) was then calculated. Tissue Doppler recording was acquired from volume samples placed at the mitral annulus using an apical 4-chamber view. Isovolumetric contraction time (IVCT) was defined as the time from the end of the annular Am wave to the beginning of the annular Sm wave. Ejection time (ET) was defined as the time from the beginning to the termination of the Sm wave. Isovolumetric relaxation time (IVRT) was defined as the time from the end of the Sm wave to the beginning of the Em wave. Tissue Doppler-derived MPI was calculated according to the formula: MPI=(IVCT+IVRT)/ET [10,16] (Figure 1).

Statistical analysis

The data analyses were performed using the SPSS program (version 22.0, SPSS, Inc, Chicago, IL, USA). For comparative purposes, participating athletes were separated into 2 groups according to the cut-off value of normal MPI in previous articles, which is also the median value of MPI (MPI ≤0.40 and MPI >0.40) of this study population [10,17]. Comparisons were made between these 2 groups. All data were reported as mean ±SD or median (minimum-maximum) for continuous variables. Normality of continuous variables was assessed using the Shapiro-Wilk test. The homogeneity of variances was evaluated with Levene's test. The *t* test or Mann-Whitney U test was used to compare continuous variables. Univariate correlation and multiple linear regression analyses were used to determine the possible confounding factors for the MPI. Variables with a p-value of <0.15 in univariate analysis were tested in the multiple regression models. A 2-sided p-value <0.05 was considered as statistically significant.

Results

Eighty athletes (68 soccer and 12 basketball players) with a mean age of 24.9±4.3 years were included in the study. The MPI values of soccer players and basketball players did not differ significantly (p=0.115). Participating athletes were separated into 2 groups according to their MPI value (MPI ≤0.40 and MPI >0.40). Baseline demographic, clinical, and biochemical variables of the participants were compared between these 2 groups (Table 1**)**. Ages and heights of the participants were similar in both groups. White blood cell count, hemoglobin, hematocrit, platelet counts, and c- reactive protein were comparable between groups. Fasting blood glucose, sodium, calcium, and magnesium levels were similar in the 2 groups; however, potassium level was significantly higher in the MPI ≤0.40 group (4.2±0.4 mmol/L *vs.* 3.9±0.3 mmol/L; p=0.004). Serum urea, uric acid, and creatinine, and as well as iron parameters and homocysteine levels, were similar in the 2 groups. Total cholesterol, high-density lipoprotein, and triglyceride levels did not differ between the 2 groups, but the low-density lipoprotein level was significantly lower in the MPI ≤0.40 group (103.8±26.0 mg/dl *vs.* 116.8±30.2 mg/dl; p=0.043). Alanine aminotransferase, aspartate aminotransferase, gamma glutamyl transferase, alkaline phosphatase, and lactate dehydrogenase levels were similar in both groups. Thyroid-stimulating hormone, vitamin B12, total protein, and albumin levels did not significantly differ between the 2 groups.

The left and right chambers diameters, wall thicknesses, and LV ejection fraction were not different between the 2 groups. E and A velocities, deceleration time, and E/A ratios were comparable in both groups. When Em, Am, Sm, and E/Em were compared, only Sm was significantly higher in the MPI ≤0.40 group compared to the MPI >0.40 group (11.9±2.5 cm/s *vs.* 10.2±2.3 cm/s; p=0.002). IVCT and IVRT were shorter in the MPI ≤0.40 group, while ejection time was similar in both groups.

In correlation analysis, only potassium (r=–0.238; p=0.034), low-density lipoprotein (r=0.335; p=0.002), Sm (r=–0.310; p=0.005), IVCT (r=0.641; p<0.001), and IVRT (r=0.743; p<0.001) were significantly correlated with MPI (Table 2**)**.

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Table 1. Clinical, biochemical and echocardiographic parameters of subjects grouped according to myocardial performance index.

	Myocardial performance index ≤0.40 (n=40)	Myocardial performance index >0.40 (n=40)	All group (N=80)	p
Age, years	25.4±4.3	24.5±4.3	24.9±4.3	0.375
Height, cm	185.6±9.9	182.3±8.9	183.9±9.5	0.133
White blood cell count, $\times 10^3/\mu L$	5.73±1.41	5.57±1.14	5.65±1.28	0.588
Hemoglobin, g/dl	15.1±0.8	15.3±1.0	15.2±0.9	0.463
Hematocrit, %	44.4±2.2	44.9±2.7	44.6±2.5	0.384
Platelet, $\times 10^3/\mu L$	215.1±42.5	217.3±36.2	216.2±39.2	0.799
Fasting blood glucose, mg/dl	90.0±6.9	89.5±8.3	89.7±7.6	0.758
Sodium, mmol/L	138.4±2.2	138.2±1.9	138.3±2.1	0.668
Potassium, mmol/L	4.2±0.4	3.9±0.3	4.1±0.4	0.004
Chlorine, mmol/L	102.5 [95–108]	102 [99–106]	102 [95–108]	0.260
Calcium, mg/dL	8.89±0.38	8.86±0.38	8.87±0.38	0.747
Serum iron, µg/dL	87 [27–179]	82.5 [35–227]	84.5 [27–227]	0.736
Total iron binding capacity, µg/dL	310.6±41.2	314.45±45.4	312.5± 43.1	0.694
Transferrin saturation, %	27 [7–53]	28.5 [10–72]	27.5 [7–72]	0.776
Unsaturated iron binding capacity, µg/dL	219.6±53.2	223.8±52.3	221.7±52.4	0.727
Urea, mg/dL	33.6±8.2	34.8±6.9	34.2±7.6	0.500
Uric acid, mg/dL	5.29±0.99	5.39±1.14	5.34±1.06	0.686
Serum creatinine, mg/dL	0.91 [0.71–1.20]	0.89 [0.70–1.28]	0.90 [0.70–1.28]	0.787
Total cholesterol, mg/dL	168.0±26.2	177.2±38.6	172.6±33.1	0.216
HDL cholesterol, mg/dL	54 [41–91]	52.5 [36–106]	52.5 [36–106]	0.525
LDL cholesterol, mg/dL	103.8±26.0	116.8±30.2	110.3±28.8	0.043
Triglycerides, mg/dL	60 [23–156]	64 [26–196]	63 [23–196]	0.310
Alanine aminotransferase, IU/L	32.5 [20–99]	33 [18–79]	33 [18–99]	0.776
Aspartate aminotransferase, IU/L	28.5 [14–155]	28 [15–65]	28 [14–155]	0.787
Gamma glutamyl transferase, IU/L	25.5 [10–257]	26 [15–65]	26 [10–257]	0.996
Alkaline phosphatase, IU/L	66.5 [36–179]	63.5 [19–99]	65 [19–179]	0.266
Homocysteine, µmol/L	11.4 [6.2–66.9]	11.5 [6.80–15.6]	11.4 [6.2–66.9]	0.814
Magnesium, mg/dL	2.0 [1.7–2.2]	2.0 [1.7–2.3]	2.0 [1.7–2.3]	0.838
Lactate dehydrogenase, IU/L	168 [122–224]	170.5 [124–259]	170 [122–259]	0.795
C-reactive protein, mg/dL	0.10 [0.08–0.40]	0.10 [0.10–1.20]	0.10 [0.08–1.20]	0.268
Total protein, g/dL	7.4±0.5	7.5±0.5	7.5±0.5	0.513
Albumin, g/dL	4.4±0.3	4.4±0.4	4.4±0.3	0.804
Thyroid-stimulating hormone, µIU/mL	2.15±0.94	2.24±0.81	2.20±0.88	0.636
Vitamin B12, pg/mL	460.5 [245–2000]	443.5 [269–1256]	453 [245–2000]	0.931

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Table 1 continued. Clinical, biochemical and echocardiographic parameters of subjects grouped according to myocardial performance index.

	Myocardial performance index ≤0.40 (n=40)	Myocardial performance index >0.40 (n=40)	All group (N=80)	p
Transthoracic echocardiography				
Left atrium, mm	35.2±3.0	34.8±2.4	35.0±2.7	0.526
Left ventricle end-diastolic diameter, mm	50.3±3.7	49.2±3.5	49.8±3.6	0.191
Left ventricle end-systolic diameter, mm	33.4±3.8	32.9±2.9	33.2±3.3	0.452
Interventricular septum thickness, mm	11 [10–14]	11 [8–12]	11 [8–14]	0.299
Posterior wall thickness, mm	12 [10–14]	12 [8–13]	12 [8–14]	0.867
Right ventricle, mm	29.8±3.4	29.2±3.2	29.6±3.3	0.447
Ejection fraction, %	0.59±0.04	0.59±0.04	0.59±0.04	0.632
E, cm/s	75.5±14.7	72.4±9.7	74.0±12.5	0.374
A, cm/s	43.5±7.8	41.1±6.7	42.3±7.3	0.227
E deceleration time, ms	205 [140–266]	208 [142–248]	205 [140–266]	0.873
E/A ratio	1.8±0.4	1.8±0.4	1.8±0.4	0.673
Em, cm/s	17.9±3.8	16.6±3.2	17.2±3.5	0.115
Sm, cm/s	11.9±2.5	10.2±2.3	11.0±2.5	0.002
Am, cm/s	6 [3–12]	6 [2–11]	6 [2–12]	0.558
E/Em ratio	4.5±1.3	4.5±1.1	4.5±1.2	0.832
Isovolumic contraction time, ms	63 [47–104]	84 [48–129]	73 [47–129]	<0.001
Ejection time, ms	314.9±25.2	310.1±27.0	312.5±26.1	0.416
Isovolumic relaxation time, ms	44.7±9.6	62.9±13.9	53.8±15	<0.001
Myocardial performance index	0.36 [0.24–0.40]	0.46 [0.41–0.63]	0.40 [0.24–0.63]	<0.001

t test or Mann-Whitney U test.

In multiple linear regression analysis, including both biochemical and echocardiographic variables with a p<0.15 in correlation analysis with MPI other than a direct participant of MPI calculation (isovolumetric contraction time, ejection time and isovolumetric relaxation time), only low-density lipoprotein was significantly associated with MPI (β=0.263; p=0.019). When echocardiographic variables were excluded in the second model, low-density lipoprotein was still significantly associated with MPI (β=0.305; p=0.005) (Table 3).

Discussion

In this study, various biochemical markers, including iron, homocysteine, electrolytes, and lipids, were evaluated for possible association with left ventricle MPI as a surrogate of cardiac performance. We found that, among these biomarkers, only low-density lipoprotein was significantly associated with MPI in elite athletes.

Improvement of physical performance may be achieved with a balance between training load and recovery. Overtraining may lead to fatigue, in turn resulting in a decrease in athletic performance and even injuries. In sports sciences, several biomarkers have been proposed to determine the degree of physical fitness, the level of training/overtraining, and chronic or acute fatigue. Despite the absence of generally accepted standard screening tests, most trainers order tests of various biomarkers for evaluation to minimize the latent risks secondary to situations such as overtraining, nutrient deficiencies, inflammation, oxidative stress, and dehydration [18,19]. Additionally, the significance of these biochemical indicators

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Table 2. Univariate correlations between myocardial performance index and clinical variables in the entire study population.

	R correlation coefficient	p
Age, years	–0.088	0.442
Height, cm	–0.121	0.291
White blood cell count, $\times 10^3/\mu L$	–0.038	0.735
Hemoglobin, g/dl	0.033	0.771
Hematocrit, %	0.134	0.235
Platelet, $\times 10^3/\mu L$	0.040	0.726
Fasting blood glucose, mg/dl	–0.083	0.462
Sodium, mmol/L	–0.057	0.615
Potassium, mmol/L	–0.238	0.034
Chlorine, mmol/L	–0.087	0.445
Calcium, mg/dL	–0.114	0.317
Serum iron, µg/dL	–0.095	0.400
Total iron binding capacity, µg/dL	0.090	0.426
Transferrin saturation, %	–0.088	0.437
Unsaturated iron binding capacity, µg/dL	0.118	0.296
Urea, mg/dL	0.021	0.855
Uric acid, mg/dL	–0.012	0.913
Serum creatinine, mg/dL	–0.050	0.659
Total cholesterol, mg/dL	0.206	0.066
HDL cholesterol, mg/dL	–0.111	0.328
LDL cholesterol, mg/dL	0.335	0.002
Triglycerides, mg/dL	0.145	0.198
Alanine aminotransferase, IU/L	–0.004	0.972
Aspartate aminotransferase, IU/L	0.027	0.814
Gamma glutamyl transferase, IU/L	–0.113	0.318
Alkaline phosphatase, IUl	–0.185	0.100
Homocysteine, µmol/L	0.044	0.699
Magnesium, mg/dL	–0.042	0.710
Lactate dehydrogenase, IU/L	0.008	0.946
C-reactive protein, mg/dL	–0.079	0.484
Total protein, g/dL	0.123	0.279
Albumin, g/dL	–0.017	0.882
Thyroid-stimulating hormone, µIU/mL	0.078	0.492

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Table 2 continued. Univariate correlations between myocardial performance index and clinical variables in the entire study population.

	R correlation coefficient	p
Vitamin B12, pg/mL	0.032	0.776
Transthoracic echocardiography		
Left atrium, mm	–0.124	0.288
Left ventricle end-diastolic diameter, mm	–0.157	0.180
Left ventricle end-systolic diameter, mm	–0.084	0.472
Interventricular septum thickness, mm	–0.074	0.525
Posterior wall thickness, mm	–0.010	0.932
Right ventricle, mm	–0.129	0.273
Ejection fraction, %	–0.030	0.800
E, cm/s	–0.148	0.280
A, cm/s	–0.099	0.474
E deceleration time, ms	–0.046	0.737
E/A ratio	0.035	0.800
Em, cm/s	–0.190	0.093
Sm, cm/s	–0.310	0.005
Am, cm/s	0.132	0.246
E/Em ratio	0.100	0.505
Isovolumic contraction time, ms	0.641	<0.001
Ejection time, ms	–0.196	0.082
Isovolumic relaxation time, ms	0.743	<0.001

Pearson's or Spearman's correlation analysis

Table 3. Results of multiple linear regression analysis.

	β standardized regression coefficient	t-statistics	p
Model 1			
LDL cholesterol, mg/dl	0.263	2.409	0.019
Alkaline phosphatase, IUl	–0.165	–1.479	0.143
Potasium, mmol/L	–0.173	–1.592	0.116
Em, cm/s	–0.141	–1.119	0.267
Sm, cm/s	–0.129	–1.041	0.301
Model 2			
LDL cholesterol, mg/dl	0.305	2.862	0.005
Alkaline phosphatase, IUl	–0.115	–1.041	0.301
Potasium, mmol/L	–0.188	–1.738	0.086

LDL – low-density lipoprotein.

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in the assessment of athletes' cardiac fitness has not been investigated sufficiently. MPI is a non-invasive, easy-to-apply, and highly reproducible tool for detection of changes in both systolic and diastolic function. Additionally, previous studies also documented the relationship between maximal oxygen consumption, a well-known indicator of performance, and MPI [14]. In this context, using MPI as an indicator of the cardiac performance of athletes may be useful for follow up of seasonal evaluations and also for research purposes as a surrogate of cardiac fitness to investigate possible biomarkers to promote cardiac health status.

MPI was first defined by Tei et al. in the normal population and severe dilated cardiomyopathy patients, and they first proposed that a normal value for MPI was below 0.40 [10]. However, there is no consensus regarding normal values in athletes. Tüzün et al. investigated 66 elite athletes and found that the athletes had better MPI compared to sedentary populations [20]. In another study, Akova et al. found that MPI values of 32 elite male athletes in their study population were similar to the control sedentary population [21]. Furthermore, Alsafi et al. found similar results in female athletes [22]. In the present study, the median value of MPI was 0.40 and was used as a cut-off value for comparison. This value was also similar to the normal value originally proposed by Tei. Although comparison of MPI between sedentary and athletic populations was not a primary aim of this study, it is one of the largest studies investigating MPI in elite athletes, and may provide a reference value for MPI in athletes.

Beyond the hemoglobin formation and anemia, iron is an essential micronutrient that plays a central role in processes associated with athletic performance, such as oxygen transport, energy production, and cell division. Iron deficiency can negatively influence athletic performance, especially in women, who are more prone to iron deficiency [23]. Additionally, endurance athletes have a higher prevalence of hereditary hemochromatosis gene compared to sedentary persons; however, the possible advantage of this genetic alteration on the exercise physiology is unclear and warrants further investigations [24]. In the present study, serum iron parameters did not show a significant association with MPI, contrary to previous studies conducted in patients with iron overload [25,26]. Nevertheless, it should be kept in mind that the present study population consisted of healthy young athletes with higher physiologic levels of iron compared to the aforementioned patients with deleterious effects of iron overload. The present results suggest that the favorable effects of iron supplementation on athletic performance may not be directly related to MPI improvement.

Homocysteine is a sulfur-containing intermediate amino acid, and high homocysteine levels are associated with increased cardiovascular and neurodegenerative disorders; nevertheless, it is not clear whether homocysteine is a risk factor or a risk marker [27]. Both acute and long-term exercise may have modulating effects on homocysteine concentrations. Acute strenuous exercise may accelerate protein catabolism in the muscle amino acid pool, which may also increase homocysteine formation; however, the effect of chronic exercise is more variable and recent studies are inconclusive [28,29]. In previous studies, cardiorespiratory fitness was inversely associated with homocysteine concentration in women [30], but there was no such association in adult males, and this difference was attributed to differences in homocysteine methylation rate and estrogen levels [31]. On the other hand, to date, the relationship between homocysteine levels and MPI in chronically and actively training males is not clear. The result of the present study showed no association between homocysteine concentrations and MPI, which is in accordance with the study conducted by Dankner et al. [31].

Low-density lipoprotein is a well-known cardiovascular risk factors, and it is also known that sedentary lifestyle is closely associated with poor low-density lipoprotein levels. Although physical performance evaluated with oxygen uptake were inversely correlated with low-density lipoprotein levels of professional athletes [32] and non-athlete subjects [33], the relationship between myocardial performance and lipid parameters was not adequately studied before. The present study demonstrates a significant association between myocardial performance index and low-density lipoprotein levels in elite athletes. This result is also compatible with a previous study showing early impairment of left ventricular function in hypercholesterolemia, even in the absence of coronary artery disease, suggesting the importance of hypercholesterolemic cardiomyopathy [34]. Fat accumulation in the myocardium, also known as cardiac steatosis, leads to lipotoxic effects on cardiomyocytes caused by the production of oxygen radicals, and triggering apoptosis may be a possible mechanism related to low-density lipoprotein levels and cardiac dysfunction [35]. On the other hand, this possibility seems less likely in this study since the participants did not have an overt metabolic derangement such as metabolic syndrome, diabetes mellitus, or very high lipid levels predisposing to cardiac steatosis. Also, this speculation was not tested with tissue sampling from myocytes or cardiac magnetic resonance imaging to quantify the fat content of myocardium, which was beyond the scope of this study. However, in this study, participants were healthy athletes with lower levels of low-density lipoprotein cholesterol, emphasizing the potential use of low-density lipoprotein levels as a biomarker and an indicator of myocardial performance and, hence, cardiac fitness in athletes rather than a hypercholesterolemic cardiomyopathy. Additionally, elucidation of the mechanisms underlying this association requires further investigations.

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Study limitations

This study evaluated the spot values of biochemical markers and echocardiographic parameters. It should be kept in mind that these variables may show seasonal or even diurnal variations. Cardiac performance was not tested simultaneously with other fitness tests such as maximal oxygen consumption, and the association between myocardial performance index and cardiac fitness was attributed to previous studies.

Conclusions

This study showed that, among a number of biochemical markers, including serum lipids, homocysteine, and iron parameters, only low-density lipoprotein cholesterol levels were a significant predictor of MPI in elite athletes. In addition to being a major risk factor for atherosclerosis, low-density lipoprotein cholesterol may have clinical significance as an indicator of cardiac fitness and performance in elite athletes or otherwise healthy actively exercising or training subjects.

Conflict of interests

None.

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