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The relationships between BNP levels with cardiac structure and function in resistance-trained athletes

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Abstract

Introduction: Echocardiography and the B-type natriuretic peptides (BNPs) provide powerful incremental assessment of cardiac function, clinical status, and outcome across the spectrum of cardiac disease. Although the previous studies demonstrated the relationships between BNP levels and cardiac structure and function in heart failure patients, but these relationships in athletes are not well known. The present study was conducted to examine the relationships between BNP levels with cardiac structure and function in resistance-trained athletes.

Material & Methods: Fifteen resistance-trained male athletes (aged: 23.0 ± 1.4 years and BMI: 24.1 ± 1.4 kg/m^2; ± SD) volunteered to participate in this study. BNP concentrations were assessed by enzyme-linked immunosorbent assay (ELISA) kits and cardiac morphology and function were assessed by echocardiography. Spearman correlation test was used to analyze the relationship between the variables.

Results: The results demonstrated that there were no significant relationships between BNP concentrations with posterior wall thickness of left ventricle at end diastole (PWTLV) (r = – 0.35, P = 0.1), interventricular septal (r = – 0.25, P = 0.3), aorta (r = 0.07, P = 0.8) and pulmonary artery (r = – 0.06, P = 0.8) diameter, diastolic left ventricle internal dimension (DLVID) (r = – 0.33, P = 0.2), systolic left ventricle internal dimension (SLVID) (r = 0.2, P = 0.4), left ventricle ejection fraction (LVEF) (r = 0.21, P = 0.4), left ventricle end-diastolic volume (LVEDV) (r = – 0.23, P = 0.4) and left ventricle end-systolic volume (LVESV) (r = – 0.23, P = 0.4).

Conclusions: In conclusion, BNP concentration is not a powerful predictor for cardiac structure and function in resistance-trained athletes.

Keywords: Cardiac structure, Cardiac function, B-type natriuretic peptide, Resistance-trained athletes

1. Introduction

The B-type natriuretic peptides (BNPs) are sensitive markers of ventricular dysfunction and are playing an increasingly important role in the diagnosis, management and prognosis of cardiac diseases (1-3). BNP is not stored but is synthesized and secreted constitutively in response to cardiomyocyte stretch (4). Increased ventricular or atrial wall stress, reflecting volume or pressure overload, is the primary driver of myocyte stretch mediated secretion, but ischemia, neurohormones, and cytokines also stimulate or modify BNP gene expression (4). Plasma levels of BNP and its split product N-terminal pro BNP (NT-proBNP) increase with age and are lower in men than in women (5). Levels are inversely related to body mass index and lean mass (6) and increase with worsening glomerular filtration rate (7).

Peptide measurements provide information complementary or incremental to echocardiography for assessment of cardiac function, clinical status, and outcome (8). Previous studies demonstrated that

BNP and NT-proBNP levels correlate positively with left ventricle (LV) dimensions, volumes, and mass in a variety of settings and populations and are inversely related to LV ejection fraction (LVEF) (9-12). Peptide levels are higher with left ventricular hypertrophy (LVH) (13) and are higher still in subjects with LVH and clinical heart failure (14).

The strongest correlations have been reported for BNP with LV diastolic wall stress consistent with stretch-mediated BNP secretion (15). BNP levels increase with greater severity of overall diastolic dysfunction, independent of LVEF, age, sex, body mass index, and renal function, and the highest levels are seen in subjects with restrictive filling patterns (5,16). Peptide levels correlate with indexes of filling pressure – including transmitral early filling velocity and its ratio to early diastolic annular velocity – as well as with indexes of compliance and myocardial relaxation (17). In subjects with normal LVEF, elevated NTproBNP or BNP are the strongest independent predictors of severe diastolic dysfunction; low peptide levels exhibit very high negative predictive value (>90%) for diastolic dysfunction (16). BNP and NT-proBNP levels also reflect left atrial size, correlating positively with left atrial volume, particularly in the general population and in patients with heart failure with preserved systolic function (18,19).

Exercise, has been shown to increase BNP secretion (20); in subjects with myocardial ischemia this elevation is more pronounced and has been found to improve the diagnostic sensitivity of the exercise-stress ECG in a selected population with normal left ventricular ejection fractions. Research results indicated that BNP levels increased after 8 weeks resistance training due to myocytes injury (21). Although the relationship between the BNP and echocardiographic measures of cardiac structure and function has been widely explored in healthy people or cardiovascular patients, there is no study was performed to examine the relationship between BNP concentrations with cardiac structure and function in athletes population. The aim of present study was to examine the relationships between BNP levels with cardiac structure and function in resistance-trained athletes.

2. Material & Methods

Subjects

The study population comprised of fifteen resistance-trained male athletes with a mean (±SD) age of 23.0 ± 1.4 years and weight of 78.3 ± 8.1 kilogram. Participants were strength trained at least 3 times per week for more than 3 years. The Islamic Azad University, Marvdasht branch Ethics Committee approved the study and written informed consent was obtained from all subjects.

Measurements

Anthropometric and body composition measurements

Height and body weight were measured, and body mass index (BMI) was calculated by dividing body mass (kg) by height (m^2). Waist circumference was determined by obtaining the minimum circumference (narrowest part of the torso, above the umbilicus) and the maximum hip circumference while standing with their heels together. The waist to hip ratio (WHR) was calculated by dividing waist (cm) by hip circumference (cm). Body fat percentage was assessed by skinfold thickness protocol. Skinfold thickness was measured sequentially, in chest, abdominal, and thigh by the same investigator using a skinfold caliper (Harpenden, HSK-BI, British Indicators, West Sussex, UK) and a standard technique.

Biochemical measurement

Blood for measurement of BNP concentrations was collected by venipuncture in Vacuette polyethylene terephthalate glycol EDTA tubes (Greiner Bio-One) on the day of the echocardiographic evaluation. Blood samples were centrifuged at 3500g for 10 min at 4°C immediately after collection. Plasma BNP was assayed via an enzyme-linked immunosorbent assay (ELISA) kits (Biomedica Immu noassay China, Inc). The sensitivity of the kits were <64 pg/ml.

Echocardiography

M-Mode and 2-dimensional images and spectral and color-flow Doppler recordings were obtained with a single commercially available instrument operating at 2.0 – 3.5 MHz. Two-dimensional imaging examinations were

performed in the standard fashion in parasternal long and short-axis views and apical 4- and 2-chamber views. All measurements were performed as recommended (22).

Two-dimensional echocardiograms were subjected to careful visual analyses to detect regional contractile abnormalities. Left ventricular end-systolic and end-diastolic volumes (LVEDV and LVESV) and ejection fraction (LVEF) were derived from biplane apical (2- and 4-chamber) views with the modified Simpson's rule algorithm. Left ventricular dimensions were measured from M-mode images by the leading-edge technique, which included interventricular septal thickness at end diastole, posterior wall thickness of left ventricle at end diastole (PWTLV), and left ventricular internal dimension at end diastole and systole (DLVID and SLVID).

Statistical analysis:

Data were analyzed using SPSS software for windows (version 17, SPSS, Inc., Chicago, IL). Shapiro-wilk t-test was used for normality. Spearman correlation test was used to evaluate the relationship between the variables. The significance level of this study was set at $P < 0.05$.

3. Results

Personal characteristics of the subjects are presented in the Table 1. As shown in the Table 1, the subjects have the normal weight, WHR and body fat percentage.

Table 1. Anthropometric, body composition and physiological characteristics of the subjects

Variables	Mean	SD
Age (y)	23.0	1.4
Body weight (Kg)	78.3	8.1
BMI (Kg.m^{-})	24.1	1.4
WHR	0.87	0.04
Body fat (%)	12.0	3.3
Fat mass (kg)	9.5	3.3
Lean body mass (kg)	68.3	5.4

BNP level and cardiac structure and function parameters of the subjects are presented in the Table 2 (See the abbreviations below the table).

Table 2. BNP level and cardiac structure and function parameters of the subjects

Variables	Mean	SD
BNP (pg/ml)	27.0	26.8
PWTLV (mm)	10.6	1.1
Interventricular septal (mm)	11.0	1.1
Aorta diameter (mm)	29.6	2.4
Pulmonary artery diameter (mm)	26.7	2.4
DLVID (mm)	48.9	2.0
SLVID (mm)	29.6	2.8
LVEF (%)	53.4	8.3
LVEDV (ml)	95.5	15.5
LVESV (ml)	42.9	9.5

BNP: B-type natriuretic peptides; PWTLV: Posterior wall thickness of left ventricle at end diastole; DLVID: diastolic left ventricle internal dimension; SLVID: systolic left ventricle internal dimension; LVEF: left ventricle ejection fraction; LVEDV: left ventricle end-diastolic volume and LVESV: left ventricle end-systolic volume.

The Shapiro-wilk test demonstrated that the data had not distribute normal, thus Spearman test was used to evaluate the relationship between BNP levels with cardiac structure and function. The correlation between the variables is presented in the Table 3. The results, demonstrated that there were no significant relationship between BNP levels and cardiac structure including: PWTLV, Interventricular septal, Aorta diameter, Pulmonary artery diameter, DLVID, and SLVID. As shown in the Table 3, no significant relationship was observed between BNP levels and cardiac function including: LVEF, LVEDV and LVESV.

Table 3. Relationships between BNP and cardiac parameters of the subjects

Variables	BNP (pg/ml)	
	r	P
Cardiac structure variables		
PWTLV	– 0.35	0.1
Interventricular septal	– 0.25	0.3
Aorta diameter	0.07	0.8
Pulmonary artery diameter	– 0.06	0.8
DLVID	– 0.33	0.2

Variables	BNP (pg/ml)	
	r	P
SLVID	0.2	0.4
Cardiac function variables		
LVEF	0.21	0.4
LVEDV	– 0.23	0.4
LVESV	– 0.23	0.4

BNP: B-type natriuretic peptides; PWTLV: Posterior wall thickness of left ventricle at end diastole; DLVID: diastolic left ventricle internal dimension; SLVID: systolic left ventricle internal dimension; LVEF: left ventricle ejection fraction; LVEDV: left ventricle end-diastolic volume and LVESV: left ventricle end-systolic volume.

4. Discussion

Although the relationship between the BNP and echocardiographic measures of cardiac structure and function has been widely explored in healthy people or cardiovascular patients, there is no study was performed to examine the relationship between BNP concentrations with cardiac structure and function in athletes population. The aim of present study was to examine the relationships between BNP levels with cardiac structure including PWTLV, Interventricular septal, Aorta diameter, Pulmonary artery diameter, DLVID, and SLVID and the relationships between BNP levels with cardiac function including LVEF, LVEDV and LVESV in resistance-trained male athletes.

The results showed that the levels of BNP in resistance-trained male athletes (27.0 pg/ml) were in normal range (<100pg/ml). The effect of resistance training on BNP is contradictory. Although Bordbar et al. (2012) noted that NT-proBNP increased after 12 weeks resistance training (21), Beltran Valls et al. (2014) reported that they failed to find such an effect after 8 weeks moderate-resistance training in elderly (23). The results of the present study suggested that BNP level had not significant changes after several years of resistance training compare to the healthy subjects.

The results showed that PWTLV, interventricular septal and DLVID in resistance-trained male athletes are greater than the normal range (10.6 mm *vs.,* 9.3 mm, 11.0 mm *vs.,* 9.2 mm and 48.9 mm *vs.,* 46.2 mm respectively). Kou et al. (2014) reported the reference ranges of PWTLV

(9.3 mm for males and 8.5 mm for females), interventricular septal (9.2 mm for males and 8.2 mm for females) and DLVID (46.2 mm for males and 43.0 mm for females) for healthy males and females (24). Previous studies in line with the present study results indicated that resistance-trained individuals have both the PWTLV and interventricular septal greater than average (25-27). Haykowsky et al. (2000) noted three mechanisms for resistance training-induced cardiac hypertrophy: (1) acute cardiopulmonary mechanisms that minimize the increase in transmural pressure and LV wall stress during exercise, (2) the underlying use of anabolic steroids by the athletes, or (3) the specific type of resistance training performed (26).

The results showed that LVEF and LVEDV in resistance-trained male athletes are lower than the normal range (53.4% *vs.,* 63.3% and 95.5 ml *vs.,* 107.1 ml respectively); however, LVESV in resistance-trained male athletes is greater than the normal range (42.9 ml *vs.,* 39.7 ml). The reference ranges of LVEF (63.3% for males and 64.1% for females), LVEDV (107.1 ml for males and 83.8 ml for females) and LVESV (39.7 ml for males and 30.2 ml for females) reported by Kou et al. (2014) previously (24). LV systolic function is generally assessed in echocardiographic studies by measuring the extent of fiber shortening, ejection fraction and velocity of circumferential fiber shortening (28), while diastolic function is assessed by studying the pattern of ventricular filling through the mitral valve (29). Abnormalities in systolic and diastolic function are generally associated with cardiac hypertrophy induced by pathological conditions, such as hypertension and valvular disease (30,31). The present study results are in agreement with those in the literature where reports demonstrate that cardiac function is not altered in resistance-trained individuals (27,32,33)

At the end the results, demonstrated that there were no significant relationship between BNP levels and cardiac structure including: PWTLV, Interventricular septal, Aorta diameter, Pulmonary artery diameter, DLVID, and SLVID and no significant relationship was observed between BNP levels and cardiac function including: LVEF, LVEDV and LVESV in resistance-trained male athletes. Previous studies demonstrated that there is a positive relationship between BNP levels with LV hypertrophy and left ventricular dysfunction (including: low

LVEF and LVEDV and high LVESV) (8,16). These results discrepancy might due to study population. The subjects of the present study were the resistance-trained athletes while the study population of those studies was the heart disease patients. Several years of resistance training induced physiological adaptation in cardiac muscle including LV hypertrophy and these exercise powered the cardiac muscle lead to increase of cardiac function. Thus the adaptation in cardiac muscle and LV hypertrophy following several years of resistance training are different with the changes from the heart disease induced cardiac muscle and LV hypertrophy.

5. Conclusion

The results indicated that BNP levels are in normal range in resistance-trained male athletes, thus BNP concentration is not a powerful predictor for cardiac structure and function in these population.

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