Effect of resistance exercise training on biochemical markers and anthropometric characteristics involved in atherosclerosis in obese women

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Abstract

Introduction: Obese individuals have elevated levels of inflammatory and cell adhesion molecules that can critically induce the occurrence of atherosclerosis. Aerobic exercise training reduces biochemical markers and anthropometric characteristics involved in atherosclerosis. However, little is known about the effect of resistance exercise training on these biomarkers. This study aimed to investigate the effect of eight weeks of resistance training on atherosclerosis biochemical markers and anthropometric characteristics in obese women.

Materials and methods: Fourteen obese women completed an 8-week resistance exercise training protocol with moderate intensity at 9 stations (exercise involving the major muscle groups: 3 sets of 8-15 repetitions of chest press, leg press, seated pulley rows, overhead press, seated leg press, leg curl, triceps extensions, biceps curls, and calf raises). Fasting blood samples were taken before and after the 8-week exercise training. Intercellular Adhesion Molecule1 (ICAM-1) and C-reactive protein (CRP) levels were measured using commercial kits by ELISA method. The data were analyzed using dependent t-test.

Results: Resistance training significantly reduced the levels of atherosclerosis biochemical markers, ICAM-1and CRP (P=0.001). Also, body fat percentage (P=0.001), waist: hip ratio, body mass and body mass index significantly decreased following resistance training (P=0.001). However, no significant change occurred in platelet counts (P=0.922).

Conclusion: Resistance training results in reduction of inflammatory biomarkers involved in atherosclerosis as well as body fat.

Keywords: Resistance exercise training, Intercellular adhesion molecule 1, Obese

Introduction

Although increased low-density lipoprotein (LDL-c) and decreased high-density lipoprotein (HDL-c) are considered to determine the risks of cardiovascular disease, reports show people who suffer from cardiovascular disease despite normal levels of LDL-c and HDL-C (1-3). Meanwhile, a lot of research suggests that cardiovascular disease has inflammatory background and that systemic inflammation plays a pivotal role in the development of atherosclerosis (4-6). Accordingly, in the past decade, more attention has been focused on inflammatory markers as independent factors that predict incidence of cardiovascular disease.

Intercellular adhesion molecule-1 (ICAM-1) and C-reactive protein (CRP) act as important inflammatory markers, which are associated with the pathogenesis of atherosclerosis (4, 7-8).

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Cell adhesion molecules exists in membrane-bound and soluble forms. Membrane intercellular adhesion molecule-1 (mICAM-1) with 90 kDa expresses constitutively on endothelial and non-endothelial cells and is up-regulated by adrenergic stimulation (4,10,11) oxidative products (8,9), shear stress (10-12), and inflammatory cytokines such as CRP (4,11,12). Circulating soluble ICAM-1 (sICAM-1) is thought to originate from proteolytic of membrane-bound ICAM-1 and has a lower molecular weight than mICAM-1 (13). mICAM-1 mediates the adherence and subsequent infiltration of circulating leukocytes across the vascular endothelium (11,13). sICAM-1 represents its expression on endothelial cells (13) and therefore provides an easy-to-measure clinical marker of vascular inflammation and endothelial activation (11). Holding 224 amino acids, CRP is synthesized and released by hepatocytes in response to inflammation (4, 14). Increased levels of CRP following inflammatory conditions result in expression of ICAM-1 on endothelial cells (1). Similarly, increased production of free radicals following adherence of immune cells to ICAM-1 results in proliferation of smooth muscle cells and re-expression of ICAM-1 on endothelial cells (4, 8, 15). This results in greater adherence of plaque and LDL particles to endothelial cells that finally leads to the development of a mature atherosclerotic plaque (4, 15, 16).

Researchers have used different nutritional and exercise training approaches to reduce the activation of endothelial cells and subsequently inhibit the expression of adhesion molecules (6-9, 17-19). In this regard, vitamin E supplementation has been suggested to decrease monocyte chemotactic activity (20) and surface expression of ICAM-1 induced by high-fat diet as it reduces the inflammatory marker (16). Also, it has been shown that daily treadmill walking (70 to 85% of maximum heart rate) (6) and cycle ergometer training (80% of maximum heart rate) (7) with modified nutritional diet (high fiber, low fat) reduce ICAM-1 and CRP levels in diabetes mellitus type 2 and coronary artery disease, respectively (6,7). Furthermore, both short- (6, 15) and long-term (21) aerobic exercise training with moderate intensity alters circulating ICAM-1 and CRP in sedentary obese and overweight adults with metabolic syndrome. Although 12-week home-based bicycle exercise training reduces serum ICAM-1 in patients with chronic heart failure, serum ICAM-1 returns to baseline upon detraining (8).

Aerobic exercise training reduces inflammatory markers involved in atherosclerosis in obese and overweight individuals in both pathological (16) and physiological conditions (14, 21). Except for one study (14), the effect of resistance exercise training (exercises that increase energy expenditure, basal metabolic rate, and body's muscle mass) on atherosclerosis biochemical markers has not been well studied. The annual rate of overweight and obesity have recently grown and can be seen in both sexes and all races (4). In a national study of diseases in Iran, cardiovascular disease was ranked third, and atherosclerosis had a higher prevalence in obese and overweight subjects (22). Furthermore, obese individuals have elevated levels of ICAM-1 than lean individuals (23-26). The present study investigated the effects of 8 weeks of resistance training on the risk factors involved in atherosclerosis in obese women.

Materials and methods

Participants: The present quasi-experimental study was approved by the Human Subjects Protection Committee of the University of Birjand (Iran). Twenty sedentary and obese women with regular menstrual periods were chosen after they completed health and Physical Activity Readiness Questionnaire (PAR-Q). Also, physical fitness level was evaluated by Baecke's physical activity questionnaire.

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Given the proposition by Bielinski and colleague (2008) that ICAM-1 concentration in serum/plasma is heritable, our exclusion criteria included subjects with any infection disease, cardiovascular disease, atherosclerosis, hyperlipidemia, hypertension, diabetes, cancer, and family background of these diseases (13). Also, we excluded any subjects who smoked or used cholesterol-lowering medications and non-steroidal anti-inflammatory drugs. All of them signed a written informed consent knowing the potential benefits and associated risks. Six subjects were excluded due to lack of regular participation. Maximal oxygen consumption (VO_2max) of subjects was determined by Storer maximal bicycle test before the exercise training protocol. Physical characteristics of the subjects are listed in Table 1.

Table 1. Subject characteristics.

Age (year)	36 ± 6
Body mass (kg)	77 ± 10
Hight (cm)	159 ± 7
BMI (kg/m^2)	30.47 ± 4.28
VO_2max, $ml.kg^{-1}.min^{-1}$	18.23 ± 3
BF %	35.93 ± 3.36

Values are presented in means ± standard deviation. BMI, Body mass index; VO_2max, maximal oxygen consumption; BF %, body fat percentage.

Anthropometric measures: Subjects' height and body mass were measured by digital Stadiometer Seca. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Waist-hip ratio (WHR) was calculated as waist measurement (at the smallest circumference of the waist, just above the belly button) divided by hip measurement (the widest part of the buttocks). Skinfold thickness was obtained with skinfold caliper (Yagami model, Japan) on the right side of the subject's body. A 3-site skin fold equation of Jackson and Pollock (triceps, thigh, and suprailiac) was used to estimate body density, and body fat percentage (BF %) was subsequently calculated using the Siri equation.

Determination of one-repetition maximum: One-repetition maximum (1RM) was determined before, at the fourth week of training, and 3 days after the last resistance exercise training (27). All strength tests throughout the study were conducted using the same equipment with identical positioning of the participant and controlled by the same investigator (28). Strength tests were always preceded by 7 min warm-up on a cycle ergometer. After warm-up, the subjects rested for 3 min. Weight was chosen according to the participant so that the participants lifted the weights at least once and up to 10 times. 1RM of agonist and antagonist muscles were measured on two separate days (27). Finally, 1RM was determined by Brzycki equation as follows: 1RM=Weight ÷ [1.0278 - (0.0278 × Number of repetitions)]

Resistance exercise training: Participants were appropriately trained under physician supervision. They performed resistance exercise training with moderate intensity for 8 weeks (1 session per day, 4 days per week, at 50 to 65% of 1-RM). Resistance exercise training was conducted at 9 stations (exercise involving the major muscle groups: chest press, leg press, seated pulley rows, overhead press, seated leg press, leg curl, triceps extensions, biceps curls, and calf raises). Each exercise was performed for 3 sets with 8 to 11 repetitions in the first 4 weeks and increased to 15 repetitions during the last 4 weeks. Exercise in each station lasted for 30 seconds and the rest between two stations was 120 seconds. One exercise session consisted of 15 to 20 min warm-up, 30 min resistance exercise training, and 10 min cool-down.

Blood sampling and biochemistry assay: Twelve-hour fasting blood samples were obtained from the antecubital vein before and 48 hours after the last resistance exercise training (14). To avoid facing the hormonal changes of the menstrual cycle,

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sampling time coincided with the luteal phase because estrogen acts as a reducing agent of serum ICAM-1 (26). The samples were centrifuged (Eppendorf Centrifuge, Mini SpinR, Germany) for 10 min at 3000 ×g at 4 °C. Serums were collected and stored immediately at −80 °C. ICAM-1 (Gen-Probe Diaclone SAS, France), and CRP (Mississauga, Ontario, Canada) levels in the serums were measured using the commercial ELISA kit according to the manufacturer's instructions. The sensitivity of the ICAM-1 and CRP kits were less than 8.68 ng/ml and 10 ng/ml, respectively.

Statistical analysis

Statistical analyses were performed with Statistical Package for the Social Sciences (SPSS) version 16. Normality of data was confirmed by Shapiro–Wilk test. Data were analyzed using the paired t-test. All data are expressed as means ± standard deviation. P values <0.05 were considered statistically significant.

Results

Mean and standard deviation of the dependent variables in the pre- and post-resistance exercise training are displayed in Table 2. In the context of atherosclerosis biochemical markers, resistance exercise training significantly reduced serum levels of ICAM-1 in post- compared to pre-resistance exercise training (P=0.001). The serum levels of CRP reduced significantly following resistance exercise training (P=0.001). In terms of anthropometric characteristics, resistance exercise training significantly reduced body mass (P=0.001), BMI (P=0.001), BF% (P=0.001) and WHR (P=0.010). In contrast, resistance exercise training did not have a significantly effect on platelet counts (P=0.922).

Table 2. Values of dependent variables in pre- and post-resistance exercise training.

Dependent variables	Time		t value	P value
	Pre	Post		
Serum ICAM1 (ng/ml)	661.90±163.19	512.42±130.92	10.5*	0.001
Serum CRP (ng/ml)	2871.64±2413	2443.91±2023.14	9.74*	0.001
Platelet counts (count/mm^3)	230360±65505	229000±38454	0.099	0.922
Body mass (kg)	76.68±10.45	73.92±10.26	7.89*	0.001
BMI (kg/m2)	30.47±4.28	29.40±4.16	7.23*	0.001
BF (%)	35.93±3.36	34.41±3.58	5.08*	0.001
WHR	0.78±0.03	0.77±0.03	3.01*	0.010

Values are presented in means ± standard deviation. The asterisk (*) indicates a significant difference. ICAM-1, intercellular adhesion molecule-1; CRP, C-reactive protein; BMI, Body mass index; BF %, body fat percentage; WHR, waist: hip ratio.

Discussion

In recent years, there has been a growing number of prospective epidemiologic studies to demonstrate that ICAM-1 and CRP are independent predictors of myocardial infarction, stroke, atherosclerosis, and sudden cardiac death, even in apparently healthy individuals (4,5,8). In the present study, we showed that reduced levels of ICAM-1 and CRP in obese women correspond with decreased levels of body fat.

There is now a substantial body of evidence suggesting that aerobic exercise training is a significant factor against incidence of atherosclerosis through reducing inflammatory markers (6-9). Here, we found reductions in ICAM-1 and CRP following resistance exercise training. The results of this study are consistent with studies that report a reduction in ICAM-1 and CRP levels, weight and BMI following both ± exercise training and dietary modification (6,7). By

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reducing fat intake, dietary modification is believed to affect adipose tissue that subsequently reduces ICAM-1 expression (21). Especially, decreased visceral adipose tissue in obese women results in greater reduction in inflammatory cytokines and ICAM-1 expression (21). In this context, a positive correlation has been showed between ICAMI, BMI and BF (%) in type 2 diabetic patients (5). Also, this finding is consistent with the study of Puglisi and colleague (2008) which attributed a reduction in plasma ICAM-1 following 6-week walking to reduced inflammatory cytokines in overweight and postmenopausal women (9). As we found a decline in ICAM-1 in obese women, a reduction in ICAM-1 is reported in obese men after daily treadmill walking (6). Serum ICAM-1 level is not different between men and women (23). In contrast, 8 to 14 weeks of rowing on a home rowing ergometer (30) and stationary cycle training (12) did not have any significant effect on concentration of ICAM-1 and CRP. This lack of change can be attributable to the inability of the mentioned exercise training in reducing body fat. Although it seems that artery remodeling induced by exercise training results in less shear stress and ICAM-1 expression (15), it seems that the major determinant of changes in ICAM-1 and CRP levels is body fat, particularly visceral fat. By producing inflammatory cytokines of tumor necrosis factor (TNF-α) and interleukin-1 (IL-1), adipose tissue increases expression of ICAM-1 on endothelial cells (6, 7, 15, 21). Inflammatory cytokines enhance activation of NF-kappaB that regulates transcription of ICAM-1 (4). Moreover, NF-kappaB promotes the attachment of monocytes and macrophages to vessel walls by synthesizing and releasing pro-inflammatory cytokines (4). Importantly, TNF-α and IL-1 stimulate synthesis of CRP in hepatocyte, while CRP increases expression of ICAM-1 (8, 23).

By increasing anti-inflammatory cytokine, exercise training has been showed to (9,14) reduce the expression of ICAM-1 and CRP induced by inflammatory cytokines of IL-1 and TNF-α that release from adipose tissue. Furthermore, exercise training improves antioxidant capacity, subsequently reducing free radical production and LDL oxidation. Decreased expression of ICAM-1 is also associated with reduced oxidation of LDL (9). Finally, it seems that part of the observed decline in levels of ICAM-1 in the present study is due to reduced shear stress induced by angiogenesis and atherogenesis after exercise training (12, 15). Shear stress results in cleavage of mICAM-1 from endothelial cells and its release into blood circulation (12).

In contrast, resistance exercise training has no effect on the number of platelets. Immune cells transfer to adjacent tissue by binding to adhesion molecules, subsequently resulting in to increased free radicals' production and LDL oxidation. Oxidation of LDL ultimately increases expression of ICAM-1 in endothelial cells. Platelets stick to ICAM-1, whereby vascular narrowing and occlusion may occur (4). In one study, reduced expression of atherosclerosis biochemical markers was attributed to decreased adhesion of monocytes to cultured endothelial cells in overweight and obese subjects who conducted both exercise training and dietary modification (6). Effect of exercise training on endothelial function is mediated by increased levels of HDL. In turn, HDL stimulates prostaglandin from vessel wall or smooth muscle cells and thereby inhibits platelet aggregation, leading finally to reduce expression of ICAM-1 (31).

Due to study limitations, the activity of immune cells and platelet adhesion was not measured in the present study and is recommended to be considered in future researches.

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Conclusion

Resistance training results in reduction of inflammatory biomarkers involved in atherosclerosis by decreasing body fat.

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