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The Effects of Moderate Swimming Training on Blood Pressure Risk Factors in Hypertensive Postmenopausal Women

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Abstract

The effects of physical activities such as jogging, running and bicycling on arteriosclerosis risk factors are well documented. However, the effect of moderate swimming exercise on arteriosclerosis risk factors and particularly oxidized low-density lipoprotein (OX-LDL) has received little attention. It was hypothesized that moderate swimming exercise reduces OX-LDL level and others arteriosclerosis risk factors in hypertensive postmenopausal women**.** Thirty-four hypertensive postmenopausal women were randomly divided into control and training groups. Swimming training was conducted four times per week for 30 min at 60-70% of maximum heart rate for eight weeks. Moderate swimming training resulted in a reduction in systolic blood pressure (p<0.05) and oxidized low-density lipoprotein (p<0.05). No changes were observed in vascular cell adhesion molecule (VCAM-1), diastolic blood pressure low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) . A positive correlation was also found between OX-LDL and systolic blood pressure (R =0.34, p<0.05) and LDL-C (r = 0.5, p<0.05). In conclusion, moderate swimming training may reduce systolic blood pressure and OX-LDL in hypertensive postmenopausal women.

Keywords: Swimming exercise, hypertension risk factors, OX-LDL

Introduction

High blood pressure in hypertensive patients is related to coronary arterial diseases and arteriosclerosis risk factors [1]. It has been reported that a quarter of hypertensive old people experience high blood pressure (>140/90 mm of mercury) [2]. The disease worsens as age increases. High blood pressure is more prevalent among postmenopausal women due to changes in female hormone levels such as estrogens and serum lipids. Estrogens increase production of nitric oxide (NO) and prostaglandin (PG), which, in turn, stimulates the blood vessels and decreases blood pressure, oxidized low-density lipoprotein (OX-LDL) and atherosclerosis risk factors (3). The enhanced arterial intimae–media thickness as result of increasing in vascular cell adhesion molecules (VCAM-1) and oxidized low-density lipoprotein has been reported as primary factor contribute to high blood pressure [4]. The presence of VCAM-1 around atherosclerotic platelets plays an important role in endothelial leucocytes movements across endothelial tissue.

Changes in oxidized LDL within the innermost layer of arterial walls connect monocytes to endothelium and this may act as a risk factor for atherosclerosis. OX-LDL containing Lisophosphatidylcholine is a strong stimulus for VCAM-1 production [5, 6]. It has been shown that regular physical activities reduce arterial resistance by decreasing VCAM-1 and OX-LDL levels, and thus lead to decreasing blood pressure [7, 8]. To date, there are very few reports on the effects of swimming training on VCAM-1 and OX-LDL [9, 10].

Moderate swimming training as non-weight bearing exercise is associated with liveliness, lower stress and less injury for weight loss and may have an effect on VCAM-1 and OX-LDL levels in hypertensive patients. This knowledge may be relevant for exercise training program to reduce arteriosclerosis risk factors in hypertensive patients. However, there are no studies that have investigated the effects of moderate swimming training on the blood pressure factors. Thus the main aim of the present study was to investigate the effects of moderate swimming training on OX-LDL and VCAM-1 in hypertensive menopausal women.

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Methods

Participants

Thirty-four hypertensive postmenopausal women (age, mean ± SD, 55 ± 7.6 yr, body mass 76.9 ± 1.4 kg, height 157.6 ± 0.8 cm) participated in the study. All participants were on medication and were not involved in any kind of regular exercise at least six months prior to the study. The blood pressure for all participants was > 140/ 90 mm of mercury. In the training group, all participants were asked to stop taking medication at least 2 days before and after the 8-week training. Five participants with high variability of blood pressure were told to take half of their routine medication. However, participants in the control group took their routine medication. The study was conducted in accordance with the Declaration of Helsinki and approved by the local ethics committee at Sabzevar University. Participants provided informed written consent before participation in the study.

Procedures

Body mass index was calculated as weight (in kilograms) divided by height (in meters) squared. Body fat mass was determined by bioelectrical impedance analysis using a body composition analyzer (BioSpace, Korea).

Blood Sampling

To analyze biochemical variables, blood samples were collected (after 12-14 hours of fasting) in two conditions: before and after the 8-week training program. For all conditions, the participants were asked to avoid performing intensive physical activity at least two days before blood sampling. Temperature and time were recorded during blood sampling. Ten ml of blood was drawn from a vein in the left hand at rest condition. The blood sample was allowed to clot at room temperature for 30 minutes and then the clots were centrifuged for 5 minutes. Then four ml of serum was separated and kept in the refrigerator (at 40^0 C).The Elisa method was used to measure the quantity of OX-LDL (kit DRG Company of Germany) and VCAM-1(Biosource Company of USA) respectively.

Exercise Training

Exercise program comprised four sessions/week with a warm-up, cool down, moderate intensity walking, and 30-min of swimming at 60–70% of maximum heart rates. Swimming exercises included front crawl foot, back crawl foot and back crawl. Maximum heart rate was calculated by age formula (220-age), and the exercise intensity was controlled by measuring pulse rates at the left wrist. The pulse rate for participants was determined at 60-70% of their maximum heart rate, in which the participants were asked to keep the target pulse rate by modulating exercise intensity.

Statistical Analysis

All variables were normally distributed as tested with the Kolmogorov–Smirnov test. T-independent statistical method was used to compare dependent variables between the control and training groups. Pearson's correlation test was used to determine the correlation between variables. All statistical operations were carried out using SPSS software and significant level was set at $P < 0.05$ for all statistical procedures.

Results

After eight weeks of moderate swimming training, systolic blood pressure ($P<0.05$) and oxidized LDL ($p<0.05$) reduced (Figure 1, 2). Moreover, a positive correlation was observed between OX-LDL and systolic blood pressure ($R=0.34$, $p<0.05$) and LDL-C ($R=0.5$, $p<0.05$).

Body weight was also reduced ($p<0.001$). However, no changes were observed in diastolic blood pressure, VCAM-1, LDL-C, body fat and HDL-C (Table 1).

Discussion

The results of present study showed a significant decrease in systolic blood pressure and OX-LDL after eight weeks of moderate swimming training. However, no significant changes were observed either in diastolic blood pressure or in VCAM-1 after 8- weeks training program.

It has been reported that VCAM-1 and OX-LDL play an important role in increasing arteriosclerosis and hypertension [6]. A high level of VCAM-1 and OX-LDL has frequently been reported in postmenopausal women [5, 1]. The observation that systolic blood pressure and OX-LDL were lowest in postmenopausal women after 8 weeks of moderate swimming training, may indicate that mechanisms associated with decreasing systolic blood pressure and OX-LDL developed during the exercise and contributed to decreasing in this risk factors.

Our results are in agreement with many pervious findings in which a significant reduction in hypertension, VCAM-1, and OX-LDL has been reported after different types of physical activities [11, 7, 12, 8]. However the main finding of the

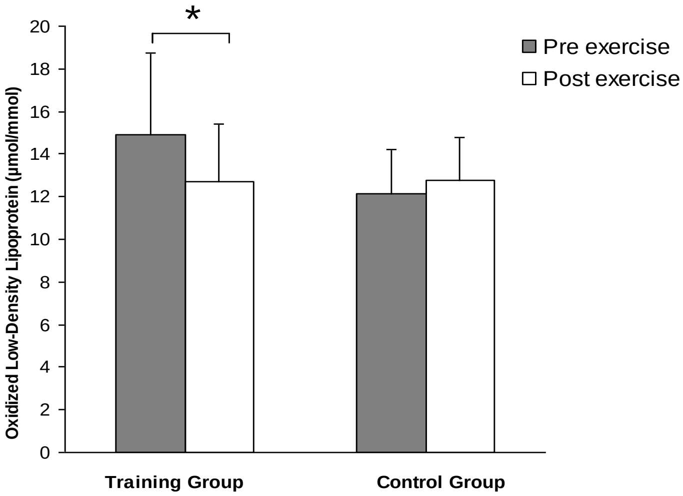

Figure 1: Oxidized low-density lipoprotein (mean ± SD, n = 34) for training and control group, measured before (pre exercise) and after eight weeks moderate swimming training at 60-70% of maximum heart rate (post exercise).* $p < 0.05$.

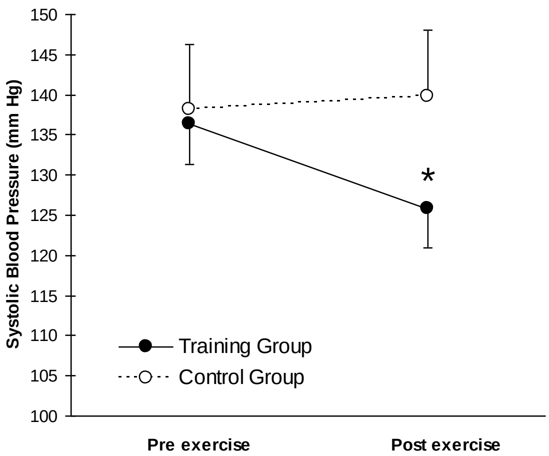

Figure 2: Systolic blood pressure (mean ± SD, n = 34) for training (black circle) and control group (whit circle), measured before (pre exercise) and after the training (post exercise).* $P < 0.05$

Table 1: Weight, body fat (BF), body mass index (BMI), low density lipoprotein cholesterol (LDL-C) high density lipoprotein cholesterol (HDL-C), diastolic blood pressure (DBP), and vascular cell adhesion molecule (VCAM) in training and control group before (pre exercise) and after eight weeks moderate swimming training at 60-70% of maximum heart rate (post exercise).* $P < 0.05$.

Variables	Group	Pre exercise	Post exercise
Weight(kg)	Training	70.97±11.35	65.22 ±18.96*
	Control	76.64±8.4	77.23± 8.6
BF (percentage)	Training	30.88± 5.52	29.21± 5.28
	Control	28.5± 5.63	29.25 ±4.62
BMI (kg/m^2)	Training	28.9± 4.34	27.71± 4.37
	Control	30.04± 4.21	30.56 ±5.22
LDL-C (mg/dL)	Training	169.77± 51.85	147.27± 28.27
	Control	143.68± 47.15	142.01± 46.44
HDL-C (mg/dL)	Training	45.52± 8.05	45.58± 9.3
	Control	44.94± 8.3	44.84± 8.84
DBP (mm Hg)	Training	88.82± 6.9	87.05± 5.01
	Control	87.64± 12.2	89.29± 14.4
VCAM-1(ng/Ml)	Training	22.87± 9.3	18.42± 7.1
	Control	21.32± 11.6	21.49± 9.5

study was that, the OX-LDL showed a significant reduction after eight weeks of elementary swimming training. Moreover, a significant relationship was also observed between OX-LDL values and systolic blood pressure, which may indicate the plausible role of OX-LDL in increasing blood pressure.

The lower value of OX-LDL after swimming training can be explained by an increase in activity of anti-oxidants and/or endothelial function associated with OX-LDL, which in turn led to reducing LDL in arterial endothelium, oxidative stress and adhesion molecules [4]. Changes in lipid profile as a result of physical activities can be another possibility of decreasing OX-LDL and systolic blood pressure [13, 14]. Physical activities stimulate muscle lipase, a factor that is known to increase high-density lipoprotein cholesterol (HDL-C) and to decrease factors associated with OX-LDL such as LDL-C, very low-density lipoprotein (VLDL), chilomicrons, triglyceride, and plasma cholesterol [13].

The significant reduction in OX-LDL as a result of swimming training may also be related to the reduced body fat. A greater number of active muscles during swimming exercise increase metabolic demands and thus accelerate the oxidation of fat tissue [15]. Moreover, the horizontal body position, lower body temperature and non-weight bearing exercising during swimming may have prolonged the exercise duration and, therefore, have led to further oxidation of fat tissue [16]. Although decrease in systolic blood pressure has been attributed to the OX-LDL reduction, we cannot reject the role of other factors such as sympathetic nervous system, C-reactive protein, increase in the sensitivity of receptors, insulin sensitivity improvement, prostaglandin E, nitric oxide and angiogenesis [17, 18, 19].

Limitations of the study

Five participants with high variability in blood pressure were suggested to start taking half of their routine medication. The problem is common to many clinical studies. However, OX-LDL and systolic blood pressure were lower in both non-medication and medication patients after the training period, while these factors remained unchanged for the control group. Thus the lower OX-LDL and systolic blood pressure in the training group probably indicate that moderate swimming exercise contributes to improvements in the anti-oxidants activity and/or endothelial function and reduces risk factors.

Conclusion

In conclusion, moderate swimming training reduced systolic blood pressure and OX-LDL in hypertensive postmenopausal women probably due to improvement of the anti-oxidants activity and/or endothelial function.

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