UNIVERSA MEDICINA

January-April, 2009 *Vol.28 - No.1*

Effect of aerobic exercise on blood lipid levels in postmenopausal women

Martiem Mawi*

ABSTRACT

*Physiology Department,
Medical Faculty,
Trisakti University

Correspondence
dr. Martiem Mawi
Physiology Department,
Medical Faculty,
Trisakti University
Jl. Kyai Tapa No.260
Grogol - Jakarta Barat
Phone: 021-5672731 ext.2803
Email:
martiemmawi@yahoo.com

Univ Med 2009; 28: 17-24

High blood total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG), and low concentrations of high-density lipoprotein cholesterol (HDL-C) are related to risk for coronary heart disease (CHD) development. Growing evidence indicates that physical exercise can prevent at least some of the negative effects on health associated with post. The purpose of this study was to evaluate the effect of regular aerobic exercise for 12 weeks on the levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides. An experimental study was conducted comprising 62 postmenopausal women, aged 50-70 years, not on hormonal therapy, consuming a regular diet, living in East and South Jakarta, and willing to perform aerobic exercises regularly. The results of this study showed that all four lipid levels differed significantly between the control group and the intervention group, the respective mean levels ± SD for TC being 228.0 ± 39.7 mg/dL vs. 171.6 ± 18.4 mg/dL, (p = 0.000); for LDL-C 149.0 ± 36.9 mg/dL vs. 97.7 ± 17.8 mg/dL, (p = 0.000); for HDL-C 50.9 ± 3.9 mg/dL vs. 71.5 ± 6.7 mg/dL, (p = 0.000); and for triglycerides 150.5 ± 67.5 mg/dL vs. 95.0 ± 37.8 mg/dL (p = 0.000). Thus practitioners recommending exercise for coronary artery disease risk reduction in postmenopausal women.

Keywords: Aerobic, exercise, blood lipid levels, postmenopausal women

INTRODUCTION

Coronary heart disease (CHD) is the leading cause of death and disability in both men and women.[1] The incidence of cardiovascular disease is low in premenopausal women, but increases in postmenopausal women, to a level similar to that in men.[2] Thirty percent of American women aged 65 years and above suffer from coronary arterial disease. After the onset of menopause, women have increased levels of total cholesterol, triglycerides, and low-density lipoprotein cholesterol (LDL-C) compared with their premenopausal counterparts. Epidemiological studies have shown a relationship between elevated cholesterol and coronary arterial disease.[3]

Every woman will experience menopause.[4] Illnesses that often occur in postmenopausal women are cardiovascular disease, osteoporosis, cancer and stroke.[2,5,6] Risk factors for cardiovascular disease include hypercholesterolemia, hypertension, and smoking.[2,4] High levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides, and a low level of high-density lipoprotein-cholesterol (HDL-C) are risk factors for coronary arterial disease. HDL-C is a strong predictor of coronary arterial disease. Research in the US found that a decrease of 1 mg/dL HDL-C would increase the risk of coronary arterial disease by 2-3 %.[7]

Coronary arterial disease increases in postmenopausal women because of a decrease in the levels of estrogen, and the occurrence of ovarian degeneration in postmenopausal women.[7-9] Estrogen has protective properties on the cardiovascular system by increasing HDL-C, and decreasing TC, LDL-C and triglycerides,[10-12] whereas a decrease of estrogen decreases HDL-C, and increases TC, LDL-C and triglycerides. Overall, the reduced estrogen level will accelerate the atherosclerotic process in the coronary arteries.[2]

In some postmenopausal women, hypercholesterolemia is also present. Typically, TC increases by 5-7%, LDL-C 10-12%, triglyceride 8-10%, and HDL-C decreases by 4-6%. According to Robergs, women who exercise regularly four times or more per week show a decrease in the risk of coronary arterial disease.[13] Physical conditioning could have an effect on coronary arterial disease through aerobic exercise, such as static ergo cycle, gymnastics, and walking. Regular exercise in postmenopausal women could increase HDL-C and decrease TC, LDL-C, and triglycerides. Besides those effects, exercise could also decrease hypertension, blood glucose, overweight, stress, and menopausal symptoms, and could increase physical fitness.[14]

The results of one study showed that there was no increase of HDL-C in postmenopausal women after 40 weeks of aerobic walking exercise. These results may be due to the short duration and low intensity of exercise done in the study. The HDL-C will increase significantly if the duration of the aerobic exercise per week is long enough.[15] A different result was obtained in Germany, where LDL-C in postmenopausal women after 4 years of intensive aerobic exercise did not show any significant difference compared to the control group.[14] In postmenopausal women, the duration of exercise per week will slightly increase the HDL-C compared to the intensity of exercise; the duration of exercise should be around 12 weeks, and the intensity of exercise should be moderate.[16]

The objective of this study was to determine the levels of blood lipids after regular aerobic exercise for 12 weeks in postmenopausal women.

METHODS

Research design

The study design was an experimental randomized non-blinded controlled trial.

Research subjects

The subjects were women aged between 50-70 years, living in East and South Jakarta, with a time window of 3 years after menopause, being in good health (not suffering from any ailments, such as diabetes mellitus, liver disease, heart disease, stroke, or lung and kidney diseases), not having received any hormonal therapy in the past three years, and consuming a balanced diet.

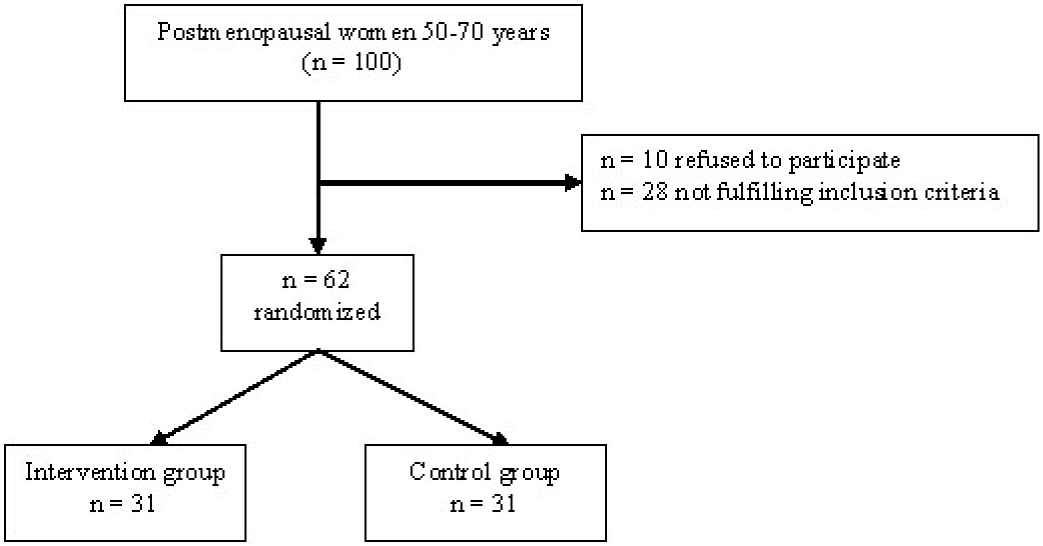

Figure 1. Screening of postmenopausal women

Intervention with aerobic exercise

The subjects in the intervention group performed aerobic exercises for 12 weeks with a frequency of 4 times a week. Before and after each round of exercises, blood pressure was measured. For the aerobic exercises, an ergocycle (Monark, Finland) was used to measure the speed and loading, and the duration of exercise was time by a stopwatch. An electrocardiograph (Cardiopax, Nihon Kohden, Japan) was used for monitoring of the heart rhythm and a Polar (Vantage NV™, Polar electro Oy, Finland) for checking of the heart rate before, during, and after the exercises. The exercises lasted for 25 minutes, of which the first 5 minutes were used for warming-up, the next 15 minutes for the main exercise, and the last 5 minutes for cooling down. The pulse rate was monitored until a value of 60% x (220 – age) had been achieved. For the first 2 weeks the exercises were conducted without addition of loads, and then loading was applied and gradually increased from 0.5 kp to 0.75 kp, and maximally to 1 kp. The weight was adjusted according to the health of the subject. If during the exercise, the pulse increased to 170 times per minute or more, or if the subject had headache, fatigue, or palpitations, then the exercise was terminated, and the subject was excluded from the study. A subject who had not completed the exercise for 12 weeks was also excluded from the study. The control group continued their habitual life style.

Measurement of variables

Recorded subject characteristics comprised age, duration of menopause, and previous or current diseases, such as liver disease, diabetes mellitus, heart disease, stroke, or lung and kidney disease. The participants were also subjected to physical examination, resting 12-lead ECG recording, and fasting and 2-hour post-prandial blood glucose, blood pressure, and body mass index [weight (kg) divided by height (m^2)] measurements. Anthropometric measurements included standing height and weight. The subjects' height (without shoes) was recorded to the nearest 0.5 cm, whilst recording of weight (with minimal clothing) was to the nearest 0.1 kg. Height was taken using an anthropometer (Microtoise ruban 2.20, France) and weight with a well-calibrated spring balance (Secca 770 Alpha, Germany).

Those subjects who completed the above measurements were further examined at a certified laboratory in Jakarta for their lipid profile, namely TC, LDL-C, HDL-C and triglycerides. The subjects were required to have fasted 14 hours prior to the examination, when 3 mL of blood was taken from the antecubital vein, and heparin or EDTA was added, or the blood was centrifuged to obtain serum. The TC was measured by CHOD PAP enzymatic calorimetry using Roche reagents. HDL-C as well as LDL-C concentrations were determined using a homogenous enzymatic method with Daiichi reagents. Triglyceride level was measured by the GPO-PAP method using Roche reagents. All of the above assessments of lipid levels were performed using Modular P 800, Roche, Germany.

According to the National Cholesterol Education program, the upper normal limits of serum lipid concentrations are as follows: TC 200–239 mg/dL, LDL-C 130-159 mg/dL, triglyceride 150-199 mg/dL, and HDL-C $\geq$ 60 mg/dL.

The present research had been approved by the Ethical Committee of the Medical Faculty, Trisakti University. Before starting the study, the subjects were given an informed consent form to fill out. After the subjects had expressed their consent by signing the form, they were given an interview and examination.

Statistical analysis

The normality of the data on TC, LDL-C, HDL-C, triglyceride, age, BMI and duration of menopause at baseline was tested using the Kolmogorov-Smirnov test. If the result of the normality test showed that the data had a normal distribution, then the independent t-test was performed. Independent t-test analysis was also used to test the differences in TC, LDL-C, HDL-C and triglyceride concentrations between the intervention and control groups. A p value of < 0.05 (2-tailed) was considered to be significant. All analyses were performed using the SPSS/PC statistical program (version 12.0) for Windows.

RESULTS

Among the one hundred postmenopausal women who were available, ten refused to be included in the study and 28 did not fulfill the inclusion criteria, thus bringing the final sample size to 62. They were divided into two groups, thirty-one subjects were randomly assigned to the control group and another 31 to the intervention group (Figure 1). The normality tests on TC, LDL-C, HDL-C, triglyceride, age, BMI and duration of menopause at baseline were done using the Kolmogorov-Smirnov test. The results of the normality tests showed that TC ($p = 0.607$), LDL-C ($p = 0.253$), HDL-C ($p = 0.959$), triglyceride ($p = 0.079$), age ($p = 0.959$), BMI ($p = 0.607$), and duration of menopause ($p = 0.959$) had a normal distribution.

To compare baseline values of important characteristics, namely TC, LDL-C, HDL-C, triglyceride, age, BMI and duration of menopause, the independent t-test was used. The results showed no differences between the two groups with regard to TC, LDL-C, HDL-C, triglyceride, age, BMI and duration of menopause at baseline.

The mean age at baseline of the control group of 59.9 ± 5.4 years was not significantly different from that of the intervention (exercise) group of 59.9 ± 4.3 years ($p = 0.959$). Similarly, mean BMI and mean duration of menopause in the control group of 26.2 ± 4.5 kg/m2 and 9.7 ± 5.6 years, respectively, did not differ significantly from the corresponding variables in the intervention group, being respectively 24.8 ± 4.4 kg/m2 (p = 0.198) and 10.5 ± 5.9 years (p = 0.425). Mean TC, LDL-C and HDL-C concentrations of the control group (210.3 ± 34.7 mg/dL, 135.0 ± 28.1 mg/dL, and 54.8 ± 11.1 mg/dL, respectively) also showed no significant differences with their counterparts in the intervention group (respectively 202.3 ± 33.1 mg/dL, 128.0 ± 27.7 mg/dL, and 55.9 ± 13.2 mg/dL), the corresponding p values being p = 0.355, p = 0.345, and p = 0.733. Mean triglyceride concentration of the control group was 137.0 ± 56.1 mg/dL, which was not significantly different from that of the intervention group of 110.0 ± 54.0 mg/dL (p = 0.063) (Table 1).

After exposure to 12 weeks of aerobic exercise, the TC concentration of the control group was 228.0 ± 39.7 mg/dL whereas that of the intervention group was 171.6 ± 18.4 mg/dL, indicating a significant difference in TC levels between the two groups (p = 0.000). The LDL-C concentration in the control group of 149.0 ± 36.9 mg/dL was also significantly different from that in the intervention group of 97.7 ± 17.8 mg/dL (p = 0.000). Similarly, HDL-C concentrations were significantly different between the control group (50.9 ± 3.9 mg/dL) and the intervention group (71.5 ± 6.7 mg/dL) with p = 0.000, while the same was true for triglyceride concentrations, being 150.5 ± 67.5 mg/dL and 95.0 ± 37.8 mg/dL, respectively (p = 0.000) (Table 2).

Table 1. Age, BMI, TC, LDL-C, HDL-C, triglycerides and menopause duration at baseline by intervention groups

Characteristics	Intervension (n = 31)	Control (n = 31)	p-value
Age (years)	59.9 ± 4.3	59.9 ± 5.4	0.959
BMI (kg/m^2)	24.8 ± 4.4	26.2 ± 4.5	0.198
TC (mg/dL)	202.3 ± 33.1	210.3 ± 34.7	0.355
LDL-C (mg/dL)	128.0 ± 27.7	135.0 ± 28.1	0.345
HDL-C (mg/dL)	55.9 ± 13.2	54.8 ± 11.1	0.733
Triglycerides (mg/dL)	110.0 ± 54.0	137.0 ± 56.1	0.063
Menopause duration (year)	10.5 ± 5.9	9.7 ± 5.6	0.425

Note: BMI = body mass index; TC = total cholesterol; LDL-C = low density lipoprotein cholesterol; HDL-C = high density lipoprotein cholesterol

Table 2. Mean of BMI, TC, LDL-C, HDL-C and triglycerides after intervention by intervention groups

Characteristics	Intervention (n = 31)	Control (n = 31)	p-value
TC (mg/dL)	171.6 ± 18.4	228.0 ± 39.7	0.000
LDL-C (mg/dL)	97.7 ± 17.8	149.0 ± 36.9	0.000
HDL-C (mg/dL)	71.5 ± 6.7	50.9 ± 3.9	0.000
Triglycerides (mg/dL)	95.0 ± 37.8	150.5 ± 67.5	0.000

Note: TC = total cholesterol; LDL-C = low density lipoprotein cholesterol; HDL-C = high density lipoprotein cholesterol

DISCUSSION

The results of this study showed that 12 weeks of aerobic exercise decreased TC, LDL-C, triglyceride and increased HDL-C levels of postmenopausal women. These results are consistent with the those of one study on early postmenopausal women who exercised over 26 months and showed significant exercise effects on serum levels of TC and triglycerides.[17] Similar investigations in exercise studies have been rather heterogeneous and depend on exercise intensity, duration, and frequency. Increases in HDL-C levels were observed if regular exercise intensity levels exceeded 80% HR max, but not at lower intensity levels.[18] The HDL-C increases with moderate intensity exercise for 60 minutes with frequency of 2 to 4 times per week[19] In exercises of moderate to light intensity of more than 30 minute duration, the energy needed for the exercises is mainly generated from fat by lipolytic enzymes and mitochondrial activity. It can be concluded from several studies that in postmenopausal women moderate intensity exercise of long duration and sufficient weekly frequency is recommended for increasing HDL-C.

Fahlman et al.[15] reported increased HDL-C levels, and decreased TC, LDL-C, and triglyceride concentrations at week 10 of an aerobic training program at 70% heart rate submaximal in elderly women. Regular exercise four times or more per week in postmenopausal women could increase HDL-C and decrease TC, LDL-C, and triglycerides. Besides those effects, exercise could also reduce hypertension, blood glucose, stress and menopausal symptoms.[13,14] For postmenopausal women it is recommended to perform dynamic types of aerobic exercises, namely static bicycle, swimming, jogging and walking. These exercises are beneficial both physically and psychologically. Physically, there will be increases in the number of red blood cells, capillary ratio and amount of muscle fiber as well as blood flow during exercise, and also increased HDL-C and fibrinolytic blood activity to decrease the risk of atherosclerosis. There is a positive relation between exercise and decreased incidence of CHD.[13]

A meta-analysis indicated that the effect of aerobic training resulted in a 2.53-mg/dL (0.065-mmol/L) elevation of net HDL-C change.[20] This is potentially of substantial importance in public health, although the effect of reducing cardiovascular risk by increasing HDL-C level might be smaller than that obtained through use of medications such as fibrates or niacin.[21] Exercise increases the capacity of skeletal muscle to oxidize both glycogen and fatty acids to carbon dioxide and water. This responsive mechanism to increase fatty acid oxidative capacity is related to increased fatty acid tissue release and enzymes related to the transport and degradation of fatty tissue. Fatty acids enter the Krebs cycle and electron transport system, and the mitochondrial changes that occur are facilitated by the enzymes involved; all have a role in increasing skeletal muscle oxidative potential for fat metabolism.[22] During sub-maximal work more than 60% of the lipids utilized comes from intramuscular fat stores during muscle contraction due to low muscle glycogen. With submaximal work, an athlete oxidizes more fat than glycogen compared to a non-athlete. Moreover, during exercise with submaximal effort, the amounts of lactic acid stored are less and fatigue is decreased.[13]

A limitation of this study is its non-blinded randomized design. However, randomization of exercise trials is much more difficult compared with placebo-controlled pharmaceutical studies because exercise studies cannot be blinded. Participants in exercise studies often refuse to be randomized to the study arm that they do not prefer. People who do not want to exercise will drop out immediately. Even worse, those subjects randomized to the control group who initially wanted to exercise may exercise without reporting, causing a bias.

CONCLUSION

Aerobic exercise in postmenopausal women of 50-70 years performed routinely for 12 weeks with a frequency of 4 times weekly and a duration of 25 minutes for each round of exercise, was able to reduce lipid levels, which are an important coronary heart disease risk factor.

ACKNOWLEDGMENTS

The authors would like to thank the Trisakti University and the Medical Faculty that funded the study. We would also like to thank the women who participated in this study.

REFERENCES

1. Welty FK. Cardiovascular disease and dyslipidemia in women. Arch Intern Med 2001; 161: 514-5.
2. Rosano GMC, Vitale C, Voltererrani M. Menopause and cardiovascular disease: the evidence. Climacteric 2007; 10 (suppl 1): 19-24.
3. Bittner V. Estrogens, lipid and cardiovascular disease: no easy answers. J Am Coll Cardiol 2000; 37: 431-3.
4. White WB. Drospirenone with 17 ß estradiol in the postmenopausal woman with hypertension. Climacteric 2007; 10 (suppl 1): 25-31.
5. Ahlborg HG, Johnell O, Turner CH, Rannevik G, Karlsson MK. Bone loss and bone size after menopause. N Engl J Med 2003; 349: 327-34.
6. Lobo RA. Menopause and stroke and the effects of hormonal therapy. Climacteric 2007; 10 (suppl 2): 27-31.
7. Assmann G, Nofer JR. Atheroprotective effects of high density lipoprotein. Ann Rev Med 2003; 54: 321-41.
8. Bulun SE, Nelson LR. Estrogen production and action. Semin Reprod Endocrinol 1999; 4: 349-58.
9. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 2001; 285: 2486-97.
10. de Aloyso D, Gambacciani M, Meschia M, Pansini F, Modena AB, Bolis PF, et al. The effects of menopause on blood lipid and lipoprotein levels. The ICARUS Study Group. Atherosclerosis 1999; 147: 147-53.
11. Hinderliter L. Cholesterol control. Am J Clin Nutr 2001; 74: 730-6.
12. Molina PE. Endocrine physiology. New York: McGraw Hill; 2004: 207-43.
13. Robergs RA, Keteyian SJ. Fundamentals of exercise physiology: for fitness, performance, and health. 2nd ed. San Francisco: McGraw Hill; 2003. p. 512
14. Kemmler W, Engelke K, von Stengel S, Wienneck J, Laurer D, Kalender WA. Long-term four-year exercise has a positive effect on menopausal risk factors: the Erlanger fitness osteoporosis prevention study. J Strength Cond Res 2007; 21: 232-9.
15. Fahlman MM, Boardley D, Lambert CP, Flinn MG. Effects of endurance training and resistance training on plasma lipoprotein profiles in elderly women. J Gerontol A Biol Sci Med Sci 2002; 57: B54-60.
16. Vella CA, Kravitz L, Janot JM. A review of the impact of exercise on cholesterol levels. Am Heart J 1999; 119: 277-83.
17. Kemmler W, Dirk Lauber D, Weineck J, Hensen J, Kalender W, Engelke K. Benefits of 2 years of intense exercise on bone density, physical fitness, and blood lipids in early postmenopausal osteopenic women. Arch Intern Med 2004; 164: 1084-91.
18. Haddock BL, Hopp Marshak HP, Mason JL, Blix G. The effect of hormone replacement therapy and exercise on cardiovascular disease risk factors in postmenopausal women. Sports Med 2000; 29: 39-49.
19. Sunami Y. Impact of exercise on cholesterol. Am Heart J 1999; 119: 277-83.

20. Kodama S, Tanaka S, Saito K, Shu M, Sone Y, Onitake F, et al. Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: a meta-analysis. Arch Intern Med. 2007; 167: 999-1008.
21. Birjmohun RS, Hutten BA, Kastelein JJ, Stroes ES. Efficacy and safety of high-density lipoprotein cholesterol increasing compounds: a meta-analysis of randomized controlled trials. J Am Coll Cardiol 2005; 45: 185-97.
22. Powers SK, Howley ET. Exercise physiology 6th ed. New York: McGraw Hill; 2007.