Effect of aerobic exercises on leptin serum, and cardiovascular risk factors in patients with type II diabetes

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Abstract: Background and Aim: Today, diabetes is the most common disease in developing community's especially urban areas. The aim of this study is to investigate the effect of aerobic exercises on leptin serum, and other factors of cardiovascular risks in patients with type II diabetes. **Materials and Methods:** The study subjects are consisted of 700 male patients with diabetes type II A. Among these, 31 patients have a history of less than 10 years and their age ranges between 35 to 55 years who are randomly assigned to the experimental group (16 cases) and control group (15 cases). The experimental group was under the effect of 50-minute aerobic exercises with the intensity of 60-70 percent HRRmax, 3 times a week for 8 weeks. Both pre-test and post-test measurements of height, weight, waist-to-hip ratio, blood pressure, heart rate, and blood sampling of subjects were performed. In this quasi-experimental test, the Kolmogrov-Smirnov test for proving the normality assumption of data and the T-Test was used to confirm the research hypotheses. **Results:** The findings showed that the selected aerobic training has an impact on leptin serum, HDL, blood pressure, body fat percentage and VO2max of subjects, ($P<0.05$) and doesn't affect total cholesterol, triglycerides, LDL, BMI and WHR. **Conclusions:** Regular aerobic exercise effectively controls diabetes and reduces the risk factors for cardiovascular disease.
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1. Introduction:

With the advancement of science in this century, the world has undergone massive changes, and sciences related to physical education and sport are no exception. One of these fields is the effect of exercise on improvement and control of the disease. Among this type of diseases, the type II diabetes, which is the most common form of diabetes, can be mentioned. According to the estimation of International Diabetes Association, in 2010 about 221 million peoples worldwide are living with diabetes, which is estimated to rise such that in the year 2025 it reaches to about 333 million people. In general we can say that 3.6 percent of the world's population have diabetes. In people with diabetes, the body's cells are not sensitive enough to the insulin hormone, and after a while, the pancreas will also have difficulties in producing insulin in sufficient quantities. Without enough insulin sensitivity, or the lack of its discharge, the body would not be able to move the sugar in the blood into cells to be used, thus blood sugar rises and causes various problems. Two main factors in type II diabetes' risk are obesity and lack of exercise. Obesity increases risky factors such as hypertension, high levels of LDL, cholesterol and triglycerides and low levels of HDL. So it gives rise to the development of diabetes. In total, about one billion people worldwide are overweight and at least 300 million of them are obese. Index attributable to high body mass index (BMI kg/m2) is 4.79% in Iran, and the overall percent of overweight 10.3%. According to the relationship between obesity and diabetes, the prevalence of this disease is 2.3 percent. The pathology caused by obesity has a series of factors including genetic predisposition, impaired metabolism of leptin, environmental factors and food intake are involved (6). It seems there is a link between diabetes and physical activity, so that increasing physical activity can help prevent or delay diabetes. One of the significant issues in the field, is the effect of exercise on leptin. Leptin was discovered by Zheng and colleagues in December 1994, and had a severe impact on the understanding of the molecular control of energy balance and obesity studies (19). Leptin controls the food intake and body weight and delays the need to eat (12, 19). This means that leptin problems cause increased susceptibility to obesity (12). Extremely high levels of leptin in obese patients compared with non-obese individuals are observed. Although leptin levels in individuals with below-normal weight have even more loss than people with normal weight (36). Leptin is considered as an anti-obesity hormone (12). Leptin is a hormone secreted by fat cells and is an indicator of total body fat in pre-breakfast form. Previously it was supposed that leptin is a signal for

weight loss, but in fact it is a signal to the brain informing the body fat amount. It looks like when leptin is not secreted, the brain thinks there is no fat in the body and stimulates the person to eat more and make more body fat. Leptin also expresses the energy balance in the body. In many studies, fasting or extreme weight loss diets reduce the levels of leptin in the body. In these terms, leptin is more sensitive to fasting than overeat. Meaning that after overeat, the leptin levels do not increase greatly. In fact, leptin levels are proportional to the amount of fat in the body. It enters the central nervous system proportional to the amount of its plasma thickness. Its receptors on the brain cells get involved in the regulation of energy. Through interaction with receptors in the hypothalamus which controls the amount of food intake and energy expenditure. In some children, the incidence of early obesity is associated with an inability to produce leptin. Been seen in obese children who are unable to lose weight, genetic defects in leptin production and leptin injections in children cause significant weight loss in them. The girls who were under treatment with leptin, leptin-deficient humans have significant weight loss (average 16.4 kg) and changes in body composition were observed in them. However, the leptin administration to leptin levels and body in normal adults was not significant change in weight. In previous studies, much pain at the injection site in some patients, leading to discontinuation of treatment with leptin, and leptin treatment in people who still work their bodies having normal leptin levels, is not clear. There are theories based on leptin level regulated by melatonin during the night. One study suggested that increased levels of melatonin decreases leptin levels (negative autoregulation). In another study it was shown that the interaction of melatonin on leptin and insulin affect appetite decreases during sleep. Extremely high levels of leptin in obese patients compared with non-obese individuals are observed. While leptin levels in individuals with below normal weight are even more than people of normal weight loss has been shown (36). It appears that the insulin regulates the leptin mRNA, although the effects vary according to glucose status. The production of leptin after the increase of insulin in response to eating occurs during fasting and after insulin reduction, reduces leptin. Researches indicate that the concentrations of physiological doses of leptin inhibit the secretion of basal insulin in the pancreas of ob mice, but have no effect on the pancreas of fa zucker mice. Use Of leptin, eliminates insulin resistance of liver. It seems that there is a relationship between leptin and insulin. It is suggested that changes in the cross-correlation of insulin happens 275 minutes before leptin. On the other hand, direct effect of insulin on mRNA ob In differentiated fat cells of rat adipose cells fully differentiated developed or newly differentiated human fat cells first appear (4). Leptin can be considered as an anti-obesity hormone (12). With fat increasing, leptin generates a signal which reduces the absorption of nutrients. Based on studies on diabetic people, leptin secretion rate varies in different races. Serum leptin in Indians, Caucasians and African Sudanese insulin levels are lower, while in hybrid Mexicans is higher. Fasting, low blood sugar, low nutrition and exercise can decrease leptin (13, 16). Hormones such as glucocorticoids, growth hormone and catecholamines in the regulation of leptin are employed by the practice and modified (9, 14). Growth hormone and cortisol promote leptin secretion (11, 18) while catecholamines prevent the secretion (10, 17). Exercise is a factor to reduce obesity, so if leptin levels are affected by exercise, how exercise effects on obesity makes clear. Practice may be impaired leptin production and reproduction performance, because leptin is associated with exercise training their attention on a series of hormones such as insulin, cortisol, catecholamines, estrogen and growth hormone, and dehydro-epiandrosterone (11, 15).

2. Methodology

The study was quasi-experimental. From the population of 700 diabetic type II men with the age range of 35 to 55 years who have medical records in Mashhad Parsian Diabetes Center, and less than 10 years of history and fasting blood glucose less than 250 mg/dl and BMI above 26 kg/m2, 35 participants of health questionnaire that participants who had no history of acute and chronic complications of diabetes complete and full consent to participate in the study were randomly selected as subjects, and the two controls (15 individuals) and experimental (16 individuals). Pre-test, demographic and physical characteristics of the subjects such as height, weight, body mass index, blood pressure and skinfold measurements in specific forms of individual measurements were recorded and subsequently blood tests was performed. Then engaged in aerobic exercise group while the control group received no exercise or special diet did not. Variables consisted of the independent variable, the training program included aerobic exercise for 60 to 70 percent HRRmax during an 8-week training period and frequency of 3 days per week and duration of each session was 50 minutes, and Dependent variables included maximal oxygen consumption, body mass index, serum leptin levels, the level of cardiovascular risk factors (cholesterol, blood pressure, LDL, HDL and TG) percent body fat and waist-hip ratio were. During the test, previous measurements and blood

tests were repeated. Data analysis and data from tests on blood samples before and after 8 weeks of training, were used for statistical analysis and descriptive data, descriptive statistics including graphs, tables, mean and standard deviation was used for the purposes of research and data normality using Kolmogrov-Smirnov test hypotheses Inferential statistics T the independent student groups were used, For data analysis, software SPSS-V13 With a significance level of $p<0.05$ was used.

Table 1. Data analysis

Stats	Group	Number	Stage	Mean + standard deviation	Pre-and post-test mean + standard deviation	Value TCalculated	Acceptance of	Significant
Leptin	Control	15th	Pretest	10/1 ± 84/4	72/0 ± 75/0-	41/2-	02/0	
			So Test	09/1 ± 09/4				
	Experimental	16th	Pretest	63/1 ± 85/4	47/2 ± 30/2-			
			So Test	02/1 ± 54/2				
HDL	Control	15th	Pretest	68/7 ± 8/39	18/8 ± 33/5-	062/2-	048/0	
			So Test	55/10 ± 13/45				
	Experimental	16th	Pretest	18/6 ± 06/36	57/6 ± 81/10-			
			So Test	83/9 ± 87/46				
LDL	Control	15th	Pretest	53/40 ± 13/117	36/26 ± 47/11-	147/0-	884/0	-
			So Test	17/32 ± 67/105				
	Experimental	16th	Pretest	73/29 ± 63/113	64/16 ± 63/12-			
			So Test	16/26 ± 00/101				
Triglycerides	Control	15th	Pretest	46/97 ± 20/177	38/112 ± 13/6-	358/0	723/0	-
			So Test	43/64 ± 07/171				
	Experimental	16th	Pretest	41/88 ± 38/196	53/80 ± 38/6			
			So Test	9/102 ± 75/202				
Cholesterol	Control	15th	Pretest	47/47 ± 47/194	08/23 ± 87/0-	606/0-	549/0	-
			So Test	44/51 ± 60/193				
	Experimental	16th	Pretest	81/39 ± 87/192	64/26 ± 31/6-			
			So Test	91/41 ± 56/186				
Systolic pressure	Control	15th	Pretest	18/1 ± 53/12	77/0 ± 43/0	56/5-	000/0	
			So Test	20/1 ± 97/12				
	Experimental	16th	Pretest	41/1 ± 44/12	57/0 ± 94/0-			
			So Test	15/1 ± 50/11				
Diastolic pressure	Control	15th	Pretest	72/0 ± 33/8	93/0 ± 37/0	832/2-	008/0	
			So Test	06/1 ± 70/8				
	Experimental	16th	Pretest	04/1 ± 19/8	89/0 ± 56/0-			
			So Test	14/1 ± 63/7				
Fat	Control	15th	Pretest	07/6 ± 14/31	61/2 ± 35/0	626/2-	014/0	

			So Test	43/5 ± 49/31				
	Experimental	16th	Pretest	35/6 ± 94/29	41/2 ± 01/2-			
			So Test	04/5 ± 92/27				
BMI	Control	15th	Pretest	90/3 ± 62/28	32/0 ± 12/0	762/1-	089/0	-
			So Test	01/4 ± 74/28				
	Experimental	16th	Pretest	04/5 ± 28/28	75/0 ± 25/0-			
			So Test	91/4 ± 03/28				
WHR	Control	15th	Pretest	067/0 ± 0001/1	022/0 ± 007/0-	002/0-	998/0	-
			So Test	068/0 ± 9927/0				
	Experimental	16th	Pretest	045/0 ± 9606/0	052/0 ± 007/0-			
			So Test	071/0 ± 9531/0				
VO2MAX	Control	15th	Pretest	30/4 ± 48/31	53/2 ± 94/0-	778/5	000/0	
			So Test	48/3 ± 54/30				
	Experimental	16th	Pretest	42/6 ± 42/31	68/1 ± 48/3			
			So Test	87/5 ± 91/34				

3. Discussion and study:

The normal level of serum leptin is between 3.7 to 11.1 (ng/ml). Aerobic exercises will be 8 weeks reduced serum leptin ($p<0.05$). Changes in leptin research, with reports of Ilahy and Ryan (1996), Tominenn and colleagues (1997), Akasaky and colleagues (1999), Hall et al (1999), Elias et al (2000), Ishi and colleagues (2001) Tong and colleagues (2002), Karmozys and colleagues (2002), Kramer and colleagues (2002), Darlin and colleagues (2003) and Ozlik (20) and colleagues (2003) were consistent with reports Hicky (1997), Voltman et al (2000) and Hommard et al (2000) is inconsistent. The findings appear to conflict with other research, the type and intensity of exercise and fitness level subjects before exercise is concerned. Cortisol secretion in obese patients, especially those with fat accumulation, increased. Elevated cortisol leads to increased secretion of leptin and leptin levels is therefore consistent with the effects of aerobic exercise, cortisol secretion is reduced, leading to a decline in leptin levels. Also on exercise, increased levels of catecholamines, which varies inversely with leptin secretion and the leptin is low. Since the amount of fat during exercise reduced leptin is derived from fat, hence reducing body fat, leptin has been another factor in the decline. Leptin is associated with insulin resistance, so it can be a cause of low blood sugar and improve insulin sensitivity was reduced.

Normal range of HDL is more than 45 mg per dL (8). Aerobic exercise training for 8 weeks significantly increased HDL ($p<0.05$) Changes of HDL with reports of Lehmann and colleagues (2001), Kaffed and colleagues (2003), Mehdi Esfahani (1381) is consistent and with reports of Mayorna et al (2002), Dunn Stan et al (2002) and Bruce et al (2004) is inconsistent. The difference in other research can be fed from different test and lack of control. Since the bulk of the building HDL protein may increase muscle mass and protein synthesis and the increase in this lipoprotein has to be very impressive. It seems that the intensity of diabetes HDL increase is desirable.

3.1. *8 weeks of aerobic exercise does not change LDL ($p<0.05$)*

In change of LDL, reports of Barnard and colleagues (1983), Krotiovsky (1985), Bill Bohr et al (2001), and M. Esfahani et al (1381) are consistent and with the reports of Lehmann and colleagues (2001) are inconsistent. Increase in blood triglyceride levels and VLDL increased hydrolysis of VLDL By lipoprotein lipase, IDL that contain lower levels of triglycerides, is generated. IDL metabolized mainly by adipose tissue and LDL make up (5). So we can deduce that the availability of resources triglycerides, the manufacturer LDL been rooted in food intake and elevated blood sugar and insulin resistance are (5). Also, because these changes can be considered during the exercises and medication.

3.2. Normal range of triglycerides is less than 150 mg per deciliter. Triglyceride levels did not change significantly after 8 weeks of training ($p<0.05$).

Changes in triglycerides in this study, with reports Lampman et al (1998), Mayorana and colleagues (2002), Dunn Stan and colleagues (2002), Kabir Anaraki (1379) and Esfahany (1381) were consistent and with Krotiovsky (1985) and Lehmann and et al (2001) were inconsistent. Triglycerides dietary source of fat needed by the body, which means that the bulk of the edible triglyceride oil is supplied from food sources. So after taking the drug, can increase triglyceride levels. Also, due to the fact that triglycerides, storage form of fat in the body can also be made from carbohydrates. Convert carbohydrates into triglycerides that occurs especially when the diet is high in carbohydrates and triglycerides are low (5). Because diabetics have high blood sugar or on a swing, the body begins to produce blood triglycerides and triglyceride increases. Seems to have been a cause of elevated triglycerides intensity. Intensity above 65 percent, followed by catabolism of fatty acids and triglyceride synthesis is enhanced (1). The difference in the values obtained, in addition to the intensity and duration of exercise is likely.

3.3. 8 weeks of aerobic exercise training does not change cholesterol level ($p<0.05$).

Cholesterol changes in the research with the reports of Dunn Stan and colleagues (2002), Kaffed et al (2003) and M. Esfahani (1381) are consistent and with the observations of Wallenberg et al (1982) and Barnard et al (1982) are inconsistent. Normal range cholesterol less than 250 mg per deciliter and food are probably the most important factor involved in the regulation of lipid accumulation. Two times more saturated fat than poly-unsaturated fats, influence on serum cholesterol levels. The liver produces cholesterol to other tissues of the body center of bile salts. In order to be normal cholesterol, the body's synthesis of some of the digestion and absorption of nutrients in the small intestine and bile salts back into the blood, to be coordinated. So you can control your cholesterol with diet and proper exercise and a balanced structure (1). Finally, the concentration of cholesterol can result in natural physiological change. For cholesterol the physiologic ratio is 6.5 percent. So when a person's cholesterol level is measured in the sample, about 13 percent in 95 percent of cases can be high or low blood cholesterol test average person is variable (7). This increase may be due to a diet high in cholesterol, fat, short duration exercises and normal physiologic changes.

3.4. 8-week of aerobic exercise can reduce systolic and diastolic blood pressure ($p<0.05$)

This study reports show that changes in blood pressure are consistent with Bill Bohr et al (2002) and M. Esfahani (1381) and with reports of Mayorana et al (2002) are inconsistent. Consistency of response in patients with diabetes is the result of exercise, increases blood vessels that directly leads to lower blood pressure. According to reports, with the loss of 5 to 10 percent of body weight in overweight patients, and reduce the risk of diabetes, dyslipidemia, and increased blood pressure (3). However, based on studies, systolic and diastolic blood pressure is associated with leptin levels (8). So in addition to the increased density of blood vessels, reducing weight, body fat and blood pressure can be attributed to the decline in leptin.

3.5. Aerobic exercises for 8 weeks reduced body fat ($p<0.05$).

Changes in fat, with the studies of Wing et al (2000), Ishi et al (2001), Cecil et al (2001) is consistent, but is inconsistent with observations of Hicky et al (1997), Christose and colleagues (2003), and Muhammad Ali Sardar (1384). For burning fat O2 is needed. The effect of aerobic exercise increases capillary density, which means more blood and oxygen to the body's muscle tissue and reaches the maximum in long-term activities that more oxygen is available to meet the energy needs of fat metabolism using. So, the intake of fat in 8 weeks of physical training, body fat percentage has dropped significantly.

3.6. 8 weeks of aerobic exercise did not change body mass index ($p<0.05$).

Changes of BMI contrasts with reports of Wing and colleagues (2000), Cecil (3) and colleagues (2001), Moina Et al (2002), and with studies of Lehmann and colleagues (2001), Tanigochi et al (2004) is consistent. These differences are probably individual differences in the control diet and anthropometric subjects. Despite the reduction in body fat, increased muscle mass and weight seem to have replaced the fat weight that way, body mass index did not significantly change subjects. Furthermore, studies have shown that patients treated with the drug compared to patients treated with sulfonylureas and metformin, BMI is lower (6).

3.7. Aerobic exercise training for 8 weeks did not change waist-hip ratio ($p>0.05$).

Changes of WHR in this study with the reports of Mayorna and colleagues (2002), Dunn Stan and colleagues (2002), Christose and colleagues (2003) are inconsistent, and with observations of Bruce and colleagues (2004) are consistent. However, it can be concluded that the reduction in fat and internal fat reserves more effective actions, and visceral fat, abdominal circumference and hip circumference were used. Because of these changes in exercise duration and lack of control groups can be attributed to their diet.

3.8. Aerobic exercise training for 8 weeks significantly increased maximal oxygen consumption ($p<0.05$)

Changes of VO2max with the results of Moina (2002), Mayorna and colleagues (2002), Bruce et al (2004), Mehdi Esfahani (1381), and Muhammad Ali Sardar (1384) is consistent, and with reports of Kaffed et al (2003) is inconsistent. The effect of aerobic exercise increased the number of muscle capillaries and muscle cross-sectional area of the muscle which leads to better blood supply. Also, the number and size of skeletal muscle mitochondrial oxidative metabolism increases and the possibility of improving muscle oxidative enzyme is markedly increased. As a result, these change an increase in muscle aerobic capacity.

4. Conclusions

In fact, adaptation caused by aerobic exercise, primarily in response to exercise in diabetic patients, which include the following:

1. Increased vascular density of muscle
2. Improvement in maximum oxygen consumption
3. Increase in skeletal muscle oxidative enzymes

Aerobic exercise, increases insulin sensitivity and the person with improved insulin sensitivity correlated inversely with leptin, we can say that aerobic exercise leads the decline in leptin (2).

In summary, this study showed that after 8 weeks of aerobic exercise training in diabetic patients of type II on serum leptin, and cardiovascular risk factors, particularly blood pressure and HDL has an impact. On the other hand, a number of studies examining the short-term training courses less than 12 weeks, showed no effect on the density of the leptin has not reported unless it is associated with fat loss. The results showed that aerobic exercise reduces fat and increases VO2max to significant level, but on triglycerides, cholesterol, HDL - BMI and WHR has no significant effect. These changes were associated with fitness level subjects, and the results indicate that this type of exercise on diabetic type II men is effective. Thomas Fritz and colleagues (2005) concluded that the research on patients with a frequency of three times a week to walk a significant effect on total cholesterol and triglycerides puts these people. It seems TG/LDL adverse effects on the walls of the arteries and can lead to accelerated atherosclerosis, exercise alone will not reduce but can be regulated by exercise intensity, duration and frequency of appropriate quantitative decreased risk of heart disease just stop. But it is noteworthy that the effect of physical exercise on triglyceride concentrations is lower than the LDL. Between TG/LDL diabetes in middle-aged men with diabetes compared with inactive there is no significant difference. So, very little difference in TG/LDL of the men was off. Peter Wood (1988) argues that the exercise will cause weight loss, LDL and reduced total cholesterol but normal or non-impact workout in the weight remains on the practice of blood serum is ineffective. VLDL-LDL analysis related to the development of platelets to prevent atherosclerosis. Indeed HDL depletion of cholesterol from peripheral tissues directly or by exchange with a very low density lipoprotein to liver transparency. But we know that TG/HDL makes a risk factor for cardiovascular disease-related factors and cholesterol and HDL. Thus with proper exercise can lower cholesterol levels and HDL increases. It helps the proportion of TG/HDL to be reduced. According to the results, possibly with the addition of exercise along with diet and medication, further changes will be seen. Insulin resistance is also associated with subcutaneous fat. Common causes of obesity and physical inactivity, resulting in insulin resistance, which reduces fat through aerobic exercise can improve insulin resistance. In the end, this type of research for diabetics type II, regular aerobic exercise on a treadmill or stationary bicycle with an intensity of 60 to 70% of maximum heart rate is recommended. Use the treadmill and stationary bike, while avoiding many sports injuries, good control of diabetes and cardiovascular disease during exercise. Drug therapy is recommended, along with regular aerobic exercise.

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