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Effects of aerobic training on the glycemic control and body composition in obese patients with type 2 diabetes

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ABSTRACT

This study was performed to investigate the effects of 10 weeks of aerobic training on glycemic control and body composition in obese patients with type 2 diabetes. Twenty one males with type 2 diabetes (40 to 50 years old) were randomly assigned to the training (n=11) and control (n=10) groups. The training group participated in an aerobic training program (three sessions per week and 45-60 min per session) for 10 weeks with the intensity of 50-65% of heart rate reserve (HRR). Weight, body mass index (BMI), percentage of body fat, glucose, insulin, hemoglobin A1c (HbA1c) and insulin resistance were measured in both groups before and after training. Ten weeks of aerobic training resulted in a significant decrease in weight, BMI, percentage of body fat, glucose, HbA1c and insulin resistance in the training group while no significant change was observed in the serum concentrations of insulin. The control group did not demonstrate any significant changes in the measured variables. There was a significant relationship between change in percentage of body fat and changes in glucose and insulin resistance. This study showed that 10 weeks of moderate intensity aerobic training can improve glycemic control and body composition in obese patients with type 2 diabetes. Our findings suggest that the improvement in glycemic control with aerobic training is associated with a decrease in percentage of body fat in these patients.

Key Words: Aerobic Training, Glycemic Control, Body Composition.

INTRODUCTION

Type 2 diabetes is a chronic metabolic disease which its prevalence is increasing rapidly throughout the world. It has been estimated that the number of diabetic patients will reach about 333 million people by 2025, 90-95% of patients will have type 2 diabetic [1]. Chronic diabetic hyperglycemia is associated with the long-term complications of various organs, especially damage to eyes, kidneys, nerves, heart and blood vessels [2].

On the other hand, obesity has been known as a major risk factor for type 2 diabetes [3] and on average, 1-kilogram increase in weight is related to 9% relative increase in prevalence of diabetes [4]. Obesity, particularly abdominal obesity, is associated with abnormalities such as insulin resistance, hyperinsulinemia, hyperglycemia, dyslipidemia and hypertension [5]. Research evidence has shown that 80-90% of diabetic patients are overweight or obese [6] and obesity worsens the diabetes-related physiologic and metabolic abnormalities, specifically hyperglycemia, hyperlipidemia and hypertension [7]. Weight loss is very important for improving glycemic control and for decreasing other risks related to obesity and diabetes [8]. Moreover, it has been proved that intentional weight loss is

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associated with the reductions in the mortality of overweight diabetic patients [9]. Therefore, decreasing hyperglycemia and body fat are two of the important goals of diabetes therapy [5].

Physical exercise along with diet and medication is considered as one of the cornerstones of type2 diabetes management [10] and preventing and treating obesity [11]. Aerobic training is a beneficial training method for decreasing hyperglycemia, which is more efficient than resistance training in improving body composition because of the relationship between weight loss and energy expenditure [12]. American Diabetes Association recommended 150 min aerobic physical activity with moderate intensity or 90 min vigorous aerobic exercise per week for diabetic patients in order for them to improve glycemic control, maintain body weight and decrease risk of cardiovascular diseases [10]. Although the effects of regular physical exercises, especially aerobic exercises, on the glycemic control in patients with type2 diabetes have been investigated in many studies, the reported results are not consistent. Some studies have reported improvement [13-16] and some others have reported no significant change [17-19] in the glycemic control in these patients in response to aerobic training. Moreover, as far as effects of aerobic training on the weight loss and body fat decrease in diabetic patients are concerned, heterogeneous findings have been reported [14, 15, 18, 20]. Additionally, reduction in hyperglycemia and insulin resistance in response to exercise training in obese patients with type 2 diabetic indicates a direct relationship between decrease in body fat and improvement in glycemic control [12]. However, some studies have stated that improvement in glycemic control is independent from the decrease in body fat [12]. Therefore, the effects of exercise training on glycemic control whether dependent on or independent from body fat are controversial. The present study was performed, first, to investigate the effects of aerobic training on indices of glycemic control (glucose, HbA1c, insulin resistance) and also indices of body composition (weight, BMI, percentage of body fat) and, second, to determine the relationship between changes in indices of glycemic control and change in body fat in response to an aerobic training program in obese patients with type 2 diabetic.

MATERIALS AND METHODS

Participants

Twenty one obese males with type 2 diabetes (with body mass index of 30.6 kg/m^2) voluntarily participated in this study. The participants were aged between 40 and 50, and their duration of disease was less than ten years. They were not participating in regular physical activities during the last year at the time of study and none of the patients were underwent diet therapy. The patients who were smoker, had complications of diabetes or other chronic diseases, and were receiving insulin were excluded. The study protocol was approved by ethics committee of Islamic Azad University, Central Tehran Branch. Consents forms were signed by the participants after they had been familiarized with the objectives and methods of the research. Then, they were randomly assigned to two aerobic training (n=11) and control (n=10) groups. The subjects were advised not to change their usual diet during the study. The descriptive characteristics of the participants in both groups are given in Table 1.

	Training group (n=11)	Control group (n=10)
Age (years)	46.36±3.3	45.8 ±3.2
Duration of diabetes (years)	4.18±2.2	3.9±2.2
Height (cm)	172.63±5.7	171.7±5.3
Weight (kg)	91.37±7.5	89.87±7.9
BMI (kg/m ²)	30.69±2.5	30.5 ± 2.6
Body fat (%)	29.16±4	28.85±2.9
Vo2max (ml.kg ⁻¹ .min ⁻¹)	25.4±3.9	25.1±3.2

Table1: General characteristics of study subjects

Values are mean $\pm SD$

Training program

The subjects of the training group participated in an aerobic training program (three sessions per week and 45-60 min per session) for 10 weeks. Each session of exercise included 10 min warm-up, 25-40 min walking or running with the intensity of 50-65% of heart rate reserve and 10 min cool down. The main part of the training started with the intensity of 50% of heart rate reserve for 25 min and gradually progressed to 65% of heart rate reserve for 40 minutes in the next sessions. Heart rates of subjects were monitored using heart rate monitors (Polar Electro, Oy, Finland).

Assessment of Body composition and VO2max

The body composition was measured with the use of a body-composition analyzer (Omron, Finland), and the VO2max was also predicted by YMCA cycle test, using a cycle ergometer (Tunturi E604, Finland).

Blood Sampling and Biochemical Measurements

To evaluate biochemical variables, blood samples were taken after 12 h overnight fast from the antecubital vein at baseline and at the end of the study. The serum was separated by centrifugation and stored at -80 °C for subsequent analysis. Fasting blood glucose and HbA1c were measured by glucose oxidase method (Pars Azmon, Iran) and the chromatography method (Biosystem, Spain), respectively. Serum insulin was measured by ELISA (demeditec, Germany); the intra- and inter-assay coefficients of variation were 2.6 and 2.88% respectively. Insulin resistance was calculated using the levels of glucose and fasting insulin by HOMA-IR formula [21].

Statistical analysis

The normality distribution of data was evaluated using Kolmogorov-Smirnov test. Paired t-test and Independent ttest were used for evaluating changes within the two groups and comparing changes between two groups, respectively. The relationship between changes in indices of glycemic control and change in percentage of body fat was evaluated by Pearson's correlation coefficient. P values less than 0.05 were considered statistically significant. The statistical analyses were performed using SPSS 15 software.

RESULTS

At the baseline, there were no significant differences in age, weight, body mass index, percentage of body fat, VO2max, duration of diabetes and indices of glycemic control between two groups (p > 0.05).

The comparison between the values obtained before and after training using the paired t-test showed a significant increase in VO2max ($p \le 0.001$) and a significant reduction in weight (p = 0.007), body mass index (p = 0.009), percentage of body fat ($p \le 0.001$), fasting blood glucose (p = 0.002), HbA1c ($p \le 0.001$) and insulin resistance (p = 0.039) in the training group. However, insulin level did not significantly change in the training group (p = 0.28) (Table 2). There were no significant differences between the pre-test and post-test values obtained from the control group (p > 0.05). Moreover, the independent t-test showed that changes in Vo2max ($p \le 0.001$), weight (p = 0.002), body mass index (p = 0.004), percentage of body fat ($p \le 0.001$), glucose (p = 0.001), HbA1c ($p \le 0.001$) and insulin resistance (p = 0.013) in the training group were significantly different from those in the control group. However, the comparison of the changes in insulin between the two groups demonstrated no significant difference (p = 0.18).

After the training, a significant relationship revealed between the change in percentage of body fat and changes in glucose (r = 0.63, P = 0.019) and insulin resistance (r = 0.54, P = 0.043) in the training group. But, the relationship between the change in percentage of body fat and change in HbA1c (r = 0.47, P = 0.071) was not significant (Table 3).

	Training group		Control group	
Variable	Before training	After training	Before training	After training
Weight (kg)	91.37 ±7.5	89.32 ±8.6*	89.87±7.9	90.18 ± 7.9
BMI (kg/m ²)	30.69 ± 2.5	29.98± 2.7*	30.5 ± 2.6	30.59 ± 2.6
Body fat (%)	29.16±4	27.22±4.5*	28.85±2.9	29.03±2.8
FBS (mg/dl)	203.54±36.5	174.6±26.9*	196.6± 34.9	202.2 ± 32.2
insulin (µIU/ml)	8.73±1.7	7.47 ± 3.6	9.23 ± 2	9.58 ± 2.2
HbA1c (%)	8.86 ± 0.7	8.25±0.7*	8.62 ± 0.9	8.71 ± 0.8
HOMA-IR	4.31±0.9	3.19±1.5*	4.45 ± 1.2	4.8± 1.4
V_0^2 max (ml kg ⁻¹ min ⁻¹)	25 4+ 3 9	31 9+ 4 5*	25 1+3 2	255 + 24

Table2: body composition and glycemic variables in before and after training

Values are mean \pm SD. *P<0.05 vs. before training

Index	R	Р	Result		
Glucose	0.63	0.019	t		
Insulin Resistance	0.54	0.043	Ť		
HbA1c	0.47	0.071			
R, Coefficient of correlation					

Table3: Correlation between change in Body fat (%) and changes in Indices of glycemic control

R, Coefficient of correlation P, Level of signification † Significant correlation (p<0.05)

DISCUSSION

In the present study, the significant decreases in fasting blood glucose, HbA1c, insulin resistance and the decreasing tendency in blood insulin indicated that 10 weeks of aerobic training with the moderate intensity had a beneficial effect on the glycemic control in obese men with type 2 diabetic. In parallel with these findings, previous studies by measurement of blood glucose, HbA1c and insulin resistance have reported that exercise training can improve glycemic control in type 2 diabetes [15, 22]. At the same time, studies which have used training with low intensity or volume have not reported any improvement in glycemic control [12]. In the present study, the 28.9 mg/dl reduction in blood glucose was accompanied by 0.61% decrease in HbA1c. Research evidence has shown that exercise training decreases HbA1c by approximately 0.66% which can decrease the risk of diabetic complications [5]. Moreover, decrease in HbA1c is associated with reduction in the cardiovascular risk in type 2 diabetes; for instance, the 0.6% decrease in HbA1c caused by aerobic exercise is associated with a 22% decrease in microvascular complications and an 8% decrease in myocardial infarction rate [23]. These results indicated the important role of aerobic exercise in improving glycemic control and decreasing hyperglycemia complications in type 2 diabetic patients. Muscular contractions can stimulate glucose transport into skeletal muscles by an insulinindependent mechanism [24, 25]. Furthermore, increased blood flow to exercising muscles can facilitate delivery of glucose to the muscles [24]. The aerobic training program used in this study caused 26% significant reduction in insulin resistance. These results may indicate the beneficial effect of aerobic training on insulin action in these patients and are in correspondence with the results of previous studies which demonstrated decrease in insulin resistance after aerobic training in type 2 diabetic patients [15, 22]. Exercise training can lead to the increase in insulin sensitivity by several mechanisms include increased glucose transporter protein (GLUT4) expression, increased glycogen synthesis activity [26] and increased lipid oxidation [27].

In the present study, the improvement in indices of glycemic control in the training group was accompanied by the improvement in their body composition. As after 10 weeks of aerobic training with moderate intensity, weight, BMI and percentage of body fat in these patients significantly decreased. Similarly, beneficial effects of exercise training on weight loss and body composition in type 2 diabetic patients have been confirmed in previous studies [14, 20]. However, some studies have not demonstrated significant changes in the indices of body composition [15, 16, 18, 28]. Contradiction of these findings may be because of using different methods for evaluating body composition (BMI, weight or body fat mass), different training methods (aerobic versus resistance) and also using or not using dietary supplements along with exercise training [12,29]. One of the most important findings of this study was the significant relationship between change in percentage of body fat and change in HbA1c was not statistically significant (probably due to small sample size). Although some studies have shown that improvement in glycemic control in type 2 diabetic patients after exercise training is independent from the decrease in body fat [12, 15], the findings of this study may suggest that aerobic training-induced improvements in indices of glycemic control (especially blood glucose and insulin resistance) in obese patients with type 2 diabetic depended on the decrease in their body fat. Nevertheless, a definitive statement on this issue requires further studies.

CONCLUSION

The results of our study show that aerobic exercises such as walking and running improve glycemic control and body composition in obese patients with type 2 diabetic. These findings suggested that improvement in glycemic control with aerobic training is associated with a decrease in percentage of body fat in these patients.

REFERENCES

[1] Horton ES. *Obesity* **2009**; 17 Suppl 3:S43-48.

[2] The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (Position Statement). *Diabetes Care* **2003**; 26:S5–S20.

[3] Hollander P. Diabetes Spectrum 2007; 20:159-65.

[4] Norris SL, Zhang X, Avenell A, Gregg E, Bowman B, Serdula M, Brown TJ, Schmid CH, Lau J. Am J Med 2004; 15; 117:762-74.

[5] Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. JAMA 2001; 286:1218-27.

[6] Kyou I, Kumar S. British Journal of Diabetes & Vascular Disease 2010; 10: 274-283.

[7] Norris SL, Zhang X, Avenell A, Gregg E, Schmid CH, Kim C, Lau J. Arch Intern Med 2004; 164:1395-1404.

- [8] Hensrud DD. Obesity Research 2001; 9: 348S-353S.
- [9] Williamson DF, Thompson TJ, Thun M, Flanders D, Pamuk E, Byers T. Diabetes Care 2000; 23:1499-1504.

[10] Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C, White RD. *Diabetes Care* **2006**; 29: 1433-1438. [11] Hill JO, Wyatt HR. *J Appl Physiol* **2005**; 99:765-770.

[12] Marwick TH, Hordern MD, Miller T, Chyun DA, Bertoni AG, Blumenthal RS, Philippides G, Rocchini A. *Circulation* **2009**; 119: 3244–3262.

[13] Sigal RJ, Kenny GP, Boulé NG, Wells GA, Prud'homme D, Fortier M, Reid RD, Tulloch H, Coyle D, Phillips P, Jennings A, Jaffey J. *Ann Intern Med* **2007**; 147: 357–369.

[14] Cuff DJ, Meneilly GS, Martin A, Ignaszewski A, Tildesley HD, Frohlich JJ. *Diabetes Care* 2003; 26: 2977–2982.

[15] Kadoglou NP, Perrea D, Iliadis F, Angelopoulou N, Liapis C, Alevizos M. Diabetes Care 2007; 30: 719-721.

[16] Kadoglou NP, Iliadis F, Angelopoulou N, Perrea D, Ampatzidis G, Liapis CD, Alevizos M. *Eur J Cardiovasc Prev Rehabil* **2007**; 14: 837–843.

[17] Fritz T, Wändell P, Aberg H, Engfeldt P. Diabetes Res Clin Pract 2006; 71: 21-27.

[18] Poirier P, Tremblay A, Broderick T, Catellier C, Tancrède G, Nadeau A. Med Sci Monit 2002; 8: CR59-65.

[19] Khan S, Rupp J, J Sports Med Phys Fitness. 1995; 35: 281–288.

[20] Nojima H, Watanabe H, Yamane K, Kitahara Y, Sekikawa K, Yamamoto H, Yokoyama A, Inamizu T, Asahara T, Kohno N; *Metabolism* **2008**; 57: 170–176.

[21] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. *Diabetologia* 1985; 28: 412-419.

[22] Yokoyama H, Emoto M, Araki T, Fujiwara S, Motoyama K, Morioka T, Koyama H, Shoji T, Okuno Y, Nishizawa Y. *Diabetes Care* **2004**; 27: 1756-8.

[23] Chudyk A, Petrella RJ. Diabetes Care 2011; 34: 1228-1237.

[24] Gulve EA. Phys Ther 2008; 88: 1297-1321.

[25] Henriksen EJ. Journal of Applied Physiology 2002; 39: 2788-796.

[26] Christ-Roberts CY, Pratipanawatr T, Pratipanawatr W, Berria R, Belfort R, Kashyap S, Mandarino LJ. *Metabolism* **2004**; 53:1233-42.

[27] Hawley JA, Lessard SJ. Acta Physiol 2008; 192: 127–135.

[28] Castaneda C, Layne JE, Munoz-Orians L, Gordon PL, Walsmith J, Foldvari M, Roubenoff R, Tucker KL, Nelson ME. *Diabetes Care* **2002**; 25: 2335–2341.

[29] Akbarpour, M., Beni , Mohsen Assarzadeh, Hamid Sadeghian, J Annals of Biological Research, 2011. 2(6): p. 123-129.