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Original Article

Effects of passive static stretching on blood glucose levels in patients with type 2 diabetes mellitus

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Abstract. [Purpose] This study determined the effects of passive static stretching on blood glucose levels in patients with type 2 diabetes. [Subjects] Fifteen patients (8 males and 7 females) with type 2 diabetes were recruited and randomly assigned to the control group or passive static stretching group. [Methods] Glycated hemoglobin was measured before and after the 8-week training period. [Results] Glycated hemoglobin levels decreased significantly in the passive static stretching group, and there were significant differences in blood glucose levels between the 2 groups. [Conclusion] Passive static stretching of the skeletal muscles may be an alternative to exercise to help regulate blood glucose levels in diabetes patients.

Key words: Passive static stretching, Blood glucose level, Type 2 diabetes

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INTRODUCTION

The incidence of diabetes mellitus is increasing worldwide; this trend is particularly strong for type 2 diabetes mellitus (T2DM)[1)]. T2DM is a chronic disease characterized by decreased insulin sensitivity and overall poor glucose control. Exercise is a generally accepted component of the nonpharmacologic treatment for T2DM[2)]. The systematic review by Boule et al. indicates structured exercise programs have a statistically and clinically significant beneficial effect on glycemic control in patients with T2DM[3)]. In addition, patients with T2DM who use insulin, low-intensity exercise significantly reduces the prevalence of hyperglycemia[4)].

Despite the benefits of physical activity, many people with T2DM do not exercise regularly. For some individuals, secondary diabetes-related complications such as lower-limb amputations, neuropathies, hypertension, nephropathies, and retinopathies can either contraindicate exercise or make it more difficult. In addition, many elderly people with T2DM do not have sufficient physical ability to perform aerobic exercise and thus have problems maintaining euglycemia[5)].

Passive static stretching occurs when sustained tension develops within a person's muscles through external forces. Several studies suggest passive stretching can increase cellular glucose uptake. Accordingly, blood glucose levels could decrease following a program of successive sustained muscle stretching. In addition, because passive stretching requires minimum effort by the person performing the stretch, people with T2DM who are reluctant or unable to exercise may be willing to follow a stretching protocol[6)]. Therefore, this study determined the effect of passive static stretching on blood glucose levels in people with T2DM.

SUBJECTS AND METHODS

Fifteen in-patients with T2DM at a hospital in Busan, Korea participated in this study. Patients were eligible if they were sedentary (i.e., not participating in regular aerobic or strengthening exercises 6 months before the study) and willing to commit to an 8-week supervised exercise program[7)]. All patients were diagnosed with T2DM confirmed by a glycated hemoglobin (HbA1c) level 6.5% or higher as a criterion for the diagnosis of diabetes[8)]. All patients meeting the inclusion criteria were given verbal and written information about this study. The patients provided informed consent prior to participating. The study protocol was approved by the Ethics Committee of Daegu University.

Patients were randomized to the control group (n = 7) or passive static stretching group (PSS, n = 8). The control group was instructed to maintain their diet and medications for diabetes and not to perform any exercise during the experiment. Meanwhile, patients in the PSS group followed the same instructions as the control group but received a 40-minute intervention consisting of 6 lower-body and 4 upper-body static passive stretches. For each stretch, the muscle was held in the stretched position for 30 seconds and was repeated 4 times. Each repetition was separated by a 15-second relaxation period, and different stretches were separated by a minimum of 1 minute. A description of stretch is provided in Table 1[6)]. The PSS group performed the stretches 3 times per week for 8 weeks.

For outcome measurements, a 10-mL blood sample was

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Table 1. Descriptions of the stretches used in the intervention

Stretch	Description
Seated knee flexor (bilateral)	The patient sat on the floor with their legs extended and arms above their head. From this position, they lowered their head toward their knees while the experimenter pushed down on their back.
Seated knee flexor–hip adductor (bilateral)	The patient sat on the floor in the cross-legged position. From this position, the patient lowered their head toward the floor while the experimenter pushed down on their back.
Seated shoulder lateral flexor (bilateral)	The patient sat in a chair with fingers interlaced behind their head. Keeping their arms in this position, the experimenter stood behind the patient and pulled the elbows back toward the body's midline.
Supine hip flexor–knee extensor (unilateral)	The patient lay on their back with their leg hanging over the edge of the table with the knee flexed at approximately 90°. The hip was then hyperextended by the experimenter while pushing down on the thigh.
Seated hip external rotators, extensors (unilateral)	The patient sat on the floor with one leg extended. The opposite leg was flexed at the knee, and the foot was placed flat against the extended leg's inner thigh. The patient then lowered their head toward the extended knee while the experimenter pushed down on their back.
Seated shoulder extensors, adductors, retractors (unilateral)	While seated in a chair, the patient extended one arm and placed it horizontally across the front of the chest. The experimenter stood behind the patient, grabbed their wrist, and pulled their arm against the chest as much as possible while keeping the arm parallel to the floor.
Supine knee flexor–plantar flexor (unilateral)	The patient lay on their back with the legs extended. The experimenter then raised one leg and simultaneously flexed the hip and dorsiflexed the ankle.
Prone hip flexor (unilateral)	The patient lay on their stomach and flexed one knee at approximately 60°. Keeping the knee in the flexed position, the experimenter lifted the thigh to hyperextend the hip.
Seated shoulder flexors, depressors (bilateral)	The patient sat on the floor with the legs extended. The experimenter then grabbed their wrists and hyperextended the shoulder by raising the arms behind the back and up toward the head while keeping the back and elbows straight.
Seated shoulder and elbow flexors (unilateral)	The patient sat on the floor with the legs extended, with one elbow flexed and brought up near the ear. From this position, the shoulder was hyperflexed by the experimenter by pushing the upper arm down toward the floor.

Table 2. Baseline characteristics of the patients

	CON (n = 7)	PSS (n = 8)	p
Age (years)	58.4 ± 1.8	49.6 ± 5.2	0.2
Duration of diabetes (years)	5.2 ± 2.9	5.4 ± 1.5	0.5
BMI (kg/m²)	24.9 ± 3.0	26.9 ± 4.1	0.7

Values are means ± SD.
CON: control group; PSS: passive static stretching group; BMI: body mass index

collected from each patient to determine blood glucose levels using an HbA1c analyzer (VARIANT™ α TURBO, Bio-Rad Laboratories, Inc., CA, USA). HbA1c values were obtained at baseline and after the 8-week intervention. Statistical analyses were performed using SPSS version 12.0. A paired t-test was used to determine whether there were significant changes in blood glucose levels before and after the intervention. Meanwhile, an independent t-test was used to analyze differences between the 2 groups. The level of significance was set at $p < 0.05$. The results are expressed as mean ± standard deviation (SD).

RESULTS

The baseline characteristics of the patients are shown in Table 2. There were no significant differences in the baseline characteristics between groups ($p > 0.05$). The results of outcome measures are summarized in Table 3. There was no significant difference in HbA1c level after the intervention in the control group ($p > 0.05$). However, HbA1c levels decreased significantly in the PSS group after the intervention

Table 3. Outcome measures

	Control group (n = 7)		Passive static stretching group (n = 8)	
	Pre-intervention	Post-intervention	Pre-intervention	Post-intervention
HbA1c (%)	7.4 ± 1.3	7.4 ± 1.4	7.4 ± 1.5	6.8 ± 1.5*†

Values are means ± SD.
*$p < 0.05$ vs. post-intervention. †$p < 0.05$ vs. control group post-intervention.
HbA1c: glycated hemoglobin A1c.

($p < 0.05$) and were significantly different between groups ($p < 0.05$).

DISCUSSION

As mentioned above, this study determined the effect of passive static stretching on blood glucose levels in patients with T2DM. The results showed HbA1c levels decreased significantly after an 8-week passive static stretching intervention. There are several possible mechanisms that could explain how passive stretching of skeletal muscles decreased blood glucose levels. According to a review by Dohm[9], glucose transport into the skeletal muscles is primarily mediated by a glucose transport protein, GLUT-4; accordingly, exercise can increase GLUT-4 levels in the skeletal muscles. Furthermore, increased metabolic activity accompanying passive muscle stretching is related to the GLUT-4 activation pathway[9, 10]. Therefore, passive muscle stretching could induce the incorporation of GLUT-4 into the stretched skeletal muscles.

Other studies also support the possibility of stretching-induced incorporation of GLUT-4 into the skeletal muscles. First, the activity of protein kinase B controls GLUT-4 incorporation; accordingly, protein kinase B is activated by passive stretching of isolated muscles[11]. Second, Sun et al. report that ischemia induces the translocation of GLUT-4 to the plasma membrane of cardiac myocytes[12]; accordingly, passive stretching of the skeletal muscles can cause ischemia[13]. Third, in an experimental study by Roberts et al., exercise-induced increases in nitric oxide levels resulted in increased glucose transport[14]; accordingly, passive stretching can increase nitric oxide release from excised soleus muscles by 20%[15]. Finally, mitogen-activated protein kinase activity stimulates glucose uptake in muscle cells[16]; Martineau et al. report that the activity of mitogen-activated protein kinase directly reflects the magnitude of mechanical stress (e.g., actively or passively generated tension) applied to the muscle[17].

The results of this study are subject to several limitations. The sample size is insufficient to generalize the results to all patients with T2DM. In addition, as HbA1c reflects the average plasma glucose level over the preceding 2–3 months, the 8-week study period might have been too short to determine changes in blood glucose levels as a result of stretching[18]. Therefore, further studies are required to ascertain the long-term (i.e., more than 3 months) effects of passive static stretching on blood glucose levels in a larger population of patients with T2DM.

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