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Antioxidant, Free Radical, and Lactate Levels in Patients with Low Back Pain

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Abstract. [Purpose] The aim of this study was to compare blood levels of antioxidants, malonaldehyde, and lactate between patients with low back pain and healthy controls. [Subjects] Ten patients with at least 6 months of low back pain and 10 healthy adult controls were recruited for this study. [Methods] To measure superoxide dismutase, glutathione peroxidase, malonaldehyde, and lactate levels on the basis of exercise intensity, we conducted a graded exercise test. Blood (20 mL) was collected 3 times (at rest and peak exercise, and in the recovery period), and analyzed. [Results] During peak exercise, levels of superoxide dismutase, glutathione peroxidase, malonaldehyde, and lactate in patients with low back pain were significantly different from those of controls. There were no significant differences between the low back pain and control subjects during the resting and recovery periods. [Conclusion] Reduced physical activity in patients with low back pain increases free radical capacity, resulting in increased antioxidant capacity and higher lactate levels. Therefore, physical therapists treating patients with low back pain must consider these characteristics and educate patients about them.
Key words: Antioxidant, Low back pain, Lactate level

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INTRODUCTION

Low back pain (LBP), with a lifetime prevalence of 60%–85%, is a major public health problem in industrialized societies[1, 2)]. The symptoms of LBP range from pain and decreased trunk flexibility and strength, to sciatica[3, 4)]. Low back pain also limits sufferers' one's social participation and decreases aerobic capacity by reducing their physical activity levels[5)].

During physical activity, oxygen is used to produce energy in the electron transport system inside mitochondria[6)]. However, when oxygen in its normal state generally absorbs two unpaired electrons, an active reaction is generated to absorb electrons from the outside. The free radicals that are produced in this process are responsible for a host of adverse effects inside the human body[7)]. There are various types of free radicals such as superoxide radical anion, hydrogen peroxide, and the hydroxyl radical, and they are continuously produced in the human body incomplete reduction of oxygen. When their concentrations exceed intracellular reduction ability, the cell membrane and DNA can be injured[8)]. This DNA injury reduces the function of the electron transport system and oxygen consumption, and mitochondria are damaged by DNA injury resulting in decrease in physical activity level[9)]. Antioxidant enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GPX) protect proteins, nucleic acids, and membranes from being damaged by free radicals[10)].

LBP is a typical disorder that reduces physical activity. Therefore, LBP can cause musculoskeletal disorders and a decrease in aerobic capacity. As cardiopulmonary rehabilitation is currently used as a type of physical therapy, research is necessary into the changes in aerobic capacity caused by physical activity. However, such research has not been conducted. Therefore, we aimed at obtaining new information to help physical therapists provide their patients with appropriate information on this issue. To achieve our aim, we compared the antioxidant, malonaldehyde (MDA), and lactate levels in 10 patients with LBP with those of 10 healthy controls (CON).

SUBJECTS AND METHODS

This research was conducted at the Metropolitan Hospital of Korea in June of 2011. The LBP group included 10 male patients with at least 6 months of LBP; the control group consisted of 10 healthy male adults. The onset period of the LBP group was 11.42 ± 3.28 month (mean ± standard

Table 1. Physical characteristics

Group	Age (year)	Height (cm)	Weight (kg)
LBP (N=10)	30.2 ± 4.18	173.6 ± 4.72	73.8 ± 6.56
CON (N=10)	30.6 ± 4.99	174.9 ± 4.23	73.6 ± 4.93

Note. All variables are mean ± standard deviation

deviation). Subjects' physical characteristics are summarized in Table 1.

We explained the purpose and procedures of the experiment to the participants. Subjects in both groups fasted for 12 h before blood testing. Blood (20 mL) was collected at rest, at peak exercise, and in the recovery period. From the collected blood samples, we extracted and analyzed antioxidant enzymes (SOD and GPX), free radical predictor (MDA), and lactate levels. To measure SOD, GPX, MDA, and lactate levels based on exercise intensity, we used a Q-stress^{TM}55 (QUINTON 2000; USA) treadmill and conducted a graded exercise test. First, we collected blood from each participant while he or she was seated on a chair on the treadmill (resting period). Later, using the Bruce protocol[11], we increased the treadmill's velocity and slope and conducted exercise at 90% of the calculated maximum heart rate (220 − age). When that point was reached, blood was collected immediately as the peak exercise period sample. Finally, the treadmill was stopped, and participants lay down on a bed. After 3 min, blood was collected for recovery period data.

To analyze the levels of SOD, 600 μL of whole blood was collected in a heparin-treated vacutainer and shaken thoroughly. It was then centrifuged for 5 min at 2500 *g* (4 °C) to obtain plasma. Then, 400 μL of ice-cold extraction reagent was added to 250 μL of the obtained plasma, and it was vortex-mixed for 30 s. Within 15 s, the optical density of the supernatant was measured at 525 nm for 1 min. The method used for GPX analysis was identical to that used for SOD analysis. The collected supernatant fluid was removed to obtain red blood cells. Cold 6% metaphosphoric acid (0–4 °C) was mixed with the red blood cells, and the mixture was centrifuged at 3000 *g* (4 °C) for 10 min. Finally, the optical density was measured at 400 nm.

For the MDA analysis, 20 mL of blood was collected, then 3 mL of whole blood was treated with heparin and centrifuged for 10 min at 3000 rpm. The resulting precipitate was added to a tube with plasma to dilute the reagent. It was then heated for 15 min in a water bath and, after cooling, was centrifuged for 10 min at 1000 rpm. Two milliliters of the centrifuged plasma was placed in a curette and analyzed using a spectrophotometer. Lactate levels were analyzed using BM Lactate test strips and an analyzer.

SPSS Version 12.0 was used to calculate the mean and standard deviation. Descriptive statistics were used to analyze the general characteristics of the subjects. Differences in SOD, GPX, MDA, and lactate levels between the 2 groups at resting, peak exercise, and recovery were tested using the independent t-test. For all data, statistical significance was accepted at values of $p<0.05$.

RESULTS

The differences in blood levels of SOD, GPX, MDA, and lactate between the LBP group and the control group are shown in Table 2. During peak exercise, all the values of all the items measured significantly differed between the LBP group and controls ($p<0.05$). There were no significant differences in the levels of the resting and recovery periods.

DISCUSSION

This research was conducted to gain knowledge about antioxidant, MDA, and lactate levels in patients with LBP. Antioxidants protect cells from free radicals that damage the cell. They remove free radicals from serum that lacks albumin and other macromolecules. Oxidative stress caused by free radicals is neutralized by antioxidants; this is one of the mechanisms by which homeostasis is maintained[12].

Free radicals mainly invade the mitochondria. Their levels increase under circumstances of vigorous activity, smoking, and extremes of environment[13]. They also show pronounced effects in individuals with disease or a sedentary life style[14].

The results of this experiment show that, in patients with LBP, all the measured items significantly increased peak exercise as compared to the controls. Previous studies have reported that antioxidant capacity does not vary with age but that total antioxidant capacity decreases as aerobic capacity increases[15]. This is because reduced physical activity increases free radical-scavenging activity, resulting in increase of antioxidant capacity. The subjects of this experiment had been in pain for more than 6 months and had not engaged in much physical activity such as walking for more than 20 minutes or climbing stairs. Their aerobic capacity had decreased due to long-term bed rest. They showed an increase the level of the free radical predictor MDA, causing increases in SOD and GPX levels as well. This result is consistent with the findings of previous studies. Our study revealed significantly different results at peak exercise, indicating that during high physical activity, the difference in the levels will be greater as in LBP sufferers than inhealthy adults. Our results suggest that people with low aerobic capacity will experience greater oxidative stress due to free radicals, causing possible harm to the cardiac proteome[16].

Overweight, obesity, high blood pressure, unfavorable blood lipid profile, and especially higher uric acid levels are associated with higher levels of antioxidants in blood serum in otherwise healthy adults. High physical activity is associated with a more favorable overall risk profile for cardiovascular and metabolic diseases, and it is also associated with lower antioxidant levels.

This experiment found that lactate levels in patients with LBP were higher than in healthy adults during exercise. Physiologically, as physical activity increases, lactate level increases. During the experiment, lactate was at its highest level at peak exercise[17]. In addition, bed rest longer than 3 days increases LL, and individuals with a sedentary lifestyle have higher lactate levels than individuals who participate

Table 2. Comparison of SOD, GPX, MDA and lactate (LAC) levels between the low back pain and control groups

	LBP				CON			
	SOD	GPX	MDA	LAC	SOD	GPX	MDA	LAC
Resting	920.50 ± 99.83	21.75 ± 3.39	0.43 ± 0.22	115.30 ± 27.38	892.00 ± 120.23	19.48 ± 3.63	0.34 ± 0.16	97.37 ± 34.90
Peak exercise	1103.70 ± 174.24	23.44 ± 4.49	0.48 ± 0.10	210.90 ± 29.28	904.10 ± 166.79*	18.89 ± 3.93*	0.34 ± 0.10**	36.23 ± 11.42*
Recovery	1007.10 ± 162.89	22.86 ± 4.04	0.27 ± 0.09	122.74 ± 25.19	913.90 ± 128.00	19.39 ± 3.60	0.28 ± 0.14	95.49 ± 35.22

Note. All variables are mean ± standard deviation. LBP, low back pain. CON, control group. $^{*}p<0.05$, $^{**}p<0.01$.

in regular aerobic exercise[18]. Thus, we conclude that the cause of increased lactate levels in patients with LBP was long-term inactivity due to pain.

It has been reported in the literature that aerobic exercise increased aerobic capacity and antioxidants and decreased MDA levels[19].

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