Immediate Effects of Core Stabilization Exercise on β-Endorphin and Cortisol Levels Among Patients With Chronic Nonspecific Low Back Pain: A Randomized Crossover Design

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Abstract

Objective: The main objective of the study was to measure the levels of plasma β-endorphin (PB) and plasma cortisol (PC) under lumbar core stabilization exercise (LCSE), placebo and control conditions in patients with chronic nonspecific low back pain.

Methods: Twenty-four participants with chronic nonspecific low back pain participated in a randomized, placebo-controlled, crossover design study. There were 3 experimental exercise conditions: control condition (positioning in crook lying and rest), placebo condition (passive cycling in crook lying using automatic cycler), and LCSE on a Pilates device tested with a 48-hour interval between sessions by concealed randomization. A blood sample was collected before and after the exercise conditions. Plasma β-endorphin and PC were measured through enzyme-linked immunosorbent assay and electrochemiluminescence in a Cobas E411 auto analyzer.

Results: A significant difference in PB level was identified before and after the LCSE condition ($P < .05$), whereas no significant differences were noted in control and placebo exercise conditions. Also, the trend of elevation of PB under the LCSE was significantly different compared with the placebo and control conditions ($P < .01$). In contrast, the PC level remained unchanged in all 3 conditions.

Conclusion: The findings of this study indicate that LCSE could possibly influence PB but not PC level among patients with chronic nonspecific low back pain. The mechanism of action of the pain-relieving effect of LCSE might be related to an endogenous opioid mechanism as part of its effects and might not be involved with a stress-induced analgesia mechanism. (J Manipulative Physiol Ther 2018;xx:1-8)

Key Indexing Terms: *Back Pain; Exercise; Beta-endorphin; Cortisol; Rehabilitation*

Introduction

Lumbopelvic core stability exercise (LCSE) training is a common therapeutic management in day-to-day practice for patients with low back pain. The LCSEs recruit and train certain specific muscles, such as transverses abdominis and multifidus, to provide spinal stabilization.[1] Evidence from the systematic reviews suggests that LCSE is an effective treatment for patients with low back pain because it reduces pain and improves function.[2] The mechanical and neurophysiological effects of LCSE, such as improved contractility of core muscle, enhanced feed forward mechanism, and improved lumbopelvic stability, are accounted as established effects for pain reduction among patients with chronic low back pain (CLBP).[3-6] Although the mechanical effects of LCSE are well documented, the biochemical effects of the LCSEs behind pain relief are yet to be fully understood. Exercise can cause endogenous induced analgesia through release of endogenous opioids.[7] However, it is not clear whether the LCSE can release the endogenous opioids that may be behind the mechanism of pain relief among patients with CLBP.

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Endogenous opioids have been found to have analgesic effects in a variety of chronic pain syndromes, including CLBP.[8] β-Endorphin (BE) is one such peptide released with adrenocorticotropic hormone from the anterior pituitary gland as a result of exercise stress.[9] The release of BE works on the endogenous opioid receptors and acts on the descending inhibitory system that modulates pain at the spinal cord level.[10] An appropriate metabolic and thermal stress is required in any exercise for the release of BE.[11] Moreover, it is suggested that exercise intensity and BE release are correlated in producing the opioid-induced analgesic effect in human beings after exercises.[12] However, it is not clear whether the LCSE has enough physical stress to induce BE release and to increase plasma BE (PB) in patients with CLBP.

Evidence suggests that functional somatic symptoms like CLBP are often associated with physical and mental stress.[13,14] Cortisol is a primary peripheral hormone from hypothalamic-pituitary-adrenocortical activity that reflects the coping mechanism of the body for stress response and pain adaptation.[13,14] The cortisol is released by the adrenal cortex by stimulation of adrenocorticotropic hormone at the anterior pituitary and corticotropin-releasing factor from the paraventricular nucleus of the hypothalamus.[15] The mental and physical stress associated with acute pain and anxiety causes an increased hypothalamus-pituitary-adrenal (HPA) axis activation.[13] Furthermore, the altered activity of the HPA axis contributes to evolution of stressful characteristics of a clinical problem into chronic pain disorder.[13] An increased concentration of plasma cortisol (PC) has been postulated to cause attenuation of pain perception in acute stress conditions.[16] Also, the cortisol regulates sympathetic and opioid mechanisms related with central pain processing.[15] If altered levels of the stress hormone cortisol influence stress-somatic complaints in functional somatic syndromes, such theories may apply in conditions like CLBP.[17] Therefore, in a common stressful condition like back pain or a physical stressor like exercise, it may be possible that both BE and cortisol are released as responses from the body to exhibit endogenous opioid–induced analgesia and stress-induced analgesia, respectively.

Although clinical studies have focused on the roles of BE and cortisol in health, the biochemical changes on PB and PC levels after LCSE have not been studied previously. Moreover, a recent systematic review concluded that there was limited evidence to determine whether or not the exercise therapy induces pain-modulating substances, and therefore, it warrants further investigation to explore the effects of LCSE on PB and PC.[18] In addition, stress-induced analgesia and placebo-induced analgesia are 2 of several other clinical reasoning mechanisms proposed to affect patients with low back pain.[19,20] Thus, it is appropriate to use deductive reasoning to differentiate and investigate the real effects of LCSE on PB and PC compared with placebo and controlled intervention.

Therefore, the main aim of the present study was to investigate the effects of 3 groups of exercise interventions—LSCE training, placebo (automated passive cycling training), and control (rest)—on the levels of PB and PC in patients with CLBP. The present study hypothesized that the LCSE might increase PB and reduce PC compared with the placebo and control interventions among patients with CLBP. Information from this study may help clinicians to understand the potential biochemical effects of LCSE compared with other exercise regimens. Such knowledge may assist clinicians to design appropriate exercise prescriptions for low back pain rehabilitation.

Methods

Participants

A total of 24 participants (7 men, 17 women; aged 33.76 ± 14.51 years) with CLBP residing in a community and university area participated in the study. An advertisement about the research study was placed around the community and in university locations, and the participants were recruited through predefined inclusion and exclusion criteria. Participants aged 20 to 35 years with mild to moderate back pain, with a visual analog scale pain score between 2 and 7 cm and presence of pain for more than 3 months with location of pain in the area between the lowest (12th) rib and the gluteal folds, participated in the study. Any participants with referred pain or neurologic involvement in the lower limbs, with a history of past surgery, with a history of smoking, or with a history of injury in the last 3 months were not recruited for the study. In addition, pregnant women and women who reported menstruation 3 days before or 3 days after the study period were not included. Also, any participants who performed any forms of physical exercises regularly were not considered for the study because routine exercises might have influenced the physiological levels of BE. None of the participants took stimulants, medications, or alcohol or was involved in heavy physical activities at least 12 hours before the test. All participants gave written informed consent to join the study, and the whole research project was conducted in an outpatient physiotherapy department of a university teaching hospital. A university ethical committee approved the human ethics for the study according to the standards of the Declaration of Helsinki, Finland.

Study Design

The effects of the 3 different groups of intervention—LCSE training, placebo (automated passive cycling training), and control (rest)—on the levels of PB and PC were investigated through a randomized, placebo-controlled crossover trial. An envelope-based concealment of random assignment and allocation of the participants was used in the study. Two independent physiotherapists who were not the part of the study assisted the process of random