Relationship of trunk muscle atrophy and provocation position in patients with chronic low back pain

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Objective: The purpose of this study is to compare chronic low back pain patients’ pain provocation position so as to identify the relevance with lumbar stabilizing muscles atrophy and pain provocation position.

Design: Cross-sectional study.

Methods: Fifty five chronic low back pain patients were participated in this study. Subjects were eligible for study participation if they were 35-55 years old and had experienced low back pain for more than 3 months. Subjects were questioned about pain and pain provocation test were done. And then they were inspected their cross sectional area (CSA) of lumbar muscles (erector spinae, iliopsoas, and multifidus) by using computed tomography. Analyze the relevance through the result data with painful area, aspect of pain and pain provocation position.

Results: CSA of erector spinae showed significant decrease on ipsilateral extension position (p < 0.05). Iliopsoas muscle showed significant decrease on contralateral position (p < 0.05). Multifidus showed significant decrease on the position of contralateral extension and contralateral flexion (p < 0.05).

Conclusions: Based on the results of our study, it may be possible to evaluate muscle atrophy by assessing causing position.

Key Words: Computed tomography, Cross sectional anatomy, Low back pain, Lumbar vertebrae

Introduction

Low back pain is one of the most common disease in society and about 80% of people were experienced it at least once in their lifetime [1]. Low back pain is also second largest reason for visits the hospital [2] so that low back pain is considerable problems of people that affects daily living and occupational activities and causes psychological as well as social problems [1,3,4]. There are several different factors and underlying mechanism responsible for lower back pain. These include herniated intervertebral discs, stenosis, spondylolisthesis, and spondyloyysis [5]. However, the prevalence of these conditions is not relatively high, and most cases of lower back pain are due to muscle sprain in this area [6,7].

If low back pain persists for a prolonged period of time, the paraspinal muscles weaken. In some cases, atrophy occurs when use of the muscle is avoided due to pain. Additionally, the other one is due to inhibiting activation of a motor neuron which controls muscle by afferent stimulation of damaged part [8,9].

There has been an interest in lumbar stabilization for treating lower back pain and a biomechanical model, showing the structural arrangement of superficial and deep muscles, was presented [10]. Additional studies later demonstrated how function could be recovered through the movement of the main muscles, multifidus and transverse abdominis, and lower back pain reduced [11-14]. To achieve lum-
bar stabilization, superficial muscles such as paraspinal muscles and rectus abdominis are needed along with deep muscles like the multifidus and transverse abdominis [15,16].

Recently, the development of body imaging techniques such as computer tomography (CT) [17], magnetic resonance imaging [18], and real-time ultrasound imaging [19] has enabled us to measure cross-sectional areas of the muscle. It is also possible to compare the shape and pain condition of the muscle involved in lumbar stabilization [20].

Danneels et al. [21] compared the CT data for the paraspinal muscles, multifidus, and iliopsoas of the trunk for chronic low back pain patients to that for healthy persons, and determined that the multifidus and paraspinal muscles had atrophied in the individuals with lower back pain. Barker et al. [22] confirmed with MRI that there is an association between pain in the multifidus and iliopsoas atrophy. Additionally, Hides et al. [23] reported that the multifidus is more atrophied in patients with chronic lower back pain than healthy individuals. Barker et al. [22] identified the atrophy of the multifidus and the iliopsoas in the pain area and no pain area and confirmed the relationship between pain and muscle atrophy.

Most cross-sectional studies have focused on the effect of spinal stabilization by studying muscles or training. Therefore, we wished to examine cross sectional area (CSA) of the multifidus, erector spinae, and iliopsoas midway between the plate and upper plates of the lumbar spine. The purpose of our study was to determine the relationship between pain provocation posture and muscle atrophy.

Methods

Subjects

Fifty five subjects participated in our study and were recruited from K hospital in Gyeonggi-do. Individuals were eligible for study participation if they were 35-55 years old and had experienced low back pain for more than 3 months (Table 1). The study included patients with chronic low back pain, who presented to the Orthopedic Surgery Department of our hospital between May 2010 and November 2011. Patients who had experienced unclear pain provocation posture, any tumors, inflammation problems, any fractures, spondylolisthesis, spondylolysis, ankylosing spondylitis, metabolic disease, structural malformation, difficulty with maintaining posture due to central nervous system problems, and neurological symptoms such as paresthesia, mental disease, intellectual disabilities, or other musculoskeletal disease were excluded. People who had undergone spinal surgery were also ineligible for our study.

Outcome measures

Imaging CSAs in the trunk muscles

CT (PQS 2000, Picker, USA) was used to evaluate the CSA in the trunk muscles. The patients reclined with a pillow between their legs and their weight symmetrically distributed in both sides. The lumbosacral was then visualized with CT, and the area between each upper and lower level of the lumbar spine was measured. An imagery analysis system (NIH Image, 1.61, US) was used to identify the CSA of the multifidus, erector spinae, and iliopsoas midway between the plate and upper plates of the lumbar spine. All measurements were recorded by one radiologist.

Data analysis

All data analyses were performed using IBM SPSS ver. 19.0 for Windows (IBM Co., Armonk, NY, USA). p-values <0.05 were considered statistically significant. An independent t-test was used to compare differences between groups. A one way ANOVA was used to compare differences between lumbar spinal level and pain provocation position.

Results

Comparison of multifidus muscle size for each lumbar spinal level according to the pain provocation test

Comparison of the CSA of the multifidus in each lumbar spinal level according to the pain provocation test results is in Figure 1. No significant differences were observed between L1 and L2, L2 and L3. Between L3 and L4, the con-

Table 1. Characteristics of all participated the subjects (N=55)

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. or Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>52.2 (28.9)</td>
</tr>
<tr>
<td>Male/female</td>
<td>21/34</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.1 (12.5)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>65.2 (11.8)</td>
</tr>
<tr>
<td>Duration of pain (week)</td>
<td>15.0 (9.3)</td>
</tr>
<tr>
<td>VAS (point)</td>
<td>7.6 (0.9)</td>
</tr>
<tr>
<td>ODI (%)</td>
<td>35.1 (13.6)</td>
</tr>
</tbody>
</table>

VAS: Visual Analogue Scale, ODI: Oswestry Disability Index.
Comparison of multifidus muscle size for each lumbar spinal level according to the pain provocation test

The contralateral extension group had the smallest size \( p < 0.05 \) and significant differences were also found between the contralateral extension group, ipsilateral flexion group, and ipsilateral extension group \( p < 0.05 \). Between L4 and L5, significant differences were observed between each group \( p < 0.05 \). Between L5 and S1 the size for the contralateral extension group was the smallest \( p < 0.05 \) and there were significant differences between the contralateral flexion group, ipsilateral flexion group, and ipsilateral extension group \( p < 0.05 \).

**Comparison of erector spinae muscle sizes for each lumbar spinal level according to the pain provocation test**

Comparison of erector spinae muscle size for each lumbar spinal level according to the pain provocation test is shown in Figure 2. No significant differences were observed between L1 and L2, L2 and L3. Between L3 and L4, significant differences were observed between each group \( p < 0.05 \). Additionally, the size for ipsilateral extension group was smaller than that of the contralateral extension group \( p < 0.05 \). Between L4 and L5, significant differences were observed between each group \( p < 0.05 \). Between L5 and S1, significant differences were observed between each group \( p < 0.05 \). The size for the ipsilateral extension group was the smallest \( p < 0.05 \).

**Discussion**

In the present study, we examined the relationship between posture which cause pain and muscle atrophy to provide a foundation for diagnosing lower back pain. We compared posture that caused pain and CSAs visualized by CT.
At each level, noticeable differences in muscle were rarely found but at lower lumbar, there was a significant difference. Atrophy of CSA was observed in subjects who experienced pain while extension the contralaterally multifidus. This finding indicates that the multifidus contracts eccentrically and needs to control the flexion of trunk so the pain is thought to caused when there is atrophy. In the erector spiniae, atrophy was found at the CSA in subjects who felt pain during ipsilateral extension. This is the main motion of the erector spiniae and causes pain when there is a concentric contraction once the trunk is extended ipsilaterally [24]. The iliopsoas had atrophied in CSAs in subjects who felt pain during contralateral extension. The iliopsoas's main motion is ipsilateral flexion but at a posture fighting against the gravity, contralateral extension of trunk caused eccentric contraction and balance controlled the trunk, which causes pain [25].

Pain is also caused by movement towards flexion way of trunk by eccentric contraction when contralateral flexion [26]. Based on the results of our study, it may be possible to evaluate muscle atrophy by assessing causing position.

References

25. Stefanidi AV, Skoromets AA, Dukhovnikova IM. Acute my-