Relationship between Scapular Upward Rotation and Gluteus Maximus Activity in Subjects with Chronic Nonspecific Low Back Pain

Mohamed Abdelmegeed1*, Warda Abdelaziz1, Salwa Abdelmajeed1, Hala Elhabashy2

1Department of Orthopedic physical therapy, Faculty of physical therapy, Cairo University, Egypt.
2Clinical neurophysiology unit, Faculty of Medicine, Cairo University, Egypt.

*Correspondence to Mohamed Abdelmegeed, Department of Orthopedic Physical Therapy, Faculty of physical therapy, Cairo University, Giza, Egypt.
Tel: +1 909 583 4966
Email: mabdelmegeed@cu.edu.eg

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Abstract:

Purpose: the purpose of this study was to investigate the relationship between the scapular upward rotation and the electromyographic (EMG) activity of gluteus maximus (GMax) and latissimus dorsi (LD) muscles in subjects with chronic nonspecific low back pain (CNSLBP).

Methods: in an EMG laboratory, 26 subjects with a diagnosis of unilateral CNSLBP with a mean age (y) of 25.15 ± 4.11 were recruited through direct referrals. EMG activity of the GMax and LD muscles was recorded while subjects were walking on a treadmill. Scapular upward rotation was measured using bubble inclinometers at 0⁰, 45⁰, 90⁰, 135⁰, and end range abduction. Pearson’s Correlation Coefficient (r) was used to correlate the outcome variables.

Results: a significant correlation between the GMax EMG activity and scapular upward rotation measured at 0-degree abduction (p= .009) was found while there was no significant correlation between the GMax activity and scapular upward rotation measured at the other four ranges (45⁰, 90⁰, 135⁰, end range abduction). Also, there was no significant correlation between the EMG activity of GMax and LD muscles (p>.05).

Conclusion: subjects with unilateral CNSLBP may have dysfunctional scapular upward rotation not related to the degree of GMax EMG activities. In addition, a correlation between increased or decreased GMax and LD muscles EMG activity could not be established.

Keywords: scapular upward rotation, low back pain, data correlation.

1.Introduction:

Chronic nonspecific low back pain CNSLBP is a common musculoskeletal problem affecting different age groups. The cause of CNSLBP is not well understood with an ill-defined diagnosis in approximately 85% to 90% (1). The high socioeconomic burden of CNSLBP suggests that more investigation of the condition is necessary (2).

Although CNSLBP does not have specific diagnostic features other than vague pain, abnormal movement, and/or functional limitation, other causes were hypothesized such as dysfunctional trunk and hip muscles (3) and movement system impairment (4).

The gluteus maximus (GMax) and latissimus dorsi (LD) muscles are interconnected through the thoracolumbar fascia (TFL) and constitute part of the myofascial sling which is important to normal load transmission, stability, and mobility of the trunk. When the oblique myofascial sling (GMax and contralateral LD muscles) operates, they produce efficient trunk extension and unload the lower back (4,5).

When there is a dysfunction in the LD muscle, abnormal movement of the scapula emerges. It was hypothesized that tightness of LD muscle can produce abnormal movement of the scapula either...
directly through reduced upward rotation or indirectly through increased upward rotation as a compensatory mechanism. This can all happen in subjects with CNSLBP due to the abnormal length of TFL (6,7).

The GMax muscle is a powerful hip and trunk extensor. Weakness of GMax muscle has been associated with low back pathologies (8). The movement system impairment described by Sahrmann et al. (4) implies that a change in muscle activity causes abnormal movement patterns which accentuate the presence of the dysfunction. This is true in subjects with CNSLBP caused by abnormal posterior oblique sling muscles (GMax and contralateral LD) activities.

According to Sahrmann et al. (4), sustained abnormal posture during activities of daily living causes abnormal tissue adaptation with resultant pain and dysfunction. This can be applied to the dysfunction of posterior oblique muscle activity in subjects with CNSLBP which can affect the position of the scapula and lumbopelvic stability. The relationship between the scapular upward rotation (through the action of the LD muscle) and the GMax muscle has not yet been investigated to the authors’ knowledge. The purpose of this study, therefore, was to investigate the relationship between the scapular upward rotation and the electromyographic (EMG) activity of GMax and LD muscles in subjects with CNSLBP.

2. Methods:

This observational analysis was conducted at the EMG laboratory of the faculty of physical therapy, Cairo university. The study was approved by the institutional review board (IRB) of the Faculty of Physical Therapy, Cairo University (approval number: P.T.REC/012/003643)

Twenty-six subjects with CNSLBP were recruited through direct referrals from their orthopedic physicians. They were included in the study if they were between the age of 20 and 40 years, have unilateral CNSLBP of at least three months in duration, have at least a score of 3 out of 10 on the visual analog scale (VAS), a body mass index (BMI) less than 30. Subjects with radiated buttock or lower extremity pain, non-mechanical low back pain, neurological dysfunctions, leg length discrepancies, shoulder dysfunction, or who do not meet the inclusion criteria were excluded. After checking eligibility to participate, each subject read and signed the informed consent form.

2.1 Procedure for assessment:

Subjects’ demographics were collected at the time of the assessment. This included the age, weight, and height to calculate the BMI. After being familiarized with the assessment procedure, the EMG activity of the GMax muscle on the same side of the low back pain and the contralateral LD muscle was recorded during walking on a regular treadmill with a speed of 3 km/h.

To measure the EMG activity of the GMax muscle, an EMG unit (Neuro-MEP.NET, Neurosoft, Ivanovo, Russia) was used. After cleaning the skin with 70% alcohol, the active electrode was placed on GMax motor point midway between the greater trochanter and the second sacral vertebra, the reference electrode was placed one inch lateral to the active one. For electrode placement of LD muscle, the active electrode was placed on the motor point of the muscle between the T9 spinous process and lateral torso, the reference electrode was placed medial and inferior to the active one at the level of the T10 spinous process. The ground electrode was placed on the spinous process of the L2 vertebra. (9,10). Surface electrocardiography adhesive electrodes were used.

The subject was made familiar with how the treadmill is operating and was taught the safety measures in case of emergency. Before placement of the surface EMG electrode, the subjects walked at his/her normal pace to get used to walking on the treadmill and were asked to increase or decrease the speed just to train for the walking required with the wires on. After the subject reported that he/she is comfortable walking, the speed was adjusted to 3 km/h, and he/she was asked to walk for 3 minutes. After that, the subject rested for 15 minutes, then the EMG electrodes and wires were hooked to the subject’s body, and he/she was asked to walk again on the treadmill for three minutes. The EMG activity from GMax and LD muscles was recorded for 30 seconds (figure 1).

To get a normalized EMG activity for each muscle for each subject, the maximum voluntary isometric contraction (MVIC) was obtained from the position of the muscle test for the corresponding muscle then the root means square (RMS) was obtained. For GMax muscle, however, it was recommended to perform the submaximal voluntary isometric contraction since the MVIC can cause pain which can interfere with the RMS value (11).

The RMS value for GMax muscle was normalized by having the patient lay prone, both knees bent to 90 degrees, and was asked to lift both thighs off the table for 5 seconds while the therapist recorded the EMG activity using the same electrode placement as before. For LD muscle, the subject was prone and was asked to extend the shoulder against the therapist’s hand keeping the elbow at 90 degrees and hold the contraction for 5 sec (11).
The amplified signal obtained during treadmill walking and the average RMS values were calculated and expressed as a percentage of the normalized value obtained during the maximum and submaximal voluntary isometric contraction from prone (12).

For measurement of scapular upward rotation, two bubble inclinometers (Baseline® Bubble Inclinometer, Fabrication Enterprises INC, White Plains, New York 10602, USA) were used. One inclinometer was strapped to the distal humerus to measure the degree of shoulder abduction, and one was placed on the spine of the scapula to measure the degree of scapular upward rotation. The scapular neutral position was measured while the arm was at the side (0-degree abduction), and then the subject was asked to abduct the arm in the frontal plane and stop per the therapist’s order at 45, 90, 135 degrees and at the end of abduction range. Scapular upward rotation was measured at each of these positions.

The average of three trials at each of the ranges was used for data analysis. Scapular upward rotation at the same side of the tested LD muscle was measured i.e., if the EMG activity of the left LD muscle was measured (and the right GMx muscle), then the left scapular upward rotation was measured (13).

2.2 Statistical analysis

To establish the correlation between the measured variables, a correlation matrix was produced using Pearson’s correlation coefficient in the statistical package for social science (SPSS) program, version 27 (SPSS Inc, Chicago, IL, USA). Pearson’s correlation was conducted because the variables were continuous, and the normal distribution of the sample was achieved using the Shapiro-Wilk test. The P value was considered significant if ≤ 0.05.

3. Results:

There was a statistically significant correlation between the GMx EMG activity and scapular upward rotation measured at 0-degree abduction (p= .009) with no significant correlation between the GMx activity and scapular upward rotation measured at the other four ranges (45, 90, 135, end range abduction). Also, there was no significant correlation between the EMG activity of GMx and LD muscles (p>.05) (Table 1).

Table 1: Pearson correlation (r) value between the outcome measure

<table>
<thead>
<tr>
<th>EMG (% MVIC)</th>
<th>LD</th>
<th>ABD_0⁰</th>
<th>ABD_45⁰</th>
<th>ABD_90⁰</th>
<th>ABD_135⁰</th>
<th>ABD_end</th>
</tr>
</thead>
<tbody>
<tr>
<td>GMx</td>
<td>-.133</td>
<td>.499**</td>
<td>.363</td>
<td>.020</td>
<td>-.144</td>
<td>-.089</td>
</tr>
<tr>
<td>LD</td>
<td>1</td>
<td>.181</td>
<td>-.372</td>
<td>-.359</td>
<td>-.343</td>
<td>-.205</td>
</tr>
</tbody>
</table>

GMx: gluteus maximus, LD: latissimus dorsi, ABD: abduction end: end range abduction **Correlation is significant at the 0.01 level (2-tailed).

4. Discussion:

In this study, the relationship between the EMG activity of GMx muscle and the contralateral scapular upward rotation was investigated in subjects with unilateral CNSLBP. Generally speaking, a such correlation could not be established. In addition, a negative correlation (although not significant) could be found between the EMG activity of the GMx and LD muscles. It is important to mention that the EMG activity of both muscles was recorded during a functional task (walking) while the scapular upward rotation was measured during a less dynamic task (shoulder abduction). Measurement of scapular upward rotation during walking could not be performed in this study due to the lack of logistics and tools needed for such recordings. In addition, the EMG lab where this study was performed was not equipped for such a purpose.

While some of the previous studies showed an inverse relationship between the EMG activity of GMx and contralateral LD in subjects with CNSLBP (5,9,15), other studies showed a direct relationship (16-21). This contradiction may stem from different levels of dysfunction of both muscles in subjects with low back pain and the different methods of assessment used in these studies. This means that with the changing severity of the CNSLBP, the level of activation is also different. Subjects with CNSLBP...
may present with different levels of LD stiffness and/or GMax weakness. Stiffness that develops as a result of pain can limit scapular upward rotation while the weakness of GMax during gait will limit the subject’s ability to move the arm as a result of guarding which will in turn increase the LD stiffness (14).

This stiffness/weakness relationship can develop over time or can be present as a compensatory mechanism when the subject is challenged during a functional task such as walking. The relationship between the posterior oblique muscles of GMax and LD is well documented in the literature although the results of the level of activation are contradictory as mentioned before. It is still important to further study such a relationship although previous literature reported that the subject’s activity level during task performance is neither predictive nor concomitant with the level of disability or pain severity (22).

Surface EMG muscle activity provides an objective method of assessment. Other studies used other tools such as the back range of motion (BROM) device (23) or Ultrasound (24) to investigate the length of the thoracolumbar fascia (TLF) connecting the GMax and LD in subjects with CNSLBP. They found that subjects with CNSLBP show a significant reduction in the length of TFL as compared to age-matched controls. It was found, however, that the strength of LD muscle as measured by the manual muscle test was not different between subjects with CNSLBP and controls (25).

Since the CNSLBP is elusive, it can still be interpreted based on movement system impairment described by Sahrmann et al. (4) which entails that malalignment and getting used to doing movement in an improper posture are associated with musculoskeletal dysfunctions. This concept involves a combination of dysfunction in the different bodily systems and can be precipitated with or without trauma. Fear of movement is also a factor in movement system impairment. The presence of these factors in subjects with CNSLBP can result in abnormal movement. Since the TFL connects LD to GMax muscles, subjects with CNSLBP may show abnormal movement patterns of the scapula giving the anatomical attachment of LD to the inferior angle of the scapula regardless of whether this abnormal movement will result in an increase or decrease in the scapular upward rotation (26).

The result of this study is contradictory to the finding of Taghizadeh et al. (26) who found that scapular upward rotation increased in subjects with chronic low back pain versus healthy controls. They also highlighted that stiffness of LD muscle can cause scapular hypomobility in subjects with CNSLBP.

They explained that if this is the cause of scapular abnormal mobility, the shoulder and the neck should be assessed. The tightness of the LD gives a diagnostic clue about where to assess and treat and not to focus only on the region of the lower back.

The result of the current study should be interpreted considering the study’s limitations. The small sample size could have contributed to the lack of correlation between the outcome variables and would not make it possible to generalize the result limiting the external validity of the study. Second, the assessment of scapular upward rotation was performed by asking the subjects to abduct the arm in the frontal plane. The scapular plane was not considered for the assessment, and it could have produced a different result given the normal anteversion of the scapula. Third, we measured the scapular upward rotation during a less dynamic task which could have had a different result if it was performed during walking or other multitasks. We recommend that future studies address these limitations.

5. Conclusion:

Based on the results and considering the limitations, subjects with unilateral CNSLBP may have dysfunctional scapular upward rotation not related to the degree of GMax EMG activities. In addition, a correlation between increased or decreased GMax and LD muscles EMG activity could not be established.

Ethical approval:

The study was approved by the institutional review board (IRB) of the Faculty of Physical Therapy, Cairo University (approval number: P.T.REC/012/003643).

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Declaration of competing interest:

The authors declare that they have no competing interests.

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