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Original Article

Correlation between Sagittal Lumbopelvic Control and Pain Sensitivity and Intensity in Chronic Lumbosacral Radiculopathy Female Patients: A Cross-Sectional Study

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Abstract

Objective: Chronic lumbosacral radiculopathy (LSR) patients frequently complain of recurrent chronic lumbopelvic pain. The poor lumbopelvic (LP) control may be related to this chronic pain in LSR. We aimed to investigate the relation of sagittal LP control with pain sensitivity and intensity in chronic LSR females. **Methods**: This study included forty females with chronic unilateral discogenic LSR (30-46 years old). Their low back pain intensity was 6.87±1.02 by visual analogue scale (VAS). The sagittal LP control and pain sensitivity and intensity were assessed for all patients using a pressure biofeedback unit during prone hip extension task, a digital pressure algometer applied at low back area and four sciatic valleix spots, and the VAS, respectively. **Results**: A moderate significant negative relation was found between sagittal LP control and pain sensitivity at low back point ($r = -0.48$, $p = 0.002$). There was a moderate to strong significant negative relation between sagittal LP control and pain sensitivity at the different sciatic valleix spots. In addition, a strong positive correlation between poor sagittal LP control and pain intensity was found ($r= 0.69$, $p < 0.001$). **Conclusion**: Poor lumbopelvic control may be highly related with pain sensitivity and intensity in chronic LSR females.

Keywords: Chronic Lumbosacral Radiculopathy, Lumbopelvic Control, Pain Sensitivity, Prone Hip Extension, Sciatic Valleix

Introduction

Lumbosacral radiculopathy (LSR) is characterised by pain caused by nerve root compression or irritation in the lumbosacral region[1]. It is one of the most common disorders of the spine which comprises about 62% to 90% of all radiculopathies[2]. The LSR affects about 3%-5% of general population worldwide[3]. It is a major cause of disability worldwide, negatively impacting communities, health care systems and patient's quality of life[4].

Patients with chronic LSR usually complain of persistent lumbopelvic (LP) pain with acute flare-ups of back pain travelling through thigh and leg with increased sensitivity[5]. In acute LSR, the pain is mainly caused by root irritation and compression originated from a herniated disc[6]. However, about 25% of LSR patients experience pain after 3 months in spite of anatomical resolution of the protruded disc. This shows that lumbar radicular symptoms are not related to MRI findings after disc prolapse[7], suggesting presence of rather factors than disc lesion related to chronic pain sensitivity of LSR patients. The recurrence and chronicity of LP pain in LSR patients may be related to poor LP control and non-optimal load transfer[6]. The LP control refers to the patient's ability to set LP orientation and control intervertebral regions[8]. Chronic low back pain

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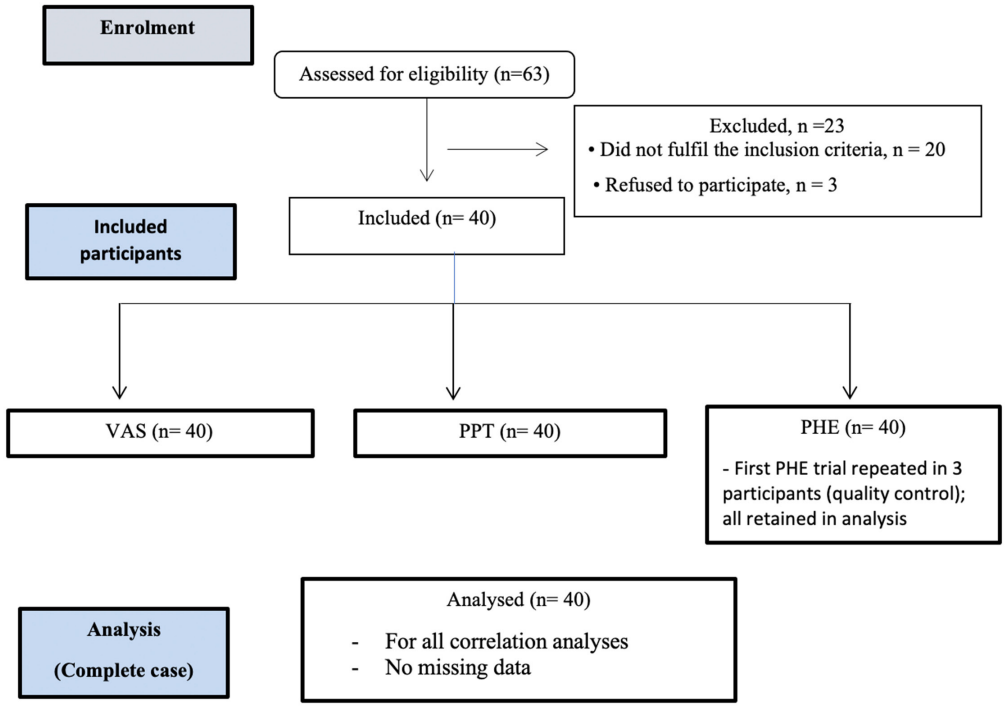

Figure 1. Participant flow diagram.

(CLBP) can impair segmental spinal control and pelvic control during functional task[9]. Females with LSR have worse pain perception, greater related disability and tend to develop more chronic manifestation as 3.3 times than for males[7,10].

Given the aforementioned, we hypothesized that there may be a relationship between LP control and pain sensitivity and intensity in chronic LSR. Up to our knowledge, there is lack of research work considering these correlations. Only one study has investigated the relationship between LP control with pain intensity measured by Visual Analogue Scale (VAS)[11]. Another study assessed the LP control in both healthy persons and CLBP patients[12]. The study of Paungmali et al (2017)[12] measured the effect of lumbopelvic stabilization exercises on pain threshold. So, the purpose of this study is to investigate the relation of sagittal LP control with pain sensitivity and intensity.

Methods

Study design

A cross-sectional study was performed between August 2025 and December 2025 at Outpatient Clinic, Faculty of Physical Therapy, Cairo University, Egypt. Aim and procedures of this study were clarified for all participants. All patients signed a written formal consent. A preliminary sample size calculation through G*Power (version 3.1.9.2; Franz Faul, Mün Kiel, Germany) revealed that forty females with chronic LSR is the minimal required number for achieving a statistical power of 0.80 at an alpha level of 0.05. This calculation was established in accordance with earlier research work[11,12].

Participants

Sixty-three female patients with chronic lumbosacral radiculopathy were initially assessed for eligibility. Twenty patients failed to fulfil the inclusion criteria (four patients were older than 50, five patients had previous lumbar surgery, three patients with fibromyalgia, four patients with bilateral radiculopathy, one patient received antidepressants and three patients have concurrent hip structural dysfunction), while three patients refused to participate in the study (Figure 1). The remaining forty chronic unilateral discogenic lumbosacral radiculopathy female patients were included in this study. All patients were diagnosed and referred by a neurosurgeon. The patients were enrolled from Outpatient Clinic, Faculty of Physical Therapy, Cairo University. They were recruited according to the following inclusion criteria: L5/S1 unilateral disc herniation with radiculopathy confirmed by MRI, age ranged from 30 to 46 years, chronic low back pain for

more than six months and body mass index (BMI) less than 30. The exclusion criteria were spinal tumors, fractures or infection, cauda equina syndrome, spondylolisthesis, spondylitis or spinal canal stenosis, any hip structural abnormality (e.g. malformations, impingements or degeneration), sacroiliac joint dysfunction, previous lumbopelvic or hip surgery, any postural deviations (e.g. scoliosis, kyphosis or lateral shift), pregnancy and severe sensory or motor loss at lower limbs. Patients used any analgesics, anticonvulsants or antidepressants in the previous three weeks were also excluded.

Procedures

Baseline characteristics of all participants including age, sex, weight, height, body mass index, side of radiculopathy and low back pain intensity were collected at the beginning of the study. However, other baseline data including disability, sleep, mood, fear-avoidance, medication history beyond 3 weeks, occupation and activity level were not assessed in this study. The low back pain intensity was evaluated by VAS. Then, LP pain sensitivity of the symptomatic side was measured by a pressure algometry. Finally, sagittal LP control was evaluated by a pressure biofeedback unit at the symptomatic side. All measurements were performed in the same order for all patients. The outcome assessor was a single, trained, and experienced physical therapist (9 years of clinical experience) and was blinded to our research hypothesis. A training session was conducted for the assessor before the beginning of the study to ensure reliability of all measurements. There was a 120 second-rest period between subsequent tests for all patients. The digital algometry and the stabilizer pressure biofeedback unit were calibrated before data collection according to the manufacturer's instructions.

Evaluation of low back pain intensity

Intensity of low back pain was measured through the Visual Analogue Scale (VAS). The VAS is a 10-cm scale with two ends, one end denotes no pain and the other one indicates the most terrible pain. Each patient was instructed to describe her level of pain by marking a point on the line[13].

Evaluation of lumbopelvic pain sensitivity

The pressure pain threshold (PPT) was used to measure LP pain sensitivity in low back area and sciatic valleix using the digital pressure algometry (Biotronix). Sciatic valleix is defined as highly sensitive localized pain spots along the sciatic nerve[14]. Four sciatic valleix spots including midpoint of gluteus maximus and popliteal fossa, mid-posterior thigh and calf cruris were assessed in the current study. From prone position, a pressure algometer with a stimulation surface area of 1 cm^2 was placed over the patient's low back area (2 cm lateral to L4 spinous process) and the four sciatic valleix spots. The pressure was applied at the following order for all patients: L4 (low back area), midpoint of gluteus maximus, mid-posterior thigh, midpoint of popliteal fossa and mid-posterior calf (cruris). Each patient was instructed to respond yes once pain was felt. The pain- producing pressure applied at a 0.1 kg/cm^2 rate was quantified as pressure pain threshold sensitivity (PPT) in kg/cm^2. Three successive measurements were conducted at each point and the average pressure of the three trials was recorded for each patient. There were ten-second rest time for each measurement of the same point[13] and 30 seconds rest interval between points. These rest periods were used to control tissue sensitization and pain temporal summation.

Evaluation of sagittal lumbopelvic control

A pressure biofeedback unit (PBU) (Stabilizer; Chattanooga Group Inc, USA) was used for measuring LP motion during active prone hip extension task (PHE) at the symptomatic side. The active PHE is a self-perturbation task used as an indirect measure for control of LP complex and treating patients with hip or trunk dysfunctions[15,16]. Before the test, each patient was asked to assume prone position on a hard surface with relaxed lumbar spine and neutral pelvis alignment. The patient was asked to perform 10 degrees of hip extension measured by a universal goniometer. An adjustable bar was fixed at this level to avoid undesirable movement. During the test, a pressure sensor of the PBU was placed anteriorly below the patient's lower abdomen and ilium. The PBU was inflated to 70 mmHg pressure (baseline pressure). Then, the patient was asked to lift the symptomatic leg off the bed while keeping a straight knee and neutral hip rotation till the heel touch the adjustable bar. For standardization of the test, all patients were instructed to breathe normally and not to perform any abdominal bracing during the test. The patients received no verbal or visual feedback for encouragement during the measurements. The amount of pressure applied by each patient was recorded and used as a proxy for sagittal LP control and LP motion. The difference between the baseline pressure (70 mmHg) and the pressure at end of PHE (end – baseline) was calculated as an absolute value. No pressure difference between baseline pressure and 10 degrees at the end of PHE represents good sagittal LP control and less LP motion. While any difference in the pressure values indicates poorer sagittal LP control and greater LP motion. All patients were instructed not to perform hip rotation during the test and if any hip rotation observed, the trial was repeated to ensure correct task performance. Three patients performed hip rotation movement during the first trial of PHE test and were asked to repeat this trial without hip rotation. Only these three trials of the three patients were excluded. All patients performed an initial trial before testing for familiarization with the test[17,18]. Three consecutive measurements were conducted with ten-second rest. The average pressure difference of the three trials was recorded for analysis. The outcome

Table 1. Patients' basic characteristics.

Variables	Mean ± SD
Age (years)	38.42 ± 6.27
Weight (kg)	79.08 ± 7.99
Height (cm)	167.84 ± 4.19
BMI (kg/m^2)	27.95 ± 2.33
Side of radiculopathy	Right: 27 (67.5%)
	Left: 13 (32.5%)

Data are represented as (Mean ± SD); SD: Standard deviation; Kg: kilogram; cm: centimetres.

Table 2. Patients' clinical characteristics.

Variable		N	Range (MIN- MAX)	Mean ± SD
VAS	Low back	40	5.20- 8.70	6.87 ±1.02
PPT (kg/cm^2)	L4 (low back area)	40	0.90- 4.00	2.27± 0.86
	Midpoint of gluteus maximus	40	0.60- 4.00	2.14± 0.74
	Mid-posterior thigh	40	0.30- 4.30	2.40 ± 1.00
	Midpoint of popliteal fossa	40	0.50- 4.00	2.45 ± 1.03
	Mid-posterior calf (cruris)	40	0.70- 5.00	2.72± 0.96
Mean pressure change (end-baseline) during PHE (mmHg)		40	10.00- 40.00	24.75 ± 7.51

Data are represented as (Mean ± SD); SD: Standard deviation; VAS: Visual analogue scale; PPT: Pressure pain threshold; PHE: prone hip extension; N: number; MIN: minimal; MAX: maximal; mmHg: millimeters of mercury; Kg: Kilogram; cm^2: square centimetre.

Table 3. Correlation of mean pressure difference during PHE (sagittal LP control and LP motion) with pressure pain threshold and visual analogue scale.

		PPT					VAS
		L4 (low back area)	Midpoint of gluteus maximus	Mid-posterior thigh	Midpoint of popliteal fossa	Mid-posterior calf (cruris)	
	N	40	40	40	40	40	40
Mean pressure difference during PHE (end-baseline) (mmHg)	**r value**	-0.48	-0.57	-0.54	-0.58	-0.34	0.69
	95% CI	-0.69: -0.20	-0.71: -0.22	-0.73: -0.27	-0.75: -0.32	-0.59: - 0.03	0.48: 0.82
	P value	0.002*	< 0.001*	< 0.001*	< 0.001*	0.03*	< 0.001*
	Adjusted P value	0.004*	< 0.001*	0.002*	< 0.001*	0.03*	< 0.001*

r value, Pearson correlation coefficient; p value: Probability value; *: Significant: (P<0.05); Strong correlation (±0.50 to ±1); Moderate correlation (±0.30 to ±0.49); Low correlation (< ±0.29); Adjusted P value: Holm–Bonferroni correction coefficient ; PHE: prone hip extension; VAS: Visual analogue scale; PPT: Pressure pain threshold; N: number; CI: Confidence Interval; mmHg: millimetres of mercury; Kg: Kilogram; cm^2: square centimetre; end-baseline: An absolute value.

assessor was blinded to our research hypothesis but not to the PPT values.

Statistical Analysis

Statistical package for the social sciences computer program (version 20 for Windows; SPSS Inc., Chicago, Illinois, USA) was utilized for analysing data. Shapiro-Wilk tests for normality revealed that all assessed variables followed normal distribution. Numeric data were calculated as mean ± standard deviation (SD) of patients' measured variables. The distribution of radiculopathy was analysed through frequency and percentage. Pearson correlation coefficient was utilized to expose the correlation between sagittal LP control and pain sensitivity and intensity in chronic LSR patients, with the level of significance at P<0.05. Prior to Pearson correlation, extreme studentized deviate (ESD) test was used to check for multiple extreme outliers (data was normally distributed). The ESD test didn't identify any significant outliers in the dataset. Also, linearity between variables was checked using scatterplots, which indicated approximately linear relationships with no influential outliers between LP control and pain sensitivity and intensity. Post hoc Holm–Bonferroni correction test was performed to handle multiplicity of multiple correlation outcomes. After correction, all correlations remained statistically significant.

Results

Patients' basic characteristics

The baseline data of all patients (age, height, weight, distribution of radiculopathy and BMI) are shown in Table 1.

Patients' clinical characteristics

The patients' clinical characteristics are presented in Table 2. Mean ±SD and ranges for VAS for low back pain intensity, PPT for the four sites (low back area at L4, midpoint of gluteus maximus, mid-posterior thigh, midpoint of popliteal fossa, and mid-posterior calf (cruris)) and PHE pressure difference (mmHg) were calculated as in Table 2.

Correlation of lumbopelvic control with pressure pain threshold and pain intensity

The bi-variate correlation coefficient was calculated for the mean pressure difference during PHE (sagittal LP control and LP motion) with VAS for low back pain intensity and PPT at both low back (L4) and four sciatic valleix spots (midpoint of gluteus maximus, midpoint of popliteal fossa, mid-posterior thigh and mid-posterior calf (cruris)) for all patients in Table 3.

There was a moderate significant negative correlation between the mean pressure difference during PHE (sagittal LP control and LP motion) and the mean values of PPT at low back point (L4) (r = -0.48, p = 0.002, 95% CI: -0.69: - 0.20) and mid-posterior calf (cruris) (r = -0.34, p = 0.03, 95% CI: -0.59: - 0.03). A strong significant negative correlation was found between the mean pressure difference during PHE (sagittal LP control and LP motion) and the mean values of PPT at midpoint of gluteus maximus (r = -0.57, p < 0.001, 95% CI: -0.71: - 0.22), mid-posterior thigh (r = -0.54, p < 0.001, 95% CI: -0.73: - 0.27), and midpoint of popliteal fossa (r = -0.58, p < 0.001, 95% CI: -0.75: - 0.32).

There was a strong significant positive relation between the mean pressure difference during PHE (sagittal LP control and LP motion) and the mean values of VAS at low back (r = 0.69, p < 0.001, 95% CI: 0.48: 0.82). The mean pressure of PHE (end − baseline) was calculated as an absolute value. Increased pressure difference at 10 degrees of PHE represents poorer sagittal LP control and greater LP motion, while decreased PPT represents increased pain sensitivity.

Discussion

The purpose of this study was to find out the correlation of sagittal LP control with pain sensitivity and intensity in chronic LSR female patients. Our major results were a presence of a moderate negative correlation between LP control and pain sensitivity at L4 (low back area) (r = -0.48, p = 0.002, 95% CI: -0.69: - 0.20) and mid-posterior calf (cruris) (r = -0.34, p = 0.03, 95% CI: -0.59: - 0.03). There was also a strong negative correlation between LP control and pain sensitivity at midpoint of gluteus maximus (r = -0.57, p < 0.001, 95% CI: -0.71: - 0.22), mid-posterior thigh (r = -0.54, p < 0.001, 95% CI: -0.73: - 0.27), and midpoint of popliteal fossa (r = -0.58, p < 0.001, 95% CI: -0.75: - 0.32). In addition, a strong positive correlation between sagittal LP control and pain intensity (r = 0.69, p < 0.001, 95% CI: 0.48: 0.82) was found. These findings can be interpreted as sagittal LP control was indirectly measured through the absolute pressure difference at 10 degrees of PHE. High pressure difference denotes poorer sagittal LP control and greater LP motion. Pain sensitivity was assessed through Pain pressure threshold (PPT) and low PPT values represent high pain sensitivity. Pain intensity was assessed by the Visual Analogue Scale (VAS) and high VAS score represents high pain intensity. So, this study assumes that decreasing the sagittal LP control may be highly related with increasing pain sensitivity and intensity, and vice versa.

Our findings partially agree with previous studies[11,12]. The study of Özalp et al (2022)[11], showed a significant negative correlation between the LP control and pain intensity in patients with lumbar disc herniation. However, there are some variations between our study and the study of Özalp et al (2022)[11]. Özalp et al (2022)[11] measured LP control through different outcome measures (trunk flexor muscle endurance and Sorensen tests), interpreting the opposite

direction of the relation between VAS and LP control measures with our study. Moreover, pain sensitivity wasn't assessed in the study of Özalp et al (2022)[11]. Paungmali et al (2017)[12] stated that the lumbopelvic stabilization training (LPST) may be associated with increasing pain threshold and may be an effective treatment for chronic low back pain. Despite different study designs, this previous study[12] may supports our finding that the LP control may be highly related with pain threshold and pain intensity.

The results disagree with two previous studies[19,20] who stated that LP control was not affected in CLBP population. The difference of the results may be due to different patients' characteristics. In addition, the study of Stöwhas et al (2024)[20] stated that the non-significant relation between pain and LP control may be due to low pain intensity in his study.

The presence of strong to moderate correlations of sagittal LP control (indirectly measured by the absolute pressure difference at 10 degrees of PHE) with pain sensitivity and intensity in LSR patients can be interpreted by some probabilities. However, the relation between the three variables in LSR patients may be overlapping. Patients with chronic LSR usually complain of chronic persistent LP pain. This chronic pain could contribute to a repeated greater LP motion during gait and PHE. This greater LP movement may be associated with poor LP control that may be manifested as hyperextension of lumbar spine and anterior pelvic tilting[21]. This may compromise the spinal and the intervertebral foramen and could contribute to lumbosacral root compression and irritation and exacerbating patient's manifestation[22]. As a result, this repeated irritative stimulus may be associated with high excitability, increasing patient pain sensitivity[23].

Moreover, chronic pain in LSR patients may contribute to the body adaptation for a single movement strategy during multiple tasks instead of using many different strategies for different tasks in healthy individuals. Consistent use of one strategy for all tasks may be associated with excessive or sustained compressive loads on LP region[6]. This excessive load may be a factor of recurrent LP pain and increased pain sensitivity in LSR patients. However, the direct correlation between pain sensitivity and pain intensity was not investigated in the current study.

Clinical implications

Demonstrating a statistically significant association between sagittal LP control with pain sensitivity and intensity in chronic LSR patients may help predict one of these variables by measuring the other ones. In addition, understanding these correlations may allow for expecting that good sagittal LP control may be related to low pain sensitivity and intensity in chronic LSR females.

Limitations and Recommendations

The current study has some limitations regarding confounding. Although, baseline patients' characteristics including age, sex, weight, height, body mass index, side of radiculopathy were assessed, statistical adjustment for these variables to assess their effect on primary correlations was not performed. In addition, other potential confounders, including disability, sleep, mood, fear-avoidance, medication history beyond 3 weeks, and occupation were not measured. Another limitation is that this study included only adult females, restricting the generalizability of our findings. However, the strategy for LP control is different between both sexes[24,25]. Moreover, this study has a potential measurement-bias risk that the outcome assessor was not blinded to prior measurement values and this could influence measurement behavior. However, this risk was minimized through standardization for all measurement protocols for all patients. Future research work including large cohorts with complete blinding of outcomes assessor and multivariate statistical adjustment are needed for more accurate assessment of these impacts. The activity level of participants was not also assessed in our study however; the strategy for LP control is task dependent[26]. Further studies included adult males and patients with different activity levels are also required.

Conclusions

Our study stated that sagittal lumbopelvic control may be highly correlated with pain sensitivity and intensity in chronic lumbosacral radiculopathy female patients.

Ethics Approval

The Ethics Committee for Clinical Research at Faculty of Physical Therapy, Cairo University approved our study (NO P.T.REC/012/005233), Date of Ethical Approval: 7 May 2024.

Consent to Participate

Written informed consent was obtained from all subjects involved in the study.

Authors' Contributions

Hend R. Kamal and Nagwa Ibrahim Rehab: Conceptualization and Methodology, Marwa Mostafa: writing and editing, Hoda M. Zakaria, Ebtesam M. Fahmy: data analysis, Visualization and editing. Azza S.A. Khalil: help and advice. All authors read and approved the final manuscript.

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