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Localized gluteal skin pinch pressure hyperalgesia in patients with chronic low-back pain

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Abstract

Background: Chronic low back pain (CLBP) is a significant health issue, causing reduced quality of life and employment limitations.
Objectives: This study aimed to compare the pinch pressure pain sensitivity of the skin in the lower back area between patients with nonspecific low back pain and a control group.
Methods: This observational, case-control study involved 193 patients with chronic low back pain (LBP) who scored more than 25 on the visual analog scale (VAS) for at least three months between October 2012 and June 2013. Mechanical pinching of the lower back region was used to test for skin hyperalgesia. A double-blind design was used to evaluate skin hyperalgesia in the control group, which consisted of 108 people.
Results: Signs of hyperalgesia were found in the upper lateral quadrant of the gluteal area, around the posterior part of the iliac crest, in both groups. However, the degree of hyperalgesia in patients with chronic nonspecific LBP (with or without symptoms) was significantly higher ($P<0.001$).
Conclusion: The results indicated that pressure-induced localized gluteal skinfold tenderness (hyperalgesia) is common in patients with chronic low-back pain.

Keywords: Chronic Low Back Pain, Hyperalgesia, Pressure algometry, Skin sensibility, Referred pain.

Introduction

Chronic low back pain (CLBP) is a serious health issue in developed nations, costing between $100 and $200 billion each year.[1] Aside from the financial burden, CLBP sufferers have a lower quality of life and a loss or limitation of work. Many people also complain of psychopathologies, including despair, anxiety, and a lack of social activities.[2] However, the pathophysiological alterations in CLBP remain unknown, necessitating greater research into the underlying pathophysiology, perhaps paving the way for successful mechanism-based treatment options.[2]

Hyperalgesia, an increased sensitivity to pain, can be a useful adaptation for protecting vulnerable tissues. However, persistent enhanced sensitivity can persist even after the initial cause of pain has disappeared, becoming a disease in its own right. Changes in signal processing within the nervous system may contribute to or cause hyperalgesia and allodynia. Localized hyperalgesia can occur as a consequence of direct tissue damage caused by sensitization of peripheral nociceptors or as a result of referred pain and tenderness, which is pain felt in a region distant from the stimulated tissue. Central changes in

processing nociceptive information may last for days, months, or years and can spread to sites remote from the primary cause of pain.[3] Widespread pain referral and generalized hyperalgesia are found in both animals and humans.[4-8] Referred pain and tenderness are common in various chronic pain conditions, including whiplash-associated disorders, fibromyalgia, degenerative knee-joint disease, irritable bowel syndrome, temporomandibular disorders, and idiopathic low-back pain.[9-14]

According to the convergence facilitation theory of referred pain, visceral and/or somatic inputs sensitize convergent spinal neurons, which originate from other parts of the body. Studies have demonstrated sensitization of dorsal horn convergent neurons by input from viscera and deep somatic structures.[15,16] These alterations show cutaneous receptive field threshold modifications and receptive field expansions in deep tissues and skin, explaining both deep soreness and cutaneous hyperalgesia. After sensitization of these secondary neurons, they respond more strongly to cutaneous input, explaining referred pain or cutaneous hyperalgesia.[17,18]

Generalized skin fold tenderness has been reported in fibromyalgia patients compared to controls.[19] Changes in pain thresholds have been demonstrated in CLBP patients, which may be interpreted as generalized pain hypersensitivity. However, the authors did not specifically investigate thresholds at the painful and nonpainful sites of CLBP patients.[20] These findings suggest that chronic pain in general, as well as nonspecific low back pain, may trigger a shared underlying mechanism, such as central sensitization, in addition to a probable initial continuous nociceptive input. The initial painful condition, such as irritation or injury to lumbar vertebra-related tissues, may function as a trigger for chronicity by gradually sensitizing nociceptive pathways.

Objectives

To our knowledge, no study exists that compares the pain pressure threshold of the skin over different parts of the total area of the low back region in chronic LBP patients against a control group. Therefore, we set out to assess the pressure-induced pain thresholds of the skin by pinching the skinfolds of different parts of the lower back using pressure algometry. This is a semi-objective method for the assessment of tenderness, the reliability of which has been reported previously.[21,22]

Methods

This observational, case-control study was conducted on 193 patients (70 males and 123 females, mean age 43.1 years) who had suffered from chronic low back pain (LBP) with an intensity score of more than 25 (using the visual analog scale, VAS; 0–100 mm) for at least three months. Exclusion criteria were as follows: pregnancy; previous surgery of the spine; a history of spinal fracture and spondylitis; serious inflammatory, rheumatic, or neurological and psychiatric conditions; uncontrolled systemic metabolic diseases; systemic infections; or having other pain syndromes. Candidates were also subjected to a clinical screening to rule out those with a high likelihood of lumbar radiculopathy, which includes radiating pain to the foot or toes, numbness and/or paraesthesia, muscular weakness (specifically testing for ankle dorsiflexion and great toe extension), impaired knee jerk and ankle reflexes, dysaesthesia and loss of light touch sensation in the medial, dorsal, and lateral aspects of the foot, an ipsilateral straight leg raising test inducing leg pain at less than 60 degrees of hip flexion, and crossed straight leg raising. Plain radiography of the lumbar spine was performed on all patients, and there were no clear pathological abnormalities to explain the source of their back discomfort. Finally, individuals with a clinical diagnosis of nonspecific LBP without radiculopathy owing to intervertebral disc herniation were eligible for inclusion based on the overall clinical impression and radiological results. Before examination, subjects confirmed they had not taken any analgesics or anti-inflammatory drugs during the last 48 hours. One hundred and eight people without a history of pain in the low back area during the previous six months and within the same age range as the test group were selected as the control group.

Pressure algometry

Using a push-force gage (Sundoo Instruments, model SN-100, capacity 100N, d=0.5N), increasing pressure was

applied over the fold of pinched skin of the entire low back area, from the inferior margin of the 12th rib to the inferior gluteal border, until the patient described the pressure as painful (up to 25 of 100 mm on the VAS, corresponding to mild pain). This was recorded as the pressure-pain threshold (PPT). Subsequently, the areas with the lowest pressure pain threshold (PPT), at pressures less than 0.7 times (100 mm VAS), and the other parts of the low back with the highest PPT were recorded. In this way, localized skinfold hyperalgesia in the low back area was detected. This was called gluteal skin pinch pressure hyperalgesia (GSPPH).

Statistical analysis

The continuous variables were expressed as the mean±SD, and the categorical variables were presented as a percentage and frequency. Chi-squared and Fisher-exact tests were used for comparisons. All statistical analyses were performed with SPSS (version 16.0, SPSS Inc., Chicago, IL, USA). A "P-value" less than 0.05 was considered significant.

Ethical considerations

The study was conducted in accordance with the Declaration of Helsinki. Institutional Review Board approval was obtained. The present study did not interfere with the process of diagnosis and treatment of patients, and all participants signed an informed consent form.

Results

A total of 193 chronic nonspecific low back pain (CLBP) patients completed the study, in addition to 108 healthy control individuals. The healthy controls reported no back pain. The mean age of the CLBP subjects was 43.1±13.5 years, while the mean age of the healthy control subjects was 46.1±15.9 years [Table 1]. In both groups, 158 (52.5%) of the participants experienced localized gluteal skin pinch pressure hyperalgesia (GSPPH) in the upper lateral quadrant of the gluteal area [Figure 1], with a threshold of less than 0.7 compared to other areas of the low back [Table 2]. In CLBP patients, 141 (73.0%) had GSPPH. The prevalence of bilateral and unilateral GSPPH was 51.8% and 21.2%, respectively. CLBP was significantly associated with GSPPH, with an odds ratio [OR] of 13.72 (95% confidence interval [CI], 7.42–25.36, p<0.001). In the healthy control group, 17 individuals (15.7%) had GSPPH, and the prevalence of bilateral and unilateral GSPPH was 9.2% and 6.5%, respectively. Age and gender were not significantly associated with GSPPH.

Table-1. Age distribution in different groups.

Age	Case (n=193)	Control (n=108)	P value
Mean±SD	43.1 ± 13.5	46.1 ± 15.9	0.08

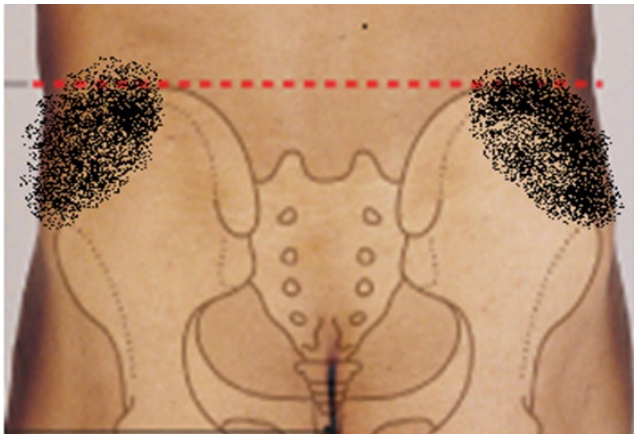

Figure-1. Skin hyperalgesia in low back area

Table-2. The values related to pinch pressure stimulation Hyperalgesia in the chronic low-back pain (Case) group and in the healthy (control) group

Skin Hyperagesia status (**GSPPH**)	**Case** (n=193)	**Control** (n=108)	**P value**
Bilateral	100(51.8%)	10 (9.2 %)	0.0001
Unilateral	41(21.2%)	7 (6.5 %)	0.0008
None	52 (27.0%)	91 (84.3 %)	0.0001

Discussion

The present study confirms the existence and higher prevalence of localized hyperalgesia in participants with LBP compared to healthy individuals. The aim of this study was to identify the prevalence and localization of skin hyperalgesia in the low back. This finding can be completely asymptomatic or may cause pain complaints in that area. In the future, this sign may be considered a treatment goal. This finding, though with a much lower prevalence, was also found in asymptomatic people. It should be considered a possible prodromal sign of LBP.

Further research is needed to evaluate the possibility of the further appearance of LBP in these individuals compared to people without this sign.

The difference in the prevalence of hyperalgesia in LBP patients in this study versus other studies.[9–11,13] may be due to the stricter definition of hyperalgesia used in this study, i.e., a decreased threshold of pain induced by pinching of the skin, which differs slightly from the definition of hyperalgesia described in previous works. This type of hyperalgesia has not been evaluated yet in healthy people or in patients with any chronic pain. This finding also agrees well with the underlying hypothesis that generalized hyperalgesia develops over time as a consequence of long-lasting pain.[22] Furthermore, self-reports of pain in the outer upper part of the gluteal area in the absence of clinical skin hyperalgesia (tenderness to pinch palpation, or GSPPH) and, on the other hand, the presence of GSPPH in the absence of any upper gluteal pain in some LBP patients and the normal population highlight the need for more researchers to discover the etiology of this sign. We used this precise definition, "GSPPH," to maximize the impression of our finding. However, this may be more specific than the usual signs and symptoms of LBP and its related radiation.

Although the mechanism underlying the reduction in skin pain threshold in these individuals is unknown, this link may be related to underlying processes[15,16] and/or the chronicity of LBP symptoms.[9] Some research has demonstrated that the central nervous system (CNS) has some role in localized and generalized decreased pain thresholds with chronic diseases.[3] This mechanism should be considered in further evaluations. This study did not detect an impact on participation in activities and quality of life, such as difficulty sitting and using some stools, in obese people. One limitation of this study was that we had difficulty finding the etiology of LBP in these studied patients.[1,2] Because this study design cannot establish causal relationships, characteristics that may predispose patients to chronic nonspecific LBP (for example, obesity, microinjuries, trauma, altered biomechanics secondary to weakness of related muscles, and congenital characteristics) limit the ability of this study to predict the prevalence of GSPPH and the most susceptible groups and types of treatment. The inability to diagnose the exact cause of GSPPH also prevents the determination of the etiology of this sign in asymptomatic populations. Longitudinal investigations are required to determine if LBP causes or is caused by GSPPH. The initial findings of this study may be useful not only in clinical care but also in generating hypotheses for studies or therapeutic trials to benefit patients with GSPPH or LBP.

Conclusions

The higher prevalence of GSPPH in individuals reporting low back pain in the absence of a known cause highlights the importance of this common symptom. Greater clinical awareness of GSPPH may help identify patients for primary prevention and therapy. This prevalence also suggests that the study of targeted rehabilitation may be useful in minimizing the impact of LBP. A longitudinal study will be necessary to identify causal factors and outcomes of interventions.

It is unlikely that sensitization develops without a continuous afferent barrage. Therefore, from a management point of view, inhibition of the initial source is advantageous. However, if this is not possible (or not sufficient), an alternative option is to attempt to treat the increased gain (hyperexcitability-hyperalgesia). Finally, recognition and treatment of these hyperexitable tissues can reduce the afferent barrage and alleviate total pain, even in the absence of knowledge of the source of the pain.

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Competing interests

The authors of this manuscript have no invested interests in products described or used in this article. The authors have no conflicts of interest.

Abbreviations

Chronic low back pain: CLBP;

Visual analog scale: VAS;

Low back pain: LBP;

Pressure pain threshold: PPT;

Gluteal skin pinch pressure hyperalgesia: GSPPH;

Central nervous system: CNS.

Authors' contributions

AK, MT, and MB were responsible for the study concept and design. AK, MT, and MB led data collection. AK, MT, and MB were responsible for the analysis and interpretation of data. MK wrote the first draft. ADB, and HA contributed to the writing of the second and third drafts. ADB, and HA provided comments on initial drafts and coordinated the final draft. All authors read and approved the final manuscript. All authors take responsibility for the integrity of the data and the accuracy of the data analysis.

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Role of the funding source

None.

Availability of data and materials

The data used in this study are available from the corresponding author on request.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. Institutional Review Board approval was obtained. The present study did not interfere with the process of diagnosis and treatment of patients and all participants signed an informed consent form.

Consent for publication

By submitting this document, the authors declare their consent for the final accepted version of the manuscript to be considered for publication.

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