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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 one of the major health problems and CLBP patients suffer from reduced quality of life and loss or limitation of employment.
Objectives: This study was aimed at comparing pinch pressure pain sensibility of the skin of total low back area in patients with nonspecific low back pain.
Methods: This observational, case-control study was conducted on patients 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 constant three months between October 2012 and June 2013. By mechanical pinching the skin of the lower back area, skin hyperalgesia was assessed in 193 patients. Following a double-blind design, skin hyperalgesia was also assessed for the skin of the control group (108 people).
Results: Signs of hyperalgesia in the upper lateral quadrant of the gluteal area, around the posterior part of the iliac crest, were found in both groups but the degree of hyperalgesia in chronic nonspecific LBP patients (with & without symptoms) was significantly higher (P<0.001).
Conclusion: The results demonstrated that pressure-induced localized gluteal skinfold tenderness (hyperalgesia) is found in most 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 one of the major health problems in industrialized countries, costing approximately US$ 100–200 billion a year.[1] Beyond this economic load, CLBP patients suffer from reduced quality of life and loss or limitation of employment. Many also complain of psychopathologies such as depression, anxiety, and loss of social activities.[2]

However, pathophysiological changes in CLBP are still obscure and call for additional efforts to understand the underlying pathophysiology, potentially allowing progress to be made towards successful mechanism-based treatment strategies.[2]

Hyperalgesia is a frequent symptom of diseases and may be a useful adaptation for the better protection of vulnerable tissues. Enhanced sensitivity for pain may, however, persist long after the initial cause for pain has disappeared. In this case, hyperalgesic pain is no longer a symptom but rather a disease in its own right. Changes of signal processing in the nervous system may contribute to or may become the sole cause for hyperalgesia and allodynia.[3]

Whereas the Localized hyperalgesia can be either following “direct tissue damage” related to sensitization of peripheral nociceptors, or due to referred pain & tenderness, which is pain that is felt in an area remote from the tissue that is stimulated.[3] Central changes in the processing of nociceptive information may potentially

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outlast their trigger events for days, months or years and also may spread to sites somatotopically remote from the primary cause of pain.[3] Widespread pain referral and generalized hyperalgesia is found in both animals and humans.[4-8] in a number of different chronic pain conditions, including whiplash-associated disorders,[9] fibromyalgia,[10] degenerative knee-joint disease,[11] irritable bowel syndrome,[12] temporo-mandibular disorders,[13] and idiopathic low-back pain.[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 body. Several studies have demonstrated sensitization of dorsal horn convergent neurons by input from viscera and deep somatic structures.[15,16] These changes manifest as threshold changes in the cutaneous receptive field, as well as receptive field expansions in deep structures and skin, explaining both deep tenderness and cutaneous hyperalgesia. After sensitization of these secondary neurons they respond more strongly to cutaneous input, thus explaining referred pain or cutaneous hyperalgesia.[17,18]

Generalized skin fold tenderness has been reported in fibromyalgia patients with respect to controls.[19] In addition, changes to pain thresholds have been demonstrated in CLBP patients that 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]

The implication of these studies is that chronic pain in general and in nonspecific low back pain patients may initiate some common underlying mechanism such as central sensitization, in addition to a possible initial continuous nociceptive input. The initial painful condition, such as irritation or damage to lumbar vertebra related tissues, may act as a trigger for chronification through gradual sensitization of nociceptive pathways.

Objectives

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

Methods

This observational, case-control study was conducted on one hundred and ninety three patients (70 males and 123 females, mean age 43.1 years) who had suffered from chronic low back pain (LBP) with intensity score more than 25 (using the visual analog scale, VAS; 0–100 mm) for at least constant three months.

Exclusion criteria were the following: 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 also received a clinical screening to exclude those with a high probability of lumbar radiculopathy, involving radiating pain to foot or toes; numbness and/or paraesthesia; muscular weakness (specifically testing for ankle dorsiflexion and great toe extension); impaired knee jerk and ankle reflexes; dysaestesia and loss of light touch sensation in the medial, dorsal, and lateral aspects of the foot; ipsilateral straight leg raising test inducing leg pain at less than 60 degrees of hip flexion; and crossed straight leg raising.

All patients were examined by plain radiography of the lumbar spine and showed no obvious pathological finding to explain the origin of their back pain.

Finally based on the overall clinical impression and radiology findings, those patients with clinical diagnosis of nonspecific LBP without radiculopathy due to intervertebral disc herniation were eligible for inclusion.

Before examination, subjects confirmed they had not taken any analgesics and anti-inflammatory drugs during the last 48 hours.

One hundred and eight persons without history of pain in low back area during the previous six months and with the same age range as the test group was selected as control group.

Pressure algometry

Using a push-force gage (Sundoo Instruments, model SN-100, capacity 100N, d=0.5N) increasing pressure over the fold of pinched skin of all the low back area, from inferior margin of 12th rib to inferior gluteal border, was applied until the patient described the pressure as becoming painful (up to 25 of 100 mm of visual analogue scale (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) the other parts of low back with highest PPT, were recorded.

In this way, localized skinfold hyperalgesia in the low back area was detected. This was named the 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 was used foe 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 LBP (CLBP) patients completed the study in addition to 108 healthy control people. No back pain was reported for the healthy controls. The CLBP subjects' mean age was 43.1±13.5 years, and the healthy control subjects' mean age was 46.1±15.9 years (Table-1).

158 (52.5%) of the subjects of both groups had localized gluteal skin pinch pressure hyperalgesia (GSPPH), in the region of the upper lateral quadrant of the gluteal area (Figure-1), with a threshold of less than 0.7 compared to other parts of 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 persons (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

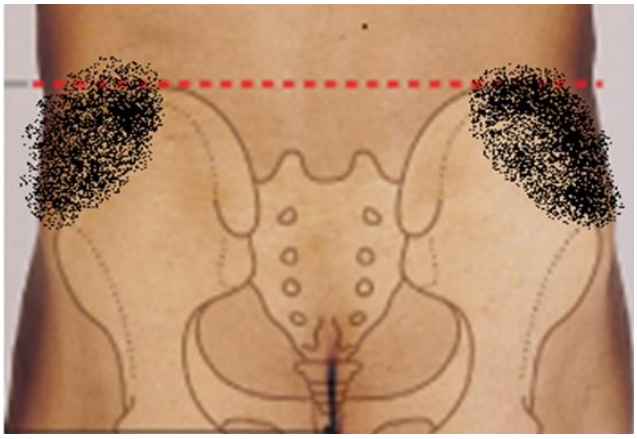

Figure-1. Skin Hyperalgesia in Low Back Area.

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 those with healthy people.

The aim of this study was identifying the prevalence and localization of skin hyperalgsia in low back. This finding

can be completely asymptomatic or may be cause of pain complaint at that area. In future this sign may be considered as a goal of treatment.

This finding, though with a much lower prevalence, was found also in asymptomatic people.

It should be considered as possible prodromal sign of LBP. Another research is needed to evaluate the possibility of further appearance of LBP in these persons, in comparison with people without this sign.

The difference in prevalence of hyperalgesia in LBP patients in this study with respect to other studies [9-11, 13] may relate to the more strict definition of hyperalgesia used in this study, i.e. decreased threshold of pain induced by pinching of skin, that differs somewhat with definition of hyperalgesia described in prior 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, the need for more researchers to find the etiology of this sign is shown by 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, existence of GSPPH in the absence of any upper gluteal pain in some LBP patients and normal population.

We used this precise definition "GSPPH" to maximize the impression of our finding. However, this may be more special than the usual sign & symptoms of LBP & its related radiation.

Although the mechanism for the decrease of skin pain threshold in these patients is unclear, this association could relate to underlying mechanisms [15,16] and/or chronicity of LBP conditions.[9] Some researches have demonstrated that central nervous system (CNS) has some role in localized and generalized decreased pain threshold 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 in sitting and using some stools, also in obese people.

One limitation of this study was that we had difficulty in finding the etiology of LBP of these studied patients.[1,2] Characteristics that might predispose patients to chronic nonspecific LBP (for example obesity, microinjuries, trauma, altered biomechanics secondary to weakness of related muscles, congenital characteristics), limits the ability of this study to predict prevalence of GSPPH and the most susceptible groups and type of treatment, because this study design cannot establish causal relations.

Inability to diagnose the exact cause of GSPPH also prevents determination of the etiology of this sign in asymptomatic populations.

To elucidate whether LBP leads to or caused by GSPPH, longitudinal studies are necessary. 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 people, who report low back pain in the absence of known cause, indicates that this common symptom is important. Therefore, 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 to minimize 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, but 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 certainly can reduce 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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