Association Between Symptoms of Central Sensitization and Cognitive Behavioral Factors in People With Chronic Nonspecific Low Back Pain: A Cross-sectional Study

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Abstract

Objective: The objective of this cross-sectional study was to analyze the relationship between symptoms of central sensitization (CS) and important cognitive behavioral and psychosocial factors in a sample of patients with chronic nonspecific low back pain.

Methods: Participants with chronic nonspecific low back pain for at least 3 months were included in the study. They completed several questionnaires and a functional test. Pearson's correlation was used to analyze associations between symptoms of CS and pain behavior, functioning, pain, pain catastrophizing, kinesiophobia, and illness perceptions. Additionally, a between-group analysis was performed to compare patients with and without clinically relevant symptoms of CS.

Results: Data from 38 participants were analyzed. Significant associations were found between symptoms of CS and all other outcomes, especially current pain ($r = 0.510$, $P = .001$), mean pain during the past 7 days ($r = 0.505$, $P = .001$), and pain catastrophizing ($r = 0.518$, $P = .001$). Patients with clinically relevant symptoms of CS scored significantly worse on all outcomes compared with persons without relevant symptoms of CS, except on functioning ($P = .128$).

Conclusions: Symptoms of CS were significantly associated with psychosocial and cognitive behavioral factors. Patients exhibiting a clinically relevant degree of symptoms of CS scored significantly worse on most outcomes, compared with the subgroup of the sample with fewer symptoms of CS. (J Manipulative Physiol Ther 2018;41:92-101)

Key Indexing Terms: *Low Back Pain; Central Nervous System Sensitization; Catastrophizing; Illness Behavior*

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Introduction

Chronic nonspecific low back pain (CNLBP) is a major problem today. Up to 84% of the general population experiences an episode of LBP during their life, and for about 90% of them, no specific medical cause can be found.[1-3] Instead, modern pain neuroscience has confirmed that the involvement of impaired central pain modulation or central sensitization (CS) might explain the patients' pain and symptoms.[4,5] Central sensitization can be defined as a process of abnormal and intense enhancement of pain caused by increased neuronal responses to stimuli in the central nervous system.[5-10] This central hyperexcitability is associated with altered sensory processing in the brain,[5,9,11] malfunctioning of endogenous pain inhibitory systems,[5,9,12] increased activity of pain facilitatory pathways,[5,9,11,13] and temporal summation of second pain and/or wind-up,[9,11,13] which leads to dysfunctional endogenous analgesic control.[12] Recent studies have also highlighted the role of an overactive network of brain areas in the CS process.[5,9,14] Increased activity is found to be present in brain areas involved in acute pain sensations (eg, insula, anterior cingulate cortex, and prefrontal cortex), as well as in other brain regions, such as different brain stem nuclei, the dorsolateral frontal cortex, and the parietal associated cortex.[14,15] Furthermore, decreased γ-aminobutyric acid neurotransmission[16] and long-term potentiation of neuronal synapses in the anterior cingulate cortex[17] are contributing to an overactive brain neuromatrix.[9] Besides these top-down mechanisms, different bottom-up mechanisms are also involved in CS, such as peripheral injury and other stressors (eg, infections), triggering the release of proinflammatory cytokines and the activation of spinal cord glia with cyclooxygenase-2 and prostaglandin E2 expression in the central nervous system.[9,18-21] Together, these mechanisms lead to hyperalgesia and allodynia in regions close to and outside the primary pain region.[5,9]

Symptoms of CS are present in a wide range of disorders, for example, fibromyalgia,[22] chronic whiplash-associated disorders,[23] irritable bowel syndrome,[24,25] and chronic fatigue syndrome.[26] However, only a subgroup of CNLBP patients appear to manifest features of CS.[6,8,27-29] Evidence exists that subgroups of CNLBP patients exhibit altered temporal summation, hyperalgesia, and allodynia for electrical, pressure, and heat stimuli in regions outside the primary painful region. Concerning the malfunctioning of descending anti-nociceptive mechanisms in CNLBP patients, conflicting evidence was found.[30,31]

It remains to be established why CS is present only in some patients with CNLBP. Genetic predisposition[4] and the influence of psychosocial and cognitive behavioral factors, such as depressive feelings,[32] pain catastrophizing,[33] and fear avoidance behavior, and more specifically kinesiophobia,[33-35] have been reported in patients with CNLBP. These factors may enhance central hyperexcitability through the activation of limbic brain regions, which, in turn, contributes to and sustains CS.[15,28,30,36,37]

The relationship between CS and pain catastrophizing or fear avoidance beliefs in patients with CNLBP has not yet been studied. By unraveling which psychosocial and cognitive behavioral factors underlie, cause, or maintain the process of CS in CNLBP patients, more targeted interventions could be integrated into their treatment, which might lead to better outcomes for this population.

The first objective of the present cross-sectional study was to analyze associations between symptoms of CS and pain behavior, functioning, pain intensity, illness perceptions, kinesiophobia, and pain catastrophizing in people with CNLBP. Second, this study aimed to compare the aforementioned outcome measures between patients manifesting and those not manifesting symptoms of CS to a clinically relevant degree.

It was hypothesized that symptoms of CS would be positively correlated with pain, pain behavior, catastrophizing, kinesiophobia, and negative illness perceptions and inversely correlated with functioning. The second hypothesis stated that persons with clinically relevant symptoms of CS have worse psychosocial outcomes related to CNLBP compared with people with fewer of these symptoms.

Methods

Study Design and Setting

This cross-sectional study was reported in accordance with the Strengthening The Reporting of OBservational studies in Epidemiology statement.[38]

Participants were initially recruited to participate in a randomized, controlled, triple-blind trial in which an experimental treatment was compared with a placebo treatment to examine the effectiveness of a new electrotherapy device for the treatment of patients with CNLBP.[39] The protocol was approved by the University Hospital Brussels (Universitair Ziekenhuis Brussel)/Vrije Universiteit Brussel ethics committee and was registered with clinicaltrials.gov (No. NCT02256410).

The study was conducted at the University Hospital Brussels, Belgium, from November 2013 to February 2015. All participants provided their informed consent prior to inclusion in the study.

The present cross-sectional study comprises a subanalysis of the baseline values of the outcome measures of the initial trial.

Participants

Participants, both male and female, were recruited in the pain clinic of the University Hospital Brussels, through an online questionnaire and through flyers and posters in health care centers and pharmacies in Brussels, Belgium. Potential participants were screened during a telephone

Inclusion

- Type of LBP
  - Chronic, at least 3 months
  - Nonspecific
  - Non-radicular
- 18-65 years
- VAS ≥4/10 in the last 24 hours
- Stable treatment regime for at least 1 month

Exclusion

- Type of LBP
  - Acute
  - With a known cause
  - Radicular syndrome
- <18 or >65 years
- VAS <4/10 in the last 24 hours
- Changes in treatment regime in the past month
- Serious or progressive neurologic deficits
- Presence of co-existing chronic pain conditions other than LBP (eg, fibromyalgia)
- Symptoms of serious underlying conditions such as tumor, infection, vertebral compression fracture, ankylosing spondylitis or clinically significant spinal stenosis
- Cardiac pacemaker, defibrillator or other metallic or electronic devices
- Sensory loss in the skin
- Skin inflammation or edema in the lower back
- Pregnant or until 1 year postnatal
- Epilepsy, cancer, arthritis (except osteoarthritis), major psychiatric disorders
- Substance abuses and addictions
- Awaiting surgery or having had surgery in the past 6 months
- BMI ≥30
- Not able to read Dutch or French

Fig 1. *Selection criteria for screening of potential patients.* BMI, *body mass index;* LBP, *low back pain;* VAS, *visual analogue scale.*

interview for selection criteria, listed in Figure 1, prior to participation.

Outcome Measures

Participants were invited to complete the assessments, which consisted of completion of a demographic questionnaire, a number of self-report outcome measures, and a functional test. Outcome measures were, first, the degree of symptoms of CS, which was assessed using the Central Sensitization Inventory (CSI), and second, pain behavior, functioning, pain intensity, pain catastrophizing, kinesiophobia, and illness perceptions.

The CSI consists of 25 symptom-related opinions that the patient has to score on a 5-point Likert scale.[6] Scores ≥40/100 indicate the presence of CS (sensitivity: 81%, specificity: 75%).[40] The CSI has good clinimetric properties for assessing symptoms of CS in patients with chronic pain.[6,41]

The results of this questionnaire were used to divide the sample into 2 subgroups, 1 containing persons with a clinically relevant degree of symptoms of CS (CSI score ≥40) and 1 containing persons with a lower degree of these symptoms (CSI score <40).[40]

Pain behavior was assessed using the 1-minute stair-climbing test, which has been found to be a very reliable test.[42] The patient was asked to climb and descend 5 stairs during 1 min as fast as possible, safely, without running. Total number of stairs climbed and descended was registered.

The Quebec Back Pain Disability Scale was used to assess the patients' limitations in functioning caused by LBP. This questionnaire includes 20 activity-related questions, which the patient has to rate on a 6-point Likert scale (0 = no problem, 5 = not capable). The total score represents a percentage of the patient's limitations.[43,44] This questionnaire is found to be valid, reliable, sensitive, and responsive[45-48] and is recommended for use in a LBP population.[45]

A 100-mm visual analogue scale (VAS) was used to rate current pain intensity and mean pain intensity during the past 7 days. The minimal clinically important difference for LBP is about 18-19 mm.[49] VAS measurements are found to have good test-retest reliability.[50]

To measure the extent of pain catastrophizing, we used the Pain Catastrophizing Scale, which consists of 13 items exploring pain-related cognition, which the patient has to score on a 5-point Likert scale (0 = not at all, 4 = all the time).[51] Scores ≥30/52 reveal a clinically relevant degree of catastrophizing.[52] This questionnaire is found to have good internal consistency and test-retest reliability, as well as proven construct and criterion validity.[53-59]

To measure kinesiophobia, the Tampa Scale for Kinesiophobia was used. Patients have to score 17 opinions on a 4-point Likert scale (1 = highly disagree, 4 = highly agree).[60] Scores ≥37/68 indicate kinesiophobia. This questionnaire has good internal consistency and test-retest reliability.[33]

Finally, patients' illness perceptions were measured based on the Brief Illness Perception Questionnaire. The patient has to rate 8 statements on a 10-point scale (1-10).[61] The higher the score, the greater is the extent to which the patient's illness perceptions are threatening him or her.[61]

Sample Size

Although the patient sample was adopted entirely from the original trial, theoretical a priori sample size calculation was performed for the present study, using G*Power 3 (Düsseldorf, Germany).[62] Effect size, based on the

correlation analysis between CSI and pain catastrophizing, was assumed to be moderate ($\rho = 0.3$); the significance level was set at $\alpha = 0.05$ and power at 0.8. This analysis revealed that the total sample should include at least 64 patients.

Statistical Methods

SPSS Version 22 (IBM, Armonk, New York) was used to perform data analyses. Baseline data were analyzed to describe group characteristics. Pearson correlation tests were used to determine if there were significant associations between CSI and other outcome measures.

After the sample was divided into 2 subgroups, 1 with and 1 without clinically relevant symptoms of CS, continuous variables were analyzed for between-group comparability with independent sample *t*-tests or Mann-Whitney *U*-tests depending on the normality of the data. Non-continuous variables were compared using Pearson χ-square tests. Subsequently, independent sample *t*-tests or Mann-Whitney *U*-tests were used to investigate differences between the 2 groups with respect to pain behavior, functioning, pain levels, illness perceptions, kinesiophobia, and pain catastrophizing. Normality of the data was analyzed using the Kolmogorov-Smirnov test. The significance level was set at .05.

Results

Participant Flow

In total, 39 participants (14 male, 25 female) were included in the original study. All included patients completed the assessments. One participant was excluded later during the initial trial because she was diagnosed with rheumatoid arthritis. Finally, data from 38 persons were analyzed.

Outcomes

Descriptive statistics of the study sample are summarized in Table 1.

Significant associations were found between symptoms of CS and all other outcome parameters (Table 2). A negative correlation was found between the CSI score and the 1-minute stair-climbing test ($r = -0.337$, $P = .039$). All other outcome parameters were positively correlated with the CSI. The highest correlation coefficients were found for the Pain Catastrophizing Scale ($r = 0.518$, $P = .001$), VAS at this moment ($r = 0.510$, $P = .001$), and mean VAS during the past 7 days ($r = 0.505$, $P = .001$).

Twelve patients scored ≥40 on the CSI and 26 scored <40, on the basis of which the sample was divided into 2 subgroups. Statistical analyses revealed no significant differences in demographic parameters between the 2 groups (Table 3). Data from the VAS at this moment, Brief Illness Perception Questionnaire, and 1-min stair-climbing test were not normally distributed; therefore, nonparametric tests were used to compare these outcomes.

Table 1. *Descriptive Statistics*

	Mean ± SD	Min-Max
Age, y	40.76 ± 13.30	18-62
Length, cm	170.11 ± 9.53	150.00-191.00
Weight, kg	72.38 ± 11.38	52.00-107.00
BMI, kg/m^2	24.98 ± 3.16	20.06-33.46
Sex, male [a]	14 (36.8)	
Language, Dutch [a]	27 (71.1)	
Language, French [a]	11 (28.9)	
CSI	32.92 ± 12.76	16-66
PCS	21.70 ± 11.86	0-46
VAS NOW	46.67 ± 27.36	2-100
VAS 7D	50.74 ± 22.81	11-100
Brief IPQ	47.74 ± 13.31	7-77
QBPDS	36.34 ± 17.35	1-79
TSK	39.74 ± 7.98	24-57
1MSCT	90.34 ± 29.94	32-194

1MSCT, 1-min stair-climbing test; *BMI,* body mass index; *Brief IPQ,* Brief Illness Perception Questionnaire; *CSI,* Central Sensitization Inventory; *Min-Max,* minimum-maximum; *PCS,* Pain Catastrophizing Scale; *QBPDS,* Quebec Back Pain Disability Scale; *SD,* standard deviation; *TSK,* Tampa Scale For Kinesiophobia; *VAS 7D,* visual analogue scale past 7 days; *VAS NOW,* visual analogue scale at this moment.
[a] Data are presented as n (%).

Both groups differed significantly on all outcome parameters except the Quebec Back Pain Disability Scale ($P = .128$), as outlined in Table 4. Patients with clinically relevant symptoms of CS scored higher on the questionnaires and lower on the functional test compared with persons with fewer symptoms of CS. The mean Pain Catastrophizing Scale score was >30/52 in persons with symptoms of CS, which indicated a clinically relevant degree of catastrophizing in this subgroup. The mean Tampa Scale for Kinesiophobia score was >37/68 in both groups, indicating that both had kinesiophobia. The scores on the Brief Illness Perception Questionnaire were high in both groups (mean scores >40/80), meaning that threatening illness perceptions seemed to be present in this sample of CNLBP, regardless of their level of CS.

Discussion

This cross-sectional study examined the relationship between symptoms of CS and pain behavior, functioning,

Table 2. *Pearson Correlation for Central Sensitization Inventory*

	n	*r*	Significance
PCS	37	0.518	.001 [a]
VAS NOW	38	0.510	.001 [a]
VAS 7D	38	0.505	.001 [a]
Brief IPQ	38	0.403	.012 [a]
QBPDS	38	0.395	.014 [a]
TSK	38	0.345	.034 [a]
1MSCT	38	–0.337	.039 [a]

1MSCT, 1-min stair-climbing test; *Brief IPQ,* Brief Illness Perception Questionnaire; *n,* number of participants; *PCS,* Pain Catastrophizing Scale; *QBPDS,* Quebec Back Pain Disability Scale; *r*, Pearson correlation coefficient; *TSK,* Tampa Scale for Kinesiophobia; *VAS 7D,* visual analogue scale past 7 days; *VAS NOW,* visual analogue scale at this moment.

[a] Statistically significant correlation.

pain intensity, illness perceptions, kinesiophobia, and pain catastrophizing in patients with CNLBP. The results suggested a significant association between symptoms of CS and all other investigated parameters. In addition, this study examined differences between CNLBP patients exhibiting a clinically relevant degree of symptoms of CS (n = 12) and those with fewer symptoms of CS (n = 26). Patients with more symptoms of CS scored worse on all outcome parameters compared with patients with fewer symptoms, except on the functioning questionnaire.

The relationship between CS and other relevant parameters has been studied in several populations, for example, patients with LBP and associated leg pain,[27] chronic whiplash-associated disorders, fibromyalgia,[63] knee osteoarthritis,[64] and chronic trapezius myalgia.[65] Psychosocial and cognitive behavioral factors such as incorrect illness perceptions, pain catastrophizing, and kinesiophobia could contribute to and sustain the mechanisms of CS,[28,30] and these associations may be bidirectional. Increased levels of CS were found to be associated with high pain catastrophizing levels, low sleep efficiency, and high clinical pain in patients with knee osteoarthritis.[64] Patients with chronic trapezius myalgia and CS exhibited more pain, anxiety, depressive feelings, poor sleep quality, pain catastrophizing, fear avoidance behavior, and disability compared with healthy control subjects.[65] Patients with LBP and leg pain classified with a dominance of CS reported more severe pain; poorer physical and mental health–related quality of life; and greater levels of back pain-related disability, depression, and anxiety compared with those classified with a dominance of peripheral neuropathic pain and nociceptive pain.[27] These results are in line with the results of this cross-sectional study in patients with CNLBP, as all investigated factors seem to be associated with symptoms of CS, particularly pain catastrophizing and self-reported pain, with correlation coefficients >0.50. Furthermore, this trial adds valuable knowledge to the findings of Smart et al[27] in patients with low back and associated leg pain, a population similar to that with CNLBP, as the present study used outcome measures focusing particularly on the patient's pain cognition, whereas the aforementioned study was focused on the association between CS and more general health-related outcomes (eg, 36-Item Short Form Health Survey [SF-36] and Hospital Anxiety and Depression Scale).[27] Sleep efficiency, quality of life, depression, and anxiety were not studied in this cross-sectional study, although these parameters might be an interesting topic for future research because they might also have an important relationship with CS in CNLBP patients.

The associations between the presence of symptoms of CS and the variables assessed in the present study may add a more biopsychological level to the onset and maintenance of chronic pain problems: it is possible that heightened CS may affect and be affected by cognition and pain perception, resulting in a vicious circle leading to chronic pain and disability.

In some pathologies (eg, fibromyalgia, chronic fatigue syndrome, and chronic whiplash-associated disorders), the presence of CS is typical.[13,23,66] Previous research has also revealed the possible presence of CS in persons with CNLBP; however, only a subgroup of CNLBP patients appears to exhibit this phenomenon.[8,9,30,67] This is in line with findings of the present study in which a minority (32%) had a clinically relevant degree of symptoms of CS. Strikingly, this subgroup scores significantly worse on all outcome parameters in comparison to the group with fewer of these symptoms. A possible explanation for the role of psychosocial factors in the development and maintenance of chronic pain might be the "fear avoidance model." This popular theory explains the role of psychosocial factors in the development of chronic pain.[68,69] It states that some patients develop pain catastrophizing during a pain episode, leading to fear avoidance behavior and resulting in more disability and chronic pain. Previous research reported the presence of pain catastrophizing and fear avoidance in persons with CNLBP[30,70] and its contribution to pain and disability.[71-73] The results of the present study indicate that patients with a clinically relevant degree of symptoms of CS have significantly more pain, worse pain behavior, and a negative, although not significant, tendency toward daily functioning in comparison to patients with fewer symptoms. According to the aforementioned fear avoidance model, this might be due to the significantly higher scores in pain catastrophizing and kinesiophobia of patients exhibiting a higher degree of symptoms of CS. Therefore, the fear avoidance model might be more applicable to a subgroup of CNLBP patients exhibiting more symptoms of CS, compared with patients exhibiting fewer of these symptoms, although kinesiophobia and threatening illness perceptions, being components of the fear avoidance model,[68,69] were also present to a clinically relevant degree in the subgroup of patients exhibiting few symptoms of CS, but to a significantly lesser extent.

An interesting topic for future research might be to unravel why some patients with CNLBP who manifest pain catastrophizing, fear avoidance behavior, and possibly worse illness perceptions develop CS, whereas others do not. In this way, important biopsychosocial factors could be

Table 3. *Descriptive Data for Subgroups With and Without Central Sensitization Symptoms*

	CSI ≥40 (n = 12)		CSI <40 (n = 26)		
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Significance
Age, y	40.58 ± 12.03	21-60	40.85 ± 14.08	18-62	.962
Length, cm	167.92 ± 9.73	153.00-184.00	171.11 ± 9.45	150.00-191.00	.594
Weight, kg	72.21 ± 14.89	55.50-107.00	72.46 ± 9.70	52.00-90.00	.169
BMI, kg/m^2	25.45 ± 3.53	21.15-31.60	24.69 ± 3.03	20.06-33.46	.291
Language, Dutch [a]	6 (50.00)		21 (80.77)		.520
Sex, male [a]	6 (50.00)		8 (30.77)		.253

BMI, body mass index; *CSI,* Central Sensitization Inventory; *Min-Max,* minimum-maximum; *SD,* standard deviation.
[a] Data are presented as n (%).

Table 4. *Outcome Measures for Subgroups With and Without Central Sensitization Symptoms and Statistical Analysis*

	CSI ≥40		CSI <40		Independent Sample *t*-Test		
	Mean (SD)	Min-Max	Mean (SD)	Min-Max	df	*F*	Significance
PCS	30.55 (11.97)	7-46	17.96 (9.80)	0-40	35	0.431	.002 [a]
VAS 7D	65.58 (23.28)	11-100	43.89 (19.41)	12-81	36	0.069	.005 [a]
TSK	44. 83 (7.43)	33-57	37.39 (7.18)	24-47	36	0.005	.006 [a]
QBPDS	42.67 (17.25)	22-79	33.42 (16.92)	1-63	36	0.015	.128

	CSI ≥40		CSI <40		Mann-Whitney *U*-test			
	Median (IQR)	Min-Max	Median (IQR)	Min-Max	Mean Rank	Sum of Rank	Significance	Group
1MSCT	77.50 (60.50-85.00)	32-94	100.00 (79.00-108.75)	44-194	12.04	144.5	.005 [a]	CSI ≥40
					22.94	596.5		CSI <40
VAS NOW	70.50 (59.50-83.75)	10-100	32.00 (22.50-56.25)	2-86	26.42	321	.009 [a]	CSI ≥40
					16.31	420		CSI <40
Brief IPQ	59.00 (43.00-69.00)	32-77	43.00 (40.25-50.75)	7-60	26.04	312.5	.013 [a]	CSI ≥40
					16.48	428.5		CSI <40

1MSCT, 1-min stair-climbing test; *Brief IPQ,* Brief Illness Perception Questionnaire; *CSI,* Central Sensitization Inventory; *df,* degrees of freedom; *F,* sum of squares; *IQR,* interquartile range (Q1-Q3); *Min-Max,* minimum-maximum; *PCS,* Pain Catastrophizing Scale; *QBPDS,* Quebec Back Pain Disability Scale; *SD,* standard deviation; *TSK,* Tampa Scale for Kinesiophobia; *VAS 7D,* visual analogue scale past 7 days; *VAS NOW,* visual analogue scale at this moment.
[a] Statistically significant difference.

defined that might contribute to, cause, and maintain the CS process. Alternatively, it would be interesting to investigate if CS, whether partly genetic or learned, might also be a driving force of such psychological factors.

Limitations

First, this cross-sectional study used a small sample of 38 participants. The sample size calculation revealed that the study was underpowered. However, post hoc power analysis based on the effect size of the correlation analysis between the CSI and Pain Catastrophizing Scale ($r = 0.581$, $P = .001$) revealed a power of 0.99 for the current sample size of 37 patients ($\alpha = 0.05$). Additionally, the study sample was divided into 2 groups to compare patients with a clinically relevant degree of symptoms of CS and patients with fewer symptoms, which led to a further decrease in patient numbers for the analyses performed on these subgroups. Post hoc power analysis for this subgroup analysis based on the results of both subgroups on the Pain Catastrophizing Scale still revealed a power of 0.88 for the current sample of 11 patients in the subgroup with a clinically relevant degree of symptoms of CS and 26 patients in the other group, with $\alpha = 0.05$. Although the sample size of this study is rather small, the results of the post hoc power analyses make it hard to ethically justify the inclusion of extra patients in this analysis.

Throughout this study, the term *central sensitization* was replaced by *symptoms of CS*, as no other evaluations but the CSI were applied to investigate the presence of CS. The CSI can give an indication of the presence of CS and indicates the presence of at least symptoms of CS. However, the classification of CS has always been difficult because no golden standard method of assessment exists.[4] Recently, Nijs et al[4] proposed the first classification criteria

for CS pain based on the main physiological and clinical features of CS, most importantly the presence of pain disproportionate to the primary injury or pathology,[4,8] generalized hyperexcitability of central nociceptive pathways,[4,8,74] and hypersensitivity of senses unrelated to the musculoskeletal system.[4,8] The CSI is 1 of the criteria used in the proposed classification algorithm by Nijs et al.[4] This classification is above all extremely valuable for the early identification of CS in clinical practice. In further research, it seems appropriate to screen study populations based on these criteria, supplemented by more objective measures that do not rely solely on self-report methods, to make a statement about the presence of CS. For example, widespread hyperalgesia, as 1 of the characteristics of CS,[5,9] can be measured by assessing pressure pain thresholds and tolerance using an algometer in locations unrelated to the lumbar area (eg, lateral epicondyles, midtrapezius regions).[30,75]

Third, based on the analyses performed in this study, no conclusions can be made on the causality of the relations, although high correlation coefficients and significance levels were found for certain parameters. Furthermore, conducting multiple correlations leads to a higher risk of type I errors, that is, false positives. No correction was applied to the analyses to overcome this issue, because, to our knowledge, there is still no "golden standard" correction for multiple correlations, apart from the most popular one, the Bonferroni correction, which is a very conservative approach, leading to large numbers of type II errors, that is, false negatives, in return. However, the results of the present study are straightforward, with all significant correlations, making it hard to question these findings based on this issue.

Because of these limitations, no strong conclusions can be drawn for the general population of patients with CNLBP, although these results provide an indication of the relationship between CS and other relevant psychosocial and cognitive behavioral factors, providing a basis for further research, especially because these results are in line with previous research about CS in chronic pain disorders.[8,63,64,76] Further research should use larger study samples and proper diagnostic criteria of CS.

Conclusion

In patients with CNLBP, we found significant relationships between the presence of symptoms of CS and pain, pain behavior, functioning, pain catastrophizing, kinesiophobia, and illness perceptions. These results are in line with findings of previous trials in different chronic pain populations. A clinically relevant degree of symptoms of CS was present only in a subgroup of the investigated sample, possibly caused by worse psychosocial factors in this subgroup. Patients manifesting more symptoms of CS scored significantly worse on the outcome measures compared with patients with fewer symptoms of CS.

Practical Applications

- This study suggests that pain cognitions might play an important role in the vicious circle of central sensitization in patients with chronic LBP. Therapists should consider implementing interventions addressing pain cognitions, such as pain neuroscience education.
- To our knowledge, previous studies investigating central sensitization in patients with LBP were rather focused on more general health-related outcome measures instead of pain cognitions, which has been found to be associated with the presence of central sensitization in many different patient populations.
- Future research should investigate why central sensitization is only present in a subgroup of patients with chronic LBP.

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