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ORIGINAL RESEARCH

Prevalence and Associated Factors of Central Sensitization Among Patients with Chronic Low Back Pain at Mbarara Regional Referral Hospital, Uganda: A Cross-Sectional Study

Marven Robert Ruyonga[1], Derrick Asiimwe[1], David Collins Agaba[1], Amon Nuwahereza[2], Richard Migisha[1], Ronald Ouma Omolo[1]

[1]Department of Physiology, Faculty of Health Sciences, Mbarara University of Science and Technology, Mbarara, Uganda; [2]Department of Physiotherapy, Faculty of Health Sciences, Mbarara University of Science and Technology, Mbarara, Uganda

Correspondence: Marven Robert Ruyonga, Email marvenrr@gmail.com

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Background: Chronic low back pain (CLBP) is a leading cause of disability worldwide and is increasingly recognized as a condition influenced by altered central pain processing. Central Sensitization (CS), characterized by amplified nociceptive signaling within the Central Nervous System (CNS), may contribute to persistent pain, disability and poor treatment response in patients with CLBP. Despite the high burden of CLBP in Uganda, there is lack of local epidemiological data on CS. This study determined the prevalence and associated factors of CS among patients with CLBP attending Mbarara Regional Referral Hospital (MRRH).

Methods: A hospital-based cross-sectional study was conducted among 304 consecutively recruited adults with CLBP attending MRRH between October 2025 and January 2026. CS was assessed using the Central Sensitization Inventory (CSI), with a score ≥40 defining CS. Sociodemographic, clinical, psychological, and lifestyle data were collected using interviewer-administered questionnaires. Modified Poisson regression identified factors independently associated with CS.

Results: The mean age of participants was 56.5 ± 16.2 years, and 55.9% were female. The prevalence of CS was 33.9% (n=103; 95% CI: 28.9–39.4). Moderate to severe anxiety (aPR=3.24, 95% CI: 2.27–4.63), depression (aPR=1.63, 95% CI: 1.24–2.16), smoking (aPR=1.41, 95% CI: 1.04–1.91), opioid use (aPR=1.36, 95% CI: 1.02–1.81) and topical analgesics (aPR=1.43, 95% CI: 1.004–2.04) were independently associated with CS.

Conclusion: Approximately one-third of patients with CLBP at MRRH exhibited features of CS. CS was associated with psychological distress, smoking, and certain analgesic use patterns. These findings reinforce the need for a biopsychosocial model of care in the assessment and management of CLBP, incorporating evaluation of central pain mechanisms, psychological distress, and other social factors that contribute to pain and disability, while prospective longitudinal studies are needed to clarify temporal relationships and causal pathways.

Keywords: central sensitization, chronic low back pain, central sensitization inventory, pain mechanisms, biopsychosocial model, Uganda

Introduction

CLBP is one of the leading causes of disability worldwide and affects people across all age groups, occupations, and socioeconomic settings.[1] Globally, an estimated 619 million people live with low back pain, making it a major contributor to health-care use, reduced productivity, and impaired quality of life.[2] In Uganda, the burden is similarly substantial, with 64.7% of older adults reported to experience low back pain, disproportionately affecting women.[3] In Mbarara, Uganda, low back pain is a common reason for seeking care, with one local study reporting that approximately 1 in 4 clients attending health centers presented with CLBP.[4] Although most patients with CLBP are classified as having non-specific low back pain, this classification does not mean that the pain is unimportant or biologically unexplained; rather, it highlights the need to understand mechanisms that may sustain pain when a clear structural cause is not identified.[5]

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CS is increasingly recognized as an important pathophysiological mechanism underlying chronic pain conditions, including CLBP. CS refers to an amplification of neural signaling within the CNS that results in hypersensitivity to pain.[6] Persistent nociceptive input from peripheral tissues may lead to increased excitability of dorsal horn neurons, reduced inhibitory pain modulation, and enhanced synaptic transmission mediated by neurotransmitters such as glutamate and substance P.[7,8] These neuroplastic changes can produce hyperalgesia, allodynia, and widespread pain that may persist even after tissue healing has occurred.[9] Psychological stress, anxiety, depression, sleep disturbances, and maladaptive pain coping strategies have also been shown to contribute to the development and maintenance of CS through dysregulation of descending pain pathways.[10,11] Contemporary evidence suggests that chronic non-specific low back pain should be understood within a biopsychosocial framework, in which persistent pain results from complex interactions between biological, psychological, and social factors rather than ongoing tissue damage alone.[12,13] Within this framework, CS is increasingly recognized as one of the dominant pain mechanisms contributing to pain persistence, disability, and poor response to treatments directed solely at structural abnormalities.[14] Consequently, rehabilitation has shifted toward mechanism-based approaches that integrate pain neuroscience education, graded physical activity, psychological interventions, and exercise therapy aimed at restoring function while addressing altered pain processing. Emerging evidence further supports combining aerobic exercise with core stabilization exercises, which has been shown to improve functional capacity, physical performance, and reduce fall risk among older adults with chronic non-specific low back pain, reinforcing the value of multidimensional rehabilitation strategies tailored to underlying pain mechanisms.[15]

Most available evidence on CS among patients with CLBP comes from high-income countries, where health-care access, pain assessment practices, rehabilitation services, and patterns of analgesic use may differ from those in low-resource settings. Although few sub-Saharan African studies have reported CS among patients with CLBP,[16] local data from Uganda remain limited. At MRRH, patients with CLBP are commonly managed in general outpatient, orthopedic, and physiotherapy clinics, yet the proportion with features of CS and the factors associated with it are unknown. Beyond addressing an important evidence gap, identifying CS has practical implications for clinical care in Uganda. Recognition of centrally mediated pain may help clinicians distinguish patients who are less likely to benefit from treatments directed solely at structural pathology and instead require mechanism-based management. Early identification may facilitate more appropriate use of pain neuroscience education, individualized physiotherapy, psychological support, rational analgesic prescribing, and multidisciplinary rehabilitation. Such an approach may improve functional outcomes while reducing unnecessary investigations and prolonged reliance on pharmacological therapies in resource-limited settings. Therefore, this hospital-based cross-sectional study aimed to determine the prevalence and factors associated with CS among patients with CLBP attending MRRH, Uganda.

Methods

Study Design and Setting

We conducted a hospital-based analytical cross-sectional study among adults with CLBP attending MRRH between October 6, 2025 and January 17, 2026. MRRH is a public regional referral hospital located in Mbarara City, southwestern Uganda, and serves patients from Mbarara City and surrounding districts, including Mbarara, Isingiro, Bushenyi, Buhweju, Ibanda, Kiruhura, Kazo, Mitooma, Ntungamo, Sheema, Rwampara, and Rubirizi. Patients with CLBP are commonly managed through the physiotherapy outpatient, general outpatient, and orthopedic clinics. The hospital receives approximately 120 patients with CLBP per month, based on hospital records.

Study Population and Participant Selection

The study population consisted of adults aged 18 years or older with CLBP attending the physiotherapy outpatient, general outpatient, or orthopedic clinics at MRRH during the study period. CLBP was defined as pain in the lumbar or sacral region lasting at least three months. Duration of pain was assessed through participant self-report during screening and verified, where available, using clinical records and previous clinic notes. Patients were eligible if they were aged 18 years or older, had CLBP, and provided written informed consent. Patients were excluded if they had low back pain lasting less than three months, were younger than 18 years, with known neurological or systemic conditions unrelated to

CLBP that might influence central pain mechanisms (for example multiple sclerosis), patients with spinal malignancy, infection, fracture and inflammatory spinal disorders, individuals unable to provide informed consent or personally complete the study questionnaires because of cognitive impairment, altered mental status, severe illness or significant communication difficulties. A consecutive sampling approach was used. Eligible patients were recruited sequentially as they presented to the participating outpatient clinics until the required sample size was achieved.

Sample Size Estimation

The sample size was estimated using the Kish-Leslie formula for a single population proportion.[17] There was no available prior Ugandan or sub-Saharan estimate of the prevalence of CS among patients with CLBP, hence the estimated prevalence of 19.62% from a study in Burkina Faso among African patients with low back pain that assessed tactile allodynia, a phenotype of CS and a clinical manifestation related to altered central pain processing was used.[18] Using a 95% confidence level, a 5% margin of error, and adjustment for 20% non-response or incomplete data, the minimum required sample size was 303 participants.

Study Variables and Measurements

The outcome variable was CS of pain among patients with CLBP assessed using the CSI.[16,19] The CSI is a validated and reliable patient-reported tool[20] and its English version was developed by Mayer et al,[21] which has also been translated into multiple other languages In several studies, the CSI demonstrated high reliability and validity, with a test-retest reliability of 0.82 and a Cronbach's alpha of 0.88. The CSI is a questionnaire divided into two sections: Part A assesses 25 somatic and emotional symptoms linked to CS, while Part B evaluates whether a patient has previously been diagnosed with one or more central sensitivity syndromes or other related conditions.[22] However, only part A was used in this study. Each item on the CSI is scored using a 5-point Likert scale, ranging from 0 = "never" to 4 = "always", with total scores ranging from 0 to 100 points.[21,23] A CSI total score of ≥40 points was used in this study as a cut-off value to identify CS among participants with CLBP.[19] The CSI has not undergone formal psychometric validation in the Ugandan population. The English version was administered because English is the standard language of healthcare delivery in Uganda. For participants requiring clarification, trained interviewers provided standardized verbal explanations in Runyankore (Local language) while maintaining the original meaning of each item.

An interviewer-administered questionnaire was used and it included information on age, sex, education level, financial income, physiotherapy history, smoking status and alcohol use. The questionnaire was developed after review of previous literature on CS and CLBP, including studies assessing psychological, clinical, and lifestyle-related factors associated with CS among patients with chronic musculoskeletal pain.[16,21] Body height was measured by a stadiometer, weight measured by a regularly calibrated digital weight scale and the Body Mass Index (BMI) was calculated (weight [kg]/ height [m^2]), and categorized as underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), or obese (≥30.0 kg/m^2).[21] Pain intensity was assessed using the 11-point Numeric Rating Scale (NRS). The NRS is a pain measurement tool where patients rate their pain on a scale from 0 (no pain) to 10 (worst possible pain).[24] This scale was included in the questionnaire.[21] Depression was assessed using the Patient Health Questionnaire-9 (PHQ-9) and categorized as no/mild versus moderate/severe depression using a cutoff score of ≥10 for moderate-to-severe symptoms[25] and anxiety was assessed using the Generalized Anxiety Disorder-7 scale (GAD-7) and categorized as no/mild versus moderate/severe anxiety using a cutoff score of ≥10 for moderate-to-severe symptoms.[16,26]

Data Collection Procedures

Participants attending the physiotherapy outpatient, general outpatient, and orthopedic clinics were consecutively screened for eligibility by trained research assistants under the supervision of the principal investigator. Screening involved confirming participant age, duration and location of low back pain, ability to provide informed consent, and absence of exclusion conditions such as neurological disorders or cognitive impairment. Duration of low back pain was determined through participant interview and review of available medical records where applicable. Research assistants received two days of training on the study objectives, ethical procedures, standardized administration of study tools, minimization of interviewer bias, handling of sensitive responses, and data management. The questionnaire and study

procedures were pretested among 15 patients with CLBP attending at the health facility, representing approximately 5% of the estimated sample size. These participants were not included in the final analysis. The pretest evaluated clarity, flow, acceptability, comprehension of questionnaire items, duration of interviews, and consistency in administration of the study tools. Minor modifications were subsequently made to improve wording clarity, sequence of questions, and interviewer guidance for selected items. The principal investigator conducted regular supervision and spot checks during data collection to ensure adherence to the study protocol and consistency across data collectors.

Written informed consent was obtained from all participants before data collection. For participants unable to read or write, the consent form was read aloud in a language they understood, and consent was documented using a thumbprint in the presence of an impartial witness. Participants were assigned unique identification codes, and no personal identifiers were recorded in the analytical dataset.

Data Management and Analysis

The collected data were entered using the KoboToolbox software and exported to Stata version 17. Descriptive statistics were used to summarize the data, with the mean and standard deviation applied to normally distributed continuous data, while the median and interquartile range were used for non-normally distributed continuous data. The prevalence of CS was calculated as number of CLBP patients with a CSI score ≥40 over the total sample size and expressed as a percentage. The prevalence was reported with its 95% confidence interval. Chi-square test (or Fisher's exact test where appropriate) was used for categorical variables. Independent *t*-tests or Mann–Whitney *U*-tests were used for continuous variables, depending on the distribution.

Modified Poisson regression with robust standard errors was used to estimate crude and adjusted prevalence ratios and corresponding 95% confidence intervals. This approach was selected because CS was a common outcome, and prevalence ratios were considered more interpretable than odds ratios. Variables associated with CS at $p < 0.20$ in bivariable analyses, together with variables considered clinically important based on prior literature and biological plausibility were entered into the multivariable modified Poisson regression model. Statistical significance in the final model was defined as $p < 0.05$. Before fitting the multivariable model, assumptions for modified Poisson regression were evaluated. Robust standard errors were used to account for potential variance misspecification. Model specification, influential observations, and multicollinearity among predictor variables were assessed before interpretation of the final model. Multicollinearity was assessed using variance inflation factors and all variables included in the multivariable model had variance inflation factors below 5, indicating no evidence of problematic multicollinearity. Adjusted prevalence ratios with 95% confidence intervals were reported.

Results

Participant Flow and Prevalence of Central Sensitization

A total of 330 patients were assessed for eligibility. Of these, 26 were excluded: 19 had low back pain lasting less than three months, six were younger than 18 years, and one had multiple sclerosis. Overall, 304 participants were enrolled and included in the analysis.

Overall, 103 of 304 participants had a CSI score ≥40, corresponding to a prevalence of CS of 33.9% (95% CI: 28.9–39.4) (Figure 1).

Characteristics of Participants

The mean age of participants was 56.5 ± 16.2 years, with female predominance (55.9%). The median CSI score was 32 (IQR 21–44). Excess body weight was common, with 51.3% classified as overweight or obese. Cardio metabolic comorbidities were frequent, particularly hypertension (33.9%) and diabetes mellitus (25.0%). Pain burden was substantial, as 96% of participants reported moderate to severe pain, and 62.8% had used analgesics for more than six weeks. Most participants (77.0%) had received physiotherapy. Psychological distress was evident, with nearly one-third (29.4%) reporting moderate to severe anxiety, while moderate to severe depression was less common (8.8%). Analgesic use was widespread: 59.2% reported paracetamol use, 52.7% NSAID use, and 28.6% opioid use (Table 1).

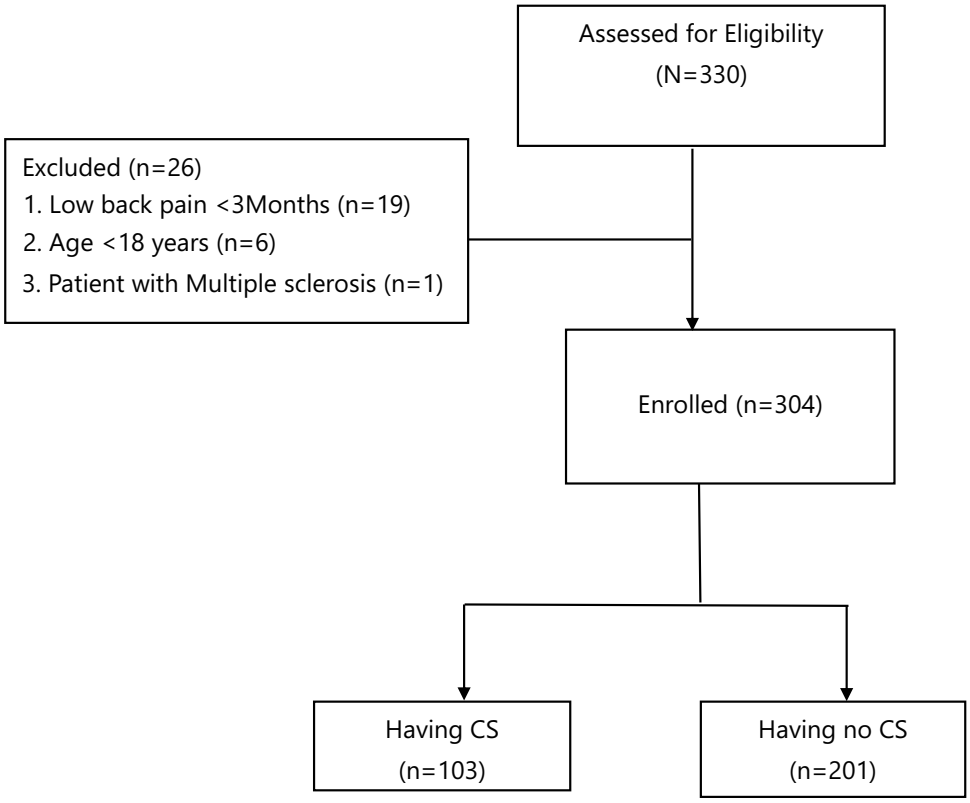


Figure 1 Flow diagram showing enrolment of patients with chronic low back pain and classification by central sensitization status at Mbarara Regional Referral Hospital, Uganda, October 2025–January 2026.

Factors Associated with Central Sensitization

In multivariable analysis, moderate-to-severe anxiety showed the strongest association with CS. Participants with moderate-to-severe anxiety had approximately three times the prevalence of CS compared with those with no or mild anxiety (aPR = 3.24; 95% CI: 2.27–4.63; p<0.001). Moderate-to-severe depression was also associated with a higher prevalence of CS (aPR = 1.63; 95% CI: 1.24–2.16; p=0.001). Smoking (aPR = 1.41; 95% CI: 1.04–1.91; p=0.029), opioid use (aPR = 1.36; 95% CI: 1.02–1.81; p=0.034), and topical analgesic use (aPR = 1.43; 95% CI: 1.004–2.04; p=0.047) were also independently associated with CS (Table 2).

Table 1 Sociodemographic, Psychological and Clinical Factors of Participants (N=304)

Variable	Frequency (N=304) n (%)	With CS (n= 103) n (%)	Without CS (n=201) n (%)	p-value
Age (years)¶				
18-35	34 (11.2)	4 (3.9)	30 (14.9)	0.002
36-59	126 (41.4)	33 (32.0)	93 (46.3)	
≥60	144 (47.4)	66 (64.1)	78 (38.8)	
Sex				
Male	134 (44.1)	48 (46.6)	86 (42.8)	0.526
Female	170 (55.9)	55 (53.4)	115 (57.2)	

(*Continued*)

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Table 1 (Continued).

Variable	Frequency (N=304) n (%)	With CS (n= 103) n (%)	Without CS (n=201) n (%)	p-value
Education				
No formal education	68 (22.4)	30 (29.1)	38 (18.9)	0.015
Primary education	73 (24.0)	31 (30.1)	42 (20.9)	
Secondary education	73 (24.0)	18 (17.5)	55 (27.4)	
Tertiary education	90 (29.6)	24 (23.3)	66 (32.8)	
Smoking status				
No	228 (75.0)	63 (61.2)	165 (82.1)	<0.001
Yes	76 (25.0)	40 (38.8)	36 (17.9)	
Alcohol use				
Yes	99 (32.6)	36 (34.9)	63 (31.3)	0.520
No	205 (67.4)	67 (65.1)	138 (68.7)	
Body Mass Index				
Underweight	8 (2.6)	1 (1)	7 (3.5)	0.429
Normal	140 (46.1)	44 (42.7)	96 (47.8)	
Overweight	110 (36.2)	41 (39.8)	69 (34.3)	
Obese	46 (15.1)	17 (16.5)	29 (14.4)	
Hypertension				
Yes	103 (33.9)	47 (45.6)	56 (27.9)	0.002
No	201 (66.1)	56 (54.4)	145 (72.1)	
Diabetes mellitus				
Yes	76 (25)	32 (31.1)	44 (21.9)	0.080
No	228 (75)	71 (68.9)	157 (78.1)	
Arthritis				
Yes	68 (22.4)	33 (32)	35 (17.4)	0.004
No	236 (77.6)	70 (68)	166 (82.6)	
Heart disease				
Yes	17 (5.6)	10 (9.7)	7 (3.5)	0.025
No	287 (94.4)	93 (90.3)	194 (96.5)	
Pain severity				
Mild	12 (4.0)	2 (2)	10 (5)	0.225
Moderate	158 (52.0)	50 (48.5)	108 (53.7)	
Severe	134 (44.0)	51 (49.5)	83 (41.3)	
Duration of CLBP				
3–12 months	200 (65.8)	63 (61.2)	137 (68.2)	0.471
1–5 years	85 (28)	33 (32)	52 (25.9)	
>5 years	19 (6.2)	7 (6.8)	12 (5.9)	
Analgesic use duration				
<6 weeks	113 (37.2)	35 (34)	78 (38.8)	0.410
≥6 weeks	191 (62.8)	68 (66)	123 (61.2)	
Physiotherapy received				
Yes	234 (77.0)	85 (82.5)	149 (74.1)	0.100
No	70 (23.0)	18 (17.5)	52 (25.9)	

(*Continued*)

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Table 1 (Continued).

Variable	Frequency (N=304) n (%)	With CS (n= 103) n (%)	Without CS (n=201) n (%)	p-value
Anxiety				
No/mild	207 (68.1)	33 (32)	174 (85.6)	<0.001
Moderate/Severe	97 (31.9)	70 (68)	27 (13.4)	
Depression				
No/mild	275 (90.5)	77 (74.8)	198 (98.5)	<0.001
Moderate/Severe	29 (9.5)	26 (25.2)	3 (1.5)	
Analgesia (paracetamol)				
No	124 (40.8)	39 (37.9)	85 (42.3)	0.458
Yes	180 (59.2)	64 (62.1)	116 (57.7)	
Analgesia (NSAIDs)				
No	144 (47.3)	54 (52.4)	90 (44.8)	0.206
Yes	160 (52.7)	49 (47.6)	111 (55.2)	
Analgesia (opioids)				
No	217 (71.4)	62 (60.2)	155 (77.1)	0.002
Yes	87 (28.6)	41 (39.8)	46 (22.9)	
Analgesia (adjuvant)				
No	281 (92.4)	92 (89.3)	189 (94)	0.142
Yes	23 (7.6)	11 (10.7)	12 (6)	
Analgesia (topical)				
No	241 (79.3)	74 (71.8)	167 (83.1)	0.022
Yes	63 (20.7)	29 (28.2)	34 (16.9)	

Notes: ¶Mean age=56.5 ± 16.2 years; Percentages are column percentages.
Abbreviations: CS, Central Sensitization; NSAIDs, Non-Steroidal Anti-Inflammatory Drugs.

Discussion

In this cross-sectional study of adults with CLBP at a regional referral hospital in southwestern Uganda, one-third had features of CS. Psychological distress, particularly moderate-to-severe anxiety and depression, showed the strongest associations, while smoking and selected analgesic-use patterns were also independently associated. These findings provide local evidence that central pain amplification is clinically relevant in routine CLBP care in Uganda and support more individualized, multidisciplinary assessment beyond structural pathology and analgesic-based management.

In the present study, the prevalence of CS among patients with CLBP was 33.9%. The observed prevalence is broadly consistent with studies from other settings that have reported clinically relevant CS among a substantial proportion of patients with CLBP.[21,27] A study from Morocco reported a higher prevalence of CS among patients with CLBP,[16] while a population-based study from Japan reported a slightly lower prevalence of CS.[21] Differences across studies may reflect variation in study populations, clinical settings, pain severity, health-care-seeking patterns, and thresholds used to define CS.

Psychological distress showed the strongest association with CS in this study. Moderate-to-severe anxiety showed the largest association with CS, with participants exhibiting more than a threefold higher prevalence of CS (aPR = 3.24; 95% CI: 2.27–4.63). In contrast, smoking, opioid use, depression, and topical analgesic use demonstrated more modest associations, with adjusted prevalence ratios ranging from 1.36 to 1.63. These findings suggest that psychological distress, particularly anxiety, may have greater clinical relevance than the other associated factors identified in this study (Table 2). These findings are consistent with previous studies showing positive associations between psychological symptoms and CS among patients with chronic pain including CLBP.[16,28] Previous studies suggest that anxiety and

Table 2 Factors Associated with Central Sensitization Among Patients with CLBP at Mbarara Regional Referral Hospital, Uganda, October 2025-January 2026

Variable	cPR	95% CI	*p*-value	aPR	95% CI	*p*-value
Age						
18–35	Ref	-	-	Ref	-	-
36–59	0.98	(0.14–2.10)	0.086	1.76	(0.83–3.70)	0.138
≥60	1.56	(1.15–2.14)	0.005	2.07	(0.97–4.43)	0.060
Education						
Not formal	Ref	-	-	Ref	-	-
Primary	0.96	(0.66–1.40)	0.843	1.10	(0.78–1.55)	0.578
Secondary	0.56	(0.35–0.91)	0.018	0.75	(0.47–1.19)	0.217
Tertiary	0.60	(0.39–0.93)	0.023	0.83	(0.56–1.23)	0.355
Smoking						
No	Ref	-	-	Ref	-	-
Yes	1.90	(1.41–2.57)	<0.001	1.41	(1.04–1.91)	0.029
Analgesia (Opioids)						
No	Ref	-	-	Ref	-	-
Yes	1.65	(1.21–2.24)	0.001	1.36	(1.02–1.81)	0.034
Analgesia (topical)						
No	Ref	-	-	Ref	-	-
Yes	1.50	(1.08–2.08)	0.015	1.43	(1.004–2.04)	0.047
Diabetes						
No	Ref	-	-	Ref	-	-
Yes	1.35	(0.98–1.87)	0.070	0.92	(0.63–1.35)	0.679
Hypertension						
No	Ref	-	-	Ref	-	-
Yes	1.64	(1.20–2.23)	0.002	0.94	(0.69–1.28)	0.703
Anxiety						
No/mild	Ref	-	-	Ref	-	-
Moderate/Severe	5.10	(3.63–7.15)	<0.001	3.24	(2.27–4.63)	<0.001
Depression						
No/mild	Ref	-	-	Ref	-	-
Moderate/Severe	3.50	(2.79–4.41)	<0.001	1.63	(1.24–2.16)	0.001

Abbreviations: cPR, crude Prevalence Ratio; aPR, adjusted Prevalence Ratio; CI, Confidence Interval.

depression may contribute to altered central pain processing through mechanisms involving dysregulation of descending pain modulation, neuroinflammation, and maladaptive neuroplasticity, particularly in regions involved in fear and pain processing such as the amygdala, anterior cingulate cortex, and prefrontal cortex.[6,28] This results in increased pain vigilance and lowered pain thresholds, both of which are characteristic of CS. Additionally, studies suggest a linkage between dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis and increased sympathetic nervous system activity, which may enhance central excitability and sustain pain hypersensitivity.[6,28] However, these mechanisms were not evaluated in the present study, and the observed associations should not be interpreted as evidence of causality.

Opioid use was also independently associated with CS. The observed association between opioid use and CS should be interpreted cautiously. Because of the cross-sectional design, the temporal relationship cannot be established, and reverse causation is plausible. Individuals with more severe pain or symptoms consistent with CS may have been more likely to receive opioid therapy rather than opioid use contributing to CS.[29] Nevertheless, prolonged opioid exposure has been associated with opioid-induced hyperalgesia (OIH).[30] OIH shares several neurophysiological features with CS,

including increased dorsal horn neuron excitability and reduced descending inhibitory control. Experimental and clinical studies in CLBP populations have shown that prolonged opioid therapy is associated with enhanced temporal summation and impaired conditioned pain modulation; hallmarks of central pain amplification.[31,32] Mechanistically, sustained opioid exposure may activate N-methyl-D-aspartate (NMDA) receptors, enhance glutamatergic transmission, increase spinal dynorphin levels, and induce microglial activation with release of pro-inflammatory cytokines, thereby facilitating central nociceptive signaling.[8,30,33] The findings reinforce concerns regarding prolonged opioid therapy in chronic non-cancer pain and highlight the importance of mechanism-based management approaches in patients with suspected CS.

Although the use of topical analgesics remained independently associated with CS, the effect size was modest and the lower confidence limit was close to the null value (aPR = 1.43; 95% CI: 1.004–2.04), indicating limited precision. Unlike opioids, topical agents primarily target peripheral nociceptive mechanisms. Their association with CS may therefore reflect treatment-seeking behavior or inadequate response to peripheral therapies among patients with centrally mediated pain. Previous studies have reported higher utilization of multiple analgesic modalities, including topical preparations, among individuals with elevated CSI scores.[6,19] This pattern may indicate a mismatch between pain mechanism and therapeutic target rather than a direct causal role of topical agents in CS. Consequently, this finding should be interpreted cautiously and requires confirmation in prospective studies.

In this study, participants with a history of smoking had a 1.4 times higher prevalence of CS compared to non-smokers. This finding is consistent with evidence linking tobacco use to heightened pain sensitivity and altered central pain modulation in chronic pain populations. Epidemiological studies have shown that smokers are at increased risk of CLBP and report greater pain intensity and disability than non-smokers.[34] More evidence from psychophysical and questionnaire-based studies suggests that smokers exhibit lower pain thresholds, enhanced temporal summation, and impaired endogenous pain inhibition; features consistent with CS.[19,35] Several biological mechanisms may underlie this association. Chronic nicotine exposure has been shown to induce neuroplastic changes within the CNS, including dysregulation of dopaminergic, serotonergic, and noradrenergic systems involved in descending pain modulation.[8] Smoking is also associated with systemic inflammation, oxidative stress, and endothelial dysfunction, which may sensitize spinal and supraspinal nociceptive circuits and impair inhibitory control.[36] Importantly, smoking frequently co-occurs with psychological distress, particularly depression and anxiety, which were independently associated with CS in this study. This suggests that the relationship between smoking and central pain amplification may involve both direct neurobiological mechanisms and indirect psychosocial pathways.[35,37] Some studies further indicate that the association may be partially mediated by factors such as physical inactivity, sleep disturbance, and pain catastrophizing.[38] The findings support smoking history as an important correlate of CS in CLBP and highlight the relevance of lifestyle assessment within a comprehensive biopsychosocial pain evaluation. Although several biological mechanisms have been proposed to explain the association between smoking, opioid use, and altered pain processing, these mechanisms remain speculative in the context of our findings because no neurophysiological assessments were performed.

These findings have important implications for clinical practice in Uganda and other low-resource settings. CLBP is frequently managed using analgesics, physiotherapy, and investigations aimed at identifying structural pathology. However, these approaches may be insufficient for patients whose symptoms are predominantly driven by CS. Identifying CS using simple screening tools such as the CSI may facilitate mechanism-based rehabilitation by helping clinicians tailor treatment according to the underlying pain mechanisms. In addition to appropriate pharmacological management, physiotherapy interventions such as pain neuroscience education, graded exercise, aerobic exercise, and core stabilization exercises may help reduce pain-related disability, improve physical function, and enhance self-management by addressing altered pain processing rather than focusing solely on tissue pathology. Contemporary rehabilitation therefore emphasizes a multidisciplinary biopsychosocial approach, integrating physiotherapy, psychological support, lifestyle modification, and patient education to optimize outcomes in individuals with CLBP. Supporting this approach, Kuzu et al (2025) demonstrated that combining aerobic exercise with core stabilization exercises significantly improved functional capacity, physical performance, and fall risk among older adults with chronic non-specific low back pain, reinforcing the value of individualized rehabilitation strategies informed by underlying pain mechanisms.[15] Collectively, these findings support incorporating assessment of CS into routine clinical evaluation to guide more targeted rehabilitation and improve patient outcomes.

Limitations

Despite its strengths, this study had a few limitations that should be considered when interpreting the findings. The cross-sectional design limits the ability to infer causality between CS and its associated factors. Temporal relationships cannot be established, and reverse causation is possible. The study also relied on self-reported measures, including the CSI and psychological screening tools, which may be subject to recall and reporting bias. Although the CSI has demonstrated good validity and reliability internationally, it is an indirect measure and should not be considered a definitive diagnostic test. The CSI captures self-reported symptoms commonly associated with altered central pain processing but cannot directly assess the underlying neurophysiological mechanisms. Consequently, CSI findings should be interpreted alongside comprehensive clinical assessment and, where feasible, objective measures of pain processing. In addition, residual confounding cannot be excluded. Although several important sociodemographic, clinical, psychological, lifestyle, and treatment-related variables were included in the analyses, other factors known to influence CS and chronic pain; including; sleep disturbances, physical activity levels, pain catastrophizing, fear-avoidance beliefs, social support, and detailed socioeconomic circumstances were not comprehensively assessed. Furthermore, although pain duration and pain intensity were measured, residual confounding related to these variables may remain. The study was conducted in a single healthcare facility, which may limit the generalizability of the findings to other populations or settings. Finally, the use of analgesic medication was assessed based on participant report without detailed evaluation of dosage, duration, or adherence, which may have affected the observed relationships.

Conclusion

Approximately one-third of patients with CLBP at MRRH exhibited features of CS. Psychological distress, smoking, and certain analgesic use patterns were associated with CS. These findings highlight the importance of considering centrally mediated pain mechanisms in the clinical assessment of CLBP. Incorporating routine evaluation of CS and psychological distress into patient management may support more individualized, multidisciplinary approaches to care in this setting. However, since this study used self-reported measures (CSI) to assess CS, the findings should be interpreted with caution. Future prospective longitudinal studies are needed to clarify the temporal relationships between CS and its associated factors. In addition, randomized controlled trials evaluating mechanism-based rehabilitation approaches, including pain neuroscience education, graded exercise, aerobic exercise, core stabilization, and multidisciplinary interventions, are warranted to determine their effectiveness in improving outcomes among patients with CLBP and CS.

Data Sharing Statement

The datasets used and/or analyzed during this study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the principles of Declaration of Helsinki. Ethical clearance was obtained from the Research and Ethics Committee of Mbarara University of Science and Technology (Approval number: MUST-2025-415). Administrative clearance was sought from the Hospital Director of Mbarara Regional Referral Hospital. All participants gave voluntary informed consent to participate in this study.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests in this work.

References

1. Aoyagi K, He J, Nicol AL, et al. A subgroup of chronic low back pain patients with central sensitization. *Clin J Pain*. 2019;35(11):869–879. doi:10.1097/AJP.0000000000000755
2. Ferreira ML, de Luca K, Haile LM. Global, regional, and national burden of low back pain, 1990-2020, its attributable risk factors, and projections to 2050: a systematic analysis of the global burden of disease study 2021. *Lancet Rheumatol*. 2023;5(6):e316–e29. doi:10.1016/S2665-9913(23)00098-X
3. Wang C, Pu R, Ghose B, Tang S. Chronic musculoskeletal pain, self-reported health and quality of life among older populations in South Africa and Uganda. *Int J Environ Res Public Health*. 2018;15(12):2806. doi:10.3390/ijerph15122806
4. Harscouët P, Chimpaye GN, Kazibwe H, et al. Managing low-back pain in rural Uganda: a qualitative study exploring the perspectives and practices of frontline health workers regarding LBP management in primary care. *BMC Musculoskeletal Disord*. 2025;26(1):168. doi:10.1186/s12891-024-08164-9
5. Pinto EM, Neves JR, Laranjeira M, Reis J. The importance of inflammatory biomarkers in non-specific acute and chronic low back pain: a systematic review. *Europ Spine J*. 2023;32(9):3230–3244. doi:10.1007/s00586-023-07717-1
6. Nijs J, George SZ, Clauw DJ, et al. Central sensitisation in chronic pain conditions: latest discoveries and their potential for precision medicine. *Lancet Rheumatol*. 2021;3(5):e383–e92. doi:10.1016/S2665-9913(21)00032-1
7. Cayrol T, van den Broeke EN. Central sensitisation: causes, therapies, and terminology. *Lancet Rheumatol*. 2021;3(8):e548. doi:10.1016/S2665-9913(21)00176-4
8. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain*. 2011;152(3 Suppl):S2–s15. doi:10.1016/j.pain.2010.09.030
9. Williams DA. Phenotypic features of central sensitization. *J Appl Biobehav Res*. 2018;23(2). doi:10.1111/jabr.12135
10. Li W, Gong Y, Liu J, et al. Peripheral and central pathological mechanisms of chronic low back pain: a narrative review. *J Pain Res*. 2021;14:1483–1494. doi:10.2147/JPR.S306280
11. Curatolo M, Arendt-Nielsen L. Central hypersensitivity in chronic musculoskeletal pain. *Physical Med Rehabil Clin North America*. 2015;26(2):175–184. doi:10.1016/j.pmr.2014.12.002
12. Meeus M, Nijs J. Central sensitization: a biopsychosocial explanation for chronic widespread pain in patients with fibromyalgia and chronic fatigue syndrome. *Clin Rheumatol*. 2007;26(4):465–473. doi:10.1007/s10067-006-0433-9
13. van Dijk H, Köke AJA, Elbers S, Mollema J, Smeets R, Wittink H. Physiotherapists using the biopsychosocial model for chronic pain: barriers and Facilitators-A scoping review. *Int J Environ Res Public Health*. 2023;20(2):1634. doi:10.3390/ijerph20021634
14. Wallden M, Nijs J. Before & beyond the pain - allostatic load, central sensitivity and their role in health and function. *J Bodywork Movement Ther*. 2021;27:388–392. doi:10.1016/j.jbmt.2021.04.003
15. Kuzu Ş, Canli M, Valamur İ, Özüdoğru A, Alkan H, Hartavi A. Effects of aerobic exercise in addition to core stabilization exercises on functional capacity, physical performance and fall risk in geriatric individuals with chronic non-specific low back pain. *BMC Sports Sci Med Rehabil*. 2025;17(1):218. doi:10.1186/s13102-025-01271-7
16. Dahmani D, Taik FZ, Berrichi I, Fourtassi M, Abourazzak FE. Impact of central sensitization on pain, disability and psychological distress in patients with knee osteoarthritis and chronic low back pain. *BMC Musculoskeletal Disord*. 2023;24(1):877. doi:10.1186/s12891-023-07019-z
17. Raimondo D, Raffone A, Renzulli F, et al. Prevalence and risk factors of central sensitization in women with endometriosis. *J Minimally Invasive Gynecol*. 2023;30(1):73–80.e1. doi:10.1016/j.jmig.2022.10.007
18. Ouédraogo DD, Nonguierma V, Napon C, et al. Prevalence of neuropathic pain among Black African patients suffering from common low back pain. *Rheumatol Int*. 2012;32(7):2149–2153. doi:10.1007/s00296-011-1945-4
19. Neblett R, Cohen H, Choi Y, et al. The central sensitization inventory (CSI): establishing clinically significant values for identifying central sensitivity syndromes in an outpatient chronic pain sample. *J Pain*. 2013;14(5):438–445. doi:10.1016/j.jpain.2012.11.012
20. Neblett R, Hartzell MM, Williams M, Bevers KR, Mayer TG, Gatchel RJ. Use of the central sensitization inventory (CSI) as a treatment outcome measure for patients with chronic spinal pain disorder in a functional restoration program. *Spine J*. 2017;17(12):1819–1829. doi:10.1016/j.spinee.2017.06.008
21. Akeda K, Takegami N, Yamada J, Fujiwara T, Nishimura A, Sudo A. Central sensitization in chronic low back pain: a population-based study of a Japanese Mountain Village. *J Pain Res*. 2021;14:1271–1280. doi:10.2147/JPR.S301924
22. Cuesta-Vargas AI, Neblett R, Chiarotto A, et al. Dimensionality and reliability of the central sensitization inventory in a pooled multicountry sample. *J Pain*. 2018;19(3):317–329. doi:10.1016/j.jpain.2017.11.006
23. Archer KR, Coronado RA, Haislip LR, et al. Telephone-based goal management training for adults with mild traumatic brain injury: study protocol for a randomized controlled trial. *Trials*. 2015;16(1):244. doi:10.1186/s13063-015-0775-1
24. Chiarotto A, Maxwell LJ, Ostelo RW, Boers M, Tugwell P, Terwee CB. Measurement properties of visual analogue scale, numeric rating scale, and pain severity subscale of the brief pain inventory in patients with low back pain: a systematic review. *J Pain*. 2019;20(3):245–263. doi:10.1016/j.jpain.2018.07.009
25. Costantini L, Pasquarella C, Odone A, et al. Screening for depression in primary care with patient health questionnaire-9 (PHQ-9): a systematic review. *J Affect Disord*. 2021;279:473–483. doi:10.1016/j.jad.2020.09.131
26. Aktürk Z, Hapfelmeier A, Fomenko A, et al. Generalized anxiety disorder 7-item (GAD-7) and 2-item (GAD-2) scales for detecting anxiety disorders in adults. *Cochrane Database Syst Rev*. 2025;3(3):Cd015455. doi:10.1002/14651858.CD015455

27. Roldán-Jiménez C, Pérez-Cruzado D, Neblett R, Gatchel R, Cuesta-Vargas A. Central sensitization in chronic musculoskeletal pain disorders in different populations: a cross-sectional study. *Pain Med*. 2020;21(11):2958–2963. doi:10.1093/pm/pnaa069
28. Miki T, Nishigami T, Takebayashi T, Yamauchi T. Association between central sensitivity syndrome and psychological factors in people with presurgical low back pain: a cross-sectional study. *J Orthopaedic Sci*. 2021;26(3):337–342. doi:10.1016/j.jos.2020.03.017
29. Fishbain DA, Cole B, Lewis J, Rosomoff HL, Rosomoff RS. What percentage of chronic nonmalignant pain patients exposed to chronic opioid analgesic therapy develop abuse/addiction and/or aberrant drug-related behaviors? A structured evidence-based review. *Pain Med*. 2008;9 (4):444–459. doi:10.1111/j.1526-4637.2007.00370.x
30. Mao J. Opioid-induced abnormal pain sensitivity: implications in clinical opioid therapy. *Pain*. 2002;100(3):213–217. doi:10.1016/S0304-3959(02)00422-0
31. Lee M, Silverman SM, Hansen H, Patel VB, Manchikanti L. A comprehensive review of opioid-induced hyperalgesia. *Pain Physician*. 2011;14 (2;3):145–161. doi:10.36076/ppj.2011/14/145
32. Younger JW, Chu LF, D'Arcy NT, Trott KE, Jastrzab LE, Mackey SC. Prescription opioid analgesics rapidly change the human brain. *Pain*. 2011;152(8):1803–1810. doi:10.1016/j.pain.2011.03.028
33. Grace PM, Hutchinson MR, Maier SF, Watkins LR. Pathological pain and the neuroimmune interface. *Nat Rev Immunol*. 2014;14(4):217–231. doi:10.1038/nri3621
34. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between smoking and low back pain: a meta-analysis. *Am J Med*. 2010;123(1):87.e7–35. doi:10.1016/j.amjmed.2009.05.028
35. Ditre JW, Heckman BW, Zale EL, Kosiba JD, Maisto SA. Acute analgesic effects of nicotine and tobacco in humans: a meta-analysis. *Pain*. 2016;157(7):1373–1381. doi:10.1097/j.pain.0000000000000572
36. Pisinger C, Godtfredsen NS. Is there a health benefit of reduced tobacco consumption? A systematic review. *Nicotine Tobacco Res*. 2007;9 (6):631–646. doi:10.1080/14622200701365327
37. Nijs J, Apeldoorn A, Hallegraeff H, et al. Low back pain: guidelines for the clinical classification of predominant neuropathic, nociceptive, or central sensitization pain. *Pain Physician*. 2015;18(3;5):E333–46. doi:10.36076/ppj.2015/18/E333
38. Vlaeyen JWS, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. *Pain*. 2012;153(6):1144–1147. doi:10.1016/j.pain.2011.12.009

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