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Central Sensitization in Chronic Low Back Pain: Moving Beyond Structural Pathology Toward a Biopsychosocial Approach

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Abstract

Chronic low back pain (CLBP) remains a major cause of disability and healthcare utilization worldwide. Although structural abnormalities are frequently emphasized in the assessment and management of CLBP, altered pain processing may contribute substantially to persistent symptoms in some patients. Central sensitization (CS) represents an important pain mechanism characterized by increased responsiveness of central nociceptive pathways. In a recent hospital-based study among adults with CLBP in Uganda, we found that features consistent with CS were present in approximately one-third of participants and were associated particularly with psychological distress, smoking, and selected analgesic-use patterns. These findings highlight the importance of considering psychological, behavioral, and treatment-related factors alongside structural pathology when evaluating persistent low back pain. In resource-limited settings, recognizing clinically relevant pain mechanisms may provide an opportunity for more individualized and multidisciplinary care. However, because CS was assessed using self-reported instruments, these findings should not be interpreted as a definitive diagnosis of neurophysiological sensitization. Future longitudinal and mechanistic studies are needed to clarify causal pathways and inform context-appropriate interventions.

Keywords: Central sensitization; Chronic low back pain; Pain mechanisms; Psychological distress; Biopsychosocial model; Uganda

Abbreviations: CLBP: Chronic Low Back Pain; CS: Central Sensitization; CSI: Central Sensitization Inventory; aPR: adjusted Prevalence Ratio

Introduction

CLBP is one of the leading causes of disability globally and affects individuals across different age groups and socioeconomic settings [1]. The traditional assessment of CLBP has largely focused on identifying structural abnormalities and excluding specific pathologies. However, structural findings do not always correspond with pain severity or functional limitation, particularly among patients with persistent non-specific low back pain [2]. CS has emerged as an important mechanism in the understanding of chronic pain. It describes increased responsiveness of central nociceptive pathways, potentially resulting in amplified and prolonged pain responses [3]. Importantly, CS should not be viewed as a diagnosis synonymous with chronic pain, but rather as a potential mechanism or phenotype that may contribute to persistent symptoms in selected patients [4]. This distinction has important implications for clinical practice. If pain is approached exclusively because of structural pathology, patients whose symptoms are maintained by altered pain processing, psychological distress, or other biopsychosocial factors may receive interventions that do not fully address their pain experience [5,6].

What Do Our Findings Tell Us?

In our recent cross-sectional study conducted at MRRH, 103 of 304 adults with CLBP had CSI scores ≥40, corresponding to a prevalence of 33.9% (95% CI: 28.9-39.4%). Moderate-to-severe anxiety demonstrated the strongest association with CS, with affected participants having more than three times the prevalence of CS compared with those with no or mild anxiety (aPR 3.24; 95% CI: 2.27-4.63). Depression, smoking, opioid use, and topical analgesic use were also independently associated with CS [7]. The particularly strong association between psychological distress and CS reinforces the importance of viewing persistent low back pain through a biopsychosocial lens. Psychological distress does not imply that pain is "psychological" or imaginary.

Rather, anxiety and depression may interact with pain processing, coping, disability, and symptom persistence in

complex ways. The association between smoking and CS is also clinically relevant. Although our cross-sectional design does not allow causal conclusions, the finding supports consideration of potentially modifiable behavioral factors when assessing patients with persistent pain. Similarly, the observed association between opioid use and CS requires cautious interpretation. Because of the cross-sectional design, it is impossible to determine whether opioid exposure contributes to altered pain sensitivity or whether patients with more severe or persistent pain are more likely to receive opioids. Nevertheless, the finding reinforces the need for careful, individualized analgesic strategies in CLBP.

Implications for Orthopedic and Rheumatology Practice

Recognition of features suggestive of CS may be particularly valuable in patients with persistent symptoms despite conventional treatment or with a combination of pain, fatigue, sleep disturbance, and psychological distress, particularly when symptoms are not adequately explained by structural findings alone [8,9]. For orthopedic and rheumatology clinicians, this does not mean abandoning structural assessment. Rather, structural evaluation and assessment of pain mechanisms should be complementary. Identifying potentially relevant psychological and behavioral factors may help clinicians develop treatment plans that extend beyond analgesic medication and repeated investigations.

A biopsychosocial approach may include appropriate patient education, reassurance regarding pain mechanisms, psychological assessment, graded physical activity and exercise, physiotherapy, and multidisciplinary rehabilitation where available [5,6]. Such approaches may be particularly relevant in low-resource settings, where expensive investigations and specialized pain services may not always be readily accessible. Importantly, the CSI should be interpreted as a symptom-assessment instrument rather than a definitive diagnostic test for neurophysiological CS [10,11]. In our study, CS was assessed using the self-reported CSI, and the instrument has not undergone formal psychometric validation in the Ugandan population [7]. Consequently, CSI findings should be interpreted alongside clinical assessment rather than in isolation.

The Way Forward in Low-Resource Settings

The burden of CLBP is substantial, yet pain mechanisms beyond structural pathology remain relatively underexplored in many low- and middle-income countries. Our findings provide preliminary local evidence that clinically relevant features of CS occur among Ugandan patients with CLBP and are associated with psychological distress and selected behavioral and treatment-related factors. Future research should move beyond prevalence estimates toward longitudinal studies capable of establishing temporal relationships between psychological distress, medication exposure, behavioral factors, CS, and clinical outcomes.

Research should also explore culturally appropriate and feasible multidisciplinary interventions for patients exhibiting features of altered pain processing. For clinicians, the immediate message is straightforward: persistent low back pain should not always be interpreted through a structural lens alone. Recognizing the potential contribution of altered pain processing and biopsychosocial factors may facilitate more individualized assessment and management, without diminishing the importance of appropriate evaluation for structural and specific causes of low back pain.

Conclusion

CS appears to be clinically relevant among a substantial proportion of patients with CLBP in Uganda. Our findings demonstrate associations between CS and psychological distress, smoking, opioid use, and topical analgesic use. While these findings cannot establish causality, they support a broader approach to CLBP assessment that considers pain mechanisms alongside structural pathology. Integrating this perspective into orthopedic, rheumatologic, physiotherapy, and primary care practice may contribute to more individualized and multidisciplinary management of persistent low back pain.

Conflict of Interest

The author declares that there is no conflict of interest.

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