CORRESPONDENCE

Commentary on "Prevalence of Central Sensitization in Notalgia Paresthetica Patients and Its Association with Vertebral Degeneration: A Cross-sectional Study"

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Dear Editor,

I read the paper by Cirakoglu et al. (1) published in your journal recently. The authors state that they *"aimed to investigate the relationships between clinical findings, radiological features, and central sensitization (CS) in notalgia paresthetica patients."* It appears that Cirakoglu et al., as several others in the field of pain neuroscience, are conflating "central sensitization" with the syndrome of "fibromyalgia." They write in their methods section: *"CSI was used to evaluate the presence of CS in the patients"* and *"In the scoring done on a scale of 100, scores of 40 or higher indicate the presence of CS."* Presenting the CSI as a tool for the screening or diagnoses of central sensitization is misleading and leads them to misrepresenting their findings, erroneous interpretation of these findings, and groundless mechanistic conclusions.

"Central sensitization" (2) refers to a neurological dysfunction of the central nervous system that gives rise to abnormal hyperexcitability of nociceptive circuits and hypersensitivity to sensory input, an improper amplified response to noxious and non-noxious stimuli with or without justified peripheral nociceptor activation, due to structural and functional alterations in the spinal cord dorsal horn and brain, postulated to involve a combination of neuronal hyperexcitability, impaired descending inhibitory control, glial reactivity and a complex neuropathological processes of dysregulated central pain processing pathways (3–5).

The Central Sensitization Inventory (CSI) (6), originally developed by Mayer & Neblett et al., (2012), is commonly used in research to assess symptoms of what pain researchers call "central sensitivity syndromes" (or "central sensitization associated symptoms"), such as fibromyalgia, migraine headache, irritable bowel syndrome, and associated chronic pain conditions. These are syndromes whose symptoms are mechanistically attributed to central sensitization according to contemporary mainstream literature (7,8). The CSI, which is a questionnaire of patient self-reported clinical manifestations, inquiring mainly into sleep problems, pain, fatigue, hypervigilance, stress, and mood (with 25 items rated on a Likert scale from 0 to 4 with the same weight for each item), appears to lack a theoretical basis tied to the underlying mechanisms of central sensitization. Instead, its construction relied primarily on knowledge on firsthand experiences reported by patients with fibromyalgia-type symptoms and by healthcare professionals dealing with patients in clinical practice. As described for its construction process by Mayer & Neblett et al. in their publication's methods section: (9) *"An interdisciplinary team that included physicians (psychiatrists and orthopedic surgeons), rehabilitation specialists, clinical psychologists, health psychologists, and psychophysiological specialists, who work exclusively with individuals with chronic pain conditions, developed the items for this Inventory."* (9) This approach, while valuable for capturing patient symptomatology, fails to employ the rigor typically expected in tools that aim to assess pathobiological or physiological mechanisms. The CSI may have good internal and external validity for identifying fibromyalgia symptomatology as a phenomenon (10), but that's not the same as diagnosing central sensitization (11).

Here's the problem: Cirakoglu et al., aiming to investigate the relationships between clinical findings, radiological features and central sensitization (CS) in *notalgia paresthetica* patients, used the following tools: Visual Analog Scale (VAS-pain) for assessing severity of pain, PainDETECT Questionnaire for neuropathic pain symptoms, and Central Sensitization Inventory (CSI) to evaluate the presence of CS in the patients. They found that notalgia paresthetica patients with "central sensitization" had significantly higher VAS pain scores (p<0.001), PainDETECT scores (p=0.005), and that "central sensitization was present in 48.9 % of notalgia paresthetica patients." Basically, they set out to test the association between variable X ("central sensitization") and variable Y (visual analogue scale for pain or painDETECT), without acknowledging that X and Y are represented by overlapping constructs, and, unsurprisingly, found a significant association. Item number 2 in the CSI questionnaire is: "My muscles feel stiff and achy" rated on a Likert scale scored from 0 to 4. Consequently, they conclude that *"central sensitization is common in notalgia paresthetica patients ..."* Do you follow? It's a fallacy. They are conflating the theory and the empirics. You have pain on one side of the equation and pain on the other side of the equation. Pain correlates with pain. Pain is significantly associated with pain. Is there anything new under the sun? High correlations are mathematically inevitable due to construct overlap, rendering the association biologically noninformative. They never truly tested the null hypothesis and their

conclusion is central sensitization irrespective of the actual results. This is clinically relevant for two main reasons: (*i*) preventing scientific misinformation: if left unaddressed, the fallacy of circular reasoning in central sensitization research risks establishing a flawed evidence base for the pathophysiology medical conditions. (*ii*) Clinical implications: since treatment protocols increasingly rely on central nervous system sensitization models (see discussion: *"Multidisciplinary evaluation considering CS status may improve management of NP."*),(1) it is important that these models are built on rigorous scientifically sound data.

The development of the CSI isn't based on anything related to a certain known mechanism, neither did it involve any neurophysiological measurements, but is based on the known symptomatology of fibromyalgia. In other words, despite its distinct name, it doesn't demonstrate anything that is theory specific. It can reflect any other mechanism just the same. The theory of the pathophysiology of fibromyalgia is still, as far as I am aware, under dispute across the field (12–15).

Dermatologists should be aware that investigating an observed phenomenon (e.g. "fibromyalgia-type symptoms") while attributing it to a supposed underlying mechanism – and then trying to investigate that same mechanism by using the phenomenon as its proxy – is a methodologic tautology, circular reasoning. It's self-persuasion, confirmation bias. Central sensitization is thus an axiom. There is a reason we don't use the Centor criteria to diagnose streptococcal pharyngitis. Anyone can call a tool "the Central Sensitization Detecting Device," but it does not make it a central sensitization detecting device. Before using a tool, a scientist should first ask: where did it come from, how was it constructed, was it validated, and what does it measure? The mechanism is not determined by terminology. Fever, melancholy, and sore throat (i.e., "streptococcal pharingitis symptoms") are not valid scientific evidence of streptococcal infection because it can be viral.

There is a theory, and there are empirics. The empirics are not the theory.

Reply to the Commentary on: "Prevalence of Central Sensitization in Notalgia Paresthetica Patients and Its Association with Vertebral Degeneration: A Cross-sectional Study"

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We would like to thank the author for the careful reading of our article and for the thoughtful and detailed critique. We appreciate the opportunity to clarify several conceptual and methodological aspects of our study, particularly regarding the interpretation of central sensitization (CS) and the use of the Central Sensitization Inventory (CSI).

We fully agree that central sensitization is a complex neurophysiological process that cannot be directly diagnosed using a single self-reported instrument. In our study, the CSI was not intended to serve as a definitive diagnostic tool for CS, but rather as a validated instrument to assess symptoms commonly associated with central sensitization. We acknowledge that certain expressions in our manuscript, such as "presence of CS," may have suggested a stronger interpretation than intended. Our aim was to identify patients with a higher burden of CS-related symptoms, rather than to establish CS as a confirmed neurophysiological mechanism.

We also agree that the CSI reflects symptomatology rather than underlying pathophysiological mechanisms. However, its use in our study was consistent with its widespread application in the literature as a tool to capture symptom clusters associated with central sensitization and related conditions. Accordingly, our findings should be interpreted within this framework, and not as direct evidence of central nervous system dysfunction.

Regarding the concern about circular reasoning, we respectfully believe that our results cannot be reduced to a simple "pain correlates with pain" interpretation. In our study, while pain intensity (VAS) and neuropathic pain characteristics (PainDETECT) did not differ significantly between patients and controls, CSI scores were significantly higher in the patient group. This suggests that the observed differences are not solely driven by pain intensity, but rather reflect a broader spectrum of symptoms associated with central sensitization. Furthermore, the CSI encompasses domains beyond pain, including sleep disturbances, fatigue, and emotional factors, supporting the interpretation that it captures a multidimensional clinical construct.

We agree that caution is required when linking symptom-based measures to specific pathophysiological mechanisms. Our study was not designed to demonstrate the existence of central sensitization as a mechanism in notalgia paresthetica, but rather to explore whether patients exhibit a higher burden

of symptoms commonly associated with central sensitization and whether these are related to clinical features such as pruritus severity, lesion distribution, and vertebral degeneration.

In this context, our findings suggest that patients with notalgia paresthetica exhibit a higher frequency of CS-related symptoms, and that these symptoms are associated with clinically relevant parameters, including pruritus severity and the extent of vertebral degeneration. These observations should be interpreted as exploratory and hypothesis-generating, rather than as definitive mechanistic conclusions.

Finally, we appreciate the author's emphasis on conceptual rigor in central sensitization research. We agree that greater precision in terminology is essential, particularly when distinguishing between symptom-based constructs and underlying biological mechanisms. We believe that our study contributes to the literature by providing clinical insights into notalgia paresthetica and by highlighting associations that warrant further investigation using objective neurophysiological methods.

We thank the author again for the valuable comments.

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