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Original Research Article

Mechanism of Peripheral and Central Sensitisation in Chronic Pain Disorders: A Prospective Observational Study

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Abstract:

Background: Chronic pain disorders are increasingly recognized as conditions involving altered nociceptive processing rather than purely structural abnormalities. Peripheral and central sensitisation play a critical role in amplifying pain perception and maintaining chronicity.

Objective: To evaluate the clinical features and underlying mechanisms of peripheral and central sensitisation in patients with chronic pain disorders.

Methods: A prospective observational study was conducted at Daradia Pain Clinic, Kolkata, over a period of 6 months. A total of 100 patients with chronic pain (>3 months duration) were included. Clinical assessment, pain scoring (VAS), and sensitisation features (allodynia, hyperalgesia) were evaluated. Statistical analysis was performed using SPSS v25 with significance set at $p < 0.05$.

Results: Peripheral sensitisation was observed in 62% of patients, while central sensitisation features were present in 48%. Significant association was found between duration of pain and central sensitisation ($p = 0.01$). Patients with central sensitisation had higher VAS scores (mean 7.8 ± 1.2) compared to those without ($p < 0.001$).

Conclusion: Both peripheral and central sensitisation contribute significantly to chronic pain disorders. Early identification of sensitisation mechanisms is crucial for targeted management.

Keywords: Chronic pain, Peripheral sensitisation, Central sensitisation, Hyperalgesia, Allodynia.

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Introduction

Chronic pain is a complex and multifactorial condition affecting millions worldwide, significantly impairing quality of life and functional capacity [1]. Unlike acute pain, which serves as a protective mechanism, chronic pain persists beyond tissue healing and is often associated with alterations in the nervous system [2].

Peripheral sensitisation refers to increased responsiveness of nociceptors due to inflammation or tissue injury. This process lowers the threshold for activation and enhances pain signals [3]. Inflammatory mediators such as prostaglandins, bradykinin, and cytokines contribute to this heightened sensitivity [4].

Central sensitisation, on the other hand, involves amplification of pain signals within the central nervous system. It is characterized by increased excitability of neurons in the dorsal horn of the spinal cord and altered pain modulation pathways [5]. This results in clinical features such as allodynia (pain from non-painful stimuli) and hyperalgesia (exaggerated pain response) [6].

The transition from acute to chronic pain is often driven by sustained sensitisation mechanisms [7]. Neuroplastic changes, including NMDA receptor activation and reduced inhibitory neurotransmission, play a crucial role [8].

Understanding these mechanisms is essential for effective management, as conventional analgesics often fail in cases dominated by central sensitisation [9]. Targeted therapies, including neuromodulators and cognitive approaches, are increasingly being explored [10].

This study aims to evaluate the prevalence and clinical correlation of peripheral and central sensitisation in patients with chronic pain disorders.

Materials and Methods

Study Design: Prospective observational study.

Study Setting: Daradia Pain Clinic, Kolkata.

Study Duration: 6 months.

Sample Size: 100 patients.

Inclusion Criteria

- Age ≥ 18 years
- Chronic pain (>3 months)
- Willing to participate

Exclusion Criteria

- Acute pain conditions
- Neurological disorders affecting sensation
- Severe psychiatric illness

Data Collection

- Demographic details
- Pain duration and intensity (VAS score)
- Clinical examination for:
  - Allodynia
  - Hyperalgesia
- Classification:
  - Peripheral sensitisation
  - Central sensitisation

Statistical Analysis

- Software: SPSS v25
- Tests: Chi-square test, t-test
- Data expressed as mean ± SD
- Significance: $p < 0.05$

Results

A total of 100 patients were included in the analysis.

Baseline Characteristics: The mean age was 45.3 ± 12.6 years, with a slight female predominance (Table 1).

Table 1: Demographic Profile

Parameter	Value
Mean Age (years)	45.3 ± 12.6
Male (%)	44%
Female (%)	56%

Prevalence of Sensitisation: Peripheral sensitisation was identified in 62% of patients, while central sensitisation was observed in 48% (Table 2, Figure 1).

Table 2: Sensitisation Distribution

Type	Number	Percentage (%)
Peripheral Sensitisation	62	62%
Central Sensitisation	48	48%

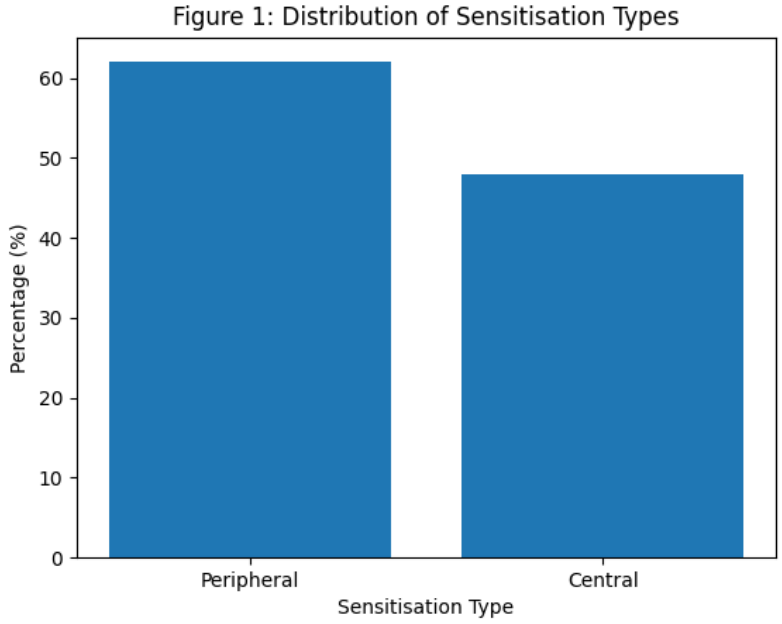

Figure 1: Distribution of Sensitisation Types

Pain Severity (VAS Scores): Patients with central sensitisation reported significantly higher pain scores (Table 3).

Table 3: VAS Score Comparison

Group	Mean VAS ± SD
Peripheral only	6.2 ± 1.1
Central sensitisation	7.8 ± 1.2

Independent sample t-test showed a statistically significant difference ($p < 0.001$).

Association with Duration of Pain: Longer duration of pain (>6 months) was significantly associated with central sensitisation (Table 4, Figure 2).

Table 4: Pain Duration vs Central Sensitisation

Duration	Central Sensitisation (%)	p-value
≤6 months	32%	
>6 months	64%	0.01

Chi-square test demonstrated a significant association ($p = 0.01$).

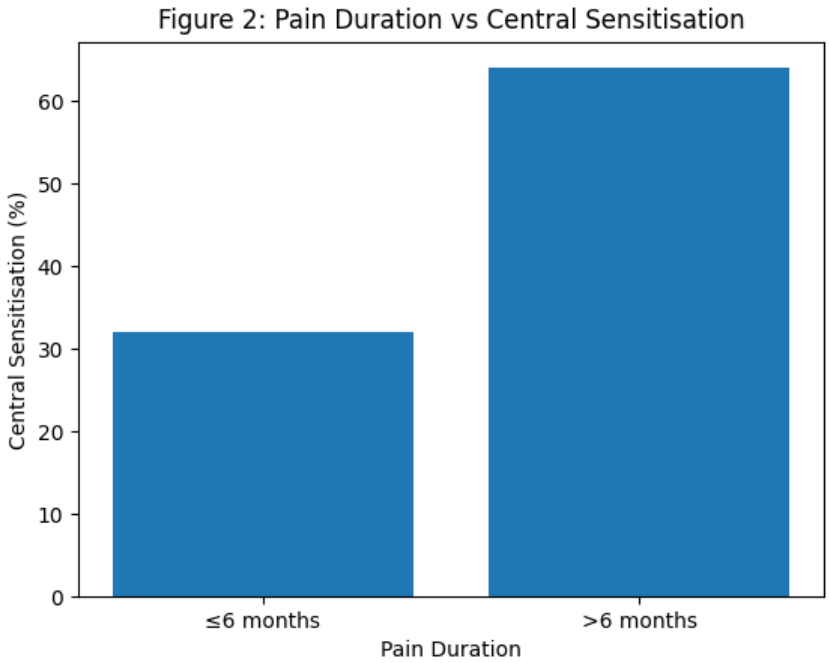

Figure 2: Pain Duration vs Central Sensitisation

Clinical Features: Allodynia was observed in 40 patients (40%), while hyperalgesia was present in 52 patients (52%). Both clinical features were significantly associated with central sensitisation, as demonstrated by Chi-square analysis ($p < 0.01$).

Summary of Statistical Findings

- Central sensitisation associated with higher pain intensity ($p < 0.001$).
- Significant association with longer duration ($p = 0.01$).
- Clinical signs (allodynia, hyperalgesia) significant ($p < 0.01$).

Discussion

This study demonstrates the significant role of both peripheral and central sensitisation in chronic pain disorders. Peripheral sensitisation was more common, reflecting ongoing nociceptive input from tissues [11].

Central sensitisation, however, was strongly associated with increased pain severity and chronicity. These findings align with previous research highlighting the importance of central mechanisms in persistent pain [12].

The association between longer pain duration and central sensitisation suggests a transition from peripheral to central mechanisms over time [13]. This has important therapeutic implications.

Allodynia and hyperalgesia were prominent clinical markers of central sensitisation, consistent with neurophysiological studies [14]. These features indicate altered pain processing at the spinal and cortical levels [15].

Neurochemical changes such as increased glutamate activity and reduced GABA inhibition contribute to sustained central sensitisation [16]. Additionally, descending inhibitory pathways may become dysfunctional [17].

Management of chronic pain should therefore focus not only on peripheral pathology but also on central mechanisms [18]. Drugs such as antidepressants and anticonvulsants have shown effectiveness in targeting central sensitisation [19].

Non-pharmacological approaches, including cognitive behavioral therapy and physiotherapy, also play a crucial role [20].

Conclusion

Peripheral and central sensitisation are key contributors to chronic pain disorders. Central sensitisation is associated with increased severity and longer duration of pain. Early identification and targeted treatment strategies are essential for effective management.

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