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Cortico-Limbic Disconnection Phenotype in Chronic Central Neuropathic Pain: A Case Report

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Background: Chronic neuropathic pain that arises after severe burns often involves central sensitization. While self-report is key for the condition to be diagnosed, tools like quantitative electroencephalograms (qEEGs), event-related potentials (ERPs), and neuropsychological tests may offer objective support.

Case Report: A 59-year-old man developed chronic multifocal pain after severe burns. Subsequently, he experienced tingling, numbness, and burning for 3 years, with mood and cognitive issues. The neurological exam revealed allodynia and hyperalgesia over the burned regions. qEEGs and ERPs revealed dysfunction in the patient's orbitofrontal, anterior cingulate, and insular regions, with high-frequency frontal hyperactivity. Neuropsychological testing confirmed deficits in the man's attention, executive function, and reaction time. Gabapentin and duloxetine gave partial relief to the patient.

Conclusion: This case highlights complex brain changes linked to central neuropathic pain. qEEGs, ERPs, and neuropsychological assessments of the patient revealed a constellation best conceptualized as a cortico-limbic disconnection with frontal hyperarousal syndrome. This construct offers an alternative framework to thalamocortical dysrhythmia. These findings support the use of clinical and medicolegal evaluation to investigate neuropathic pain and emphasize the need for further validation of this emerging phenotype.

Key words: Chronic neuropathic pain, central sensitization, qEEG, event-related potentials (ERPs), neuropsychological testing, medicolegal evaluation

BACKGROUND

Burn injuries can induce chronic neuropathic pain through direct nerve damage and maladaptive central sensitization (1,2). Although patients' clinical histories remain the basis for diagnosing neuropathic pain, objective neurophysiological tests such as quantitative electroencephalograms (qEEGs) and event-related potentials (ERPs) can enhance diagnostic certainty and guide therapy (3,4).

Persistent centralized pain states correlate frequently with cognitive deficits, especially in attention, memory, and executive function, further complicating patient care (5,6). The following case illustrates the use of advanced neurophysiological methods in confirming and guiding therapy for refractory neuropathic pain, while also underlining those methods' emerging implications in medicolegal settings.

CASE PRESENTATION

Clinical History

A right-handed 59-year-old man with no significant medical history sustained extensive chemical burns in 2020 when industrial caustic material splashed onto his

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face, left torso, abdomen, left groin area, and genitalia. The burns were categorized as second- and third-degree, requiring the patient's admission to the intensive care unit. He received immediate decontamination and acute pain management. Although his wounds healed, he developed chronic neuropathic pain in the affected regions within several months. He was referred to the Pain Clinic after failing to achieve adequate relief with over-the-counter analgesics and opioids.

He described burning, tingling, and "buzzing" sensations, with marked allodynia triggered by minimal stimuli. He also reported depression, anxiety, painful erections, and cognitive difficulties (slowed thinking and poor concentration). During a physical exam, allodynia and hyperesthesia were observed over the patient's previously affected regions. No motor deficits were detected. The patient was diagnosed with peripheral neuropathy following burn injuries. He also underwent a comprehensive neurophysiological and psychological assessment to delineate the pathophysiology and assess his cognitive burden. He was started on an adequate pain control regimen for central pain.

Neurophysiological Assessment

Quantitative EEG (qEEG)

To enhance the robustness of our neurophysiological assessment, we used 3 complementary qEEG/stereo-electroencelography (sEEG)-based systems: BrainMaster, NeuroGuide™ (Applied Neuroscience, Inc.), and BrainView. Each system offers overlapping but distinct capabilities. BrainMaster is an established EEG/neurofeedback hardware/software suite (Freedom/Discovery/Atlantis series) that allows for the measurement of amplitude, phase, frequency, coherence, and cross-frequency metrics. The suite supports rapid feedback and high reliability in basic EEG spectral and coherence analysis. NeuroGuide™ delivers surface and LORETA (low-resolution brain electromagnetic tomography) source imaging with dynamic databases, Z-score mapping, and phase/coherence and cross-frequency metrics; the system offers fine localization via standardized weighted LORETA/Z-score tools and batch processing for comparison with normative data. BrainView integrates qEEGs, evoked responses (ERPs), source analysis (3D/LorE/exact LORETA [ELoreta]), and connectivity metrics, with an extensive standardized normative database. Using all 3 methodologies allowed us to triangulate our findings, including surface spectral metrics, source localization, and connectivity, strengthening our confidence in the results (e.g., hypoactivation in the orbitofrontal cortex [OFC]/anterior cingulate cortex [ACC]/insula and network inefficiency, as described below) to a greater extent than relying on a single system would (3).

Evoked Potentials

Evoked potentials were obtained using the BrainView platform, which integrated ERP paradigms into qEEG acquisition. A standard 21-channel EEG was recorded at a sampling rate of 500 Hz with a common reference montage, the band-pass filtered at 0.5–60 Hz, and the notch filtered at 60 Hz. During acquisition, the patient underwent eyes-open and eyes-closed hyperventilation (180 seconds), and photic stimulation (2–30 Hz). ERPs were elicited using visual stimuli (checkerboard reversal paradigm) to extract the N2 component, and auditory stimuli (oddball paradigm) to derive P2, P3, and N4 latencies and amplitudes. Results were automatically compared to age-normative databases. The derived measures included sensory (P2), attentional (P3), and working memory (N4) indices, which were interpreted in this clinical context (5).

Behavioral Performance Testing

Behavioral attention and response inhibition were assessed with a computerized continuous performance paradigm (BrainView). The task measured the patient's reaction time to visual and auditory stimuli, variability of response speed, omission errors (missed responses), and commission errors (false responses). These indices provided objective markers of sustained attention, processing speed, and inhibitory control. Performance was compared automatically to age-referenced normative data provided by the testing platform.

Neuropsychological Testing (CNS Vital Signs)

Neuropsychological evaluation was carried out with the CNS Vital Signs system (CNSVS), a computerized, validated cognitive battery. CNSVS provides domain scores normed to age- and education-matched populations. Composite scores generated included indices for executive function, memory, processing speed, reaction time, and attention. The patient's mood was assessed with the Depression, Anxiety, and Stress Scale (DASS-42) and Patient Health Questionnaire (PHQ-9). His global cognition was additionally assessed using common screening measures; i.e., the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA) (7,8).

The CNSVS includes embedded validity indicators that automatically flag inconsistent performance patterns, excessive errors, or improbable response styles. These internal checks ensure that the results obtained are reliable and reflect genuine cognitive performance rather than poor effort or malingering. In this case, all validity indicators were passed, supporting the accuracy of the neuropsychological and behavioral findings.

RESULTS

Quantitative EEG (qEEG)

Quantitative EEG recordings were consistent across the 3 analytic platforms, though each highlighted different aspects of the patient's cortical activity. At the surface level, the BrainView and BrainMaster platforms demonstrated that the posterior dominant rhythm was preserved within the normal alpha range, approximately 11–12 Hz (12.1 Hz in one session). This finding argues against the possibility of a global downward shift of the dominant rhythm characteristic of canonical thalamocortical dysrhythmia. The normal expected alpha peak in the posterior region with eyes closed was absent (Fig. 1), and alpha reactivity was markedly blunted: the eyes-closed/eyes-open alpha power ratio measured only ~1.01 (normative >1.2), reflecting impaired vigilance and arousal modulation.

In addition, there were focal increases in delta power, which appeared most prominently in circumscribed occipital clusters. These were localized (focal) phenomena rather than instances of global slowing, and they coexisted with other abnormalities observed on source analyses.

The sLORETA maps generated through the Applied Neuroscience platform showed a complementary pattern (Fig. 2). Here, theta and alpha power were reduced in frontal, temporal limbic regions. Specifically, the ACC (Brodmann area 25) showed a theta Z-score of –2.2 (7 Hz) and an alpha Z-score of –3.0 (11 Hz), indicating significant hypoactivity compared to normative databases figures. These reductions were evident as blue shading on the voxel maps and consistently highlighted diminished current source density within the ACC, OFC (BA 11), and anterior insula (BA 13). Thus, rather than excess slow activity, the source analyses indicated underactivation of major limbic regulators (Fig. 2).

Connectivity and phase coherence analyses further highlighted network inefficiency (Fig. 3). Maps from Applied Neuroscience and BrainView showed predominantly reduced coherence (blue lines) linking the limbic and cortical regions, particularly connections involving the ACC, OFC, and insula. These were interspersed with small islands of increased coherence in frontal networks, yielding a heterogeneous pattern that reflected reorganization and a reduction in global efficiency rather than a uniform rhythmopathy.

By contrast, higher-frequency bands revealed the opposite pattern. Both the BrainView/BrainMaster outputs and the Applied Neuroscience sLORETA figures demonstrated focal increases in high-beta and gamma power within the medial and superior frontal cortex (Brodmann area 6) (Fig. 4). Z-scores reached +3.4 for hi-beta at 32 Hz and +3.0 for gamma at 35 Hz, results that were consistent with hyperactivity of the frontal motor–attention hub. This frontal hyperarousal occurred alongside the limbic hypoactivity described above.

These findings showed a consistent cross-platform profile: the patient exhibited an absent posterior alpha rhythm, localized delta foci, hypoactivity of limbic regulatory hubs (ACC, OFC, anterior insula), hyperactivity of frontal high-beta/gamma generators in BA6, and inef-

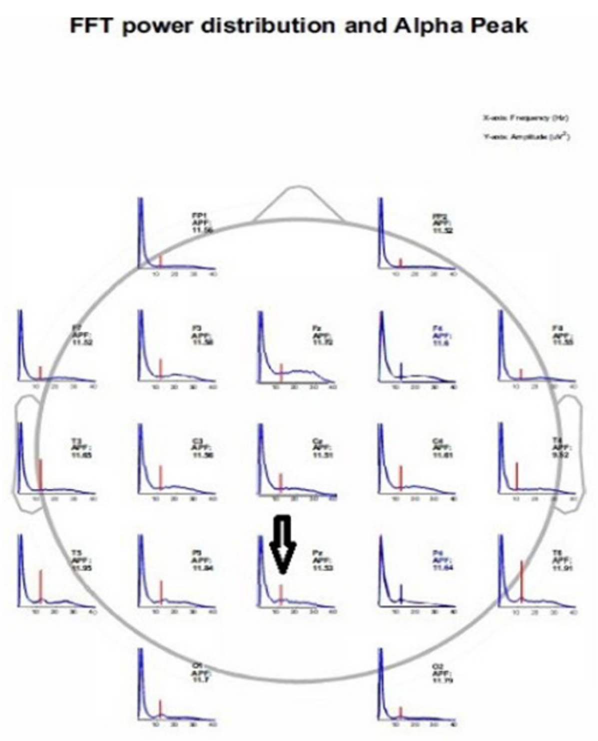

Fig. 1. Quantitative EEG surface power distribution. The arrow demonstrates an absent/blunt alpha peak in the posterior region.

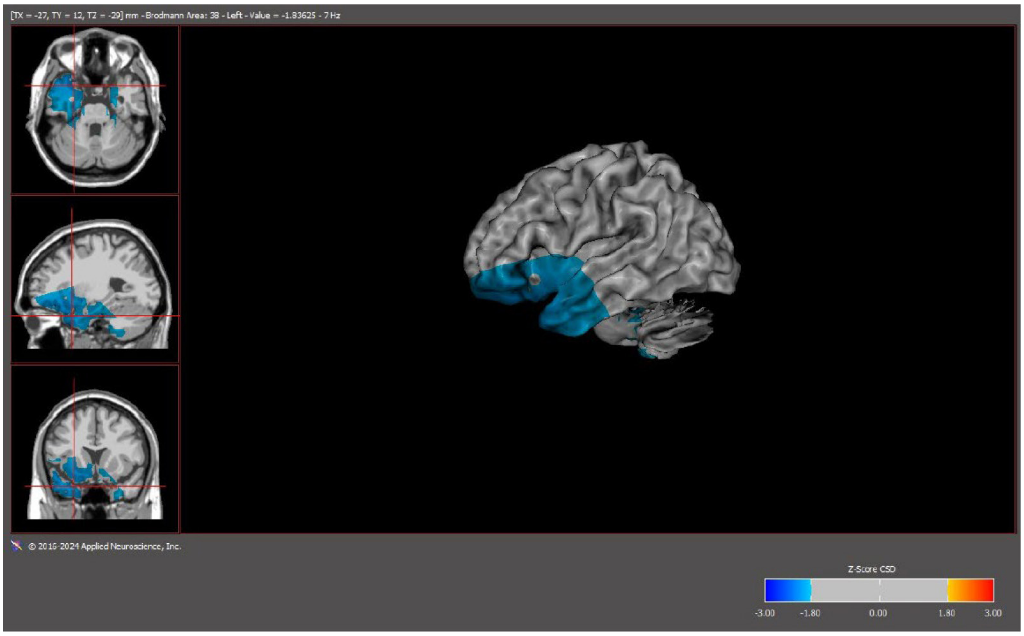

Fig. 2. sLORETA source localization demonstrating hypoactivation in the left frontal and temporal regions. Three-dimensional reconstruction of the brain shows areas of reduced current source density (blue shading) involving the orbitofrontal cortex and anterior temporal lobe (Brodmann area 38). These regions are implicated in emotional regulation, pain valuation, and sensory integration. Reduced activation here is consistent with impaired top-down modulation in chronic neuropathic pain.

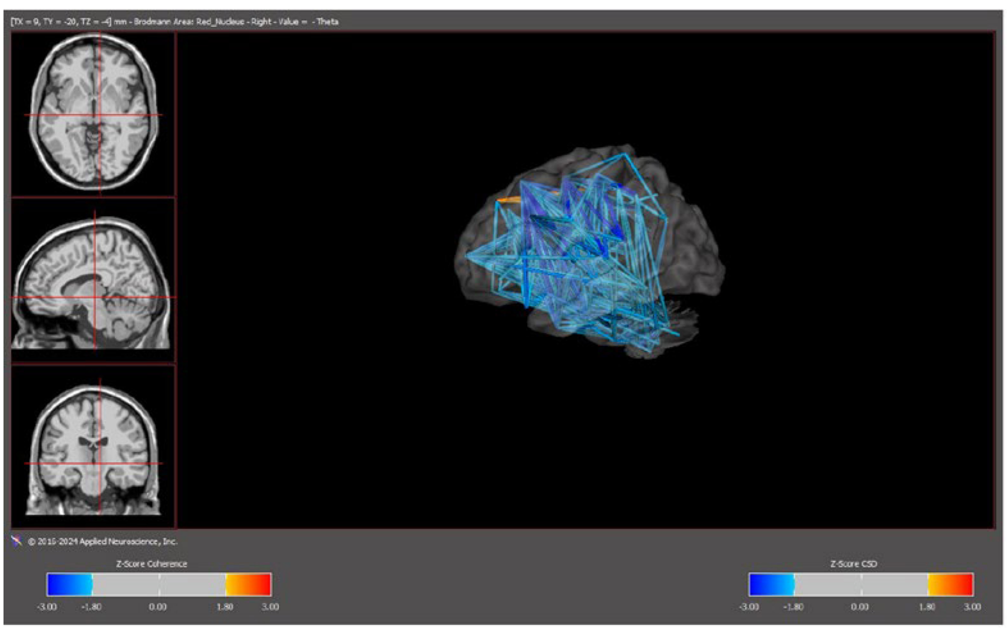

Fig. 3. Connectivity analysis reveals inefficient cortical communication in chronic pain. Functional connectivity map derived from EEG coherence analysis. Blue lines indicate reduced interregional connectivity, while orange lines (minimal here) indicate hyper-coupling. The predominance of hypoconnectivity demonstrates impaired integration across cortical networks, a feature consistent with network inefficiency observed in chronic centralized pain states.

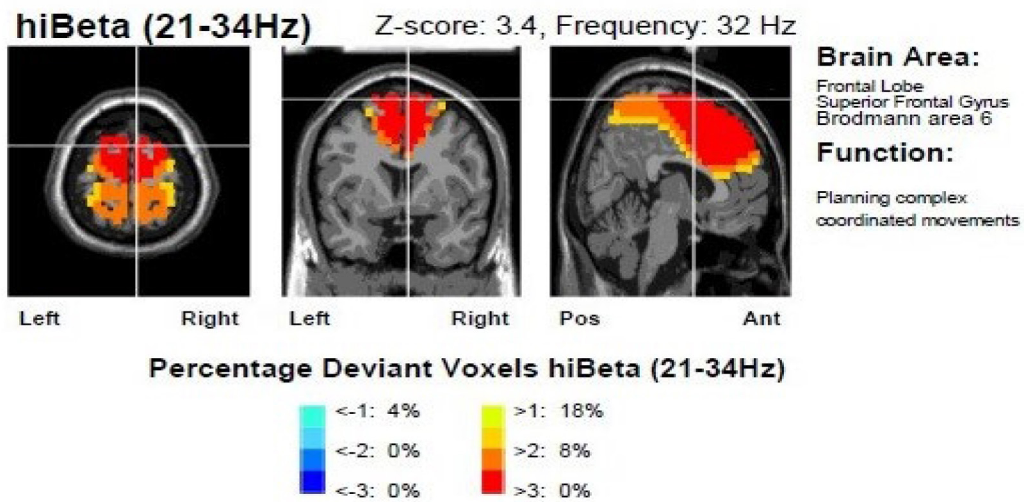

Fig. 4. BrainView/BrainMaster outputs and Applied Neuroscience sLORETA maps reveal focal increases in high-beta (32 Hz, Z = +3.4) and gamma (35 Hz, Z = +3.0) power within the medial and superior frontal cortex (Brodmann area 6). This activity pattern is consistent with hyperactivation of the frontal motor–attention hub.

ficient connectivity characterized by mixed hypo- and hyper-coherence. This pattern is best understood as cortico-limbic disconnection with frontal hyperarousal and inefficient network organization rather than a classic thalamocortical dysrhythmia.

Event-Related Potentials (ERPs)

ERP analysis evaluated sensory and cognitive processing during visual and auditory paradigms. Visual ERP (N2) showed normal latency (~204 ms) with borderline low normal amplitude, and auditory ERP (P2) showed prolonged latency (~164 ms; normal < 120 ms) and low amplitude, consistent with impaired visual and auditory sensory gating (5). The P3 potential showed prolonged latency in auditory attention (P3 ~356 ms), indicating slowed cognitive processing and reduced efficiency in attentional control. Importantly, these results were in agreement with the qEEG maps.

Behavioral Performance Testing

On behavioral testing, the patient demonstrated a mean reaction time of 523 ms (slower than the normal reference range of 350-500 ms) with increased reaction time variability (14.8 ms; normal < 10 ms). Missed responses were low (1.7%; reference ≤ 6%), indicating preserved vigilance, but commission errors were elevated (7.5%; abnormal > 4%), consistent with poor inhibitory control. Taken together, these findings reflected slowed cognitive processing, increased variability of attentional performance, and deficient response inhibition.

Table 1.Cognitive profile highlights.

Domain	Percentile Score	Interpretation
Visual Memory	10th	Low Average
Reaction Time	1st	Very Low
Executive Function	7th	Low
Reasoning	2nd	Very Low
Attention/Processing	Below average	Slowed performance
Mood (PHQ-9)	18	Moderate Depression
Stress (DASS subscale)	26	Elevated Stress

Abbreviations: BVMT-R = Brief Visuospatial Memory Test–Revised; CPT-3 = Conners Continuous Performance Test, Third Edition; TMT-B = Trail Making Test, Part B; WAIS-IV = Wechsler Adult Intelligence Scale, Fourth Edition; PHQ-9 = Patient Health Questionnaire–9; DASS-21 = Depression, Anxiety, and Stress Scale–21.

Neuropsychological Testing

A standardized neuropsychological battery assessed the patient's memory, attention, executive function, and mood (Table 1). Testing revealed deficits in attention, executive function, and reaction time, consistent with impaired higher-order cognitive control in chronic pain states. The patient's reasoning and visual memory were also below normative expectations. Mood measures indicated moderate depressive symptoms and high stress burden. These findings are consistent with the literature on the cognitive and emotional impact of centralized neuropathic pain (6).

Therapeutic Interventions and Current Status

The patient was followed up at a university hospital's burn center, where he received standard wound management. His neuropathic pain responded partially to gabapentin (up to 800 mg TID) and duloxetine, consistent with first-line management strategies, providing ~50% initial relief that later plateaued (5,6). The residual pain remained refractory to opioids and nonsteroidal anti-inflammatory drugs (NSAIDs). For pain and mood symptoms, he was prescribed duloxetine. Despite those measures, he continued to report fragmented sleep, averaging 5-6 hours of restless slumber per night. Three years after the injury, his pain remained marked by flares lasting one-3 hours at a time, often triggered by physical activity. Neurological examination revealed patchy sensory deficits with preserved motor strength and reflexes.

As a future consideration for ongoing refractory symptoms, adjunctive approaches may be considered, including neurofeedback to modulate abnormal EEG patterns and cognitive behavioral therapy to address mood disorders (3). A framework integrating pharmacological, neurophysiological, and psychological strategies remains essential for optimizing the outcomes of treatments for complex chronic pain following burn injury.

DISCUSSION

Chronic neuropathic pain that arises after burn injury is a complex, often refractory condition with manifestations that extend beyond the sensory domain to impair cognition, mood, and daily functioning. This case illustrates how advanced neurophysiological testing, including qEEGs, event-related potentials, and neuropsychological assessments, can provide objective evidence of cortical dysregulation, supporting both clinical interpretation and medicolegal evaluation.

Our neurophysiological results revealed a consistent pattern of impaired top-down regulation. At the cortical surface level, the posterior alpha peak was absent (~11-12 Hz), and alpha reactivity was blunted (eyes-closed/eyes-open ratio ≈ 1.01), indicating impaired vigilance regulation without a thalamocortical dysrhythmia–type shift. Source analyses demonstrated hypoactivation in the OFC (BA11), subgenual/ACC (BA25), and anterior insula (BA13), regions involved in descending pain control, salience appraisal, and interoception (9). In contrast, high-frequency power (hi-beta/gamma) was increased in the medial frontal cortex (BA6), a signature consistent with hyperarousal. Connectivity analyses revealed a heterogeneous pattern of hyper- and hypocoherence, suggesting an inefficient network organization. These findings support a cortico-limbic disconnection phenotype: underactive inhibitory hubs coupled with overactive arousal networks(3).

The anterior insula (BA13) is widely recognized as a core interoceptive hub responsible for integrating internal bodily signals and contributing to subjective awareness of physiological state, while the orbitofrontal cortex (BA11) supports valuation, emotional appraisal, and expectation of relief or harm. Dysfunction in these regions has been linked to altered pain perception and impaired salience attribution in neuroimaging studies that use tasks of interoceptive awareness and pain modulation (10-12).

ERP results converged on this interpretation. The auditory P2 latency was prolonged (~164 ms) and the auditory P3 delayed (~356 ms) with reduced amplitude, while the visual N2 (~204 ms) had borderline low amplitude. These abnormalities indicated slowed sensory-cognitive processing and reduced attentional resource allocation. Neuropsychological testing provided convergent evidence: reaction time was at the first percentile, executive function at the seventh percentile, reasoning at the second percentile, and visual memory at the tenth percentile. Mood scales confirmed moderate depression and elevated stress. The convergence across qEEGs, ERPs, and neuropsychology strengthens the conclusion that the central nervous system dysfunction is real, systemic, and mechanistically linked to impaired top-down control.

A further point of convergence comes from the behavioral performance results. The combination of slowed reaction time, increased variability, and elevated commission errors provides task-based evidence of impaired inhibitory control and attentional inefficiency (6). These abnormalities map closely onto the neurophysiological profile: delayed and attenuated auditory P3 responses, hypoactivation of OFC and ACC, and mixed hypo-/hypercoherence on connectivity analysis (13). In other words, both electrophysiology and behavioral testing independently highlight a failure of top-down regulation, a finding consistent with a compromised descending pain modulatory system (DPMS) and the patient's clinical picture of chronic centralized pain.

Pain remains a fundamentally subjective experience, but this case illustrates how neurophysiological and cognitive assessments can provide objective support in the evaluation of complex chronic pain. Converging data from multiple modalities can guide treatment planning, support clinical impressions, and, in some cases, contribute to forensic evaluations.

Given the subjective nature of pain, the integration of objective physiological evidence that pertains to it, particularly functional neuroimaging, has become increasingly influential in medicolegal settings. As discussed by Reardon, functional MRI offers potential as a tool to visualize pain-related brain activity, while Salmanowitz further explores the applicability of functional MRI in validating pain claims in civil litigation (14,15). Similarly, the CNSVS incorporates embedded validity indicators, which detect inconsistent or improbable response patterns. In the present case, all validity checks were passed, providing strong evidence that the deficits observed were genuine and not attributable to suboptimal effort or malingering. This result reinforces the reliability of the findings and strengthens their probative value in both clinical and medicolegal contexts.

Importantly, courts have begun to consider such neurophysiological findings as admissible evidence in litigation. In Martinez v. North Shore (2023), abnormal qEEG findings were accepted as supporting evidence of chronic pain (16). While the legal acceptance of such data remains relatively novel and subject to scrutiny, these developments illustrate the evolving convergence of neuroscience and jurisprudence.

While qEEGs, ERPs, and neuropsychological assessments offer valuable insights into assessing pain and related cognitive impairment, the use of these tools' interpretations in legal settings remains controversial. Neurophysiological markers such as altered alpha reactivity, ERP delays, or abnormal coherence patterns lack specificity, as they may also appear in conditions such as mood disorders, sleep disturbances, or traumatic brain injury. Despite these limitations, these markers remain valuable adjunctive tools in the appropriate clinical context. Much like other laboratory findings, they cannot be used in isolation to prove or disprove pain but must be interpreted within a biopsychosocial framework.

Therefore, while these tools provide critical supporting information, they should remain complementary in assessing pain during medicolegal evaluations until stronger diagnostic validity is established. Multimodal approaches, integrating clinical history, neurophysiology, neuropsychology, and imaging, provide the most reliable basis for understanding and validating persistent pain states.

Access to advanced neurophysiological assessments such as qEEGs, event-related potentials, and computerized neuropsychological testing varies across clinical settings and may be limited by availability. In many institutions, these tools are concentrated in specialized centers, which can create logistical barriers to implementation. Future work should focus on standardizing acquisition protocols and evaluating cost-effectiveness to facilitate broader clinical adoption.

CONCLUSION

This case provides a rare and integrative demonstration of neurophysiological and cognitive alterations in a patient with chronic neuropathic pain secondary to chemical burns. In contrast to the canonical thalamocortical dysrhythmia pattern, this patient's profile corresponds more closely to what has been increasingly described across chronic pain cohorts: frontal high-frequency (beta/gamma) activity linked to ongoing pain perception (17), aberrant cortico-limbic function within the ACC, OFC, and insular regions responsible for salience appraisal, interoception, and valuation (10,13,18), and network disruption characterized by mixed hyper- and hypocoherence (9). This constellation is consistent with a compromised descending pain modulatory system (DPMS), which has been highlighted repeatedly in both human and preclinical models of chronic pain. The findings of this case highlight the value of combining clinical observation with objective data to understand and validate complex pain syndromes. We propose that this emerging profile may be conceptualized as a cortico-limbic disconnection with frontal hyperarousal syndrome, a distinct electrophysiological pattern in chronic pain that offers an alternative framework to thalamocortical dysrhythmia and warrants further validation as a clinical construct.

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