Via Dolorosa: impact of nociplastic pain on hospitalisation outcomes. A retrospective cohort study

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Abstract

Objective

Nociplastic pain, characterised by altered central pain processing, is associated with increased healthcare utilisation. This study investigated the relationship between nociplastic pain, based on clinical diagnoses of fibromyalgia, chronic pain, or myofascial pain, and hospital outcomes in patients.

Methods

A retrospective cohort study was conducted using hospital records of hospitalised patients. Patients aged ≥18 years with a diagnosis of fibromyalgia, chronic pain, or myofascial pain were classified as the research group, while a comparison group of hospitalised patients without these diagnoses was selected. Outcomes included length of stay (LOS) for the index hospitalisation, recurrent hospitalisations within 6 months, Opioid use disorder (OUD), and 5-year mortality. Statistical analyses included t-tests, Wilcoxon tests, linear and Poisson regressions, adjusting for age and sex.

Results

Of 18,393 patients, 3,326 (18.1%) were in the research group. The research group was older (mean age 62.35 vs. 50.57 years, p<0.001) and had a higher proportion of females (72.7% vs. 56.6%). Adjusted analyses showed longer LOS in the research group (mean 6.89 vs. 5.59 days, p=0.002), higher recurrent hospitalisations (p<0.001), and increased OUD (87.5% of cases in the research group, p<0.001). Surprisingly, 5-year mortality risk was lower in the research group (HR 0.523, p<0.001).

Conclusion

Nociplastic pain diagnoses are associated with prolonged hospitalisations, increased readmissions, and OUD, but lower long-term mortality. These findings highlight the need for targeted pain management strategies.

Key words

nociplastic pain, hospitalisation length, readmissions, opioids, retrospective cohort study

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Introduction

Nociplastic pain is defined by the International Association for the Study of Pain (IASP) as pain that arises from altered nociception, despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain (1, 2). This mechanistic descriptor distinguishes it from both nociceptive pain, which results from tissue damage or inflammation and neuropathic pain, stemming from nervous system lesions (3). Nociplastic pain often manifests in conditions such as fibromyalgia, chronic fatigue syndrome (CFS), irritable bowel syndrome (IBS), myofascial pain syndrome, interstitial cystitis and chronic headache syndromes, where it can occur in isolation or alongside other pain types (4, 5). Central sensitisation is considered to play a key role in the pathophysiology of nociplastic pain, involving amplified neural signalling, lowered pain thresholds, and symptoms such as widespread pain, fatigue, sleep disturbances, and cognitive impairments (6).

The prevalence of nociplastic pain varies across populations and conditions. In the general population, estimates for related syndromes such as fibromyalgia range from 2–6%, with higher rates (10-48%) among individuals with rheumatic diseases (7). Overall, nociplastic features may affect up to 5–15% of individuals, with a higher incidence among females (8). This pain type contributes significantly to global disability, imposing a substantial healthcare and economic burden, due to its chronic nature and what appears as resistance to conventional treatments (9, 10).

Patients with nociplastic pain, particularly those with fibromyalgia, exhibit heightened healthcare utilisation, including more outpatient visits, hospitalisations, and emergency department encounters compared to controls (11, 12). For example, fibromyalgia is linked to longer hospital stays post-surgery, such as total hip arthroplasty, due to increased complications, opioid requirements, and resource use (13, 14). These patterns may arise from unmanaged symptoms, comorbidities, and disparities in access to non-pharmacologic therapies, leading to elevated costs and strained healthcare systems (15).

Opioid prescribing for chronic non-cancer pain has escalated, despite evidence of limited long-term efficacy and significant risks, including dependence, tolerance, overdose, and opioid-induced hyperalgesia (16, 17). In nociplastic pain, where central sensitization predominates, opioids may exacerbate pain sensitivity and contribute to a cycle of escalation (18). Recent studies suggest a bidirectional relationship between chronic pain and OUD, potentially mediated by shared neurobiological mechanisms (19).

Mortality outcomes in chronic pain populations are inconsistent. Some meta-analyses indicate a 20–30% increased risk of all-cause mortality, particularly from cardiovascular, respiratory, and accidental causes, while others find no overall elevation after adjusting for confounders (20, 21). In fibromyalgia specifically, evidence leans toward no increased all-cause mortality, despite higher morbidity (22, 23). Interestingly enough, in a later study with a larger cohort, mortality rates were larger (24). Despite these insights, retrospective studies examining nociplastic pain based on clinical diagnoses in hospital settings are limited. The current study evaluates the impact of nociplastic pain on hospital LOS (primary), recurrent hospitalisations, OUD, and mortality, aiming to inform targeted interventions.

Methods

Study design and setting

This was a retrospective cohort study using anonymised data from medical records of hospitalised patients. The study was approved by the hospital's Institutional Review Board (IRB), with a waiver of informed consent due to the retrospective nature and use of de-identified data. Data were collected over a 2-year period following IRB approval.

Participants

Inclusion criteria. Patients ≥18 years with a clinical diagnosis of fibromyalgia, chronic pain, or myofascial pain

syndrome, and at least one hospitalisation at or after the diagnosis.

Exclusion criteria. Age <18 years, history of myeloma at any stage, inflammatory bowel diseases, or malignancy (other non-melanoma skin cancer).

Patients with active malignancy, multiple myeloma, or inflammatory bowel disease were excluded to reduce confounding from conditions frequently associated with severe nociceptive or mixed pain mechanisms, high opioid exposure, increased hospitalisation frequency, and elevated baseline mortality risk, which could obscure evaluation of nociplastic pain-related outcomes.

Patients meeting inclusion criteria formed the research group (n=3,326). A control group (n=15,067) was selected from the same wards, consisting of patients without these pain diagnoses but otherwise similar in admission characteristics.

Because nociplastic pain cannot be retrospectively identified using mechanistic biomarkers or IASP grading criteria, we used clinical diagnoses commonly associated with nociplastic mechanisms (fibromyalgia, chronic pain syndrome, and myofascial pain syndrome) as a pragmatic clinical proxy. This approach reflects real-world diagnostic practice but does not fully capture the biological construct of nociplastic pain and carries a risk of misclassification. Therefore, findings should be interpreted as reflecting outcomes associated with these clinical diagnoses rather than mechanistically confirmed nociplastic pain.

Data collection

Data were extracted from electronic medical records and included:

- Baseline: Demographics (age, sex), primary diagnosis, comorbidities.
- Index hospitalisation: LOS.
- Follow-up: Recurrent hospitalisations within 6 months, opioid use disorder (OUD was defined as a new diagnosis documented by the treating physician during hospitalisation or follow-up, based on clinical assessment and medical record coding), 5-year mortality via registry linkage.

Data were anonymised, stored securely on hospital computers with password protection, and handled only by authorised researchers.

Statistical analysis

Data normality was assessed; non-normal distributions led to use of parametric (t-tests) and non-parametric (Wilcoxon rank-sum) tests. Linear regression was adjusted for age and sex for LOS. Poisson regression was utilised for recurrent hospitalisations and Cox proportional hazards for mortality. Chi-square was utilised for opioid addiction. Analyses were performed using SPSS version 31.0.0.0(117), with $p<0.05$ considered significant. Sample size provided >80% power for detecting medium effect sizes.

Table I. Baseline demographic and clinical characteristics of hospitalised patients with nociplastic pain-related diagnoses (research group) and controls.

Variable	Research group (n, %)	Control group (n, %)	Total (n, %)
Sex: Male	909 (27.3%)	6539 (43.4%)	7448 (40.5%)
Sex: Female	2417 (72.7%)	8528 (56.6%)	10945 (59.5%)
Diabetes	773 (21.2%)	1910 (12.1%)	2683 (13.8%)
Hypertension	1548 (42.5%)	3850 (24.5%)	5398 (27.9%)
Hyperlipidaemia	1332 (36.6%)	2638 (16.8%)	3970 (20.5%)
Tobacco use	118 (3.2%)	180 (1.1%)	298 (1.5%)

Values are presented as n (%). Continuous variables are presented as mean ± standard deviation.

Table II. Length of stay (LOS) during index hospitalisation in patients with nociplastic pain-related diagnoses *versus* controls.

	n	Mean LOS	Std. deviation
Control	15,052	5.59	10.62
Research	3,311	6.89	13.97

LOS values are presented as mean ± standard deviation (days). Group comparisons were evaluated using both parametric and non-parametric tests due to non-normal distribution of LOS.

Results

A total of 18,393 patients were included: 3,326 (18.1%) in the research group and 15,067 (81.9%) in the control group.

Demographics

The research group had a higher proportion of females (72.7% *vs*. 56.6%) and was older (mean age 62.35±17.29 years *vs*. 50.57±21.13 years, $p<0.001$). All subsequent analyses were adjusted for age and sex.

Primary outcome: length of stay (LOS)

Mean LOS was longer in the research group (6.89±13.97 days) *vs*. control (5.59±10.62 days). Due to non-normality, both t-test and Wilcoxon tests were performed.

T-test: t = -6.000, $p<0.001$ (equal variances); t =-5.047, $p<0.001$ (unequal).

Wilcoxon: Z = -2.030, $p=0.042$.

Adjusted linear regression confirmed significance ($p=0.002$ for group effect).

Secondary outcomes

Recurrent hospitalisations (within 6 months). Significantly higher rates of recurrent admission were observed in the research group compared with the control group. Wilcoxon: Z = -20.507, $p<0.001$.

In adjusted Poisson regression, the group coefficient was –0.424 (control *vs*. nociplastic pain group; $p<0.001$). This corresponds to an incidence rate ratio (IRR) of 0.654, indicating that the control group had 65% of the recurrent hospitalisation risk observed in the nociplastic pain group, equivalent to a 1.53-fold higher risk in the research group (Exp(B)=0.654 for control).

Opioid use disorder

OUD diagnosis rates (added by the attending physician during hospitalisation) was retrieved. OUD was significantly more prevalent in the research group (28/32 cases, 87.5%, Chi-square: 104.278, $p<0.001$)

Table III. Multivariable Poisson regression model for recurrent hospitalisations within 6 months.

	B	Std. error	Wald Chi-Square	*p*	Exp(B)
Intercept	-1.344	0.054	613.174	<0.001	0.261
Group=0 (Control)	-0.424	0.030	194.535	<0.001	0.654
Male=0	-0.102	0.027	14.452	<0.001	0.903
Age	0.011	0.001	277.946	<0.001	1.011

Model adjusted for age, sex, and study group.

Table IV. Prevalence of opioid use disorder (OUD) in nociplastic pain group *versus* controls.

OUD	Control (count/expected)	Research (count/expected)	Total
No (0)	15,063 / 15,040.8	3,298 / 3,320.2	18,361
Yes (1)	4 / 26.2	28 / 5.8	32
Total	15,067	3,326	18,393

Mortality (5-year)

Survival analysis demonstrated a statistically significant difference between groups (Kaplan-Meier log-rank χ^2 = 9.294, p=0.002), with the nociplastic pain group showing higher 5-year survival compared to controls.

In the multivariable Cox proportional hazards model adjusting for age and sex, nociplastic pain diagnosis remained independently associated with lower mortality risk (HR=0.523, 95% CI 0.467–0.586, p<0.001). Older age and male sex were both associated with higher mortality risk.

After adjustment for age and sex, nociplastic pain diagnoses were associated with a lower observed risk of death within 5 years. However, given the observational design and potential residual confounding, this finding should be interpreted cautiously.

Discussion

The longer length of stay and higher readmission rates observed in patients with nociplastic pain-related diagnoses are consistent with prior literature on chronic pain and fibromyalgia populations (11-13). However, due to the retrospective design of the study, the underlying mechanisms cannot be determined. Potential explanations may include differences in healthcare utilisation patterns, system-level care processes, differences in discharge planning, or increased clinical complexity. These interpretations should be considered hypotheses rather than demonstrated causal mechanisms.

More importantly, the study design cannot establish whether prolonged stays reflect genuine medical complexity, inadequate pain management protocols, or healthcare provider bias toward patients with subjective pain complaints. Alternative explanations include iatrogenic complications from polypharmacy, delayed discharge due to social factors, or unconscious provider bias against patients with chronic pain diagnoses. The lack of severity-adjusted comparisons limits our ability to distinguish between these possibilities.

Furthermore, the healthcare system's response to chronic pain patients may create self-perpetuating cycles. Emergency departments and internal medicine services often lack specialised pain management resources, potentially leading to suboptimal acute interventions that necessitate return visits. This represents a systems-level failure rather than inherent patient factors.

An additional systems-level contributor may be the institutional reliance on "quality measures" that emphasise patient-reported outcomes such as pain visual analogue scale (VAS) scores at discharge. In some settings, clinicians are implicitly encouraged to achieve low VAS values before discharging patients, a practice that may inadvertently promote short-term pharmacologic solutions, including the re-prescription of opioids over more sustainable multimodal pain management strategies. These dynamic risks reinforcing dependence patterns and distorting the true goal of care from functional recovery toward numeric satisfaction metrics.

Multidisciplinary approaches, integrating physical therapy, psychology and pharmacology, have demonstrated efficacy in reducing utilisation and improving outcomes in chronic pain (25, 26).

The higher OUD rate in the research group aligns with known risks, where chronic pain patients face elevated OUD incidence due to misconceptions and overprescribing (16). Central sensitisation may underlie this link, perpetuating a cycle of pain and dependence (18, 19). Guidelines advocate limiting opioids, favouring multimodal therapies, yet barriers persist (27). Additionally, surveillance bias should be considered, as increased healthcare contact and opioid exposure in chronic pain populations may increase the likelihood of OUD recognition and documentation.

The lower observed 5-year mortality in the research group represents a paradoxical finding. Importantly, persistence of this association after adjustment for age and sex alone does not imply a true survival benefit. Given the higher comorbidity burden and the absence of adjustment for overall comorbidity burden or disease severity, this association is likely non-causal and may reflect residual confounding, differences in healthcare exposure, or surveillance effects.

Literature addressing mortality in chronic pain populations remains heterogeneous. While some studies demonstrate increased mortality risk, particularly related to cardiovascular and external causes, others, especially in fibromyalgia cohorts, show no increase in all-cause mortality (20-23).

Due to the retrospective observational design, causal relationships between nociplastic pain-related diagnoses and hospitalisation outcomes cannot be established. While the associations observed in this large real-world cohort are clinically relevant, confirmation in prospective and mechanistically characterised cohorts is required, particularly

regarding long-term mortality patterns. Recent contemporary reviews of fibromyalgia and nociplastic pain populations further emphasise the complex and multifactorial nature of chronic pain conditions, including the contribution of comorbidity burden and systemic health factors to long-term clinical outcomes. These data support cautious interpretation of mortality associations in these populations and reinforce the need to interpret survival differences as healthcare utilisation-associated observations rather than direct biological effects of nociplastic mechanisms (31, 32).

Limitations include potential coding bias in diagnoses, reliance on administrative data, and lack of detailed comorbidity adjustments. Although analyses were adjusted for age and sex, adjustment for overall comorbidity burden or disease severity indices was not performed. Therefore, residual confounding remains likely, and results should be interpreted accordingly. The large sample strengthens generalisability but may introduce heterogeneity. Future research should incorporate mechanistic biomarkers, interventional trials, and longer-term economic analyses.

Conclusion

Nociplastic pain diagnoses are associated with greater hospital utilisation and higher opioid-related risks, highlighting the substantial healthcare burden within this population. Although our data suggest no increase in long-term mortality, this finding should be interpreted cautiously given the study's observational design and potential confounding. Strengthening clinician education on nociplastic pain, improving access to multidisciplinary pain management services, and reorienting care goals from short-term pain reduction toward functional recovery may help reduce unnecessary pharmacologic escalation and improve overall patient outcomes.

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