EDITOR'S CHOICE

A Nationwide Propensity Score-Matched Analysis Identifying Preinjury Predictors of Complex Regional Pain Syndrome Following Distal Radius Fracture

Nirav K. Mungalpara, MD,* William K. Reiser, BS,† Logan Van Poucke, BS,* Gautam Malhotra, MD,* Alfonso Mejia, MD,* Mark H. Gonzalez, MD, PhD†

Purpose Complex regional pain syndrome (CRPS) is an uncommon but debilitating complication following distal radius fractures (DRFs). This study aimed to evaluate a broad set of preexisting comorbidities, including substance-use disorders, psychiatric conditions, compressive neuropathies, upper-limb traumatic nerve injuries, and cervical radiculopathy and determine their associations with CRPS development after DRF.

Methods A retrospective analysis of the PearlDiver database (2010−2022) was performed. Patients with and without CRPS following DRF were matched 1:1 using propensity scores based on age, sex, fracture type, and DRF management (Current Procedural Terminology 25605−25609). Multivariable logistic regression evaluated the association between CRPS and 12 preexisting comorbidities. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) and *P* values were reported.

Results Among 2,685,041 DRF patients, 3,796 developed CRPS (incidence 0.18%). In the matched cohort (n = 26,186), several strong associations emerged. Neuropathic conditions demonstrated the highest risk: upper-limb nerve injury (OR 3.20, 95% CI 2.42−4.30), radial nerve lesions (OR 2.43, 95% CI 1.72−3.50), cubital tunnel syndrome (OR 2.27, 95% CI 2.01−2.57), carpal tunnel syndrome (OR 2.01, 95% CI 1.88−2.14), and cervical radiculopathy (OR 1.80, 95% CI 1.68−1.94). Psychiatric conditions showed modest associations: anxiety disorders (OR 1.25, 95% CI 1.18−1.32), depression (OR 1.07, 95% CI 1.01−1.13), and fibromyalgia (OR 1.58, 95% CI 1.44−1.74). Substance-use factors demonstrated mixed effects: opioid use increased risk (OR 1.39, 95% CI 1.27−1.52), alcohol use decreased risk (OR 0.86, 95% CI 0.79−0.94), and tobacco/cannabis were not significant.

Conclusions Preexisting neuropathic disorders, particularly traumatic nerve injuries, compressive neuropathies, and cervical radiculopathy, are the strongest predictors of CRPS after DRF. Psychiatric conditions confer a smaller but consistent risk, while substance-related factors vary. Identifying preexisting neural vulnerability may improve risk stratification after DRF. *(J Hand Surg Am. 2026;51(7):763−769. Copyright © 2026 by the American Society for Surgery of the Hand. All rights are reserved, including those for text and data mining, AI training, and similar technologies.)*

Type of study/level of evidence Prognosis II.

*From the *Department of Orthopaedics, University of Illinois at Chicago College of Medicine, Chicago, IL; and the †Department of Orthopaedics, University of Illinois at Chicago College of Medicine, Rockford, IL.*

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Corresponding author: Nirav K Mungalpara, MD, Department of Orthopaedics, University of Illinois at Chicago College of Medicine, 835 South Wolcott Avenue, Office No E270, Chicago, IL 60612; e-mail: Nirav@uic.com or Nirav.orthodoc@gmail.com.

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COMPLEX REGIONAL PAIN syndrome (CRPS) is a rare, yet debilitating disorder, causing unrelenting, continuous, and disproportionate pain after the inciting event.[1–3] CRPS is categorized into two types: CRPS type 1, which is caused by antecedent trauma or fracture, and CRPS type 2, which involves a specific nerve injury. The condition is marked by severe chronic pain, pathological changes in bones, joints, and skin, excessive sweating, and hypersensitivity.[4] The pain often persists long after the initial injury has healed and can spread beyond the original site of injury. The exact mechanisms of CRPS leading to a cycle of pain and dysfunction remain poorly understood.[5]

CRPS type 1 involves the upper extremity more often than the lower extremity.[1,2,6–8] Fractures have been implicated among the most common triggering events for the development of CRPS, with one study finding 44% of CRPS cases developed after a fracture.[7,9,10] The distal radius is the most common fracture associated with CRPS in the contemporary literature. This is likely due to the rich nerve supply in the area of the distal radius, leading to significant neuro-inflammatory responses that can trigger CRPS.[9] A recent study using data from 2007 to 2011 reported an overall incidence rate of 0.07% of CRPS type 1 in a sample of 33,406,123 admitted patients.[11] The incidence of CRPS after distal radius fracture (DRF) has been reported between 1.1% and 36.7% in different studies.[12–15]

The focus of this study was to evaluate various risk factors that have been reported in the literature for the development of CRPS after DRF and to investigate the association of the preexisting cervical radiculopathy with the development of CRPS following DRF.

MATERIALS AND METHODS

Data source

A retrospective analysis was conducted of data from 2010 to 2022 using the PearlDiver database[16] (PearlDiver Inc), an extensive, all-payer, national claims database that includes 165 million unique inpatient records from various health-care providers across the United States. The database is publicly accessible and compliant with the Health Insurance Portability and Accountability Act. The data set encompasses information from the International Classification of Diseases, Ninth and Tenth Revisions (ICD-9 and ICD-10), Current Procedural Terminology (CPT) codes, as well as demographic, medical, and surgical details. The supplementary tables include the list of ICD and CPT codes that were used in this study. This study did not require institutional review board approval because the data were deidentified.

Study cohort creation

The DRF cohort was divided into two—those who developed CRPS after the DRF and those who did not (Fig. 1).

Because there is enough evidence in the body of literature suggesting that age,[17] female sex,[17] complex and high energy DRF presentations,[18] and more invasive fixation procedures[18] predispose the patient to develop CRPS after DRF. Propensity scores were used to match DRF with CRPS, postfracture, to their controls with age, sex, three types of DRF (closed DRF, open DRF, and DRF that end up in fracture-related complications like nonunion and malunion), and five types of DRF management (CPT-25605, CPT-25606, CPT-35607, CPT-25608, CPT-25609). We used a 1:1 nearest-neighbor matching approach with a 0.2 standard deviation caliper size to ensure the closest possible match. We used 1:1 nearest-neighbor propensity score matching without replacement based on age, sex, obesity, fracture type (closed, open, and complicated), and index procedure CPT codes (25605−25609). Covariate balance before and after matching was assessed using standardized mean differences (SMDs), with an absolute SMD <0.1 considered indicative of adequate balance. Hypothesis testing (t tests, χ^2 tests) was reported descriptively but not used as the primary criterion for balance.

Analysis on propensity score-matched cohorts

After 1:1 propensity score matching, a combined cohort of patients with and without CRPS was used for multivariable logistic regression analysis, with

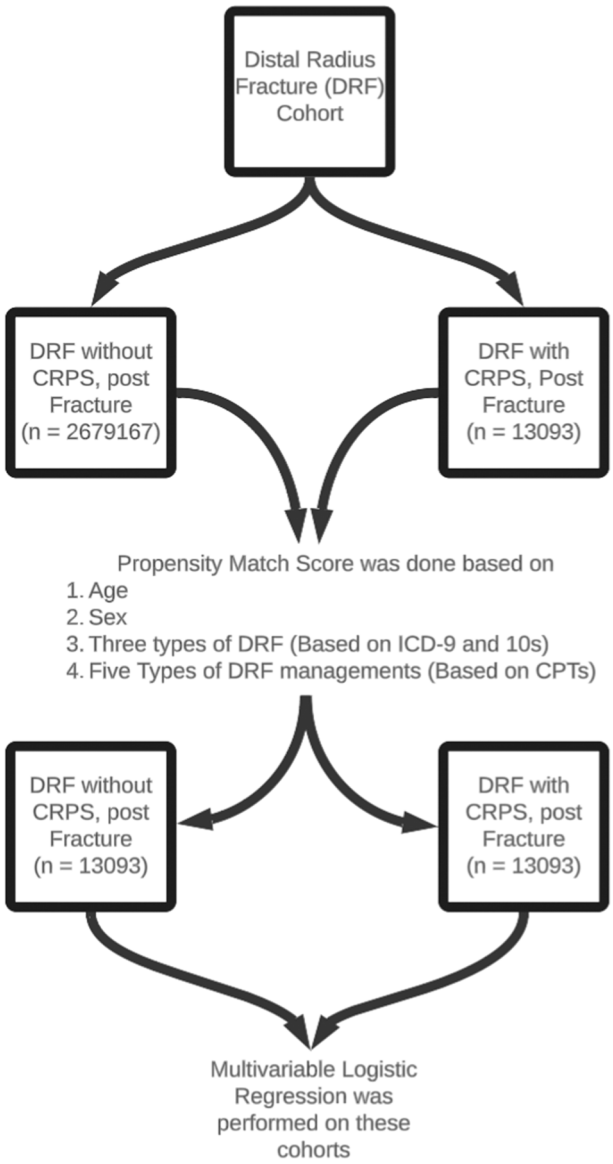

FIGURE 1: Study cohort creation: DRF cohort was divided into two—those who developed CRPS after the DRF and those who did not.

the presence of CRPS as the dependent variable. Independent variables included a priori clinically relevant comorbidities. Psychiatric conditions, anxiety disorders and depression,[19] were included based on prior evidence demonstrating their association with the development of CRPS after DRF. Fibromyalgia,[20,21] another condition consistently linked to heightened pain sensitivity and increased CRPS risk, was also incorporated.

Neuropathic conditions affecting the upper limb were evaluated as potential predictors, including carpal tunnel syndrome (median nerve compression), cubital tunnel syndrome (ulnar nerve compression), radial nerve lesions, documented traumatic nerve injuries, and cervical radiculopathy. These variables were selected because of their biological plausibility and prior reports suggesting increased susceptibility to disproportionate pain responses or altered neurogenic inflammation following fracture.

All models were adjusted for the specified covariates, and results were reported as adjusted odds ratios (ORs) with corresponding 95% confidence intervals (CIs).

RESULTS

Propensity score matching

Before matching, substantial differences were present between CRPS and non-CRPS cohorts across all baseline characteristics (Table 1). After 1:1 propensity score matching (13,093 vs 13,093), all covariates demonstrated excellent balance, with absolute SMDs <0.01 for every matching variable. Corresponding hypothesis tests (t tests and χ^2 tests) were nonsignificant for all variables after matching, confirming successful equalization of baseline characteristics between groups.

Multivariable regression analysis

A multivariable logistic regression analysis was performed on the propensity score-matched cohort (n = 26,186) to identify independent predictors of CRPS following DRF. Twelve clinically relevant covariates were included in the fully adjusted model, encompassing preexisting psychiatric comorbidities (anxiety disorders, depression, fibromyalgia), preexisting upper-extremity neuropathies (carpal tunnel syndrome, cubital tunnel syndrome, radial nerve lesions, traumatic nerve injury), and preexisting cervical radiculopathy, along with preexisting substance-use factors (alcohol, tobacco, cannabis, and opioids).

Variance inflation factors (VIFs) for all predictors were <1.40 (Table 2), well below the common multicollinearity threshold of 5−10,[22] indicating the absence of multicollinearity and supporting the stability of coefficient estimates.[22]

In the fully adjusted analysis (Table 2, Fig. 2), several factors were independently associated with the development of CRPS after a DRF. Alcohol use disorder was associated with a modest reduction in CRPS risk (OR 0.86, 95% CI 0.79−0.94), while tobacco use and cannabis use were not significant predictors. Chronic opioid abuse demonstrated a meaningful increase in CRPS risk (OR 1.39, 95% CI 1.27−1.52). Psychiatric comorbidities showed significant associations, including anxiety disorders

TABLE 1. Comparison of Demographic, Fracture, and Management CPTs Before and After Propensity Score Matching in DRF Patients With and Without CRPS

	Before Matching			After Matching			
Characteristic	DRF With No CRPS (n = 2,679,167)	DRF With CRPS (n = 13,093)	*P* Value	DRF With No CRPS (n = 13,093)	DRF With CRPS (n = 13,093)	*P* value	SMD (After)
Demographics							
Age, y (mean ± SD)	43.8 ± 27.7	55.6 ± 14.9	<0.001	56.0 ± 16.0	55.9 ± 16.0	1	0.00
Women, *n* (%)	1,738,652 (64.9%)	4,385 (84.8%)	<0.001	10,972 (83.8%)	10,962 (83.7%)	0.9	0.00
Fracture types, n (%)							
Closed DRF	2,655,686 (99.1%)	5,096 (98.6%)	<0.001	12,924 (98.7%)	12,917 (98.7%)	1	0.00
Open DRF	44,738 (1.7%)	217 (4.2%)	<0.001	451 (3.4%)	432 (3.3%)	1	0.01
DRF with malunion/ nonunion	48,132 (1.8%)	429 (8.3%)	<0.001	666 (5.1%)	665 (5.1%)	0.97	0.00
Management CPT codes, n (%)							
CPT 25605	295,862 (11%)	789 (15.3%)	<0.001	1,754 (13.4%)	1,750 (13.4%)	0.98	0.00
CPT 25606	48,575 (1.8%)	119 (2.3%)	0.010	345 (2.6%)	340 (2.6%)	1	0.00
CPT 25607	110,119 (4.1%)	458 (8.9%)	<0.001	1,036 (7.9%)	1,036 (7.9%)	1	0.00
CPT 25608	91,892 (3.4%)	424 (8.2%)	<0.001	934 (7.1%)	927 (7.1%)	1	0.00
CPT 25609	156,748 (5.9%)	809 (15.7%)	<0.001	1,678 (12.8%)	1,669 (12.7%)	1	0.00

(OR 1.25, 95% CI 1.18−1.32) and depression (OR 1.07, 95% CI 1.01−1.13). Fibromyalgia exhibited one of the strongest nonneuropathic associations (OR 1.58, 95% CI 1.44−1.74).

Neuropathic and nerve-related conditions demonstrated the highest magnitude of risk. Upper-limb nerve injury was the strongest predictor of CRPS (OR 3.20, 95% CI 2.42−4.30), followed by radial nerve lesions (OR 2.43, 95% CI 1.72−3.50), cubital tunnel syndrome (OR 2.27, 95% CI 2.01−2.57), and carpal tunnel syndrome (OR 2.01, 95% CI 1.88−2.14). Cervical radiculopathy also remained a significant independent predictor (OR 1.80, 95% CI 1.68−1.94). These findings suggest that preexisting neuropathic vulnerability—whether peripheral nerve compression, nerve injury, or proximal radiculopathy—may sensitize the upper limb to the disproportionate neuroinflammatory response characteristic of CRPS.

DISCUSSION

The prevalence of CRPS following DRFs reported in the literature varies widely, making it challenging to determine a standard reference value. Previous studies have shown rates ranging from 0.2% to as high as 22%.[14,23] Lipman et al[21] reported an incidence of 0.51% for CRPS within 1 year after a DRF, whereas our study had an incidence rate of 182.31 per 100,000 patients in a cohort of 1,966,974 DRF. Our study has a 0.18% incidence of CRPS after DRF, which ranges from 0.17% to 2.25% according to the type of DRF. This discrepancy might be due to previous studies[14,23] overestimating the prevalence, or it could indicate that CRPS is underdiagnosed or undercoded in the Medicare database. Another reason for such a large variance is criteria-based diagnosis of CRPS, which introduces the physician's bias to document the CRPS diagnosis. In our study, the detailed analysis of the cumulative CRPS incidence rate after DRF shows a large difference between the cohort with preexisting cervical radiculopathy and those without preexisting cervical radiculopathy.

The risk of developing CRPS after a DRF is well established to be higher in females,[24−26] individuals aged 61−70 years,[7] those undergoing open reduction compared to closed manipulation, and those receiving surgical management rather than conservative treatment.[18] However, medical risk factors for CRPS are not well understood, and there is limited evidence about the comorbidities examined in previous studies. Factors such as smoking, obesity,

TABLE 2. Result of Multivariable Logistic Regression Analysis with VIFs

Predictor	Adjusted or	*P* value	VIF
Substance abuses			
Alcohol use disorders	0.86 (0.79−0.94)	**<0.001**	1.135
Tobacco use	1.04 (0.98−1.10)	0.175	1.149
Opioid use	1.39 (1.27−1.52)	**<0.001**	1.03
Cannabis use	1.06 (0.93−1.20)	0.375	1.1
Psychiatric illnesses			
Anxiety disorders	1.25 (1.18−1.32)	**<0.001**	1.34
Depression	1.07 (1.01−1.13)	**0.026**	1.362
Fibromyalgia	1.58 (1.44−1.74)	**<0.001**	1.05
Neuropathies			
Upper-limb nerve injury	3.20 (2.42−4.30)	**<0.001**	1.015
Carpal tunnel syndrome	2.01 (1.88−2.14)	**<0.001**	1.06
Cubital tunnel syndrome	2.27 (2.01−2.57)	**<0.001**	1.059
Radial nerve lesion	2.43 (1.72−3.50)	**<0.001**	1.004
Cervical radiculopathy	1.80 (1.68−1.94)	**<0.001**	1.045

Bolded *P* values indicate statistical significance ($P < 0.05$).

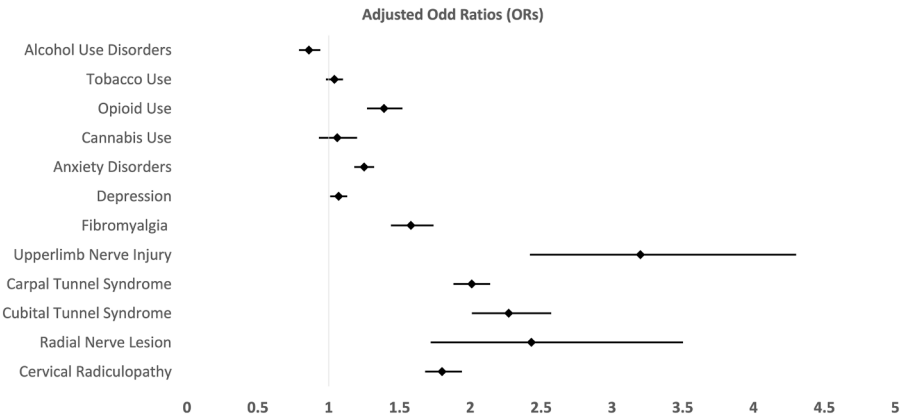

FIGURE 2: Fully adjusted forest plot analysis showing that several factors were independently associated with the development of CRPS after DRF.

hypertension, congestive heart failure, and diabetes have been investigated,[18,27] but none were found to be significant. Additionally, anxiety and depression, historically considered components of a "Sudeck" personality, were among the earliest risk factors proposed for the development of CRPS. However, some recent reviews have questioned the validity of this association.[26] Given these gaps in knowledge, we sought to investigate the association between CRPS and a broader set of clinically relevant variables. Specifically, we examined four categories of substance use (alcohol, tobacco, opioid, and cannabis), three psychiatric conditions (depression, anxiety, fibromyalgia), and five neuropathic disorders (upper-limb nerve injury, carpal tunnel syndrome, cubital tunnel syndrome, radial nerve lesions, and cervical radiculopathy). This analysis was performed in a propensity score-matched cohort of patients with and without CRPS following DRF, matched on age, sex, fracture type, and five distinct management strategies based on CPT codes. Hence, our base population was homogenous in regard to types and management of DRF, as well as age and sex of the population.

It is established that CRPS is seen more commonly in patients with chronic pain conditions.[6]

Because of the neurogenic origin of CRPS, we hypothesized that preexisting neuronal lesions like cervical radiculopathy, carpal tunnel syndrome, and cubital tunnel syndrome, radial nerve lesion and preexisting documented upper-limb neuronal injuries might predispose patients with DRFs to developing CRPS. A systematic review from 2020 estimated the prevalence of cervical radiculopathy was 1.14% for males and 1.31% for females.[28] Hence, its associations with the development of CRPS after DRF, which is the most common fracture of the wrist, have been worth analyzing.

In our multivariable regression analysis, several clear patterns emerged regarding which preexisting conditions most strongly predispose patients to developing CRPS after a DRF. Neuropathic disorders consistently demonstrated the highest predictive value, with upper-limb nerve injury, radial nerve lesions, and compressive neuropathies, such as cubital and carpal tunnel syndromes, showing the strongest associations. These conditions not only had the largest effect sizes but also relatively wide CIs, reflecting both their substantial impact and the underlying heterogeneity of patients who present with preexisting nerve pathology. In contrast, conditions such as anxiety, depression, and fibromyalgia showed more modest associations and narrower CIs, indicating smaller but more precise effects across the large matched cohort. Substance-related factors revealed a mixed pattern: chronic opioid use was a meaningful predictor, whereas alcohol and cannabis use showed either minimal or inverse associations. Among all predictors, preexisting nerve injury and compressive neuropathies provided the strongest signal for CRPS risk, suggesting that prefracture neuronal vulnerability rather than psychiatric or metabolic factors plays the most influential role in shaping postfracture CRPS. These findings collectively indicate that the best predictors of CRPS after DRF are disorders that already compromise neural integrity of the upper limb, supporting the hypothesis that a sensitized or dysfunctional peripheral−central pain pathway may set the stage for an exaggerated neuroinflammatory response following fracture.

Limitations

This study has certain limitations because of the reliance on an administrative database for patient information. It may introduce selection bias and limit the ability to establish causality. The PearlDiver database encompasses the complete Medicare Standard Analytical Files from 2010 to 2022. Consequently, our analysis depends on the accuracy of diagnosis and procedure codes provided by physicians and hospitals, with potential errors arising from miscoding or missing codes. Nevertheless, a recent report on improper payments from 2022 indicated an overall coding error rate of 0.6% in 2022, which was 1.6% in 2014.[29] Thus, although this presents a limitation, the error rate remains relatively low. Additionally, diagnoses that are not commonly linked to billing might be undercoded in administrative databases. This undercoding, however, is likely consistent across different cohorts and would not affect the identification of associations, although it may lead to an underestimation of the prevalence and magnitude of these associations. Additionally, given the use of administrative data, a key limitation is the inability to verify the clinical validity of CRPS diagnoses assigned through coding; this is particularly important for CRPS, where diagnostic criteria remain controversial and inconsistently applied, distinct from general miscoding errors. The study population, while large, may not be representative of the true general population, and the findings may not be generalizable. Future research should focus on prospective studies like randomized controlled trials to confirm these findings and explore the underlying mechanisms linking cervical radiculopathy to CRPS. Also, investigating the role of specific interventions and their impact on the development of CRPS in patients with preexisting cervical radiculopathy could provide valuable insights for clinical practice. Our multivariate regression models exhibit relatively low pseudo-R^2 values ($\sim$10%); however, our base population comprises 165 million patients, and we have incorporated all risk factors previously established in the literature for CRPS development following DRF. Certain key factors that are responsible for the development of CRPS after DRF, such as higher scores postfracture[30] and higher peripheral and central pain sensitization,[31] cannot be assessed in large administrative databases.

CONFLICTS OF INTEREST

No benefits in any form have been received or will be received related directly to this article.

REFERENCES

1. Bharwani KD, Dirckx M, Huygen FJ. Complex regional pain syndrome: diagnosis and treatment. *BJA Educ*. 2017;17(8):262−268.
2. Veldman PH, Reynen HM, Arntz IE, Goris RJ. Signs and symptoms of reflex sympathetic dystrophy: prospective study of 829 patients. *Lancet*. 1993;342(8878):1012−1016.
3. Harden NR, Bruehl S, Perez RSGM, et al. Validation of proposed diagnostic criteria (the "Budapest Criteria") for complex regional pain syndrome. *Pain*. 2010;150(2):268−274.
4. Birklein F, Ajit SK, Goebel A, Perez RSGM, Sommer C. Complex regional pain syndrome - phenotypic characteristics and potential biomarkers. *Nat Rev Neurol*. 2018;14(5):272−284.

5. Bruehl S. Complex regional pain syndrome. *BMJ*. 2015;351:h2730.
6. Ferraro MC, O'Connell NE, Sommer C, et al. Complex regional pain syndrome: advances in epidemiology, pathophysiology, diagnosis, and treatment. *Lancet Neurol*. 2024;23(5):522−533.
7. de Mos M, de Bruijn AGJ, Huygen FJPM, Dieleman JP, Stricker BHC, Sturkenboom MCJM. The incidence of complex regional pain syndrome: a population-based study. *Pain*. 2007;129(1−2):12−20.
8. Petersen PB, Mikkelsen KL, Lauritzen JB, Krogsgaard MR. Risk factors for post-treatment complex regional pain syndrome (CRPS): an analysis of 647 cases of CRPS from the Danish Patient Compensation Association. *Pain Pract*. 2018;18(3):341−349.
9. Dutton LK, Rhee PC. Complex regional pain syndrome and distal radius fracture: etiology, diagnosis, and treatment. *Hand Clin*. 2021;37(2):315−322.
10. Świta M, Szymonek P, Talarek K, et al. Complex regional pain syndrome after distal radius fracture-case report and mini literature review. *J Clin Med*. 2024;13(4):1122.
11. Elsharydah A, Loo NH, Minhajuddin A, Kandil ES. Complex regional pain syndrome type 1 predictors - epidemiological perspective from a national database analysis. *J Clin Anesth*. 2017;39:34−37.
12. Bickerstaff DR, Kanis JA. Algodystrophy: an under-recognized complication of minor trauma. *Br J Rheumatol*. 1994;33(3):240−248.
13. Atkins RM, Duckworth T, Kanis JA. Features of algodystrophy after Colles' fracture. *J Bone Joint Surg Br*. 1990;72(1):105−110.
14. Dijkstra PU, Groothoff JW, ten Duis HJ, Geertzen JHB. Incidence of complex regional pain syndrome type I after fractures of the distal radius. *Eur J Pain*. 2003;7(5):457−462.
15. Jellad A, Salah S, Ben Salah Frih Z. Complex regional pain syndrome type I: incidence and risk factors in patients with fracture of the distal radius. *Arch Phys Med Rehabil*. 2014;95(3):487−492.
16. PearlDiver Technologies. PearlDiver—custom research at your fingertips [Internet]. Accessed July 28, 2024. https://pearldiverinc.com
17. Lorente A, Mariscal G, Lorente R. Incidence and risk factors for complex regional pain syndrome in radius fractures: meta-analysis. *Arch Orthop Trauma Surg*. 2023;143(9):5687−5699.
18. Roh YH, Gong HS, Baek GH. Prognostic value of pain sensitization during early recovery after distal radius fracture in complex regional pain syndrome. *Pain Med*. 2019;20(6):1066−1071.
19. Pereira DE, Momtaz D, Gonuguntla R, et al. Patients with preexisting anxiety and mood disorders are more likely to develop complex regional pain syndrome after fractures. *Clin Orthop Relat Res*. 2024;482(2):222−230.
20. Crijns TJ, van der Gronde BATD, Ring D, Leung N. Complex regional pain syndrome after distal radius fracture is uncommon and is often associated with fibromyalgia. *Clin Orthop Relat Res*. 2018;476(4):744−750.
21. Lipman MD, Hess DE, Werner BC, Deal DN. Fibromyalgia as a predictor of complex regional pain syndrome after distal radius fracture. *Hand N Y N*. 2019;14(4):516−522.
22. Kim JH. Multicollinearity and misleading statistical results. *Korean J Anesthesiol*. 2019;72(6):558−569.
23. Zollinger PE, Tuinebreijer WE, Kreis RW, Breederveld RS. Effect of vitamin C on frequency of reflex sympathetic dystrophy in wrist fractures: a randomised trial. *Lancet*. 1999;354(9195):2025−2028.
24. Kim YD. Diagnosis of complex regional pain syndrome. *Ann Clin Neurophysiol*. 2022;24(2):35−45.
25. Taylor SS, Noor N, Urits I, et al. Complex regional pain syndrome: a comprehensive review. *Pain Ther*. 2021;10(2):875−892.
26. Pons T, Shipton EA, Williman J, Mulder RT. Potential risk factors for the onset of complex regional pain syndrome type 1: a systematic literature review. *Anesthesiol Res Pract*. 2015;2015:956539.
27. Zyluk A. Complex regional pain syndrome type I. Risk factors, prevention and risk of recurrence. *J Hand Surg Br*. 2004;29(4):334−337.
28. Mansfield M, Smith T, Spahr N, Thacker M. Cervical spine radiculopathy epidemiology: a systematic review. *Musculoskeletal Care*. 2020;18(4):555−567.
29. Centers for Medicare & Medicaid Services (CMS). 2022 Medicare fee-for-service supplemental improper payment data [Internet]. Accessed July 29, 2024. https://www.cms.gov/files/document/2022-medicare-fee-service-supplemental-improper-payment-data.pdf
30. Wang AWT, Lefaivre KA, Potter J, et al. Complex regional pain syndrome after distal radius fracture: a survey of current practices. *PLoS One*. 2024;19(11):e0314307.
31. Abd-Elsayed A, Stark CW, Topoluk N, et al. A brief review of complex regional pain syndrome and current management. *Ann Med*. 2024;56(1):2334398.