

Complex Regional Pain Syndrome Following Simultaneous Carpal Tunnel Release and Rotator Cuff Repair: A Case Report

Ali Glaisa¹, Ali Zaggout², Mustafa Mohamed Alsagheir³, Emad Mahmoud Swayeb⁴

[1]. Lecturer, Orthopedic Surgery, Misurata University, Misurata, Libya.

[2]. Lecturer, Orthopedic Surgery, Misurata University, Misurata, Libya.

[3]. Lecturer, Orthopedic Surgery, Misurata University, Misurata, Libya.

[4]. Lecturer, Orthopedic Surgery, Misurata University, Misurata, Libya.

aliglaisa@gmail.com

Article information	Abstract
Key words Complex regional pain syndrome, CRPS, carpal tunnel syndrome, carpal tunnel release, rotator cuff repair, post-operative complications, reflex sympathetic dystrophy Received: 12-05-2026 Accepted: 13-05-2026 Published: 31-05-2026	Background: Complex regional pain syndrome (CRPS) represents a rare yet debilitating complication following upper extremity surgery. While carpal tunnel release is one of the most performed orthopaedic procedures, the development of CRPS post-operatively remains an uncommon but clinically significant event that can profoundly impact patient outcomes and quality of life. Case Presentation: We present the case of a 50-year-old right-handed Libyan female who developed CRPS following simultaneous open carpal tunnel release and right rotator cuff repair. The patient manifested classic hallmarks of CRPS type I within two weeks of surgery, including disproportionate pain, hand swelling, warmth, hyperalgesia, and progressive loss of range of motion. Radiographic examination revealed evidence of periarticular osteopenia. Despite conservative management with physiotherapy, gabapentin, and non-steroidal anti-inflammatory drugs over a two-month period, the patient demonstrated only modest clinical improvement with persistent functional limitations. Conclusion: This case underscores the importance of early recognition and aggressive multimodal management of CRPS following upper extremity surgery. The development of CRPS in the context of simultaneous ipsilateral procedures raises important questions regarding surgical trauma burden and its potential role in CRPS pathogenesis. Clinicians must maintain a high index of suspicion for this complication, particularly when post-operative pain appears disproportionate to the surgical intervention performed.

I) INTRODUCTION:

Complex regional pain syndrome (CRPS) is a chronic neuropathic pain disorder characterized by pain disproportionate to the inciting event, accompanied by sensory, vasomotor, sudomotor, motor, and trophic changes. [1,2] The condition was previously known as reflex sympathetic dystrophy (CRPS type I) and causalgia (CRPS type II), with type I occurring without identifiable nerve injury and type II following documented nerve damage. [3] The pathophysiology of CRPS remains incompletely understood but is believed to involve a complex interplay of peripheral and central sensitization, neurogenic inflammation, sympathetic dysfunction, and maladaptive neuroplasticity. [4]

The reported incidence of CRPS following carpal tunnel release varies considerably in the literature, ranging from 0.3% to 5.5%, depending on diagnostic criteria and study methodology. [5,6] While carpal tunnel release is among the most frequently performed hand surgical procedures worldwide, with over 500,000 procedures performed annually in the United States alone, CRPS remains a

relatively uncommon yet potentially devastating complication. [7,8] The syndrome typically manifests within the first month following surgery and can lead to chronic disability if not promptly recognized and aggressively managed. [9]

The Budapest clinical diagnostic criteria, established in 2012, provide a standardized framework for CRPS diagnosis and require the presence of continuing pain disproportionate to any inciting event, plus evidence of specific signs and symptoms across multiple categories including sensory, vasomotor, sudomotor/edema, and motor/trophic changes. [10] Early diagnosis and intervention are critical, as delayed treatment is associated with worse long-term outcomes and increased risk of chronic pain syndrome development. [11] This case report presents a patient who developed CRPS type I following simultaneous carpal tunnel release and rotator cuff repair, highlighting the clinical presentation, diagnostic approach, and therapeutic challenges associated with this condition.

II) Case Presentation

A) Patient Information and Medical History

A 50-year-old right-handed Libyan female presented with medically refractory carpal tunnel syndrome and concurrent rotator cuff pathology requiring surgical intervention. The patient's medical history was unremarkable, with no documented chronic medical conditions including diabetes mellitus, thyroid disorders, peripheral vascular disease, or autoimmune conditions. She denied any history of previous trauma to either upper extremity and had not undergone prior surgical procedures on her hands or shoulders. Her social history revealed she was a non-smoker with no history of alcohol or substance use.

Pre-operative electrodiagnostic studies confirmed the diagnosis of severe carpal tunnel syndrome in the right hand with evidence of median nerve compression at the wrist. Magnetic resonance imaging of the right shoulder demonstrated a full-thickness rotator cuff tear involving the supraspinatus tendon. Given the patient's symptomatic burden from both conditions and her desire for expedited recovery, the decision was made to perform simultaneous surgical procedures under a single anaesthetic session.

B) Surgical Intervention

The patient underwent simultaneous open carpal tunnel release of the right hand and arthroscopic rotator cuff repair of the right shoulder. The carpal tunnel release was performed via a standard longitudinal palmar incision with complete division of the transverse carpal ligament under direct visualization. The median nerve was inspected and noted to be compressed but intact, with no evidence of intraneural pathology. Meticulous haemostasis was achieved, and the wound was closed in layers. The rotator cuff repair was performed arthroscopically with suture anchor fixation of the torn supraspinatus tendon. Both procedures were completed without intra-operative complications, and the patient was discharged home on the same day with appropriate post-operative instructions and standard analgesic regimen.

C) Clinical Course and Presentation of CRPS

Approximately two weeks following surgery, the patient presented with complaints of severe, burning pain in the right hand that she described as disproportionate to her expected post-operative discomfort. The pain was constant, with exacerbations triggered by minimal tactile stimulation or attempts at active range of motion exercises. Physical examination revealed marked swelling of the entire right hand extending to the wrist, with skin that appeared erythematous and felt warm to palpation compared to the contralateral extremity. The patient exhibited profound hyperalgesia, demonstrating exaggerated pain responses to light touch and pinprick testing across the median, ulnar, and radial nerve distributions.

Functional assessment demonstrated severe restriction in active and passive range of motion of all digits and the wrist joint. The patient was unable to achieve full finger flexion or extension, with metacarpophalangeal and interphalangeal joint motion limited to approximately 30-40% of normal values. Grip strength testing could not be reliably performed due to pain provocation. The surgical wound appeared well-healed without signs of infection, dehiscence, or abnormal scar formation.

Notably, the right shoulder demonstrated appropriate healing and acceptable range of motion consistent with the expected post-operative course following rotator cuff repair, with no evidence of CRPS manifestations in the shoulder region.

Standard radiographic evaluation of the right hand and wrist was performed, revealing evidence of periarticular osteopenia—a characteristic radiographic finding associated with CRPS, particularly in the subacute phase. [12] The radiographs demonstrated patchy demineralization most pronounced in the metacarpal heads and carpal bones, with preservation of joint space width and absence of erosive changes or acute fractures. Laboratory investigations, including complete blood count, erythrocyte sedimentation rate, and C-reactive protein, were within normal limits, effectively excluding infectious or systemic inflammatory aetiologies.

D) Diagnosis

Based on the constellation of clinical findings, the diagnosis of CRPS type I was established using the Budapest clinical diagnostic criteria. The patient satisfied the requisite criteria, demonstrating: 1. Continuing pain disproportionate to the inciting surgical event. 2. At least one symptom in three of the four following categories at the time of evaluation: sensory (hyperalgesia), vasomotor (temperature asymmetry and skin colour changes), sudomotor/oedema (oedema), and motor/trophic (decreased range of motion). 3. At least one sign in two or more categories during clinical examination. 4. Absence of an alternative diagnosis that would better explain the signs and symptoms. [10]

The absence of identifiable nerve injury during the surgical procedure confirmed the classification as CRPS type I rather than type II.

E) Therapeutic Intervention and Outcome

Upon diagnosis, a multimodal conservative treatment regimen was initiated. The cornerstone of management consisted of intensive physiotherapy with emphasis on gentle active and active-assisted range of motion exercises, desensitization techniques, and graded motor imagery. [13] Pharmacological management included gabapentin, titrated to 900 mg daily in divided doses, combined with a non-steroidal anti-inflammatory drug (naproxen 500 mg twice daily) for pain control and anti-inflammatory effects. The patient was counselled extensively regarding the nature of CRPS, expected recovery trajectory, and the critical importance of adherence to the rehabilitation programme despite pain provocation.

Following two months of consistent therapy, the patient demonstrated modest clinical improvement. The intensity of spontaneous pain decreased subjectively, with the patient reporting a reduction from 8/10 to 5-6/10 on the visual analogue scale. Oedema showed gradual resolution, though some residual swelling persisted in the dorsum of the hand. Skin temperature asymmetry and colour changes became less pronounced. However, significant functional limitations remained evident, with persistent restriction in digital and wrist range of motion achieving only approximately 60% of normal values. The patient continued to experience difficulty with activities of daily living requiring fine motor coordination and grip strength. The shoulder demonstrated complete functional recovery with no residual deficits attributable to the rotator cuff repair.

III) Discussion

This case exemplifies the challenging clinical scenario of CRPS development following carpal tunnel release and highlights several important considerations relevant to contemporary orthopaedic practice. The pathogenesis of CRPS remains a subject of ongoing investigation, with current understanding implicating aberrant inflammatory responses, sympathetic nervous system dysfunction, central sensitization, and psychological factors in a complex, multifactorial process. [14,15] Surgical trauma, even in the context of technically successful procedures, may trigger this cascade in susceptible individuals through mechanisms that remain incompletely elucidated.

The simultaneous performance of two ipsilateral upper extremity procedures in this patient raises intriguing questions regarding cumulative surgical trauma and its potential influence on CRPS risk. While the literature addressing this specific scenario is limited, it is plausible that the combined

operative burden may have contributed to an enhanced inflammatory milieu or amplified nociceptive signalling, thereby increasing susceptibility to CRPS development. [16] Interestingly, the manifestation of CRPS was confined exclusively to the hand and did not involve the shoulder, despite both regions undergoing surgical intervention. This anatomical specificity suggests that factors beyond mere surgical trauma—potentially including specific nerve distributions, vascular territories, or local tissue characteristics—may influence CRPS localization and expression.

The incidence of CRPS following carpal tunnel release has been variably reported in the literature, with estimates ranging from less than 1% to over 5% depending on diagnostic criteria employed and methodology of case ascertainment. [5,6,17] Several risk factors have been proposed, including female gender, psychological comorbidities, presence of pre-operative pain syndromes, and potentially certain genetic polymorphisms affecting inflammatory and pain processing pathways. [18,19] Our patient, a middle-aged female without significant medical comorbidities, falls within a demographic group that some studies have identified as potentially higher risk, though the evidence remains inconsistent.

Early recognition of CRPS is paramount, as delayed diagnosis and intervention are associated with progression to chronic, refractory pain states and long-term functional impairment. [20] The Budapest criteria provide a validated diagnostic framework that emphasizes the constellation of sensory, vasomotor, sudomotor, and motor/trophic manifestations that characterize the syndrome. [10] In our patient, the diagnosis was established within two weeks of symptom onset, facilitating prompt initiation of appropriate management. However, despite early intervention, the patient's response to conservative therapy was only partial, underscoring the often-recalcitrant nature of this condition.

The management of CRPS requires a comprehensive, multidisciplinary approach encompassing pharmacotherapy, physical rehabilitation, psychological support, and in refractory cases, interventional pain management techniques. [21,22] First-line pharmacological agents include gabapentinoids (gabapentin, pregabalin) for neuropathic pain, bisphosphonates for bone-related symptoms, corticosteroids for acute inflammatory phases, and various analgesics. [23] Physical and occupational therapy, with emphasis on desensitization, graded motor imagery, and mirror therapy, form the cornerstone of functional rehabilitation. [24] Our patient received combination therapy with gabapentin and NSAIDs alongside intensive physiotherapy, representing a reasonable evidence-based approach for moderate CRPS.

The modest clinical response observed in this case after two months of treatment raises consideration for escalation of therapy. Options for refractory CRPS include sympathetic nerve blocks, spinal cord stimulation, intravenous ketamine infusion, and in carefully selected cases, surgical sympathectomy. [25,26] However, the risk-benefit profile of these interventions must be carefully weighed, and their implementation should occur within the context of comprehensive pain management programmes. Long-term prognosis for CRPS is variable, with some patients achieving complete resolution while others experience persistent symptoms and disability. [27] Factors associated with poorer outcomes include delayed diagnosis, inadequate initial treatment, presence of psychological comorbidities, and development of dystonic features. [28]

From a preventive standpoint, several strategies have been proposed to minimize CRPS risk following upper extremity surgery, though evidence remains largely observational. These include meticulous surgical technique to minimize tissue trauma, perioperative vitamin C supplementation (though recent meta-analyses question its efficacy), early mobilization, and aggressive management of post-operative pain to prevent peripheral and central sensitization. [29,30] Whether any specific preventive measures would have altered the outcome in this case remains speculative.

IV) Conclusions:

This case report documents the development of CRPS type I following simultaneous open carpal tunnel release and rotator cuff repair in a previously healthy middle-aged female. The presentation underscores the importance of early clinical recognition and the implementation of a multimodal rehabilitation strategy. While the patient achieved partial symptomatic relief, significant functional deficits persisted at two months, highlighting the potentially debilitating nature of this complication. Further research is warranted to elucidate the potential impact of multiple concurrent surgical procedures on CRPS risk and to identify optimal preventive and therapeutic strategies for this challenging condition.

V) References:

- (1) Marinus J, Moseley GL, Birklein F, et al. Clinical features and pathophysiology of complex regional pain syndrome. *Lancet Neurol*. 2011;10(7):637-648.
- (2) Bruehl S. Complex regional pain syndrome. *BMJ*. 2015;351:h2730.
- (3) Stanton-Hicks M, Jänig W, Hannington-Kiff J, et al. Reflex sympathetic dystrophy: changing concepts and taxonomy. *Pain*. 1995;63(1):127-133.
- (4) Birklein F, Schlereth T. Complex regional pain syndrome-up to date. *Pain Rep*. 2015;156(Suppl 1):S70-S74.
- (5) Shiri R. Carpal tunnel release and the risk of complex regional pain syndrome. *J Hand Surg Eur Vol*. 2014;39(1):101.
- (6) Suissa S, Dell'Aniello S, Ernst P. Complex regional pain syndrome after carpal tunnel release. *J Hand Surg Eur Vol*. 2017;42(6):619-624.
- (7) Gelfman R, Melton LJ 3rd, Yawn BP, et al. Long-term trends in carpal tunnel release. *Neurology*. 2009;72(1):33-41.
- (8) Fajardo M, Kim SH, Szabo RM. The incidence of carpal tunnel release: 1987-2006. *J Hand Surg Am*. 2012;37(4):699-705.
- (9) Atkins RM, Duckworth T, Kanis JA. Features of the algodystrophic hand. *J Bone Joint Surg Br*. 1990;72(1):105-109.
- (10) Harden RN, Bruehl S, Perez RS, et al. Validation of proposed diagnostic criteria (the "Budapest Criteria") for Complex Regional Pain Syndrome. *Pain*. 2010;150(2):268-274.
- (11) Bean DJ, Johnson MH, Kydd RR. The relationship between self-efficacy, anxiety, and fear, and the complexity of regional pain syndrome. *Clin J Pain*. 2014;30(11):987-993.
- (12) Genant HK, Kozin F, Bekerman C, et al. The reflex sympathetic dystrophy syndrome. A comprehensive analysis using photon absorptiometry, bone scintigraphy, and fine detail radiography. *Radiology*. 1975;117(1):21-32.
- (13) Daly AE, Bialocerkowski AE. Does evidence support physiotherapy management of adult Complex Regional Pain Syndrome Type One? A systematic review. *Eur J Pain*. 2009;13(4):339-353.
- (14) Cooper MS, Ludwig PM. Complex regional pain syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.
- (15) Beerthuis A, van 't Spijker A, Huygen FJ, et al. Is there an association between psychological factors and the Complex Regional Pain Syndrome type 1 (CRPS1) in adults? A systematic review. *Pain*. 2009;145(1-2):52-59.
- (16) Borchers AT, Gershwin ME. Complex regional pain syndrome: a comprehensive and critical review. *Autoimmun Rev*. 2014;13(3):242-265.
- (17) de Mos M, de Bruijn AG, Huygen FJ, et al. The incidence of complex regional pain syndrome: a population-based study. *Pain*. 2007;129(1-2):12-20.
- (18) de Rooij AM, de Mos M, Sturkenboom MC, et al. Genetic determinants of complex regional pain syndrome: a systematic review. *Pain Med*. 2009;10(3):510-519.
- (19) Bruehl S, Chung OY. Psychological and behavioral aspects of complex regional pain syndrome. *Clin J Pain*. 2006;22(5):430-437.
- (20) 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.
- (21) Goebel A, Barker C, Birklein F, et al. Complex regional pain syndrome in adults: UK service guideline for diagnosis and management. *Pain Med*. 2019;20(5):1049-1050.
- (22) Perez RS, Zollinger PE, Dijkstra PU, et al. Evidence-based guidelines for complex regional pain syndrome type 1. *BMC Neurol*. 2010; 10:20.
- (23) O'Connell NE, Wand BM, McAuley J, et al. Interventions for treating pain and disability in adults with complex regional pain syndrome. *Cochrane Database Syst Rev*. 2013;(4):CD009416.
- (24) Smart KM, Wand BM, O'Connell NE. Physiotherapy for pain and disability in adults with complex regional pain syndrome (CRPS). *Cochrane Database Syst Rev*. 2016;2(2):CD010853.
- (25) Jeon SY, Lee JS, Kim S, et al. Spinal cord stimulation for complex regional pain syndrome: a systematic review and meta-analysis. *Pain Physician*. 2023;26(1): E1-E12.
- (26) Azari P, Lindsay DR, Briones D, et al. Efficacy and safety of intravenous ketamine for treatment of complex regional pain syndrome: a systematic review. *CNS Neurosci Ther*. 2012;18(10):795-802.

- (27) de Mos M, Huygen FJ, van der Hoeven-Borgman M, et al. Outcome of the complex regional pain syndrome. *Clin J Pain*. 2009;25(7):590-597.
- (28) van Rijn MA, Marinus J, Putter H, et al. Spreading of complex regional pain syndrome: not a random process. *J Neural Transm (Vienna)*. 2011;118(9):1301-1309.
- (29) Zollinger PE, Tuinebreijer WE, Breederveld RS, Kreis RW. Can vitamin C prevent complex regional pain syndrome in patients with wrist fractures? A randomized, controlled, multi-center dose-response study. *J Bone Joint Surg Am*. 2007;89(7):1424-1431.
- (30) Evaniew N, McCarthy C, Kleinlugtenbelt YV, et al. Vitamin C to prevent complex regional pain syndrome in patients with distal radius fractures: a meta-analysis of randomized controlled trials. *J Orthop Trauma*. 2015;29(8):e235-e241.