UDC 616.728.3-089.843-06:616-008.6-009.7-031.3
DOI: 10.14739/2310-1210.2026.1.338147

Complex regional pain syndrome as a complication following total knee arthroplasty: clinical significance and impact on functional outcomes

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Aim. To evaluate the impact of complex regional pain syndrome (CRPS) on outcomes following primary and revision total knee arthroplasty (TKA). The study assesses the incidence, clinical manifestations, and functional consequences of CRPS, while evaluating current diagnostic and treatment approaches and the need for standardization.

Materials and methods. A systematic scientific review was conducted, covering literature from PubMed, Scopus, and Web of Science published between 2015 and 2025. Inclusion criteria focused on studies of adult patients undergoing TKA who developed CRPS diagnosed by IASP or Budapest criteria. The analysis targeted incidence, clinical manifestations, risk factors, diagnostic approaches, treatment efficacy, and impact on rehabilitation outcomes.

Results. CRPS remains a significant complication of TKA, with a reported incidence of approximately 0.2–2.0 % after primary procedures and 2–5 % following revision surgery. Clinical presentation is characterized by refractory pain, vasomotor dysfunction, and restricted range of motion, leading to significantly lower WOMAC and SF-36 scores and prolonged rehabilitation compared to uncomplicated TKA. Diagnosis remains challenging due to the lack of standardized early-detection protocols and symptom overlap with other complications, and underutilization of international diagnostic criteria. While multimodal management (pharmacotherapy, physiotherapy, and interventional techniques) is effective, its success is highly dependent on early initiation. High-quality evidence regarding CRPS in the specific context of revision TKA remains limited.

Conclusions. CRPS adversely affects functional outcomes after TKA, with higher prevalence observed in revision cases. The absence of standardized preventive measures and optimized therapeutic regimens highlights an urgent need for personalized rehabilitation strategies and the implementation of international diagnostic criteria in routine clinical practice. Further research is needed to establish precise diagnostic criteria, effective prevention strategies, and personalized therapeutic and rehabilitation approaches.

Keywords: pain, endoprosthetics, knee joint.

Zaporozhye Medical Journal. 2026;28(1):47-52

Синдром комплексного регіонального болю як ускладнення після тотального ендопротезування колінного суглоба: клінічне значення та вплив на функціональні результати

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Мета роботи – проаналізувати вплив синдрому комплексного регіонального болю (СКРБ) на результати тотального ендопротезування колінного суглоба (ТЕП), визначити вплив СКРБ на результати; вивчити частоту ускладнення, клінічні прояви та функціональні наслідки, оцінити наявні підходи до діагностики, лікування та потребу в стандартизації.

Матеріали і методи. Здійснили огляд наукової літератури, що ідексується в базі даних PubMed, та інших рецензованих джерел за період 2015–2025 рр. До дослідження залучено праці, присвячені вивченню СКРБ після первинного або ревізійного ТЕП у дорослих пацієнтів, із чіткими діагностичними критеріями (IASP / Будапештські). Аналізували інцидентність, клінічні прояви, фактори ризику, діагностичні підходи, ефективність лікування та вплив на реабілітаційні результати.

Результати. СКРБ – важливе і значуще ускладнення ТЕП колінного суглоба з частотою 0,2–2,0 % після первинних і 2–5 % випадків після ревізійних операцій. Це ускладнення супроводжується вираженим болем, вазомоторними і руховими порушеннями, суттєво знижує показники WOMAC і SF-36 та подовжує терміни реабілітації. Діагностика залишається складною, оскільки досі немає стандартизованих протоколів, симптоми неспецифічні, схожі на інші ускладнення, а міжнародні критерії недостатньо використовують на практиці. Лікування багатокомпонентне, передбачає фармакотерапію, фізіотерапію, застосування інтервенційних методів, потребує раннього початку для кращого прогнозу. Водночас бракує уніфікованих рекомендацій і даних досліджень з високим рівнем доказовості, особливо щодо ревізійного ТЕП.

Висновки. СКРБ суттєво погіршує результати ендопротезування колінного суглоба, особливо після ревізійних операцій. Досі не укладено чіткі протоколи діагностики, не стандартизовано профілактичні заходи та не оптимізовано схеми лікування, і це ускладнює менеджмент таких пацієнтів. Доцільно продовжити дослідження СКРБ для визначення точних діагностичних критеріїв, ефективних профілактичних стратегій і персоналізованих підходів до терапії та реабілітації.

Ключові слова: біль, ендопротезування, колінний суглоб.

Запорізький медичний журнал. 2026. Т. 28, № 1(154). С. 47-52

Total knee arthroplasty (TKA) is considered the "gold standard" for treating end-stage knee osteoarthritis, providing significant pain relief and functional restoration for the majority of patients [1]. However, postoperative complications occur in 10–20 % of cases, substantially reducing patient satisfaction [2]. Among these, complex regional pain syndrome (CRPS), historically referred to as reflex sympathetic dystrophy, represents a challenging complication. CRPS is characterized by severe pain, along with sensory, autonomic, and motor impairments that are typically disproportionate to the inciting surgical trauma [3,4].

The incidence of CRPS following primary TKA (pTKA) is estimated at 0.2–2.0 %, but this risk increases to 2–5 % following revision TKA (rTKA) [5,6]. Revision procedures are associated with higher complication rates due to extensive soft tissue dissection, repeated surgical trauma, and potential perioperative nerve damage [7,8]. This systematic review synthesizes current literature regarding CRPS after pTKA and rTKA, compares incidence rates, evaluates the impact on functional outcomes, and identifies diagnostic and therapeutic gaps to guide future clinical research.

Aim

To evaluate the clinical significance of complex regional pain syndrome as a complication following TKA, assess its impact on functional recovery, and identify current evidence-based strategies for early diagnosis and optimized rehabilitation in affected patients.

Materials and methods

This systematic review was conducted through a targeted analysis of scientific literature on CRPS in patients following TKA. A comprehensive literature search was performed across international electronic databases, including PubMed / MEDLINE and others, covering the period from 2015 to 2025. The search strategy utilized combinations of the following terms and keywords: "complex regional pain syndrome", "CRPS", "total knee arthroplasty", "revision arthroplasty", and "postoperative complications".

Inclusion criteria: clinical studies evaluating CRPS after pTKA or rTKA; human participant trials; publications in English or Ukrainian that utilized validated diagnostic frameworks (the IASP / Budapest criteria).

Exclusion criteria: non-clinical studies; case reports with limited sample sizes, commentaries, or studies lacking full-text access; research focusing exclusively on surgical technique without reporting postoperative follow-up data.

The methodological quality of the included studies was appraised based on study design, cohort size, and the validity of the diagnostic tools employed. A total of 24 peer-reviewed sources met the final inclusion criteria and addressed various aspects of CRPS following pTKA and rTKA. The analysis focused on the quality of study design, sample characteristics, assessed parameters, and relevance to the objectives of this review.

Results

The incidence of CRPS following pTKA ranges from 0.2 % to 2.0 % [9,10,11]. A study by J. D. Kosy et al. reported no cases of CRPS among 100 patients after pTKA based on the Budapest criteria, underscoring the rarity of this condition and the paramount importance of accurate diagnosis [12]. This underscores the condition's rarity but also suggests potential underdiagnosis due to significant diagnostic challenges [9]. Similarly, M. Duenes et al. noted that up to 50 % of patients with unexplained pain following TKA may present with a neuropathic pain component, necessitating a careful differential diagnosis; however, their study did not specifically address revision TKA [6].

The incidence increases to 2–5 % after rTKA, which is attributed to extensive tissue trauma, scarring, and prolonged operative time [13,14,15]. The elevated risk following revision surgery is further associated with more challenging surgical access and potential perioperative nerve damage *(Fig. 1)*.

Clinical manifestations. CRPS following TKA is characterized by severe pain, often described as burning or shooting, accompanied by allodynia (pain from non-noxious stimuli) and hyperalgesia (an exaggerated pain response).

Additional clinical signs include edema, skin discoloration (erythema, cyanosis, or mottling), temperature asymmetry, abnormal sweating (sudomotor changes), and motor impairments such as weakness or tremors [3,6]. Following revision TKA, symptoms may be more pronounced due to repeated tissue trauma and heightened inflammatory responses [14,16]. Differential diagnosis remains challenging due to the significant overlap of symptoms with other common postoperative complications, such as periprosthetic joint infection or prosthetic instability [2,13].

CRPS diagnostic framework. Several diagnostic frameworks, including the Bruehl (Budapest), Atkins, and Veldman criteria, are employed to clinically confirm CRPS. Currently, the Budapest Criteria, endorsed by the International Association for the Study of Pain (IASP), are recognized as the global gold standard for CRPS diagnosis [17]. These criteria necessitate the presence of pain that is disproportionate to the inciting event, accompanied by a specific threshold of subjective symptoms and objective clinical signs across four distinct categories, provided that no other diagnosis can better explain the clinical presentation *(Table 1)*.

Differential diagnosis. The diagnosis of CRPS following TKA is challenging due to significant symptom overlap with other common postoperative complications. A thorough differential diagnosis is essential to ensure appropriate and timely intervention *(Table 2, Fig. 2)*.

Risk factors for CRPS following TKA include female sex (3:1 ratio), younger age (<60 years), psychological disorders (depression, anxiety, pain catastrophizing), severe preoperative pain, sleep disturbances, and prolonged tourniquet use [7,18]. For rTKA, additional risk factors include a history of prior knee surgeries, infectious complications of the primary prosthesis, and extended operative time [13,19]. In rare cases, hypersensitivity to metals (nickel) in the endoprosthesis may contribute to CRPS development, although other etiologies, such as periprosthetic joint infection, must be rigorously excluded *(Table 3)* [14,17,20].

Impact on clinical outcomes. CRPS significantly impairs functional outcomes following TKA. Patients with CRPS exhibit significantly lower WOMAC scores (mean 45 vs. 75 in the non-CRPS group, $p < 0.001$) after pTKA and even lower scores (mean 38 vs. 70, $p < 0.001$) following rTKA [9,14].

Table 1. Diagnostic criteria

Criteria	Diagnostic approach	Clinical signs / symptoms	Additional comments, treatment
Budapest criteria	All 4 conditions must be present: persistent pain, at least 1 sign in 2+ categories, at least 1 symptom in 3+ categories, exclusion of other diagnoses	4 categories: sensory (allodynia, hyperalgesia); vasomotor (change in skin temperature / color); sudomotor (edema, sweating); motor / trophic (movement restrictions, dystonia, nail / skin / hair changes)	Confirmation of diagnosis only after exclusion of other conditions. Used in international protocols
Atkins criteria	Clinical confirmation in the presence of a complex of signs	Neuropathic pain (burning, allodynia); vasomotor / sweating disorders; edema; mobility restrictions, degenerative changes	Suitable for initial diagnosis. Focused on the functional state of the limb
Veldman criteria	≥4 of 5 core symptoms required + assessment of additional factors	Pain when moving; difference in temperature / color / volume of the affected limb; restrictions on movement; spread beyond the lesion; refractory pain after surgery / injury	The stages of treatment are described in detail: medications, physiotherapy, psychotherapy, neurostimulation, surgery. Emphasis on early diagnosis and a comprehensive approach

Table 2. Differential diagnosis of CRPS after TKA

Condition	Main symptoms	Distinguishing features from CRPS	Diagnostic methods
Infection	Pain, swelling, redness, local hyperthermia, systemic signs (fever, increased C-reactive protein, leukocytosis)	Systemic manifestations, elevated inflammatory markers, pain localized to the joint	WBC count, C-reactive protein, ESR, joint fluid aspiration / culture
Mechanical instability or aseptic loosening	Pain, instability during weight-bearing, limited range of motion	"Start-up" pain, mechanical symptoms, changes on serial imaging	Plain radiography, CT, bone scintigraphy
Deep vein thrombosis	Diffuse swelling, calf pain, skin discoloration	Absence of vasomotor / trophic changes; positive Homan's sign	Duplex ultrasonography
Peripheral neuropathy	Neuropathic pain, sensory disturbances (numbness / tingling)	Symptoms strictly follow a dermatomal or peripheral nerve distribution; no autonomic dysfunction	Electromyography, nerve conduction velocity studies
Psychosomatic disorders	Chronic pain without clear organic cause, emotional distress	Inconsistency between subjective complaints and objective clinical findings	Psychological assessment, multidisciplinary evaluation
Metal hypersensitivity	Chronic pain, localized swelling, persistent dermatitis	Atypical cutaneous reactions near the prosthesis, symptoms refractory to standard analgesics	Patch tests, blood tests for metal levels, tissue histology

The duration of rehabilitation is typically prolonged by 3–6 months after pTKA and by 6–12 months after revision surgery [6,10]. Quality of life, as assessed by the SF-36 scale, is also substantially reduced, with mean scores of 40 after pTKA and 35 after rTKA ($p < 0.01$), indicating a higher degree of disability following revision procedures *(Fig. 3)*.

The treatment of CRPS following TKA is multimodal and comprises the following strategies. Pharmacotherapy includes nonsteroidal anti-inflammatory drugs, gabapentin, pregabalin, and corticosteroids to mitigate pain and inflammation [17]. Physiotherapy is focused on manual lymphatic drainage, mobility exercises, and graded motor imagery [9]. Interventional methods: sympathetic nerve blockade and spinal cord stimulation are reserved for refractory cases [10].

The efficacy of treatment is highly dependent on the timing of intervention. Following pTKA, clinical remission is achieved in approximately 60–80 % of cases within 6–12 months. In contrast, after rTKA, remission rates drop to 50–70 % and require a longer duration of 12–18 months [17] *(Fig. 4)*.

Future research directions and therapeutic innovations. In addition to established clinical protocols, emerging therapeutic modalities and comprehensive investigations into the pathophysiology of CRPS offer significant potential for improving patient outcomes.

Objective inflammatory markers: the quantification of local heat flux and inflammatory cytokine profiles (TNF-α) may serve as objective metrics for assessing vasomotor dysfunction and facilitating the early identification of CRPS predisposition [13]. Preliminary data suggest that elevated preoperative TNF-α levels are associated with a heightened

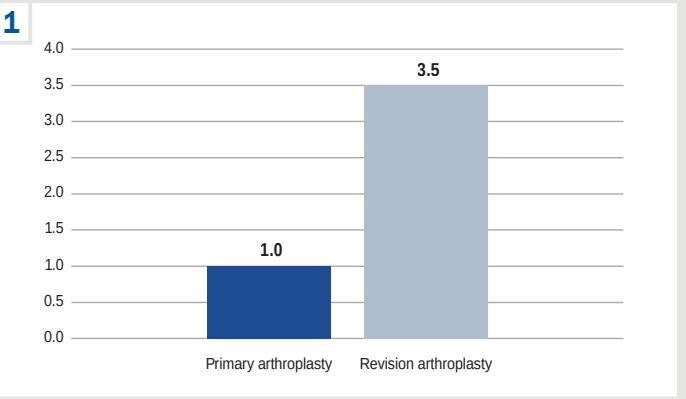

Fig. 1. Comparison of the CRPS incidence after pTKA and rTKA.

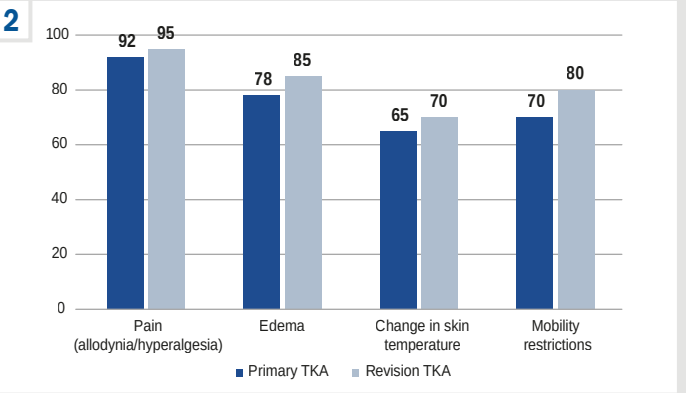

Fig. 2. Frequency of major clinical symptoms of CRPS following TKA.

Table 3. Primary risk factors for CRPS following TKA

Risk factor	Description
Psychological status	Depression, anxiety, and catastrophizing increase central pain sensitization
Preoperative pain	Intense, widespread pain prior to surgery as a predictor of poor outcomes
Sleep disturbances	Impairment of descending pain inhibitory pathways
Metal hypersensitivity	Rare etiology, specifically related to nickel-containing components
Systemic factors	Female sex, smoking, diabetes mellitus, and autoimmune diseases

Table 4. Summary of literature coverage on CRPS following TKA

Study type	Examples	Limitations
Case reports	Individual reports of CRPS following TKA	Limited generalizability
Retrospective series	Analysis of CRPS incidence following revision TKA	Small sample sizes, selection bias
Prospective studies	J. D. Kosy et al.: 100 patients monitored for CRPS	Rarity of the condition, potential for underestimation
Epidemiological forecasts	Projections of TKA volume to 2030	General focus; lacking specific data on CRPS complications

Table 5. Directions for future research in CRPS following TKA

Research direction	Description	Clinical example / Target
Biomarker development	Identification of markers for early CRPS diagnosis	Analysis of preoperative inflammatory cytokine levels (TNF-α)
Prevention assessment	Evaluating surgical techniques and intraoperative constraints	Impact of tourniquet time and minimally invasive approaches on CRPS risk
Neuromodulation	Assessing efficacy of advanced interventions for refractory cases	Spinal Cord Stimulation for chronic, treatment-resistant pain
Long-term outcomes	Longitudinal analysis of CRPS following rTKA	Assessment of quality of life and functional recovery 5 years post-surgery
Protocol standardization	Developing unified diagnostic and therapeutic recommendations	Global implementation of the Budapest criteria in orthopedic oncology and trauma

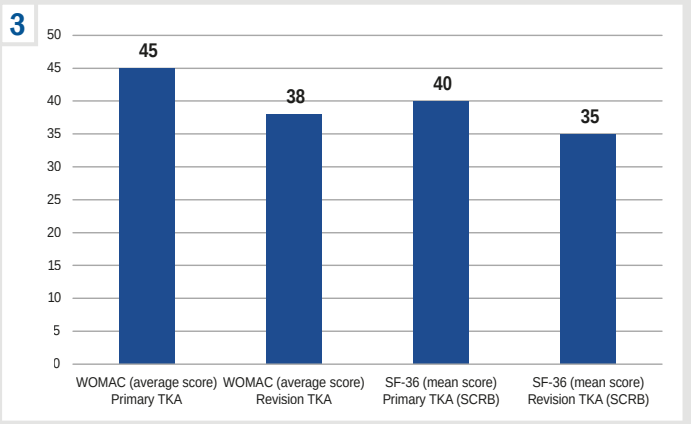

Fig. 3. Comparison of functional outcomes between pTKA and rTKA in patients with CRPS.

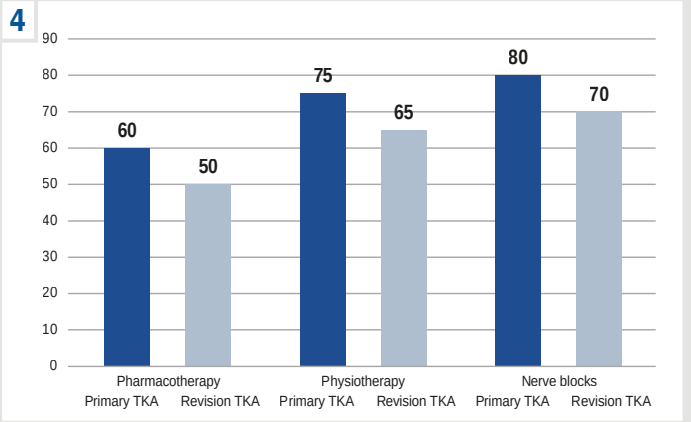

Fig. 4. Efficacy of multimodal treatment across pTKA and rTKA cohorts

risk of developing CRPS [7], though these findings necessitate further large-scale validation.

Innovative diagnostic and rehabilitation technologies: the integration of robotic-assisted systems (LokomatPRO or LokoHelp) allows for the precise quantification of motor impairments, including range of motion, muscle strength, and neuromuscular coordination. Such technologies are critical for the objective evaluation of the motor components of CRPS and for the longitudinal monitoring of rehabilitation efficacy.

Autonomic cardiovascular assessment: autonomic testing provides a valuable adjunct for evaluating patients following TKA. These assessments objectively measure the physiological response to postoperative stress, specifically regarding orthostatic blood pressure regulation and the sympathovagal balance (the interplay between sympathetic and parasympathetic influences).

Novel pharmacological agents: the development of targeted therapies focusing on specific nociceptive pathways, such as N-methyl-D-aspartate (NMDA) receptor antagonists and purinergic receptor blockers, may provide vital therapeutic alternatives for refractory cases of CRPS.

Coverage of the problem in the literature. The current literature on CRPS following TKA primarily comprises case reports, retrospective series, and limited prospective studies. There is a notable scarcity of high-quality, multicenter research, particularly regarding rTKA.

Data on revision TKA are even more limited. Previous studies reported a higher incidence of CRPS (2–5 %) in revision cases but provided limited epidemiological data [7]. This highlights significant gaps in the understanding of the epidemiology, risk factors, and optimal management of CRPS following revision surgery. The literature emphasizes the importance of early intervention; some

studies suggest that timely multimodal treatment, including early physiotherapy, pharmacotherapy, and interventional therapies, can improve the prognosis, yielding outcomes comparable to those in patients without complications [14]. However, comparative studies evaluating the effectiveness of these approaches specifically between pTKA and rTKA are currently lacking.

In summary, the evidence base for CRPS following TKA is weakened by small sample sizes and a reliance on incidental observations. Larger, prospective, multicenter studies are essential to enhance our understanding of this condition, particularly given that the number of rTKA procedures is projected to increase significantly by 2030 [10,13,22] *(Table 4)*.

The need for further research and standardization of approaches. CRPS following TKA is a multifaceted condition warranting further investigation, particularly in the context of rTKA. In these cases, the risk is significantly elevated due to cumulative tissue trauma and extensive scarring. Recent studies emphasize the potential of biomarkers for early diagnosis; for instance, elevated preoperative TNF-α levels have been shown to correlate with a higher risk of developing CRPS [3,10]. Specifically, S. Bruehl et al. (2022) demonstrated that preoperative TNF-α levels serve as predictive markers for CRPS emergence six months post-TKA [10].

Standardization of diagnostic protocols, specifically the Budapest criteria, is essential to minimize misdiagnosis in the postoperative period, when CRPS symptoms frequently overlap with other surgical complications. Similarly, treatment algorithms require rigorous standardization. Current approaches, including multimodal pharmacotherapy, specialized physiotherapy, and interventional techniques, exhibit variable efficacy depending on the timing of initiation. Establishing consistent, evidence-based guidelines is critical to enhancing clinical practice and optimizing patient recovery.

Future research should prioritize the evaluation of preventive strategies, such as the adoption of minimally invasive surgical techniques and the restriction of tourniquet application time to less than 120 minutes [10]. Furthermore, exploring novel therapeutic modalities, such as neuromodulation (spinal cord stimulation), is vital for managing refractory cases. Investigating the long-term outcomes of CRPS following rTKA, where current data remain scarce, is essential for the development of personalized, patient-centric treatment approaches [7] *(Table 5)*.

Discussion

CRPS is a multifaceted condition characterized by neuroinflammation, central sensitization, and autonomic dysfunction [4,5]. Surgical trauma during TKA triggers a localized inflammatory response, releasing pro-inflammatory cytokines such as TNF-α) and Interleukin-1β (IL-1β), which significantly heighten pain sensitivity. Prior investigations have found that central sensitization, evidenced by the increased temporal summation of pain, and persistent inflammatory processes, particularly elevated TNF-α levels, are pivotal contributors to CRPS pathogenesis [3,20]. Furthermore, the risk of developing CRPS is notably higher following rTKA due to the more extensive surgical trauma and repeated tissue injury [13].

Strategies for the CRPS prevention include the employment of minimally invasive surgical techniques, limiting pneumatic tourniquet time to under 120 minutes, and the initiation of early postoperative rehabilitation [6]. Future research must prioritize the development of sensitive diagnostic biomarkers, the rigorous assessment of preventive strategy efficacy, and the exploration of novel therapeutic modalities, such as neuromodulation. Additionally, longitudinal data on the long-term prognosis of CRPS following rTKA are essential to optimize rehabilitation protocols and patient-centered care [7,23].

The diagnosis of CRPS following TKA remains challenging due to the non-specific nature of early symptoms, which frequently overlap with other postoperative complications, including periprosthetic joint infection or prosthetic instability [9]. The Budapest Criteria, which require the presence of disproportionate pain and specific symptoms across multiple clinical categories, remain the gold standard for diagnosis [17]. A thorough exclusion of differential diagnoses is imperative during the postoperative period to avoid diagnostic errors.

The prompt initiation of a multimodal therapeutic regimen is critical for improving clinical outcomes. Evidence suggests that timely interventions, including specialized physiotherapy, targeted pharmacotherapy, and neurostimulation when indicated, can yield functional outcomes comparable to those observed in patients without CRPS [13,24]. Furthermore, psychological factors such as anxiety and depression significantly influence the clinical course of CRPS, particularly after rTKA, where patients may have been predisposed by previous negative surgical outcomes [14].

Conclusions

1. Complex regional pain syndrome is a significant complication following total knee arthroplasty, with a markedly higher incidence after revision procedures (2–5 %) compared to primary ones (0.2–2.0 %). Complex regional pain syndrome negatively impacts surgical outcomes, leading to lower functional scores (WOMAC, SF-36), prolonged rehabilitation (up to 12 months in revision cases), and diminished quality of life. This underscores the necessity for intensified focus on early prevention and proactive management strategies.

2. The diagnosis of complex regional pain syndrome relies on the internationally recognized Budapest Criteria, which necessitate the presence of disproportionate pain and multi-categorical symptoms after the exclusion of alternative etiologies. Given the complexity of postoperative differential diagnosis, there is an urgent need for standardized screening protocols and improved methods for early detection.

3. The management of complex regional pain syndrome must be multimodal and interdisciplinary, encompassing pharmacotherapy, physical therapy, and interventional techniques. Early treatment initiation significantly enhances the probability of clinical remission. Rehabilitation requires a multidisciplinary approach focused on early mobilization and individualized physical activity programs tailored to the specific needs of both primary and revision total knee arthroplasty patients. These strategies are essential to mitigate the risk of pain chronification and to optimize the patient's postoperative quality of life.

Funding
This study received no external funding.

Conflicts of interest: authors have no conflict of interest to declare.
Конфлікт інтересів: відсутній.

Надійшла до редакції / Received: 27.08.2025
Після доопрацювання / Revised: 09.12.2025
Схвалено до друку / Accepted: 15.12.2025

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