

Review Article

Rheumatoid Arthritis and Carpal Tunnel Syndrome: A Scoping Review

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Abstract

Background: Carpal Tunnel Syndrome (CTS) is caused by compression of the median nerve within the carpal tunnel of the wrist. CTS is frequently observed in patients with autoimmune or chronic inflammatory conditions, especially inflammatory joint diseases like rheumatoid arthritis (RA). RA is a chronic inflammatory condition that leads to joint degradation, characterized by pain, stiffness, and swelling in synovial joints.

Objective: CTS and RA are both distinct conditions, but in some cases, CTS may arise from inflammatory processes linked to RA. In this scoping review, we evaluate the core connection between the diseases. An increase in pressure in the carpal tunnel, leading to median nerve compression, can serve as a clinical manifestation of RA.

Key findings: Chronic RA may lead to histopathological changes in tendons of the carpal tunnel, causing inflammation and fluid buildup in the synovium of the tendons. Due to structural and inflammatory changes in the carpal tunnel associated with chronic RA, timely clinical evaluation for diagnosing CTS is essential.

Conclusion: This study emphasizes the significance of identifying RA risk in CTS patients and the necessity for timely referral to rheumatology, reflecting substantial concern. Improving early identification and encouraging ongoing research will be crucial for improved patient outcomes.

Introduction

Carpal Tunnel Syndrome (CTS) is an entrapment neuropathy of the median nerve. It is the most prevalent peripheral nerve compression syndrome observed in clinical settings, representing around 90% of all nerve entrapment conditions. Its occurrence varies from 7 to 16% in the overall population, with a greater frequency noted in females aged 30–60 years. The occurrence in women is 5–6 times greater than in men [2]. Its symptoms include pain, numbness, weakness, and a tingling sensation in the hand or wrist [1]. While CTS can stem from multiple local and systemic factors, it is linked to autoimmune and chronic inflammatory conditions like rheumatoid arthritis (RA).

RA is a chronic inflammatory disease that affects joints in 0.4–1.3% of adults, making it one of the most chronic inflammatory conditions [2]. Its clinical manifestations include pain, stiffness, and swelling in synovial joints. In addition to joints, inflammation associated with RA affects periarticular structures, which may alter the function of tendons and ligaments and increase the possibility of median nerve entrapment [3].

A population-based study showed that CTS is the most common localized entrapment neuropathy diagnosed both before and after the onset of RA. This association is observed in both seropositive and seronegative RA patients [4]. CTS is primarily a compressive neuropathy; however, it can

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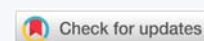
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be a sterile (non-infectious) inflammatory condition with significantly higher levels of inflammatory markers such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF- α) and C-reactive protein (CRP) [5]. Tenosynovitis is an early and prevalent characteristic of RA, based on imaging studies, and it may be a contributor to median nerve compression in CTS [6]. Additionally, in RA, there is an increase in synovial tissue and the development of invasive synovial tissue known as the pannus [7]. Tendon inflammation and synovial proliferation can lead to swelling in the carpal tunnel, which puts more pressure on the median nerve and contributes to CTS.

In RA, persistent synovitis, autoantibodies, and widespread inflammation are frequently observed. Neuropathy in RA can also result from entrapment, vasculitis, and drug toxicity. CTS is the most frequent neurological observation in RA. Agarwal, et al. found that 10.1% of 108 RA patients experienced CTS. In research conducted by Gray and Gottlieb, patients with RA and flexor tendinopathies experience CTS more often than those with RA who do not have flexor tendinopathies (47% vs. 13%). A recent study conducted by Sakthiswary and Singh found no definitive or persuasive evidence connecting laboratory or clinical parameters in RA to the median nerve involvement. The research combined data from eight studies involving patients with RA, showing that 86 out of 1,561 (5.5%) patients with RA were diagnosed with CTS. In contrast, the pooled prevalence of subclinical CTS was found to be 14.0%. The research found that the occurrence of CTS in RA is similar to that in the general population, and there was no relationship between the characteristics of the median nerve and the clinical parameters of the disease. A recent cross-sectional study indicated that 95.9% of the wrists of individuals with RA exhibited CTS. The wide variation in reported prevalence indicates the importance of mapping the available evidence systematically [7].

Numerous studies have investigated the link between rheumatoid arthritis and carpal tunnel syndrome, but the evidence differs regarding prevalence, clinical features, proposed mechanisms, and diagnostic results. The variations in reported results emphasise the necessity to systematically map the existing literature and pinpoint areas where evidence is still scarce or contradictory. Although previous reviews have addressed CTS in the context of rheumatic diseases, this review concentrates specifically on the relationship between RA and CTS, mapping the evidence across various facets of this connection. This scoping review thus seeks to outline the existing evidence regarding the link between RA and CTS, focusing specifically on their epidemiological connection, clinical and pathological characteristics, proposed mechanisms, diagnostic methods, and related factors. By synthesising these findings, the review could clarify the existing evidence landscape and pinpoint priorities for future research.

Method

A scoping review was conducted in September 2026 to map the available literature on the link between rheumatoid arthritis (RA) and carpal tunnel syndrome (CTS). The review sought to identify and summarise existing evidence concerning the epidemiological association, clinical and pathological features, proposed mechanisms, diagnostic findings, and associated factors linking RA with CTS. The review was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) to support transparent and structured reporting of the review methodology.

In September 2026, a search of the literature was conducted using PubMed and Google Scholar. The search strategy used combinations of terms related to rheumatoid arthritis (RA) and carpal tunnel syndrome (CTS), including “rheumatoid arthritis,” “carpal tunnel syndrome,” “systematic review,” “autoimmune disease,” and “initial symptoms of rheumatoid arthritis.” The search aimed to identify existing literature examining the connection between RA and CTS, including epidemiological findings, clinical and pathological features, proposed mechanisms, diagnostic findings, and associated factors. The search was limited to English-language publications published between 2016 and 2025. Articles relevant to the searches were evaluated for inclusion based on their connection to the goals of the scoping review.

Studies were deemed eligible if they examined the relationship between rheumatoid arthritis (RA) and carpal tunnel syndrome (CTS) in adult human populations. Studies investigating CTS, median nerve entrapment, or median neuropathy in individuals with RA were considered relevant. Eligible publications were restricted to English-language publications released from 2016 to 2025. Original observational and clinical studies, such as cohort, cross-sectional, and case-control studies, were considered eligible for inclusion. Relevant review articles were also considered when they contributed to the identification or synthesis of evidence related to the objectives of the scoping review.

Studies were excluded if they involved animals or did not include adult individuals with rheumatoid arthritis (RA). Studies that did not focus on carpal tunnel syndrome (CTS), median nerve entrapment, or median neuropathy were also excluded. Publications outside the specified period of 2016 to 2025 and those published in languages other than English were excluded. Duplicate records were removed, and articles that mentioned RA or CTS only incidentally, without addressing their relationship, were excluded. Articles that did not align with the objectives of the scoping review were similarly excluded.

Records identified through PubMed and Google Scholar were screened according to the predefined eligibility criteria.

Duplicate records were identified and removed before screening. The titles and abstracts of the remaining records were reviewed to identify potentially relevant studies. Potentially eligible articles were assessed at the full-text phase, after which the inclusion and exclusion criteria were applied. Studies that met all predefined eligibility criteria were included in the final synthesis. The screening and selection of studies were performed by a single reviewer.

Data from the included studies were recorded using a standardised extraction method. The extracted information included the author and publication year, country, study design, sample size, characteristics of the rheumatoid arthritis population, methods used to assess carpal tunnel syndrome, diagnostic approaches, key findings, proposed mechanisms, and reported risks or associated factors. The extracted data were organised to facilitate comparison of findings across

Author, Year	Country	Study Design	Sample Size	Population/ RA Characteristics	CTS Assessment	Diagnostic Method	Main Findings	Proposed Mechanism	Risk/ Associated Factors
Wipperman, et al. 2016 [1]	USA	Review	N/A	Not specifically focused on RA	Clinical features of CTS	Clinical examination and electrodiagnostic testing	Describes clinical presentation, diagnosis, and management of CTS	Median nerve compression within the carpal tunnel	Age, obesity, diabetes, pregnancy, repetitive hand use and other recognized CTS factors
Gong, et al. 2024 [2]	China	Bidirectional two-sample Mendelian randomization study	RA GWAS: 14,361 cases/43,923 controls CTS GWAS: 11,208 cases/195,047 controls	Genetic data for RA	Genetically predicted CTS	Mendelian randomization using GWAS data	Evidence of a positive causal effect of RA on CTS; no evidence of reverse causality	Possible causal contribution of RA to CTS	Genetic instruments associated with RA and CTS
Abdullah, et al. 2023 [3]	Sudan	Case-control study	154 RA patients	Patients with RA; wrist/median nerve involvement assessed	Median nerve involvement in RA	Ultrasound and/or electrophysiological assessment	CTS/median nerve involvement was reported in RA patients; 49/154 (31.8%)	RA-related inflammatory and structural changes may affect the median nerve	RA-related wrist involvement and disease characteristics
George, et al. 2024 [4]	USA	Population-based study	1,335 RA; 1,331 non-RA	Incident RA population	CTS before/ on and after RA diagnosis	Population-based clinical-record ascertainment	CTS was more frequent before/ on RA diagnosis and incidence remained increased after RA diagnosis	Possible shared inflammatory/ autoimmune mechanisms	BMI, ESR and RA serostatus were examined
Arshad, et al. 2024 [5]	-	Case-control study	32 CTS; 32 controls	Not specifically focused on RA	CTS patients	Clinical/ electrophysiological assessment	CTS was associated with increased IL-6 and TNF- α levels, supporting sterile inflammation	Sterile inflammation and oxidative stress	Inflammatory and oxidative-stress pathways
Sudol-Szopinska, et al. 2017 [6]	-	Review	N/A	RA patients with musculoskeletal involvement	Not specifically CTS-focused	MRI and ultrasonography	MRI and US demonstrate synovitis, tenosynovitis and structural changes in RA	Synovitis and tenosynovitis can contribute to compression around the carpal tunnel	Joint inflammation, synovitis, tenosynovitis
Birsanu, et al. 2024 [7]	Romania	Review	N/A	Rheumatic disease including RA	CTS in rheumatic disease	Clinical, electrophysiological and imaging approaches	Reviews the association between CTS and rheumatic diseases, including RA	Inflammation, synovitis and tenosynovitis may increase carpal tunnel pressure	Rheumatic disease, inflammation, structural changes
Sevy, et al. 2023 [8]	USA	Narrative/ clinical review	N/A	Not specifically focused on RA	Clinical CTS assessment	Clinical examination and electrodiagnostic testing	Reviews CTS clinical features, diagnosis and management	Median nerve compression at the wrist	Diabetes, hypothyroidism, obesity, pregnancy, repetitive wrist activity and other factors
Butt, et al. 2023 [9]	Pakistan	Cross-sectional study	154 RA patients	Patients with RA; disease severity assessed	CTS among RA patients	Clinical/ electrophysiological assessment	Reported prevalence of CTS in RA and examined its association with disease severity	-	Age, sex, disease duration and RA-related factors were assessed; no significant associations reported for several factors
Chang, et al. 2025 [10]	USA	Retrospective propensity-matched study	896 matched patients	Patients with autoimmune rheumatic diseases; RA subgroup included	CTS requiring carpal tunnel release	Clinical/ electrodiagnostic diagnosis and surgical outcomes	Rheumatic-disease patients undergoing CTR had differences in postoperative outcomes/ complications compared with controls	Chronic inflammatory/ rheumatic disease may influence nerve/tissue healing	Autoimmune rheumatic disease and other clinical factors
Nakamura, et al. 2024 [11]	Japan	Retrospective study	235 newly diagnosed RA patients	Newly diagnosed RA; 11 had concomitant CTS	CTS at initial RA diagnosis	Ultrasonography and clinical/laboratory assessment	11/235 (4.7%) had concomitant CTS; all 11 showed PD-positive tenosynovitis	Tenosynovitis and synovitis around the carpal tunnel may contribute to median nerve compression	Older age, female sex and ACPA-negative status were reported characteristics



studies and to support thematic synthesis of the existing evidence.

The extracted data were narratively synthesised and thematically organised based on the objectives of the scoping review. The findings were categorised into primary themes, including the epidemiology and prevalence of carpal tunnel syndrome in individuals with rheumatoid arthritis, the temporal relationship between rheumatoid arthritis and carpal tunnel syndrome, proposed pathophysiological mechanisms, clinical manifestations, diagnostic approaches, and reported risk or associated factors. Variations and similarities in findings across studies were identified and examined within each theme. Areas where evidence was limited, inconsistent, or conflicting were also identified to highlight gaps in the current literature and priorities for future studies.

Results

A literature search was performed using PubMed and Google Scholar to identify studies investigating the relationship between rheumatoid arthritis (RA) and carpal tunnel syndrome (CTS). The searches identified a total of 158 records, including 107 records from PubMed, 50 records from Google Scholar, and 1 record identified from other sources. Following the removal of 5 duplicate records, 153 records remained for screening. Titles and abstracts were reviewed according to the predefined eligibility criteria, resulting in 116 records being excluded. The remaining 37 articles were assessed at the full-text stage, of which 26 were excluded after full-text evaluation. A total of 11 studies met the eligibility criteria and were included in the final scoping review. The PRISMA-ScR flow diagram illustrates the study selection process.

PRISMA Stage	Working Numbers
Pubmed records identified	107
Google Scholar results returned	50
Records identified from other sources	1
Total records identified	158
Duplicates removed	5
Records screened	153
Records excluded after title/abstract	116
Full-text articles assessed	37
Full-text articles excluded	26
Full-text articles included	11

Across the included studies, carpal tunnel syndrome (CTS) was reported among patients with rheumatoid arthritis (RA), although the reported prevalence varied across study populations and diagnostic approaches. A 2023 study involving 154 patients with RA reported CTS in 49 patients (31.8%). The mean age of the study population was 43.6 ± 13.7 years, and the mean disease duration was 8.3 ± 6.1 years. The relatively high frequency of CTS reported in this population indicates that CTS may represent an important clinical manifestation among individuals with RA [9].

Other studies reported varying frequencies of CTS in RA populations. Agarwal, et al. reported CTS in 10.1% of 108 patients with RA, whereas Gray and Gottlieb reported a higher frequency among RA patients with flexor tendinopathies than among those without flexor tendinopathies (47% versus 13%). In addition, a pooled analysis of eight studies identified CTS in 86 of 1,561 patients with RA (5.5%), while the pooled prevalence of subclinical CTS was 14.0%. These findings demonstrate substantial variation in the reported frequency of CTS among patients with RA [7].

The variation in reported prevalence may reflect differences in study populations, sample sizes, RA characteristics, diagnostic criteria, and methods used to identify CTS. Studies using electrodiagnostic or ultrasound-based assessment may detect median nerve involvement that is not identified through clinical assessment alone. Overall, the available evidence indicates that CTS occurs among individuals with RA, although its reported prevalence varies considerably across studies.

Findings from a population-based study conducted by George et al. indicate that carpal tunnel syndrome (CTS) can occur both before and following the onset of rheumatoid arthritis (RA). Among 1,335 patients with RA, 179 (13%) had experienced CTS at some point before or at the incidence of RA, compared with 85 (6%) individuals without RA (OR 2.30, 95% CI 1.75–3.03). The occurrence of CTS was also increased at least two years before RA diagnosis (OR 1.55, 95% CI 1.14–2.10), indicating that CTS may precede the clinical diagnosis of RA in some individuals. During subsequent follow-up, 154 patients with RA developed CTS, and after adjustment for age, sex, smoking, and obesity, the risk of developing CTS remained higher among patients with RA than among individuals without RA (HR 1.80, 95% CI 1.39–2.31) [4].

Results from a bidirectional Mendelian randomization study conducted by Gong et al. further support an asymmetric relationship between RA and CTS. The inverse-variance weighted analysis demonstrated a positive causal relationship between genetically predicted RA and CTS (OR 1.001, 95% CI 1.001–1.002, *p* = 0.001), whereas the reverse Mendelian randomization analysis found no evidence of a positive causal relationship from CTS to RA. Although these findings do not establish the clinical timing of CTS in relation to RA diagnosis, they support the possibility that RA-related processes contribute to the development of CTS rather than CTS itself causing RA [2].

The development of carpal tunnel syndrome (CTS) in individuals with rheumatoid arthritis (RA) appears to be associated with inflammatory and structural changes within and surrounding the carpal tunnel. RA-related synovitis, synovial expansion, flexor tenosynovitis, and progressive joint and tendon changes can alter the structure of the carpal tunnel and contribute to increased pressure on the median

nerve. Proliferation of synovial tissue and pannus formation may further occupy the limited space within the tunnel, contributing to median nerve compression.

Tendon involvement may represent an important component of this process. In individuals with RA, inflammation and structural damage to the flexor tendons may increase the volume of tissues passing through the carpal tunnel. Filippucci et al. found that around 50% of the 90 patients with RA in their study had at least one inflamed tendon, while tendon damage was also frequently observed. These inflammatory and structural tendon abnormalities may contribute to neurotendinous changes and further compromise the limited space within the carpal tunnel.

When the carpal tunnel becomes narrowed, or its contents increase, compression of the median nerve and its associated microvasculature may occur. Persistent compression may impair intraneural blood flow and axonal transport, resulting in venous congestion, endoneurial oedema, ischemia, and disruption of the blood-nerve barrier. Continued nerve compression may subsequently cause demyelination and, in more severe cases, axonal injury, providing a pathological basis for the sensory and motor manifestations of CTS [5,8].

In addition to inflammatory changes, local anatomical factors may contribute to CTS in individuals with RA. The presence of a persistent median artery or additional muscle bundles, together with RA-related synovial and joint changes, may further reduce the available space within the carpal tunnel. Consequently, CTS in RA is likely to result from an interaction between inflammatory, structural, vascular, and local anatomical factors rather than from a single pathological mechanism.

A significant clinical issue in individuals with rheumatoid arthritis (RA) is the potential overlap between RA-associated hand and wrist symptoms and carpal tunnel syndrome (CTS). RA often involves inflammatory changes in the wrist and flexor tendons, such as flexor tenosynovitis and radiocarpal synovitis, which may occur alongside median nerve compression and contribute to hand and wrist symptoms. Furthermore, persistent or recurrent tenosynovitis and other inflammatory changes associated with RA may contribute to persistent or recurrent median nerve compression [3,10]. Consequently, symptoms such as pain, tingling, numbness, and weakness in patients with RA may reflect CTS, RA-related musculoskeletal involvement, or a combination of both. This overlap may complicate the recognition of concomitant CTS and emphasizes the importance of considering median nerve involvement when sensory symptoms develop in patients with RA.

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CTS was mainly evaluated through clinical assessments focusing on typical symptoms and examination results. Multiple research investigations employed nerve conduction studies (NCS) and electromyography (EMG) to validate median nerve involvement and evaluate severity, especially in atypical cases. These methods could be beneficial in rheumatoid arthritis (RA) since carpal tunnel syndrome (CTS) signs can coincide with RA-related symptoms in the hands and wrists.

Ultrasonography served as a non-invasive diagnostic technique, encompassing evaluation of the median nerve's cross-sectional area and related tendon or synovial issues. Nevertheless, results concerning the median nerve cross-sectional area in RA were variable, with certain studies noting elevated measurements while others detected no notable difference. MRI was not typically recommended for diagnosing CTS [1].

Nakamura, et al. noted that patients presenting with concomitant CTS at the initial RA diagnosis were characterised by older age, female sex, and ACPA negativity. Among the cohort, all 11 patients with concurrent CTS were female, had a mean age of 73 years, and were tested negative for ACPA. Many had mild or absent articular manifestations at presentation, suggesting that CTS may occur during an early or preclinical phase of RA. However, the small number of CTS cases limits the generalizability of these findings [11].

Discussion

This scoping review identified consistent evidence of an association between rheumatoid arthritis (RA) and carpal tunnel syndrome (CTS), although the reported frequency of CTS varied considerably across studies [2,4,7,9]. The variation in prevalence appeared to reflect differences in study populations, RA characteristics, diagnostic approaches, and methods used to identify CTS [7,9]. Importantly, the reviewed evidence indicated that CTS may occur before, around the time of, or after the diagnosis of RA, suggesting that it is not exclusively a complication of established RA [4]. Population-based findings further demonstrated an increased risk of CTS among individuals with RA, while Mendelian randomization evidence supported a possible causal contribution of RA to CTS but did not support the reverse relationship [2,4]. Overall, these findings suggest that the relationship between RA and CTS is multifactorial and may involve processes associated with RA that contribute to median nerve compression and subsequent CTS [2,4,7].

The studies provided consistently demonstrated the presence of CTS among patients with RA, although the

prevalence reported varied considerably across different populations. This variation may be associated with differences in sample characteristics, the duration of RA, clinical features, study design, and the criteria used to diagnose CTS. Diagnostic heterogeneity may also have contributed to these differences, as research employing electrodiagnostic or ultrasonographic techniques may detect median nerve involvement, including subclinical cases that may not be identified through clinical assessment alone. In general, the existing evidence indicates that CTS is a significant manifestation among patients with RA. At the same time, the considerable variation in reported prevalence highlights the influence of population traits and diagnostic methods on its estimated occurrence [7,9].

The development of CTS in individuals with RA appears to be linked to inflammatory and structural alterations within and around the carpal tunnel. Synovitis related to RA, synovial growth, flexor tenosynovitis, and alterations in tendons can increase tissue volume within the tunnel and lead to increased pressure on the median nerve. Persistent compression can subsequently impair intraneural blood circulation and result in oedema, ischemia, demyelination, and, in severe cases, axonal damage [5,8]. Research has also emphasised the significance of tenosynovial inflammation, with RA-associated CTS exhibiting notable synovial alterations surrounding the carpal tunnel, unlike the marked median nerve swelling observed in idiopathic CTS [3,5]. Nevertheless, findings concerning median nerve cross-sectional area in RA have varied, with some studies indicating enlargement and others reporting no significant difference. These results indicate that CTS linked to RA could primarily be caused by adjacent inflammatory and structural alterations instead of solely by median nerve swelling.

Diagnosing CTS in patients with RA can be difficult since the joint symptoms of RA might be subtle or nonexistent when CTS occurs. Nakamura et al. noted that several patients showing concurrent CTS at the onset of RA exhibited mild or absent arthritis symptoms, and 6 of the 11 patients did not meet the 2010 ACR/EULAR classification criteria at presentation. This indicates that CTS might sometimes occur in an early or preclinical stage of RA and could thus be missed when significant joint symptoms are not present. Significantly, ultrasonography revealed synovitis in the carpal tunnel and adjacent small joints in all 11 patients, suggesting that ultrasound could assist in detecting underlying inflammatory alterations in those presenting with CTS. These results underscore the significance of evaluating RA in individuals with CTS, especially when symptoms arise alongside subtle or unusual inflammatory indicators [11].

Recent research has provided additional insights into the relationship between RA and CTS. Nakamura et al. showed that CTS might occur at the initial diagnosis of RA, even when articular symptoms are mild or absent, as 6 of 11 patients did not meet the 2010 ACR/EULAR classification criteria at initial

assessment. This finding reinforces the possibility that CTS may develop during an early or preclinical stage of RA [11]. Imaging findings have also indicated differences between RA-associated and idiopathic CTS. Smerilli, et al. reported that idiopathic CTS was mainly characterized by median nerve swelling associated with persistent compression, whereas RA-associated CTS appeared to involve more rapid median nerve compression related to synovial inflammation[2]. Additionally, George et al. reported that a higher BMI was associated with a greater incidence of CTS after RA diagnosis, whereas the higher incidence observed in seronegative RA compared with seropositive RA did not reach statistical significance [4]. Collectively, these contemporary findings suggest that CTS in RA may represent a distinct and multifactorial process involving early inflammatory changes rather than merely being a consequence of chronic structural joint disease.

Earlier reviews have addressed carpal tunnel syndrome within the framework of rheumatic diseases, including rheumatoid arthritis, yet typically regarded CTS as one manifestation among various rheumatic disorders. In contrast, this scoping review specifically examines the relationship between rheumatoid arthritis and carpal tunnel syndrome, outlining the available evidence across various facets of this relationship. The analysis consolidates evidence regarding the epidemiological link and timing of CTS in relation to RA, clinical and pathological features, proposed inflammatory and structural mechanisms, diagnostic methods, and related factors. It also emphasizes variations in reported prevalence and diagnostic findings among studies and points out areas where the evidence is still insufficient or inconsistent. This review concentrates on the RA-CTS relationship and incorporates findings from epidemiological, clinical, imaging, electrodiagnostic, and mechanistic studies, offering a detailed overview of the current evidence landscape and pinpointing priorities for future research.

Despite increasing evidence on the relationship between RA and CTS, several knowledge gaps remain. Differences in diagnostic criteria and methods may contribute to the variation in reported CTS prevalence, highlighting the need for standardized diagnostic approaches. Longitudinal studies are also needed to clarify the temporal relationship between RA, tenosynovitis, and CTS. In addition, the effects of RA disease activity and treatment on CTS development remain incompletely understood. Further studies using combined ultrasonographic and electrophysiological assessment may help clarify the differences between RA-associated and idiopathic CTS.

This review has several limitations. The literature search was limited to PubMed and Google Scholar, and the broad search structure of Google Scholar may have resulted in inconsistencies in identifying relevant studies. The restriction to English-language publications may have introduced language bias. Moreover, the included studies varied in their

populations, diagnostic methods, and study designs, limiting direct comparison of findings. Publication bias cannot be excluded, and the available evidence was not suitable for quantitative meta-analysis.

Conclusion

The existing literature indicates a clinically relevant association between RA and CTS. CTS can develop before, during, or following the diagnosis of RA, and inflammation along with structural alterations near the carpal tunnel may lead to compression of the median nerve. Nonetheless, variations in diagnostic methods and a lack of extensive longitudinal data complicate the definitive understanding of the temporal relationship and its underlying mechanisms. Standardised diagnostic methods and future studies are required to better understand the connection between RA and CTS, thereby improving their early detection and management.

Declarations

Availability of data and materials: No primary data or database was used in preparing the manuscript. The data used in this study are available from publicly accessible sources, as cited in the manuscript. For detailed access, please refer to the references provided.

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