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FLEXOR TENOSYNOVITIS OF THE WRIST: A COMPREHENSIVE REVIEW OF CURRENT KNOWLEDGE

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ABSTRACT

Introduction and purpose. Flexor tenosynovitis of the wrist is inflammation of the volar (palmar) flexor tendon sheaths, an under-recognized but clinically important cause of volar wrist pain, swelling, and median-nerve symptoms. It spans mechanical, calcific, inflammatory, and infective forms that mimic one another and delay correct treatment. This review consolidates current knowledge on its anatomy, etiology, diagnosis, and management.

Materials and methods. This review synthesized 55 articles, spanning 1940 to 2026, identified through an extensive search of PubMed and limited to English-language publications; no date restriction was applied, so that landmark anatomical descriptions could be retained alongside contemporary practice. Data were synthesized descriptively, organized by pathophysiology, clinical presentation, diagnosis, and management.

Description of the state of knowledge. The condition comprises mechanical (notably flexor carpi radialis), calcific, inflammatory (rheumatoid), and infective (pyogenic, tuberculous, nontuberculous mycobacterial, and fungal) forms that overlap clinically and may compress the median nerve, causing secondary carpal tunnel syndrome. Diagnosis combines clinical examination with radiographs, ultrasound, and magnetic resonance imaging, supplemented by histopathology or culture when the cause is unclear. Management is form-specific, ranging from immobilization and anti-inflammatory drugs for self-limiting calcific disease, through corticosteroid injection, to tenosynovectomy with carpal tunnel release and urgent debridement for pyogenic infection, in which delay worsens outcomes.

Conclusions. Flexor tenosynovitis of the wrist is uncommon, etiologically diverse, and frequently misdiagnosed; early differentiation of its forms is the key to correct treatment. The evidence base rests largely on case reports and retrospective series rather than controlled trials.

KEYWORDS

Tenosynovitis, Wrist, Tendons, Carpal Tunnel Syndrome, Rheumatoid Arthritis

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Introduction

Flexor tenosynovitis of the wrist denotes inflammation of the synovial sheaths that envelop the flexor tendons as they cross the volar (palmar) aspect of the wrist, including the digital flexors within the carpal tunnel and the radial and ulnar bursae [1]. Although individually uncommon, these disorders are a clinically important cause of volar wrist pain, localized swelling, and reduced hand function, and they are frequently misattributed to more common conditions on first presentation [2,3]. Flexor carpi radialis (FCR) tendinitis, for example, is an infrequently recognized but clinically meaningful cause of radial-sided volar wrist pain that is difficult to diagnose and to treat [3], while acute calcific tendinitis of the wrist flexors is repeatedly mistaken for cellulitis or other infections, leading to unnecessary antibiotic treatment [2,4].

The clinical significance of flexor tenosynovitis at the wrist derives from both its anatomical location and its diverse etiology. Because the flexor tendons share the carpal tunnel with the median nerve, synovial thickening and effusion within the flexor sheaths can produce secondary median-nerve compression and a clinical picture of carpal tunnel syndrome [5,6]. The underlying process may be mechanical and stenosing, calcific, inflammatory (as in rheumatoid disease), or infective, and these forms overlap closely in their presentation, so that chronic volar wrist swelling is a recognized diagnostic dilemma, particularly in regions where tuberculosis is endemic and inflammatory and infectious causes must both be considered [7,8]. At its most severe, pyogenic flexor tenosynovitis is among the most serious closed-space infections of the hand and is associated with a high risk of stiffness, contracture, and tendon necrosis [9,10]. Systemic conditions, most notably diabetes mellitus, further raise the burden of flexor-side tendon-sheath disease and complicate its course [11].

Despite this clinical importance, the literature on flexor tenosynovitis of the wrist is scattered across case reports, small surgical series, and disease-specific studies, with little consolidated guidance. The goal of this review is to draw these findings together into a single, accessible overview of the anatomy and

pathophysiology, epidemiology, clinical presentation and diagnosis, and management of flexor tenosynovitis of the wrist, with an emphasis on the practical strategies that distinguish its mechanical, calcific, inflammatory, and infective forms. Based on our PubMed search of English-language publications, we did not identify a prior narrative review consolidating flexor-side wrist tenosynovitis across its full etiologic range; to the best of our knowledge, no such synthesis exists, although individual forms are addressed in disease-specific summaries or in broader hand-and-finger reviews. In drawing these threads together, this review also addresses the wider determinants of the condition relevant to contemporary health systems: the rising musculoskeletal burden of diabetes, work-related mechanical exposure, the socioeconomic gradients in serious hand infection, and the diagnostic technologies, including point-of-care ultrasound and machine-learning image analysis, that are beginning to change how it is detected and managed.

Methodology

This narrative review synthesizes 55 articles identified through an extensive search of the PubMed database and restricted to English-language publications. Search terms combined the concepts of flexor tenosynovitis, tendinitis, and tendinopathy with the anatomical structures of the volar wrist (flexor carpi radialis, flexor carpi ulnaris, carpal tunnel, and flexor tendon sheaths and bursae) and with the major etiologic and management themes (calcific, rheumatoid, pyogenic, and atypical infection; ultrasound and magnetic resonance imaging (MRI); corticosteroid injection and surgical treatment). No date restriction was applied: the search favored contemporary diagnostic and therapeutic practice but deliberately retained landmark anatomical and clinical descriptions, the oldest dating to 1940 and the most recent to 2026, where these remain the primary source for an enduring concept. For the purposes of this review, “wrist-level” disease was taken to encompass the volar carpal region (the carpal tunnel and its flexor tendons, the radial and ulnar bursae, and their proximal extension into the distal forearm, the space of Parona), together with the immediately adjacent distal forearm and proximal palm. Reports were screened for relevance to flexor-side disease in this region; extensor-compartment disorders (such as De Quervain’s and intersection syndrome) and isolated digital trigger finger were excluded. Trigger finger, although it is itself a stenosing flexor tenosynovitis, was excluded because it arises at the digital A1 pulley rather than at the wrist and has its own extensive dedicated literature. Because the flexor sheath–bursa system is anatomically continuous from the distal forearm to the digits, and because wrist-specific data are sparse for some forms (particularly pyogenic infection, which is most often reported at the digital and palmar level), evidence from hand- and finger-level series was included where it informs volar-wrist disease and is identified as such in the text. Consistent with narrative-review methodology, the articles were purposively selected for relevance and representativeness rather than exhaustively retrieved, and the 55 sources represent the final included set; the search was limited to a single database (PubMed) and to English-language publications, and non-indexed and non-English-language literature was not systematically retrieved, which may underrepresent infective forms endemic to regions where such reports are more commonly published in local-language journals (see Conclusions for further discussion). The collected data underwent descriptive synthesis organized by pathophysiology, clinical presentation, diagnosis, and management.

Anatomy and Pathophysiology

The flexor tendons of the wrist are enclosed in synovial sheaths that reduce friction as the tendons glide through fibro-osseous and retinacular tunnels [1]. Fluid accumulation and synovial thickening — the hallmarks of tenosynovitis — are the primary targets of imaging [1]. On the radial side, the FCR runs through a tight fibro-osseous tunnel at the distal wrist, and it is at this confined distal segment, rather than at the proximal myotendinous junction, that FCR tendinitis almost exclusively arises; this distribution points to a mechanical, stenosing mechanism [12]. Within the carpal tunnel, the digital flexor tendons and the radial and ulnar bursae lie immediately adjacent to the median nerve, so that expansion of the flexor synovium readily encroaches on the nerve [5,6].

The synovial anatomy of the volar wrist is more elaborate than that of a simple tendon sheath, and it underlies the characteristic patterns of disease spread. The digital flexor tendons enter the wrist through two large synovial bursae: the radial bursa, which invests the flexor pollicis longus, and the ulnar bursa (the common flexor sheath), which surrounds the digital flexor tendons [13,14]. Cadaveric and imaging studies show that these compartments communicate with one another, and with selected digital sheaths, in a substantial but inconstant proportion of wrists: ultrasound-guided tenography demonstrated that the radial and ulnar bursae communicate in an “hourglass” configuration in all cadaveric specimens; passive dye injection in a

separate cadaveric series confirmed that direct radio-ulnar communication is patent in roughly one-third of wrists, with the lower rate likely reflecting the greater resistance of passive injection to opening potential communicating channels compared with active tenography [14,15]. This continuity explains how infection can spread in the classic “horseshoe” pattern: ascending infection from the ulnar bursa enters the Space of Parona — the potential space of the distal forearm bounded anteriorly by the digital flexor tendons (flexor digitorum superficialis and profundus) and posteriorly by the pronator quadratus — before descending via the radial bursa to the thumb sheath, or vice versa [16]. This pathway also warns the clinician that the routes of spread are variable rather than fixed: in a classic dissection series of 46 hands, the ulnar bursa had no normal communication with the little-finger sheath in a quarter and only a small, valve-like one in more than half [13,15].

The pathophysiology of flexor tenosynovitis is heterogeneous. A mechanical, stenosing process predominates in FCR disease, where repetitive load within the fibro-osseous tunnel produces tendinitis that may progress to partial or complete rupture [3,17]. A calcific form, in which calcium hydroxyapatite crystals are laid down inside the tendon, affects both the FCR and the flexor carpi ulnaris (FCU); it is typically an acute, self-limiting inflammatory condition that presents with sudden localized pain and tenderness, most often over the pisiform when the FCU is involved [2,4,18,19]. This acute, infection-mimicking behavior is shared by hydroxyapatite deposition disease more broadly, which is an easily overlooked differential in the emergency setting, where it is frequently mistaken for infection and prompts unnecessary investigation and treatment [20]. Calcium pyrophosphate deposition (CPPD) disease has also been documented in upper limb tendon sheaths outside the carpal tunnel, where it produces acute tenosynovitis resembling both calcific hydroxyapatite disease and infection [21]; crystal identification is the distinguishing investigation. An inflammatory form occurs in rheumatoid arthritis, in which hypertrophied flexor synovium and fibrinous “rice bodies” may form within the tendon sheaths and bursae; flexor tenosynovitis can occasionally be the sole presenting manifestation of rheumatoid disease, even when serology is negative [7,8]. A further non-infective, non-inflammatory cause is dialysis-related amyloidosis (DRA), in which β 2-microglobulin accumulates in osteoarticular tissues after long-term hemodialysis; DRA produces chronic carpal tunnel syndrome through deposition in the flexor retinaculum and synovium, and flexor tenosynovitis and carpal bone erosion occur more frequently in amyloid than in non-amyloid carpal tunnel syndrome among dialysis patients [22]. Finally, an infective form ranges from acute pyogenic flexor tenosynovitis, a closed-space bacterial infection of the sheath [9,10], to indolent granulomatous infections caused by *Mycobacterium tuberculosis*, nontuberculous mycobacteria, and fungi, which characteristically produce chronic swelling with rice bodies and are frequently mistaken for inflammatory disease [23–26].

A range of host and mechanical factors predispose to flexor tenosynovitis of the wrist. Diabetes mellitus is associated with a spectrum of hand disorders, including flexor tenosynovitis, carpal tunnel syndrome, and limited joint mobility [11]. The mechanism is thought to be a hyperglycemic milieu in which non-enzymatic glycation of collagen, chronic low-grade inflammation, and microvascular dysfunction stiffen and thicken periarticular and tendon-sheath connective tissue, producing the cluster of diabetic hand conditions of which flexor tenosynovitis forms part [27]. Occupational exposures contribute on the mechanical side: repetitive forceful gripping, sustained awkward wrist posture, and hand-arm vibration are recognized work-related risk factors for soft-tissue and tendon disorders of the upper limb, and such exposures are incorporated into the World Health Organization and International Labour Organization joint methodology for estimating the global work-related musculoskeletal burden [28]. Immunocompromise predisposes to atypical mycobacterial and fungal sheath infection [23], while penetrating trauma and animal bites are common antecedents of infective flexor tenosynovitis [25,29]. Flexor tenosynovitis is also a recognized iatrogenic complication of volar locking-plate fixation of distal radius fractures, where prominent or incorrectly positioned hardware irritates the overlying flexor tendons; plate placement distal to the watershed line, a prominent Soong grade, and postoperative plaster immobilization have been identified as risk factors [30].

Specific Clinical Entities

Several named entities recur in the literature on flexor tenosynovitis of the wrist, each with a characteristic presentation; their principal features are summarized in Table 1. FCR tendinitis may be inflammatory or calcific and, when untreated, can progress from tendinitis to partial or complete rupture, which underlies the rupture-based treatment algorithm discussed below [3,12,17]. Mechanical tendinopathy of the FCU at its pisiform insertion is a distinct entity from calcific FCU disease, arising from repetitive overuse strain at the enthesis rather than from crystal deposition. It presents with ulnar-sided wrist pain localized over the pisiform and is encountered in those performing repetitive ulnar-loading activities [31]. High-frequency ultrasonography demonstrates tendon thickening, peritendinous effusion, and hyperemia at the pisiform insertion, with cystic fluid collections from the pisotriquetral joint in overuse-related cases, distinct from the calcific deposits that characterize acute FCU tendinitis [31]. Conservative treatment (relative rest, activity modification, nonsteroidal anti-inflammatory drugs (NSAIDs), and physiotherapy) is the first-line approach; pisiform excision achieves sustained symptomatic relief in refractory cases, with all patients returning to work at a mean follow-up of six years [32]. Calcific tendinitis of the FCU can closely mimic infection; in one reported case a 65-year-old patient was treated for cellulitis with unnecessary antibiotics before the correct diagnosis was made [2,4].

Flexor tenosynovitis within the carpal tunnel may be idiopathic or secondary to systemic disease and is an important cause of secondary median-nerve compression; reported cases include noninfective tenosynovitis in a patient with newly diagnosed type 2 diabetes and tuberculous tenosynovitis presenting as chronic volar swelling with median-nerve symptoms [5,6]. Chronic granulomatous and fungal infections form a distinct, indolent group; described presentations include a tuberculous compound palmar ganglion with three years of progressive swelling and flexor dysfunction, resolved by excision and a twelve-month course of antitubercular therapy; nontuberculous mycobacterial (*Mycobacterium intracellulare*) tenosynovitis in an immunocompromised patient; and post-traumatic *Candida* infection causing bilateral wrist rice-body tenosynovitis with recurrence after incomplete excision [23,25,26]. Tuberculous tenosynovitis is itself protean: a series of four wrist and hand cases reproduced all three of Kanavel's anatomic-clinical forms (serofibrinous, rice-bodied, and caseous synovitis), included a rupture of the flexor carpi radialis, and was uniformly diagnosed late after months of indolent swelling. Histology, culture, and molecular testing are therefore needed in any suspicious case [33]. Nontuberculous disease behaves similarly: in a ten-year, two-center series of 23 hand cases, *M. intracellulare* predominated (70%), several patients had received corticosteroid injections before the correct diagnosis was reached, and the average symptom duration before diagnosis was eight months [34]. At the acute end of the spectrum, pyogenic flexor tenosynovitis is a surgical emergency [9,10]; staging reflects this severity — in a single-unit series of 120 operated hand cases, patients were staged by the Michon–Sokolow staging system (stage I: serous sheath fluid; stage II: purulent fluid with viable tendon; stage III: necrosis of the tendon or pulley system) and showed a stepwise worsening of digital motion, disability, and return-to-work time from stage I to stage III, with delayed surgery, group-A β -hemolytic streptococcal infection, smoking, and diabetes identified as poor prognostic factors [35]. *Staphylococcus aureus* is the most commonly isolated organism, identified in up to 75% of culture-positive cases; methicillin-resistant *S. aureus* (MRSA) accounts for up to 29% of isolates and is an increasingly prevalent pathogen, with *Staphylococcus epidermidis*, β -hemolytic streptococci, and *Pseudomonas aeruginosa* among additional common isolates and *Pasteurella multocida* relevant after animal bites [36]. Because the condition is high-risk yet low-prevalence, it is also a recognized diagnostic trap in emergency practice, where early recognition rests on pattern recognition rather than any single confirmatory test [37].

Table 1. Forms of flexor tenosynovitis of the wrist.

Form	Typical tendon(s)	Mechanism	Hallmark presentation	Key diagnostic	First-line management
Mechanical / Stenosing (FCR)	Flexor carpi radialis (distal fibro-osseous tunnel)	Repetitive load within a tight fibro-osseous tunnel; may progress to rupture	Volar radial wrist pain ± palpable tender lump	MRI (extent of rupture)	Activity modification, NSAIDs; tenosynovectomy or FCR-sacrificing procedure by extent of rupture
Mechanical / Stenosing (FCU)	Flexor carpi ulnaris (pisiform entheses)	Repetitive ulnar-loading overuse; enthesopathy at pisiform insertion	Ulnar-sided wrist pain over pisiform	Ultrasound (tendon thickening, peritendinous effusion, pisotriquetral cyst)	Activity modification, NSAIDs, physiotherapy; pisiform excision for refractory cases
Calcific (hydroxyapatite)	Flexor carpi ulnaris (pisiform), flexor carpi radialis	Calcium hydroxyapatite deposition; acute, self-limiting	Sudden localized pain, erythema, tenderness; mimics cellulitis	Radiograph / ultrasound (calcific focus)	Immobilization + NSAIDs; ultrasound-guided barbotage
Crystal arthropathy (CPPD)	Flexor tendon sheaths and peritendinous structures of the wrist	Calcium pyrophosphate deposition	Acute volar wrist pain, swelling, and tenderness; mimics calcific and infective disease	Radiograph (chondrocalcinosis); synovial fluid crystal analysis	NSAIDs, colchicine; joint aspiration
Inflammatory (Rheumatoid)	Flexor tendon sheaths and bursae	Autoimmune synovial proliferation; rice bodies	Chronic volar swelling ± median-nerve symptoms; may be the sole sign of RA	MRI + histopathology (rheumatoid nodule)	Treat underlying RA; tenosynovectomy ± carpal tunnel release
Infective (Pyogenic)	Any volar flexor sheath	Acute closed-space bacterial infection	Kanavel signs (fusiform swelling, flexed posture, sheath tenderness, pain on passive extension)	Clinical (Kanavel signs); culture	Urgent debridement, drainage, irrigation + antibiotics for established disease; antibiotics alone may suffice if recognized early (operative vs non-operative balance unsettled; see Management)
Infective (Granulomatous / Fungal)	Flexor sheaths and bursae	<i>M. tuberculosis</i> , nontuberculous mycobacteria, fungi	Indolent chronic swelling with rice bodies	MRI + histopathology and culture	Radical excision/synovectomy + prolonged targeted antimicrobials

Much of the evidence for the infective forms, particularly pyogenic disease, derives from digital and hand-level series rather than wrist-specific cohorts and is extrapolated to the wrist with that caveat.

Abbreviations: FCR, flexor carpi radialis; MRI, magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs; RA, rheumatoid arthritis.

Epidemiology

Reliable population-level prevalence figures for flexor tenosynovitis of the wrist are scarce, and much of the literature consists of case reports and single-center retrospective series; this follows from the relative rarity of the individual entities. Calcific tendinitis of the hand and wrist is explicitly described as a rare occurrence that, owing to its unfamiliarity, is often misdiagnosed [2,4], and FCR tendinitis is likewise considered uncommon, with calcific involvement of the proximal tendon at its myotendinous junction being extremely rare [3,12].

The infective form is the best quantified, though these data derive predominantly from hand- and finger-level cohorts rather than wrist-specific series. Pyogenic flexor tenosynovitis is consistently reported to constitute between about 2.5% and 9.4% of all primary hand infections in a systematic review of 24 studies totaling 1108 patients [38], and it accounted for 5.3% of all purulent diseases of the hand in one single-center series [9]. Higher estimates exist: one large series put the figure at around 20% of hand infections, and the spread across reports likely reflects differing denominators (all versus primary hand infections) and referral patterns rather than a true difference in frequency [35]. Surgical cohorts are correspondingly substantial, ranging from 60 to 174 patients in individual series [9,29,35,39,40]. These cohorts skew male and middle-aged, with a mean age of 47.2 years in one series of 174 patients [29] and 46 men among 66 patients in another [39], and they consistently carry a high comorbidity burden, most often end-stage renal disease, peripheral vascular disease, and diabetes [41]. Penetrating trauma and animal bites are common inciting events, the latter accounting for 23.7% of cases in one cohort [29]. A Swedish nationwide registry study confirmed that pyogenic flexor tenosynovitis is uncommon at the population level (approximately 0.04% of a nationwide cohort of approximately 5.7 million residents) and that male sex, older age, and comorbidity are associated with higher infection risk, alongside a socioeconomic gradient in incidence (specifically by occupational qualification) and a higher risk of subsequent opioid use [42].

Several systemic and iatrogenic contexts also shape the epidemiology. Among 702 patients with diabetes mellitus, musculoskeletal disorders of the hand were highly prevalent, affecting 45.9% of the cohort; carpal tunnel syndrome (17.7%) was the most common, followed by Dupuytren's contracture (14.4%) and adhesive capsulitis (13.8%), with flexor tenosynovitis among the recognized manifestations, and their frequency tracked with glycemic control and microvascular complications [11]. An independent cross-sectional study of 539 people with type 2 diabetes similarly found that 31.9% had at least one musculoskeletal disorder; osteoarthritis was the single most common (16.7%), with the diabetes-associated hand conditions of carpal tunnel syndrome and diabetic cheiroarthropathy (each 9.3%) and trigger finger (4.1%) also represented, yet only about a third of patients had ever been counseled on the link between diabetes and musculoskeletal health [43]. The economic stakes of this association are considerable: a one-year cost analysis of surgically treated diabetic hand infections estimated a total cost of roughly USD 24,600 per patient, driven chiefly by inpatient care, which frames timely diagnosis and prevention as a health-economic as well as a clinical priority [44]. Flexor tenosynovitis is also a defined complication of volar locking-plate fixation of distal radius fractures, requiring hardware removal in 3.7% of patients in one case-control study, with a mean time to symptom onset of 19.4 months [30].

Clinical Presentation and Diagnosis

The cardinal features of flexor tenosynovitis of the wrist are volar pain, localized swelling, and tenderness over the affected tendon, although the precise picture depends on the underlying cause and the tendon involved. FCU disease, including its calcific form, typically produces tenderness localized over the pisiform [4,19], whereas FCR tendinitis presents as volar radial wrist pain, sometimes with a palpable, tender lump over the tendon at the level of the distal radius [3,18]. Because the flexor synovium lies within the carpal tunnel, progressive thickening and effusion can compress the median nerve and produce secondary carpal tunnel syndrome, with paresthesia of the radial digits, volar swelling, and, on MRI, volar bowing of the retinaculum with edema of the median nerve, confirmed by nerve conduction studies [5,6]. Although the

median nerve is the classic site of compression, a distended ulnar bursa can less commonly compress the ulnar nerve in Guyon's canal, producing ulnar rather than median symptoms [8].

A central diagnostic challenge is that the different forms resemble one another and other conditions. Acute calcific tendinitis is readily mistaken for cellulitis [2,4], whereas chronic volar wrist swelling poses a recognized differential dilemma encompassing abscess, tuberculous tenosynovitis, tenosynovial giant cell tumor (diffuse type, formerly pigmented villonodular synovitis), volar wrist ganglion, calcium pyrophosphate deposition (CPPD) arthropathy, and inflammatory arthritis, further blurring the distinction from infection [7,8]. The presence of fibrinous "rice bodies" within the sheaths and bursae is characteristic of chronic inflammation but is itself etiologically nonspecific, occurring in tuberculous, nontuberculous mycobacterial, fungal, and rheumatoid disease alike [8,23–26]. For acute pyogenic flexor tenosynovitis, the Kanavel signs (fusiform digital swelling, semiflexed resting posture, tenderness along the tendon sheath, and pain on passive extension) remain the diagnostic foundation, and they are most informative when considered in aggregate; in one 128-patient tertiary surgical series, in which established advanced disease predominates, most patients presented with all four signs simultaneously, with pain on passive extension and a resting flexed posture the most frequent [41]; this is consistent with the upper bound of cross-study estimates, which place the complete tetrad in approximately 22% to 56% of patients at presentation, the lower figure reflecting earlier and less severe cases encountered across broader clinical settings [45]. An incomplete tetrad must not be taken to exclude the diagnosis [45]. Kanavel's signs were, however, originally described for digital-level pyogenic tenosynovitis of the finger flexor sheaths; at the proximal bursal level, where the radial or ulnar bursa is affected, diffuse palmar and volar-wrist swelling with point tenderness over the affected bursa may be the dominant presentation, and the complete tetrad is less reliably elicited in this anatomical context [10,37]. Systemic inflammatory markers are non-specifically elevated in both pyogenic tenosynovitis and cellulitis and have low discriminatory value between the two [10].

Imaging supports and refines the clinical diagnosis. Plain radiographs identify a calcific focus and localize it to the symptomatic tendon; in a series of six patients with acute wrist or hand pain, four calcific deposits proved to be in the FCU at the pisiform [19]. Ultrasound and MRI are the principal modalities for tenosynovitis itself: both detect the fluid accumulation and synovial thickening that define the condition, with ultrasound also permitting dynamic, real-time assessment and detection of hypervascularity, and MRI mapping the extent of soft-tissue inflammation on fluid-sensitive sequences and after the administration of contrast [1]. Advances in high-resolution transducers have made ultrasound a first-choice tool for the tendons of the hand and wrist, and the addition of color or power Doppler is specifically recommended to detect the hyperemia of tenosynovitis in overuse, inflammatory, infective, and post-traumatic conditions [46]. Standardized positioning of the volar wrist facilitates both sonographic evaluation and ultrasound-guided intervention [47]. The increasing portability of ultrasound has extended this capability to the bedside: emergency physicians have been shown to recognize the hypoechoic peritendinous fluid of pyogenic flexor tenosynovitis on point-of-care ultrasound, and the modality, with a previously reported sensitivity of about 94%, can both support an early diagnosis and monitor the response to non-operative treatment, though this figure derives from small case reports and series rather than a validated diagnostic-accuracy study. The reach of bedside ultrasound into the emergency department has thus changed the assessment of a condition traditionally diagnosed on clinical grounds alone [45,48]. In undifferentiated arthritis, MRI-detected tenosynovitis carries prognostic weight, although in a cohort of 91 patients (of whom 31.9% progressed to rheumatoid arthritis — a coincident figure from a separate cohort unrelated to the diabetic series above) the predictive signal was specific to symmetrical metacarpophalangeal involvement, which was independently associated with progression (odds ratio 4.96; specificity 87%, sensitivity 35%), and was not significant for tenosynovitis at the wrist level [49]. Machine-learning analysis of ultrasound radiomics has been proposed to make the sonographic diagnosis of tenosynovitis more objective: in a single proof-of-concept study, not specific to the wrist, an XGBoost model distinguished tenosynovitis at an area under the curve of 0.914 and a sensitivity of 0.881 on the test set [50]. By contrast, in pyogenic flexor tenosynovitis, imaging remains investigational and is not routinely required to establish the diagnosis, which rests primarily on clinical assessment [10]. When the cause is uncertain, histopathology is often decisive: caseating granulomas with Langhans giant cells indicate tuberculous disease, noncaseating granulomas suggest nontuberculous mycobacterial infection, and a rheumatoid nodule confirms rheumatoid tenosynovitis, while microbiological culture identifies pyogenic and fungal organisms [5,7,23–25].

Management

Treatment of flexor tenosynovitis of the wrist is dictated by the underlying form, and ranges from simple immobilization for self-limiting calcific disease to urgent surgical debridement for pyogenic infection. A practical approach escalates from conservative measures to injection and, where indicated, surgery.

Conservative and non-invasive treatment

For acute calcific tendinitis and mild mechanical disease, conservative management is usually sufficient. Short periods of wrist immobilization combined with NSAIDs typically resolve calcific FCU tendinitis within about two weeks [2,4], and calcific tendinitis of the FCR has resolved gradually over several months with hand therapy and NSAIDs, confirmed by radiographic disappearance of the calcific focus [18]. In FCR tendinitis, patients with less than 50% tendon rupture on MRI are appropriately managed conservatively in the first instance, with complete relief and return to activity in a proportion of cases [3,17].

For rheumatoid tenosynovitis, systemic disease-modifying antirheumatic drug (DMARD) therapy is the cornerstone of medical management. Current European Alliance of Associations for Rheumatology (EULAR) recommendations advocate methotrexate as the first-line conventional synthetic DMARD, with biological agents (tumor necrosis factor inhibitors, abatacept, rituximab, or interleukin-6 inhibitors) or targeted synthetic DMARDs (JAK inhibitors) added when initial therapy is insufficient, based on risk stratification and a treat-to-target strategy [51]. Adequate systemic disease control frequently suppresses the synovial proliferation and rice-body formation driving rheumatoid tenosynovitis [51].

Injection therapy

Corticosteroid injection is widely used for hand and wrist soft-tissue pathology that fails initial conservative care. Injections into the hand and wrist are largely considered safe, with significant side effects uncommon, although isolated serious complications such as flexor tendon rupture and necrotizing fasciitis have been reported; in the absence of dedicated guidelines, common practice limits treatment to no more than four injections in a year, spaced at least three months apart, favoring betamethasone or methylprednisolone, on a relatively weak evidence base [52]. Corticosteroid injection is contraindicated when infection cannot be excluded; clinical red flags warranting tissue sampling before injection include constitutional symptoms, immunocompromise, an atypical indolent course, and failure of a prior injection — circumstances in which injection before correct diagnosis has been documented to delay treatment by months [34]. The corticosteroid dose may also influence outcomes, though the relevant evidence comes from hand soft-tissue conditions that are not specific to the wrist flexors, so the dosing principle is only plausibly transferable. In a retrospective comparison of 466 patients (649 injections) treated for carpal tunnel syndrome, trigger finger, and de Quervain tenosynovitis, high-dose (40 mg) triamcinolone outperformed low-dose (5–10 mg) injections, yielding fewer repeat injections (18% vs 35%), less conversion to surgery (12% vs 21%), and longer intervals between repeat injections (12.0 vs 5.6 months), without major complications in either group [53]. For calcific disease, ultrasound-guided barbotage combined with injection provides a simple and effective option, achieving significant immediate and sustained (six-month) relief [19], and standardized volar-wrist positioning aids accurate ultrasound-guided injection [47].

Surgical and invasive treatment

Surgery is reserved for disease that fails conservative measures, for compressive or chronic tenosynovitis, and, urgently, for pyogenic infection. In FCR tendinitis, a treatment algorithm based on the degree of tendon involvement guides the procedure: patients with less than 50% rupture undergo tenosynovectomy with excision of trapezial osteophytes, whereas those with greater than 50% rupture undergo an FCR-sacrificing procedure [3,17]. In a single 16-patient series reported across two companion papers, five were managed conservatively (with complete relief and return to full activity in three), while the 11 surgical patients (four with less than 50% and seven with greater than 50% tendon involvement) all achieved satisfactory functional and clinical outcomes [3,17]. When chronic flexor tenosynovitis compresses the median nerve, carpal tunnel release combined with total tenosynovectomy relieves symptoms and, in tuberculous or rheumatoid disease, simultaneously yields tissue for definitive histopathological diagnosis [5–7].

Pyogenic flexor tenosynovitis is a surgical emergency in established disease, treated with debridement, drainage, and irrigation followed by early rehabilitation [9,10]; the evidence that follows is drawn predominantly from digital and hand-level series rather than wrist-specific cohorts. In a series of 60 patients,

drainage with primary closure sufficed in 61.7%, while 38.3% required repeated debridement [9]. Empiric antibiotic selection should therefore cover staphylococci, with agents active against MRSA guided by local epidemiology and host risk factors, and extended to cover relevant Gram-negative organisms after animal bites or in immunocompromised patients [36]. However, when the infection is caught early and given antibiotics before it advances, cure may be achieved without surgery [10]. The balance between operative and non-operative management is genuinely unsettled: a systematic review of 24 studies (1108 patients) found insufficient evidence to establish the superiority of either approach for length of stay or patient-reported outcomes, and called for the prospective comparisons that the prevalence of the disease warrants [38]. Selected early presentations do well without operation: intravenous antibiotics under close hand-surgeon surveillance achieved outcomes comparable to surgery in a cohort of patients with animal-bite pyogenic flexor tenosynovitis, only one of whom ultimately required an operation [54]. Others argue from large operative experience that any genuine suspicion of established sheath infection should prompt emergency surgical exploration rather than a trial of primary antibiotics [35]. Timing is the dominant prognostic factor: in a multivariate analysis of 66 patients, a longer interval from onset to debridement and polymicrobial infection (odds ratio 7.80, 95% CI 1.56–38.8) were independent predictors of unfavorable outcomes, and preoperative conservative treatment merely delayed definitive surgery [39]. Surgical technique appears less critical than timeliness: among 174 patients, open debridement (121 cases) and minimally invasive approaches (53 cases) yielded comparable outcomes [29]; the two principal techniques — extensile open debridement versus closed-catheter sheath irrigation (CCI), in which the sheath is continuously irrigated through an indwelling catheter without large skin openings — were directly compared in a 47-case retrospective study, with CCI producing fewer complications than open drainage (3 vs 9), though the difference was not statistically significant and no randomized evidence establishes the superiority of either technique [55]. In the same 128-patient series, outcomes did not differ significantly between hand-trained and non-hand-trained orthopedic surgeons (return-to-operating-room rates 26% vs 22%; amputation 5% vs 7%), so treatment should not be delayed for specialist availability [41]. Antibiotic route can likewise be individualized: in 110 patients, postoperative outpatient oral antibiotics shortened hospitalization (0.6 vs 3.6 days) without significantly increasing readmission (10.7% vs 5.7%), repeat surgery, or amputation compared with inpatient intravenous therapy, except where surrounding structures were involved [40]. In granulomatous and fungal tenosynovitis, thorough synovectomy with complete removal of all inflammatory tissue foci and rice bodies, combined with prolonged targeted antimicrobial therapy, is essential, as incomplete excision is the principal cause of recurrence [23–26]. The antimicrobial courses are long and tailored to the organism: roughly six to eight months of post-operative antitubercular chemotherapy achieved cure without recurrence in a wrist and hand tuberculous series [33], while nontuberculous disease required a mean of about ten months of treatment and more than two debridement procedures per patient, frequently complicated by delayed wound healing and drug toxicity [34]. Finally, flexor tenosynovitis arising after volar plate fixation of a distal radius fracture is managed by hardware removal, which should be considered early in high-risk cases [30].

Prognosis and Outcomes

Prognosis varies markedly by form. Calcific tendinitis is benign and self-limiting, resolving with conservative treatment [2,4,18,19], and FCR tendinitis managed by the rupture-based algorithm achieves satisfactory functional outcomes [3,17]. Pyogenic flexor tenosynovitis, by contrast, carries a substantial risk of permanent morbidity, although these outcome data again derive largely from digital and hand-level series: the most frequent residual problems are stiffness and contracture [9,10], tendon necrosis occurred in 13.3% of the 60-patient series [9], and permanent deficits were recorded in roughly 38% of the 66-patient cohort [39]. The severity of these sequelae tracks closely with the stage at presentation: in the 120-case series, total active motion, QuickDASH disability scores, and time to return to work each worsened stepwise from stage I to stage III, the most advanced cases facing up to a year away from work or ending in amputation [35]. Outcomes are worse with delayed debridement and polymicrobial infection [39]; diabetes is associated with the need for further procedures [29], and anaerobic infection and diabetes are linked to amputation [40]; the best functional recovery is seen when tendon viability is preserved [9]. Beyond the local outcome, a diagnosis of pyogenic flexor tenosynovitis markedly increases the risk of subsequent opioid overuse, with an adjusted odds ratio of 13.23 in a nationwide registry cohort [42]. Patients with granulomatous disease generally do well after timely tenosynovectomy and prolonged antimicrobial therapy, with full functional recovery and no recurrence reported when excision is complete [5,24,26], whereas incomplete excision predisposes to relapse even under

targeted therapy [25]. Recovery is nonetheless often incomplete when diagnosis is delayed: in the nontuberculous mycobacterial series most patients retained some restriction of joint motion at final follow-up, and the best outcomes were confined to those with single-digit involvement [34]. In the inflammatory context, MRI-detected tenosynovitis is both a source of morbidity and a prognostic marker in undifferentiated arthritis, predicting progression to rheumatoid arthritis, although in the source cohort this signal was specific to metacarpophalangeal involvement and was not significant at the wrist [49].

Conclusions

Flexor tenosynovitis of the wrist is an uncommon but clinically important and etiologically diverse condition that spans mechanical, calcific, inflammatory, and infective forms. Its principal challenge is diagnostic: the forms closely mimic one another and other disorders, and chronic volar wrist swelling in particular demands that infective, inflammatory, and neoplastic causes all be considered [7,8]. Because the flexor tendons share the carpal tunnel with the median nerve, the condition is also an under-recognized cause of secondary carpal tunnel syndrome [5,6]. Plain radiographs, ultrasound, and MRI, supplemented where necessary by histopathology and culture, allow the forms to be distinguished, which is the prerequisite for correct, form-specific treatment [1,19]. Management ranges from immobilization and NSAIDs for self-limiting calcific disease, through corticosteroid injection, to tenosynovectomy and, most urgently, prompt debridement of pyogenic infection, in which any delay worsens outcomes [3,39,52].

The forms reviewed present along two axes that frame the condition's remaining challenges. On urgency, calcific and pyogenic disease are acute presentations that are both regularly mistaken for cellulitis; the consequence of misdiagnosis is greatest in pyogenic infection, where delayed debridement is the dominant predictor of poor outcome. The chronic forms — rheumatoid, granulomatous, and fungal — share indolent swelling and rice-body formation that is nonspecific without histopathology and culture, requiring a systematic diagnostic approach even when the clinical course appears stable. On evidence quality, pyogenic disease has the largest evidence base, including a systematic review of 24 studies and cohorts of up to 174 patients, yet even that body of evidence was insufficient to establish whether operative or non-operative management is superior [38]; the rarer forms rest almost entirely on case series. Together, these axes converge on a single priority: etiologic differentiation at first contact, backed by prospective comparative data that the field currently lacks.

Several limitations temper these conclusions. As a narrative rather than systematic review, the synthesis drew on a single database (PubMed) and English-language sources only, did not apply a formal risk-of-bias assessment or quantitative pooling, and is therefore subject to selection and language bias. The underlying evidence is itself weak: it rests largely on case reports and small retrospective series rather than controlled trials, and even the largest pooled analysis of pyogenic disease found insufficient evidence to favor operative over non-operative management [38]. Because wrist-specific cohorts are scarce for several forms, some infective and injection data are extrapolated from hand- and finger-level studies; although the flexor sheath system is anatomically continuous, this extrapolation should be made with caution. Prospective multicenter comparisons remain the unmet need [10,29]. The condition's burden extends beyond individual anatomy: it is rising with the prevalence of diabetes and is shaped by occupational exposure and socioeconomic inequality [27,42,44], and diagnostic technologies, including point-of-care ultrasound and emerging machine-learning tools, may support earlier and more equitable identification in these populations [48,50].

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