

# De Quervein's Tenosynovitis - A Systematic Review

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**Abstract:** *De Quervain's Disease (DQD) is a painful condition that affects the tendons that control the movement of the thumb, specifically the abductor pollicis longus and extensor pollicis brevis. Although this condition was discovered many years ago, its exact etiology remains unknown. However, two theories exist, an inflammatory-mediated pathway and the other myxoid degenerative changes. Substantial evidence exists for both theories, thus requiring further studies into the etiology of DQD. Finkelstein's and Eichhoff's tests have been used for physical examination to clinically diagnose DQD. However, these tests have been shown to have low specificity, hence, the emergence of the wrist hyperflexion and abduction of the thumb test [WHAT]. Ultrasonography may become a critical diagnostic tool to identify anatomical variations, reducing the risk of further complications. The management of DQD is typically conservative, with escalation to steroid injections before surgery is indicated. While current research has suggested possible novel approaches for diagnosing and treating DQD, more studies are required to gain greater insights into the effectiveness of these interventions.*

**Keywords:** Eichhoff test, Finklestein test, wrist hyperflexion and abduction of the thumb test (what), extensor pollicis brevis, abductor pollicis longus, De quervain's disease

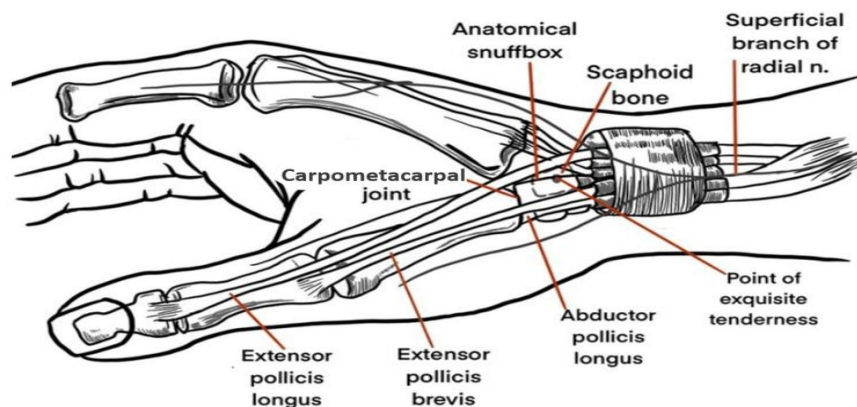
## 1. Introduction

De Quervain's tenosynovitis is a painful condition of the wrist which leads to difficulty in performing daily activities. It is also known as De Quervain's disease (DQD) or "Trigger thumb". It affects the tendons on the lateral side of the wrist. It is caused by inflammation of the tendons that control the movement of the thumb, especially the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) [1]. These tendons run through a narrow tunnel known as the wrist's first extensor compartment as shown in figure 1. It is a fibro-osseous sheath that becomes inflamed and constricted in De Quervain's tenosynovitis [1].

Fritz De Quervain, a swiss surgeon first reported this phenomenon in 1895 [2]. De Quervain's tenosynovitis typically affects adults between 30 and 50 years of age; mostly affecting women than men, especially in those who are pregnant or have recently given birth [3]. People who use repetitive hand or wrist motions in their daily activities such as typing, knitting, gardening, or playing sports such as

golf or tennis [4]-[6] that strain the tendons in the wrist and hand are most commonly affected by DQD [4], [5]. People with certain medical conditions, such as rheumatoid arthritis or diabetes, may also be at a higher risk of developing this condition. However, it can affect anyone who engages in activities that strain the tendons on the thumb side of the wrist [4], [5].

History and clinical examination are sufficient to diagnose the disease [7]. The condition causes pain and tenderness on the lateral side of the wrist and may radiate up the forearm. Patients may also experience swelling and difficulty moving the thumb or grasping objects [2]. Activities involving repetitive hand and wrist movements, such as grasping, twisting, or pinching, can worsen the condition [2]. The treatment methods, including physical therapy, corticosteroid injections, and therapeutic ultrasound, may need to be adjusted to each patient's unique wrist anatomy [8], [9]. This article provides information on the current discussion around the etiology, diagnosis, and various treatment options available for De Quervain's tenosynovitis.



**Figure 1:** Standard arrangement of abductor pollicis longus, extensor pollicis brevis, and point of tenderness in De quervein's tenosynovitis [10]

## 2. Etiology of De Quervain's Disease

The exact etiology of DQD is a subject of investigation. However, two theories exist, an inflammatory-mediated pathway and the other myxoid degenerative changes.

### 2.1 Inflammatory mediated pathway

Inflammatory mediators are molecules released during the inflammatory response and play a crucial role in the pathogenesis of DQD by promoting the accumulation of immune cells to the site of inflammation, increasing blood vessel permeability, and inducing pain [5], [11]. These molecules also cause tissue damage by increasing the production of reactive oxygen species and promoting the activation of matrix metalloproteinases, which are enzymes that degrade the extracellular matrix of tissues [11]-[13]. Classically, researchers have accepted the pathogenesis of DQD to be angiogenic and fibrotic rather than inflammatory. Anti-inflammatory drugs, however, continue to be the universally accepted conservative treatment. [14], [15]. Thus, there is reason to believe that the downstream cascade that results in DQD is mediated by an underlying inflammatory condition. The first to investigate this possibility was Kuo et al. in 2015. They not only found the inflammatory mediators like neutrophil elastase, macrophages, and COX-2 in specimens with DQD, but also found a correlation between the expression of these mediators and the severity of symptoms [11]. In addition, Kuo published a study showing increased expression of the inflammatory mediators interleukin (IL)-20 and tumor necrosis factor-alpha (TNF- $\alpha$ ) in patients with DQD [12].

The inflammatory pathways may also explain the predisposition of DQD for the female sex. Shen et al. revealed that estrogen-B receptors (ER-Bs) (inducers of COX-2 expression) show increased expression in DQD patients, and greater expression is correlated with more severe symptoms [3]. Thus, an estrogen surge may explain the predilection of DQD for women of childbearing age and menopause. With further research, therapeutic plans that target ER-B may reduce these symptoms, particularly in women. Shen et al. also revealed the expression of inflammatory factors IL-1B, IL-6, vascular endothelial growth factor, and Von Willebrand factor, which were also positively correlated with symptom severity [3]. Shen et al. hypothesized that macrophagic invasion into the synovial membrane might induce the production of inflammatory factors, which result in chronic inflammation and angiogenesis [3].

Other findings in support of the inflammatory theory include the presence of IL-1 $\beta$  and IL-6, which are elevated in the tenosynovial tissue of patients with DQD [3], [12]. Additionally, prostaglandins, lipid mediators have been found to be increased in the tenosynovial tissue of patients with De Quervain's tenosynovitis that play a role in inflammation and pain, [3]. Thus, it is undeniable that inflammation plays a crucial role in the pathogenesis of DQD. As future research sheds light on these specific pathways, treatment must adapt to target them accordingly.

## 2.2 Chronic Myxoid Degeneration

Few publications argue that the inflammatory components of DQD are merely superimposed on myxoid degeneration occurring within the tendon sheath and synovium [5], [6]. Clarke et al. examined the tendon sheaths of 23 patients with DQD and observed notable increases in vascularity and mucopolysaccharides compared to controls [6]. Only four of the 23 specimens in this study contained lymphocytes within the tendon sheaths, and no lymphocytes were found within the synovium of any test subjects [15]. Clarke et al. argued that although inflammation may be present in patients with DQD, it is not an etiological factor but rather an overlying process that masks myxoid degeneration [2]. Read and colleagues observed intramural deposits of mucopolysaccharides below the synovium of six women who developed DQD during pregnancy or within 12 months of childbirth [6]. Interestingly, not one specimen in their study showed any signs of inflammation [6]. Clarke et al. stated that the term 'stenosing tenovaginitis' is a misnomer and that de Quervain's disease is a result of intrinsic, degenerative mechanisms rather than extrinsic, inflammatory ones [6].

## 2.3 Other Etiological Factors

Numerous paradigms have been used to study DQD, shedding light on the pathophysiological factors involved. A genome-wide investigation by Kim et al. in 2017 confirmed that a reference SNP cluster on chromosome 8 (rs35360670) is linked with DQD [16]. To our knowledge, this was the first study to demonstrate an association between allelic variation and DQD. Apart from genetic factors, a case study by Yurdakul et al. in 2017 reported a potential link between the administration of somatotropin treatment and the development of the condition. The study involved a 14-year-old girl who presented a persistent DQD after being treated with somatotropin hormone therapy for growth hormone (GH) deficiency [17]. GH and insulin-like growth factor 1 levels are associated with musculotendinous collagen expression [17]. Increased collagen synthesis causes thickening of the flexor tendons and synovial oedema, which may lead to tenosynovitis of the frequently used tendons of the hand [5]. A paper published by Lipscomb in 1951 argued that angulation on the radial side of the wrist is farther in female anatomy and may, therefore, partly explain why women are affected more than men [18].

## 3. Diagnosis

History and clinical examination are commonly used to diagnose the disease. Clinical examination includes Finkelstein and Eichhoff tests [19]. In the Finkelstein test, the examiner holds the patient's thumb firmly with one hand while applying firm traction longitudinally and in the direction of slight ulnar deviation to the wrist with the other hand. In contrast, the Eichhoff test requires the patient to oppose the thumb into the palm and clench the fingers while the examiner passively applies ulnar deviation to the wrist [20]. Finkelstein test has a higher specificity and fewer false positives when compared to the Eichhoff test [19], [20].

A new active diagnostic strategy called the wrist hyperflexion and abduction of the thumb test (WHAT) has emerged. During WHAT, the patient is asked to actively hyperflex the wrist and abduct their thumb as the examiner's index finger provides counter pressure, which will evoke pain if DQD is present [19], [20]. This test identifies DQD exacerbation while minimizing shear between APL/EPB and the bony floor of the first extensor compartment [19], [20]. This test has been proven to be more accurate in diagnosing DQD, in both specificity and sensitivity values when compared to the Eichhoff test [20].

Most studies suggest that patients with septation in the first dorsal extensor compartment of the wrist tend to develop De Quervain's and have post-treatment complications [21]. An excellent diagnostic tool for identifying this septation before treatment is ultrasonography [22]. This method could be beneficial in diagnosing De Quervain's and identifying the possible anatomy of the patient's wrist before executing the treatment plan to reduce the risk of postoperative complications. A septum will usually present as a hypoechoic area in ultrasonography. Other lesions, such as intratendinous degeneration, synovial proliferation, or fluid, could also be perceived as a hypoechoic area and should be differentiated by the physician [21], [22]. In general, ultrasonography could be the key to identifying anatomic variations, which may help in reducing the incidence of post-treatment complications and symptom regression [21].

#### 4. Treatment and Rehabilitation Strategies

Once a physical examination concludes with a newly diagnosed DQD patient, the treatment steps are divided between a multitude of non-surgical approaches and the last-resort surgical approach if symptoms fail to resolve. Current non-surgical methods commonly include rest, massage, cold and heat application, diathermy, thumb spica splints to immobilize the irritated tendons, bracing, physical therapy, non-steroidal anti-inflammatory drug (NSAID) prescriptions, and local corticosteroids injections to reduce the inflammatory swelling and irritation of the APL/EPB tendons. Although these non-surgical approaches relieve immediate pain, there remains a considerable incidence of pain recurrence. It was initially understood that corticosteroid injections alone had almost a six times greater cure rate than splints alone [23], [24]. Later studies exploring the efficacy of comparing individual versus combined non-surgical approaches further illustrate that multimodal treatment plans of hand therapy with corticosteroid injection minimally reduce visual analog scale pain scores more than using the steroid injection method exclusively [9], [25].

Steroid injections directly proximal to the radial styloid process remain the treatment of choice for newly diagnosed DQD patients [14]. Initial treatment of DQD should be with a local injection of long acting corticosteroid derivative. The patients should be informed about the local adverse reactions such as pain and fat atrophy that are minor and most often self-limited. Initial corticosteroid injections have proven a cure rate ranging from 62% to 100% with the failure-to-cure associated with a present APL/EPB septum or specific mechanical triggering of the first dorsal compartment [24],

[26]. A second injection is usually administered for patients with pain recurrence two weeks after the first injection. If pain remains two weeks later, then it is expected that a third injection would be ineffective to reduce the symptoms, therefore, requiring a surgical approach [26], [27]. Efficacy of the intra-sheath injection of triamcinolone acetonide (TC), which is a long-acting and lyophobic steroid has been mentioned in few reports for patients with snapping fingers [28], but very few reports describe the clinical outcomes with the same in de Quervain's disease. A study conducted by Orlandi et al. concluded that Hyaluronic acid addition to ultrasound steroids injection may improve outcome and reduce the recurrence of DQD [29].

Another non-surgical physical agent that can be utilized is therapeutic ultrasound. It is a rehabilitative modality used for different musculoskeletal injuries to enhance tissue extensibility, reduce pain, and improve healing in wounds, tendons, and bones [30]. It is based on high-frequency sound waves at varying parameters depending on the presenting condition and treatment goals [27]-[31]. A 3 MHz frequency is applied for superficial structures and is commonly used for DQD [30]. Once swelling and pain have been treated, therapeutic exercises can be incorporated. This approach is based on performing different range of motion (ROM) exercises that enhance the gliding of the APL and EPB tendons in the first dorsal compartment, starting with isometrics and then completing ROM against gravity [31], [32]. However, ultrasound has been found to be contraindicated for patients with acute inflammation or surgical tendon repairs within the last six weeks [33].

Another mechanism for rehabilitation is therapeutic Kinesio taping (KT). It focuses on positioning Kinesio tapes to release interstitial pressure and reduce inflammation [34]. To achieve a diminished contraction, KT must be applied from the insertion of the muscle to its origin [34]. It is thought to enable decompression of subcutaneous nociceptors, thereby reducing pain. [34].

A surgical release of the first extensor compartment is required for extreme cases of DQD patients failing to resolve symptoms within six months of corticosteroid injections or other non-surgical treatments. When accessing the first dorsal compartment, it is crucial to longitudinally incise the EPB subcompartments (the tendon most likely needing decompression) and the septum dividing APL/EPB tendons if present [35]. Failure to properly incise the septum will ultimately result in failed decompression and refractory DQD symptoms. Another complication during the surgical release is EPB tendon subluxation which can be prevented by avoiding complete excision of the EPB tendon sheath [27], [30]. After the procedure, postsurgical intervention recommends a 1-2-week thumb spica splint followed by weeks of active range-of-motion exercises, scar/edema management, and strengthening exercises [9]. It is in interest of patient to use non-surgical modes before going for surgical release. Higher costs and complications limit the use of surgical procedures. [36].

#### 4.1 Modern Conservative Treatment Approaches

Unique novel therapeutic methods for tenosynovitis that can assist and even accelerate healing along the treatment plan for DQD patients, such as ultrasound, phonophoresis, iontophoresis, and the Graston technique have been summarized. Phonophoresis/sonophoresis uses ultrasound to direct topical anti-inflammatory medications deeper into tissues. In contrast, iontophoresis uses an electrical current/gradient to deliver anti-inflammatory medication to shallow regions of the hands/feet to reduce oedema, inflammation, scar tissue, and pain [33], [37]. These techniques are commonly used for patients with hyperhidrosis but have also been helpful for chronic overuse tendinopathies and stenosing tenosynovitis such as DQD. It is also unclear if these methods of administration properly reach the desired tissues before being diluted by microvasculature. The Graston technique aims to cause controlled microtrauma to the desired soft tissue to augment mobilization and regeneration by following the principles of Wolf's Law, referring to tissue remodelling per the stress placed on it [23], [33]-[38].

These new treatment approaches reduce the time necessary for a patient to recover from persistent pain and regain strength. Additional research needs to be conducted on the effectiveness and relative value to existing surgical and non-surgical treatment modalities.

#### 5. Conclusion

There exists a lot of debates regarding the etiopathogenesis, diagnosis and treatment of dequervein's tenosynovitis. Conservatively, DQD is managed using oral NSAIDs, including physical therapy, splinting, therapeutic ultrasound approaches, and microtrauma assisted-healing techniques before starting corticosteroid injections or even surgery when indicated. This review has demonstrated that ultrasonography may be advised in order to reduce the risk of surgical complications and provide a clearer understanding of how anatomical variation may interact with the pathological factors that cause this condition. A combination of ultrasound steroid injections with hyaluronic acid improves the outcome and prevents recurrence of DQD. Overall, more research is required to elucidate and expand on our present understanding of DQD.

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