Title: A Contemporary Overview of De Quervain's Disease

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ABSTRACT

De Quervain's Disease (DQD) is the debilitating stenosing tenosynovitis of the first radial dorsal compartment of the wrist. Anatomical variation within this compartment is commonly exhibited in those afflicted. Various modalities have been postulated to elucidate the microscopic processes which bring about DQD. Evidence for inflammatory and degenerative changes have both been brought forth. Clinical tests have been useful in establishing a diagnosis however they have low specificity and commonly present with false positive results. Ultrasound and other imaging studies are forthcoming tests for diagnosing DQD with enhanced accuracy. The treatment for DQD is escalatory in nature, beginning with simple conservative measures, definitive treatment is first compartment release surgery. There are a few novel therapies which have emerged in the modern epoch that offer promising results for those who suffer from this condition.

KEYWORDS

De Quervain's Disease, Stenosing Tenosynovitis, Abductor Pollicis Longus, Extensor Pollicis Brevis, First Dorsal Compartment, Rehabilitation, Finklestein, Eichnecoff, Radial First Compartment,

ABBREVIATIONS

APL:	Abductor Pollicis Longus
COX-2:	Prostaglandin-Endoperoxide Synthase 2
DQD:	De Quervain's Disease
EPB:	Extensor Pollicis Brevis
ER-B:	Estrogen Receptor-β
LCM:	CarpoMetacarpal Ligament
LCCR:	Radial Collateral Carpal Ligament
LST:	Scaphotrapezial Ligament
NSAID:	Non-Steroidal Anti-Inflammatory Drugs
WHAT:	Wrist Hyperflexion and Extension of the Thumb

Introduction:

The extensor muscles of the forearm, responsible for extension of the wrist and thumb, emanate from the lateral epicondyle of the humerus. They traverse through one of six compartments in the dorsal aspect of the wrist while encased within a fibro-osseous sheath (1). Stenosing tenosynovitis of the Abductor Pollicis Longus (APL) and Extensor Pollicis Brevis (EPB) occurs within the first compartment towards the radial aspect of the wrist (2). This phenomenon was first reported by Fritz De Quervain in 1895, and has since then been termed *"De Quervain's Disease" (DQD)(3)*. De Quervain theorized that this condition was a consequence of the repetitive strain that would occur amongst common laborers who bore jobs onerous on the wrist (i.e. assembly workers) (3). The preliminary literature suggests that DQD results from myxoid degeneration rather than an underlying inflammatory process (4). On the contrary, present-day investigation places considerable emphasis on the inflammatory markers that may cause a predisposition to DQD (5),(6),(7). Beyond inflammatory predisposition, other risk factors have recently been brought to notice, including somatotropin exposure and genetic propensity (8). Anatomical variations of the first dorsal extensor compartment have been identified in numerous trials and it has been shown that these variations influence treatment outcome (9). As a result of the high anatomical variation, different therapeutic regimens have shown inconsistent rates of success (10). The methods of healing which include physical therapy, corticosteroid injections and therapeutic ultrasound may need to be tailored specifically to each patient's unique wrist anatomy (11). Paralleling the effectiveness of these therapies in extensive patient populations has provided insight to both the pathogenesis and ever evolving approach to treating DQD (12). We evaluate the present literature not only to provide a contemporary perspective about DQD but also elucidate the ongoing debate towards its etiology, anatomy and treatment.

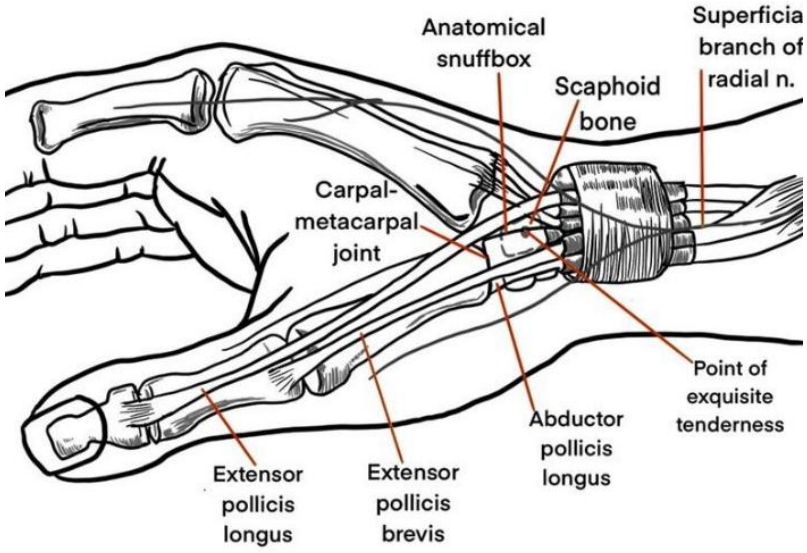

Figure 1- Both the Extensor pollicis brevis and abductor pollicis longus are depicted here. They Emanate from the lateral epicondyle and traverse through the first compartment of the dorsoa-lateral aspect of the wrist.

Methods

Several procedures were conducted to ensure a well-performed review of the current literature on DQD was accomplished. First our data was obtained from archives of biomedical and life-science literature consisting of PubMed and ResearchGate. The scholarly research search engine Google Scholar was also used to find topic specific peer reviewed journals through using keyword searches. We used a variety of keywords including: “DeQuervain tenosynovitis”,” APL and EPB tendon slips”, “1st dorsal compartment septation”, “Anatomical variations”, “First dorsal extensor compartment”, “accessory tendons of APL and EPB”, “preoperative ultrasonography of DQD patients”, “intracompartmental septum”, “Inflammation and De Quervain's Disease”, “DQD and manufacturing”, “De Quervain's Disease and pregnancy”, “Myxoid degeneration and DQD”, “Somatotropin treatment and effects”, “genetics and De quervain's tenosynovitis”, “Inflammatory mediators of DQD”, “Markers of DQD”, “De Quervain’s tenosynovitis Diagnosis”, “DQD and Corticosteroid injections”, “Surgical Release of the 1st dorsal compartment”, “Non-surgical treatment for DQD patients”, “Surgical treatment for DQD patients”, “Ultrasound and DQD”, “Iontophoresis”, “Phonophoresis”, and “Graston Technique”. This Search process uncovered 44-peer reviewed articles published from 1951-2019.

Anatomical Variations of the First Dorsal Compartment (Clinical Significance)

De Quervain's Disease (DQD) results from stenosing tenosynovitis of the first radio dorsal compartment of the hand which contains the APL and EPB tendons. The complex intrinsic musculature of the human thumb is evolutionarily novel and thus the anatomy of the first dorsal compartment is quite diverse from person-to-person (insert study that supports this). The impact of anatomical variation on the likelihood of developing DQD is a fundamental point of interest and may explain why some individuals are more susceptible to developing this condition. Here we summarize the relationship between anatomical variation and the propensity for developing DQD.

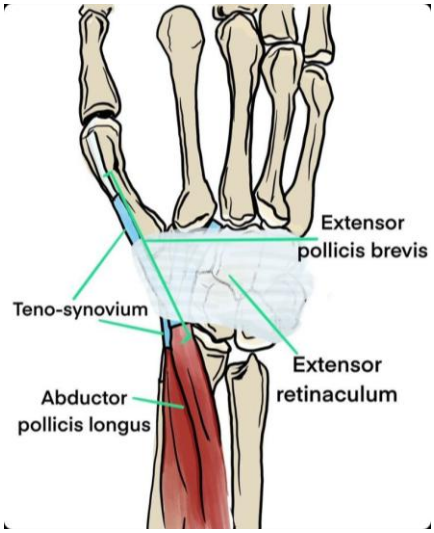

Figure 2- The normal type anatomy of the first radio dorsal compartment contains 1 APL tendon and 1 EBV tendon. Deviation from this typical structure is commonly seen in DQD.

The standard arrangement of the first dorsal extensor compartment is comprised of one APL tendon inserted into the base of the first metacarpal bone and one EPB tendon inserted into the proximal phalanx, both traverse through the first fibro-osseous compartment of wrist dorsum. (25) Investigation conducted on patients with DQD commonly reveals anatomical variation which differs from the standard anatomical model. It is likely that these patterns increase the likelihood for developing this condition.

One prevalent anatomical variant seen in many DQD patients is the presence of complete or incomplete septi within the first dorsal compartment. This septum is a fibrous tunnel which extends through the compartment and divides the APL and EPB tendon slips into different subcompartments at the level of the radial styloid. (26, 28) The subcompartmentilization of the EPB and APL not only compresses the local structures of the wrist dorsum causing inflammation but commonly results in inadequate decompression during surgical treatment, leading to the eventual relapse of symptomatology (25,27).

Other APL and EPB findings across multiple studies illustrate the number of tendon slips in different patients and cadavers. APL tendons range from 1 to 6 tendon slips in most studies. Usually only 2 slips are seen in cadavers, although recent studies have shown that a single APL tendon slip is more commonly seen in DQD patients intraoperatively. (28,29) On the other hand, EPB tendons are commonly described as one single tendon slip in the majority of cases. Absence of an EPB tendon has also been illustrated in some patients, and very rarely have there been cases identified with multiple EPB slips. The importance of these tendon slip anatomical variations has been theorized by multiple studies that different insertion sites result in different gliding resistances or friction coefficients which potentially predispose certain individuals to stenosing tenosynovitis

of the 1st dorsal compartment (30, 31). The most common anatomical combination of tendon slips between normal cadavers and DQD patients includes 1 EPB tendon compartment (with potential accessory slips) and 2 APL tendon slips (28). However, amongst the compartments analyzed, only the DQD patients presented more consistently with 1 APL and 1 EPB tendon slip separated into subcompartments by a septum.

Etiology of De Quervain's Disease

An Overture:

The exact etiology of DQD is a subject of investigation; many prevailing theories have arisen since De Quervain first postulated its origin in his preliminary paper (3). He speculated that DQD is an occupational disease which was augmented through industrial expansion and thus increased demand for repetitive motions of the wrist to complete assembly production (3). A cross-sectional study conducted in 2011 provides support for this theory, when 3710 factory workers from France were examined between 2002-2005, logistic regression modeling revealed that work involving pronation and supination of the wrist (ie screwdriving) was associated with DQD (19). Although the original postulation by De Quervain remains popular, its merit has been called into question by several classic publications. A quintessential paper by Thomas et al. looking at peritendinitis in 544 Luton Car Plant workers between the 1940s-1950s disclosed that there were only 2 recorded cases (from 544) of DQD which was not any more prevalent than in the common population (15). Furthermore, if labor related microtrauma was the chief culprit behind DQD, the disease would be more prevalent in men who predominated the assembly lines, yet DQD is three times more common in women, particularly women of child bearing age (16). What's more, if DQD is truly labor related, it should show proclivity for the wrist of the dominant hand, yet this has been shown to be false in several studies (16)(21). It would seem then, at best, work related torsion is merely a catalyst for eliciting DQD symptoms. It may well be the case that rather than inflicting DQD, the industrial expansion elicited flares of existing cases that were previously inconspicuous. Several explanations for the underlying cause of DQD have been put forth since its discovery. We will review and discuss the prominent etiological theories in this section.

Inflammation:

Classically, researchers have accepted the pathogenesis of DQD to be angiogenic and fibrotic rather than inflammatory in nature. Be that as it may, anti-inflammatory medication remains the universally accepted conservative treatment (15). Thus there are grounds for suspecting an underlying inflammatory process mediating the downstream cascade that leads to DQD. Kuo and

Colleagues were the first to explore this avenue in 2015. Not only did they find inflammatory mediators (Neutrophil elastase, Macrophages and COX-2) to be present in specimens with DQD, they also found that as the severity of the symptoms increased so did the expression of these mediators (5). The inflammatory pathways may also explain DQD's propensity for the female sex. Shen et al. revealed that estrogen-B Receptors (Inducers of COX-2 expression) are expressed with greater magnitude in DQD patients, and greater expression is correlated with harsher symptomatology (7). Thus, an estrogen surge may explain DQD's predilection for women of child bearing age and menopause. With further research, therapeutic plans that target ER-B may ameliorate symptomatology, particularly in women. If DQD's pathogenesis is truly inflammation-associated, then the expression of interleukins which mediate inflammatory processes would be amplified in DQD specimens. This has been shown to be the case, as IL-20 mRNA (a chemotaxis mediator in the inflammatory pathway) is not only increased in the tenocytes of patients with DQD but is also positively correlated with the severity of symptoms (6). In consequence, it cannot be denied that inflammation plays a critical role in the pathogenesis of DQD and as future research begins to shed light on these specific pathways, treatment must adapt to target them in accordance.

Chronic Myxoid Degeneration:

The presence of inflammatory activity within DQD specimens has been demonstrated in multitudinous studies. With so much evidence, It is enticing to assume that inflammation within the synovium and tendon sheath gives rise to DQD and not further inquire about its etiology. However, there is a stream of research that suggests inflammation may just be a confounding variable that cloaks the true pathological nature of this condition. It has been argued by several classic publications that the inflammatory components of DQD are merely superimposed on myxoid degeneration occurring within the tendon sheath and synovium. Clarke et al, Examined the tendon sheaths of 23 patients with DQD and observed notable increases in both vascularity and mucopolysaccharides compared to controls (20). Furthermore, only 4 of the 23 specimens in his study contained lymphocytes within the tendon sheaths, and no lymphocytes were found within the synovium of any test subjects (20). Clarke argued that although inflammation may be present in patients with DQD, it is not an etiological factor but rather an overlaying process that masks myxoid degeneration (20). Read and colleagues observed intramural deposits of mucopolysaccharides below the synovium of 6 women who developed DQD during pregnancy or within 12 months of child birth (21). It is interesting to note that not one specimen in Read's study showed any signs of inflammation (21).

Other Etiological factors:

DQD has been investigated from multifarious paradigms, which provide insight into the factors that may contribute to its pathogenesis. A genome-wide investigation conducted by Kim et al. 2017 confirmed that a reference SNP cluster located on chromosome 8 (rs35360670) is linked with DQD ($p=1.9\times10-8$; OR =1.46; 95%CI = 1.38–1.59) (17). This was the first study, to our knowledge, to demonstrate an association between allelic variation and DQD. In addition to genetic predisposition, a case report of a 14-year-old girl presented by Yurdakul et al. 2017 revealed a possible association between the use of somatotropin treatment as a cause of pathogenesis. The pediatric patient presented a persistent DQD after being treated with somatotropin hormone therapy for Growth Hormone (GH) deficiency (22). GH and IGF-1 levels are associated with musculotendinous collagen expression (22). Increased collagen synthesis causes thickening of the flexor tendons and synovial edema, which may lead to tenosynovitis of the frequently used tendons of the hand (23). 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, in part, explain why women are affected more than men (18).

Pathology

Histopathology- What do the slides say

In Clarke's classic study, the tendon sheaths of DQD patients were shown to be 5x thicker than in controls with considerable dense mucinous deposition within the synovium. Exaggerated vascularity within the central portion of the fibrous sheath was also demonstrated. Of note only 4 of the 23 patients in this study presented with lymphocytic infiltration in the tendon sheath. Interestingly, no histological support for an active inflammatory process underlying DQD was demonstrated in this study (20). Hooper and colleagues examined the histological appearance of fibrous tendon sheaths in postpartum patients with DQD (4). Just like in Clarke's study, histological examination revealed striking amounts of mucinous and myxoid degeneration within the synovium (4). The tenosynovial regions of the specimens revealed marked accumulation of mucopolysaccharides. Similarly to the results found in Clarke's study, the central portion of the fibrous sheath also demonstrated mild angiogenesis (4). Hooper's results also did not show much evidence for an inflammatory mediated pathway. In the same way as Clarke and Hooper, Kuo and colleagues found intense thickening of the fibrous tendon sheath with varying degradation of collagen structure and angiogenesis (5). Unlike Clarke and Hooper however, Kuo performed

immunochemical staining which revealed increased expression of inflammatory markers including neutrophil elastase, macrophages, MAC387 and COX-2 positive cells (5). Statistical analysis revealed that the expression of these inflammatory markers was positively correlated with severity of symptoms (Except MAC387 which was maximally expressed in moderate cases of DQD) (5). Kuo went on to publish another study that showed an increased expression of the inflammatory mediators IL-20 and TNF-a in patients with DQD (6). In the same year Kuo published his results on IL-20, Shen published a similar study underlying the expression of ER-B in DQD patients (7). The overall expression of this receptor was shown to be positively correlated with symptom severity (7). Furthermore, Shen's study indicated that the expression of inflammatory factors IL-1B, IL-6, COX-2, VEGF, and vWF are positively correlated with symptom severity (7). Thus Shen' study suggests that macrophagic invasion into the synovial membrane may induce the generation of inflammatory factors which give rise to chronic inflammation and angiogenesis (7).

Diagnostics

The Finkelstein and Eichhoff maneuvers are the most used clinical tests for diagnosing DQD. To perform the Finkelstein test, the examiner grasps the thumb firmly with one hand, while the other holds the forearm on the ulnar side in a resting position in neutral supination. A firm traction is then applied on the patient's thumb, pulling it longitudinally and in the direction of slight ulnar deviation to the wrist. When performing the Eichhoff test, the patient is asked to oppose the thumb into the palm and then clench the fingers over the thumb. Ulnar deviation is applied passively to the wrist with one hand while the examiner's other hand holds the forearm in the same way as for the Finkelstein test (24). Despite being effective, arguments have always arisen due to false-positive results and discomfort during the examination. Such findings are ascribed to the fact that they are passive tests that have the disadvantage of stressing different structures that are not directly involved in the pathology of DQD. Due to the controversy generated, a new active diagnostic strategy called the wrist hyperflexion and abduction of the thumb test (WHAT) have emerged. During WHAT, the patient is asked to hyperflex the wrist actively and put their thumb actively into abduction while the examiner uses his index finger to counter the maneuver, which will cause pain if there are actual de Quervain's problems with the APL and EPB (24). The mechanism of the WHAT test minimizes the shear between APL/EPB and the bony floor of the first extensor compartment, and since the patient is performing this test, they are effectively controlling the tensioning of the LCCR, the LST, and LCM (24).

Most studies suggest that patients with septation in the first dorsal extensor compartment of the wrist have a tendency to develop De Quervain's and to have post-treatment complications. (32) This is due to the fact that a septum has been identified more often than not in patients with DQD than in cadavers. An excellent diagnostic tool that has been recently published by some authors in respect to locating septation before giving treatment of DQD is ultrasonography of the wrist. Some

studies have focused on proving the efficacy of ultrasonography in detecting both APL and EPB tendons, their sizes, and the presence or absence of a septum in between them. In Nagaoka's clinical study, preoperative ultrasonography was done on 32 wrists of patients with DQD and it successfully identified septation in 25 of them before their surgeries. (33) This method could be very useful both in diagnosing De Quervain's and to identify the possible anatomy of the patient's wrist before executing the treatment plan to greatly reduce the risk of postoperative complications. Some limitations to this tool do exist. First, the physician has to be aware that 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 as well. (32) Overall, ultrasonography could be the key to decreasing the incidence of post treatment complications and regression of symptoms.

Treatment:

Current DQD treatment & rehabilitation methods

Once a physical exam concludes with a newly diagnosed DQD patient, the next steps for treatment are divided between a multitude of non-surgical approaches and the last-resort surgical approach if symptoms fail to subside. Current non-surgical methods commonly include: hand physical therapy, thumb spica splints to immobilize the irritated tendons, anti-inflammatory NSAID prescriptions, and corticosteroid injections to reduce the inflammatory swelling and irritation of the APL/EPB tendons. Although these non-surgical approaches solve the concerns of immediate pain, there remains a considerable incidence of pain recurrence which has borne the question of "how efficient are these methods for curing DQD patients". It was initially understood that corticosteroid injections alone had almost a 6 times greater cure rate than splints alone (34). 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 reduces VAS (visual analogue scale) pain scores more than using the steroid injection method exclusively (12). To-date, steroidal injections directly proximal to the radial styloid process remains the treatment of choice for newly diagnosed DQD patients (28). Initial corticosteroid injections have proven a cure rate ranging 62-100% with the failure-to-cure associated with a present APL/EPB septum or specific mechanical triggering of the 1st dorsal compartment (35). For patients with pain recurrence 2 weeks after the 1st injection, a second injection is usually administered and if pain remains 2 weeks later then it is expected that a third injection would be futile to alleviate the symptoms, therefore requiring a surgical approach (36).

For extreme cases of DQD patients failing to resolve symptoms within 6 months of corticosteroid injections or other non-surgical treatments, a surgical release of the 1stextensor compartment is required. When accessing the 1st dorsal compartment, it is pivotal to longitudinally incise the EPB sub-compartments (the tendon most likely needing decompression) and the septum dividing APL/EPB tendons if present (27). Failure to properly incise the septum will ultimately result in

failed decompression and refractory DQD symptoms. Another complication during the surgical release is the EPB tendon subluxation which can be prevented by avoiding complete excision of the EPB tendon sheath (37,38). This solution to tendon subluxation does not commonly apply to the APL tendon as dorsal decompression avoids damaging the tendon sheath surface towards the EPB (39). After the procedure, postsurgical intervention recommends 1-2 week thumb spica splint followed by weeks of active range-of-motion exercises, scar/edema management, and strengthening exercises (11).

Consequences of anatomical variations on steroid injections & surgery

As reporting has shown across multiple studies, a consistent incidence of DQD patients remains for those having pain recurrence post-initial injection and thus require a second corticosteroid injection. Interestingly, most DQD patients receiving a second corticosteroid injection do not require a third dosage to resolve their symptoms (40,36). The necessity of the second dosage is commonly attributed to the anatomical variations between DQD patients. Across multiple studies, the variation of the septum beginning at the radial styloid and dividing APL/EPB tendons at the 1st dorsal compartment has been commonly theorized to enclose the initial injection to only one tendon compartment therefore leaving the other tendon untreated and allowing for symptoms to resurface. Therefore, it is common practice for the 2nd injection to target dorsally the potential EPB tendon sub-compartment as it is the most likely to be more irritated than the APL (28). The injection site and the presence of sub-compartments or septation are crucial factors that must be considered for both the efficacy of corticosteroid injections and surgical release of the 1st dorsal compartment.

As mentioned previously, surgical release of the 1st dorsal compartment for refractory DQD patients requires severing the EPB subcompartment and the septum if present to ensure a successful procedure. Interestingly, reporting shows that approx. 62.2% of surgically treated DQD patients have a septum and among those only approx. 58.5% are incomplete septums (28). The lack of properly making a transverse incision at these two structures results in failed decompression of the 1st dorsal compartment and a recurrence of pain weeks later (41,42). It has been noted as well that DQD patients present with a greater sub-compartment incidence therefore complicating the surgical release procedure intending to cure the patient and avoid post-op complications or failed treatment.

Other modern & developing treatment approaches

Here we summarize 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. High 3MHz frequency ultrasound (both continuous and 88.25 but has been found to be contraindicated for patients with acute inflammation or surgical tendon repairs within the last 6 weeks (43). Phonophoresis/sonophoresis uses ultrasound to direct topical anti-inflammatory medications deeper into tissues while Iontophoresis uses an electrical

current/gradient to deliver anti-inflammatory medication to shallow regions of the hands/feet to reduce edema, inflammation, scar tissue, and pain (43). Both of these techniques are commonly used for patients with hyperhidrosis but has also provided useful for chronic overuse tendinopathies and stenosing tenosynovitis like DQD. It is also reportedly uncertain 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 remodeling in accordance with the stress placed on it" (34, 44). With these new treatment approaches and more developing, the DQD treatment timeline presents a trend enhancing the rehabilitation of stenosing tenosynovitis and reducing the time necessary for a patient to recover from persistent pain and regain strength. The greatest caveat of these practices is the lack of available research regarding the efficacy of such treatment and comparable utility to current surgical/non-surgical treatment methods.

Conclusion:

Since Fritz De Quervain first postulated stenosing tenosynovitis within the radial dorsum of the wrist, a considerable amount of research has been conducted to provide further insight. A wide array of unique anatomical variation within the first dorsal compartment exists within the population. Numerous studies have shown that structural divergence from normal anatomy is, at least in part, related to contingency for developing DQD. The exact pathological pathway which leads to DQD is a subject of debate. Two schools of thought have emerged in the present epoch, one that contends an inflammatory mediated pathway and one of degenerative changes. Strong evidence is provided for both explanations and further scrutinization is required to better understand the patterns that bring about DQD. Classically, Finkelstein's and Eichhoff's test have been used as the physical exams of choice for making a clinical diagnosis of DQD, however these tests have been shown to have low specificity. There is evidence which suggests ultrasonography may become a key diagnostic tool for identifying DQD. The management of DQD is typically conservative in nature, with escalation up to steroid injections before surgery is indicated. Future research into DQD should focus on establishing a clearer picture of how anatomical variation and other pathological factors may interplay to bring about this condition. While current research has suggested possible novel approaches for diagnosing DQD, more studies are required to gain greater insight into the effectiveness of these interventions. Thus further investigation will be necessary to refine and further elaborate our current understanding of DQD.

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