Trigger Digits

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Trigger digits, a relatively simpler ailment, was brought to the fore by a young medical intern, Alphonse Notta, serving at *l'hôpital Saint-Louis.* Reporting the clinical presentation of a seamstress, he wrote "When all the fingers of the right hand are flexed and the patient wants to straighten them, all the fingers straighten out comfortably except the middle finger which begins to straighten then it stops. The patient then forcefully straightens out her extensors, a brisk movement occurs. The middle finger then straightens out easily. However, very often this extra effort of the extensors is not enough and her finger remains flexed. The patient then uses the extremity of the right thumb and lifts the last phalanges of the middle finger a little, this then allows her finger to be extended"[1].

Notta, published his observation in 1850 stating that, "the medical condition described in these case studies pertains to a nodosity on the span of the flexor tendons of some fingers. This nodosity encounters obstacles when fingers are being bent and stretched… What is the cause of this nodosity? What obstacles does it encounter?''[1].

Before venturing into the works of pioneers to answer these questions, what does the term "trigger finger" mean for a clinical practitioner? Often referred to as stenosing tenosynovitis, this condition is diagnosed when a patient presents with a symptomatic locking or clicking of a finger or the thumb produced by a mismatch between the volume of the flexor tendon sheath and its contents.

Epidemiology

The life time risk of developing a trigger finger is 2-3% and it occurs six times more frequently in women in their fifth and sixth decades of life[2]. The ring finger and the thumb are most frequently affected and least symptomatic in index and little fingers. The risk is increased to as much as 10% among people with diabetes mellitus[3]. More than one trigger digit can be present in the same hand.

Etiological factors and associations:

Though described 167 years ago, there is no clear consensus present in the literature about the causative factors of trigger digits. The first case report by Notta described the clinical presentation of a seamstress and hence he speculated that the cause for the pathological lesion would be a result of "repetitive occupational micro trauma to the tendon" from her constant sewing[1]. But majority of these are cited as being of an idiopathic nature, collectively called as primary trigger finger where the site of obstruction is the first annular pulley. Secondary trigger digits are those seen in association with other diseases like carpal tunnel syndrome and other systemic diseases like diabetes mellitus, rheumatoid arthritis, gout, amyloidosis, mucopolysaccharoidosis etc.

Coexistence of carpal tunnel syndrome and trigger finger is a common observation. An inflammatory process, both at the level of A1 pulley and the carpal tunnel may be the cause for association. Conflicting evidences exist in the literature whether this association is coincidental or one leading to the other.

Association between trigger digits and diabetes mellitus provides an interesting research material where exists an unclear evidence that diabetes mellitus increases the risk for development of trigger digits. Koh et al suggested that "screening for diabetes may be warranted in patients with involvement of more than three trigger digits"[3].

Pathophysiology

Under normal circumstances, the flexor tendons must glide quietly through the annular pulley system during flexion and extension of the digits. They are encased in a double layered synovial sheath which facilitate smooth gliding. This synovial sheath is closely related to the tendons and the pulley system. During forceful flexion of the digits or power grip, there occurs a considerable angulation of the tendons at the proximal edge of the A1 pulley which acts as a fulcrum or the constricting element.

Two pathological variants have been described namely the nodular variant, where the swelling is contained, which moves along with the movement of the tendon and the diffuse variant, where the swelling is diffuse and less defined[4]. The development of each variant has a different pathophysiology.

In 1874, Arthur Menzel, a German physician, proposed a theory for the origin of a trigger digit. Artificially constructing a Notta's node by looping thread around the flexor tendons and manually inserting grains of rice into the flexor sheath in cadavers, he concluded that the "contraction of the sheath" and the "circumscribed tumour of the tendon" which are "the products of inflammation" should act mutually as the causative agents of the ailment[5].

In 1954, Bollinger and Fahey, wrote that the nodule was actually, "the markedly thickened fibrous sheath, two to three times the normal thickness" and a fibrocartilaginous metaplasia is noted in the inner layer of the pulley[6].

In 1972, Hueston and Wilson proposed the popular "needle and thread analogy" in which they studied the arrangement of the fibers within the flexor tendons. They found that these fibers follow a spiral pattern. When a recurrent deforming force is exerted upon the tendons during flexion and extension, by the constricted tendon sheath, particular segment of fibers "bunch up" into a nodule distal to the constriction, the A1 pulley in this case (Figure 1A and B). They also noted that the flexor tendon would be forced through the constriction during active flexion and there was no such force against the constriction during active extension. This explains why the nodule formation occurs always distal to the constriction and when this nodule slips proximal to the pulley, the finger remains in the characteristic posture of trigger finger[7] (Figure. 1C).

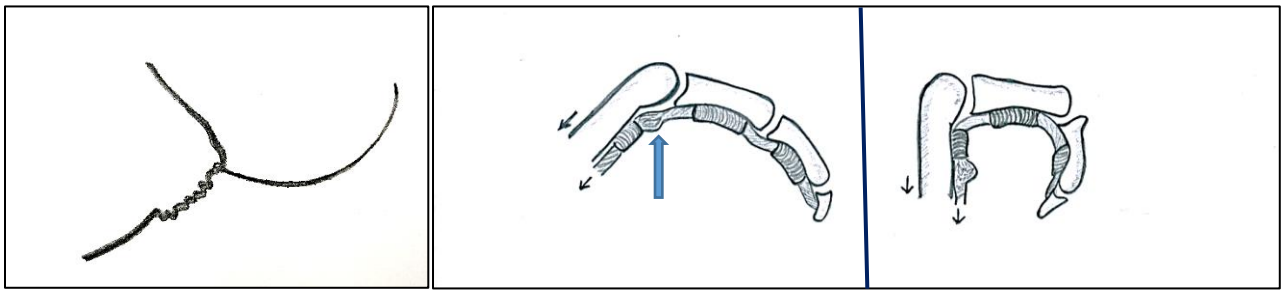

Figure. 1A, B and 1C- The 'needle and thread analogy' concept of the cause of nodule formation in the trigger finger. The nodule formation is just distal to the A1 pulley (arrow), the site at which the maximum force is exerted by the flexor tendon.

In trigger digits associated with systemic diseases, the fibrous sheath thickening has been attributed to the accumulation of abnormal proteins in the tenosynovium. In patient with diabetes mellitus, glycosylation of collagen,

confers them with increased cross linking, packing and stiffening of collagen resulting in thickening of the fibrous sheath which in turn leads to trigger digits[3].

However, the pathophysiology in cases associated with rheumatoid arthritis is the diffuse inflammation of the tenosynovium that produces a mismatch between the size of the contents of the sheath and the enclosing fibro- osseous canal.

Electron microscope studies of the A1 pulleys have helped to elucidate the pathogenesis of trigger digits. The normal A1 pulleys were characterized by the presence of amorphous extracellular matrix and chondrocytes lining the inner layer of the fibrous sheath. The pathologic pulleys were characterized by the presence of loss of extracellular matrix, chondrocyte proliferation and type III collagen production[8].Thus it can be postulated that this fibrocartilaginous metaplasia results from the repeated friction and compression between the flexor tendon and the corresponding inner layer of the A1 pulley.

Classification

Various classification systems have been proposed according to the mechanical disorders and symptoms (Newport and Froimson) [9, 10], based on the severity of symptoms (Quinnell and Eastwood) [11, 12] and based on mechanical disorders only (Patel and Bassini) [13]. Though there is no effective and uniform clinical classification, the most frequently used are those proposed by Froimson and Green[14]. Comprehensively, the trigger digits can be graded as:

Grade I: Pre-triggering-pain; tenderness over the A1 pulley; history of catching but not demonstrable on physical examination
Grade II: Triggering, active; demonstrable catching, patient can actively extend
Grade III: Triggering, passive; demonstrable catching requiring passive extension or inability to actively flex
Grade IV: Contracture; demonstrable catching, with a fixed flexion PIP joint contracture

Clinical presentation

The patient presents with a slight discomfort or pain in the involved digit localized to the base of the digit. Initially, the triggering may not be painful. The patient may feel a mild click in the finger or may report inability to fully flex the finger. With progression of the disease, there is distinct discomfort on the palmar side of the metacarpophalangeal (MCP) joint, with pain frequently radiating into the forearm. When triggering occurs, the patient often perceives the snapping as occurring at the proximal interphalangeal joint. Triggering, noted in early morning becomes less bothersome as the day progresses when the patient carries out his/her activities of daily life which is not the case in patients with advanced disease. On physical examination, there might be tenderness at the palmar base of the involved digit, indicative of early tenosynovitis. Once deformation of the tendon has occurred, "catching" of the digit will be manifested as the patient tries to extend the fingers from a fist position. More severe forms of the disease will lock the finger or thumb in flexion, requiring the patient or examiner to push the finger into extension; there will be noticeable "give way" on unlocking. A careful history and a thorough physical examination to rule out the associated medical conditions such as rheumatoid arthritis, diabetes, gout and carpal tunnel syndrome should be carried out.

Treatment

Duration of symptoms and the type of pathological variant, nodular or diffuse form the basis of the treatment protocol. Symptoms that have been present for more than six months were shown to respond poorly to non-surgical treatment.

Non-surgical treatment

Avoidance of inciting activities might be adequate in mild early cases. However, some form of treatment should be instituted with the occurrence of symptoms.

Nonsteroidal anti-inflammatory drugs along with massage, ice therapy, splinting and injections can address majority of the symptoms of early nodular tenosynovitis.

A low profile customized thermoplastic MCP joint blocking splint is found to decrease triggering of a single isolated trigger finger. The MCP joint of the involved finger is held in a position of 10^{0-} $15^{0}$of flexion leaving the interphalangeal joints free. For better results, the splint should be worn for a period of four months[15].

Long acting corticosteroid injection into the tendon sheath forms the mainstay of initial treatment in symptomatic trigger digits. Introduced by Howard et al in 1953[16], this form of treatment gained popularity and has been studied in detail with ample evidences suggesting that all the grades of tenosynovitis respond to this form of treatment to various extent. Water soluble corticosteroids like, Betamethasone sodium phosphate is the most commonly used as it does not precipitate in the tendon sheath following injection. Methyl prednisolone and triamcinolone have also been found to be effective. The approach can be either lateral or palmar, of which the lateral approach is found to be easier and less painful as the neurovascular bundle lies palmar to the area of injection. A 1-cm^3 syringe with a 25 or 27-gauge 0.5- inch needle is used. From the radial border of the finger, the needle is inserted into the mid-lateral area of the proximal phalanx above a line connecting the proximal and distal interphalangeal joint creases over the first cruciate pulley (the neurovascular bundle lies palmar to that line). The skin and subcutaneous area are anesthetized with 1% xylocaine without epinephrine. The needle is inserted only until slight resistance is felt. The patient is asked to wiggle the finger. Slight grating can be felt at the end of the needle. If the needle is in the tendon proper, there is paradoxical motion of the needle and syringe (i.e., with digit extension, the syringe moves proximally). The rest of the anesthetic is then injected into the tendon sheath. The needle is disconnected from the 1-cm^3 syringe but left in place, and the syringe is reloaded. When a corticosteroid is used, 0.75 mL of such an agent and 0.25 mL of 1% xylocaine are loaded in the syringe, which is reconnected to the needle left in the finger. The patient is again asked to wiggle the finger to ascertain the correct position of the needle. The injection is administered, and the needle is withdrawn. The treated digit should remain anesthetized for 3 to 4 hours. Benefits from the steroid injection should persist for 2 to 5 days after the procedure[17].

Success rate ranges from 67% to 90% following the first injection, with treatment of the thumb being most successful and this rate falls down to 36% following second injection[2]. Response rate to steroid injection diminishes with increased duration of symptoms and number of injection.

In case of diffuse tenosynovitis, steroid injection can be used only once and only when the symptoms have been present for less than four months and if more than four months, surgical release is indicated.

The success rate with steroid injection falls down to 30% in patients with diabetes mellitus[18]. With reports of hyperglycemia[19] and increased recurrence rate of about 50%, steroid injection is not favored for treating trigger digits in patients with diabetes mellitus.

By instituting proper technique, complications noted with corticosteroid injection are rare and include hypo-pigmentation of the skin, tendon rupture and fat atrophy at the injection site.

Surgical treatment

With a success rate of 99%, surgical release of the first annular pulley is the traditional gold standard method of treatment in all cases of primary trigger digits and those associated with diabetes mellitus. Two methods namely percutaneous release and open release have been described.

Percutaneous release of A1 pulley

Percutaneous release of the A1 pulley first was described in 1958 by Lorthioir[20]. The technique has gained popularity recently and a number of studies have evaluated the safety and efficacy of percutaneous release. Several instruments have been advocated for the procedure including a hypodermic needle, a tenotome, or specially designed knives.

Percutaneous release can be performed as a day care procedure. Local anesthetic mixed with corticosteroid is administered and the palmar base of the affected finger is prepared. The patient is asked to flex the affected digit actively. The surgeon then hyperextends the finger which brings the flexor tendon sheath directly under the skin and allows the

neurovascular bundles to displace to either side. An 18-gauge needle or other device is inserted at the proximal aspect of the A1 pulley. Care should be taken to stay centered over the flexor tendon sheath to avoid neurovascular structures and to enter the skin perpendicularly with the bevel of the needle parallel to the tendon. The proximal edge of the A1 pulley is located near the distal horizontal palmar crease for the small, ring, and middle fingers. For the index finger it is located at the proximal horizontal palmar crease. Release of the ring and middle fingers is believed to be relatively safe. Surface landmarks for needle insertion have been studied in detail and the injection sites are shown below (Figure. 2). The needle is inserted one third the distance from the distal palmar crease to the base of the long, ring, or small finger. In the case of the index finger, the needle is inserted one third the distance from the distal thenar crease and the base of the finger. These locations have been found to consistently correlate with the middle of the A1 pulley and to allow cutting both proximally and distally to completely transect it[21].

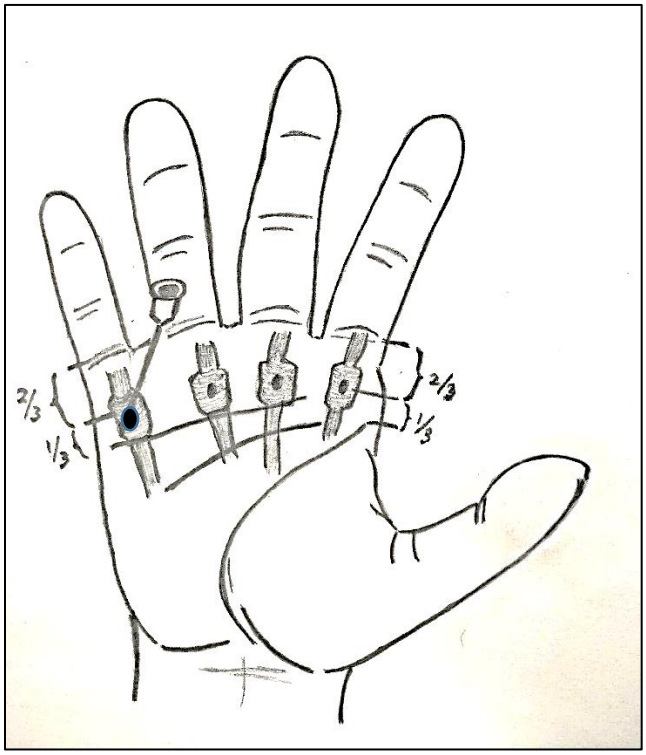

Figure 2: Needle entrance points (dots) are located approximately one third the distance from the distal palmar crease and two thirds the distance from the proximal digital crease. This corresponds to the center of the A1 pulley.

In the thumb, since the neurovascular bundles are more central and closer to the flexor pollicis longus tendon the percutaneous release is considered risky and authors recommend not to use the percutaneous technique for pulley release in the thumb. Particularly the radial digital nerve is at great risk as it obliquely courses across the flexor tendon.

Short duration of surgery, shorter duration of postoperative pain, quicker recovery of full hand function and faster return to work have been proposed by the investigators as the advantages of percutaneous release[22].

One of the major complications noted with percutaneous release is injury to the digital nerve. Cadaver studies have shown that the digital nerves in the index finger and thumb lie within 2 to 3 mm of the needle puncture site. Hence, authors recommend to avoid using the percutaneous release technique for these two digits. Incomplete release of the pulley is another concern though Pope and Wolfe hypothesized that triggering resolves even if the distal edge of the A1 pulley is not released[23]. Post-operative painful tenosynovitis without triggering, a reactive inflammation due to scoring of the flexor tendon is a common complication. This can be minimized with pre-operative local administration of corticosteroid with anesthetic agent.

Open release of A1 pulley **(preferred method of treatment at our center):**

Open release of the A1 pulley has been used to treat trigger digits for more than 100 years. We prefer to perform an open A1 pulley release under local anesthetic so that the complete release can be confirmed intra-operatively before closure of the wound as evidenced by the full range of active flexion and extension. Others believe that local anesthetic distorts the surgical anatomy and therefore prefer a regional anesthesia. Transverse, longitudinal, or oblique incisions on the volar aspect of the hand overlying the MCP joint and A1 pulley all have been described and we prefer a transverse incision at the level of an imaginary line joining the distal palmar crease at the ulnar border and the proximal palmar crease at the radial border of the palm (Figure 3).

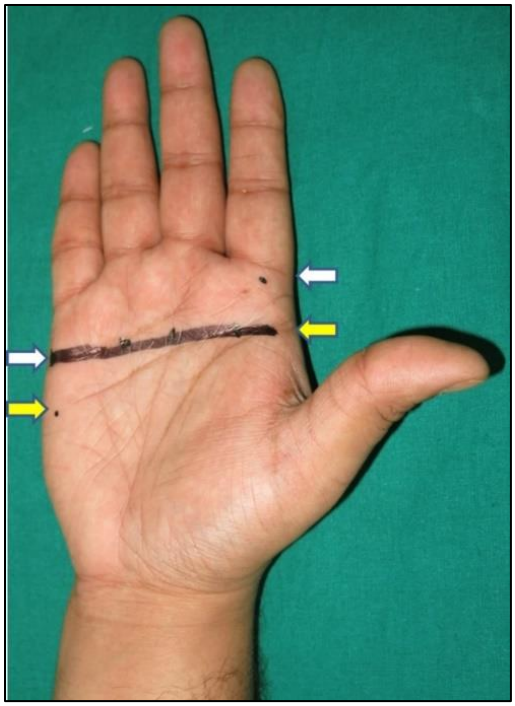

Figure 3: Transverse incision made at the level of an imaginary line joining the distal (white arrow) and proximal (yellow arrow) palmar creases.

Blunt dissection is continued down to the level of the flexor tendon and the entire A1 pulley is visualized with care taken to protect the neurovascular bundles that are located on the radial and ulnar sides of the tendon sheath. The radial neurovascular bundle to the thumb is most at risk for injury because it takes an oblique ulnar to radial course across the A1 pulley. This bundle is also subcutaneous, averaging 1.19 mm deep to the dermis at the thumb MCP flexion crease, and may be transected with a deep skin incision. The A1 pulley should be released completely for symptoms to resolve reliably. The patient is then asked to flex and extend the digit intra-operatively. If triggering is still occurring, the release should be checked for completeness and further release of the A1 pulley may be warranted. In patients operated under regional anesthesia, the complete release of the A1 pulley can be ensured by performing the ‘squeeze’ test where the distal one third of the forearm is ‘squeezed’ following which the fingers fall into complete flexion. Similarly, the completeness of trigger thumb release can be checked by passive hyper-flexion of the wrist which results in complete extension of the thumb if the pulley has been completely released. After achieving hemostasis, the wound is closed with absorbable

sutures. Generally, only supportive dressings are needed after surgery. Full range of active mobilization is allowed from the second post-operative day.

Inadvertent release of A2 pulley followed by bowstringing and digital nerve injury are the complications noted following open surgical release. The former can be avoided with proper visualization of the entire pulley. Hazani et al described a technique of inserting a hypodermic needle 5mm proximal to the palmar digital crease marking the distal edge of A1 pulley[23]. Thermal injury to the digital nerves while using electro-cautery should be avoided.

In patients with trigger digits associated with rheumatoid arthritis, release of the A1 pulley is not recommended as there will be an increased tendency for the ulnar drift of the fingers (Figure. 4). It has been noted that despite pulley release, the finger motion will be still limited due to the presence of diffuse tenosynovitis or rheumatoid nodules in the region of other pulleys. These patients require synovectomy while preserving the pulleys. Release of ulnar slip of flexor digitorum superficialis might be required in certain cases to achieve unhindered gliding of the flexor tendons.

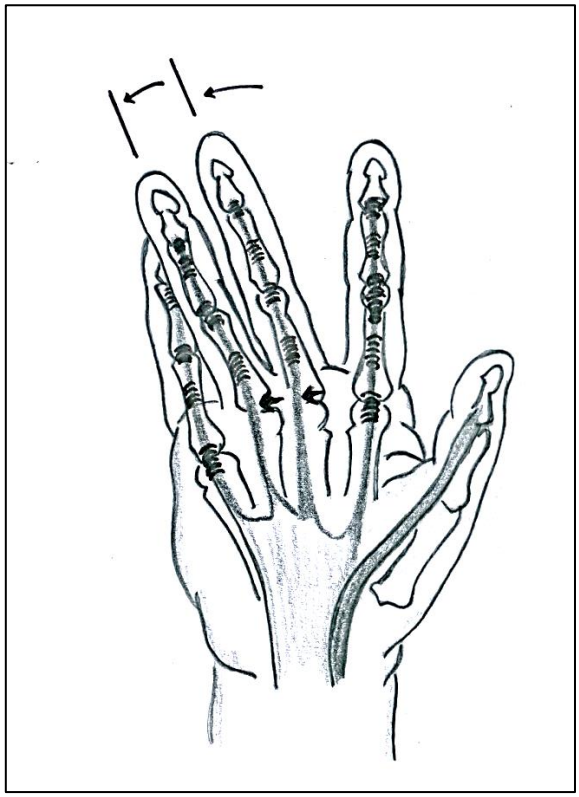

Figure 4: The tendency toward ulnar drift of the flexor tendons after the A-1 pulley of the middle and ring fingers

Conclusion

Trigger digit is a relatively simpler mechanical problem caused by a mismatch between the relative size of the flexor tendon and its sheath. Measures to release sites of sheath impingement or to reduce the local volume of the flexor tendon will relieve symptoms. If non-operative measures like activity modification, anti-inflammatory medication, splinting, corticosteroid injection fail operative treatment with open or percutaneous A1 pulley release is indicated.

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