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RESEARCH ARTICLE

A Hole in One

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ABSTRACT:
This case report discusses the presentation, diagnosis, and treatment of trigger finger in a 62-year-old right-handed male with a history of two months of right index finger pain and locking sensation. The patient's occupation and activities, such as typing, golfing, running, and biking, potentially contributed to the development of trigger finger. Physical examination revealed tenderness near the A1 pulley, a palpable nodule, and limited extension and abduction of the affected finger. Imaging showed mild degenerative changes and a small sesamoid bone. The patient received a corticosteroid injection to the tendon sheath, along with high-velocity low-amplitude (HVLA) manipulation and myofascial release to correct associated somatic dysfunctions. The patient experienced reduced pain and improved range of motion after treatment, and follow-up at one month showed complete resolution of symptoms.

Keywords: Trigger finger, A1 pulley, corticosteroid injection, somatic dysfunctions.

Introduction

Trigger finger is a frequently encountered problem in the outpatient setting that leads to pain and deformity in one of the digits on the hand[16]. It leads to a painful catching sensation that typically occurs as the finger is moved from a flexed to extended position[16]. Due to its progressive nature, it can lead to significant functional deficits and even deformity if not treated[16].

Trigger finger is caused by inflammation from irritative forces between the tendon and tendon sheath. This leads to a thickening of the tendon as well as a narrowing of the flexor pulley sheath[16]. This combination reduces the ability for the tendon to move smoothly through the sheath during finger motion. Trigger finger most frequently affects the ring finger or thumb on the right hand but can present on any finger. The tendon and tendon sheath thickening most often occurs at the A1 pulley in the hand (Figure 1).[1]

The condition is graded by its severity with Grade I consisting of some pain and a history of catching. As trigger finger progresses, Grade II presents with more consistent catching where the patient can induce catching but still maintains the ability to actively move the finger through full flexion and extension. In Grade III, the patient is unable to actively extend the finger once catching occurs and must use their other hand to move the finger into its fully extended position. In the last and most debilitating phase, Grade IV, the finger is fixed in its contracted position.[17]

While trigger finger can present in a wide array of patients, this condition most often occurs between the fourth and sixth decades of life.[6,16] In children, it typically occurs before age eight[16]. It carriers a lifetime risk of 2.6% with women being diagnosed with it at least 2x more frequently than men.[7] Those with diabetes are four times more likely to develop this condition than the average non-diabetic population.[3] Other conditions that may predispose a patient to experiencing trigger finger include: rheumatoid arthritis, amyloidosis, gout, thyroid disease and carpal tunnel[16]. In children, the condition is typically developmental due to an incompatibly sized tendon sheath and flexor tendon where the tendon is too large for the sheath[16]. Due to its inflammatory causes, it typically presents in those completing repetitive tasks involving their hands or experience repetitive compressive forces on the hand[16]. These repetitive actions lead to inflammation and tendon thickening due to fibrocartilaginous metaplasia where the tendon and pulley meet[16].

Upon patient presentation, patients typically report an insidious onset palm pain and at least occasional painful catching where the affected finger is unable to move smoothly from flexion to extension.

Patients may note a palpable nodule in the palm proximal to metacarpophalangeal joint. Swelling or pain may be present in the area of the nodule. Due to the symptomatology, predominance for the right hand and its occurrence in those who utilize their hands for work, frequently patients will report difficulty with work-related tasks.

The diagnosis is typically made after collecting patient history and performing a physical exam. If imaging is obtained, diagnostic ultrasound is the most helpful and most cost-effective modality. During evaluation, tendon thickness can be compared between affected and non-affected sides. Dynamic ultrasound evaluation can also be performed where the catching is able to be seen while passively moving the finger from flexion to extension. Xray can be obtained to rule out other conditions.

Most cases can be managed conservatively. Steroid injection remains a first line treatment so help decrease the inflammation and therefore the thickness of the tendon. Typically, patient symptoms including pain and function improve following injection however symptoms can recur. Those with a more recent onset of symptoms are more likely to respond to injection[16]. Other conservative treatment options include: non-steroidal anti-inflammatory drugs (NSAIDs), therapeutic exercises, joint mobilization, and splinting.[4] Again, these are more likely to be successful earlier in the disease course[16]. Surgical release of the A1 pulley is considered if conservative treatment fails or Grade IV cases[4,16].

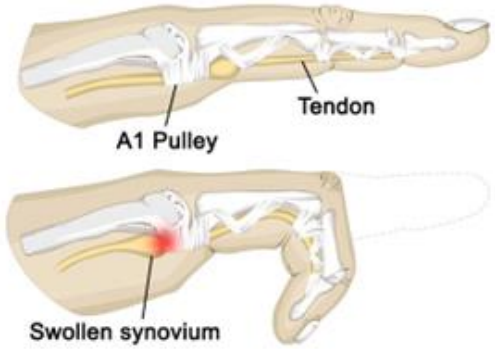

Figure 1: A1 pulley of the finger[4]

Case History

A 62 year-old, right-handed male presented with a two-month history of pain in his right index finger and reported a sensation of the finger locking. The discomfort was localized to the distal area of his

palm. The patient worked as a statistician that required frequent typing. Notably, he had not undergone any prior treatments for this condition, and there had been no ergonomic assessments or adjustments before his presentation. The patient had no history of injuries or surgeries on his hand and had not previously experienced this type of pain. He enjoyed regular golfing (three times per week), running, and biking, and had no relevant medical or family history that could contribute to his current condition.

PHYSICAL EXAM

No signs of deformity, ecchymosis, or swelling were observed in the right hand. However, tenderness was noted upon palpation at the base of the second metacarpophalangeal (MCP) joint. A discernible palpable nodule was identified near the A1 pulley region. Notably, attempts at passive motion failed to elicit any triggering response in the affected finger. Active range of motion assessment revealed full flexibility in finger flexion, abduction, and opposition of the right hand's fingers. Nevertheless, limitations were observed in terminal extension of the second digit, as well as in right wrist flexion and abduction. Dermatome and myotome testing for C5-8 and T1 demonstrated normal results. Furthermore, the patient displayed an abducted right ulna, radiocarpal flexion, and a posterior glide of the right metacarpal bone.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis considered for the patient's condition included trigger finger, sesamoiditis, capsulitis, second metacarpal posterior glide, as well as the possibility of a flexed right capitate and an abducted right ulna. Each of these potential diagnoses was evaluated based on the patient's symptoms, physical examination findings, and imaging results to accurately pinpoint the underlying cause of the finger discomfort and locking sensation.

Figure 2. Pinch View Xray: Sesamoid bone volar to 2nd MCP Head (arrow).

IMAGING, DIAGNOSIS, AND PATIENT COURSE

Imaging studies were conducted, including three-view hand x-rays, which revealed the presence of mild degenerative changes within the hand's structures (Figure 2). Notably, no evident fractures or dislocations were observed. Additionally, the imaging captured the presence of a small sesamoid bone located volar to the second metacarpophalangeal (MCP) head, providing valuable insights into the anatomical configuration of the hand.

Our final diagnosis was trigger finger of the second digit. His associated somatic dysfunctions were a flexed right capitate, abducted right ulna, and posterior glide of the second metacarpal. The patient's treatment plan comprised a corticosteroid injection guided by ultrasound to the tendon sheath, supplemented by osteopathic manual therapy (OMT) with high-velocity low-amplitude (HVLA) manipulation of the right capitate, and myofascial release applied to the second metacarpal and ulna (Figure 3 & 4). The patient exhibited favorable tolerance to both interventions, manifesting diminished pain and restoration of normal range of motion on the contralateral side. Subsequent evaluation after one month revealed complete alleviation of pain and symptoms, empowering the patient to resume occupational and recreational pursuits at his initial functional level.

Figure 3. Positioning and force application for HVLA for radiocarpal flexion dysfunction.[5]

Figure 4. Positioning and force application for ME for ulnar dysfunction.[5]

Discussion

This case highlights the clinical presentation, diagnosis, and successful management of trigger finger in a middle-aged male with the standard of care treatment in addition to OMT. Trigger finger, characterized by painless clicking or catching of a finger, results from inflammation at the A1 pulley of the hand[16]. This condition most commonly affects patients between the fourth and sixth decades of life[16].

This patient's age aligns with the typical age group affected by this condition. The patient's occupation as a statistician involving repetitive finger motions and his active lifestyle, including golfing, running, and biking, likely contributed to the development of trigger finger. These activities can lead to cumulative strain on the hand's tendons and sheaths, increasing the risk of inflammation and subsequent triggering. Typical associated conditions with trigger finger include: arthritis, carpal tunnel, renal disease, gout, diabetes, hypothyroidism and overuse.[6,8,9] Those with diabetes are at particularly high risk as up to 25% of patients presenting for trigger finger have diabetes and half of these patients will have multiple fingers with trigger finger.[6] The diabetic population is also at high risk of development of trigger finger following carpal tunnel release (CTR), with 10% of diabetic patients experiencing development of trigger finger following surgical CTR.[6]

Physical examination played a crucial role in confirming the diagnosis. The palpable nodule near the A1 pulley and limited finger extension and abduction were indicative of trigger finger. Trigger finger can be graded using the Quinnell Grading system which characterizes the degree of trigger finger by the patient's ability to move the finger from a flexed position to extended. Grade I, is the most mild, is used for those with uneven movement. Grade II is used when the patient is able to actively overcome the triggering by continuing to move the finger from flexed to extended without help from any other fingers or their other hand. Grade III is assigned if the patient can still passively move the finger after catching occurs with the other hand, whereas in Grade IV the patient has a fixed deformity where neither active nor passive movement allows them to fully extend the finger.[6] Our patients symptoms were consistent with Grade II which along with relatively short-term symptoms, 2 months, his prognosis was favorable with conservative management. Imaging, in the form of three-view hand x-rays, revealed mild degenerative changes and a sesamoid bone, providing valuable insights into the hand's structural condition.

Early treatment for trigger finger may include immobilization to limit continued inflammation and irritation of the already enlarged tissue or NSAIDs to reduce inflammation. If this is not successful in reducing pain and improving function of the finger, steroid injection, with triamcinolone as the preferred agent, is recommended. In comparison to dexamethasone which has a success rate of 30%, triamcinolone relieves symptoms of trigger finger in 83% of patients.[12]

Our patient's treatment plan included an ultrasound-guided corticosteroid injection (CSI) to the tendon sheath as the patient had previously tried taking anti-inflammatory medications at home. CSI helps reduce the thickness of the A1 pulley by decreasing inflammation. Prior studies have demonstrated a significant reduction in the thickness of the pulley from 1.1mm to 0.7mm after 1 month, if injected within the tendon sheath.[10] The flexor tendon also decreases in thickness however not as much as the A1 pulley. This is likely due to the difference in tissue density between the two sites.[11] Care must be taken when performing CSI for treatment of trigger finger due to its risk of tendon rupture if performed repetitively or directly into the tendon. If the first injection is not successful, then a repeat one at 6 weeks-post the first injection can be performed. Therefore, the use of ultrasound-guidance for direct visualization of the structures of the hand as well as the location of the injection is helpful.

Moreover, addressing associated somatic dysfunctions through Osteopathic Manipulative Therapy (OMT), high velocity low amplitude (HVLA) manipulation and myofascial release targeted the biomechanical factors contributing to the condition. OMT is a constellation of hands-on manual therapy techniques that improve range of motion deficiencies leading to decreased pain and improved function. OMT is useful for many conditions and there are appropriate techniques for various parts of the body. Additionally, OMT has been documented to be helpful in treatment of other hand and wrist conditions such as carpal tunnel.[13,14,15] The patient's positive response to treatment, including pain reduction and improved range of motion, alongside corticosteroid injection, highlights the value of this comprehensive approach.

Follow-up at one month demonstrated complete resolution of symptoms and restoration of the patient's functional capabilities. This case highlights the importance of a holistic approach to trigger finger management, considering both mechanical factors and patient-specific activities contributing to the condition. Clinicians should be attentive to

patient history, occupational factors, and activity patterns when diagnosing and managing trigger finger, tailoring treatment to address both the underlying inflammation and associated biomechanical dysfunctions.

Conclusion

This case demonstrates the importance of recognizing trigger finger as a potential consequence of repetitive activities, even in individuals with active lifestyles. Conservative management, incorporating corticosteroid injections and manual techniques, can lead to substantial improvements in symptoms and function. Clinicians should consider associated somatic dysfunctions that could contribute to the condition and incorporate relevant interventions.

Ultimately, early diagnosis and appropriate interventions play a crucial role in achieving favorable outcomes and allowing patients to resume their regular activities. Further research could explore the mechanistic relationship between trigger finger and associated somatic dysfunctions to enhance the understanding of this condition and refine treatment strategies.

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