COMPARISON BETWEEN TRIAMCINOLONE INJECTION AND HYDROCORTISONE INJECTION IN TREATMENT OF TRIGGER FINGER: A PROSPECTIVE SINGLE-BLINDED RANDOMIZED CONTROLLED STUDY.

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INTRODUCTION: Trigger finger is a term for stenosing tenovaginitis affecting the excursion of the long flexor of the hand at the area of zone II of the digit. Steroid injection is one of the treatment option. Triamcinolone is the steroid mainly used for trigger finger treatment. Hydrocortisone is used only in paeditric trigger finger. Therefore result

and outcome of hydrocortisone in treatment of adult trigger finger still not establish.

PURPOSE: This study was designed to test the null hypothesis that there is no difference in resolution of trigger finger in term of pain, tenderness and triggering/locking in 3 months after injection with triamcinolone, a depot form of potent steroid or hydrocortisone, a highly soluble form but less potent steroid.

METHODS: Seventy patients were enrolled in a prospective randomized controlled study comparing triamcinolone and hydrocortisone injection for idiopathic trigger finger. They were randomized into two groups; triamcinolone group and hydrocortisone group. All patients required to answer DASH questionnaire, give VAS score and evaluated for triggering/locking and tenderness at A1 pulley before injection, immediately after injection and 3 months after injection. Fifty-nine patients completed the 3 months follow-up (28 triamcinolone arm, 31 dexamethasone arm). Outcome measures included the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, presence/absence of triggering/locking finger, presence/absence of A1 pulley tenderness and pains severity base on visual analog scale. A Chi-square test and student t-test were used to compare both groups.

RESULTS: Immediately after injection, absence of triggering was documented in 24 of 34 patients (70.6%) in the triamcinolone group and

in 28 of 36 patients (77.8%) in the hydrocortisone group. The rates of resolution of triggering 3 months after injection were 22 of 28 (78.6%) in the triamcinolone group and 26 of 31 (83.9%) in the hydrocortisone group. In term of tenderness of A1 pulley, immediately after injection, absence of tenderness was documented in 26 of 34 patients (76.5%) in the triamcinolone group and in 28 of 36 patients (77.8%) in the hydrocortisone group. The rates of resolution of tenderness 3 months after injection were 18 of 28 (64.3%) in the triamcinolone group and 22 of 31 (71%) in the hydrocortisone group. There were no significant differences between Disabilities of the Arm, Shoulder, and Hand scores and Visual Analog Scale score for pain immediately after injection and the 3-month follow-up. After the close of the study, there was no complication in both treatment groups.

CONCLUSIONS: There is no significant different in term of resolution of tenderness over A1 pulley, resolution triggering/locking, pain and physical disabilities score improvement between the two types of steroid injection.

Keywords: trigger finger, A1 pulley, triamcinolone, hydrocortisone, steroids injection.

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Abstract

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Abstrak

‘Trigger finger’ adalah istilah umum untuk stenosing tenovaginitis yang mengganggu ekskursi tendon-tendon tangan di zon II. Suntikan steroid adalah salah satu rawatan untuk ‘trigger finger’. Triamcinolone adalah steroid utama yang diguna untuk rawatan ‘trigger finger’. Suntikan hidrokortison hanya diberi untuk rawatan ‘trigger finger’ kanak-kanak. Oleh itu persoaln timbul tentang keputusan dan hasil rawatan ‘trigger finger dengan hidrokortison.

TUJUAN: Penyelidikan ini direka untuk menguji hipotesis null bahawa tiada perbezaan dalam resolusi dari ‘trigger finger’ daripada aspek kesakitan, tenderness dan mencetuskan / mengunci dalam 3 bulan selepas penyuntikan dengan triamsinolon, steroid di dalam bentuk depot atau hidrokortison, steroid yang sangat larut air tetapi kurang poten.

TATACARA: Tujuh puluh pesakit mengambil bahagian dalam kajian prospektif terkawal rawak membandingkan triamsinolon dan suntikan hidrokortison untuk ‘trigger finger’ idiopatik. Mereka dibahagikan kepada dua kumpulan secara rawak iaitu kumpulan triamcinolone dan kumpulan hidrokortison. Semua pesakit di dalam kedua-dua kunmpulan dikehendakki menjawab soalan DASH, member skor VAS dan diperiksa untuk kehadiran mencetus/mengunci jari dan kehadiran /ketiadaan tenderness pada puli A1 sebelum suntikan, sesudah suntikan dan 3 bulan selepas suntikan. Lima puluh sembilan pesakit menyempurnakan 3 bulan rawatan susulan (28 dalam kumpulan triamsinolon, 31 dalam kumpulan deksametason). Keputusan kajian termasuk skor DASH, kehadiran /

ketiadaan mencetuskan / mengunci jari, kehadiran / ketiadaan tenderness pada puli A1 dan skor VAS.

KEPUTUSAN: Selepas penyuntikan, ketiadaan ‘trigger finger’ didokumentasikan dalam 24 daripada 34 pesakit dalam kumpulan triamsinolon dan di 28 daripada 36 pesakit dalam kumpulan hidrokortison. Kadar resolusi symptom di dalam 3 bulan selepas suntikan, sebanyak 22 daripada 28 di dalam kumpulan triamsinolon dan 26 daripada 31 di dalam kumpulan hidrokortison. Untuk ketiadaan tenderness pada puli A1 didokumentasikan dalam masa 26 dari 34 pesakit dalam kumpulan triamsinolon dan di 28 daripada 36 pesakit dalam kumpulan hidrokortison. Kumpulan triamsinolon dan di 28 daripada 36 pesakit dalam kumpulan hidrokortison. Kadar resolusi symptom di dalam 3 bulan selepas suntikan diberikan, 18 dari 28 pada kelompok triamsinolon dan 22 daripada 31 pada kelompok hidrokortison. Tiada perbezaan yang signifikan antara skor DASH dan skor VAS selepas penyuntikan dan 3bulan setelah rawatan sususlan dilakukan. Pada penghujung kajian, tiada komplikasi pada kedua-dua kumpulan perlakuan.

KESIMPULAN: Tiada perbezaan yang signifikan dalam resolusi tenderness pada puli A1, resolusi ‘trigger finger’, rasa sakit dan perbezaan skor VAS dan DASH di antara dua suntikan steroid.

Kata kunci: mencetuskan jari, pulley A1, triamcinolone, hidrokortison suntikan steroid.

CHAPTER 1

1. Introduction

Trigger finger is a lay man term for stenosing tenosynovitis of long flexor of the fingers. Its main pathology is situated at A1 pulley where the pulley is thickened and entrapped the tendon subsequently affecting the tendon smooth gliding within its pulley system. Besides the long flexor of fingers, extensor tendon of the hand may also involved in stenosing tenosynovitis. De Quervain's disease is the common stenosing tenosynovitis involving first compartment of extensor of the hand[1].

Due to dissatisfactory results of treatment with splinting, researchers use intra-sheath steroids injection which resulted in high proportion of good outcomes[2]. Most commonly use steroid for trigger finger injection are triamcinolone, dexamethasone and hydrocortisone. Only triamcinolone is used widely for treatment of trigger finger because triamcinolone has more rapid effect on treatment of trigger finger. Relief of pain at A1 pulley resolved in three to five days and triggering or locking resolved in two to three weeks[87]. Dexamethasone has less rapid effect compared to triamcinolone but believed to be more durable comparing to triamcinolone injection[122].

Hydrocortisone is used only in paeditric trigger finger[129]. Therefore result and outcome of hydrocortisone in treatment of adult trigger finger still not establish. Moreover the cost of hydrocortisone cheaper than triamciolone per ampoule. Benefit from this study is to provide a different choice of corticosteroid beside triamcinolone.

CHAPTER 2

2. Literiture Review

2.1 Relevant Anatomy (condensation from Last's Anatomy Regional and Applied textbook 11th ed)[128]

Long Flexor of the Hand: The tendon of flexor digitorum superficialis enters the fibrous flexor sheath on the palmar surface of the tendon of flexor digitorum profundus. It divides into two halves which flatten and spiral around the profundus tendon and meet on its deep surface to form chiasma or partial deccusation. This forms a tendinous bed in which lies the profundus tendon. Distal to the chiasma, the superficialis tendon insert to the margin of the volar aspect of the middle phalanx. The profundus tendon enters the fibrous sheath deep to the superficialis tendon later to lie superficial to superficialis tendon partial decussation. Profundus tendon then passing distally to insert onto base of distal phalanx. Meanwhile in thumb, there is only a single long flexor tendon passing over its phalanges that is flexor pollicis longus tendon together with its investing flexor sheath.

Fibrous flexor sheath: This is a strong unyielding fibrous sheath in which long flexor tendons lies inside its fibro-osseous tunnel. It starts from the metacarpal heads to the distal phalanges of all five digits. In the thumb, the fibrous sheath contain only tendon of flexor pollicis longus while the rest of the four digits contain superficialis and profundus tendons. Over the small joints of the hand, flexor sheath becomes dense and stiff where the arcuate fibre forms pulleys. These pulleys function to prevent bowstringing of the flexor tendon and increase the tendon excursion. The two types of pulleys are annular (A) and cruciate (C). Annular pulleys are composed of single fibrous bands while cruciate pulley composed of two crossing fibrous bands.

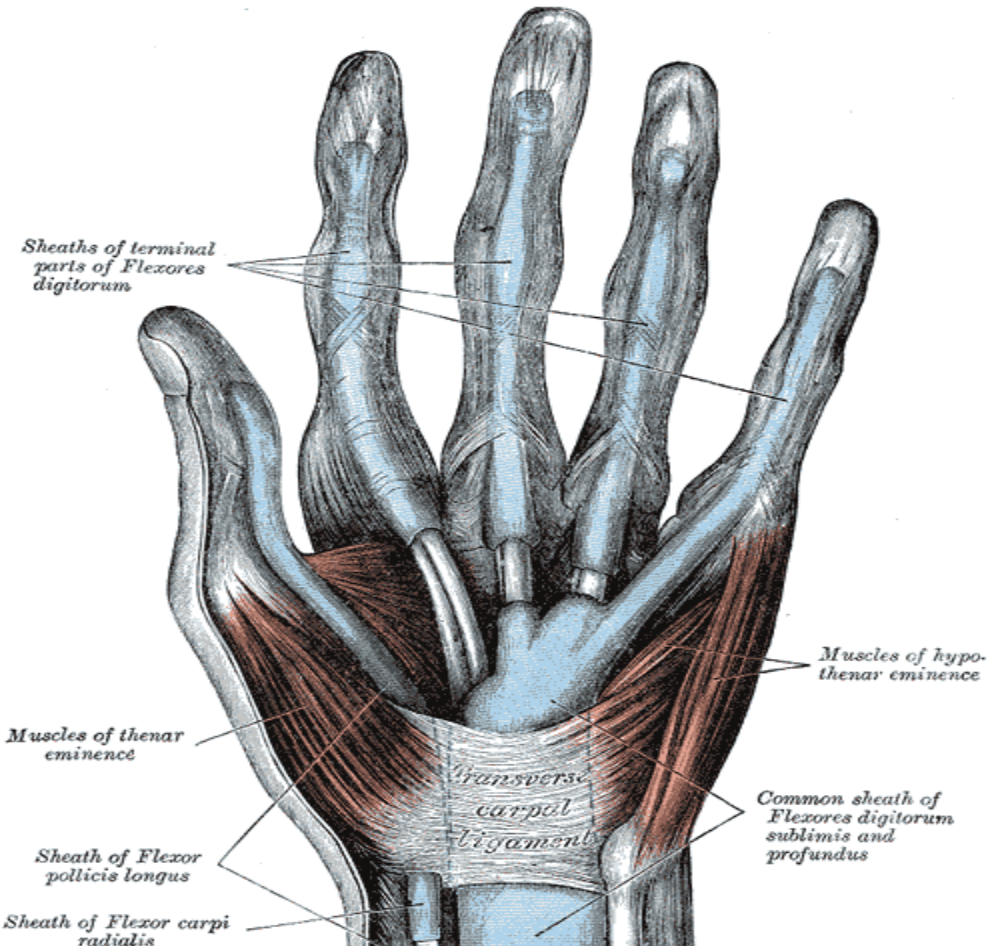

Figure 1 : The mucous sheaths of the tendons on the front of the wrist and digits

(Adopted from: IV. Myology. 1F. The Muscles and Fasciæ of the Hand. Gray, Henry. 1918. Anatomy of the Human Body. http://bartleby.com/107/126.html)

In thumb, there are two annular pulleys that are located over the metacarpophalangeal and interphalangeal joint whereas oblique pulley lies over the proximal half of the proximal phalangeal. The A2 and A4 pulleys are vital in preventing bowstringing of the flexor tendons and have to be preserved or reconstructed following any damage to these pulleys.

Synovial flexor sheath: In the carpal tunnel, the flexor tendon sheath are invested by synovial sheath which extend proximally for about 2.5cm into the lower part of the forearm and proceed distally to varying extent. Flexor pollicis longus has a synovial

sheath extending from above the flexor retinaculum to insertion of the tendon onto the base of distal phalanx of the thumb. For the superficialis and profundus tendon, they are invested inside a common synovial sheath which extends into the palm until just distal to flexor retinaculum. However, this sheath continued along the whole extent of long flexor of the little finger until its insertion. In the index, middle and ring fingers where synovial sheath ends beyond the flexor retianculum, a separate synovial sheath lines the fibrous sheath over the phalanges. It commences at level of distal transverse crease of the palm until the insertion of the tendon over the base of distal phalanx.

Detail anatomy and function of digital flexor tendon sheath have been described widely. Digital flexor tendon sheath comprised of two components, the membranous synovial component and ligamentous retinacular component[3-10]. The membranous synovial component is a double hollow tube sealed at both end[5-8,10-12]. For the index, middle and ring fingers, this layer starts at the level of distal palmar crease at the level of metacarpal neck and end just proximal to the distal interphalangeal joint. For thumb and little finger, this layer extended proximally into the carpal tunnel[3-4,6-8,11-15]. Inside this tube-like and hollow membranous synovial component contained a synovial fluid[6,8,13,16]. All the long flexor tendons of the hand lie inside this membranous synovial component of sheath in which they were aligned longitudinally[17]. The layer in which in contact with the tendons is called visceral layer and the layer not in contact with tendons is called parietal

layer[17].

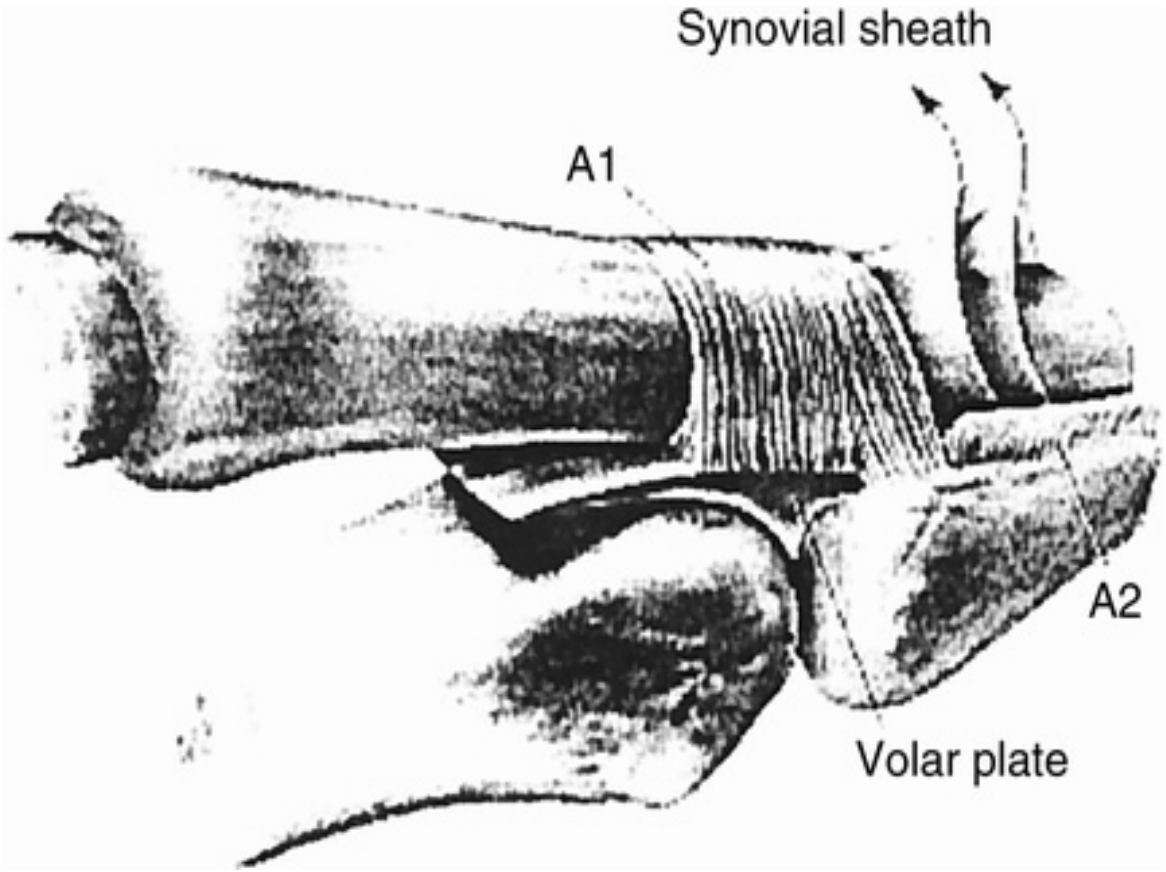

Figure 2: Microstructural of flexor tendon sheath (Moore, J. Steven MD, MPH. Flexor Tendon Entrapment of the Digits [Trigger Finger and Trigger Thumb]. *Journal of Occupational and Environmental Medicine*. 2000; 42(5):526-545)

Ligamentous retinacular part of the flexor sheath form a series of intermittent ligamentous structure called pulleys[4,5,6,8-11,15], Two types of pulley present: annular pulley and cruciate pulley[8-10,22]. A2 and A4 pulleys cover the tendon at the shaft of proximal phalanx and middle phalanx. The functions of A2 and A4 pulleys are to keep a constant relationship between tendons and joint axes of rotation and to prevent bowstringing of the tendons over the joint during flexion and over the curve surface of the shaft of proximal and middle phalanx[9]. Another three annular pulleys: A1, A3 and A5 cover the tendons over the

metacarpophalangeal, proximal interphalangeal and distal interphalangeal joint. Whereas cruciate pulleys cover the tendons as they cross the joints[9]. Cruciate pulley restraint bowstringing the least compare to annular pulley. During flexion of the long flexor tendons, A1, A3 and A5 pulleys shorten up to 50% whereas A2 and A4 pulleys shortened up to 20%. However cruciate pulleys shortened the most during flexion of the tendons. This arrangement and configuration of retinacular portion prevent buckling of the pulleys or impingement of the tendons during flexion of the tendons[8,10].

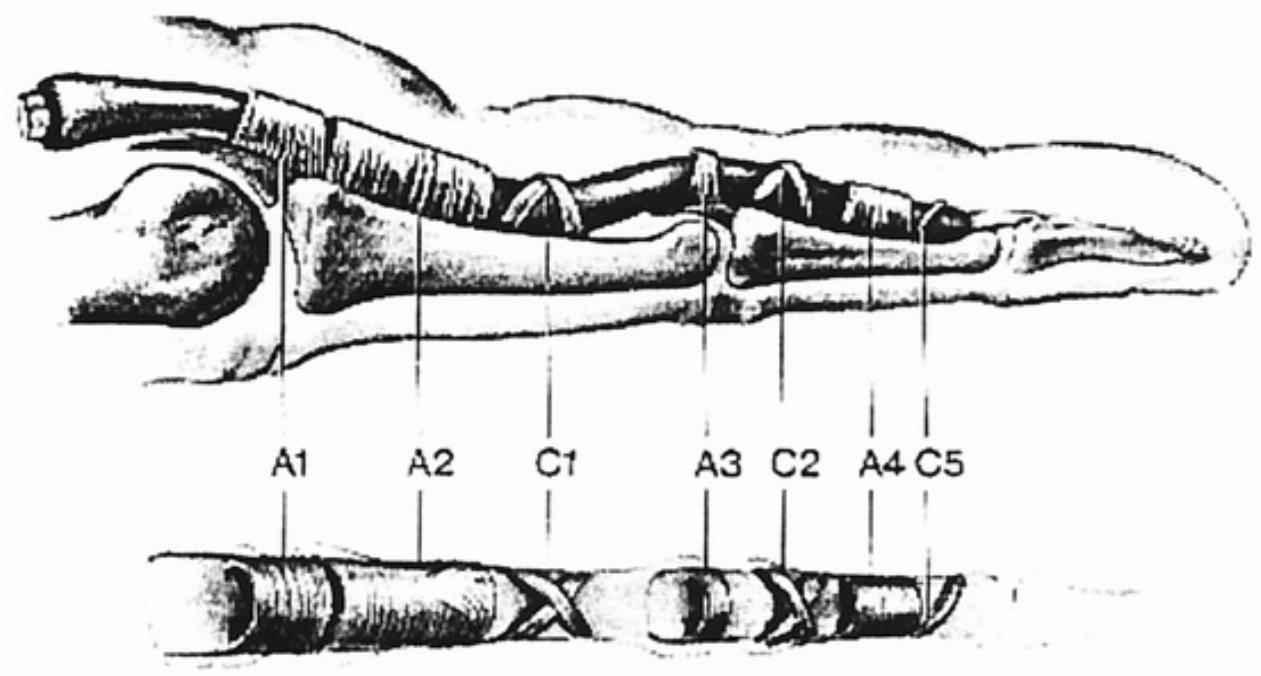

Figure 3: Pulleys of the flexor tendons of the hand (Moore, J. Steven MD, MPH. Flexor Tendon Entrapment of the Digits [Trigger Finger and Trigger Thumb]. *Journal of Occupational and Environmental Medicine*. 2000; 42(5):526-545)

The A1 pulley begins at 5mm proximal to the metacarpophalangeal joint[8,22]. It is about 0.5mm thick, and about 5mm to 10mm in length[3,4,10,19]. There is a gap about 1 to 3mm between the distal edge of A1 pulley and proximal edge of the A2 pulley[4,6,8,22]. During flexion of the digits, the synovial portion bulged through the gaps between pulleys in order to minimize buckling or deformity of the pulleys[8,15]. During full flexion of the digits, A2 pulley is the most important pulley in preventing bowstringing followed by A4 pulley[8,22]. Loss of A1 pulley resulting in no noticeable loss of digital flexion or applied force as long as A2 pulley, A4 pulley and palmar aponuerosis are intact[6,24,25]. Therefore preservation of A2 and A4 during tendon repair is recommended[23,26].

Microscopically, A1 pulleys composed of two layer, the outer vascular layer and inner friction layer[27]. The outer layer composed of dense capillary network whereas the inner layer is composed of collagen, fibroblast and chondrocytes-like ovoid cells[27]. The relatively avascular surface of the pulleys received its nutrient through diffusion from the synovial fluid[5,22].

Biomechanically, pulleys deflect the flexor tendons at their point of contact. Hume et al developed and validated a mathematical model to estimate tension on a pulley based on applied fingertip force, tendon tension, tendon excursion, joint range of motion, pulley position and pulley geometry[30]. For a fingertip force of 3N with joint flexion angle between 0 to 30° the tension value at proximal end and distal end of the pulley were predicted to be 11N and 11.5N respectively. However at more

extreme flexion degree of 90°, the predicted tension value at proximal and distal end of the pulleys were increased to 112.9N and 28.3N respectively[31]. A conclusion drawn that, at the extreme flexion degree, maximum pulley tension would be predicted despite decreased tendon tension at this angle[31].

From a cadaveric study, it is estimated that a normal A1 pulley will rupture when the pulley tension is around 250N to 300N and for A2 pulley is around 400N[30,32]. Hume et al again demonstrated that 3N fingertip force will generate up to 107N of tension in A1 pulley and with fingertip force of 12N, the tension will raised up to 428N thus placing the pulley at risk of mechanical failure[54]. Schuind et al managed to demonstrate that a daily hand activities such as hand gripping and pincer grip will generate a force of tension well below the average strength of the A1 pulley[33]. Azar et al measured intrapulley pressure of A1 pulley. In his study, pressures ranged from 0 to 50mm Hg in neutral position and raised up to 500 to 700mm Hg with full finger flexion[34].

2.2 Pathology of the trigger finger

A histological study by Sampson et al showed that the pathognomonic changes for trigger finger is fibrocartilagenous metaplasia involving the A1 pulley[27]. In his study the number of chondrocytes and extracellular matrix were increased in the trigger digits as compared to the normal digits[27,112]. However there was absence of inflammatory cells as suggested by the suffix “itis” in the term of stenosing tenovaginitis which was noted by several authors in their studies[37,38, 39,40].

Grossly, there was compromising of the cross-sectional area of the fibro-osseuos tunnel at the level of A1 pulley due to thickening of the A1 pulley[12,27,35,36,39,40]. Because of stenosing of the fibro-osseous tunnel at A1 pulley, the underlying flexor tendons may exhibit a nodular or fusiform swelling[27,36,40,123].

2.3 Pathophysiology of trigger finger

Snapping or triggering effect is mainly caused by disproportion between diameter of flexor tendons and fibro-osseous tunnel form by A1 pulley[12,35,40-42,44-,47,49]. Because of flexor tendon is stronger compare to extensor tendons, patient are usually able to the affected digits but however they have difficulty in extending the digit which further explained the snapping effect[47,49].

Rarely, trigger finger occurs at the level of carpal tunnel and A2 pulley[49,59]. Uncommon factor causing trigger finger were include

extensor tendon slipping off the metacarpal head[50], sesamoid bones abnormalitiesat metacarpal and interphalangeal joint[51], catching of the collateral or palmar ligaments on osteophytes over the metacarpal head[50,52], tenosynovial mass lesion[55,56,57] and flexor tendon mass lesion[46,58,59,60].

2.4 Frequency of the trigger finger

It is more common in women compared to man in a ratio of 2-6:1. Several series found the peak incidence of trigger digit to be in individuals age between 55 year-old to 60 year-old. According to De La Parra-Marquez et al [107] trigger finger was more common in female compared to male with increased prevalence at age of more than 53 year-old and may be related to diabetes mellitus, obesity whereas in occupation-related he found significant incidence of trigger finger in occupation of secretary, seamstress and homemaker. In his study , he found that trigger finger was more common in right hand compare to left hand, whereas in digit involvement, middle finger was the most common digit involved followed by thumb, ring, index and little finger in decreasing order. A meta-analysis done by Fleisch et al [108] showed that trigger finger was greatest in women at age average of 52 to 62 year-old. Age distribution has not changed significantly despite an increase in computing and repetitive tasks. Trezies et al [131] investigated a relationship between occupation and development of trigger finger and he found there was no relationship between the two groups. A cross-

sectional study by Gorsche et al [132] among workers at meat-packing plant showed a hand-tool use occupation may increase the rate of trigger finger development. Triggering seems to be more frequent in patients with rheumatoid arthritis or diabetes mellitus[55-57,60-63,72-77,109]. These patients were more resistant to injection treatment[78,79]. It is also frequent to be found among patients with psoriatic arthritis, amyloidosis[68,109], hypothyroidism[64], sarcoidosis and infection[68,109]. Other studies have reported a relationship between trigger finger and carpal tunnel syndrome[64-66], Dupuytren's contracture[67] and congestive heart failure[70]. However the was no reported relationship with pregnancy[71]. Trigger finger also may occurred in paediatric age group with occurance rate at age 1 year old is 3.3 per 1000 live birth[110]. However this condition was expected to resolve without any treatment in more than 60% cases[111].

Strom reported that prevalence among non-diabetic patient age above 30 was 2.2%[80]. According to Blower, patients presented with multiple affected digits were at 45% of risk to have a subsequent digit affected however patients presented with single affected digit only have 16% of risk to have a subsequent digit affected[81].

2.5 Staging of the trigger finger

Green's classification of triggering is used only for clinical grading and documentation. No correlation was established between the grading system and the outcome of type of treatment given.

Green's classification consists of four grades with subdivision of grade III into IIIa and IIIb.

Grade I	pain, history of catching that is not demonstrable clinically with tenderness over A1 pulley
Grade II	demonstrate catching but with ability to actively extend the involved digit
Grade IIIa	demonstrate locking in which passive extension is required
Grade IIIb	unability to actively flex the involved digit
Grade IV	catching with fixed flexion deformity of PIPJ

Table 1 : Green classification for trigger finger.

Another staging that commonly used is Quinnell classification[82].

Grade 0	Mild crepitus in the non-triggering finger
Grade 1	No triggering, but uneven finger movements
Grade 2	Triggering is actively correctable
Grade 3	Usually correctable by the other hand
Grade 4	The digit is locked

Table 2: Quinnell classification for trigger finger.

(Adopted from Quinnell RC: Conservative management of trigger finger. Practitioner 1980; 224:187-190. (s)

2.6 Clinically features of trigger finger

Patient may presents with snapping, locking or catching sensation together with pain over the distal palm. Sperling described a snapping or locking sensation as “spasmodically arrested, irregular movement” in which snapping, clicking, locking, stiffness and difficulty to extend a flexed digit often associates with pain or discomfort sensation[83]. In one study, there were 17% cases reported as being pain free, 60% reported as moderate pain and 23% reported as severe pain[84]. For frequency of triggering, 11% reported as absent, 52% as occasional, 22% as frequent, 8% as constant and 7% as block. For hand disability, 25.5% reported as nil, 66.5% as moderate and 8% as significant[35,36,40,85].

On examination, there will be a tenderness and/or palpable nodules over the A1 pulley[35,83,86]. Palpation also may reveal a clicking or snapping sensation on digit motion[36,40,49],. Anderson and Kaye suggested a diagnostic test with 1% lidocaine injection in which resolving pain on palpation and passive motion of the affected digits in which resolution of pain either on palpation or with provocative flexion/extension post injection as positive test[87]. However, a differential diagnosis of early stage of Dupuytren’s contracture should be bare in mind as it may be confused with trigger finger as suggested by Hadler[88]. Parker reported five patients presented with concomitant Dupuyten’s contracture and trigger finger in which the thickened palmar fascia extended to A1 pulley[89].

2.7 Treatment of trigger finger

Among option of treatments for trigger finger are conservative treatment, steroid injection surrounding the A1 pulley and surgical release of the A1 pulley. Results of the treatment much depends on the associated disease with the trigger finger such as rheumatoid arthritis, diabetes mellitus or stages of the disease at the presentation[78,79, 114]. However, spontaneous recovery has been reported in 20% to 29% of cases[36,90,113].

Splinting of the involved digit is part of the conservative management prior to corticosteroid injection development. The most recent study by Evan and colleague[91] reported a success rate of 73% with the use of splinting protocol for 3 weeks. In his study, the splint is applied over the metacarpophalangeal joint in 15 degrees flexion leaving the interphalangeal joint mobile. The splint is usually made of thermoplastic splint and worn throughout the day. All patients in his study prescribed to a hand exercise with activities required grasping, acute flexion without repeatitve stress and hook fist in order to promote tendon gliding and nutrition. From his study, Evan postulated tendon gliding exercise and massage to the entire digital sheath and palm may prevent or reduce the progression of symptoms of the affected finger. He also concluded that there was no beneficial effect of oral anti-inflammatory medications. Colbourn et al [124] managed to show in his series that splinting only managed to reduce the pain and number of triggering events in ten active fists. In this series, splinting failed to improve hand gripping and totally relieved the symptom. Rogers et al [125] advocated a splinting at the level of distal interphalangeal joint in study among workers at meat-packing

plant. In his study, eighty-one percents of the digits were successfully treated with this method in one year follow-up. This was explained in his later cadaveric study which showed a reduction in flexor digitorum profundus tendon excursion in distal interphalangeal splinting. Patel et al [126] also successfully showed a good result of splinting treatment for trigger finger. In his study, 66% of the digits were successfully treated with splinting over the metacarpophanlangeal joint at 10 degrees to 15 degrees flexion. As a conclusion from multiple studies mentioned above, splinting could be a first line option of treatment in early presentation of trigger finger.

Currently steroid injection is the first choice of the treatment for trigger finger after its introduction in 1951[92-99]. Most commonly used steroid for trigger finger injection were triamcinolone, dexamethasone and hydrocortisone. Only triamcinolone is used widely for treatment of trigger finger. The reason is because triamcinolone has more rapid effect on treatment of trigger finger. According to Anderson and Kaye [87], relief of pain at A1 pulley resolved in three to five days and triggering or locking resolved in two to three weeks. Dexamethasone has less rapid effect compared to triamcinolone but believed to be more durable comparing to triamcinolone injection based on a study by Ring et al [122].

To the date hydrocortisone was only used for treatment of traumatic trigger finger in the pediatric age groups[129]. Dexamethasone and hydrocortisone belong to glucocorticoid group with dexamethasone potency is 25 times more than triamcinolone. Comparing triamcinolone with hydrocortisone which also belong to glucocorticoid group,