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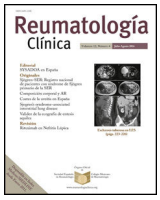

Original Article

Determinants of therapeutic success of corticoids injections in trigger finger syndrome☆

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ABSTRACT

Background and objective: Trigger finger is a frequent complaint in which corticosteroid infiltrations play a relevant therapeutic role in intermediate degrees of severity when conservative treatment has not worked. However, there are no criteria to select which patients will benefit most from this procedure. The present study aimed to identify the factors leading to the therapeutic success of corticosteroid infiltration in these patients.

Materials and methods: We designed a prospective longitudinal study based on routine clinical practice with adult patients with a clinical diagnosis of trigger finger grade II or III on the Quinnell scale, who underwent an infiltration of 20 mg of triamcinolone acetate. The outcome variables were to achieve a Quinnell grade I or reduce the severity of the symptoms by at least one category two months after the procedure. To identify the determinants of complete or partial therapeutic success, binary logistic regression predictive modelling was performed using those variables that had a satisfactory univariate correlation.

Results: 74 patients were included over three years, 42 of whom (61.8%) were classified as Quinnell grade III. After infiltration, 22 (32.4%) achieved complete resolution and 50 (73.5%) partial resolution. The variables tendon thickening (HR 10.72; 95%CI 2.88-39.93; $P<.001$) and progression time (HR 1.23; 95%CI 1.02-1.49; $P=.027$) proved to be predictors of therapeutic success in complete resolution. For the modelling for partial resolution, the same variables proved to be determining predictors (HR 5.57; 95%CI 1.38-22.41; $P=.016$ and HR 1.18; 95%CI .99-1.41; $P=.051$, respectively). Pulley thickening did not demonstrate predictive ability in either model.

Discussion and conclusions: Our results indicate that the demonstration of finger flexor apparatus thickening is the main determining factor for the success of corticosteroid infiltrations in this pathology. This is in agreement with the histological findings of specimens obtained from both tenosynovial and pulley tissue. In the former, in addition to an infiltrate of inflammatory characteristics, the presence of chondrocytoid cells producing hyaluronic acid is demonstrated. Although the therapeutic success of infiltrations in previous studies reaches 70%, the recurrence rate is similar after 12 months. The selection of patients with tendon thickening ensures therapeutic success in the short term, could reduce recurrence in the long term, and avoid delay in release surgery.

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Condicionantes del éxito terapéutico de las infiltraciones de corticoides en el síndrome del dedo en resorte

Palabras clave:
Dedo en resorte
Tenosinovitis flexora estenosante
Infiltración de corticoides

R E S U M E N

Antecedentes y objetivo: El dedo en resorte es un motivo de consulta frecuente en el que las infiltraciones de corticoides juegan un papel terapéutico relevante en los grados de severidad intermedios cuando el tratamiento conservador no ha funcionado. Sin embargo, no existen criterios que permitan seleccionar qué pacientes se beneficiarán más de este procedimiento. El objetivo de nuestro estudio es identificar los condicionantes de éxito terapéutico de las infiltraciones de corticoides en estos pacientes.
Materiales y métodos: Diseñamos un estudio prospectivo longitudinal basado en práctica clínica habitual con pacientes adultos, con diagnóstico clínico de dedo en resorte grado II o III, a quienes se les realizó una infiltración de 20 mg de acetato de triamcinolona. Las variables desenlace fueron el alcanzar un grado Quinnell I o reducir en al menos una categoría la severidad del cuadro clínico, 2 meses después del procedimiento. Para determinar los condicionantes del alcance de los objetivos se realizó una modelización predictiva de regresión logística binaria utilizando aquellas variables que tuvieron una satisfactoria correlación univariante.
Resultados: Se incluyeron 74 pacientes a lo largo de 3 años, 42 de los cuales (61,8%) tenían un grado Quinnell III. Tras la infiltración, 22 (32,4%) alcanzaron la resolución completa y 50 (73,5%), la resolución parcial. Las variables engrosamiento tendinoso (HR: 10,72; IC 95%: 2,88-39,93; p < 0,001) y tiempo de evolución (HR: 1,23; IC 95%: 1,02-1,49; p = 0,027) demostraron ser condicionantes predictoras del éxito terapéutico en la resolución completa. Para la modelización para resolución parcial las mismas variables demostraron ser condicionantes predictoras (HR: 5,57; IC 95%: 1,38-22,41; p = 0,016 y HR: 1,18; IC 95% 0,99-1,41; p = 0,051, respectivamente). El engrosamiento de la polea no demostró capacidad predictiva en ninguno de los 2 modelos.
Discusión y conclusiones: Nuestros resultados indican que la demostración de engrosamiento del aparato flexor del dedo es el principal condicionante del éxito de las infiltraciones de corticoides en esta afección. Esto concuerda con los hallazgos histológicos de especímenes obtenidos tanto de tejido tenosinovial y de poleas. En los primeros se demuestra, además de un infiltrado de características inflamatorias la presencia de células condrocitoides productoras de ácido hialurónico. Si bien el éxito terapéutico de las infiltraciones en estudios previos alcanza el 70%, la tasa de recidiva es semejante a los 12 meses. La selección de pacientes con engrosamiento tendinoso asegura el éxito terapéutico a corto plazo, podría reducir la recidiva a largo plazo, y evitar el retraso de la cirugía de liberación.

Introduction

Trigger finger or flexor stenosing tenosynovitis of the finger flexor apparatus is a common reason for consultation in primary care, rheumatology and hand surgery. Its prevalence in the general population has been estimated at 2% and up to 7% in diabetics, with a slight predominance in the female sex and in the dominant hand[1].

Trigger finger is a limitation for the extension of the finger once it has been flexed[2]. It is due to a biomechanical incongruence between the space left by the pulleys (flexor canal)[3], mainly A1, and the volume occupied by a specific portion of the finger flexor apparatus composed of the flexor *digitorus superficialis* and flexor *digilatil profundus* in the long fingers and by the flexor *pollicis longus* in the thumb. This incongruence may be due to a thickening of the pulley, a thickening of the tendinosous apparatus or a combination of both these factors[4].

Diagnosis is eminently clinical, based on the history of episodes of an impossibility to extend the finger which requires passive assistance[2,4,5]. Occasionally the thickening is palpable and infrequently the patient has pain or loss of strength[2,6]. Performing imaging tests is not mandatory for diagnosis. but due to its low cost, innocuousness and relative accessibility, ultrasound is able to distinguish the aetiology of the process on a level of pulley-tendon complex and can be used to guide local treatment[3,4,7]. Stratification of symptom severity is made using the Quinnell[8] clinical scale. This scale has 4 ordinal categories: 1) Crepitus without blockage of mobilisation, 2) actively resolving extension blockage, 3) extension blockage resolved by assistance and 4) irreducible blockage. Traditionally, when conservative management fails, and before release surgery, treatment with corticosteroid infiltrations at the level of the flexor sheath at pulley height isused[9]. This procedure does not, however, ensure a response in all cases, as approximately 30% will require surgery[10].

The aim of this study was to determine the factors associated with the therapeutic success of corticosteroid infiltrations in patients diagnosed with trigger finger at A1 pulley level.

Method

A prospective longitudinal study was conducted between May 2019 and June 2021. Consecutive patients were recruited from a musculoskeletal practice, a rheumatology practice and a general orthopaedic and trauma practice. Inclusion criterion was clinical diagnosis of the trigger finger at A1 height, confirming in the corresponding consultation any finger of the hands and the therapeutic failure of general measures. Patients with trigger fingers on specular fingers of both hands were excluded as they made comparative ultrasound study impossible. Patients who had been subjected to infiltrations with corticosteroids or fibrinolytic agents, radiotherapy or previous trigger finger operations, or previous palmar fascia disease in either hand were also excluded.

Once the patient had signed a consent form to participate in the study an ultrasound study was performed and an injection was made in accordance with standard clinical practice of a preparation of 20 mg of triamcinolone acetate and 0.5 cc of 2% mepivacaine. The total volume of the mixture was 1 ml, although only 0.3-0.5 ml was administered during infiltration. The needle was placed at the proximal palmar crease as a starting point for position adjustment using ultrasound guidance, coinciding with the anatomical position of the A1 pulley[11]. The ultrasound was performed using a Logiq 9S (General Electric®) ultrasound scanner with an 18 MHz “Hockey stick” probe. During the ultrasound scan, the thickness of the A1 pulley

Table 1
Baseline, demographic and clinical characteristics of the patients included according to the affected group on the Quinnell scale. All comparisons were made using the Chi-square or Student's t test.

Characteristic	Total	Quinnell-II N = 26	Quinnell-III N = 42	*P* value
Age (years)	59.08 ± 6.91	57.46 ± 7.59	60.09 ± 6.35	.128
Male sex	40 (58.8%)	16 (61.5%)	24 (57.21%)	.720
Comorbidities				
Diabetes	18 (26.5%)	6 (23.1%)	12 (28.6%)	.618
Hypothyroidism	14 (20.6%)	4 (15.4%)	10 (23.8%)	.514*
Rheumatoid arthritis	2 (2.9%)	1 (3.8%)	1 (2.4%)	NA
Progression time (months)	9.72 ± 3.65	9.73 ± 4.45	9.75 ± 3.19	.987
Dominant hand affected	52 (76.5%)	18 (69.2%)	34 (81%)	.268
Affected finger				.691
First	2 (2.9%)	1 (3.8%)	1 (2.4%)	
Second	11 (16.2%)	4 (15.4%)	7 (16.7%)	
Third	26 (38.2%)	12 (46.2%)	14 (33.3%)	
Fourth	29 (42.6%)	9 (34.6%)	20 (47.6%)	

NA: Not applicable.
* Exact Fisher test.

and the flexor tendon of the affected and contralateral finger were measured. Measurements were made with the finger in full extension. All infiltrations were performed under real-time ultrasound guidance with the probe positioned transverse to the longitudinal axis of the corresponding flexor tendon. The infiltrations was made inside the flexor tendon sheath in its adjacent portion and at the level of the A1 pulley, using a subcutaneous needle and standard aseptic measures.

Patients were grouped according to the detected ultrasound finding: thickening of the pulley (> 15% of the thickness of the pulley of the affected finger compared with the contralateral one) or thickening of the flexor tendon (> 15% of the thickness of the affected finger compared with the contralateral one). To determine the severity of the clinical symptoms, the original Quinnell scale was used before and 8 weeks after infiltration.

Statistical analysis was fixed at 2 primary endpoints: achieving Quinnell grade I and lowering to at least one ordinal category on the Quinnell scale. A bivariate association analysis was performed between the independent demographic, clinical and baseline ultrasound variables. A predictive (forward Wald) modelling approach was planned in which the variables that reached statistical significance $P < .10$ were included.

This study was approved by the Research Ethics Committee of our centre on 5th June 2019 (EXP 126/19).

Results

The study included 74 patients. Three patients rejected the therapeutic approach with infiltrations, 2 did not return for a check-up and one was referred for surgery with a Quinnell IV. Forty-eight patients (64.8%) came from a musculoskeletal practice, 15 (20.2%) from a general orthopaedic and trauma practice and 11 (14.8%) from a rheumatology practice. Mean ± standard deviation of the 68 patients who participated in the study was 59.08 ± 6.91 years. Forty (58.8%) were men. Twenty-six patients (38.2%) were classified as Quinnell II and 42 (61.8%) as Quinnell III. Table 1 summarises the participant group characteristics according to the Quinnell scale medical symptom grading.

Eight weeks after infiltration, 22 patients (32.4%) achieved a Quinnell grade I and 50 (73.5%) recorded an ordinal reduction of their clinical scale of at least one grade.

In the bivariate correlation study with complete resolution (Quinnell I), tendon thickening was detected in 18/22 patients (81.8%) who achieved this objective and in 13/46 (28.3%) who did not achieve the primary objective (OR: 1.43; CI 95%: 3.24-40.24; $P < .001$). Progression time of patients who achieved clinical resolution was 11.5 ± 3.56 months and 8.86 ± 3.41 of those who did not achieve the primary objective (t = 2.92; $P = .005$). In the bivariate correlation study with partial resolution (reduction of a Quinnell grade), tendon thickening was detected in 28/50 patients (56%) who achieved this objective and in 3/18 (16.7%) who did not (OR: 6.36; 95% CI: 1,63-24,78; $P = .005$). Progression time in the group who achieved partial resolution was 10.36 ± 3.54 and in the group who did not achieve it this was 7.94 ± 3.45 (t = 2.49; $P = .015$). The other variables were not statistically significant. Table 2 summarises the results of the bivariate correlation studies.

For binomial logistic modelling, the tendon thickening variables (HR: 10.72; 95% CI: 2.88-39.93; $P < .001$) and progression time (HR: 1.23; 95% CI: 1.02-1.49; $P = .027$) were included in the predictive model of treatment success (primary objective: Quinnell I). For the partial success model (reduction by one Quinnell grade), the variables which were statistically significant within the model were tendon thickening (HR: 5.57; 95% CI: 1.38-22.41; $P = .016$) and progression time (HR: 1.18; 95% CI: .99-1.41; $P = .051$). No multicollinearity was identified between the variable time of progression and tendon thickening.

Regarding adverse events reported by patients, these were presented by 6 individuals over follow-up: 4 cases of pain and functional limitation, which required anti-inflammatory treatment and which in all cases was limited to under 2 of the first weeks, one case of vagal decompensation without loss of consciousness during the procedure and one case of local erythema which persisted for 72 h before subsiding without the need for any treatment. No infections, tendon ruptures or hyperglycaemic decompensations were reported.

Discussion

The detection of tendon thickening at pulley level is the most important factor in the outcome of trigger finger infiltrations with corticosteroids. The chronic nature of the process also appears to lead to favourable infiltration outcomes.

This study was not exempt from limitations. On the one hand, we exclusively focused on the diagnosis of A1 pulley dependent trigger finger which —although the most common— does not represent all cases. The A1 pulley is larger and easier to identify by ultrasound. This case selection homogenized the type of condition studied. Another limitation was that the infiltration was ultrasound guided to guarantee administration of the treatment in the location recommended by the literature. However, this type of approach is not the one most commonly used in standard clinical practice. For this reason, the effectiveness described in our results could be higher than that observed in settings where infiltration was performed using only anatomical references. Notwithstanding, ultrasound guidance is relevant in putting treatment at pulley height without demonstrating that intra-synovial administration really is more effective than extra-synovial[9]. Finally, our study did not cover recurrence as an outcome variable. Other studies report that trigger fingers resolved with infiltrations may recur in 30% of cases after 6 months and in 65% after a year[10]. The population included in our study had a low representation of patients with immune mediated rheumatological diseases. This was due to asymmetry in the proportion of patients who came from the 3 different practices involved in this study.

The flexor canal, where the obstruction in patients with trigger finger originated, comprises the pulley and the flexor tendon apparatus. In our study we did not identify that the thickening of the pulley was relevant in infiltration effectiveness. Two explanations may be given for this phenomenon: firstly, that the increase in thickness was produced at the expense of the collagen fibres which were most distant from the flexor canal. Secondly, that the thickening of the pulleys was in keeping with a non-inflammatory adaptive phenomenon and is therefore not affected by corticosteroid exposure. This second hypothesis is supported by the absence of inflammatory findings in the histopathology of the studied pulleys of patients with trigger finger[7]. Permeability of the tendinous synovium and the similar efficacy outcomes in comparative studies between the administration with or without ultrasound guidance support both hypotheses[9,12]. However, it has been demonstrated that in the tenosynovial tissue of specimens obtained from patients with trigger finger there is inflammatory infiltration, neovascularisation and synovial hyperplasia. Although the most prevalent histological finding is the infiltrate of CD44+ hyaluronic acid-producing chondrocytoid cells[3], these findings would account for the greater efficacy of infiltration in patients with tendon rather than pulley thickening.

To a lesser extent, progression time is another predictor of infiltration treatment success. The longer the progression time the more probable it is that the patient achieves complete or partial

Table 2
Results from bivariate analysis between the objective variables and the independent variables.

	Complete resolution			Partial resolution		
	Achieved	Not achieved	*P* value	Achieved	Not achieved	*P* value
Age (years)	57 ± 7.49	60.08 ± 6.46	.085	59.06 ± .7	59.16 ± 6.66	.956
Progression time	11.5 ± 3.56	8.86 ± 3.41	.005	10.36 ± 3.54	7.94 ± 3.45	.015
Male sex	13 (32.5%)	27 (67.5%)	1.00	29 (72.5%)	11 (27.5%)	1.00
Diabetes	3 (16.7%)	15 (83.3%)	.143	11 (61.1%)	7 (38.9%)	.143
Hypothyroidism	3 (21.4%)	11 (78.6%)	.523	12 (85.7%)	2 (14.3%)	.323
Dominant hand affected	15 (28.8%)	37 (71.2%)	.36	38 (73.1%)	14 (26.9%)	1.00
Affected finger			.763			.716
First	1 (50%)	1 (50%)		2 (100%)	0	
Second	3 (27.3%)	8 (72.7%)		7 (63.6%)	4 (36.4%)	
Third	10 (38.5%)	16 (61.5%)		19 (73.1%)	7 (26.9%)	
Fourth	8 (27.6%)	21 (72.4%)		22 (75.9%)	7 (24.1%)	
Tendon thickening	18 (58.1%)	13 (41.9%)	< .001	28 (90.3%)	3 (9.75)	.005
Pulley thickening	13 (36.1%)	23 (63.9%)	.605	30 (83.3%)	6 (16.7%)	.061

resolution. Theoretically this observation seems difficult to explain, but if we go back to the recognized histological findings in the trigger finger, it may be interpreted that the first episode to take place is the thickening of the pulley (non inflammatory process, probably induced by the physiological adaption of repeated action) and later, the tendon response with the chondrocytoid and inflammatory infiltrate, which is susceptible to the anti-inflammatory treatment.

There are no existing studies that identify patients who would benefit from treatment with corticosteroids. Considering the greatest weakness of the use of this technique is the risk of recurrences, the identification of tendon thickening would mean that patients who would benefit from infiltration could be selected. Further longitudinal studies could back up whether this finding would also prevent long-term recurrences.

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Conflict of interests

The authors have no conflict of interests to declare with this study.

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