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Proteomic profiling reveals a distinct inflammatory microenvironment in trigger finger

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Proteomic profiling reveals a distinct inflammatory microenvironment in trigger finger

Abstract

Trigger finger is a common hand disorder, but the contribution of local inflammation to its pathophysiology remains unclear. This study investigated the inflammatory protein profile of the tendon–pulley apparatus in patients with trigger finger, with particular attention to mediators associated with atopic inflammation. Biopsies were obtained during surgery from the A1 pulley and flexor tendon of affected and adjacent asymptomatic fingers in 16 patients. Sixty-two tissue samples were analyzed using a multiplex panel of 71 cytokines, chemokines, and growth factors. Multivariate analysis demonstrated a distinct inflammatory protein profile in A1 pulleys from trigger fingers compared with controls. The levels of eleven cytokines had the capacity of discriminating between trigger and control pulleys according to multivariate analysis, including several mediators associated with leukocyte recruitment, fibrosis, and atopic inflammation. No significant multivariate separation was observed in tendon samples. These findings demonstrate a localized inflammatory microenvironment in the A1 pulley of trigger fingers and suggest that inflammatory, fibrotic, and atopy-associated pathways may contribute to disease pathophysiology. This molecular characterization may provide a basis for further molecular studies and development of targeted therapeutic approaches.

Introduction

Trigger finger is one of the most common hand disorders and is characterized by painful snapping or locking of the affected finger during flexion or extension. Biomechanically it is caused by a thickening of the first annular ligament (the A1 pulley) and impaired tendon gliding. In advanced cases, the affected finger may become locked in a flexed position. Trigger finger affects approximately 2% of the population and represents one of the leading causes for which patients consult healthcare professionals regarding hand-related concerns [1].

The exact pathophysiology leading to trigger finger is still unclear. Several mechanisms have been proposed which can explain different aspects of the clinical picture associated with trigger finger, although none of those are able to clarify every clinical characteristic of this condition. Various risk factors have been identified such as diabetes and rheumatic disease. There is also an association to carpal tunnel syndrome [2].

Trigger finger is a clinical condition which can be considered as a part of a family of disorders called “tendovaginitis stenosans”. De Quervain’s tenosynovitis and extensor pollicis longus stenosing tenosynovitis are examples of other tendinopathies in this family. As the name implies, there is a mismatch between the pulley and the tendon itself. Therefore, stenosing tendovaginitis may represent a common pathological outcome arising from multiple pathways with different etiologies.

Although trigger finger has been referred to as an inflammatory condition, the nature of inflammation has not been well described. Many authors even question inflammation, and instead stress degenerative factors and mechanical wear, as main pathogenic factors [3-6]. Further, it is unknown how the tendon and its pulley differ in their inflammatory responses [4]. The aim of this study was to describe the differences in composition of inflammatory biomarkers in trigger finger. The use of proteomics methodology in this study enables analysis of the actual, functioning molecules in the tissue.

A recent study on outcome of trigger finger surgery showed an association between atopic diseases and poor outcomes [7]. Therefore, factors of importance for atopic reactions such as eotaxins were therefore of special interest.

Methodology

In this cross-sectional single-center study, 16 consecutive patients older than 18 years of age who needed surgery for trigger finger were included. Exclusion criteria comprised concomitant Dupuytren's contracture, and steroid injection to the hand performed within three months before the operation.

A hand surgeon evaluated trigger finger patients referred to the clinic, and those assessed as requiring trigger surgery were invited to participate in the study. At surgery, biopsies were taken from the A1 pulley and tendon of the trigger finger and from the adjacent asymptomatic finger. The latter biopsies served as controls. Written informed consent was obtained from all patients. All methods were performed in accordance

105 with the relevant guidelines and regulations, and the study was approved by Uppsala
106 Regional Research Ethics Board (approval number Dnr 2012/386).
107
108 **Surgical Technique**
109 Under local infiltration anesthesia and tourniquet control, a transverse incision was
110 made just distal to the distal palmar crease of the trigger finger, and extended 1-1,5 cm
111 over the adjacent, asymptomatic control finger. The first annular ligament, the A1
112 pulley, was exposed by blunt dissection and released with a longitudinal incision.
113
114 A longitudinal biopsy was obtained from the A1 pulley, and a typical biopsy strip was 10
115 mm long and 2 mm wide. A similar strip was excised from the flexor digitorum
116 superficialis tendon at the level of the A1 pulley.
117
118 A total of 20 biopsies were taken from pullies from trigger fingers along with 17 control
119 biopsies from asymptomatic fingers. There were 14 tendon biopsies taken from trigger
120 fingers and 11 from controls.
121
122 **Sample preparation and biochemical analysis**
123 The frozen biopsy samples were heat stabilized using Denator Stabilizer T1 (Denator,
124 Gothenburg, Sweden) at quick frozen mode as described by Olausson et al. 2015 [8]. The
125 biopsy materials were transferred to a tube and 300 µl of PBS buffer solution including
126 0.1% Tween® were added to each sample. The biopsies were homogenized using
127 TissueLyser III Bead Mill Homogenizer (QIAGEN, Germany). Samples were then stored
128 at 4°C for two hours, and the homogenate was centrifuged at 16000 g at 4°C for 30

minutes. The supernatant was removed to a new tube, and total protein concentrations were measured using Quant kit (GE Healthcare, Little Chalfont, UK) according to the manufacturer's recommendation. Analyses of inflammatory proteins (cytokines, chemokines, and growth factors) were performed using the multiplex immunoassay technology Meso Scale (MSD) (Maryland, USA) which is an electro-chemo-luminescence method. The commercially available multiplex panel of 71 inflammatory substances was used as previously described [9]. Data was collected and analyzed using MESO QUICKPLEX SQ 120 instrument equipped with DISCOVERY WORKBENCH® analysis software (Meso Scale Diagnostics, Rockville, MD, USA).

Statistics

Descriptive analyses included means and ranges for age, body mass index (BMI), numeric rating scale (NRS) and Disabilities of the Arm, Shoulder and Hand questionnaire (DASH) scores.

Multivariate Data Analysis (MVDA) was chosen since it allows extracting information from data with multiple variables by using all the variables simultaneously. MVDA is the recommended method in omics that can handle large numbers of variables and small numbers of observations (short and wide data tables) [10]. MVDA was performed using SIMCA-P + v.17.0 (Sartorius, Umeå, Sweden), following the recommendations by Wheelock and Wheelock and as described by Gerdle et. al. [11,12]. First principal component analysis (PCA) was used as an unsupervised method for an overview of all data and to detect the presence of any outliers. Then Orthogonal Partial Least Squares Discriminant Analysis (OPLS-DA) was used to analyze differences between the groups (trigger fingers vs controls) based on the 71 inflammatory proteins. The validity of OPLS-

DA model was assessed using cross-validated analysis of variance (CV-ANOVA). An OPLS-DA model with p-value < 0.05 for the CV-ANOVA was considered as significant. Other model diagnostic parameters include also R^2 and Q^2. R^2 describes the goodness of fit – the fraction of sum of squares of all the variables explained by a principal component. Q^2 describes the goodness of prediction – the fraction of the total variation of the variables that can be predicted by a principal component using cross-validation methods. The R^2 value should not be higher than Q^2 and the difference should be less than 0.3, otherwise it is overfitted and not robust. Proteins with a variable of importance for projection (VIP) higher than 1.0 and with the predictive, correlation scaled loading component (p(corr)) higher than 0.3 were considered as significant proteins that contribute to the differences between the groups.

Paired T-test was also performed but no correction for multiple comparisons was made.

Results

The study group consisted of 4 male and 12 female patients with a mean age of 60 at the time of surgery. The patient characteristics are summarized in Table 1.

Eleven cytokines (MCP-1, MIP-5, MCP-2, IL-15, GRO-α, TRAIL, Eotaxin-2, IL-17B, MIP-1β, CTACK, and M-CSF) had the capacity to discriminate trigger from control samples of pulleys in OPLS-DA analysis (VIP > 1.0 and p(corr) > 0.7), (Figure 1 and Table 3). There was no significant OPLS-DA model regarding the tendons.

In univariate analysis 17 cytokines were significantly altered in pulley samples, and five in tendon samples (Table 4). Out of these MIP-3β, Eotaxin-1, TPO and CTAK were also significantly elevated in tendons, and MCP-4 was only elevated in the tendons.

Out of the 11 cytokines with the capacity to discriminate trigger- from control-samples, seven also differed significantly in the univariate, paired analysis. Out of these, MCP1 was both the cytokine that best distinguished between the groups and the one that showed the greatest difference compared to the controls (Table 3 and 4).

Discussion

Multivariate proteomic analysis demonstrated a clear separation between pulley samples from trigger fingers and adjacent asymptomatic fingers. This separation was driven by a distinct chemokine profile characterized by increased levels of various inflammatory and atopy-associated mediators. Several of the discriminating cytokines are known to promote inflammation and fibrosis, indicating that a localized inflammatory microenvironment may coexist with changes of mechanical wear and degeneration in trigger finger.

Histopathological changes in A1 pulley specimens taken from patients with trigger fingers are described in the literature in detail. The number of chondrocytes and adjacent extracellular matrix was found to be significantly increased in trigger digits compared to controls [13]. Another study concluded that the histologic abnormalities observed in the A1 pulley of trigger digits are characteristic and not related to inflammation. Sbernardori and Bandiera demonstrated that the innermost layer of A1

pulley specimens consists of irregular connective tissue, characterized by fine collagen fibers and a substantial presence of extracellular matrix. There was also a significant amount of chondroid metaplasia found in this layer [6].

In our study, biopsies from the tendon-pulley apparatus of affected fingers contained higher concentrations of inflammatory cytokines than those from adjacent control fingers. The cytokines identified by proteomic analysis as discriminating between trigger fingers and controls (MCP-1, MIP-5, MCP-2, IL-15, GRO-α, TRAIL, Eotaxin-2, IL-17B, MIP-1β, CTACK, and M-CSF) may be associated with histologic changes reported in previous studies. These findings do not contradict previous histological studies describing degenerative or metaplastic changes in the A1 pulley. Rather, they suggest that such structural alterations may coexist with or be driven by a low-grade, localized inflammatory microenvironment not readily detected by routine histology.

OPLS-DA analysis failed to detect discriminating cytokine levels of trigger and control tendons. This may be attributable to the relatively small number of biopsy samples, resulting in limited statistical power. However, univariate analysis of the tendon data revealed significant differences in five proteins. Four of these, MIP-3β, Eotaxin-1, TPO, and CTAK, showed changes consistent with those observed in the pulley samples. The fifth protein, MCP-4, also fits well within the overall pattern of inflammatory and atopy-related signaling identified in the pulley tissue.

MCP-1 is strongly pro-fibrotic. Its role in several conditions arising from fibrosis such as renal fibrosis is well established [14,15]. It is also known that micro-trauma from repetitive

mechanical load on tenocytes increases expression and secretion of MCP-1, also known as CCL2 (C-C motif ligand 2) [16]. MCP-1 attracts monocytes to loading sites and activates resident macrophages initiating an inflammatory response. It contributes to the pathogenesis of atopic asthma and eczema by promoting inflammatory cell recruitment and activating downstream signaling [17,18]. However, because of its cross-sectional design, the present study cannot establish whether increased MCP-1 is a cause or consequence of the local tissue changes.

GRO-alpha can induce hypertrophy through CXCR2 (C-X-C chemokine receptor 2) found on chondrocytes [19]. Although it has mostly catabolic effects on chondrocytes, it may play a role in changing the composition of chondrocytes seen histologically in the A1 pulleys.

MIP5 belongs to the CC chemokine family that is also known as Chemokine (C-C motif) ligand 15 (CCL15). It attracts neutrophils, monocytes, and lymphocytes by binding to chemokine receptors such as CCR1 and CCR3 on cell surfaces. Patients with severe asthma have higher levels of CCL15 in their blood, and treatment with omalizumab, a humanized anti-IgE antibody, has been shown to decrease these levels [20,21]. Bronchial biopsy samples from individuals with moderate-to-severe asthma showed increased CCL15 mRNA expression [22].

MCP-2 (CCL8) is also important in controlling allergic inflammation by influencing Th2 responses and attracting immune cells. In allergic conditions like asthma, allergic

247 rhinitis, and atopic dermatitis, the CCL8–CCR8 interaction in mice leads to an increase
248 in eosinophils, mast cells, and Th2 lymphocytes within affected tissues [23].
249
250 CTACK, also referred to as CCL27, is a chemokine that is primarily produced by
251 keratinocytes in the skin. The concentration of CTACK is found to be elevated
252 significantly in children with atopic dermatitis than in healthy controls [24].
253
254 The Eotaxin gene family (eotaxin, eotaxin-2, and eotaxin-3) is involved in recruiting
255 eosinophils, basophils, and Th2 lymphocytes, which play a key role in allergic disease.
256 These agents function via CCR3 and exhibit strong eosinophil chemoattractant activity
257 in both vitro settings and animal models. CCR3 has been implicated in several fibrotic
258 diseases, and multiple chemokines, including MCP-2 (CCL8) and eotaxin 2 (CCL24),
259 both identified in this study as elevated, are capable of activating CCR3 [25].
260
261 The overall pattern of proteomics of the biopsies shows a significant difference
262 between trigger fingers and controls in multivariate analysis (Figure1, Table 3). Our
263 results show a tissue specific elevation of chemokines promoting inflammation and
264 fibrosis. This reinforces the view that inflammatory and fibrotic components are
265 involved in the pathophysiology of trigger finger.
266
267 A major strength of this study is the within-subject design, allowing direct comparison
268 between affected and unaffected tendon-pulley tissues under identical systemic
269 conditions. By minimizing inter-individual variability, this approach enhances

confidence that the observed differences reflect local pathological processes rather than patient-specific factors.

This study has several limitations. The sample size is modest, reflecting the invasive nature of tissue sampling and the within-subject design. No functional assays or immunohistochemical localization were performed. The cross-sectional study design with patients having established disease cannot address events associated with disease induction. Yet, it provides a snapshot of the molecular conditions that sustain the disease and are relevant to the need for surgery. These factors limit causal inference but do not diminish the observed local differences between affected and unaffected tissues.

In conclusion, our study strengthens the evidence of a local inflammatory signaling in the A1 pulley and provides additional indications of atopy associated inflammatory pathways in trigger finger.

Author contribution

U.A. prepared the initial draft of the manuscript and prepared figures and tables.
B.G.H contributed to the research design, conducted the surgery, wrote parts of the manuscript and helped prepare figure 1 and tables.
M.W. provided guidance in research design, assisted in study implementation, data analysis and manuscript revision.
B.G. provided methodological support, carried out the lab analyses, performed the statistical analyses and prepared figure 1 and tables.

All authors reviewed the manuscript and approved the final submitted version.

Data availability statement

The datasets generated during the current study are available from the corresponding author on request through collaboration and after ethical approval.

Competing interests

The authors declare no competing interests.

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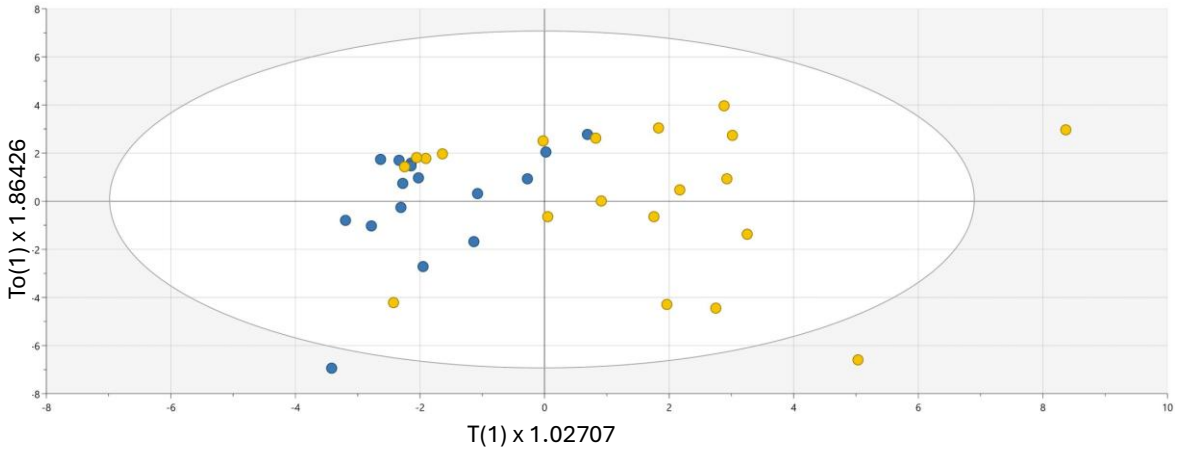


Figure 1. OPLS-DA (orthogonal partial least-discriminant analysis) score plot showing separation in proteomics between biopsies from trigger fingers (yellow circles) and control fingers (blue circles).

Table 1. Patient characteristics

Patient characteristic (n=16)	Value
Women, n (%)	12 (75%)
Age, years (mean/ range)	60 (42-78)
BMI (mean/ range)	27 (23-33)
Pain (NRS) (mean/ range)	4.6 (2-9)
DASH (n=14) (mean/ range)	27 (0.8-70.5)

DASH: Disabilities of the Arm, Shoulder and Hand questionnaire
BMI: Body mass index
NRS: Numeric rating scale

Table 2. List of cytokines in the panel used for the study

CTACK	GRO-alpha	IL-16	IL-1α	IL-31	MCP-1	MIP-3β
ENA-78	I-309	IL-17A	IL-1β	IL-33	MCP-2	MIP-5
Eotaxin	IFN-α2a	IL-17A/F	IL-2	IL-4	MCP-3	SDF-1alpha
Eotaxin-2	IFN-β	IL-17B	IL-21	IL-5	MCP-4	TARC
Eotaxin-3	IFN-γ	IL-17C	IL-22	IL-6	M-CSF	TNF-α
EPO	IL-10	IL-17D	IL-23	IL-7	MDC	TNF-β
FLT3L	IL-12/IL-23p40	IL-17E/IL-25	IL-27	IL-8	MIF	TPO
Fractalkine	IL-12p70	IL-17F	IL-29/IFN-L1	IL-9	MIP-1α	TRAIL
G-CSF	IL-13	IL-18	IL-2Ra	IP-10	MIP-1β	TSLP
GM-CSF	IL-15	IL-1RA	IL-3	I-TAC	MIP-3α	VEGF-A
						YKL-40

Table 3. Cytokines that discriminate trigger finger pulleys from controls according to Orthogonal Partial Least Squares Discriminant Analysis (OPLS-DA).

Proteins	*VIP*	*p(corr)*	*Trigger finger Median pg/ml (min-max)*	*Control finger Median pg/ml (min-max)*
MCP-1	1.36	0.92	52.35 (5.82–175.50)	14.28 (4.40-107.68)
MIP-5	1.33	0.90	131.68 (21.36-537.60)	66.04 (22.13-281.44)
MCP-2	1.32	0.89	1.60 (0.27-3.82)	0.68 (0.09-1.77)
IL-15	1.31	0.88	0.68 (0.17-7.17)	0.70 (0.09-6.02)
GRO- α	1.18	0.81	2.41 (0.58-9.54)	1.56 (0.8-6.50)
TRAIL	1.17	0.80	1.47 (0.06-53.62)	1.24 (0.01-46.17)
Eotaxin-2	1.13	0.76	38.26 (2.24-119.33)	20.23 (0.3–124.19)
IL-17B	1.11	0.66	1.41 (0.02-13.82)	0.79 (0.06-8.97)
MIP-1β	1.10	0.75	1.76 (0.09-9.97)	0.95 (0.04-4.00)
CTACK	1.09	0.74	18.48 (2.98–59.78)	11.67 (4.10-38.71)
M-CSF	1.08	0.73	4.04 (0.32-59.21)	3.19 (0.88–62.70)

The proteins are listed in descending order based on VIP (variable of importance for projection). p(corr) =correlation scaled loading component.

Table 4. Cytokines with significantly altered levels in trigger finger pulleys compared to controls, paired T-test, univariate analysis.

Cytokine	P-value	Cytokine	P-value
MCP-1	0.001	VEGF-A	0.015
MCP-2	0.001	YKL-40	0.017
Eotaxin-2	0.002	CTACK *	0.020
GRO-alpha	0.002	IP-10	0.026
MIP-5	0.003	Eotaxin *	0.031
MIP-1β	0.005	TPO *	0.038
IL-16	0.007	IL-15	0.039
TARC	0.008	MCP-3	0.046
MIP-3β *	0.011		

* Elevated in both tendons and pulleys.