Correlations Between Clinical Presentations of Adult Trigger Digits and Histologic Aspects of the A1 Pulley

K. Drossos, MD, M. Remmelink, MD, PhD, N. Nagy, MD, V. de Maertelaer, PhD, J.L. Pasteels, MD, PhD, F. Schuind, MD, PhD

Purpose We aimed to report by light microscopy the normal histology of the A1 pulley, describe the histologic abnormalities of A1 pulleys in trigger digits, and look for possible correlations between these findings and the severity of the disease.

Methods In a series of 104 trigger digits operated on in 80 adult patients, the A1 pulleys were removed and histologically studied. The findings were compared with 55 normal A1 pulleys obtained from fresh-frozen cadaveric specimens.

Results The normal A1 pulley was composed of 3 layers: layer I, an inner, avascular, concave unicellular or bicellular gliding layer containing cartilage-like cells; layer II, a middle layer, also avascular, characterized by spindle-shaped fibroblasts; and layer III, an outer, richly vascularized layer, continuous with the membranous tendons sheath. We used a 3-grade classification, increasing in severity, to describe the histologic abnormalities observed in trigger digit A1 pulleys. Mild abnormalities (grade 1) were those with a fibrocartilaginous gliding surface almost intact. The margin between the fibrocartilaginous and membranous portions of the pulley was well delineated. In moderate abnormalities (grade 2), the avascular fibrocartilaginous gliding surface appeared fissured and thinner. The inner layer (I) was interrupted and replaced by fibrous tissue, with fissures that did not cross through the middle layer (II). A mild vascular network hyperplasia was observed in the outer layer (III), which began to invade the fibrocartilage. In severe abnormalities (grade 3), the fibrocartilaginous gliding surface was thin, discontinuous, or even completely destroyed. The vascular network hyperplasia became excessive and reached the synovial space of the flexor tendon sheath. The histologic features were correlated with the severity of the clinical symptoms ($p < .001$).

Conclusions The histologic abnormalities observed in the A1 pulley of trigger digits are characteristic and not related to inflammation. As the trigger digit worsens, the gliding surface begins to wear and is gradually replaced by a secondary invasive hyperplasia from the outer layer. These abnormalities could be caused by a modification or an increase of the mechanical stresses along the flexor tendons. *(J Hand Surg 2009;34A:1429–1435. © 2009 Published by Elsevier Inc. on behalf of the American Society for Surgery of the Hand.)*

Key words Trigger digit, A1 digital pulley, light microscopy, fibrocartilage, metaplasia.

From the Department of Orthopaedics and Traumatology, Hôpital Erasme, Cliniques Universitaires de Bruxelles; and the Laboratories of Histology and Statistics, Brussels School of Medicine, and IRIBHN-SBIM, Université libre de Bruxelles, Brussels, Belgium.

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Corresponding author: F. Schuind, MD, PhD, Department of Orthopaedics and Traumatology, Hôpital Erasme, Cliniques Universitaires de Bruxelles, 808 Lennik Street, B-1070, Brussels, Belgium; e-mail: frederic.schuind@erasme.ulb.ac.be.

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Detailed anatomic studies of the annular pulley system have increased our knowledge of the critical components of the flexor apparatus,[1–4] allowing better understanding of the etiology of trigger digit. The inner aspect of the normal digital canal is not a continuous smooth surface. The thin part of the flexor tendon sheath (membranous portion[1]) overlaps the pulleys (retinacular component[1]) before attaching to their palmar surfaces, so that the pulleys demonstrate free edges within the digital canal.[5] Cohen and Kaplan[6] observed synovial cells on the inner gliding surface of the annular pulleys. In contrast, Lundborg and Myrhage[7] found no evidence of a continuous synovial cell layer on the inner gliding surface of normal pulleys. A comparative study with histology, immunohistochemistry, and transmission electron microscopy of normal and trigger digits A1 pulleys by Sampson et al.[8] reported that normal and trigger digits' A1 pulleys are composed of an outer, vascularized, convex layer and an inner, concave, flexor tendon gliding layer. In the latter, the cells and adjacent matrix have several characteristics of fibrocartilage, including the presence of chondrocytes. In trigger digits, the number of chondrocytes and adjacent extracellular matrix are significantly increased. Sampson et al. believed that the underlying pathobiological mechanism for triggering at the A1 pulley is fibrocartilage metaplasia. Sbernardori identified in trigger digits a trilayer structure, the deepest layer composed of irregular connective tissue with chondroid metaplasia, the middle layer containing dense connective tissue, and the outermost layer formed of loose connective tissue.[9] The same group performed scanning electron microscopy of the gliding surface of the A1 pulley and found alterations of the normally smooth gliding surface in trigger digits, with fragmentation and detachment of the surface layer, exposing the underlying collagen fibrils and chondroid cells.[10] However, those authors neither proposed a staging of the histologic abnormalities nor searched for possible correlations with the clinical symptoms.

The aims of the present study were to report the histology of normal A1 pulleys, describe the histologic abnormalities observed in A1 pulleys of trigger digits, propose a histologic classification of these lesions, and look for possible correlations among these histologic findings and the clinical severity of the disease.

MATERIALS AND METHODS

We obtained 55 normal A1 pulleys from all digits of 11 fresh-frozen adult cadaver hands. The proximal border of the pulley was at the metacarpophalangeal skin crease of the thumb, proximal palmar crease of the index finger, halfway between the proximal and distal palmar creases for the long finger, and distal palmar crease for the ring and little finger.[11] To recognize the distal border of the pulley, we searched for the separation between the A1 and A2 pulleys, present in 95% according to Doyle.[1] Pathological A1 pulleys were obtained from 104 trigger digits (Table 1) in 80 adult patients in whom triggering was observed preoperatively and resolved as a result of the surgery. At the time of the study, in our unit, no trigger digit was given steroid injection and all cases were treated surgically. The same surgical protocol was followed: under local anesthesia, a full diameter portion of the A1 pulley was resected and sent for histologic analysis. No specimen was obtained from the flexor tendon's surface. After resection, the patient was asked to attempt an active full flexion to confirm the resolution of triggering.

TABLE 1. Distribution of A1 Pulley Specimens by Digit

Digit	Digits (n)	%
Thumb	32	31
Index	9	9
Long	21	20
Ring	33	32
Small	9	9

The patients (41 men and 39 women) ranged in age from 18 to 79 years (mean, 56.8 ± 13.1 years). Trigger digit specimens were obtained from 71 right hands and 33 left hands. There was no correlation between affected and dominant sides (Fisher's test). The symptoms had been present for an average of 6.9 months before surgery (standard deviation, 6.4 months). In the medical history, we noted that 15 patients suffered from diabetes mellitus, 15 from carpal tunnel syndrome, 8 from Dupuytren's disease, and 3 from de Quervain's tenosynovitis. The majority (65%) were or had been heavy or light manual workers. Congenital, rheumatoid, and posttraumatic trigger digit cases were excluded.

To assess the severity of the preoperative symptoms, we used the classification proposed by Newport et al.[12] In stage I, the patients report tenderness and pain at the palmar surface of the A1 pulley without a nodule or triggering. Stage II concerns those patients who describe tenderness, swelling, or tendon nodule with associated catching or triggering during active movements. Finally, in stage III, the symptoms are equivalent to those of stage II but triggering or catch-