Editorial

The role of the A0 pulley in trigger finger

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Received: 25 January 2022; Accepted: 03 March 2022; Published: 30 March 2022.

doi: 10.21037/jxym-22-4

View this article at: https://dx.doi.org/10.21037/jxym-22-4

Trigger finger, also known as stenosing tenosynovitis, is one of the most common pathophysiologic hand conditions in the general population, affecting an estimated 2–3 percent of people worldwide (1). It may affect one or multiple digits and is found to be more common among diabetics (1). Other risk factors include trauma, carpal tunnel syndrome, gout, acromegaly, glycogen storage diseases, and rheumatoid arthritis (2). Due to the prevalence of this disease, it is important to have an appropriate understanding of the pathophysiology of trigger finger, as well as have a firm grip on effective treatment options in order to ensure the highest probability of success and patient satisfaction.

The classic pathophysiology of trigger finger is thought to be abnormal thickening or inflammation of the A1 pulley. Differences between triggering and non-triggering A1 pulleys have been demonstrated on scanning electron microscopy (SEM) and on histology (3,4). Abnormal A1 pulleys have increased type III collagen and chondrocytes with loss of extracellular matrix (3). Histologically, the A1 pulley consists of 3 layers, an outer vascularized convex layer and two inner avascular concave layers. In trigger finger, the inner fibrocartilage becomes replaced by fibrous tissue; elongated fibroblast nuclei are replaced by chondrocyte nuclei and there is vascular hyperplasia with uneven distribution of chondromyxoid matrix (4).

Treatment options for this condition have been described to include (I) conservative management with splinting, non-steroidal anti-inflammatory drugs, and avoidance of aggravating activities; (II) hand therapy; (III) corticosteroids injections; (IV) open surgical or percutaneous release of the A1 pulley; or (V) some combination of these options to stop the painful locking and catching of the flexor tendons at the affected A1 pulley.

Some patients continue to experience trigger finger despite release of the A1 pulley, as described by the authors of the manuscript under review as well as by others (5,6). Initial descriptions of the pulley system by Doyle did not discuss a palmar aponeurosis pulley, however, later work by Manske and Lesker as well as a later paper by Doyle propose the likely existence of the palmar aponeurosis pulley and acknowledge its role as a pulley of the flexor tendons akin to the numbered pulleys (7-9).

Some authors have referred to this palmar aponeurosis pulley as the "A0 pulley", as the current authors do; they have found on cadaveric dissection that the A0 pulley does not have any connections to the palmar aponeurosis and is described as an independent structure (5). Thus, there is an interest in the literature in determining whether or not the A0 pulley is the primary cause of trigger finger in some patients, whether or not it should be the only pulley released, and/or if it should be routinely released alongside the A1 pulley during the surgical treatment for trigger finger.

The authors have provided scintillating preliminary data serving as an important stepping stool in determining the importance of the A0 pulley, building off a study by Liu *et al.* that established an accurate cadaveric model for trigger finger by using such a model as the basis for its own experiments (10). The authors use two cadaveric hands, cable ties, tensiometers and simulated crimp and slope grips (simulating *in vivo* grips) to determine minimal tension normalized by circumference (mTNC) to induce triggering and work of flexion (WOF).

In this report, the mTNC for the small finger and ring finger of Hand #1 was found to be lower for the A0 pulley, whereas the reverse was true in Hand #2 for the middle finger and index finger in which the mTNC was lower for the A1 pulley. Triggering was elicited under all three FDS tensions (0, 5 and 10 N) at the mTNC for the middle finger

and index finger with A1 and A0 constriction, but triggering was not induced under 0 N at the mTNC for the small or ring finger with A0 constriction (5). There are other variable results reported regarding the WOF at the mTNC, maximal force of flexion at the mTNC and magnitude of triggering.

While this manuscript presents some interesting preliminary data, no definitive conclusions can be made as of yet as the sample size is too small and the results are too variable—both weaknesses acknowledged by the authors. For example, the authors were able to induce triggering in the ring and small finger of Hand #1 but were unable to do so in the index nor the middle finger of that same hand. In Hand #2, no experiments were performed on the ring and small fingers due to tissue degeneration. The mTNC was lower for the A0 pulley for the ring and small fingers but lower for the A1 pulley for the middle and index fingers. Increasing FDS tension was found to increase the magnitude of triggering for the small and ring fingers at the level of the A0 pulley but decreased the magnitude of triggering for the middle and index finger at the A1 pulley (5). These findings suggest that triggering at the A0 versus the A1 pulleys (or both) might depend on patient factors as well as the complex interactions of the FDS, FDP and intrinsic muscles—which could not be replicated in this model. In this case, a larger sample size would have been beneficial as would an even more accurate cadaveric model of trigger finger.

It is important to note here that most of the recent rigorous research performed and presented on the A0 pulley has been conducted by the same group or proteges herein (11,12). Past manuscripts have reported on the A0 pulley also known as the palmar aponeurosis pulley but these were not prospective, randomized studies (6-9). We ask ourselves why this might be the case?

Perhaps, the large time gap between the identification of this structure in Manske's 1975 report (7) and the interval lack of attention in the literature to the A0 pulley is suggestive that it is routinely released by surgeons and need not be further investigated? Alternatively, it might be the case that trigger finger is so rarely caused by the A0 pulley alone that these account for an insignificant slice of this pathology; or that persistence or recurrence of triggering secondary to an unreleased A0 pulley is mistakenly attributed to incomplete release of the A1 pulley; or that triggering resulting from combined A0 and A1 pulley pathology is simply under-reported in the literature. In any case, identification, proper anatomic description, and pathology attribution should be pursued if for no other reason than for completeness.

To this end, despite the limitations addressed here, we applaud the authors for using a cadaveric model to investigate the role of the A0 pulley in inducing triggering as well as for investigating the importance of differential FDS/FDP tensions. The effort to recreate *in vivo* grips is admirable. We remain hopeful that with additional research, surgeons of the hand may be able to—with more precision—accurately determine which pulley or pulleys are responsible for trigger finger, henceforth minimize morbidity and optimizing patient outcomes and satisfaction.

Acknowledgments

Funding: None.

Footnote

Provenance and Peer Review: This article was commissioned by the editorial office, *Journal of Xiangya Medicine*. The article did not undergo external peer review.

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jxym.amegroups.com/article/view/10.21037/jxym-22-4/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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doi: 10.21037/jxym-22-4
Cite this article as: Mukit M, Moss CA 3rd, Walker ME. The role of the A0 pulley in trigger finger. J Xiangya Med 2022;7:1.