









Unique skin findings in a case of the A3 pulley trigger finger due to an osteochondroma

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Trigger finger is characterized by catching or locking of the finger due to the inability of smooth movement of the flexor tendon. In most cases, trigger finger is caused by stenosing tenosynovitis and hypertrophy of the retinacular sheath.^[1] The etiology of trigger finger is usually idiopathic; however, it also occurs in patients with rheumatoid arthritis, diabetes mellitus, gout,^[2] or tumors.^[3] Furthermore, the site of tendon triggering is the A1 pulley in most cases; however, the A2 and A3 pulleys and palmar aponeurosis may also be involved.^[1]

Although A3 pulley trigger finger is rare, a few cases caused by hypertrophy of the retinacular sheath^[4] and ganglion^[5] have been reported previously. In this article, we, for the first time, report a case of the A3

ABSTRACT

Trigger finger is usually caused by stenosing tenosynovitis and hypertrophy of the retinacular sheath, and the most common site of tendon triggering is the A1 pulley. Although the A3 pulley trigger finger has been described in a few cases caused by hypertrophy of the retinacular sheath and ganglion, associated skin findings have not been reported to date. Herein, we report a rare case of the A3 pulley trigger finger due to osteochondroma with unique skin findings in a 50-year-old woman. In this case, we observed a V-shaped skin depression on the palmar side of the proximal interphalangeal joint of the right middle finger during finger locking. Additionally, we observed bilateral linear skin depressions on the sides of the proximal phalanx. These findings might be caused by the traction force on the A3 pulley, connected to the skin via the Grayson and Cleland ligaments, which are fibrous tissues that connect the skin and tendon sheath.

Keywords: Finger joint, osteochondroma, snapping fingers, trigger finger disorder.

pulley trigger finger due to osteochondroma with unique skin findings.

CASE REPORT

A 50-year-old female patient presented with an enlarged mass on the volar aspect of the proximal interphalangeal (PIP) joint of her right middle finger. She noticed the mass a year before presentation and experienced painful triggering during finger extension over the last several months. Initially, she visited the family clinic due to the increasing frequency of triggering and catching, and a tentative diagnosis of a ganglion development was made in the absence of imaging examination. The symptoms worsened and, consequently, she was referred to our department, following which skin tethering on the volar and lateral sides of the proximal phalanx was also observed.

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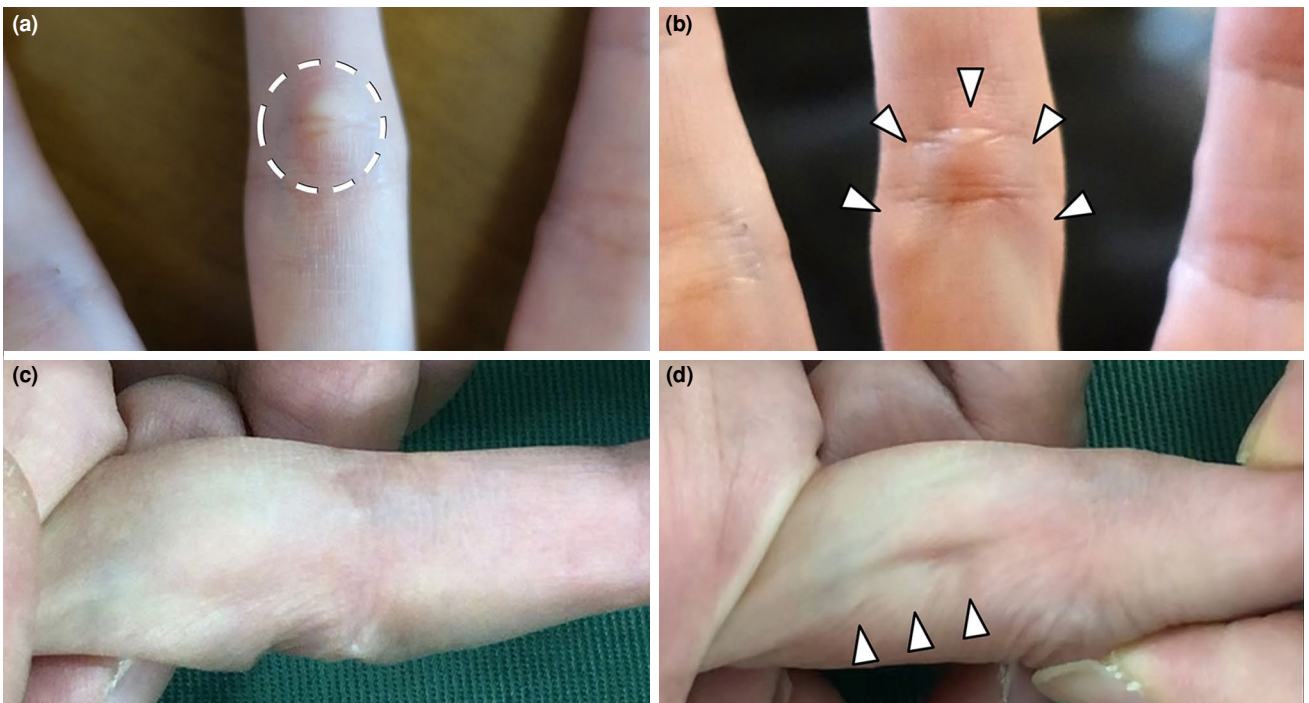


FIGURE 1. Unique skin findings upon finger locking in the A3 pulley trigger finger. (a, b) The volar side of the right middle finger. A round and hard mass measuring 2 mm is palpated around the proximal interphalangeal (PIP) crease (A, circle). During locking in finger extension, a V-shaped skin depression is seen (B, arrowheads). (c, d) The lateral side of the right middle finger. Linear skin depressions are observed on the bilateral sides of the base of the middle finger, when it is locked during finger extension (D, arrowheads).

On her physical examination, finger triggering, catching, and locking were observed during extension. A round, hard, 2-mm-wide non-tender mass was palpable on the volar side of the PIP joint of the middle finger. Skin findings showed a V-shaped skin depression on the PIP crease during finger locking (Figure 1a, b, Video 1) and linear skin depressions bilaterally on the sides of the proximal phalanx (Figure 1c, d) during finger locking. Radiography revealed a bone tumor protruding from the volar side of the base of the middle phalanx (Figure 2a). Computed tomography showed that the cortex and medulla of the tumor were in continuity with the underlying bone (Figure 2b-d). Magnetic resonance imaging findings did not show a defined cartilage cap due to the small size of the tumor.

The patient was diagnosed with trigger finger caused by an osteochondroma, and a surgical resection was planned. A Bruner incision was made around the PIP crease of the middle finger, and intraoperative findings revealed that the C1 pulley was torn and the apex of the tumor was exposed beneath the skin (Figure 3a). The apex of the tumor interfered with the A3 pulley during finger extension (Figure 3b). Interestingly, linear skin depressions on

the sides of the proximal phalanx were found even after a skin incision that prevented direct contact between the tumor and overlying skin. The bone tumor compressed the flexor digitorum profundus (FDP) tendon to the ulnar side and penetrated the radial insertion of the flexor digitorum superficialis



VIDEO 1. The lateral side of the right middle finger. The proximal interphalangeal joint is locked, and linear skin depressions are observed on the bilateral sides of the base of the right middle finger.



FIGURE 2. Osteochondroma of the middle phalanx. (a) Radiography shows a bone tumor protruding from the base of the middle phalanx (arrowhead). (b, c) Computed tomography. Sagittal (b), axial (c), and 3-dimensional image of the bone tumor (arrowhead). The cortex and medulla of the tumor are in continuity with the underlying bone.

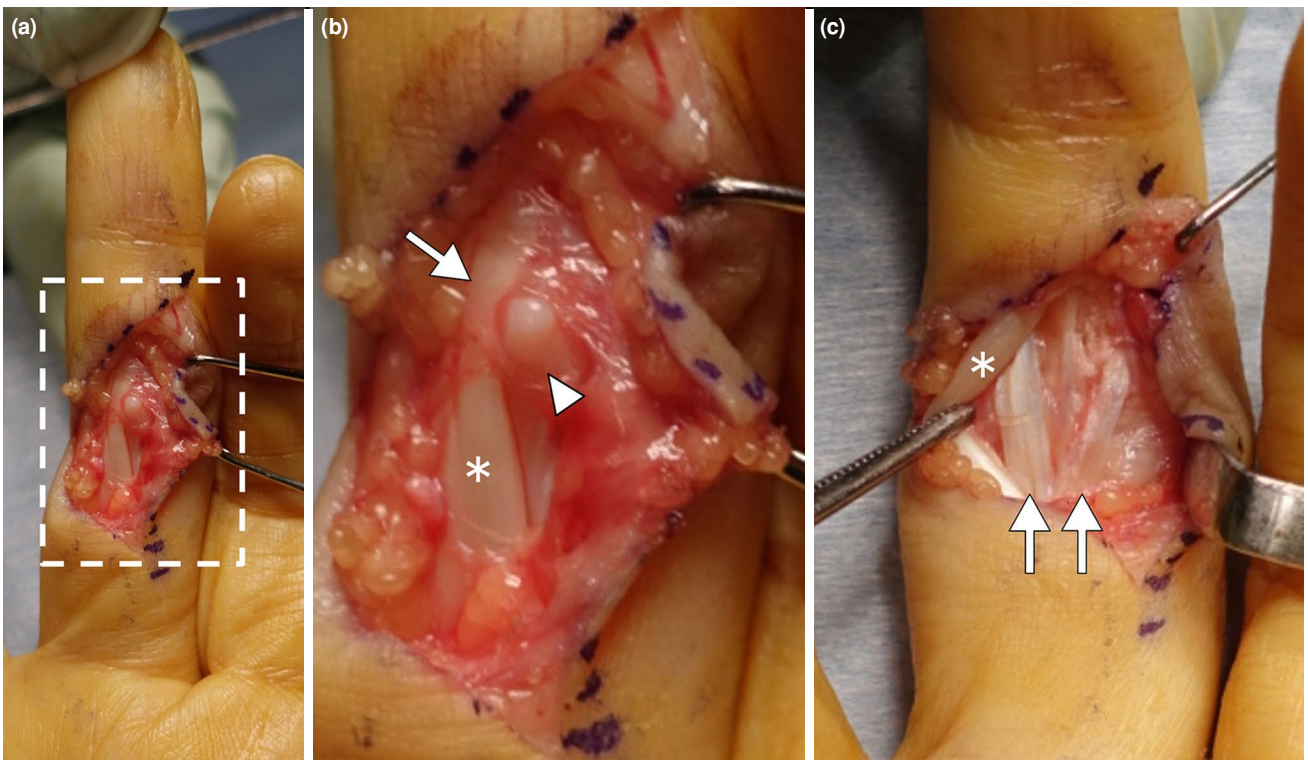


FIGURE 3. The A3 pulley tethered by the bone tumor during finger extension. (a) A Bruner incision was made around the PIP crease. The C1 pulley was torn, and the apex of the tumor was exposed just beneath the skin. (b) A magnified image of the boxed area in A. The apex of the tumor (arrowhead) interfered with the A3 pulley (arrow) during finger extension. *: Flexor digitorum profundus tendon. (c) After tumor resection. The tumor penetrates the radial side of the FDS tendon (arrow). *: Flexor digitorum profundus tendon.

PIP: Proximal interphalangeal; FDS: Flexor digitorum superficialis.

(FDS) tendon. The tendon was dissected from the base of the tumor, and the tumor was ultimately resected (Figure 3c). The FDS tendon and A3 pulley were repaired using a 5-0 nylon suture. The tumor was pathologically diagnosed as an osteochondroma. The triggering and locking phenomena disappeared after surgery, and the osteochondroma did not recur for a year.

DISCUSSION

Trigger finger usually occurs at the A1 pulley site. In idiopathic A1 pulley trigger finger, repeated friction during finger flexion-extension causes hypertrophy of the retinacular sheath and flexor tendon, which restricts the movement of the flexor tendon.^[1] The A1 pulley trigger finger is also caused by other pathological processes such as rheumatoid arthritis, diabetes mellitus, gout,^[2] and tumors.^[3] Although osteochondroma of the hand accounts for only 4% of all cases, osteochondroma at the base of the proximal phalange resulting in an A1 pulley trigger finger has been reported.^[6]

A few cases of the A3 pulley trigger finger have been reported,^[3,7,8] and the etiology of these cases was flexor tendon thickening^[4] or a ganglion.^[5] In the current patient, unique skin findings were observed upon finger locking which were not described in previous report. We observed a linear depression on both sides of the proximal phalange of the right middle finger and a V-shaped skin depression on the PIP crease.

Initially, we predicted that the skin findings were because of the direct interference between the osteochondroma and overlying skin, although we could not explain the precise mechanism of lateral linear skin depression before surgery. The involvement of the A3 pulley was indicated from the following operative findings: (i) The apex of the tumor interfered with the A3 pulley during finger extension. (ii) Even after a skin incision, which prevented direct contact between the tumor and overlying skin, lateral skin depression occurred when the finger was extended. These points support the theory that the skin findings occurred due to an interference between the tumor and A3 pulley and not between the tumor and overlying skin.

Other key structures possibly resulting in these findings are the Grayson and Cleland ligaments, which are fibrous tissues that connect the skin and tendon sheath. The ventral fibers of the Grayson ligament form a trabecular network on the ventral side of the finger, connecting the tendon sheath

with the ventral side of the skin. In addition, the Grayson ligament is transversely oriented during flexion and becomes more obliquely oriented during extension.^[9] Furthermore, the fibers are arranged in a V-shape during finger extension. Intraoperative findings indicated that a distal traction force was applied to the A3 pulley, when the top of the osteochondroma interfered with the pulley during finger extension. Thus, we assumed that the skin in contact with the Grayson ligament was also pulled distally, followed by the unique V-shaped skin tethering on the palmar aspect of the PIP joint. Similarly, the Cleland ligament and dorsal fibers of the Grayson ligament connect the tendon sheath to the bilateral sides of the skin.^[10] On retraction of the A3 pulley, the Grayson dorsal fibers and Cleland ligament are assumed to pull the skin on the lateral side of the proximal phalange. This mechanism causes a unique bilateral linear skin depression. The characteristic V-shaped and linear depressions have not been described in previous reports.^[11]

In conclusion, we encountered a rare case of the A3 pulley trigger finger due to an osteochondroma at the base of the middle phalanx. Unique skin findings on the palmar and lateral sides of the finger were observed, when the A3 pulley was distally retracted. The Grayson and Cleland ligaments are assumed to be the key structures that contribute to these unique skin findings.

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Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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