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Reference:

Favril Alexander, Vanhoenacker Filip, Goubau Yannick, Jager Tjeerd.- Camptodactyly resulting from anatomical variation of lumbrical muscles: imaging findings Skeletal radiology - ISSN 0364-2348 - 48(2019), p. 2009-2014
Full text (Publisher's DOI): https://doi.org/10.1007/S00256-019-03202-3

To cite this reference: https://hdl.handle.net/10067/1588270151162165141

Camptodactyly resulting from anatomical variation of lumbrical muscles: imaging findings

Abstract

We report three cases of camptodactyly in adolescent patients, presenting with a passive flexion deformity of the fifth finger. Ultrasound findings include aberrant lumbrical insertion and decreased lumbrical volume size, confirmed with magnetic resonance imaging, and aberrant dynamics. Surgery confirmed these findings in one patient. To the best of our knowledge, these imaging findings have not been reported previously.

Keywords: camptodactyly - lumbricals - ultrasound

Introduction

Camptodactyly is derived from a Greek term meaning "bent finger". It is a non-traumatic congenital flexion deformity, occurring in the proximal interphalangeal (PIP) joint in one or several fingers. Most commonly the fifth finger is affected. The metacarpophalangeal and distal interphalangeal (DIP) joints are usually not affected. The deformity may be flexible or fixed and can be isolated or associated with a number of syndromes. Camptodactyly is usually gradually progressive if not treated. [1]

The differential diagnosis includes locked trigger finger, Boutonnière deformity, Dupuytren disease and clinodactyly. Clinodactyly describes a nontraumatic flexion deformity with angulation in the radioulnar plane.

Unlike plain radiographic findings, resulting from long-standing flexion deformity of the PIP joint, imaging findings on ultrasound and MRI have never been reported. The purpose of this article is to present the US and MRI findings in camptodactyly attributed to an anatomical variation of the lumbricals in a series of 3 sporadic cases.

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Case series

Three adolescent patients presented with an isolated flexion deformity of the fifth finger (patient demographics, see table 1). There was no prior trauma. Physical examination revealed inability to actively extend the proximal interphalangeal (PIP) joint of the little finger (Fig. 1).

Plain radiography confirmed the flexed position of PIP 5 (Fig. 2). Ultrasound showed three similar findings in each patient. First an aberrant insertion of the lumbrical of the fifth finger was seen. In individuals without camptodactyly, the fourth

lumbrical muscle belly follows a curved course, deep and distal to the metacarpal heads (Fig. 3a). Its distal tendinous extension partly blends with the radial side of the extensor expansion of the fifth finger. In our series, a superficial distal course of the muscle belly was seen, inserting on the radial side of superficial flexor tendon (FDS) of the affected fifth finger, at the level of the fifth metacarpophalangeal joint (Fig. 3b).

Second a decreased lumbrical muscle volume size is was noted on the affected side (Fig. 4a, Fig. 4b).

Finally-dynamical ultrasound confirmed the aberrant lumbrical insertionshowed altered dynamics at the involved finger. Passive motion of the superficial flexor tendon (passive flexion at the PIP with maintained extension of DIP) caused motion of attached lumbrical muscle, whereas passive motion of the deep flexor tendon (passive flexion at DIP) did not affect motion of the lumbrical muscle (Suppl. Material 1). Passive motion of the superficial flexor tendon did not cause coordinated lumbrical muscle motion in the remaining non-affected fingers (Suppl. Material 2).

MRI was performed on 3-Tesla MRI scanner (Discovery; General Electric Medical Systems, Milwaukee, WI, USA) with dedicated coils and protocols in three planes including axial and coronal fat-suppressed (FS) T1-weighted images (WI), coronal FS T2-WI and axial, coronal and sagittal FS proton-density fast spin echo (FSE). The slice thickness was 2 mm, except for the sagittal FS proton-density FSE, which were 1 mm thick.

MRI confirmed the aberrant course, insertion (Fig. 3c, Fig. 3d) and volume size loss (Fig. 4c, Fig. 4d) of the fourth lumbrical muscle in all patients.

One patient was treated surgically. Surgery confirmed a hypotrophic lumbrical muscle inserting on the superficial flexor tendon of the fifth finger. Release of the lumbrical muscle and the checkrein ligaments was performed. Full extension was achieved and the joint was locked in extension with the use of a Kirschner wire through the PIP joint. Despite the uneventful immediate postoperative result, long-term cosmetical result was poor. Two patients were treated conservatively with serial casting which resulted in unchanged and slightly improved cosmetical result, respectively.

Discussion

Camptodactyly is a congenital flexion deformity, occurring either as a sporadic anomaly, inherited as an autosomal dominant trait with incomplete penetrance or as part of an often ill-defined malformation spectrum such as orofaciodigital syndrome, trisomy 13-15 and Jacob-Downey syndrome [2].

It usually involves the proximal interphalangeal (PIP) joint of the fifth finger, but other digits may be affected as well, with a decreasing frequency towards the radial side of the hand. The disorder is usually gradually progressive. Distal interphalangeal joint or metacarpophalangeal joint involvement suggests a posttraumatic cause rather than true camptodactyly [1]. The prevalence of camptodactyly is less than 1% [2]. It may be unilateral (33%) or bilateral (66%)

Met opmerkingen [F1]: Altijd verleden tijd.

Met opmerkingen [F2]: Beter zo past beter bij opmerking van R1.

[1]. It may be diagnosed at birth or during (pre)adolescence and can be divided into a reducible or flexible and a fixed or irreducible type. According to the age of onset, it can also be classified in three subtypes (infantile, adolescent and neonatal/syndromic) [3]. Camptodactyly is very rarely associated with significant functional deficits. It is painless and does not cause motor or sensory deficits [1].

The precise pathogenesis of camptodactyly is still a matter of debate and has been attributed to many factors [1]. These include contractures of the skin or fascia or abnormalities of volar plate or collateral ligaments. Anomalies in the collateral and other restraining ligaments have been described as a cause of camptodactyly. The flexor digitorum superficialis can be contracted, underdeveloped or devoid of normal function.

The lumbrical muscle may have an aberrant origin, from the transverse carpal ligament and/or ring flexor tendon. Aberrant insertion on the superficial flexor tendon, metacarpophalangeal joint, ring finger extensor apparatus or in the lumbrical canal or absence of lumbrical muscle have been described as well in the surgical literature [1,2].

The lumbrical muscle is thought to be the principal cause of camptodactyly. In a series of 74 consecutive operations for camptodactyly, McFarlane et al. found that the lumbrical muscle IV to the small finger had an anomalous insertion in all cases [4], although other authors have suggested that other factors may cause the deformity [5].

According to anatomical textbooks, the lumbrical muscles usually arise in the palm from the deep flexor tendons and insert on the radial sides of the extensor expansion of the second to fifth fingers, distal to the metacarpophalangeal joint level [6,7] (Fig. 5). The first and second lumbricals are unipennate and innervated by the median nerve. The third and fourth are bipennate, arising from the sides of adjacent flexor tendons, and are supplied by the ulnar nerve [7]. The lumbricals consist of a unique muscle group, connecting two extrinsic antagonistic muscles.

Most authors believe that the lumbricals act as a deflexor of the PIP joint, contributing to extension of the PIP joint. Their role in metacarpophalangeal flexion is rather limited [8].

Until present, only radiographic features of long-standing cases of camptodactyly have been described, showing changes of the PIP joint secondary to prolonged flexion deformity. No soft tissue imaging findings have been described. In our small series, ultrasound findings in camptodactyly were threefold: aberrant lumbrical insertion, volume size loss and aberrant dynamics.

In our opinion, ultrasound and plain radiographs are the imaging modalities of choice to evaluate patients with camptodactyly. Plain films are used to evaluate the degree of flexion. Ultrasound and MRI may directly visualize the aberrant lumbrical insertion,—and decreased size. The advantage of ultrasound is the ability for dynamic evaluation.

Camptodactyly is difficult to treat, and inconsistent results have been reported [1]. Conservative treatment is favored in most cases and is best for PIP contracture less than 30°. Surgical treatment is preferred in progressive/severe deformity or in case of failure of conservative treatment. Surgery includes a global approach that addresses all the potential

causes of camptodactyly, with or without tendon transfer [1]. Variable surgical techniques and disparate outcomes confuse interpretation. As the functional result of surgical correction of camptodactyly is not always satisfactory, it may be important to select those patients that may potentially benefit from surgical correction. In this regard, ultrasound may contribute to document anatomic variations in the lumbricals as causative factors in camptodactyly. More precise preoperative cartography on patients refractory to conservative treatment may allow a targeted surgical approach with smaller incision, faster recovery and possibly better functional results. However, like demonstrated in our three cases, the outcome of both conservative and surgical treatment remains relatively poor. This is in line with the literature of the topic. In a case series of 57 patients (79 fingers), Siegert et al compared the outcome of surgery and conservative treatment. Of the surgically treated patients who were available for follow-up 6 year after treatment, there were 66% poor, 16% fair, 18% good and no excellent results. In the conservative group, there were 15% poor, 20% fair, 65% good and no excellent results.

Although this report of abnormal ultrasound and MRI findings in camptodactyly in a small series may not have a clear direct effect-impact on the choice of the treatment option, on the ultimate prognosis and outcome of treatment, we believe that further documentation of abnormal imaging findings will be helpful to unravel the complex anatomy and pathogenesis of camptodactyly.

For definite conclusions, further studies -however- are mandatory in a larger series of patients.

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Met opmerkingen [F3]: Wil je alle referenteis nog eens extra goed nakijken?

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Supplementary Material

Suppl. Material 1 16-year-old male with camptodactyly, oblique longitudinal probe position. With the passive flexion of the PIP joint, the superficial flexor tendon of the fifth finger (yellow arrows) moves. Moving along with the tendon, we notice the passive motion of the fourth lumbrical muscle (red arrow).

Suppl. Material 2 13-year-old female with camptodactyly, ultrasound of the non-affected finger. Passive motion of the superficial flexor tendon (yellow arrow) does not cause coordinated lumbrical muscle (red arrow) motion.

Suppl. Material 3 Permission to reprint from Sobotta Atlas of Human Anatomy, 14th Edition.

Figure Legends

Fig. 1 13-year-old female with camptodactyly: clinical picture.

Fig. 2 The flexed position of the fifth PIP joint is illustrated on plain radiography in a 16-year-old male with camptodactyly.

Fig. 3 Aberrant lumbrical insertion: 13-year-old female with camptodactyly, oblique longitudinal ultrasound of the fifth finger (a, b). On the unaffected side (a), the lumbrical muscle of the fifth finger (red arrow) distally curves towards dorsal where its distal tendinous extension blends with the radial extensor apparatus at the level of the proximal phalanx (not seen). On the affected side (b) a straight and superficial course of the lumbrical muscle is seen, and the muscle inserts on the superficial flexor tendon (yellow arrow). MC5 = fifth metacarpal. P1= proximal phalanx fifth finger.

Axial (c) proton-density fast spin echo (FSE) fat-suppressed MRI of the affected hand at the level of the fifth metacarpal head shows a superficial position of the lumbrical of the fifth finger (red arrow), closely related to and partially covering the superficial flexor tendon (yellow arrow). The fourth lumbrical has a large contact surface with the adjacent flexor tendon. The third lumbrical (blue arrow) is more deeply positioned. Axial (d) proton-density fast spin echo (FSE) fat-suppressed MRI of the unaffected side at the same level as (c) shows the fourth lumbrical muscle coursing deep between metacarpal 4 and 5 towards the extensor expansion (not seen).

Fig. 4 15-year-old male with camptodactyly of unknown cause (no aberrant lumbrical insertion, not included in table 1). Decreased size of the fourth lumbrical muscle. 16-year-old male with camptodactyly.

Axial (a) and longitudinal (b) ultrasound shows an important decrease in muscle size of the fourth lumbrical (red arrows) on the affected side (images on the left). Note the adjacent flexor tendon (yellow arrows). On the affected side, the lumbrical lies close to and has a large contact surface with the fourth lumbrical. Axial proton density fat-suppressed MRI shows small volume size of the fourth lumbrical muscle (red arrow) on the affected side (d).

Fig. 5 Normal lumbrical anatomy. Note the bipennate fourth lumbrical, inserting on the extensor expansion of the fifth finger (red arrow). Image from Sobotta Atlas of Human Anatomy, 14th Edition [6]. With permission to reprint (Suppl. Material 3).

Figures



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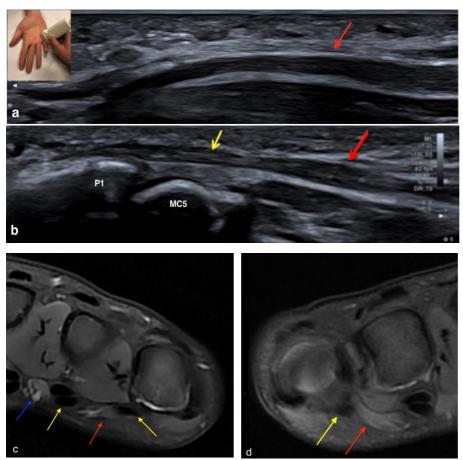


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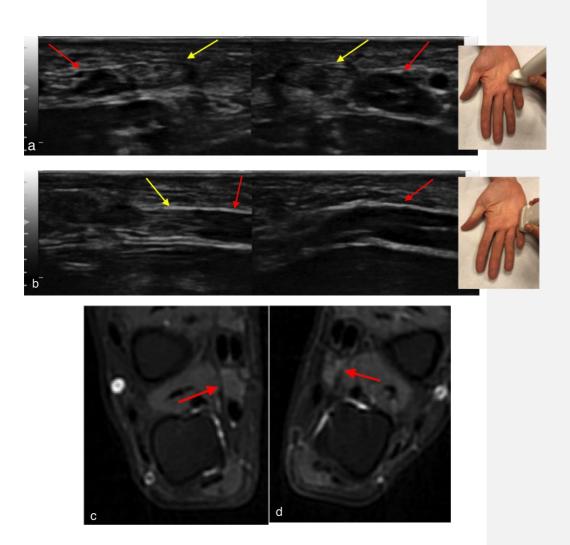


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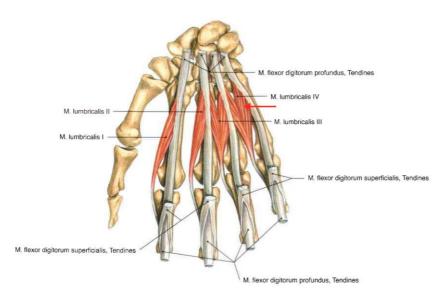


Fig. 5 Normal lumbrical anatomy. Note the bipennate fourth lumbrical, inserting on the extensor expansion of the fifth finger (red arrow). Image from Sobotta Atlas of Human Anatomy, 14^{th} Edition [6]. With permission to reprint (Suppl. Material 3).