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CASE REPORT

Trigger finger in a hereditary multiple exostoses disease: A unique case report

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Abstract

Trigger finger is one of the most common upper extremity problems seen by hand surgeons. Lesions occupying space in the tendon bed can prevent tendon gliding. We describe a unique case of trigger finger in a patient known for a hereditary multiple exostoses disease where an exostosis in the tendon bed constricted the tendon sheath, leading to triggering and locking. Open surgical treatment was performed by removing of the exostosis which relieved the problem.
Level of Evidence: Level V, therapeutic study.

Keywords Trigger finger · Hand surgery · Hereditary multiple exostoses

Introduction

Trigger finger (TF) is one of the most common upper extremity problems seen by hand surgeons. The majority of triggering is due to the disproportionate size of the flexor and its overlying pulley at the level of the metacarpal head [1]. The lifetime risk of TF development is between 2 and 3% but increases to up to 10% in diabetic patients [1, 2]. Lesions occupying space in the tendon bed can prevent tendon gliding and cause TF. We present a unique case of trigger finger in a patient known for a hereditary multiple exostoses (HME) disease where an exostosis in the tendon bed constricted the tendon sheath, leading to triggering and locking. HME is an autosomal dominant disorder that affects 1 out of 50,000 individuals in the general Caucasian population [3] with males being more affected than females [4]. We describe our case and discuss the management of TF in patients with HME.

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Case report

A 64-year-old man with a known multiple exostoses disease presented for plastic surgery consultation after experiencing persistent pain on the volar aspect of the proximal phalanx and intermittent triggering of the left middle finger for several months. He had consulted his family doctor who concluded the diagnosis of tenosynovitis and intermittent triggering of the left middle finger.

Clinical examination of the left middle finger revealed tenderness along the flexor tendon sheath with a palpable and painful nodule in the region of the A1 pulley. Mobilization of the finger revealed significant triggering.

Ultrasonography (US) showed tenosynovitis of the flexor tendon sheath of the left middle finger with thickening of the A1 pulley and an exostosis engaging with the flexor tendon at the proximal part of the first phalanx of the same finger (Fig. 1). X-ray examination confirmed the diagnosis of exostoses, showing a bony irregularity over the volar aspect of the proximal phalanx in the lateral view (Fig. 2).

Open surgical treatment was performed under locoregional anesthesia in order to release the flexor tendon with the incision of the A1 pulley and resect the exostosis. The flexor tendon had a frayed aspect which suggests a risk of tendon rupture if the surgery was not performed (Figs. 3 and 4).

Histology of the excised tissue confirmed the presence of exostosis. The postoperative course was uneventful. At 1 week after the operation, at which time the stitches were removed, the patient experienced no triggering during mobilization. At

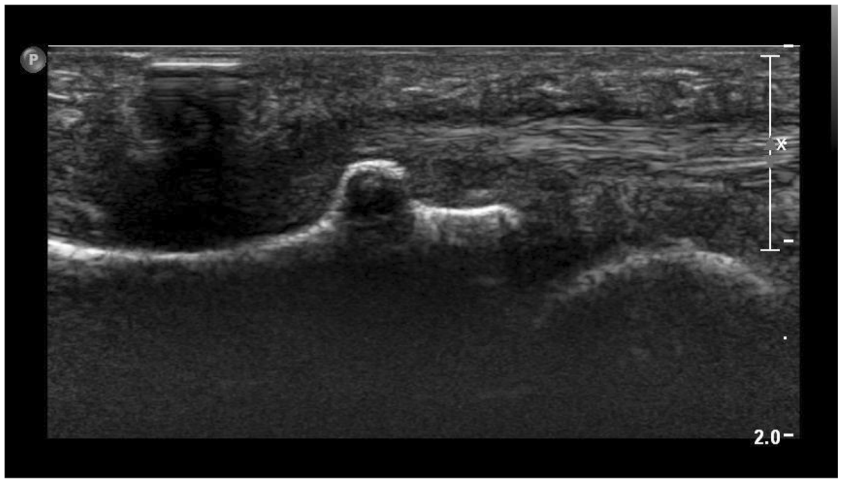

Fig. 1 Ultrasonography (US) in front of the A1 pulley of the third finger (left hand) showing tenosynovitis of the flexor tendon sheath and an exostosis engaging with the flexor tendon at the proximal part of the first phalanx of the same finger

6-week follow-up, the scar was flat and soft, while the operated finger showed a complete range of motion without triggering.

Discussion

Trigger finger (TF) is one of the most common upper extremity problems seen by hand surgeons. The lifetime risk of TF development is between 2 and 3% but increases to up to 10% in diabetic patients [1, 2]. We present a unique case of TF in a patient known for a hereditary multiple exostoses disease where an exostosis in the tendon bed constricted the tendon sheath, leading to triggering and locking.

The majority of triggering is due to the disproportionate size of the flexor and its overlying pulley at the level of the metacarpal head [1]. Clinically, the patient may present with a digit locked in a particular position, usually in flexion, which may need gentle passive manipulation into full extension [2], as well as tenderness in the region of the A1 pulley with a possible palpable nodule [1]. In our case, the patient presented a particular form of TF due to a space-occupying lesion.

Indeed, two types of TF have been described. The primary type is the most common form of TF and is most often seen in middle-aged women rather than men [5]. The causes of primary TF are often multiple, and a precise etiology has not been elucidated [5]. However, the first annular pulley at the metacarpal head seems to be affected the most often [5], suggesting that its location is subject to the highest force and pressure gradient in both a normal and power grip [2], which thus contributes to TF. The secondary type concerns patients with chronic illnesses such as diabetes, rheumatoid arthritis, gout, renal disease, hypothyroidism, and space-occupying lesions.

Our patient developed an exostosis in the tendon bed that constricted the tendon sheath, leading to triggering and locking. In addition to exostoses, patients with space-

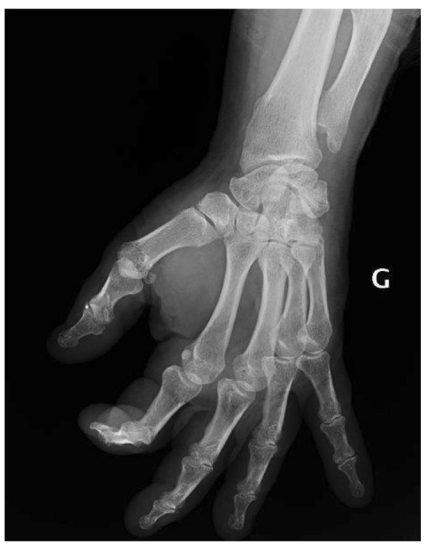

Fig. 2 X-ray examination: Lateral view of the left hand showing a bony irregularity over the volar aspect of the proximal phalanx

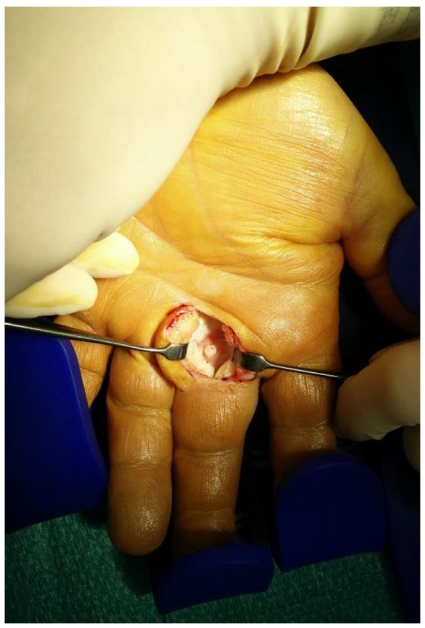

Fig. 3 Incision of the A1 pulley revealed a very damaged flexor tendon

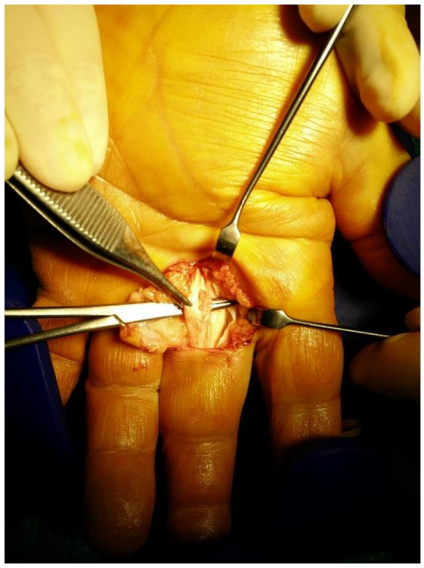

Fig. 4 Skin and tendon was raised to expose the underlying exostoses arising from the volar aspect of the neck of the proximal phalanx

occupying lesions could be affected by intratendinous granulation tissue, amyloid deposits, tendon tumors, tendon sheath tumors, post-traumatic capsule, fractures, cartilage lesions, lipoma, turret exostosis, and so on.

Usually, clinical examination is sufficient to diagnose TF, but given our patient's particular presentation with known hereditary multiple exostoses (HME) and the failure of conservative treatment, we decided to perform radiological testing. Unlike the common form of TF, X-ray examinations may be necessary to obtain complementary diagnosis information in the case of suspected space-occupying lesions [1, 5]. However, with HME, the radiological appearance may not be obvious in routine X-ray examinations. A small bony protuberance can only be visualized on true lateral view of the digit, while computed tomography scans can provide a better estimation of the lesion size [1]. Moreover, these examinations allow us to differentiate "classical" exostosis (osteochondroma) from turret exostosis, which is an ossifying hematoma. Unlike osteochondroma, the separation between the lesion and the cortex of the underlying phalanx is clear in the case of turret exostosis. Histological analysis also allows us to differentiate between these two types of exostoses: on the one hand, osteochondroma with the typical histological aspect presenting a central spongy bone core surrounded by immature cortical bone with a cap of hyaline cartilage associated with endochondral ossification, calcification, and continuity with the cortex as well as an additional cartilage cap [1], and on the other hand, turret exostoses that appear as cancellous bone with cortical bone without continuity with the adjacent cortex [6].

In our case, exostosis leads to symptomatology of TF. Lee et al. [1] already described TF secondary to exostoses of the hand, but without a history of HME. HME is an autosomal dominant disorder that affects 1 out of 50,000 individuals in the general Caucasian population [3] with males being more affected than females [4]. It is a benign condition with multiple bony tumors with cartilage caps (osteochondromas), mainly presenting in the long and flat bones [3].

Hand involvement in HME varies from 0.7 [4] to 79% [7]. The common presentation of HME hand involvement is brachydactyly, phalangeal and metacarpal cone-shaped epiphyses, and clinodactyly [5]. Given the available data, TF does not appear to be a typical presentation of HME.

Our patient had not received non-invasive treatment and was immediately referred for open surgery. Although the overall complication rates of open surgery (including reflex sympathetic dystrophy, infection, stiffness, nerve transection, incision pain, flexion deformity, flexor tendon bowstringing, and recurrence) [5] may be slightly higher or equal to the percutaneous release [8], the necessity to have an open view of the patient led us to open surgery. Indeed, the choice of open surgery combined the need to both treat TF and respect the tumor. The complete excision of the lesion and overlying periosteum with careful curettage of the bed was crucial in order to avoid the high risk of local recurrence [1] and the most severe complications of HME [3]. We can add that the presence of exostosis weakened and frayed the flexor tendon which increases the risk of rupture hence the need for surgery. The prevalence of chondrosarcoma transformation in HME has been observed by many authors with wide variations reported in the different studies, partly because exostoses are essentially asymptomatic and mostly remain undiagnosed, and partly because of variations in the selection criteria, age, study group distribution, and follow-up duration [3]. The prevalence of chondrosarcoma transformation in HME ranges widely from 0.57 to 25% [9].

In conclusion, our patient presented a particular form of TF generated by an exostosis in a context of a hereditary multiple exostoses disease, which resulted in the disproportionate size of the flexor tendon and its overlying pulley due to its particular location at the level of the metacarpal head. The management of this space-occupying lesion was different than a classical TF because it was dictated by the presence of the exostosis. These elements led us to proceed to open surgery with very satisfactory results and no complications at 6 months and no recurrence at 2-year follow-up. We recommend that patients with known HME who present a TF to undergo radiological exams for an optimal management of the case.

Compliance with ethical standards

Conflict of interest C.Vrancken, Y. Farid, and R. Matasa declare that they have no conflict of interest.

Ethical approval Ethical approval was not needed for this research.

Informed consent Informed consent is not applicable for this research.

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References

1. Lee SJ, Pho RW (2005) Report of an unusual case of trigger finger secondary to phalangeal exostosis. Hand Surg 10(1):135–138
2. Akhtar S, Bradley MJ, Quinton DN, Burke FD (2005) Management and referral for trigger finger/thumb. BMJ 331(7507):30–33
3. Ohkuma R, McCarthy EF, Deune EG (2011) Hereditary multiple exostoses in the hands and fingers: early presentation and early surgical treatment in family members. Case reports. Hand (N Y) 6(2): 209–216
4. Alvarez C, Tredwell S, De Vera M, Hayden M (2006) The genotype-phenotype correlation of hereditary multiple exostoses. Clin Genet 70(2):122–130
5. Makkouk AH, Oetgen ME, Swigart CR, Dodds SD (2008) Trigger finger: etiology, evaluation, and treatment. Curr Rev Musculoskelet Med 1(2):92–96
6. Wissinger HA, McClain EJ, Boyes JH (1966) Turret exostosis. Ossifying hematoma of the phalanges. J Bone Joint Surg Am 48(1):105–110
7. Solomon LS (1974) Chondrosarcoma in hereditary multiple exostosis. Afr Med J 48(16):671–676
8. MalletL L, Munnich A, Maroteaux P, LeMerrer M (1997) Incomplete penetrance and expressivity skewing in hereditary multiple exostoses. Clin Genet 52(1):12–16
9. Jaffe HL (1958) Tumors and tumorous conditions of the bones and joints. Léa&Febiger, Philadelphia, pp P117–P141