Bilateral stenosing flexor tenosynovitis in a pediatric patient. A case report

Luis Enrique Cortés Gutierrez M.D.
Juan Daniel Arreola Pérez M.D.
Luis Arturo Rubio Arteaga M.D.
Adrian Cano Padilla M.D.
Josue Vásquez Simón M.D.
Alejandro García Peñaloza M.D.

Edo. Mex, Mexico

Case Report

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Background: Introduction: Trigger finger most commonly occurs in two age groups: children under 8 years of age and adults between 40 and 60 years. The incidence in the pediatric population is approximately 3.3 per 1,000 live births. The etiology remains unclear, with many authors proposing both hereditary and acquired causes. Approximately one-third of cases managed conservatively fail to resolve and subsequently require surgical release.

Methods: A 1-year and 5-month-old male patient with no relevant medical history. The current condition began at birth, presenting with difficulty in extending the third finger of the left hand and the third and fourth fingers of the right hand. Targeted physical examination revealed forced flexion of the third finger of the left hand with a palpable snap (+/A1 pulley); on the right hand, forced flexion of the third and fourth fingers with a similar snapping sensation (+/A1 pulley).

Results: Following an unsuccessful course of conservative treatment, the patient was scheduled for surgical intervention consisting of exploration of the flexor mechanism of the aforementioned digits.

Discussion: Unlike flexor stenosing tenosynovitis in adults, the condition is rare in children, and its pathophysiology is poorly understood. Currently, there is no consensus regarding the optimal treatment for pediatric flexor stenosing tenosynovitis. Treatment options vary and include both surgical and non-surgical approaches.

Conclusion: Flexor stenosing tenosynovitis in pediatric patients is an uncommon condition with diverse etiologies and an incompletely understood pathophysiology. A comprehensive diagnosis should consider both systemic factors and local anatomical abnormalities. While initial management is typically conservative, surgery becomes necessary when symptoms persist, potentially revealing bony anomalies beyond the involvement of the A1 pulley. Further studies are needed to establish accurate diagnostic and therapeutic protocols for the pediatric population.

Keywords: Tenosynovitis.

Stenosing tenosynovitis, commonly known as trigger finger, is the most frequent cause of wrist pain in adult patients. In the general population, its prevalence is estimated to range between 2% and 3%. It occurs most commonly in two distinct age groups: children under 8 years of age and adults between 40 and 60 years (1). In the pediatric population, the incidence is estimated at 3.3 per 1,000 live births (5). Parents typically report an initial inability to fully extend the affected finger (2).

The etiology of pediatric trigger finger remains incompletely understood. Several authors have proposed both hereditary and acquired causes (7). Regarding the hereditary pathway, approximately 29% of patients have been found to present with genetic abnormalities, most frequently mucopolysaccharidosis and other lysosomal storage diseases (4).

Alternatively, the condition may occur secondary to trauma or infections of the upper limbs, often related to habits such as thumb sucking or nail biting (3).

Pediatric trigger finger thus represents a rare clinical entity with a poorly understood pathophysiology, complicating the establishment of standardized treatment protocols. Further research and surgical case reports are required to define the most appropriate therapeutic approach in this population (6).

The diagnostic evaluation of a pediatric trigger finger should include a thorough search for underlying conditions. Standard radiography is often unremarkable, although joint abnormalities or bony masses may occasionally be detected. Ultrasonography has become a valuable tool for identifying nodules,

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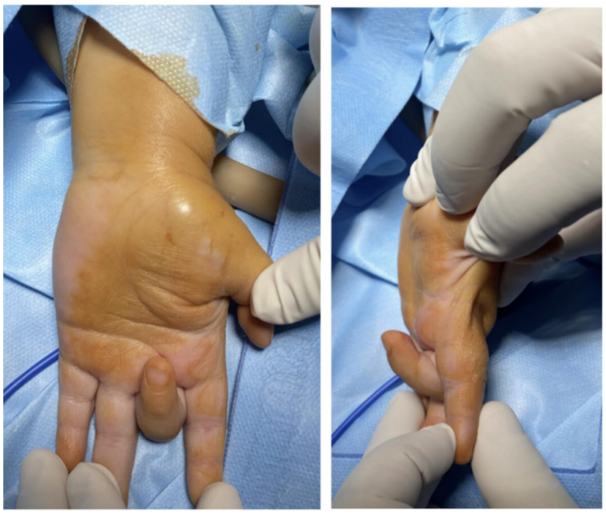

Figure 1. Preoperative image of the left hand showing flexion deformity of the third finger.

thickening of the flexor tendons, or pulley abnormalities. While magnetic resonance imaging (MRI) can provide highly detailed soft tissue characterization, its use in pediatric patients may require sedation and, in general, does not usually alter the treatment plan (4).

Several studies have analyzed the natural history of stenosing tenosynovitis in children, reporting spontaneous resolution rates of up to 65% following a follow-up period ranging from 6 to 48 months (2). However, approximately one-third of cases managed conservatively do not resolve and eventually require surgical release (4). A meta-analysis published in 2014 reported a surgical success rate of 82% (5).

Surgical intervention should begin with a longitudinal incision over the A1 pulley. Careful inspection of the flexor tendons at this level is recommended to identify the presence of nodules, abnormal gliding of the flexor digitorum superficialis (FDS), atypical lumbrical insertions, or adhesions between adjacent tendons. Any abnormal structure should be resected. If triggering persists after A1 pulley release, the incision should be extended distally to explore the A2 and A3 pulleys. In patients with mucopolysaccharidosis, initial conservative management is advised, with surgical intervention reserved for those who do not show improvement within six months (4).

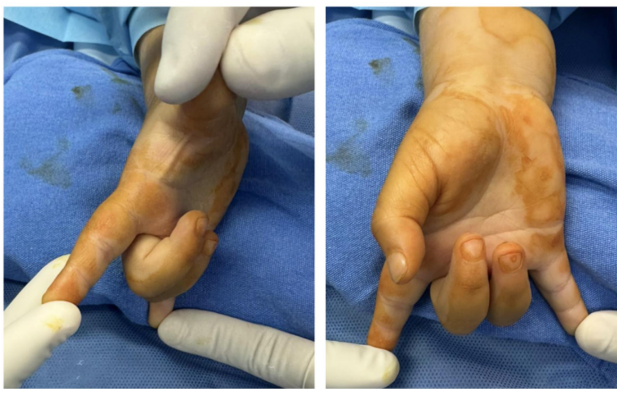

Figure 2. Preoperative image of the right hand showing flexion deformity of the third and fourth fingers.

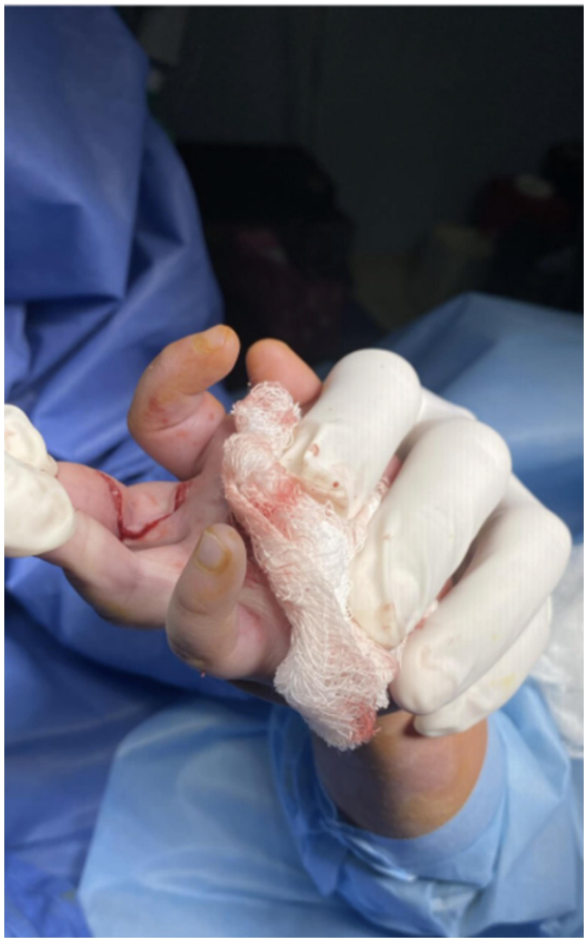

Figure 3. Intraoperative image after release of the A1 pulley of the third and fourth fingers of the right hand, demonstrating persistent flexion contracture at the proximal interphalangeal joint.

Case report

A 1-year and 5-month-old male patient was presented accompanied by his mother. An indirect interview was conducted, documenting the following history: no chronic-degenerative diseases; no known allergies; no history of blood transfusions, trauma, or substance abuse. The only relevant surgical history was congenital umbilical hernia.

The mother reported that the condition had been present since birth, characterized by an inability to fully extend the third finger of the left hand and the third and fourth fingers of the right hand.

On focused physical examination, flexor and extensor muscle tone in both forearms was preserved, with no evidence of thenar atrophy or neurovegetative symptoms. In the left hand, an abnormal digital

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Figure 4. Intraoperative fluoroscopic imaging demonstrates a wedge-shaped deformity of the articular head of the middle phalanx, ecident with the middle finger in a flexed position, leading to a loss of joint congruity.

cascade was noted, attributable to forced flexion of the third finger, along with a positive snapping phenomenon at the level of the A1 pulley. In the right hand, an abnormal digital cascade was also observed, secondary to forced flexion of the third and fourth fingers, with a similarly positive snap at the A1 pulley level.

As part of the preoperative workup, comprehensive laboratory testing was performed, including complete blood count, blood chemistry panel, and coagulation studies. The patient was also evaluated by the pediatric service at our institution, which reported no abnormalities and classified the surgical risk as low. A chest X-ray was taken on 02/06/2024; although no official radiology report was available in the system, a preliminary review revealed no osseous alterations or pathological findings in the lung parenchyma.

In addition, the patient was evaluated by the Medical Genetics Department of our institution, which ruled out any syndromic association based on the clinical features presented.

Following initial clinical and radiological evaluation (Figures 1 and 2), as well as the implementation of a conservative therapeutic trial which showed no clinical improvement, and after completing the preoperative protocol with assessment by the Pediatrics Department, the patient was scheduled for surgical intervention consisting of exploration of the flexor mechanism of the previously affected digits.

Intraoperative findings revealed the following: the A1 pulley of the flexor tendons of the third and fourth fingers of the right hand measured approximately 1 cm in diameter and 2.5 mm in thickness. The A1 pulley of the third finger of the left hand measured 1 cm in diameter and 2 mm in thickness.

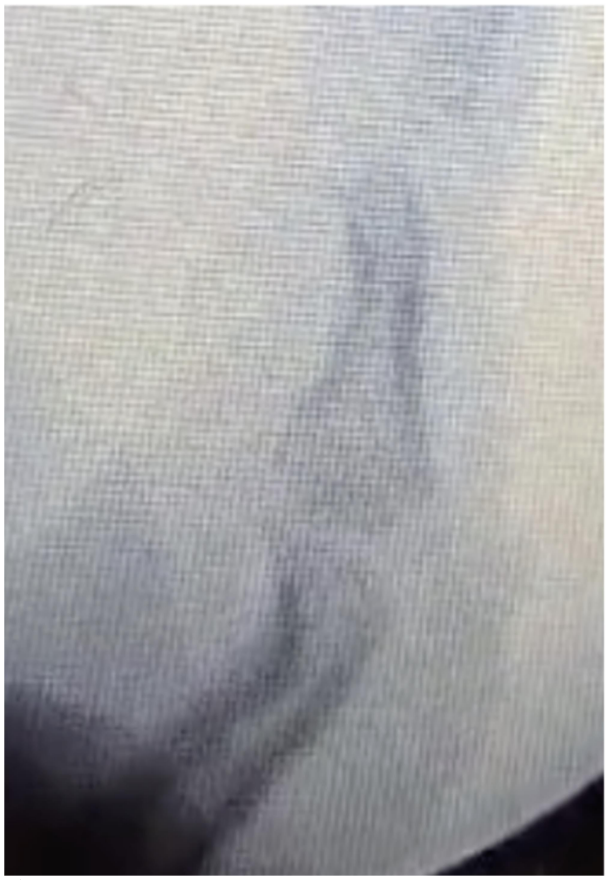

Figure 5. Intraoperative fluoroscopic imaging demonstrates a wedge-shaped deformity of the articular head of the middle phalanx, evident with the middle finger in an extended position, leading to loss of joint congruity.

After releasing the affected pulleys and confirming adequate excursion of both the flexor digitorum profundus and superficialis tendons, persistent forced flexion of approximately 60 degrees at the proximal interphalangeal joint of the third finger was observed (Figure 3).

Subsequent surgical exploration of the PIPJ of the third finger revealed a deformity of the articular head of the proximal phalanx, which was responsible for the snapping phenomenon and the extension deficit.

Intraoperative fluoroscopic imaging was obtained (Figures 4 and 5), revealing an alteration in the bony structure of the proximal phalangeal head, likely secondary to prolonged flexion associated with the chronic course of stenosing flexor pathology.

The patient demonstrated adequate postoperative progress and remains under follow-up by the Plastic Surgery Department in conjunction with the Rehabilitation Service, with the goal of achieving functional ranges of motion in both flexion and extension.

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Discussion

The exact etiology of stenosing flexor tenosynovitis in infants remains unknown. However, it may be associated with trauma, anatomical abnormalities, or underlying conditions such as mucopolysaccharidosis and diabetes (1). Unlike adult stenosing tenosynovitis, the condition is rare in children, and its pathophysiology is poorly understood (2). Despite its low incidence, numerous case reports have described pediatric trigger finger.

Currently, there is no consensus on the optimal treatment for pediatric stenosing flexor tenosynovitis, likely due to the rarity of this diagnosis in the pediatric population (3). Treatment options vary and include both surgical and non-surgical approaches. Conservative management may involve immobilization of the affected joint, use of NSAIDs to control pain, and corticosteroid injections directly into the tendon sheath (4–5).

Multiple studies included in the systematic review by Womack et al. advocate for early surgical management over conservative treatment. Of the 118 affected fingers, 54 underwent initial surgical treatment, with a successful symptom resolution rate of 87% (47/54) (3).

Conclusion

Stenosing tenosynovitis of the flexor mechanism in pediatric patients is a rare clinical entity with multifactorial etiology, and its pathophysiology presents a valuable opportunity for further research. Our case highlights the importance of a multidimensional diagnostic approach that integrates the assessment of underlying systemic diseases and local anatomical anomalies, which may be overlooked in conventional imaging studies.

While conservative management remains a reasonable initial therapeutic option, the persistence or worsening of symptoms should prompt timely surgical intervention. In this case, intraoperative identification of a structural malformation at the head of the proximal phalanx—alongside the classic A1 pulley stenosis—suggests that surgical management should aim not only at pulley release but also at systematic inspection of adjacent osseous structures. This finding underscores two key points: (1) the necessity of meticulous surgical exploration to directly confirm imaging-based preoperative findings, and (2) the importance of reassessing the relationship between the chronicity of the pathological process and subsequent articular adaptations.

These findings highlight the need for multicenter studies to better define the natural history of this condition in the pediatric population. Such evidence will be essential in developing precise management protocols, promoting individualized therapeutic strategies, optimizing resource utilization, and ultimately improving prognoses in this age group.

Conflicts of interests

None declared by the authors.

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Luis Enrique Cortés Gutierrez
Cirugía Plástica y Reconstructiva
Hospital Bicentenario de la Independencia ISSSTE
Edo. Mex. México.

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