

Paediatric calcified tendinitis in extensor carpi radialis brevis: A case report

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Abstract

Paediatric calcified tendinitis is a rare entity. Although trauma is a triggering factor, the aetiology is unknown. Conservative management is the treatment of choice with most patients experiencing symptom resolution within six weeks. We describe the case of a six-year-old male child with persistent wrist pain despite conservative treatment. Exploration revealed involvement of the extensor carpi radialis brevis, and complete recovery after surgical excision of the lesion identified on X-ray.

Keywords: Paediatric, Calcified tendinitis, Extensor carpi radialis brevis.

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Introduction

Calcified tendinitis is a very rare entity in children; only a few paediatric cases of calcified tendinitis causing triggering finger after trauma have been reported.^{1,2} Calcified tendinitis is best described as calcium hydroxyapatite deposition in tendons leading to an inflammatory condition with localised pain.² Although several theories have been proposed, the aetiology remains idiopathic. The unusual deposition of calcium in or around the hand and other sites with symptoms may mimic other inflammatory or infectious conditions leading to a misdiagnosis of such a condition. Here, we report our case of calcified tendinitis in the extensor carpi radialis brevis tendon of a six-year-old male child.

Case Report

An approval was obtained from the Royal Medical Services Human Research Ethics Committee number (11/2018) to perform the retrospective review of medical record, X-rays and histopathology report for the case.

A previously healthy six-year-old child presented with right wrist pain of one week duration following a fall six weeks earlier. Examination revealed local swelling and tenderness over the mid dorsum of the right wrist, full range of movement of all fingers with no pain, mild pain

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Figure-1: Pre-operative anteroposterior X-ray of the wrist showing a radiopaque shadow over the radial side of the wrist joint.

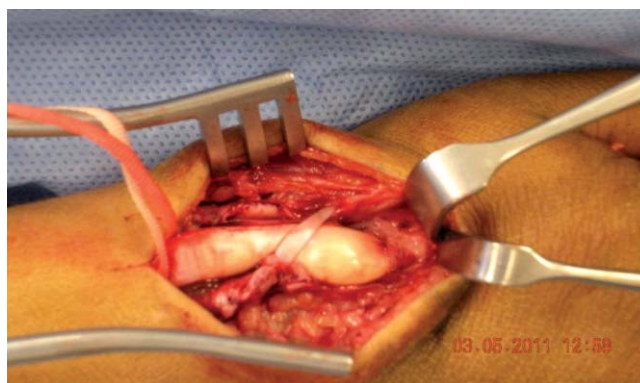


Figure-2: Intra-operative picture of the lesion seen on X-ray.

with passive and active wrist movement, no redness or local wounds, and no palpable axillary lymph nodes.

X-ray of the hand and wrist showed a tube-like calcified mass on the dorsum of the wrist (Figure-1). Blood examination showed an erythrocyte sedimentation rate of 10 mm/h, and a C-reactive protein level of 2 mg/L. From history, examination and investigation gout, pseudogout, hand space infection or septic joint were excluded.

The patient was diagnosed with acute calcific tendinitis, which was treated by splinting the wrist and non-steroidal anti-inflammatory medication was initiated. He was followed for six weeks, but there were no clinical or



Figure-3: Postoperative anteroposterior X-ray of the wrist taken 10 days after resection, confirming complete disappearance of the lesion.

radiographic changes.

With the failure of conservative treatment, a local exploratory surgery was planned. Surgical intervention was performed under general anesthesia with the use of tourniquet. Intraoperative findings showed calcification around the extensor carpi radialis brevis (Figure-2). Upon opening the sheath, a white chalk-like material exuded from the wound. Small, hard, bone-like material was observed with curettage, and the surrounding membranous tissue was removed by curette. The area was washed with saline, and the tendon was closed with continuous Vicryl sutures. Post operatively, we used tramal according to body weight for the first 24 hours followed by paracetamol suppositories 250mg twice daily. Postoperatively the wrist joint was immobilized in a neutral position by a back slab for two weeks soon after gaining consciousness the patient was encouraged to start a range of motion in the fingers. The wound with dressing was checked 48 hours post operatively.

Histopathology report described the samples as small pieces of bone and calcifying aponeurotic fibroma. The patient was free of symptoms two weeks after surgery, and a follow-up X-ray appeared normal (Figure-3).

Discussion

Calcium hydroxyapatite deposition in the tendon is termed calcific tendonitis.²⁻⁴ This condition was first described by Cohen in 1924.⁵

Most cases in the literature report acute calcific tendinitis in adults (15-57 years), with pre and perimenopausal women affected five times more frequently than men. Lesions usually affect the index finger of the dominant

hand, and the most common sites are the flexor carpi ulnaris near the pisiform, long flexor and long extensor of the finger.^{6,7}

The pathogenesis of calcification remains unknown but may be related to local hypoxia within the tendon and subsequent fibrocartilage metaplasia and dystrophic calcification deposition.⁸⁻¹⁰

Greene et al proposed that acute calcification may be similar to other crystal-induced diseases, but biochemical profiles are often normal, and hypercalcaemia is not seen in acute calcification.^{6,7,11} Association with collagen diseases such as scleroderma and dermatomyositis has been proposed, but acute calcification is not a feature of these diseases.^{12,13} This condition is often misdiagnosed as a bacterial infection, but it is well known that infection is not a cause of acute calcification.

Acute calcific tendinitis can present in one of three ways: chronic symptoms, acute inflammation, or completely asymptomatic. Rupture of the calcific deposit into adjacent soft tissue induces acute inflammation that can cause pain, oedema, and occasionally erythema.^{3,7,11}

Only a few cases of acute calcific tendinitis in paediatric patients have been reported. Seiler and Kerwin⁹ described trigger finger in a child after old trauma by a pencil, and they found a calcified mass in the flexor tendon of the third digit. Hansen and Battista¹⁴ reported a case of trigger finger after calcified tendinitis affecting the flexor digitorum superficialis tendon. Contrary to adult patients, most paediatric cases occur after trauma.

The condition is self-limiting, with most symptoms resolving in untreated patients within three weeks; calcification on X-ray tends to disappear over the course of several weeks.^{3,8,15} Immobilisation along with non-steroidal anti-inflammatory drugs may shorten the symptom duration to seven days. The most dramatic results have been observed with local injection of a mixture of steroid and local anaesthetic. Surgery is rarely indicated but may be considered if symptoms persist longer than six weeks.

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