Case Report

# Teutschländer Disease Mimicking Extraskeletal Osteochondroma- Dilemma in Diagnosis and Decision Making: A Case Report and Review of Literature

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## Abstract

Tumoral calcinosis (TC) or Teutschländer disease is a rare entity in which there is deposition of calcium salts in periarticular soft tissues. It is a benign metabolic condition of unknown aetiology. It is most commonly seen around large joints such as the hip, knee and elbow. It may be associated with calcium and phosphate metabolism disorders. Extraskeletal osteochondromas are benign lesions which are slow-growing. These can occur around the small joints of the hands and feet and cause displacement of digits. We report a case of a nine-year-old girl presenting with a recurrent swelling around the left little toe causing displacement of the toe. The clinical and radiological diagnosis suggested a possibility of extraskeletal osteochondroma, whereas the final histopathological examination (HPE) confirmed a diagnosis of Teutschländer disease or tumoral calcinosis.

**Keywords:** Tumoral calcinosis, extraskeletal osteochondroma, Teutschländer disease

## Introduction

Tumoral calcinosis or Teutschländer disease is an uncommon metabolic disorder involving the deposition of calcium salts in soft tissues.[1] It typically affects large joints such as the hip, shoulder, and elbow. [2,3] Lesions around smaller joints of the hand and foot are rarely reported.[2,4] The exact pathogenesis is incompletely understood, but it is linked to loss of local homoeostasis of calcium and phosphate metabolism. Although initially, this systemic metabolic role was refuted and it was thought to be a localised problem due to tissue injury and degeneration of bursae.[5] The idiopathic form is called primary TC which can again be divided into primary normophosphatemic and primary hyperphosphataemic subtypes. The secondary form is due to disease conditions like renal failure, parathyroid dysfunction, vitamin D toxicity etc. However, the end point of all these three types is the deposition of amorphous calcium crystals in periarticular soft tissue with identical histopathological features. Clinical examination and radiology aid in diagnosis, while histopathology confirms the diagnosis. A diagnostic dilemma arises when the lesions are either located in atypical locations or show ambiguous features on radiological examination.

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## Case Report

A 9-year-old girl was brought to the outpatient clinic by her parents with a complaint of a painless swelling on her left little toe, which had been gradually increasing in size over the past 1 year. There was a history of similar swelling 4 years ago, for which she had undergone surgical excision 2 years ago. She developed a similar swelling at the same site one year ago. There was no similar lesion elsewhere in the body. The mass initially appeared on the dorso-medial aspect of the toe but soon enlarged and extended in all directions and encircled the toe completely. As a result, the toe was deviated upwards and laterally (Figure 1) The child was unable to wear covered footwear due to the toe deformity. The skin over the mass became thinned out and eroded, leading to a small ulcer. The mass was painless and non-tender. Later on, the child complained of mild pain due to the ulcer. There was no history of similar swelling in either parent or sibling. The child did not have signs of any systemic illness. The mass was firm to hard, encircling the base of the left little toe with gross deviation of the toe outwards and laterally.

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**Figure 1:** Pre-operative image showing lesion with overlying ulcer and deviation of the left little toe outwards and upwards



**Figure 2:** X-ray image showing dense lobulated calcified lesion over the middle phalangeal region of left little toe with no clear visualization of the phalanx

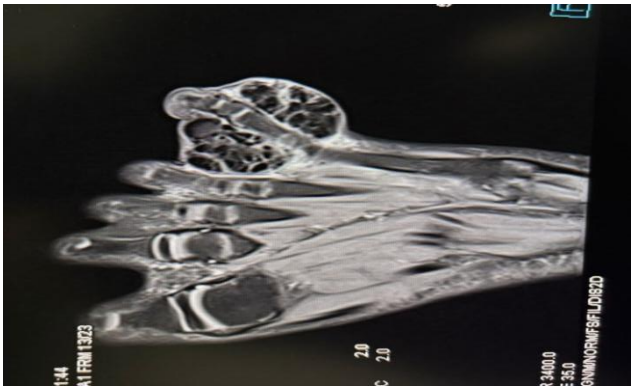


**Figure 3:** MRI coronal cuts showing 34mmx 27 mm x 32 mm irregular T1 & T2 hypointense mass encircling the left 5th toe with displacement of flexor and extensor tendons but without any changes in the underlying bone




**Figure 4:** MRI axial cuts showing 34mmx 27 mm x 32 mm irregular T1 & T2 hypointense mass encircling the left 5th toe with displacement of flexor and extensor tendons but without any changes in the underlying bone



**Figure 5:** Intra-operative image showing complete excision of the lesion with preservation of the extensor apparatus



**Figure 6:** Excised fragmented pieces of the lesion

The overlying skin was hyperpigmented with a longitudinal scar of previous surgery. A 1 cm x 1 cm ulcer was present on the lateral aspect.

## Radiographic Examination

X-ray revealed a dense lobulated calcified lesion over the middle phalangeal region of the left little toe with no clear visualization of the phalanx as such. Distal phalanx was visualised distal to the lesion (Figure 2).

MRI scan – A 34 mm x 27 mm x 32 mm irregular T1 and T2 hypointense mass was seen encircling the left 5th toe with displacement of flexor and extensor tendons but without any changes in the underlying bone. It was suggestive of extra skeletal osteochondroma. Differential diagnosis: benign ossified lesion (Figures 3 and 4).

## Incisional biopsy

An incisional biopsy was done through the previous surgical scar. It showed extensive dystrophic calcification containing numerous vascular channels. The surrounding fibrocollagenous stroma showed numerous osteoclastic giant cells with no evidence of atypia.

## Laboratory studies

All blood parameters were within normal limits except for the

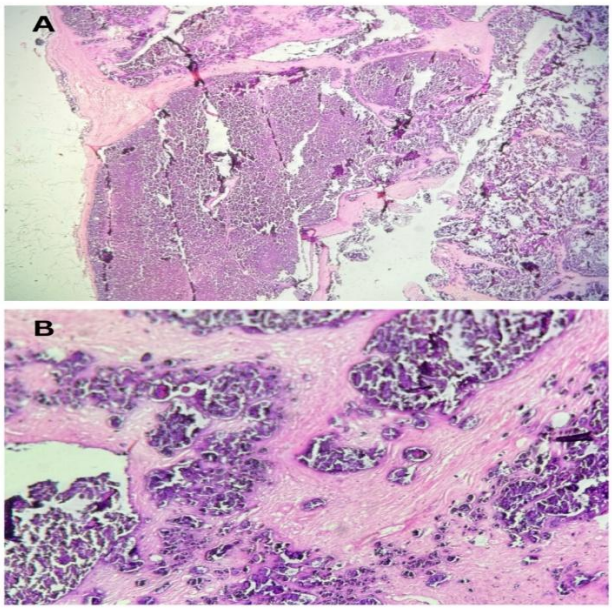



**Figure 7:** Histopathology Images-Lobules of irregular deposits of amorphous granular calcium crystals separated by collagenous stroma A. H&E 100x & B. H&E 400x



**Figure 7:** At 9 months follow up with no recurrence and correction of the deformity

phosphate level, which was marginally elevated (6.1 mmol/dl).

### Management

Given the conflicting and ambiguous diagnostic data from clinical, radiological and incisional biopsy, a decision was taken to operate and send the mass for excisional biopsy to reach a final diagnosis. Under general anaesthesia the lesion was opened through the previous surgical scar. A multilobulated mass was seen infiltrating irregularly into the soft tissue. The lobules contained cystic spaces that ruptured easily, exuding milky fluid with chalky granules. The lesion was completely encircling both digital arteries and digital nerves. Careful dissection was performed under loupe magnification, and the mass was removed piecemeal to preserve the neurovascular structures. Both tendons were found to be uninvolved (Figures 5 and 6). After a thorough wash, ensuring clearance of all particles, the incision was closed over a mini-vacuum drain. A soft padded dressing with a below-knee slab was applied for temporary immobilization. The histopathology report showed lobules of irregular deposits of amorphous granular calcium crystals separated by collagenous stroma (Figure 7).

Postoperative period was uneventful apart from some marginal discolouration of the suture line, which healed with conservative management. Weight-bearing was allowed as soon as the child was comfortable, which was about one week after surgery. She was put on a low phosphorus diet as per the direction of the dietician.

Suture removal was done at 2 weeks. The toe deformity was found to be completely reversed, and the child was able to wear closed footwear. At 9 months' follow-up she remains symptom free (Figure 8)

## Discussion

Duret was the first to describe the rare entity in 1889. German pathologist Otto Teutschlander extensively studied this pathology from 1935 onwards and hence the condition, especially the hereditary form of the disease, was named after him and was called Morbus Teutschlander across Europe. Cuban pathologist, Alberto Inclan, unaware of the European discoveries, used the term "tumoral calcinosis" for localised deposits of calcium within soft tissues that he reported in three patients.[5,6] This entity is characterised by deposition of calcium salts, mostly calcium hydroxyapatite and amorphous calcium phosphate in soft tissues around large joints with common sites around the hip, knee and elbow. Diagnostic dilemma arises when it is seen in distal parts of the appendicular skeleton such as hands and feet. In fact, very few cases have been reported in the foot.[4] The diagnosis is based on clinical, radiological and histopathological findings. It can be classified into three subtypes:

1. Primary normophosphatemic TC with no metabolic abnormalities
2. Primary hyperphosphataemic TC (familial pattern),
3. Secondary TC with co-existing disease or metabolic anomaly such as renal failure, milk-alkali syndrome, hyperparathyroidism, hypervitaminosis D and malignancy.[7]

Most common among these three is the normophosphatemic TC. Slavin et al., postulated that the mechanism behind this entity is a two-step process: a) trauma initiating a multistep response to injury in juxta-articular tissue, b) multifactorial elevation of calcium phosphate products principally due to hyperphosphataemia as the injured cells release their intracellular phosphates. This tissue injury leading to a metabolic response finally causes stereotypical deposition of calcium causing specific lesions.[8] The familial form is due to specific genetic mutations that alter phosphate metabolism. Mutations involving GALNT3 and KLOTHO genes cause a downregulation of FGF-23(Fibroblast Growth Factor – 23) which causes hyperphosphatemia by increasing gut absorption and decreasing urinary excretion of phosphate.[7]

The third variant, i.e. secondary TC, can occur due to a multitude of conditions leading to an abnormality of calcium and phosphate metabolism. Uraemia, adynamic bone disease / hypoparathyroidism, secondary hyperparathyroidism, aluminium

overload, vitamin D toxicity and malignancy with bone destruction are some of the reported causes. Baums reported a case in a patient with renal failure on long-term haemodialysis and stated that increasing the frequency of dialysis per week, keeping a relatively low dialysate calcium concentration, maintaining serum calcium level below 2.7 mmol/L and serum phosphorus below 1.5 mmol/L would be effective in control of the disease.[4]

One typical feature which differentiates TC from other disorders of calcium deposition, such as dystrophic calcification is the tendency of these lesions to grow slowly and increase in size, like a tumour.[3] The lesions enlarge because of failure to reduce the calcified burden by the compartment lining histiocytes and dysmorphic osteoclasts that are unable to solubilise the hydroxyapatite. As a result, there is continued new locule formation.[8]

In the case reported, the atypical location in the foot and characteristic toe deformity led to a diagnostic dilemma. Extra-skeletal osteochondroma, a rare benign tumour, has a very similar presentation. In fact, the MRI report in our case suggested extra-skeletal osteochondroma as a diagnosis. While osteochondromas are common benign bone tumors, the extra-skeletal form is relatively rare. Foot is the second most common site for such tumours after the hand. In fact, Kho *et al*., proposed that the finding of a discrete osseous mass in the soft tissues of the extremity should make the clinician consider extra-skeletal osteochondroma as a diagnosis.[9] Balaji *et al*., reported a case of a 9-year-old boy with exactly the same presentation as our case. The child in their case report had a slow-growing mass at the base of

the 4th toe that caused a similar type of splaying deformity of the toe. Excisional biopsy in their case proved it to be extra-skeletal osteochondroma. Although osteochondroma usually occurs in adults, rare forms occurring in children are also reported, hence should be kept in mind in atypical cases.[10]

Histopathology plays a very crucial role in diagnosis. The finding of variously sized cysts separated by thin and thick fibrous septa and containing amorphous calcium deposits surrounded by histiocytes and occasional giant cells and other inflammatory cells is characteristic of tumoral calcinosis.[7] Histopathologically, the lesions of TC are classified into three stages. Stage I or early active lesions are mostly seen in children. Surgical excision is the recommended treatment but they are prone to recurrence. Stage II is the intermediate stage with less florid cellular proliferation. Stage III is the inactive form occurring mostly in adulthood and may not require surgery unless they are causing any specific problem.[11] On the other hand, extra-skeletal osteochondroma is characterised by the presence of mature chondroid tissue with bland-looking chondrocytes. Foci of mature bone may also be seen.[9,10] In our case, it was the typical gross finding of cysts containing thick milky and chalky components and the typical histopathological finding that clinched the diagnosis.

## Conclusion

Tumoral calcinosis is a rare disorder but should be kept in mind for calcified lesions in different body parts, even in atypical locations like the hand and feet. Clinical and radiological findings may not always corroborate and it is the final histopathology that gives a proper diagnosis.

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### Conflicts of interest

None to declare

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