

CASE REPORT

DYSTROPHIC CALCINOSIS IN A CHILD WITH A THUMB SUCKING HABIT: CASE REPORT

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We present an uncommon case of a 3-year-old boy with a finger sucking habit who developed dystrophic calcification in his left thumb. Two years after excision, there was no recurrence, and the thumb retained full range of motion. We also discuss its probable pathogenesis and present a brief review of the literature about orthopedic complications in the hand due to this habit.

DESCRIPTORS: Calcinosis. Hand. Finger. Child. Thumb sucking.

Thumb and finger sucking is a common phenomenon in the pediatric age group¹ and represents the earliest form of habitual manipulation of the body². It is usually associated with oral pleasure and self-comforting behavior, persisting approximately into the fifth year³. Most of the normally developing children lose interest in the habit, with spontaneous disappearance well before school age. Intermittent persistence is frequent at bedtime, during illness, and in times of emotional tension¹. Prolonged compulsive activity may indicate the need for psychological support. Vigorous attempts to break a pre-school child of this habit are not recommended, in despite of complications such as dental malocclusions, finger deformities, and chronic paronychia⁴. We describe here an uncommon dystrophic calcinosis from a thumb sucking habit.

CASE REPORT

A 3-year-old white male was brought to our service in January 1997 by his mother who was concerned about the swollen aspect of her infant's left thumb. She emphasized that the child had been sucking his thumb since birth.

Examination showed a large, tender periarticular mass just on the volar side of the first metacarpophalangeal joint (Fig. 1). The boy was healthy, and laboratory data, including serum calcium (9.3 mg/100 ml) and serum phosphorus (4.8 mg/100 ml), were all normal.

Radiographs of the hand confirmed a multilobulated calcific mass in this area (Fig. 2).

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Excision of the calcific mass was carried out. Upon operation, the mass was whitish, pasty, and chalky, with no true lining capsule.

Microscopically, the mass was a dystrophic calcific deposit, with fibrous connective tissue and foreign-body giant cells (Langhans cells).

The wound healed uneventfully, and in a couple of weeks there was no more tenderness. The patient regained full function of the thumb in 3 weeks. Follow-up 2 years later revealed no recurrence, and the thumb retained full range of motion (Figs. 3-4).

DISCUSSION

Hand complications in children from digital sucking have been reported^{3,5-10}. Langford¹¹ first described finger sucking as a harmless habit.



Figure 1 - Periarticular tumor near the metacarpophalangeal joint of the thumb.



Figure 3 - Aspect of the thumb 2 years after excision of tumor.



Figure 2 - Radiograph showing calcific mass in thumb area.



Figure 4 - Radiographs showing no more calcinosis 2 years after excision of tumor.

Nowadays, it is well known that finger sucking can cause palatal, dental, and occlusional deformities¹². Less frequently, finger deformities, paronychia, and even oral hemorrhage have been reported³⁻¹⁰. All deformities occurred in index fingers. Broadway and Orht⁶ in 1956 described the backward displacement of the proximal interphalangeal joints of 2 fingers resulting from digital sucking. Lloyd-Roberts⁷ presented a case of index finger deviation and rotation toward the radial side, treated with osteotomy. Reid and Price⁹ had 3 similar cases and 2 additional ones

with hyperextension of the proximal interphalangeal joints. Rankin et al.³ reported 3 cases of index fingers with radial deviation deformity. In the same year, Blöem et al.⁵ described 5 cases of rotational deformity of the index finger caused by reversed finger sucking. Rayan and Turner⁸ reported 2 digital deformities and 3 thumbnail infections. Stone and Mullins¹⁰ considered thumb sucking to be the most primary predisposing factor for chronic paronychia in children. Phelan et al.⁴ published an article about an unusual complication from thumb sucking. It was a severe

oral hemorrhagic ulceration under the tongue caused by thumb tip pressure directly over the mucosal lesion on the floor of the mouth. This development led to shock and required emergency hospital treatment.

We report the first description of dystrophic calcinosis due to thumb sucking. Chen and Eng¹³ reported a similar lesion in a thumb tip after injury during carpentry work. In 1969, Serre¹⁴ et al. reported calcinosis circumscripta in a 7-year-old boy who had such lesions in the right hand and knee.

Dystrophic calcification should be differentiated from lesions in other calcification syndromes¹⁵⁻¹⁷. A significant number and variety of disorders cause extraskeletal deposition of calcium,

Table 1 - Disorders associated with extraskeletal calcification or ossification^{17,18}.

A. Metastatic calcification	
I.	Hypercalcemia
a.	Milk-alkali syndrome
b.	Hypervitaminosis D
c.	Sarcoidosis
d.	Hyperparathyroidism
e.	Renal failure
II.	Hyperphosphatemia
a.	Tumoral calcinosis
b.	Hypoparathyroidism
c.	Pseudohypoparathyroidism
d.	Cell lysis following chemotherapy for leukemia
e.	Renal failure
III.	Crystal-Deposition Diseases
a.	Gout
b.	Calcium Pyrophosphate Deposition Disease
B. Dystrophic calcification	
I.	Calcinosis (universalis or circumscripta)
a.	Childhood dermatomyositis
b.	Scleroderma
c.	Systemic lupus erythematosus
II.	Post-traumatic
C. Ectopic ossification	
I.	Myositis ossificans (post-traumatic)
a.	Burns
b.	Surgery
c.	Neurologic injury
II.	Fibrodysplasia (myositis) ossificans progressiva

phosphate, and sodium (Table 1). In some disorders, mineral is precipitated as amorphous calcium-phosphate or as hydroxyapatite and mono-sodium urate crystals¹⁷; in others, bone tissue is formed. The pathogenesis of the ectopic mineralization in these conditions is generally attributed to one of three mechanisms. First, a supranormal calcium-phosphate and crystal solubility product in extracellular fluid can cause metastatic calcification. Alternatively, mineral may be deposited as dystrophic calcification into metabolically impaired or dead tissue despite normal serum levels of calcium and phosphate. Third, true bone formation occurs ectopically in a few disorders in which the pathogenesis is poorly understood¹⁸.

Injured tissue of any kind is predisposed to dystrophic calcification^{13,19}. Apparently, such tissue can release material that has nucleating properties. The local factor that predisposes the precipitation of salts is unknown^{13,17}. Indeed, several mechanisms seem likely. It is clear that mineral precipitation into injured tissue is even more striking and more severe when either the extracellular calcium or phosphate level is increased^{16,18}. Pathologic calcification usually is initiated by the biological membranes of mitochondria or matrix vesicles through the interaction of phosphate enzymes with calcium-binding phospholipids. Hydroxyapatite crystals are formed first within the protective microenvironment of the membrane microspace^{19,20}.

The term “calcinosis” refers to an important type of dystrophic calcification that commonly occurs in, or under, the skin in connective tissue disorders (dermatomyositis, scleroderma, and systemic lupus erythematosus). Other etiologies for calcinosis include metastases and trauma that produce necrotic tissue. Calcinosis may involve a relatively localized area with small deposits in the skin and subcutaneous tissues, especially over the extensor aspects of the joints and the fingertips (calcinosis circumscripta); or it may be widespread, and not only in the skin and subcutaneous tissues, but also deeper in periarticular regions and areas of trauma¹⁷⁻¹⁸. The lesions of calcinosis are small or medium-sized hard nodules that can cause muscle atrophy and contractures¹⁸.

We attribute the development of dystrophic calcification in our case to repetitive trauma by thumb sucking, producing areas of abraded tissue and leading to deposition of circulating calcium salts forming calcific mass.

In cases of surgically accessible dystrophic calcinosis, surgical excision of the lesion may achieve satisfactory symptom relief and cure.

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RESUMO

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Os autores apresentam caso incomum de uma criança de três anos de idade com o hábito de chupar o dedo que desenvolveu calcinose distrófica no polegar esquerdo. Dois anos após a ressecção cirúrgica, não ocorreu recidiva e o polegar mantém todos os movi-

mentos. Discutem, ainda, sua provável patogênese e fazem breve revisão da literatura a respeito das complicações ortopédicas na mão devido a este hábito.

DESCRITORES: Calcinose. Mão. Dedo. Criança. Chupar o Dedo.

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