Mirror image calcified lesions of the finger tip: Two case reports

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AA Waitzman, A Freiberg. Mirror image calcified lesions of the finger tip: Two case reports. *Can J Plast Surg* 1993;1(1):52-54. Calcified lesions in the pulp of the finger tip are quite rare. Two case reports of mirror image lesions are presented with clinical, radiological and tissue pathology evaluation. All laboratory parameters were normal, there were no systemic signs or symptoms, and no evidence of crystal disease, neoplasm or previous trauma. These lesions may represent dystrophic calcification or tumoral calcinosis, but the etiology is far from clear. Excision and closure under local anesthesia was successful in managing these lesions.

Key Words: *Calcified, Finger tip, Hand Surgery, Tumour*

Image en miroir de lésions calcifiées des extrémités digitales: deux rapports de cas.

RÉSUMÉ: Les lésions calcifiées au niveau de la pulpe de l'extrémité du doigt sont plutôt rares. Deux rapports de cas de lésions présentant l'aspect d'une image en miroir sont illustrés ici avec une évaluation clinique, radiologique et histologique. Tous les paramètres de laboratoire se sont révélés normaux. Il n'y avait aucun signe ou symptôme systémique ni manifestation de maladie du cristal, de néoplasie ou de traumatisme antérieur. Ces lésions pourraient représenter une calcification dystrophique ou une calcinose tumorale, mais l'étiologie est loin d'être résolue. L'exclusion et la fermeture sous anesthésie locale ont traité ces lésions avec succès.

Calcified lesions of the finger tip are a rare finding. Recently two patients presented to the plastic and hand surgery service at the Toronto Hospital, Western Division, for evaluation of such a lesion.

A 63-year-old woman was referred to the hand surgery clinic for evaluation of a mass in the pulp of her left long finger tip in July 1989. The mass had been present for three years, was nontender, and had slowly enlarged (Figure 1). She was otherwise well and on no medication. There was no history of trauma or infection, and no history of rheumatological conditions such as gout, pseudogout or collagen vascular disease involving either the patient or her family. She had no systemic signs or symptoms of a rheumatic disorder. All laboratory parameters including serum calcium, magnesium, phosphate, urate, creatinine, alkaline phosphatase and total protein were within normal limits. Plain x-rays of the digit revealed trabeculated calcification in the pulp of the third distal phalanx (Figure 2).

The patient was taken to minor surgery and the mass explored and excised under digital block anesthesia. A white-tan mass was dissected off the periosteum and nail bed (Figure 3). There was no capsule or pseudocapsule. The defect was closed with a local V to Y advancement flap (Figure 4).

Figure 1 Left long finger of patient in case 1. Volar (left) and lateral views (right)

Figure 2 X-ray of left long finger of patient in case 1. Anterior-posterior (left), oblique (middle) and lateral views (right)

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Figure 3) Left long finger of patient in case 1. Intraoperative view after excision of lesion

Figure 4) Left long finger of patient in case 1. Closure of defect with V to Y flap (left). Healed incision with no recurrence two-and-a-half years after surgery, volar (middle) and lateral views (right)

Figure 5) Right long finger of patient in case 1. Volar view

Figure 6) Left thumb of patient in case 2. Volar (left) and lateral x-rays (right)

The pathology report described a calcified mass $0.6 \times 0.4 \times 0.3$ cm. The microscopic findings were dystrophic calcification with a foreign body-type granulomatous reaction. There were no crystals seen. The pathological diagnosis was calcinosis, etiology not apparent.

The wound healed well with no recurrence. However, two-and-a-half years later, the patient presented with a mass in the pulp of the right long finger tip (Figure 5). The mass had been present for six months and was identical to the previous lesion on the opposite hand. All laboratory parameters tested above were again normal. The radiologist's report from plain x-ray films described a calcification of soft tissue at the distal end of the distal phalanx of the right long finger, query calcinosis or dystrophic calcification.

Under digital block anesthesia the right long finger was explored and again a white-tan mass was resected from the pulp. The pathology report described a subcutaneous calcified nodule measuring $0.9 \times 0.6 \times 0.4$ cm with no crystals seen. There has been no recurrence in either of the operated fingers up to present time (September 1992).

A 67-year-old woman was referred to the hand surgery clinic for evaluation of a hard painful mass in the pulp of the left thumb. The mass resembled a gouty tophus (Figure 6). The patient was otherwise well and on no medication. She had no signs or symptoms of a rheumatic disorder, and there was no history of gout or any other rheumatic disease involving the patient or her family. Laboratory investigations including serum calcium, magnesium, phosphate, total protein, urate, cholesterol and triglycerides were within normal limits.

Under digital block anesthesia the left thumb was explored. A hard yellow-white mass was excised in toto. The wound was closed directly. The pathology report described a $1.3 \times 1.4 \times 0.7$ cm mass with dense collagenous fibrosis of the dermis. There were microcalcifications and some areas contained foreign body-type multinucleated giant cells. There were no crystals seen.

The wound healed normally with no local recurrence; however, in early 1992 a similar lesion began to form in the pulp of the right thumb. At the time of report it is being simply observed as it is asymptomatic.

DISCUSSION

Abnormal deposits of calcium may be categorized on an etiological basis. Metastatic calcification may develop when there is an abnormality of calcium or phosphate regulation, or both. Examples of this include hyperparathyroidism, renal failure, milk-alkali (Burnett's) syndrome, vitamin D toxicity and sarcoidosis. In these conditions internal organs are more commonly involved than the skin and subcutaneous tissues. Metabolic calcinosis results from tissue injury. This includes physical trauma, vascular insufficiency, infection or collagen vascular diseases (1). Dystrophic calcification, when calcium is deposited in dead or degenerating tissue, may be included in this category (2). Interstitial calcinosis consists of calcinosis universalis and circumscripta. Calcinosis circumscripta is a condition most common in middle aged women, usually associated with scleroderma or dermatomyositis. Calcium is deposited subcutaneously near joints or in flexor tendon sheaths. Calcinosis universalis may present with deposits in the skin, subcutaneous tissue or muscle. Radiologically the calcification is of homogenous density, and aggregates of foreign body giant cell surrounded by fibrosis are seen microscopically. It is a rare disease of children and young adults with no known etiology (1,2).

A tophus containing calcified material may be seen in gout and pseudogout, with the presence of appropriate crystals seen on microscopy (3-6). Tumours, both benign and malignant, or cysts may also calcify. In these cases the diagnosis should be apparent from microscopic examination of the tissue or capsule. Finally, tumoral calcinosis is a rare condition usually reported in black children. In one-third of cases there is hyperphosphatemia, and in one third a familial pattern is present. The calcium deposits are juxta-articular and may be very large; however, skin involvement is unusual (1,2,7,8). Typically, large joints are involved, but deposits in the hand have been reported (7). The deposits are usually

encapsulated and contain multiple lobes. Microscopically, foreign body giant cells may be present (2).

In the two cases reported here, there were no abnormalities of calcium or phosphate regulation, systemic signs or symptoms, monosodium urate or calcium pyrophosphate dihydrate crystals, infectious agents, or a capsule or pseudocapsule. In each case there was a second mirror image lesion in the opposite digit. This makes the diagnosis of dystrophic calcification less likely, even if the patients failed to remember antecedent trauma. The lack of a capsule or loculations, however, make the diagnosis of tumoral calcification somewhat less likely. The clinical, radiological and histological findings are most consistent with calcinosis universalis, however, the patients' age and small number of lesions are not. The rarity of such calcified lesions in the pulp of the finger tip makes an etiological diagnosis difficult, and there will likely never be a clear answer for the two cases presented. Fortunately the strategy of local excision and closure has so far been successful in the management of these two patients.

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