CASE REPORTS

Deposition of calcium pyrophosphate dihydrate crystals in a soft tissue chondroma

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Abstract

Calcium pyrophosphate dihydrate (CPPD) crystal deposits were found in an extra-articular chondroma of the soft parts overlying the distal phalanx of the right middle finger. The lesion appeared to arise from the flexor tenosynovium. The pathogenesis of soft tissue chondroma and the relation of cartilage metaplasia to the process of CPPD crystal deposition were investigated.

Extraskeletal chondroma of the soft parts is a slowly enlarging cartilaginous tumour occurring primarily in the soft tissue of the hands and feet.^{1,2} The most common single site is the fingers and the lesion is commonly associated with periarticular structures such as the tendon, tendon sheath, or joint capsule. The deposition of calcium pyrophosphate dihydrate (CPPD) crystals occurs mainly in the joint hyaline cartilage and fibrocartilage and in articular and periarticular structures, including the synovium and joint capsule.³ It has also rarely been reported in extra-articular locations including bursae,⁴ tendons,^{5,6} subcutaneous tissue,⁷⁻¹⁰ and within the dura mater.¹¹ We report the finding of CPPD crystals in an extraskeletal calcified chondroma of the soft parts.

Patient

A 67 year old woman presented with a 14 month history of a swelling on the palmar aspect of the distal phalanx. This was a painful, firm 1.5 cm long lesion, mobile with respect to the bone.

The patient had experienced pain in the right shoulder, fingers, and lower back for 18 years, with more recent development of pain in the right elbow and right knee. There was a strong family history of osteoarthritis with associated Heberden's nodes. The patient herself had Heberden's nodes on her right index finger. Radiographic examination (fig 1) showed a partly calcified lesion which appeared to be extra-articular. Plain x rays of the lumbar spine, right shoulder, elbow, wrist, hand, and knee showed features consistent with a degenerative arthritis.

A white, calcified nodule 1.2×1×0.6 cm was found during surgery and this was removed. The lesion appeared to arise from the synovium around the flexor tendon sheath. It was not attached to the joint capsule.

Microscopically, the lesion was a well defined nodule composed largely of hyaline and fibrocartilage arranged in a lobular fashion (fig 2). These lobules were separated by cellular connective tissue and within the cartilage lobules and in the fibrous septae there were numerous and extensive deposits of basophilic calcified material. These were shown to contain abundant positively birefringent short rod-like crystals when viewed with compensated polarised light (fig 3). In addition, there was focal calcification in some of the cartilage lobules. This took the form of large areas of calcification occupying most of the centre of the cartilage lobules (fig 4) in addition to more diffuse granular deposits of calcium around chondrocytes in the cartilage matrix (fig 5). Histiocytes and giant cells were seen around the cartilage lobules and around deposits of pyrophosphate.

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Figure 1 Anteroposterior and lateral radiographs of the soft tissue chondroma of the right middle finger.

Figure 2 Whole mount section of the cut surface of the soft tissue chondroma showing numerous crystal deposits (large arrows) and lobules of cartilage, some of which are calcified (small arrows).

Figure 3 Deposit of calcium pyrophosphate dihydrate crystals viewed with polarised light. There is a surrounding fibrosis and a histiocytic and giant cell response.

Figure 4 Deposits of calcium in the vicinity of chondrocytes within the soft tissue chondroma.

Figure 5 Focal granular deposits of calcium around chondrocytes in one of the cartilage lobules.

The lesion was completely encapsulated by fibrous tissue and was seen to lie in relation to the synovium of the tendon sheath. The cartilage matrix was metachromatic with toluidine blue and there was no amyloid material seen on Congo red staining.

The lesion was diagnosed as an extraskeletal calcified chondroma of the soft parts containing deposits of CPPD.

Discussion

The clinical history of chronic arthritis and identification of CPPD crystals by compensated polarised light microscopy makes it highly probable that this patient has CPPD crystal deposition disease.¹² Although CPPD crystals have occasionally been found in periarticular sites such as tendons,^{5,6} they have not previously been recorded in a chondroma of the soft parts or any other predominantly chondroid lesion. Tumour like deposits of CPPD in the finger, however, have previously been reported.⁷⁻¹⁰ Some of these have been related to the flexor tenosynovium¹⁰ and some have shown small areas of chondroid metaplasia.⁹

The absence of CPPD deposition in cartilaginous tumours is surprising given the fact that CPPD deposition in cartilage is relatively common and that hyaline and fibrocartilage are the most common sites of CPPD crystal deposition.¹² A possible explanation for this may lie in a consideration of the nature of the lesion designated as a chondroma of the soft parts. These lesions are benign, do not metastasise and are closely related to, or appear to arise from, the synovial lining.² Histological features resembling those of a giant cell tumour of the tendon sheath (benign synovioma), a lesion thought to be of synovial origin,¹³ have previously been noted in a soft tissue chondroma.^{1,2} In the case reported here, the lesion was closely related to the flexor tenosynovium and numerous histiocytes and macrophage polykaryons were present in the lesion. It is therefore possible that the chondroid element in a soft tissue chondroma could arise by cartilaginous metaplasia in a pre-existing benign synovioma. CPPD crystals are known to be found only in the phagocytic cells of the synovial membrane.¹⁴ In this patient, where it is probable that CPPD crystal deposition disease is present, CPPD crystals may have been taken up by the mononuclear phagocytes and macrophage polykaryons within the lesion, leading to the formation of large crystalline deposits. It is also of interest that there is a report of CPPD crystal deposits in association with synovial osteochondromatosis,¹⁵ a lesion in which the chondroid element is also thought to arise by metaplasia.¹⁶ It is also possible that cartilaginous metaplasia in connective tissue could underlie the deposition of CPPD in extra-articular tissues such as the dura mater and tendon.

It could also be argued that the lesion is a cartilaginous neoplasm and that the CPPD crystals are formed by chondrocytes within the lesion. Such crystals are known to be formed by articular cartilage and fibrocartilage *in vitro*.¹⁷ The infiltration of histiocytes and macrophage

polykaryons would then be regarded as part of the normal cellular response to crystalline material and calcific deposits.

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