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Non-traumatic calcifications/ossifications of the bone surface and soft tissues of the wrist, hand and fingers: A diagnostic approach

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Abstract In the absence of obvious trauma, the calcifications/ossifications of the bone surface and soft tissues of the wrist, hand and fingers can be challenging and may not be noticed or lead to unnecessary examinations and monitoring. Although these are usually benign conditions and despite a favorable spontaneous outcome, surgical resection may be required and recurrence may occur. In practice, only paraneoplastic syndromes such as secondary hypertrophic osteoarthropathy (Pierre Marie-Bamberger syndrome) may reveal a malignant tumor, most often pulmonary. We suggest a diagnostic approach based on the initial clinical presentation (acute pain, chronic pain, growth ± pain) and the radiological features.
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Califications and ossifications that develop on the surface of bone and in the soft tissue of the wrist, hand and fingers are usually benign. In practice, only paraneoplastic syndromes such as Pierre-Marie-Bamberger syndrome may reveal a malignant tumor that is usually pulmonary and cyanotic. Primary malignant tumors in the soft or paraosteal tissues are rare in the wrist, hand and fingers. Acral metastases are rare and usually develop in advanced, previously diagnosed tumoral diseases.

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The morphological features of these calcifications are less discriminant than the underlying anatomical structure where they are located, an anatomical structure that is not specific for the causative agent. The clinical features (slow growing, swelling, acute or chronic pain) are essential diagnostic criteria (Table 1) to help determine the location and morphology of these calcifications or ossifications. In the absence of any obvious trauma, and because of the many different possible benign conditions, it is important to help the clinician define those that may require surgical management (osteocondromatosis, Nora’s lesion, vascular malformation, calcifying aponeurotic fibroma), those that may reveal a metabolic disorder or a systemic disease (calcium pyrophosphate dehydrate crystal deposition disease, gout, kidney failure, connective tissue disease, psoriatic arthritis) and those that can be treated symptomatically (hydroxyapatite crystal deposits, tendinosis).

**Context of non-traumatic acute pain**

**Hydroxyapatite crystal deposits**

Hydroxyapatite is one of the most stable forms of basic calcium phosphates (Fig. 1). It is naturally present in bone, enamel and dentin, where it is the main mineral component. An acute and solitary periarticular, peritendinous or intraarticular hydroxyapatite deposit may occur, possibly due to

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![Figure 1](image_url)  
**Figure 1.** Acute deposits of hydroxyapatite crystals in the soft tissues of three different patients: on conventional radiography at the base of P1 of the 2nd ray (arrow) (A), on axial CT scan at the distal insertion of the extensor carpi radialis longus muscle on the 2nd metacarpal base (arrow) (B), on conventional radiography (arrows) (C), ultrasound (D) and Doppler ultrasound near the triquetral bone and the distal insertion of the extensor carpi ulnaris (E). Note on ultrasound the echogenic mass with the calcium spots and no posterior shadow cone (D-star) and the Doppler hyperemia consistent with the local inflammation (E).
local hypoxia leading to crystalline precipitate. Recent, or fairly recent traumas are found in certain cases when the patient is questioned.

Theoretically this acute deposit may develop anywhere, but certain zones are more common (“disease with calcium phosphate crystals in multiple tendons”), such as the apophyseal insertion of the greater or lesser tuberosity of the humerus, the deltoid V-shaped tendinous confluence and insertion on the deltoid tuberosity of the humerus, the insertion of the gluteus maximus on the linea aspera as well as the cervical spine (periodontoid soft tissue and transverse ligament of the atlas, insertion of the long muscle of neck). The calcification may be located in the tendon, its sheath or in the adjacent serous bursa. The sudden inflammatory reaction associated with partial or total resorption of this calcium deposit is the cause of the violent clinical reaction as well as the biological inflammatory syndrome. These calcium crystal deposits may also be found in the wrist, the hand or the fingers, although this is less frequent, where they may mimic arthritis or tenosynovitis [1]. The insertion of the flexor carpi ulnaris on the pisiform bone is frequently involved in the wrist [2]. Whatever the location is, these deposits are often solitary or fragmented, fairly round or oval shaped, of irregular density, milky and without trabecular or cortical bone, differentiating them from a process of ossification. Cortical erosions may be found in contact with the deposits. Intramedullary penetration with a cancellous bone edema may be observed especially on the femoral or humeral diaphyses. The outcome is usually favorable within several days. In the hand or fingers, persistent deposits may require surgical resection because of discomfort, swelling, or impingement of adjacent anatomical structures.

**Proliferative periostitis, Fori d reactive periostitis, Nora’s Lesion, Turret exostosis**

These entities may be expressions of the same disease. They may be diverse presentations of so-called reactive periosteal lesions that are observed on radiographic imaging at different times [3]. A traumatic or microtraumatic origin of these entities is a subject of debate, because trauma is not always reported. These different lesions are usually found in the extremities and in particular the proximal phalanges of the fingers.

Florid periostitis (Fig. 2) begins with painful swelling of the soft tissues (dactylitis) associated with a periosteal reaction that progresses along the phalangeal diaphysis within several weeks. It may be circumferential and mimic an infectious (osteomyelitis) or tumoral (osteosarcoma) process in the bone [4]. The periosteal lesion is often compact and irregular with a crenelated appearance that may result in fusiform enlargement of the phalanx. Cortical erosion has occasionally been described in contact with the periosteal lesion. Unlike exostosis, the lesion progresses and produces more mature bone with no visible continuity with the medullary canal of the underlying bone. Histologically, different quantities of fibrous tissue, cartilage and an osteoid matrix are visible associated with a proliferation of fibroblast/myofibroblast cells.

Clinically, bizarre parosteal osteochondromatous proliferation or Nora’s lesion (Fig. 3) presents as a moderately painful mass that mainly affects the hand (55%), the long bones (27%) or the feet (15%). The ossified mass may have a pedunculated or sessile attachment to the underlying periosteum, with no periosteal reaction or medullary continuity. The histological analysis shows fibrous tissue, cartilage, areas of enchondral ossification and a clear cap of cartilage in certain cases. There is a high degree of cellularity with no atypia. Surgical resection is usually performed but there is a risk of recurrence.

Turret exostosis (Fig. 4) is a bone growth that develops on the surface of the bone, usually on the dorsal aspect of the proximal or middle phalanx. It is a result of a minor trauma of the deep aspect of the extensor tendon of the finger resulting in a subperiosteal hematoma, which ossifies as it progresses. [5]. Turret exostosis is attached to the periosteum with a sessile base and has a cartilage peripheral cap [6] which, for some authors, suggests a lack of ossification because of the distance from the periosteal vascularization [3].

**Myositis ossificans circumscripta**

The term myositis ossificans circumscripta (MOC) corresponds to a sudden process of heterotopic ossification that is preceded in certain cases by often minor injury. In fact, the term is not accurate because this “ostegenic storm” can occur outside the muscle, in contact with the fascias, the tendons or the adipose tissue. MOC usually occurs in the deep tissue in contact with the large muscles of the extremities. Moreover, this process does not initially involve inflammatory cells [7]. The pathological examination shows that MOC is organized into zones with proliferation of fusiform pseudosarcomatous cells during the first week followed by the development of annular ossification which progresses centripetally. The development of MOC is rare in the hand, although a few cases have been reported in the literature [8–11].
Context of chronic pain

Tendinosis and mechanical enthesopathy

Although it is not a separate pathological entity, mechanical enthesopathy may be associated with calcifications and/or periostosis. It is rare in the hands or fingers. It usually involves the insertion of the extensor and flexor carpi radialis on the dorsal and ventral 2nd and 3rd metacarpal bases, respectively. In the wrist, it involves the flexor carpi ulnaris, which is the only extrinsic muscle of the wrist that inserts on the carpus (pisiform). Injury to the radial extensors is favored by the presence of a carpal boss, which develops due to the presence of an accessory ossicle (os styloideum) or the presence of osteophytes due to osteoarthritis of the 2nd or 3rd metacarpal bases in contact with the trapezoid and the capitatum [12]. Distal enthesopathy of the flexor carpi radialis is usually associated with trapezio-metacarpal or scapho-trapezio-trapezoidal osteoarthritis.

Psoriatic arthritis

Psoriatic arthritis is an asymmetric mono- oligo- or polyarthritis that affects the small joints, in particular the interphalangeal joints of the hands and feet (Fig. 5). In 25% of cases, it affects one or several fingers, involving different joints (MCP, IPP, IPD) of a same digital ray. Clinically there is dactylitis. Psoriasis of the skin, scalp or nails is an important diagnostic feature. Nevertheless, arthritis may precede the skin condition in 10% of cases. The rheumatoid factor is
always negative. Radiography shows bone resorptions and hyperostosis. Extended periostosis appears as bone wispy and dense outgrowths (whiskering and spurs respectively) associated with marginal articular erosions and enthesitis [13] that may even be visible at a distance from the joint, for example on the radial styloid process [14]. These bony outgrowths should not be confused with the short bone spurs that may be visible on the tendon insertions on the lateral sides of the proximal phalanges, which are normal variants [15] and have no clinical significance.

Osteochondromatosis

Synovial osteochondromatosis is a primary or secondary osteocartilaginous metaplasia of the synovial tissue (Fig. 6). It may involve the joint or the synovial membrane of a tendon or bursa. In the hand and wrist, osteochondromatosis mainly involves the joints of fingers or the flexor tendon sheaths [16,17].

Intraarticular osteochondromatosis is rarer in the wrist or hand than in the knee, hip, elbow, shoulder or ankle. However, because of an extensive synovial territory, the tendon sheaths of the hand are frequently involved [18]. Conventional radiography identifies the soft tissue swelling and the presence of more or less calcified loose bodies. Erosion of the cortical bone (“scalloping”) in contact with the loose bodies may be found. Treatment includes a synovectomy and excision of the loose bodies. Postoperative recurrence is frequent and may occur at a distance from the initial location along the tenosynovial sheath. [19].

Calcium pyrophosphate dihydrate crystal deposition disease (CPPD)

Diverse clinico-radiographic presentations are observed from solitary articular and para-articular calcifications to arthropathy with articular degeneration. Chondrocalcinosis is defined by the presence of hyalin cartilage and fibrocartilage whatever the type of crystal is, although it usually involves CPPD crystals. Most diseases involving CPPD deposits are sporadic forms and usually involve women over the age of 50, usually after the age of 70. In younger
patients, a family form of the disease should be looked for which usually develops between 20 and 40 years old (with a predominance in France in Alsatian families) or a secondary form (revealing hyperparathyroidism, hemochromatosis or hypomagnesemia in particular) [20].

The disease may be asymptomatic (10–20% of cases) and discovered by chance on X-ray. CPPD can also mimic a degenerative arthropathy (pseudo-arthritic appearance on X-ray in 35–60% of cases) with bilateral symmetric polyarticular involvement. More rarely, a rapidly degenerative arthropathy may also be observed. In 10–20% of cases, the clinical features include pseudo-gout, acute arthritis, occasional fever, and significant local inflammation.

Calcifications of the periarticular soft tissues involve the synovial membrane, the capsule, the tendons and the ligaments. They are thin and linear. Synovial calcifications are located throughout the joint space and form ill-defined blury opaque calcifications of low density. The metacarpo-phalangeal joints are frequently involved in the hand. In the wrist, synovial calcifications are mainly radiocarpal at the carpal triangular fibrocartilaginous complex and the distal radioulnar joint. Tendinous calcifications are found along the main axis of the tendon, and may have a stratified appearance. Real calcified masses may be visible in the soft tissues and can mimic gout [21] (Fig. 7).

Gout

Osteoarticular involvement in gout corresponds to the presence of intra- or para-articular monosodium urate crystals. This is the sign of chronic hyperuricemia, above the monosodium urate saturation point, which causes the slow formation of crystals. Except for secondary forms and enzymopathies, primary gout is usually found in men over the age of 40. Tophi usually develop several years after the onset of symptoms and are tissue deposits of urea whose volume is correlated to the level of hyperuricemia and the duration of disease without treatment. Calcifications occur later with variable densities. Subcutaneous tissues, tendons, paraarticular soft tissues, and osteoarticular tissues are involved while the joint is preserved for a long time. In the hand, involvement of interphalangeal joints (proximal and distal) is more frequent than that of metacarpo-phalangeal joints. Involvement is asymmetric associating erosions and bone spurs (enthesophytosis and osteophytosis) [22].

The association of gout and para-articular CPPD deposits is possible in particular in the fingers. In this case, the calcifications are related to CPPD crystals, and not the result of gout tophus calcifications.

Connectivitis

Calcinosis of the soft tissues (calcinosis cutis) is frequently associated with connectivitis and more particularly with systemic scleroderma and dermatomyositis [23] (Fig. 8).

Scleroderma is a disease of the connective tissue, arterioles and microvessels resulting in fibrosis and vascular obliteration. It is characterized by the presence of antcentromere or antitopoisomerase I antibodies. There is a diffuse and progressive form and a form that is more limited to the cutaneous tissues named CREST syndrome (abbreviation for Calcinosis, Raynaud’s syndrome, Esophageal dysmotility, Sclerodactylyla and Telangiectasia). Both forms can present with calcinosis of the soft tissues, which is frequent in the hand, and which corresponds to aggregates of sometimes large, pseudotumoral hydroxyapatite crystals. The physiopathogenesis of these deposits is a subject of debate. They are painless, may become ulcerated and in this case have a chalky appearance. Osteoarticular involvement is possible and includes erosive and degenerative arthritis with epiphyseal erosions, joint space narrowing and acro-osteolysis [24].

Dermatomyositis is a diffuse inflammatory myopathy that is found in children in 50% of cases and adults around 40 years old, especially men. There is a bilateral symmetric and proximal motor deficit of the limbs associated with cutaneous symptoms including subcutaneous calcinosis. This calcinosis is especially frequent in the pediatric form of the disease. Calcifications are linear and curved, mainly observed in the knee, elbow and fingers, sometimes associated with a milky liquid discharge and resorption of the tips of the distal phalanx. Muscle calcifications are mainly observed in the proximal limbs and intermuscular fascias [25].

Figure 7. ‘‘Pseudo-tophus’’ of calcium pyrophosphate dihydrate (CPPD) crystals. Coronal (A) and axial (B) CT scans of the calcified mass (arrows) that has eroded the metacarpal cortex and penetrated the medullary canal.
Non-traumatic calcifications/ossifications

Chronic Renal failure

Hydroxyapatite deposits are found in the soft tissues of patients with chronic renal failure (Fig. 9). The frequency seems to increase with the duration of the renal failure and dialysis of patients. They are due to secondary hyperparathyroidism, the increase in phosphocalcium and alkalosis. These deposits form round or oval radio-opaque aggregates, usually located in the para-articular areas of the shoulder and wrist and the interphalangeal joints of the fingers, hips and ankles [26]. They are usually asymptomatic but may be associated with joint pain and osteodystrophy. Calcium oxalate crystals may also be visible in cartilage, the synovial membrane, the joint capsule and the periarticular and subcutaneous soft tissues [27]. These calcifications may be cloudy, reticulated, or punctuate depending on the tissue they are located in. Secondary oxalosis is favored by vitamin C supplements, for which oxalate is a catabolite. Finally, vascular calcifications are frequent in chronic renal failure in relation to arteria media calcification, which may be seen on radiography and earlier on ultrasound [28].

Hypertrophic pulmonary osteoarthropathy (Pierre Marie-Bamberger syndrome)

This syndrome is generally associated with a cyanotic pulmonary condition, which may or may not be tumoral (Fig. 10). Non-tumoral causes include cyanotic cardiopathies, chronic respiratory failure, and cystic fibrosis. However, this syndrome is frequently paraneoplastic, associated with lung cancer, which it may reveal [29]. VEGF (Vascular Endothelial Growth Factor) and PDGF (Platelet-Derived Growth Factor) are probably involved in the pathogenesis of this disease. VEGF is secreted during chronic hypoxia and by the tumor itself. Clinically, joint stiffness is frequent. Arthralgia and joint effusion associated with hypertrophic wrists, hands and digital clubbing are also observed. Periostitis is visible on conventional radiography with a periosteal proliferation forming peridiaphyseal sheaths involving the phalanges, the metacarpal and carpal bones and the diaphysis of the long bones.

More or less painful swelling

Chondromas

Bone chondromas (enchondromas, subperiosteal chondromas) frequently involve the fingers (Fig. 11). They may be solitary or multiple in enchondromatosis (Ollier’s disease). They may be asymptomatic or revealed by a stress fracture. They can grow within the bone and expand it presenting extraosseous growth that clinically appears as a deformed finger. Chondromas with an epicenter in soft tissues are rare except for chondromas of the respiratory tract and remnants of cervical cartilage of the brachial fissures. In most cases, they occur in the extremities, the hands in 54–64% and the feet in 20–28% of cases. This benign tumor, which is usually solitary, is a firm, mobile, slow growing mass that may be painful, and may adhere to the joint capsule or the tendon sheath [30]. The MR scans show a lesion with hyperintensity on T2-weighted images and a hypo- or isointensity on T1-weighted images that is more or less heterogeneous depending on the extent of mineralization [31]. Post-contrast enhancement is usually peripheral and septal, highlighting the lobulated architecture of the tumor [32]. Calcifications are visible in 33% - 77% of cases on radiography or CT scans and may suggest the mineralization pattern of a chondroid matrix [33]. These calcifications are usually “pop-corn” like with rings and arcs but may be punctiform depending on the maturity of the tumor. They are central or peripheral defining the lobulated architecture of the lesion. The chondromas in fingers are less often calcified which may be due to an earlier clinical diagnosis.

Vascular malformation

Vascular malformations may be found throughout the body but are more often found in the pelvis, the limbs and the skull (Fig. 12). Fifty to sixty-six percent of the lesions are
Figure 9. Calcium deposits in a dialysis patient with chronic renal failure. Oval shaped, cloudy calcified masses (arrows) of the phalanges of the toes (A) and fingers (B).

Figure 10. Hypertrophic pulmonary osteoarthopathy (Pierre Marie-Bamberger syndrome) associated with a bronchopulmonary carcinoma. Periosteal proliferation with peridiaphyseal sheaths involving the radius and ulna (A) and the metacarpal bones (B).

Figure 11. Enchondromatosis (Ollier’s disease) involving the fingers (amputation of the right 5th and left 3rd fingers). A. On conventional radiographs, the enchondromas appear as bone radiolucent and expanding lesions of the phalanges particularly visible on the 3rd and 4th rays of the right hand and phalanges of the 2nd left finger. B. The extraosseous expansion of the chondromas of the 2nd left finger accounts for the swelling and calcifications of the soft tissues (arrows).
Non-traumatic calcifications/ossifications

Figure 12. Numerous phleboliths related to a large venous malformation involving the forearm and the palmar aspect of the hand, visible on conventional radiographs in anterior-posterior view (A) and sagittal view (B) (courtesy of Dr. Annouk Bisdorff, Hôpital Laribosière, Paris).

Venous malformations. In the hands and feet, only 10% of vascular malformations are arteriovenous [34].

Venous malformations or those with a main venous component (because they may be associated with lymphatic or capillary vessels) are usually diagnosed in the upper limb distal extremity because of their coloration that is more or less blue when it is superficial. These venous malformations frequently show phleboliths (visible on X-ray or CT scans) that must not be confused, on MR images, with flow voids that are related to the rapid flow in arterial vessels [35].

Calcifying aponeurotic fibromas

Calcifying aponeurotic fibromas are rare (< 1% of the benign soft tissue tumors) and usually develop in children and young adults with a peak between 8 and 14 years old predominantly in males (ratio of males to females of 2:1). In 70% of cases, they develop in contact with aponeuroses, fascias and tendons of the palmar side of the hands. The second most frequent location is the foot (plantar side). They are slow-growing painless tumors that do not limit the joint. They are poorly differentiated and heterogeneous on MRI. Calcifications are not always present at first; they are thin, punctiform and generally centrally distributed. Before surgical removal, a biopsy should be performed to exclude a synovial sarcoma, which is the main differential diagnosis [36].

Conclusion

Calcifications and ossifications of the surface of bones and soft tissues may reveal metabolic, systemic, arthritic, benign or pseudotumoral entities, requiring additional investigation, surgical resection or simply treatment of symptoms. The clinical presentation is essential to narrow down the diagnostic possibilities and must be considered in relation with the anatomical location and imaging features.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References