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Periarticular calcification causing acute carpal tunnel syndrome: a case report

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ABSTRACT

We report a case of acute carpal tunnel syndrome caused by periarticular calcification (hydroxyapatite deposition disease) around the wrist joint in a 64-year-old woman. She had acute severe pain, exacerbated by wrist movements and extension of the fingers. Her full blood count, urea, electrolytes, uric acid, calcium, phosphate, alkaline phosphatase, and thyroid function levels were all within normal ranges, and her serum was negative for rheumatoid factor. Computed tomography revealed lobulated calcification close to the volar capsule. She underwent an emergency surgical decompression of the carpal tunnel under general anaesthesia within 3 hours of presentation. The flexor tendon sheaths were excised, and 'toothpaste-like' chalky material (hydroxyapatite crystals) in the capsule was removed. The pain was relieved dramatically and her median nerve function recovered. She was symptom-free at the one-year follow-up.

Key words: *calcification, physiologic; carpal tunnel syndrome; median nerve*

INTRODUCTION

Acute carpal tunnel syndrome is uncommon, and is usually caused by a carpal injury or radial fracture. We report such a case caused by periarticular calcification around the wrist joint.

CASE REPORT

In June 2007, a 64-year-old woman presented with a 2-day history of severe pain in her right wrist. She had had mild discomfort in her wrist but no loss of function for 6 weeks following a mild 'jarring' while gardening. The acute severe pain was worst at night (visual analogue scale score of 10) and exacerbated by wrist movements and extension of the fingers. It had not responded to paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs). She also had

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a progressively worsening sensation of 'pins and needles' in her hand. Her medical history was not suggestive of gout or hyperparathyroidism.

On examination the patient was afebrile, with a normal heart rate and blood pressure. She held her fingers in a flexed position, and attempts at active and passive movement of the wrist and extension of the fingers were markedly limited, owing to pain. The volar aspect of her wrist was diffusely swollen. Light touch sensation was reduced and objective sensation was lost (2-point discrimination of >20 mm) over the median nerve distribution. The muscles of the forearm were neither tense nor tender to palpation. The radial pulse and capillary refill appeared normal. The pain prevented elicitation of Phalen's or Tinel's sign, or testing of muscle power supplied by the median nerve. Her full blood count, urea, electrolytes, uric acid, calcium, phosphate, alkaline phosphatase, and thyroid function levels were all within normal ranges, and her serum was negative for rheumatoid factor. Wrist radiography showed a shadow measuring 2x2 cm near the ulnar and volar aspect of the right radiocarpal joint; computed tomography showed lobulated calcification close to the volar capsule (Fig. 1).

She underwent an emergency surgical decompression of the carpal tunnel under general anaesthesia within 3 hours of presentation. There was considerable tenosynovitis in the common flexors, and the median nerve appeared congested (Fig. 2). The flexor tendon sheaths were excised, and 'toothpaste-like' chalky material in the capsule in the 2 areas of nodular swelling was removed.

Bacterial cultures were negative and there were no birefringent crystals under polarising lights. The nodule was histologically consistent with hydroxyapatite crystals. The tenosynovium revealed non-specific inflammatory cells.

Postoperatively her pain was relieved dramatically, and she recovered her median nerve function. The wrist was held in a slab for a week, and a course of NSAIDs was given for 2 weeks. Active mobilisation was commenced after removal of the slab. At the one-year follow-up, she remained free of symptoms.

DISCUSSION

Fractures of the distal radius are the most common causes of acute carpal tunnel syndrome,[1–3] followed by carpal injuries.[4–6] Non-traumatic acute carpal tunnel syndrome has been reported to be secondary to infective tenosynovities,[7] coagulopathies,[8] false aneurysm,[9] gout or rheumatologic disorders.[3]

Pressure from a haematoma or thickened synovium may contribute to fibrosis and ischaemic damage within the nerve. Regardless of the cause,

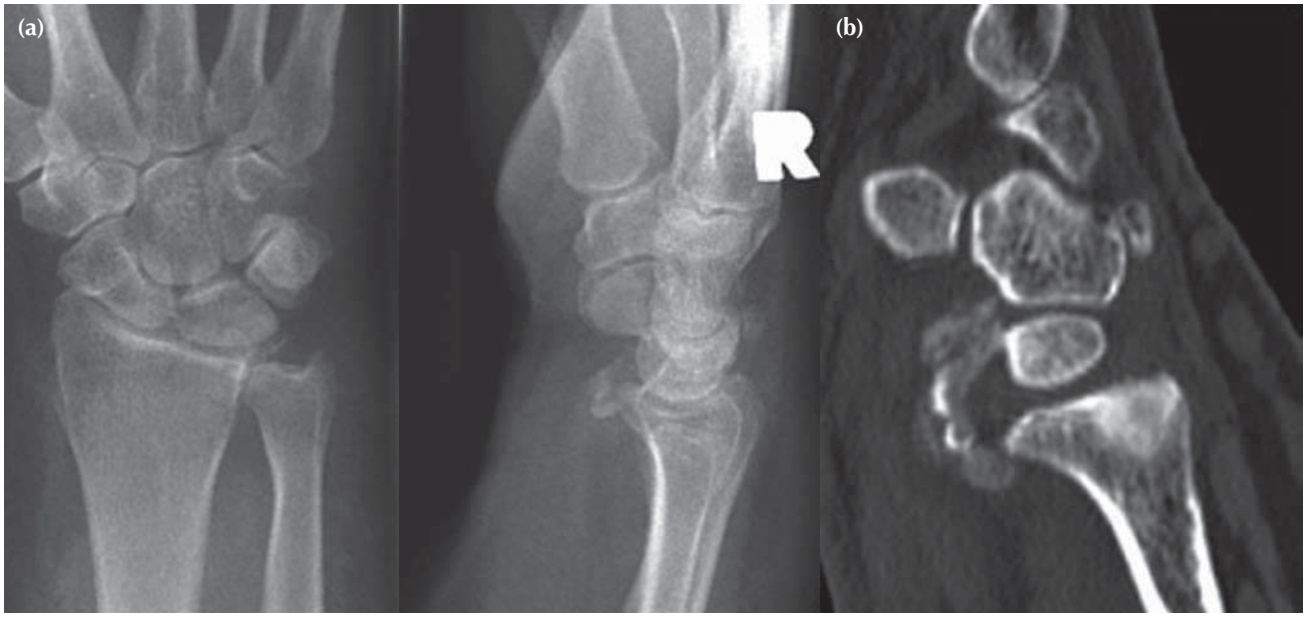

Figure 1 **(a)** An amorphous opacity is seen at the anterior wrist. **(b)** Deposits of high signal intensity suggestive of a calcified mass anterior to the carpal bones.

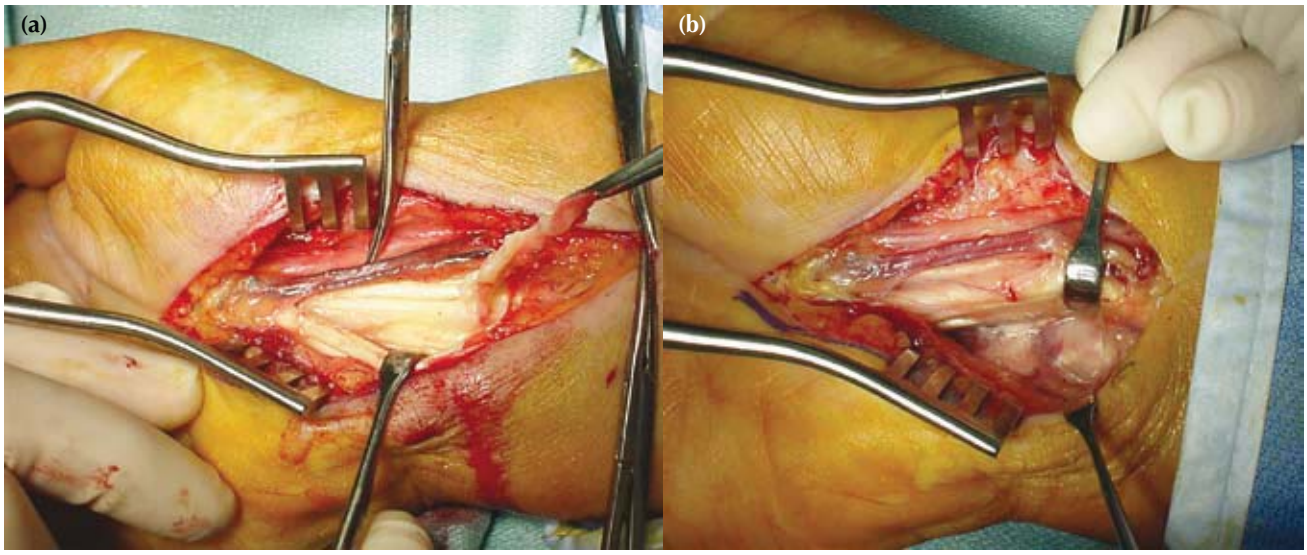

Figure 2 **(a)** The median nerve is congested with tenosynovitis of the flexor tendons. **(b)** Chalky, granular deposits in the volar capsule of the wrist consistent with hydroxyapatite crystals.

Table
Differences between calcium pyrophosphate and calcium hydroxyapatite crystals

Characteristics	Calcium pyrophosphate	Calcium hydroxyapatite crystals
Crystalline arthropathy	Most common	Less common
Site	Usually polyarticular: knee>hip>shoulder, elbow	Usually monoarticular: shoulder>elbow, metacarpophalangeal joint, wrist
Tendon involved	Usually the supraspinatus, triceps, quadriceps, Achilles tendons	Usually the flexor carpi ulnaris tendon near its attachment to the pisiform
Manifestation	Acute/subacute/chronic and usually self-limiting	Acute/subacute/chronic and usually self-limiting
Radiographic appearance	Prominent linear or punctuate deposits and parallel to the subjacent subchondral bone	Calcifications are more homogeneous or cloud-like

emergency surgical decompression is indicated when acute carpal tunnel syndrome is suspected. Delayed treatment may result in long-term median neuropathy and incomplete recovery.[3,10,11]

Calcific periarthritis is uncommon and characterised by periarticular deposition of calcium hydroxyapatite crystals in bursae, tendons, and ligaments. Local trauma may precipitate periarticular calcification. Recurrence is uncommon. The shoulder is the most commonly affected joint, but any joint is susceptible. Crystal deposition is often asymptomatic and detected incidentally. Nonetheless, it may cause acute calcific periarthritis (due to rupture of the deposit into surrounding soft tissues, leading to an inflammatory response) with localised pain, tenderness, and loss of function. The acute episodes typically resolve over 2 to 3 weeks without treatment, only rarely causing chronic pain or dysfunction.

Hydroxyapatite deposition disease is characterised by calcifications with a homogeneous cloudlike appearance in specific sites, with no underlying disorder.[12] This distinguishes it from most other differential diagnoses. Calcium pyrophosphate disease is more linear and diffused (Table). Gouty tophi are more faintly calcified and associated with elevated urate levels. Heterotopic bone and myositis ossificans have a trabecular pattern with a cortical rim. Tumoural calcinosis, either primary idiopathic or secondary to renal disease, is a metabolic disorder. Collagen vascular diseases such as scleroderma or dermatomyositis usually produce widespread calcifications involving the subcutaneous tissues and are associated with an underlying disease. Periarticular metastatic calcification is usually

associated with sarcoidosis, hypervitaminosis D, hypoparathyroidism, and milk-alkali syndrome.

It is rare to find acute calcific periarthritis affecting the carpal joints and causing acute carpal tunnel syndrome. Most such cases involve small calcifications that are self-limiting and resolve with immobilisation and NSAIDs within 3 weeks.[13–17] In only 2 reported cases has a large hydroxyapatite deposit caused severe pain and a neurological deficit, threatening the median nerve to such an extent that immediate surgical decompression was indicated,[18,19] as seen in our patient who failed to respond to treatment with anti-inflammatories and had acute neurological deficits. Periarticular calcifications should be considered in the differential diagnosis of acute carpal tunnel syndrome.

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