INTRODUCTION

Acute carpal tunnel syndrome is an uncommon condition. When present, it usually follows a carpal injury or fracture of the lower end of the radius.

We describe a case of acute carpal tunnel syndrome in a 64 year old woman caused by periarticular calcific deposits around the wrist joint. Emergent surgical decompression of the carpal tunnel with removal of the calcified material was required and has led to remission of pain and recovery of median nerve function.
CASE REPORT

A 64 year old woman presented to the emergency department with severe pain in her right wrist of two days duration. This had been preceded by 6 weeks of mild discomfort but no loss of function following a mild ‘jarring’ while gardening. She reported that this acute severe pain was worst at night (10 out of 10) and exacerbated by wrist movements and extension of the fingers. It had not responded to paracetomol and NSAIDs prescribed by her general practitioner the preceding day. There was a sensation of ‘pins and needles’ in her hand. This progressively got worst. Her past medical history was not suggestive of gout or hyperparathyroidism.

On examination the patient was afebrile with heart rate and blood pressure within normal limits. She was in obvious discomfort and held the fingers of her right hand in a flexed position. A diffuse swelling of the volar aspect of the right wrist was noted. Attempts at active and passive movement of the wrist and extension of the fingers were markedly limited as they caused excruciating pain. A reduction to sensation of light touch was noted in the distribution of the median nerve. There was objective sensory loss (2-point discrimination greater than 20 mm) in the median nerve distribution. The muscles of the forearm were neither tense nor tender to palpation. Radial pulse and capillary refill appeared to be normal.

Due to the degree of pain she experienced on movement it was difficult to elicit Phalen’s or Tinel’s sign, nor to test power in the muscles supplied by the median nerve.

Full blood count, urea, electrolytes, uric acid, calcium, phosphate, alkaline phosphotase and thyroid function were all within normal ranges and rheumatoid factor was negative. Plain radiographs [Fig 1] of the wrist showed a 2 cm x 2cm large, dense shadow near the ulnar and volar aspect of the right radiocarpal joint with no other bony abnormality or joint lesion. An urgent CT scan of the same region showed lobulated calcification in the wrist joint close to the volar capsule [Fig 2].

The patient was admitted for pain relief and emergent surgical decompression of the carpal tunnel. This was performed within three hours of her presentation to the emergency room under general anaesthesia. An extended carpal tunnel incision was used to decompress the median nerve. There was an evidence of significant tenosynovitis of common flexors. The median nerve appeared congested. Tenosynovectomy of flexor tendons was performed. On retracting flexor tendons, there were two areas of nodular swelling [Fig 4] in the capsule.
which on exploration revealed chalky material with “tooth paste like consistency”.

The biopsy of the nodule reported to be acute calcification around the joint which was histologically consistent with acute periarticular calcification [hydroxyl apatite crystals]. The tenosynovium revealed nonspecific inflammatory cells. Culture was negative and polarising light did not reveal negative birefringent crystals. With this clinical, radiological, histological findings were suggestive HADD [periarticular calcification]

There was dramatic relief after surgery. Wrist was held in a slab for a week and was given a course of NSAID for two weeks. After removal of slab active mobilization is commenced. At one year, patient was symptom free.
DISCUSSION

The syndrome of acute compression of the median nerve has only recently been reported (Bauman et al 1981). Although fractures of the distal radius are the most common underlying injury, the syndrome may also be seen with carpal injuries. Nontraumatic acute carpal tunnel syndrome been described secondary to: infective tenosynovities, coagulopathies, false aneurysm, gout or rheumatologic disorders.

Local pressure from haematoma or thickened synovium may contribute to fibrosis, probably by causing ischemic damage within the nerve. It is important, however, for medical staff to recognise the symptoms of acute carpal tunnel syndrome, as there is potential for long-term median neuropathy if treatment is delayed. Regardless of the underlying cause, operative decompression is indicated as soon as possible when the diagnosis of acute carpal tunnel syndrome is suspected. Delay in treatment may result in incomplete recovery and may be responsible for long term sequelae.

Calcific periarthritis is an uncommonly reported condition characterised by periarticular deposition of calcium hydroxyapatite crystals in bursae, tendons and ligaments. It was first described in 1870 in the shoulder and this remains the most commonly affected joint, but there is the potential for deposition around any joint. Crystal deposits are often asymptomatic and detected as incidental findings, but may cause acute calcific periarthritis (thought to be due to rupture of the deposit into surrounding soft tissues leading to an inflammatory response) with localised pain, tenderness and loss of function. These acute episodes typically resolve gradually over two to three weeks without treatment, only rarely causing chronic pain or dysfunction.

One of the confusing aspects of periarticular calcification is the associated nomenclature. A variety of terms have been used. A very characteristic homogeneous cloudlike appearance distinguishes HADD [as the present case] from most other disorders. Finding calcifications in the specific sites favoured by HADD without an underlying disorder should separate this entity from others in the differential diagnosis.

HADD should not be confused with the more linear and diffuse CPPD crystal calcifications [Table 1]. Gouty tophi are more faintly calcified and are associated with elevated urate levels. Heterotopic bone and myositis ossificans have a trabecular pattern with a cortical rim that can be distinguished from HADD and CPPD calcifications. Tumoral calcinosis, either primary
idiopathic or secondary to renal disease, may mimic HADD if it presents in small amounts. One should look for a metabolic disorder in the latter situation to distinguish this cause. Collagen vascular disease such as scleroderma or dermatomyositis can also produce calcifications that can mimic HADD. These calcifications are usually more widespread, can involve the subcutaneous tissues, and are associated with a known underlying disease. Periarticular metastatic calcification may be seen in association with sarcoidosis, hypervitaminosis D, hypoparathyroidism, and milk–alkali syndrome.

The carpal joints are infrequently affected by acute calcific periartthritis and reports in the literature of acute carpal tunnel syndrome caused by calcific deposits are very rare, consisting only of case reports1-18. Most of these reports13-17 describe cases where small calcifications within the confines of the carpal tunnel led to symptoms of carpal tunnel syndrome which responded well to conservative treatment with immobilisation and NSAIDs. In most cases the symptoms resolved completely within 3 weeks with gradual resorption of the calcifications. 2 cases18,19 such as the one we present here in which a large apatite deposit produced severe pain deficit and acutely threatened the median nerve such that immediate surgical decompression was indicated is very rare.

The reason for the development of periarticular calcific deposits remains unclear, but a local trauma (as is the case in our patient) may precipitate calcific lesions. Recurrence appears to be uncommon and our patient continues to have good function of her hand and wrist with no further pain.

In conclusion, this rare case should remind orthopaedic surgeons and radiologist to consider periarticular calcifications when abnormal deposits seen in the vicinity of the joint within the carpal tunnel when evaluating for acute carpal tunnel syndrome.
REFERENCES


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LEGENDS

Fig 1
A plain anteroposterior radiograph of the right wrist, showing an amorphous opacity at the wrist level anteriorly

Fig 2
CT demonstrating an amorphous deposit of high signal intensity suggestive of calcified mass anterior to the carpal bones

Fig 3
Intraoperative photograph of the wrist, showing congested median nerve with tenosynovitis of the flexor tendons.

Fig 4
Intraoperative photograph of the wrist, showing chalky, granular deposits in the volar capsule of the wrist consistent with hydroxyapatite crystals