Acute calcific tendinitis of the flexor carpi ulnaris causing acute compressive neuropathy of the ulnar nerve: a case report

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ABSTRACT

This study reports a case of acute calcific tendinitis of the flexor carpi ulnaris in a 64-year-old woman. She presented with symptoms of acute ulnar nerve compression mimicking a volar compartment syndrome. Owing to rapidly progressive symptoms, emergency surgical exploration was carried out. Intra-operatively a large mass of calcium phosphate carbonate was noted in association with the flexor carpi ulnaris near its insertion at the wrist compressing the ulnar nerve and artery in Guyon’s canal. Postoperatively the patient had complete resolution of symptoms. Conservative management with non-steroidal anti-inflammatory drugs, rest, splinting, and steroid therapy is recommended for acute calcific tendinitis, but this case suggests a role for surgical treatment when there is acute neural compression and severe pain.

Key words: compartment syndrome; tendinopathy; ulnar nerve

INTRODUCTION

Acute calcific tendinitis (ACT), also known as calcific peritendinitis or periarthritis, is an acute inflammatory condition of unknown aetiology commonly occurring in the pericapsular area of the shoulder, and less often in the hand or wrist.1,2 ACT of the flexor carpi ulnaris (FCU) may cause ulnar nerve compression as the nerve traverses Guyon’s canal.3 Its symptoms can be of rapid onset and severity, with pain, swelling, localised erythema, and loss of function. It often causes diagnostic confusion, and patients may be inappropriately treated for infection, crystal arthropathy, or even fractures.3–5 This study reports a case of ACT of the FCU causing acute ulnar nerve compression in Guyon’s canal. The diagnostic difficulty was highlighted, as was the optimum treatment strategy when associated with acute compressive neuropathy.

CASE REPORT

In September 2010, a 64-year-old, right-hand-dominant woman presented with a 12-hour history
of increasing pain and restricted range of movement of the left wrist. She had no fever or malaise, nor any trauma, pathology, or overuse of the wrist. In the past 2 hours, she started to develop paraesthesia in the ring and little fingers. She had undergone a total knee replacement for osteoarthritis 4 weeks earlier.

On examination, the wrist was swollen with tenderness on the volar-ulnar side. There was altered sensation in the little finger and the ulnar border of the ring finger, with a positive Tinel’s test over Guyon’s canal. Radial and ulna pulses were palpable and distal capillary refill time was under 2 seconds. Range of movement at the wrist was restricted by pain to an arc of 30°. Her white cell count was 12.7 x10⁹/l, C-reactive protein level was 23 mg/l, and erythrocyte sedimentation rate was 34 mm/hr. Other parameters, including serum urea and electrolytes were within normal limits.

The provisional diagnosis was acute compressive neuropathy of the ulna nerve at the level of Guyon’s canal secondary to a septic or inflammatory arthritis. Ultrasonography of the wrist revealed no effusion.

The patient was prescribed anti-inflammatories, and the wrist was splinted. Her pain increased over the following hours and persisted, despite >120 mg of morphine sulphate having been given in <10 hours. Repeated clinical examinations revealed a progressive deficit of ulna nerve sensory and motor function, with median nerve involvement. Palpation over Guyon’s canal became intolerable, and the distal volar compartment of the forearm became tense. All active wrist movement was lost and passive extension of the wrist or fingers caused severe pain. Radiographs were obtained en route to the operating theatre (Fig.).

Owing to the rapidly progressive nature of the neurological deficit and signs of an early volar compartment syndrome, urgent surgical exploration of the wrist was performed to decompress Guyon’s canal through a volar approach. The wrist was severely swollen and tense over the ulnar side. The ulnar pulse was poor. On incising the volar carpal ligament, a significant volume of chalky fluid was extruded under pressure. Within the canal, a large mass of soft chalky material associated with the tendon of the flexor carpi ulnaris was extracted piecemeal. At this stage the forearm compartments became soft. No further decompression or exploration was necessary. The diagnosis was ACT of the FCU.

Postoperatively the patient had an immediate return of ulnar and median sensory function, and then pain-free motor function on the following day. The retrieved specimen showed 80% calcium phosphate carbonate and 10% magnesium phosphate, consistent with ACT. Microscopy and culture confirmed no infection.

**DISCUSSION**

ACT causes acute inflammation in periarticular locations. Its aetiology is poorly understood. One theory postulates microvascular trauma and localised tissue hypoxia, in reaction to soft-tissue deposition of calcium that give rise to the inflammatory responses (pain, swelling, and erythema). The deposits are usually composed of calcium carbonate apatite, which can usually be seen on radiographs as fluffy opacification. Blood tests may help to exclude infection but not to establish the diagnosis.

ACT of the FCU is a potential cause of compression of the ulnar nerve at the level of Guyon’s canal. A case of ACT compressing the median nerve in the carpal tunnel has been reported, where surgical exploration was performed owing to diagnostic uncertainty, despite a slower onset of symptoms. Normally, ACT should be treated nonsurgically as it is self-limiting and resolves over the course of several weeks. Management includes splinting and non-steroidal anti-inflammatory drugs. Injected or systemically administered steroids have variable success and can provide rapid resolution.
of symptoms.\textsuperscript{9} Surgical exploration is no longer advocated.

Thorough diagnostic workup is important. In our patient, as there was no history of trauma, radiographs of the wrist were not arranged until the patient was \textit{en route} to the operating theatre. Ultrasonography is user dependant and failed to assist in the diagnosis. Radiographs would have hastened the diagnosis, but would not have changed the management of the patient. Although conservative management is advocated for ACT, surgical exploration was performed in our patient, because the diagnosis was uncertain and the wrist pain was severe despite strong opioid analgesia. Recovery of neural function is favoured by reducing the duration of compression, as such has been noted in burns patients.\textsuperscript{11} Thus, when ACT causes acute nerve compression, urgent surgical decompression may be necessary.

\section*{REFERENCES}