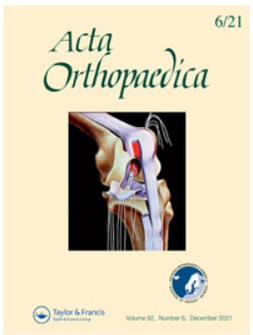

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Dislocating medial head of triceps—awareness of the condition could avoid inappropriate surgery—a case report

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A 62-year-old man was referred to our unit by his general practitioner with the preliminary diagnosis of a cyst on the medial aspect of his left elbow. On clinical examination, he was found to have a painful snapping beyond 70° of elbow flexion. Ulnar nerve dislocation was implicated as he had pain radiating down the ulnar side of his forearm. A soft tissue mass was palpable above the medial epicondyle, which was initially thought to be a lipoma pushing the ulnar nerve forward. An ultrasound scan failed to show a discrete abnormal mass and the conclusion of the report by the radiologist was that an accumulation of subcutaneous fat could be responsible for the swelling. His ulnar nerve symptoms had vanished at this stage, but the discomfort from the clicking sensation over the medial side of the elbow still persisted. We decided to perform an exploration with the presumptive diagnosis of a dislocating anomalous medial head of triceps leaving the option open for an anterior transposition of the ulnar nerve.

Operative findings

No lipoma was visible. The ulnar nerve was being pushed anteriorly by a large anomalous medial head of triceps (Figure 1) and they dislocated together in flexion (Figure 2). There were in effect two clicks on passive flexion of the elbow, the first caused by the ulnar nerve and the second by the dislocating medial head of triceps moving across the medial epicondyle. A wedge of muscle was excised, the tendon partially divided on its medial side, rolled in and sutured. Flexion did not cause any dislocation of the triceps at the end of

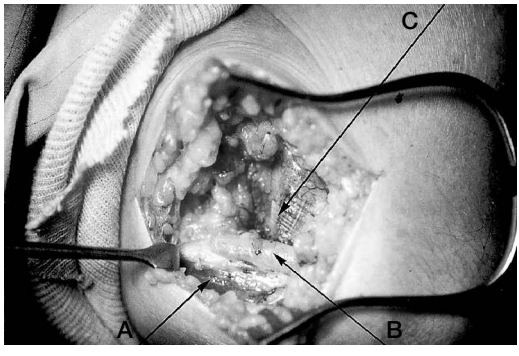

Figure 1. Medial aspect of the left elbow in extension with anterior up and posterior down. Large anomalous medial head of triceps (A) behind the ulnar nerve (B) pushing it anteriorly. Arrow marked C points at the medial epicondyle.

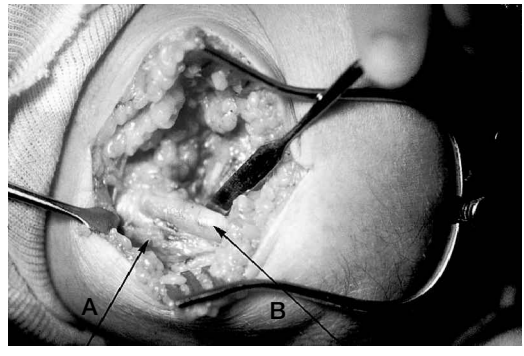

Figure 2. Same elbow as in Figure 1 in flexion. Both the medial head of triceps (A) and the ulnar nerve (B) are dislocating anterior to the medial epicondyle. The tip of the instrument lies beneath both structures on the medial epicondyle.

the procedure. The ulnar nerve was still dislocating but since it was not symptomatic we felt it appropriate to leave it alone. The wound was closed in layers.

Postoperative course

The patient was discharged in a collar and cuff and was instructed to mobilize his elbow immediately and gently. There were no ulnar nerve symptoms postoperatively, neither could dislocation of the nerve be elicited during follow-up examination at 6 weeks postoperatively. It was thought to be anchored by the scar tissue either in front or behind the medial epicondyle. The patient was kept under further follow-up. He was seen at 6 and then 12 months postoperatively. He had neither any ulnar nerve symptoms nor clicking at the elbow with full range of motion on both occasions.

Discussion

Variations of the musculature around the elbow have been reported. These may present as an abnormal insertion of the medial head of the triceps into the medial epicondyle (Matsuura et al. 1994), an anconeus epitrochlearis muscle originating from the medial head of triceps and inserting into the lateral epicondyle (O'Hara and Stone 1996), or a prominent medial head that covers the ulnar nerve or even extends into the cubital tunnel (Dellon 1986). Clinical consequences may be sensory and motor disturbance of the ulnar nerve (Rolfsen 1970, Dreyfuss and Kessler 1978, Reis 1980, Hayashi et al. 1984, Spinner et al. 1997), or an uncomfortable or painful snapping sensation on the medial side of the elbow (Reis 1980, Spinner et al. 1997). Rolfsen (1970) also described instability in the hand and fingers during the snapping. A dislocating medial head of triceps has also been reported in conjunction with Waardenburg syndrome, a rare constellation of facial, ophthalmic, neural and cutaneous anomalies (Spinner et al. 1997).

Ulnar nerve symptoms caused by a snapping medial head of the triceps muscle were first reported by Rolfsen (1970) and subsequently by Dreyfuss and Kessler (1978), Reis (1980) and Hayashi et al. (1984). Haws and Brown (1995) reported a bilateral case. Dreyfuss and Kessler noticed dislocation of the ulnar nerve together with the medial head of the triceps during the exploration as was the case with our patient. Hayashi et al. (1984) found a tendinous arch which prevented the nerve from moving anteriorly, thereby causing compression. They postulated that dislocation of the nerve was not an important factor contributing to the development of the cubital tunnel syndrome, but it was compression. They released the arch which was tethering the nerve and also performed a medial epicondylectomy to solve the snapping problem. Dreyfuss and Kessler (1978) reported that the snapping itself is well tolerated by the patient and they seek medical attention only when the sensory symptoms appear. This statement disagrees with the experience of Rolfsen (1970), Reis (1980) and Haws and Brown (1995). Although the nerve symptoms of patients reported by these authors improved considerably after anterior transposition of the ulnar nerve, which was the initial surgical intervention, they required additional surgery for uncomfortable or painful snapping. Our experience also confirms this.

As to the surgical procedure, Reis (1980) and Haws and Brown (1995) preferred to excise the anomalous slip of the medial head, whilst Dreyfuss and Kessler (1978) detached the medial head from its insertion and diverted it radially to suture it to the central portion of the tendon. Our operative technique was most similar to Rolfsen's (1970), who excised the dislocating part of the tendon and inserted "correcting" sutures to abolish the dislocation.

This condition may easily be missed in routine clinical practice because the clinician may simply not be aware of its existence. Patients may present with or without nerve symptoms. The diagnostic dilemma, however, exists in both cases. There are a variety of different mechanisms causing ulnar nerve symptoms of which dislocation of the nerve from its groove is the commonest. Indeed, most of the reports on this condition reveal that patients had to undergo repeated surgical explorations to shed light on the actual pathology (Rolfsen, 1970, Reis 1980, Haws and Brown 1995, Spinner et al. 1997). Whilst the focus of diagnostic and therapeutic attention would invariably be directed towards the ulnar nerve in the presence of nerve

problems, ill-defined symptoms may cause confusion and delay treatment.

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Increased levels of chondrocalcin in knee joint fluid in synovial chondromatosis—a case report

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A 70-year-old man was admitted to our department with a 5-month history of spontaneous pain and limitation of motion in his left knee. Radiographs of the knee were normal, but MRI showed fluid in the joint. The concentration of chondrocalcin in articular fluid measured by EIA was 18 ng/mL.

Arthroscopy revealed only mild cartilage erosion. During surgery, abundant cellular debris was removed. Histological evaluation of the cellular debris showed typical findings of synovial chondromatosis, but the synovium was normal or only slightly inflamed. The patient had no complaints at 7 months postoperatively.

Discussion

Synovial chondromatosis is a rare disorder characterized by the formation of multiple cartilaginous nodules in the synovial joint, bursae or tendon sheaths (Murphy et al. 1962, Sim et al. 1977). Although the disease is considered by most authors to be metaplastic rather than neoplastic, little is known about its etiology (Sciot et al. 1998).

In cases with no calcification or ossification of the cartilage, it is difficult to diagnose of synovial chondromatosis without the use of arthrography or arthroscopy (Norman and Steiner 1986, Nokes et al. 1987, Burnstein et al. 1988).

Ryan et al. (1982) reported that chondromas obtained from the synovium of a patient who had typical synovial chondromatosis mainly contained type II collagen. This composition is distinct from that of rheumatoid and normal synovium. Normal synovium usually produces only types I, III, and V collagen.

Choi et al. (1983) reported a newly discovered protein that was isolated from fetal epiphyseal cartilage; it was called "chondrocalcin". Thereafter, the primary structure of chondrocalcin was found to be identical with that of the C-propeptide of type II procollagen (Van Der Rest et al. 1986). The joint fluid levels of this molecule reflect the synthesis of type II collagen increase in primary osteoarthrosis and traumatic arthritis joint fluid