

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

Dellon A L. Musculotendinous variations about the medial humeral epicondyle. *J Hand Surg (Br)* 1986; 11 (2): 175-81.

Dreyfuss U, Kessler I. Snapping elbow due to dislocation of the medial head of the triceps. A report of two cases. *J Bone Joint Surg (Br)* 1978; 60 (1): 56-7.

Haws M, Brown R E. Bilateral snapping triceps tendon after bilateral ulnar nerve transposition for ulnar nerve subluxation. *Ann Plast Surg* 1995; 34: 550-1.

Hayashi Y, Kojima T, Kohno T. A case of cubital tunnel syndrome caused by the snapping of the medial head of the triceps brachii muscle. *J Hand Surg (Am)* 1984; 9A (1): 96-9.

Matsuura S, Kojima T, Kinoshita Y. Cubital tunnel syndrome caused by abnormal insertion of triceps brachii muscle. *J Hand Surg (Br)* 1994; 19 (1): 38-9.

O'Hara J J, Stone J H. Ulnar nerve compression at the elbow caused by a prominent medial head of the triceps and an anconeus epitrochlearis muscle. *J Hand Surg* 1996; 21B (1): 133-5.

Reis N D. Anomalous triceps tendon as a cause for snapping elbow and ulnar neuritis: A case report. *J Hand Surg* 1980; 5: 361-2.

Rolfen L. Snapping triceps tendon with ulnar neuritis. Report on a case. *Acta Orthop Scand* 1970; 41: 74-6.

Spinner R J, Davids J R, Goldner R D. Dislocating medial triceps and ulnar neuropathy in three generations of one family. *J Hand Surg (Am)* 1997; 22A (1): 132-7.

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

Shito Fukuhara, Yosuke Kanazawa, Soshi Uchida, Shoujirou Akahoshi, Toru Yoshioka and Toshitaka Nakamura

Department of Orthopaedic Surgery, University of Occupational and Environmental Health, School of Medicine, Kitakyushu 807-8555, Japan. Tel +81 93-691 7444. Fax -692 0184
Submitted 99-08-08. Accepted 99-12-27

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 au-

thors 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 osteoarthritis and traumatic arthritis joint fluid

(Shinmei et al. 1995). Shinmei et al. (1995) reported that the mean (SE) and median values of chondrocalcin in joint fluids of osteoarthritis (94 cases), rheumatoid arthritis (141 cases), and traumatic arthritis (30 cases) groups were 4.9 (4.71), 1.1 (0.1), and 2.1 (2.0) ng/mL, respectively. The mean chondrocalcin value of normal joint fluids drawn by the injection of physiological saline from 15 healthy controls was 0.3 (0.1) ng/mL.

Since the degree of cartilage erosion was mild in our case, we conclude that the increased level of chondrocalcin reflected an increased synthesis of type II collagen in synovium caused by synovial chondromatosis. Estimates of chondrocalcin in joint fluid may be useful for diagnosing synovial chondromatosis.

- Burnstein M I, Fisher D R, Yandow D R, Hafez G R, De Smet A. Case report 502. *Skeletal Radiol* 1988; 17: 458-61.
- Choi H U, Tang L H, Johnson T L, Pal S, Rosenberg L C, Reiner A, Poole A R. Isolation and characterization of a 35,000 molecular weight subunit fetal cartilage matrix protein. *J Biol Chem* 1983; 258: 655-61.
- Murphy F D, Dahlin D C, Sullivan R. Articular synovial chondromatosis. *J Bone Joint Surg (Am)* 1962; 44 (1): 77-86.
- Nokes S R, King P S, Garcia R Jr., Silbiger M L, Jones J D, Castellano N D. Temporomandibular joint chondromatosis with intracranial extension. MR and CT contributions. *AJR* 1987; 148: 1173-4.
- Norman A, Steiner G C. Bone erosion in synovial chondromatosis. *Radiology* 1986; 161: 749-52.
- Ryan L M, Cheung H S, Schwab J P, Johnson R P. Predominance of type II collagen in synovial chondromatosis. *Clin Orthop* 1982; 168: 173-7.
- Sciort R, Cin P D, Bellemans J, Samson I, Van den Berghe H, Van Damme B. Synovial chondromatosis: clonal chromosome changes provide further evidence for a neoplastic disorder. *Virchows Arch* 1998; 433: 189-91.
- Shinmei M, Kobayashi T, Yoshihara Y, Samura A. Significance of the levels of carboxy terminal type II procollagen peptide, chondroitin sulfate isomers, tissue inhibitor of metalloproteinases, and metalloproteinases in osteoarthritis joint fluid. *J Rheumatol (Suppl 43)* 1995; 22: 78-81.
- Sim F H, Dahlin D C, Ivins J C. Extra-articular synovial chondromatosis. *J Bone Joint Surg (Am)* 1977; 59: 492-5.
- Van Der Rest M, Rosenberg L C, Olsen B R, Poole A R. Chondrocalcin is identical with the C-propeptide of type II procollagen. *Biochem J* 1986; 237: 923-5.

Delayed diagnosis and treatment of Tillaux fracture—a case report

Giovanni Zatti¹, Fabio D'Angelo¹ and Alberto Giughello²

¹Clinica Ortopedica e Traumatologica, Ospedale di Circolo, Viale Borri 57, IT-21100 Varese, Italy. Tel +39 332-282682. Fax -288956; ²Divisione di Ortopedia e Traumatologia, Ospedale "S. Anna", IT-22100 Como, Italy
Submitted 99-09-07. Accepted 99-11-22

A 14-year-old boy injured his left ankle while playing football at school and was brought to the local hospital, where anteroposterior and lateral roentgenograms were taken (Figure 1). The physician in the Emergency Department diagnosed an infraction of the lateral malleolus and a short leg cast was applied for 30 days.

After the cast was removed the patient continued to complain of ankle pain during walking and joint movements. He was referred to our department where new radiographs showed a Salter Harris type III injury of the lateral portion of the distal tibial physis (Salter and Harris 1963). Reexamination of the first radiographs showed a Tillaux frac-

ture with widening of the tibiofibular mortise. A CT scan confirmed this diagnosis and also showed a rotational displacement of the fragment (Figure 2).

5 weeks after the injury, the fracture was exposed through a small anterolateral incision. The tibiofibular mortise appeared widened and unstable and the bone was soft. The displaced fragment was cleaned respecting the intact anterior tibiofibular ligament. The original site was cleaned with a curette; the fragment was reduced and fixed with a 3.5-mm cancellous screw to the intact epiphysis.

The boy was discharged from hospital after 3 days with the ankle free and he was told not to