Case reports

Ulnar nerve compression—a case of giant uremic tumoral calcinosis

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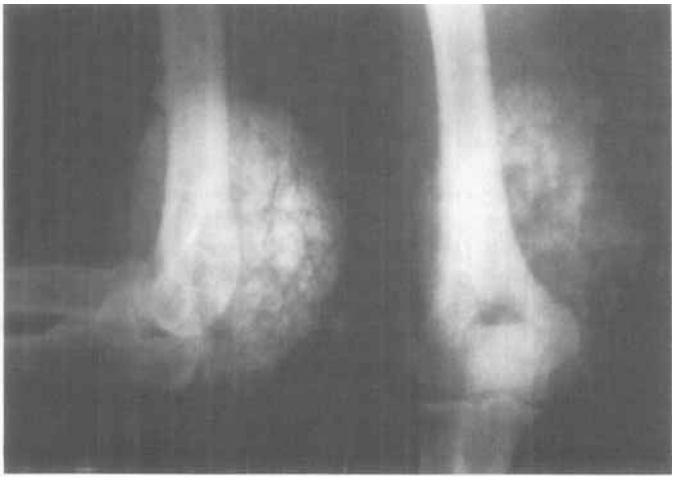
A 30-year-old man had been on chronic hemodialysis for 9 years because of chronic renal failure secondary to nephrosclerosis. During the preceding 6 months, he had noted round, firm tumors in both elbows, the left shoulder and the right hip. The tumors were painless and increased in size. He also reported paresthesias in the 4th and 5th fingers of the left hand during the previous month. Tinel's sign at the elbow was positive. Motility and sensibility were normal

Radiographs showed extensive homogeneous, lobulated, calcified masses around both elbows, left shoulder and right hip. An electrophysiological study disclosed cubital nerve impairment at the left elbow. Blood chemistry showed Ca 10.1 mg/dL, Ph 7.8 mg/dL, alkaline phosphatase 138 U/L and parathyroid hormone (PTH) 121 ng/L (normal value < 60 ng/L). The calcium-phosphorus product had been constantly above 70 in the last few years, due to an uncontrolled hyperphosphoremia (mean phosphorus 9.9 mg/dL) related to an inappropriate diet and insufficient dialytic treatment and due to moderate hypercalcemia related to the continuous administration of calcium carbonate and calcitriol. Hyperparathyroidism was excluded since parathyroidectomy had been performed 6 months before, and because the patient had maintained low PTH levels (< 150 ng/L).

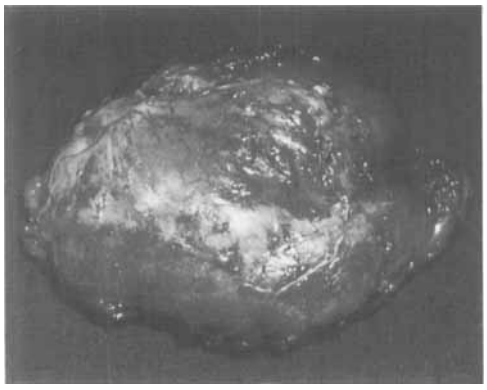
The tumors were surgically removed from both elbows. In the right elbow, the tumor was located in the subcutaneous tissue and did not compress the adjacent structures. A 10 cm tumor, with well defined edges, was excised from the left elbow. This tumor was located below the triceps muscle and above the cubital tunnel adhering to the cubital nerve. Decompression of the cubital nerve and external neurolysis were performed. The tumors were encapsulated by fibrous tissue and contained a white pasty mass. Microscopy showed an amorphous calcified material with macrophages and some giant cells surrounded by thin fibrous tissue septa, which were diagnosed as tumoral calcinosis. The duration of dialysis was increased and calcitriol was stopped. Paresthesias disappeared in the early period after surgery and an electrophysiological study at the left elbow performed 3 months later was normal. 6 months after surgery, the patient received a renal transplant. No relapse of tumoral calcinosis has been documented in a 2-year follow-up.



An extensive multinodular calcified mass. The joint space is normal.

Discussion

Tumoral calcinosis is an uncommon disease characterized by calcified periarticular, often large, soft tissue masses, containing calcareous material and usually located around large joints. Tumoral calcinosis can be pri-



Macroscopically, a well circumscribed and encapsulated tumor with a yellowish pasty material on cross-section is observed.

mary, with a family history in one third of the cases. Moreover, it has been described in association with primary or secondary hyperparathyroidism, vitamin D intoxication, scleroderma and chronic recurring multifocal osteomyelitis (Noyez et al. 1993, Majeed 1994, Geirnaerdt et al. 1995, Steinbach et al. 1995). Patients on dialysis because of chronic renal failure can develop tumoral calcinosis, alhough the pathogenesis has not been completely established. Precipitation of calcium phosphate salts in the soft tissues occurs when the solubility of the calcium-phosphorus product reaches a critical value, generally greater than 70. Classically, the commonest cause has been severe uncontrolled hyperparathyroidism. However, in our patient, tumoral calcinosis was secondary to persistent hyperphosphoremia and was due to an iatrogenic hypercalcemia (excessive administration of calcitriol or calcium carbonate). Today, the elevated calcium-phosphorus product represents the main etiology of tumoral calcinosis in the dialysis patient. Less frequent causes include aluminum intoxication, secondary to a bone mineralization defect (Zins et al. 1992, Fernández et al. 1993).

The lesion consists of an encapsulated soft-tissue mass, of variable diameter (up to 20 cm) having fibrovascular septa. The surrounding areas contain yellowish-white calcified pasty material composed of calcium phosphate, calcium carbonate or a mixture of these. Microscopy shows an amorphous calcified material with macrophages and some multinucleated giant cells. In some cases, the calcified material forms small psammoma body-like masses (Steinbach et al. 1995).

Conventional radiographs show large homogeneous masses of lobulated appearance, confluent and separated by thin fibrous septa that give the mass a 'chicken wire' appearance. The masses are periarticular and the joint spaces are maintained. CT discloses the presence of fluid-calcium levels (sedimentation sign) and MRI displays multiple cavities of low signal density on T1- and T2- weighted sequences (Geirnaerdt et al. 1995, Steinbach et al. 1995).

Clinically, the patients present painless periarticular round tumors that have a firm consistency and grow progressively. Usually, large joints are involved and, less frequently, hands and feet. Decreased joint mobility, nerve compression or ulceration of the overlying skin, sometimes with discharge of chalky milky fluid, appear with large tumors (Asunción and Tzarnas 1994, Geirnaerdt et al. 1995).

The basic treatment of uremic tumoral calcinosis consists of correct control of the calcium-phosphorus product metabolism, together with excision of the tumors when they cause compression. The induction of a negative calcium balance by an increase in the number of dialysis sessions, with calcium-low dialysate, reduces the masses. Renal transplantation also leads to progressive reduction of the calcified masses (Fernández and Montoliu 1994).

Development of dialysis-related amyloidosis ($ß_2$-microglobulin deposit) in patients undergoing long-term dialysis is a well known cause of carpal tunnel syndrome and, less frequently, ulnar nerve compression (Gilbert et al. 1988, Konishiike et al. 1994). However, nerve compression due to tumoral calcinosis is uncommon in dialysis patients. Tumoral calcinosis causing compression of the ulnar nerve in Gunyon's canal has been described in 2 patients with systemic scleroderma (Thurman et al. 1991, Chammas et al. 1995) and a carpal tunnel syndrome secondary to tumoral calcinosis has been reported in one patient (Weiber and Linell 1987). However, no renal failure was observed in any of these 3 patients.

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Lipoma arborescens with osteochondral metaplasia—a case mimicking synovial osteochondromatosis in a lateral knee bursa

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A 52-year-old woman for 4 years had noted a slowly growing swelling of the right knee. There was no history of trauma. Physical examination revealed a firm, fixed, tender mass, measuring 6 × 7 cm over the lateral part of the knee. Knee motion was normal without crepitus. Routine laboratory studies were normal. Serological tests for rheumatoid factor were negative. Needle aspiration of the lesion yielded sterile, clear, yellow fluid.

Radiography showed a calcified soft tissue mass lateral to the knee, but no arthrosis. MRI revealed a well-defined cystic lesion which contained a villous fatty area interspersed with fluid. Chondromatous changes showing intermediate signal intensity on both T1 and T2 weighted images were seen in the fatty tissue (Figure 1). Arthrography showed no communication with the mass. Arthroscopy revealed no intraarticular abnormalities.

At operation, the mass was found under the iliotibial tract. It extended posteriorly between the fibular collateral ligament and the popliteus muscle. No communication with the knee joint or the proximal tibiofibular joint could be found. The patient had no recurrence 4 years after the marginal excision.

The excised specimen was a fluid-filled cystic synovial lesion with 5 loose bodies. The luminal surface

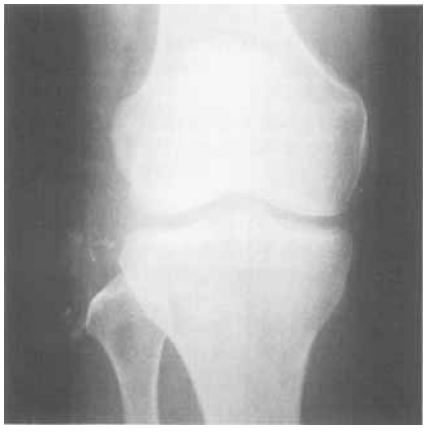
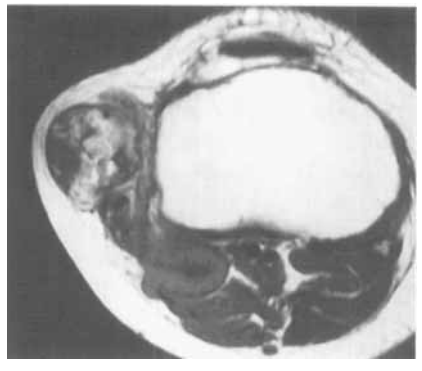


Figure 1. A. Extraarticular radiolucent mass with calcified nodules.



B. Transverse T1 weighted MR image of a distended lateral knee bursa shows villous synovial proliferation with tissue identical to subcutaneous fat in signal intensity, interspersed with fluid. Cartilaginous islands are embedded in the fatty tissue.