soft tissue mass. Radiographs may show a soft tissue mass with or without calcification. Synovial chondromatosis is unusual in the hand, the calcific mass being in close proximity to a joint. Traumatic lesions, such as myositis ossificans, can present as intramuscular calcific densities, but this is rarely seen in the hand. Systemic and metabolic conditions can be associated with soft tissue calcification, including chronic renal failure, hyperparathyroidism, hypercalcemia, vasculitis disorders, and scleroderma.

The majority of vascular hand lesions can be diagnosed with a thorough history and complete physical exam. Additional diagnostic tests include Doppler ultrasonic flow detection, radioisotope angiography, and contrast arterial angiography. Plain radiographs may assist in confirming the diagnosis.

The majority of vascular tumors of the hand are benign and can be treated with observation. Other reported operative therapies for these tumors include sclerosing agents, ionizing radiation, and cryotherapy. Results with these methods have been generally poor. Several authors have recommended surgical excision for symptomatic vascular tumors of the hand.

The same general principles that apply to other types of hand surgery apply to removal of tumors. Care must be taken to protect small digital arteries and nerves that often lie in close proximity, if not adherent, to the tumor. Tributary vessels should be identified and ligated as far away from the tumor as practical. When the tumor involves the overlying dermis, it is often necessary to excise the skin superficial to the tumor.

This patient’s cavernous hemangioma is unique in its radiographic appearance. While the radiographic presence of phleboliths is pathognomonic of a vascular tumor, typically they are small, punctate calcifications within the lesion. In this case, radiographs showed a rather large, soft tissue calcification, which was atypical of a phlebolith.

It is the purpose of this report to document the unusual radiographic appearance of a hemangioma within the hand, alerting the practicing orthopedic surgeon to the differential diagnosis if confronted with a similar soft tissue calcific mass in the hand.

References

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Bilateral Capitellar Steroid-Induced Avascular Necrosis
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Atraumatic avascular necrosis has been attributed to many etiologic factors, including alcoholism, diabetes, and sickle cell disease. However, steroid use is one of the more common factors implicated. 1 Avascular necrosis secondary to steroid use tends to affect many anatomic locations including, in decreasing order of frequency, proximal femur, proximal humerus, distal femur, and proximal tibia, as well as other miscellaneous sites. 2, 3 Involvement of the distal humerus—specifically, the capitellum—has been reported infrequently. 4, 5 Bilateral distal humerus involvement associated
with renal transplantation and steroid use has been reported. However, details are lacking about the exact anatomic location and extent of the involvement. We present a case of bilateral steroid-induced capitellar avascular necrosis.

**CASE REPORT**

A 37-year-old woman presented with a history of dermatomyositis of 5 years' duration. She had been treated with prednisone, with a maximum dose of 120 mg/day. She had bilateral femoral head avascular necrosis, which had been treated by bilateral total hip arthroplasty. At presentation she reported increasing right elbow pain and stiffness, for 11 months, with nocturnal awakening. She denied having any symptoms referable to her left elbow. Her elbow pain had been treated elsewhere by a period of immobilization, with no relief.

On examination, the neurovascular findings were within normal limits. Her shoulders had a full range of motion bilaterally. The range of motion of her right elbow was: 30° short of full extension with flexion to 115°; supination, 45°; and pronation, 40°. For her left elbow: hyperextension by 10° with flexion to 135°; supination, 90°; pronation, 90°.

Her current medications included azathioprine (Imuran) and salsalate (Disalcid). She had been off oral prednisone therapy for 3 weeks at presentation.

Plain radiographs revealed sclerotic changes and fragmentation of the capitellum and a portion of the trochlea involving the right elbow (Fig 1). The left elbow was normal radiographically (Fig 2). Tomograms revealed the sclerosis and fragmentation of the articular surface of the distal humerus, specifically the capitellum. A bone scan revealed no additional sites of involvement. Magnetic resonance imaging (MRI) of the elbows revealed the involvement of the subchondral bone of the capitellum and a portion of the trochlea bilaterally (Fig 3).

Arthroplasty was performed with resurfacing and a Pritchard ERS total elbow prosthesis (Fig 4). Intraoperatively, erosive changes of her cartilage with frank loosening and fragmentation of the cartilage and subchondral bone over the capitellum were seen. The radial head had eburnation of the articular cartilage. In addition, the lateral one third of the trochlea was fragmented with dissociation of the articular surface from the underlying bone.
Fig 3: MRI shows involvement of capitellum and lateral third of trochlea of the right elbow (A, B) and involvement of the capitellum and trochlea of the left elbow (C, D).

Fig 4: Radiographs after total elbow arthroplasty.

Fig 4A.

Fig 4B.

Fig 4C.

The patient was seen approximately 22 months following surgery. With respect to her left elbow, her radiographs continued to be normal. She had no symptoms attributable to the left elbow, had full range of motion, and no difficulties thus far.

**DISCUSSION**

Atraumatic avascular necrosis has been associated with many etiologic factors. Steroid use is second only to alcoholism as an associated factor. Several theories regarding the mechanism by which steroids cause avascular necrosis have been proposed: 1) a cortisone-induced hypercoagulable state associated with vasculitis, resulting in sludging; 2) a cortisone-induced osteoporosis, resulting in trabecular fracture and compression of subchondral vessels; and 3) a cortisone-induced fatty liver with hyperlipidemia, leading to systemic embolization of fat and avascular necrosis of bone.

Certainly, the femoral head is affected most commonly, as illustrated in this patient who had bilateral total hip arthroplasties for avascular necrosis. However, reports of the involvement of the distal humerus have been sporadic. Atraumatic avascular necrosis of the femoral head has been reported to occur bilaterally in nearly 90% of cases. Therefore, bilateral capitellar involvement could be anticipated. Our MRIs document this. Multiple joint necrosis is not unusual, and occurred in our patient.

Surgical treatment options in this patient included radial head excision, hemiarthroplasty, and total elbow arthroplasty. When intraoperative inspection revealed eburnation of the radial head and fragmentation of the capitellum and lateral third of the trochlea, resurfacing total elbow arthroplasty was the apparent choice to remove diseased tissue and to maintain maximal bone stock in the event revision might be needed in the future, particularly in a young patient.

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