Avascular Necrosis of the Hamate Treated With Capitohamate and Lunatohamate Intercarpal Fusion

GEORGE A. MAZIS, MD; VASILEIOS I. SAKELLARIOU, MD, PHD; ZINON T. KOKKALIS, MD

abstract

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This article describes a case of a 58-year-old man with no significant underlying disease who had chronic pain due to osteonecrosis of the hamate. Following physical examination, imaging, and laboratory findings, he underwent surgical exploration via a limited dorsal approach. The hamate bone did not have a normal anatomical appearance or structure. It was marble-like in appearance, soft, friable, and lacking physiologic contour and bone strength. We removed the dorsal aspect of the hamate piece-meal. The articular surfaces of the adjacent carpal bones (capitate and lunate) were excised, and the lesion was packed with iliac autograft. Three months postoperatively, fusion of the remaining hamate and the adjacent capitate and lunate carpal bones was evident, and the patient reported mild wrist pain, moderate grip strength improvement, and mild improvement of wrist range of motion and stiffness.

Currently, no standard treatment algorithm exists for patients with avascular necrosis of the hamate. However, delayed diagnosis of this clinical entity can be debilitating. A high index of clinical suspicion and an early course of treatment offers considerable benefits to patients with osteonecrosis of the hamate.

Drs Mazis, Sakellariou, and Kokkalis are from the Department of Orthopedics, School of Medicine, National and Kapodistrian University of Athens, Attikon University General Hospital, Athens, Greece. Drs Mazis, Sakellariou, and Kokkalis have no relevant financial relationships to disclose.

Correspondence should be addressed to: George A. Mazis, MD, Department of Orthopaedic Surgery, University of Athens, Medical School, Attikon University General Hospital, 1 Rimini Street, 12462, Chaidari, Greece (mazis.giorgos@gmail.com).

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Avascular necrosis of the carpal bones is a rare cause of wrist pain and is associated with limited range of motion (ROM), weakness, and the development of arthritis as the endpoint. Few cases of hamate osteonecrosis have been reported. Magnetic resonance imaging (MRI) is the diagnostic tool of choice because it is characterized by high sensitivity and specificity rates for the detection of early phases of avascular necrosis, offering the advantage of early diagnosis at a precollapse stage of the carpal bones. Its sensitivity allows for differentiation of subtle changes in the bone marrow signal. Several surgical options are associated with successful outcome; however, the whole course of treatment is challenging and unpredictable.

The cause of idiopathic avascular necrosis is probably multifactorial; corticosteroids, alcohol, hemoglobinopathies, systemic disorders (eg, systemic lupus erythematosus and Gaucher’s disease), and dysbarism are well-known causative factors. Cumulative stress is probably not a key factor in the pathogenesis of avascular necrosis. However, several cases were classified as idiopathic, with no directly identifiable etiology.

CASE REPORT

This study was approved by the Institutional Review Board. A 58-year-old man with no significant underlying disease presented with unremitting chronic right wrist pain. He had no history of trauma of the right wrist. He reported no fever or hand, wrist, or forearm numbness. His history was uneventful. He was taking no medications and reported no allergies, smoking, or alcohol or illicit drug use. A review of systems was otherwise unremarkable.

For 2 years, he was treated conservatively with splints and physiotherapy and received several regimens of nonsteroidal anti-inflammatory drugs (NSAIDs) but had never been treated with steroid injection.

On physical examination, the patient had tenderness over the hypothenar eminence. Right wrist ROM was 20° of dorsiflexion to 20° of palmar flexion. Supination and pronation of the wrist were not affected, ranging from −90° to +90°. Grip strength was diminished, and his forearm muscles were wasted. On examination, he had tender swelling on the ulnar aspect of his right wrist.

Laboratory peripheral blood studies showed that complete blood count, erythrocyte sedimentation rate, and C-reactive protein (CRP) were within normal ranges.

Radiographically, mixed radiolucent and sclerotic areas existed in the bone of the hamate, with dominance of the radiolucent lesion (Figure 1). Computed tomography (CT) scan of the wrist, which was performed with a high-resolution protocol with 1-mm slices parallel to the longitudinal axis of the forearm, showed an extended lucency of the hamate forming a pseudocyst and sclerosis of the surrounding cancellous bone. Magnetic resonance imaging revealed focal central hypointense bone marrow edema with marrow fat inhomogeneity of the hamate in coronal plane of the T1-weighted MRI sequence (Figure 2). In addition, a marrow edema was revealed as hyperintense signal in the coronal plane in the T2-weighted MRI sequence (Figure 3).

The patient underwent surgical exploration with preservation of the dorsal branch of the ulnar nerve. The procedure was performed via a limited dorsal approach directly over the body of the hamate. After the identification of the dorsal cutaneous branch of the ulnar nerve, the retinaculum overlying the fifth dorsal compartment was sharply incised. Extensor digiti quinti tendon was retracted radially, and a radially based capsular flap over the hamate was raised. The hamate bone did not have a normal anatomical appearance and structure. It was marble-like in appearance, soft, friable, and lacking physiologic contour and bone strength.

We removed the dorsal aspect of the hamate piecemeal. The volar cortical plate was preserved to protect the deep branch of the ulnar nerve and retain the space of the defect. The articular surfaces of the adjacent carpal bones (capitate and lunate) were excised, and the lesion was packed with iliac cancellous bone autograft. The reconstructed hamate was fixed using two 1.5-mm Kirschner wires with the adja-
cent capitate and lunate carpal bones. The wound was closed in anatomical layers. The dorsal capsule was closed using 2-0 Ethibond braided nonabsorbable sutures (Ethicon, Somerville, New Jersey) and figure-8 sutures. The subcutaneous tissue was closed with 3-0 Polysorb absorbable sutures (Tyco Healthcare UK Ltd, Gosport, United Kingdom), and the skin with interrupted horizontal mattress sutures of 5-0 Prolene monofilament nylon (Ethicon).

Postoperatively, the wrist was protected with a cast for 4 weeks. Kirschner wires were removed 6 weeks postoperatively. The patient was encouraged to do ROM and grip-strengthening exercises as tolerated.

Three months postoperatively, he described his wrist pain as mild and reported moderate grip strength improvement and mild improvement of wrist ROM (30° of dorsiflexion to 30° of palmar flexion) and stiffness. His postoperative Disabilities of the Arm, Shoulder, and Hand (DASH) score was 11.7, whereas his preoperative DASH score was 25.

Fusion of the remaining hamate and the adjacent capitate and lunate carpal bones was evident radiographically (Figure 4).

**Discussion**

Avascular necrosis of the hamate is uncommon and can be debilitating when left untreated. The few reported cases of hamate avascular necrosis show a relation with major or repetitive minor trauma.3,6,7

Studies of the vascular anatomy of the hamate demonstrate that blood supply to the proximal pole of the hamate is entirely intraosseous from the distal pole because the wedge-shaped proximal pole of the hamate is almost entirely intra-articular.2,5 Panagis et al5 described the vascular supply of the carpal bones and classified them according to their relative risk for avascular necrosis. The hamate and trapezoid were categorized as group II bones because they lack intraosseous anastomoses but are considered to be at less risk for avascular necrosis because they have >1 nutrient vessel.5

The palmar vessels are branches that originate from the palmar carpal and deep palmar branches of the ulnar artery. The dorsal vessels originate from nutrient vessels of the dorsal carpal artery. The latter is a branch of the ulnar artery or a perforating branch of the anterior interosseous artery. The proximal segment is supplied solely by intraosseous vessels that originate from the dorsal and palmar vessels anastomoses into an arcade. Therefore, fractures proximal to this arcade, which commonly involve the proximal pole, may place this segment at risk for vascular compromise.

In our patient, surgical treatment followed by cast immobilization and ROM and grip-strengthening exercises resulted in a positive outcome. However, due to the limited number of similar cases, no standardized treatment algorithm exists for these patients.

Van Demark and Parke4 reported a case of a 45-year-old man who sustained a crush injury of the hand leading to nondisplaced scaphoid, capitate, and transverse hamate fractures. Wrist bone scan showed abnormal vascular status of the proximal hamate fragment. The authors followed conservative treatment by casting for 3 months. The capitate and scaphoid fractures healed; however, the hamate fracture was delayed in healing, and radiographs revealed evidence of proximal pole avascular necrosis. This patient reported limited ROM and pain at 1 year but declined surgery because he was able to return to work with no serious limitations.4

Telfer et al6 reported the case of a 16-year-old boy with wrist pain after falling on a flexed wrist while playing football. Radiographs 5 months after injury showed an abnormal trabecular pattern of the hamate. Bone scan showed an increased uptake of technetium-99. The patient reported persistent wrist pain radiating to the forearm, grip weakness, and limited ROM after conservative treatment with splinting and physical therapy. Radiographic evaluation at 18 months revealed sclerotic and lucent regions in the hamate, and MRI confirmed avascular necrosis of the hamate. Capitohamate intercarpal fusion with the use of iliac crest bone graft was performed 2 years posttrauma, and the patient was pain free, with slight limitation of wrist ROM 1 year postoperatively.6

Tukenmez et al7 reported a case of a 25-year-old man with osteonecrosis of the hamate. He reported pain and swelling in his right wrist. The diagnosis was accomplished with plain radiographs and MRI. The case was treated surgically and included resection of the necrotic bone. The cavity was filled with autogenous cancellous bone graft. In addition, capitohamate arthrodesis was performed. Postoperative histopathological examination demonstrated avascular necrosis of the hamate. The arthrodesis was obtained 4 months postoperatively.3

Juon et al7 reported a case of avascular necrosis of the hamate in a young boy after repetitive stress in his left wrist. After progression of the necrosis with cyst formation on follow-up MRI and persisting pain, the patient underwent successful revascularization with a vascular bone graft of the distal radius.7

Currently, no standardized treatment algorithm exists for patients affected by avascular necrosis of the hamate. However, delayed diagnosis of this clinical entity can be debilitating. A high index of clinical
suspicion and an early course of treatment can offer a considerable benefit in patients with avascular necrosis of the hamate.

REFERENCES


