Avascular Necrosis of the Capitate

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ABSTRACT

Avascular necrosis of the capitate is a rare entity. The most common reported etiology is trauma. We report a case of avascular necrosis of the capitate in a patient with chronic wrist pain that began after a single episode of remote trauma.

CASE REPORT

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32 year old woman presented to the hand clinic with chronic wrist pain seeking a second opinion for treatment of avascular necrosis of the capitate. Aside from a 12 pack year smoking history, she sustained a closed decelerating wrist injury in a motor vehicle accident 14 years previously. She has experienced intermittent pain since that time treated by non-steroidal anti-inflammatory drugs and opiates. The initial diagnosis was a carpal bone fracture. Recently, the pain had increased, severely limiting wrist motion.

Radiographs of the wrist showed collapse and fragmentation of the proximal pole of the right capitate, with increased density of the mid to distal pole (Fig 1). MR imaging of the wrist, showed a low signal intensity bone fragment at the proximal capitate and heterogeneous predominately intermediate signal of the mid to distal pole on both T1-weighted and intermediate-weighted images (Fig 2a, b). Fat-suppressed T2-weighted images showed corresponding low signal of the proximal pole fragment and heterogeneous high signal involving the distal pole (Fig 3). Findings were consistent with avascular necrosis of the capitate. Surgery confirmed a necrotic, collapsed proximal pole of the capitate.

DISCUSSION

Avascular necrosis (AVN) of the capitates is a rare clinical entity, first reported by Jonsson in 1942 [1]. Since then, there have been approximately 30 cases reported in the literature [2]. The infrequency of AVN of the capitate compared with the scaphoid is thought to be related to its relatively protected position in the center of the distal carpal row. Although the majority of reported cases have been associated with an acute episode of trauma, other reported etiologies have included repetitive micro-trauma, dorsal instability, steroid injections, and gout [2, 3, 4]. Fracture through the waist of the capitate, together with a fracture through the scaphoid, the so-called naviculo-capitate fracture syndrome, has been reported to predispose to avascular necrosis of the capitate [2, 5, 6]. In this entity, the proximal pole of the capitate may be rotated up to 180 degrees, further complicating the healing process. True idiopathic cases are rare, and it is speculated that underlying predisposing factors such as anomalous blood supply, ligamentous instability, and unrecognized or repetitive trauma are thought to be factors, leading to a final common pathway of inadequate blood supply to the capitates [2, 3, 7, 8].

The external vascular supply of the carpus is supplied by transverse arches formed by the anastamosis of the radial, ulnar and anterior interosseous arteries. Three patterns of intraosseous blood supply have been described, with vessels entering the bone at ligamentous attachments on the palmar surface, and to a varying degree from the dorsal surface [6, 8]. In all patterns, blood supply to the proximal pole is in a retrograde fashion. Thus, the vascular supply to the proximal pole of the capitate is tenuous, and analogous to the vascular supply to the scaphoid. The head of the capitate has no collaterals, thus is at further risk for AVN [9].

In our case, imaging showed a fracture with fragmentation of the proximal pole. Because of the chronic nature of the

injury, it cannot be determined whether the fracture occurred at the initial time of injury or whether it is a result of the avascular necrosis. In addition, because of significant fragmentation of the proximal pole, the degree of rotation could not be assessed, thus the possibility of association with prior fracture of the scaphoid, the "naviculo-capitate" fracture syndrome, could not be determined. Although this would have been a possibility, surgery confirmed the scaphoid was not fractured.

The early stages of avascular necrosis are difficult to detect using conventional radiographs. It has long been known that MR imaging is more sensitive than radiographs in the detection of AVN [10, 11]. Prior studies involving MRI for avascular necrosis have predominately focused on the scaphoid and lunate, as these are more commonly involved with AVN, but similar findings would be presumed for the capitate, as well. Sensitivity is increased with the use of gadolinium intravenous contrast to evaluate the presence or absence of enhancement, suggesting viability [12]. The presence of low signal intensity on T1-weighted images has been shown to a quite sensitive sign of AVN of the scaphoid [13]. Murakumi et al. reported MR imaging of a case of avascular necrosis of the capitate that showed low-signal intensity areas on T1-weighted images and high-signal-intensity areas on T2 images, corresponding to an area of revascularization and regeneration noted on angiography and histologic examination [4]. However, in recent studies involving the scaphoid, signal intensity on T2-weighted or STIR images, and enhancement on T1-weighted fat suppressed images have proven to be quite variable and not as helpful a determinate of AVN [12-14]. Thus, low signal intensity on T1-weighted images is the most reliable indicator of the presence of AVN. Lapinsky and Mack reported a low-signal-intensity lesion on T1-weighted MR imaging corresponding to avascular necrosis of the capitate bone [15].

In our case, the proximal pole of the capitate demonstrated extremely low signal intensity on both T1 and T2-weighted images of the wrist. By all known standards and grading systems, this corresponds to a late stage of avascular necrosis. The more heterogeneous signal intensity within the distal pole is of less certain significance, the possibilities including moderate to severe ischemia versus revascularization or reparative process.

Several surgical treatments have been described in the literature, including interposition arthroplasty using tendon graft, intercarpal arthrodesis, silicone athroplasty, resection of the proximal pole and drilling, and iliac crest bone grafting. Recently, Hattori et al have reported the technique of harvesting a vascularized pedicled distal radius bone graft based on the fourth extensor compartmental artery (ECA) with retrograde flow through the fifth ECA from the dorsal intercarpal arch and inserting it into the capitate from a dorsal approach, with revascularization of the capitate being evident on follow up MRI reported by one author [6, 16]. In addition, it is important to unload the capitate during the revascularization period either by application of an external fixator or temporary pinning of the midcarpal joint.

Interestingly, Moran retrospectively reviewed MRI results of vascularized pedicle graft for lunate avascular necrosis of 26 patients. Normalization of T2-weighted values was seen in the first 3 to 6 months. This was followed by normalization of T1-weighted values at 18 to 20 months [17].

Our 32 year old patient was strongly advised to quit smoking, and she subsequently underwent vascularized distal radius bone grafting based upon the 4th ECA with retrograde supply via the 5th ECA and temporary capitate unloading with .065 in. K- Wires from the distal scaphoid to the distal capitate. Results are pending.

We have reported a case of avascular necrosis of the capitate, which was presumably secondary to a remote episode of trauma. In our case, it is possible that this may have been present and unrecognized over the many years during which the patient experienced chronic pain.

TEACHING POINT

Avascular necrosis of the capitate is a rare entity thought to be related to its relatively protected position in the center of the distal carpal row. It should be a consideration in the evaluation of wrist pain, even in the absence of recent trauma.

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FIGURES

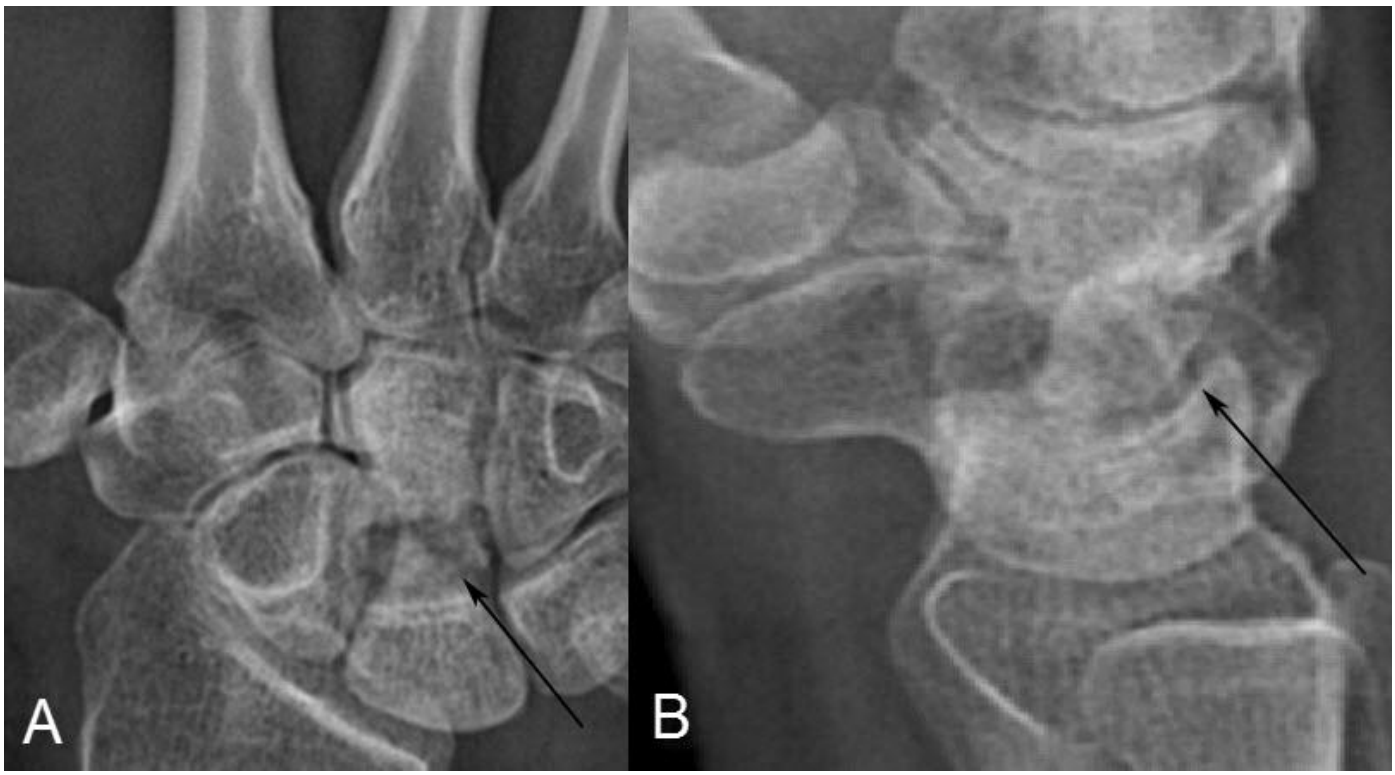

Figure 1: 32 year old woman with avascular necrosis of the capitate. PA radiograph (a) showing increased density in the distal capitate corresponding to the zone of avascular necrosis. Probable fracture and fragmentation is better demonstrated in the lateral view (b).

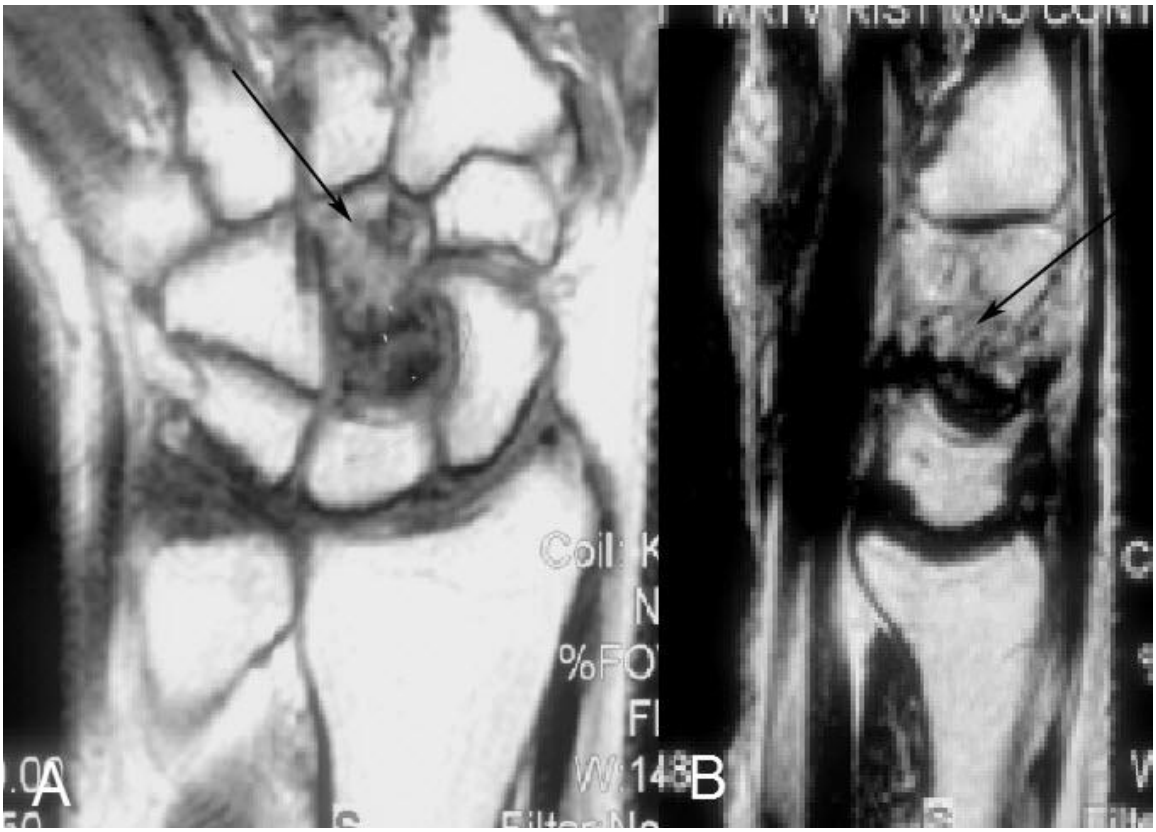

Figure 2: 32 year old woman with avascular necrosis of the capitate. Coronal T1-weighted without contrast (TR:500 ms) (a) and sagittal intermediate-weighted without contrast (TR:3884ms) (b) images show low signal intensity in the proximal capitate and heterogeneously low signal in the distal pole.

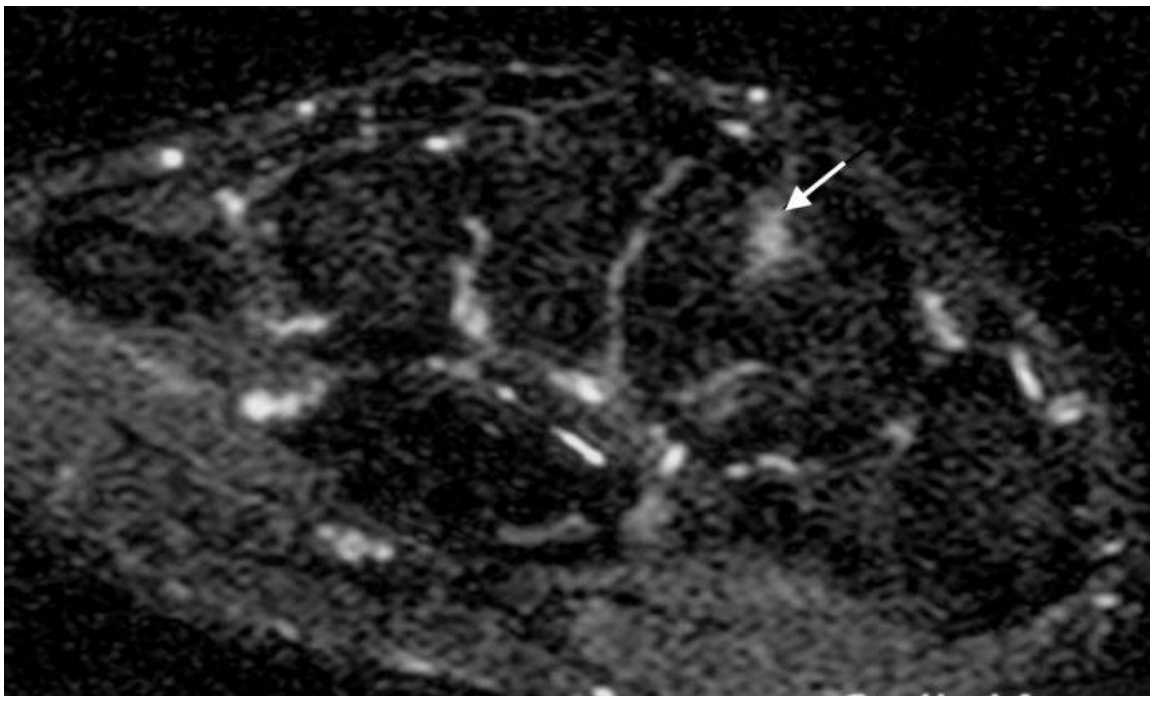

Figure 3: 32 year old woman with avascular necrosis of the capitate. Axial T2-weighted fat saturated without contrast (TR: 6867ms) images show mild areas of high signal intensity in the distal capitates (arrow).

Incidence	Rare entity with approximately 30 reported cases in the literature
Gender Ratio/Age Predilection	No gender or age predilection
Etiology	Anomalous blood supply and ligamentous instability with a final pathway of inadequate blood supply to the capitate
Risk Factors	Acute episode of trauma, repetitive micro-trauma, dorsal instability, steroid injections, smoking, pregnancy and gout.
Treatment	Interposition arthroplasty using tendon graft, intercarpal arthrodesis, silicone athroplasty, resection of the proximal pole and drilling and iliac crest bone grafting. However, from the number of various surgical treatment options that no consensus exists even in treating the same stage of the disease.
Prognosis	Dependent on stage of diagnosis with reported surgical outcomes of full function of the wrist with minimal to no pain and advanced osteoarthritis with lunocapitate fusion.
Imaging Finding	Plain radiograph may demonstrate increased density in the distal capitates, fractures or fragmentations corresponding to the zone of avascular necrosis. T1 weighted MRI sequence may show low signal intensity in the proximal capitate with T2 weighted MR sequences demonstrating high signal intensity in the zone of avascular necrosis.

Table 1: Summary table of avascular necrosis of the capitate.

	X-Ray	MRI T1	MRI T2
Avascular necrosis of the Capitate	Increased density in the distal capitates, fractures or fragmentations corresponding to the zone of avascular necrosis	Low signal intensity in the proximal capitate	High signal intensity in the zone of avascular necrosis.
Subchondral insufficiency fracture	Sclerotic band along the joint line	Subchondral, slightly irregular, low signal line with edema between the line and the joint	High signal between the fracture and joint space especially in fluid sensitive sequences
Subchondral cyst	Subchondral well defined cystic lucencies	Subchondral low signal cystic space, often follows fluid signal	Subchondral high signal cystic space, often follows fluid signal

Table 2: Differential diagnosis table for avascular necrosis of the capitate.

ABBREVIATIONS

AVN: Avascular Necrosis
MR: Magnetic Resonance
ECA: extensor compartmental artery
STIR: Short TI Inversion Recovery

KEYWORDS

Avascular Necrosis; Capitate; Scaphoid; Vascular Supply Carpus; Stages Avascular Necrosis; Extensor Compartmental Artery; Distal Radius Bone Graft