Avascular Necrosis of the Trapezoid Bone Following Carpometacarpal Arthroplasty

PATRICK KANE, MD; GREG WARYASZ, MD; JULIE KATARINCIC, MD

ABSTRACT

A 58-year-old female developed avascular necrosis of her trapezoid approximately 3 months after undergoing carpometacarpal arthroplasty. The patient was treated conservatively with immobilization and had complete resolution of her clinical symptoms during her year of follow-up. Additionally, radiographic examination showed complete restoration of the height of her trapezoid approximately 1 year after the index procedure. Avascular necrosis of the trapezoid is extremely rare with very few cases described in the literature. This is the first description of avascular necrosis following carpometacarpal arthroplasty.

KEYWORDS: Carpal Bone Avascular Necrosis, Carpometacarpal Arthroplasty, Surgical Complication

INTRODUCTION

Avascular necrosis of the carpal bones is rare, with the most frequently reported cases involving the scaphoid, lunate and capitate.[1] We present a case report of avascular necrosis of the trapezoid following carpometacarpal arthroplasty. The patient developed symptoms approximately 3 months post-operatively and with conservative treatment the patient had full resolution of her symptoms.

CASE REPORT

A 58-year-old woman was treated with a right thumb carpometacarpal arthroplasty with flexor carpi radials tendon transfer in August 2011 for stage III CMC arthritis. Prior to surgical intervention the patient had failed conservative treatment with splinting and a corticosteroid injection given in April 2011. The patient tolerated the procedure well, there were no intraoperative complications. However, at 12 days post-op the patient presented complaining of increased pain and erythema around the surgical wound and the patient was started on a 10-day course of Keflex for presumed treatment of a superficial cellulitis; the erythema resolved uneventfully. The remainder of her course is benign. She started hand therapy at 4 weeks post-op. X-rays at 4 (**Figure 1**) and 7 weeks were unremarkable with standard post surgical changes observed.

At 15 weeks (**Figure 2**) the patient returned to the office complaining of increased acute onset of pain in her wrist; x-rays taken at this time showed increased sclerosis and

Figure 1. AP radiograph of right wrist 4 weeks post-op from CMC arthroplasty.

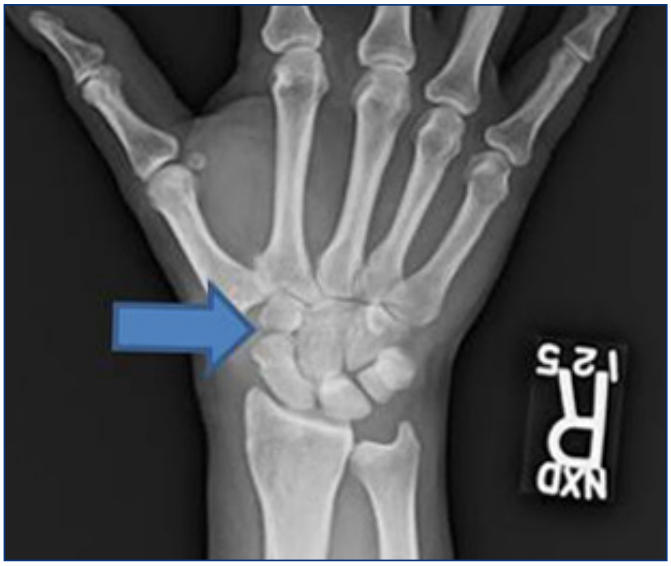


Figure 2. AP radiograph of right wrist 15 weeks post-op from CMC arthroplasty.

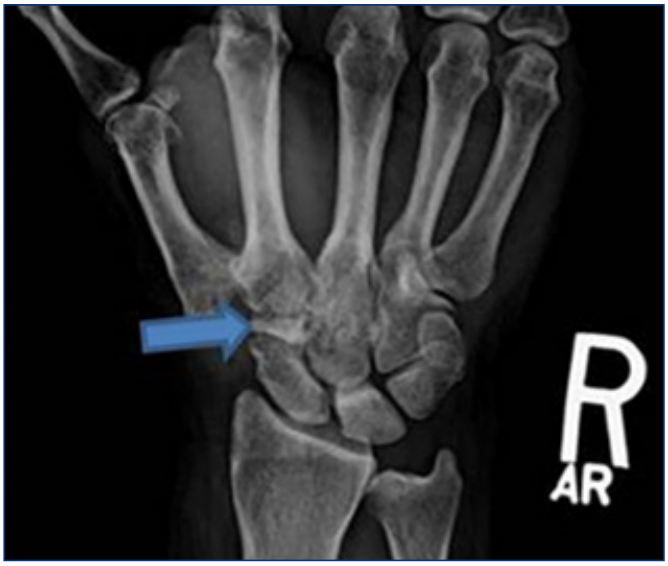


Figure 3. MRI of right wrist showing avascular necrosis of trapezoid.

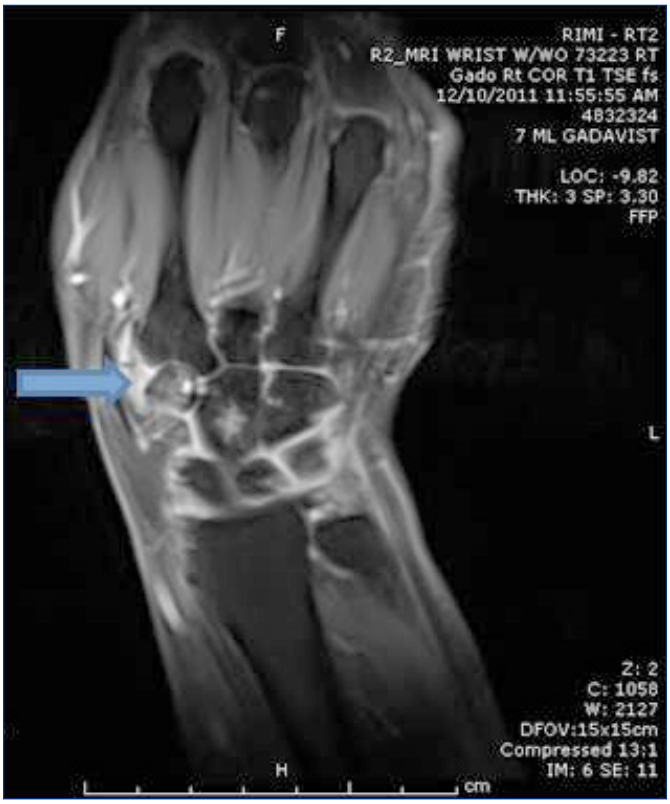
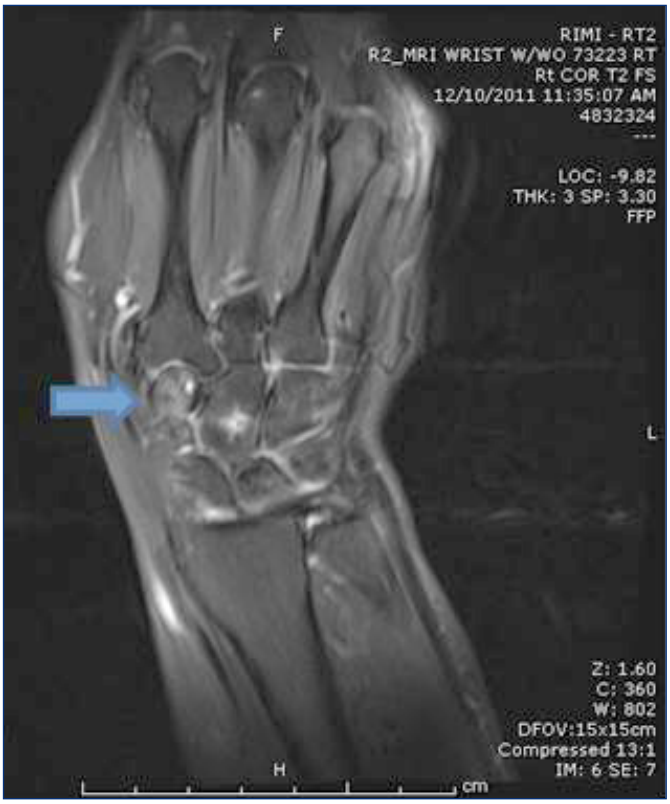


Figure 4. Coronal T1 with Gado and Coronal T2.



collapse of the trapezoid. Given the radiographic findings an MRI of the carpus was obtained which showed imaging consistent with avascular necrosis of the trapezoid (**Figure 3 and 4**).

The patient was treated conservatively with splinting for comfort, and gradually the patient's symptoms improved. The patient was last seen in follow-up August of 2012, repeat x-rays (**Figure 5**) were obtained which showed restoration of bone height and reconstitution of the trapezoid. Patient has been clinically asymptomatic since March 2012.

Figure 5. AP radiography of right wrist approximately 1 year post-op from CMC arthroplasty.

DISCUSSION

Avascular Necrosis (AVN) of the carpal bones is rare. The most frequently reported cases involve the scaphoid, lunate and capitate.[1] Risk Factors for AVN include a history of trauma, alcohol intake and steroid medication use. In this case, there were no identifiable risk factors; however, there was a potential precipitating event. Other cases of AVN described in the literature had no identifiable cause.[2,3] Disruption of the blood supply from trauma resulting in enucleation of the trapezoid from its bed have been proposed as a mechanism for AVN[4,5]; however, even with an open dislocation and nearly all soft tissue attachments being stripped, there is no guarantee of AVN.[5] In our review of the literature, AVN of the trapezoid following CMC arthroplasty has not previously been described.

Most of the research into AVN has been extrapolated from the femoral head model. Death of bone is thought to be due from either arterial occlusion, venous congestion, elevated intraosseous pressure or microtrauma.[6] After the insult to the bone is complete, the bone begins angiogenesis to revascularize. The next step is resorption of dead bone. Since the articular surface is supplied nutrition from the synovial fluid, the bone's ability to rebuild its subchondral surface will determine the degree of collapse and fracturing.[6]

MRI is a capable imaging modality to determine AVN from other pathologies that can cause pain related to the trapezoid. Fracture, dislocation and arthritis are known pathologies, but there have also been reports of intraosseous ganglion[7] and coalition.[8] MRI of AVN can show subtle changes such as increased signal due to initial edema and then decreased signal as there is ischemia and necrosis.

In a cadaveric vascularity study by Gelberman et al. various vascular patterns of the carpal bones were correlated with the clinical incidence of avascular necrosis and at-risk patterns of vascularity were identified.[1,9,10] The carpal bones were divided into 3 groups, which correlated with risk pattern. The first group of carpal bones which were considered to be most at risk for post traumatic AVN consisted of the scaphoid, capitate and 20% of the lunates; all carpal bones had a large area of bone dependent on a single intraosseous vessel. The trapezoid was placed into group 2 along with the hamate. The authors concluded that although these bones were theoretically at risk because of the lack of internal anastomoses, they do not undergo AVN.

The trapezoid benefits from a varied extraosseous blood supply. Branches from the intercarpal, dorsal radiocarpal and basal metacarpal arches as well as the radial recurrent artery all contribute blood supply to the trapezoid. This rich supply of blood vessels may explain why AVN is such a rare occurrence in the trapezoid.[11]

AVN of the trapezoid in other case reports has been usually treated conservatively with casting/immobilization[3]; when this fails curettage, iliac crest bone graft, and core revascularization using graft from the dorsal metacarpal vascular bundle to the second ray has been used to achieve partial revascularization.[2] In our case, only immobilization was required to achieve imaging consistent with revascularization.

In our case, there was not a clear vascular injury or enucleation of the trapezoid from its soft tissue bed to explain the resultant AVN. Given the dual blood supply to the trapezoid, it is possible that our patient only had a single vessel supplying the trapezoid or some other vascular anomaly making it behave more like the lunate/scaphoid/capitate bone. Based on the imaging performed and the improvement in patient symptoms and plain radiographs, there can be no conclusion made as to the exact cause of the trapezoid AVN in our case after CMC arthroplasty.

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Disclosures

The authors have no conflicts of interest, did not violate any animal or human rights and ensured that identifying information for patients has been excluded with regards to this manuscript; additionally informed consent was obtained from the patient.

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