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Case Report

Avascular Necrosis of an Adolescent Distal Radius: A Literature Review

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A 12-year-old boy was tackled mid-air, resulting in a fall on the outstretched hand without fracture. The patient was treated conservatively but developed sharp pain and stiffness 6 months later. Imaging revealed distal radius avascular necrosis with physeal involvement. Owing to the injury chronicity and location, we treated the patient conservatively with hand therapy. After 1 year of therapy, the patient returned to normal activities without pain and with a resolution of findings on imaging. Avascular necrosis is more frequent in carpal bones (eg, Kienböck disease of the lunate and Preiser disease of the scaphoid). Growth arrest at the distal radius can lead to ulnocarpal impaction, triangular fibrocartilage complex injury, or distal radioulnar joint injury. In this case report, we discuss our treatment rationale and review the literature on pediatric avascular necrosis for hand surgeons.

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Distal radius fractures are the most common type of fracture in children aged <16 years; however, avascular necrosis (AVN) of the distal radius is rare.¹ Hand surgeons primarily encounter AVN in adults as Kienböck disease of the lunate or Preiser disease of the scaphoid. The etiology and pathophysiology of AVN are multifactorial.

Pediatric AVN at an open physis complicates management and requires additional considerations. Disruption of a physis can cause complete or partial growth arrest, leading to a limb-length discrepancy or limb angulation. Physeal AVN is most common in femoral head and neck fractures and as a sequela of slipped capital femoral epiphysis, for which surgeons may pursue operative reductions.

We present a case of chronic, posttraumatic distal radius AVN in an adolescent epiphysis, discuss our management rationale, and review the existing literature on upper extremity AVN. The patient was informed and consented to allow photographs and case data for publication.

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Case Report

A 12-year-old African American boy sustained a fall on the outstretched hand while playing football. While in mid-air, he was tackled, resulting in an extra-articular Salter-Harris class 2 fracture of the right radius. He was promptly taken to an outpatient hospital and treated conservatively with a short arm cast.

The patient had a history of phalangeal fractures and a thumb collateral ligament injury (Gamekeeper's thumb), both of which were sports-related injuries with appropriate mechanisms. There was no previous injury or deformity of the distal radius. The patient had his cast removed 8 weeks later and could return to playing sports owing to appropriate healing on follow-up examination and radiographs. No appreciable AVN or damage to the physis was present at that time. The patient intermittently used a standard cock-up wrist splint after cast removal.

One year after the initial injury, the patient presented to our orthopedic clinic complaining of increasing pain and stiffness in the right wrist that began approximately 6 months after his initial injury. The patient reported no new injuries to the wrist. On physical examination, the patient's pain was aggravated by axial wrist loading and direct palpation on the dorsal distal radius. The patient had limited wrist extension and flexion at 10° and 30°, respectively, and reduced radioulnar deviation by 50% but normal pronation and supination. The remainder of the examination was unremarkable.

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Figure 1. One year after injury, the patient presented without obvious deformity; however, MRI demonstrated signal changes in the distal radius and lunate consistent with AVN. **A** Posteroanterior wrist radiograph. **B** T1-weighted wrist MRI. **C** T2-weighted wrist MRI.

Plain radiographs revealed no signs of active pathology. Magnetic resonance imaging (MRI) scans were ordered for further evaluation, revealing AVN of the distal radius epiphysis (Fig. 1). After discussion, the parents and orthopedic surgeon agreed to pursue conservative management with dedicated hand therapy to improve range of motion. The patient was also advised to continue to use the cock-up wrist splint, and he was temporarily stopped from playing sports.

The patient was seen 2 months later with an improved range of motion. Wrist extension and flexion were now at 40° and 70°, respectively. Radiography was repeated and revealed no concerning changes or abnormalities. On the basis of these findings, we advised the patient and family to continue hand therapy and undergo a new MRI scan to evaluate the status of the epiphysis. This MRI scan revealed the ongoing resolution of AVN (Fig. 2).

At a follow-up visit 1 month later, the patient demonstrated continued progress on examination. The range of motion of the wrist was fully flexed to 75° and lacked 20° of terminal extension to

50°. The patient discontinued hand therapy appointments. At his final follow-up visit, approximately 18 months after the index injury, his motion was unchanged. Radiography demonstrated no interval changes at the wrist (Fig. 2). The patient and his parents were advised to continue home exercises and adjust his splint to maximize extension. The patient returned to full activity, including impact sports, and was satisfied with the clinical outcome.

Discussion

Avascular necrosis is a rare cause of chronic wrist pain in pediatric patients. In the order of decreasing frequency, AVN in carpal bones occurs in the scaphoid, capitate, hamate, trapezium, trapezoid, triquetrum, and pisiform. However, AVN of the distal radius has not been reported in a pediatric patient, and evidence about the management of secondary physeal involvement is limited. Therefore, we reviewed pertinent literature on AVN and have presented our management rationale for this case.

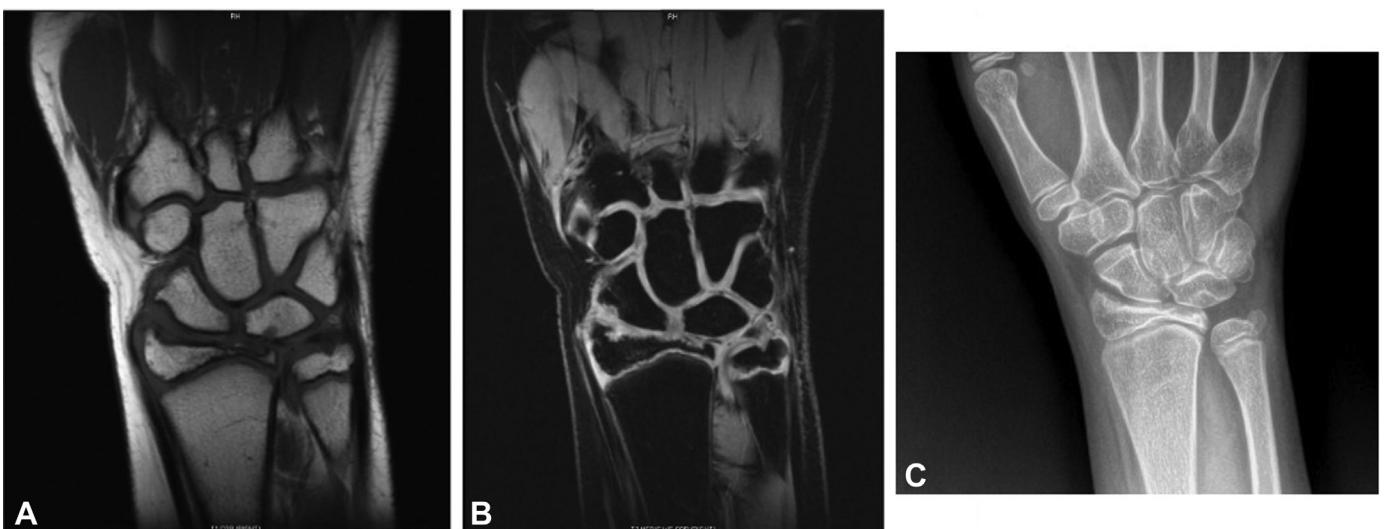


Figure 2. MRI 2 months into therapy revealed continuing resolution of AVN with no collapse or progression, and final radiographs 6 months into therapy demonstrated no acute changes. **A** T1-weighted wrist MRI. **B** T2-weighted wrist MRI. **C** Posteroanterior wrist radiograph.

The etiology of AVN is multifactorial, involving genetic predispositions, local vascular factors or injury, intraosseous pressure, and mechanical trauma. Our patient may have had any combination of these mechanisms. Repetitive athletic activities, such as those undertaken by our patient, are a known risk factor for AVN.² However, overuse or traumatic injury is not a prerequisite. Stroh et al³ reported on 10 wrists with atraumatic distal radius osteonecrosis in a case series. They found that high-dose corticosteroids were a risk factor for AVN of the distal radius, seen in 60% of reported cases.³ Autoimmune disorders and blood dyscrasias were also seen in the cases.³ Our patient did not have any risk factors identified in the literature, although autoimmune and blood diseases can be latent during adolescence.

Certain vascular patterns and venous outflow blockages in the lunate are predictors of Kienböck disease. By comparison, the redundant blood supply at the distal radius may account for the rarity of AVN in this location.⁴ In our case, however, the vascular supply is significantly altered with an active and open physis in an adolescent. The physis may have increased the likelihood of nonunion, through steal syndrome phenomena or otherwise, and progressed to AVN. In addition, because our patient was initially conservatively managed at a different institution, we cannot ensure that our patient was adequately immobilized. Prommersberger and Fernandez⁵ described that, in their experience with 23 distal radius nonunions, instability secondary to inadequate immobilization is likely related to nonunion.

A common presenting symptom of AVN is chronic pain. This pain often worsens with weight bearing or motion. However, some patients may be asymptomatic in the early stages, which may delay presentation, as in our case. Avascular necrosis has a typical progression pattern in the wrist on plain radiographs: normal appearance, sclerosis, fragmentation, cyst formation, fracture, bone collapse, wrist collapse, and degenerative joint changes. However, initial radiographs rarely detect earlier stages of AVN.⁶ The physis is more susceptible to injury than the surrounding bone because of differences in biomechanical properties, which may not be seen in radiography.⁷ Therefore, the gold standard for AVN diagnosis is MRI, regardless of location, with a sensitivity of up to 100%.⁸ Magnetic resonance imaging at the time of diagnosis showed well-demarcated initial focal lesions of AVN on T1-weighted images and hyperintense vascular granulation tissue on T2-weighted images. These findings constitute the “double-line sign,” pathognomonic of AVN.

Owing to the shortage of reports, the natural history of distal radius AVN is not well characterized. However, because AVN of both the scaphoid and lunate progresses, distal radius AVN will likely progress as well. With progression in mind, we provided treatment using the following principles: avoiding bone collapse, restoring anatomy, and reducing the risk of degenerative joint disease.⁹ We were fortunate to detect AVN in our patient at an early stage. With activity modification and splinting, we could see regression in his symptoms and bone healing on subsequent imaging. Our primary

concern with operative management was physeal compromise and worsening convalescence. However, had the disease progress failed to improve or worsened after 2 months dedicated to nonsurgical management, we would have counseled surgical treatment.

Early AVN is typically approached nonsurgically with nonsteroidal anti-inflammatory drugs and wrist immobilization. Hungerford and Jones¹⁰ investigated nonsurgical management of AVN and found that smaller lesions have a low chance of progression. They also suggested that surgical management should primarily be guided by lesion size and stage in symptomatic and asymptomatic patients.¹⁰ Stroh et al³ recommended nonsurgical management in their case series of atraumatic distal radius AVN. Nevertheless, they questioned the role of palliative percutaneous intralesional drilling to provide pain relief and return of function.³ However, our decision to manage conservatively was based on the location near the active physis. We felt assured in both our decision to manage conservatively and the ongoing resolution of AVN in this patient because of the reduction in symptoms and encouraging findings on imaging at follow up. Given these findings, we believed it was clinically appropriate to have him follow up as needed after his last visit, which was 6 months after the initial AVN diagnosis and 18 months after the initial index injury.

Pediatric AVN of the distal radius epiphysis has not been previously reported. We postulate that the unique anatomy of the growth plate predisposes a susceptibility to trauma, which can kink or injure retinacular vessels and compromise blood supply, leading to AVN. We managed the patient nonsurgically using activity modification and splinting and observed resolution in symptoms and imaging. Nevertheless, this case raises awareness that AVN can cause chronic progressive posttraumatic wrist pain in skeletally immature patients.

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