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Kienbock's disease in a varsity football player: a case report and review of the literature

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Objective: *To present the diagnostic, clinical features, and management of Kienbock's disease and create awareness of the differential diagnosis of this condition in patients presenting with insidious, progressive dorsal wrist pain.*

Clinical Features: *A 23-year old male varsity football player presented with insidious progressive dorsal sided wrist pain with reduced wrist flexion and extension. A diagnosis of Kienbock's disease was made based on radiographs and magnetic resonance imaging.*

Intervention and Outcome: *A 3mm ulnar-minus variance was found and a joint leveling procedure to shorten the radius was performed. Conservative therapy was provided pre and post surgical management.*

Summary: *This case report demonstrates the importance of findings on radiographs, MRI, and*

Objectif : *Présenter le diagnostic, les caractéristiques cliniques et le traitement de la maladie de Kienböck, et susciter une conscientisation quant au diagnostic différentiel de ce trouble chez les patients qui présentent une douleur insidieuse et progressive à la face dorsale du poignet.*

Caractéristiques cliniques : *Un joueur de football universitaire de 23 ans présente une douleur insidieuse et progressive du côté dorsal du poignet avec réduction de la flexion et de l'extension du poignet. Le diagnostic de maladie de Kienböck est établi d'après des radiographies et l'imagerie par résonnance magnétique.*

Intervention et résultat : *Une divergence ulnaire négative de 3 mm est décelée et une procédure de nivellement de l'articulation visant à raccourcir le radius a été effectuée. Une thérapie conservatrice a été administrée avant et après l'intervention chirurgicale.*

Résumé : *La présente étude de cas témoigne de l'importance des éléments trouvés au moyen des radiographies, de l'IRM et de l'examen clinique dans l'établissement d'un diagnostic exact et dans*

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clinical examination in the accurate diagnosis and management of a patient with wrist pain.
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KEY WORDS: Kienbock, Kienbock's disease, lunatomalacia, avascular necrosis, osteonecrosis, lunate

l'intervention menée chez le patient souffrant de douleur au poignet.
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MOTS CLÉS : Kienböck, maladie de Kienböck, malacie du semi-lunaire, nécrose avasculaire, ostéonécrose, os semi-lunaire

Introduction

Kienbock's disease, also known as lunatomalacia, was first described in the literature by Robert Kienbock in 1910 as ligamentous trauma to the lunate resulting in the interruption of internal arterial supply to the bone.[1] It has been defined as a specific form of osteonecrosis affecting the lunate bone of the carpus. Kienbock's disease is a form of osteonecrosis affecting the lunate characterized by sclerosis, cystic changes, fragmentation, and articular surface collapse as present on plain film radiographs.[2,3] Kienbock's disease is a rare condition with an unknown etiology.[2,3] Among athletes, the incidence and prevalence of Kienbock's disease are unknown. Due to its unknown etiology the management is highly controversial with both conservative and operative management alleviating pain, improving function, and limiting progression of the disease.[2,3] It can present with similar symptomatology to a scapholunate sprain, lunate fracture, or lunate dislocation, which can make differentiating this condition from other wrist conditions a challenge for many health care practitioners. This report highlights a case of Kienbock's disease in a 23-year old male varsity football player and reviews the relevant literature on the disease. The patient consented to release all information in regard to this case for publication.

Case report

A 23-year old male, varsity football player presented to a chiropractor with a primary complaint of a 3-year history of intermittent left wrist pain lasting one to two weeks in duration. At presentation, the patient reported progressive pain in the left wrist for a five-month duration with no relief of symptoms. The chief complaint was dull and achy pain poorly localized to the dorsal and palmer aspect of the left wrist distal to the ulnar styloid process. The patient attributed the wrist pain to a history of heavy bench-pressing in the gym during his varsity football career, as well as, repeated throwing as a quarterback. The pain was described as dull and achy at rest and sharp with bench-pressing weights and push-ups. The patient's history revealed he had never injured or sustained any minor or major trauma to the left wrist prior to presenting to the chiropractor. Pain in the left wrist was aggravated with terminal wrist extension and resistance training. The pain the patient experienced would recede for several months duration and would reappear unexpectedly without any direct wrist trauma or repetitive use. Pain was relieved with intermittent immobilization for one to two weeks until pain had fully subsided. The patient never sought out any form of medical or conservative treatment during painful periods. The patient reported that he had not taken any nutriceuticals or pharmaceutics for pain control.

Upon physical examination by the chiropractor, observation revealed no swelling, redness, or bruising on the dorsal or palmer aspect of the left wrist. There was pain on palpation overlying the left triangular fibrocartilage and left scapholunate ligament. There was bony tenderness on palpation of the left lunate, triquetrum, and capitate bones. Active range of motion revealed a limitation in flexion by 10 degrees and extension by 10 degrees of the left wrist due to pain. Passive range of motion revealed pain at 80 degrees of flexion and extension due to pain. Axial loading of the left wrist with full extension reproduced sharp pain felt with bench pressing and push-ups. Resisted range of motion of the left wrist reproduced dull and achy pain surrounding the lunate bone in flexion, ex-

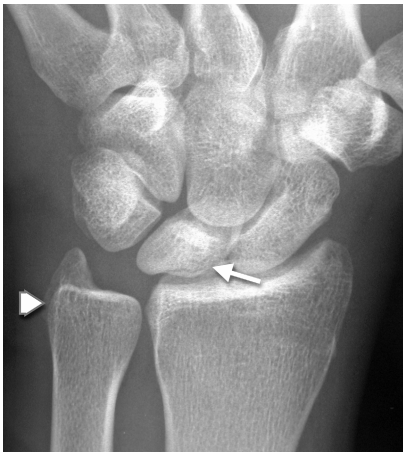

Figure 1: *Left AP wrist radiograph at initial presentation shows collapse and subchondral fracture of left lunate (arrow) and a 3mm negative ulnar variant (arrowhead)*

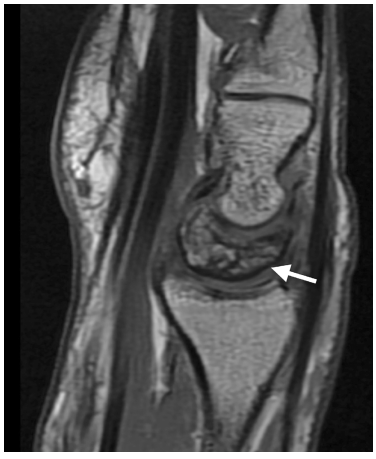

Figure 2: *Left Sagittal TI MRI taken at initial presentation shows fragmentation and collapse of left lunate*

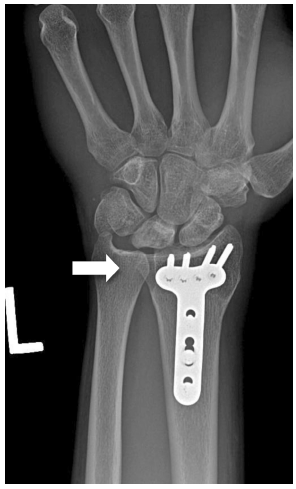

Figure 3: *Left AP wrist radiograph, the arrow shows a left radial shortening procedure of 3mm 6 months after initial diagnosis Kienbock's disease*

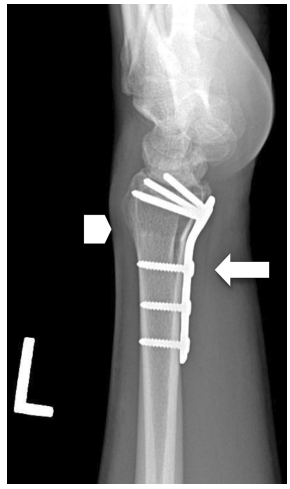

Figure 4: *Left lateral radiograph, the arrow shows the left radial shortening procedure of 3mm after initial diagnosis of Kienbock's disease. Arrowhead shows osteotomy scar in the proximal radius.*

tension, pronation, supination, ulnar deviation, and radial deviation. Neurological examination including deep tendon reflexes, sensation, and motor strength of the upper extremities was reported as unremarkable.

Bilateral radiographs of the wrists were obtained. The right wrist exhibited a three-millimeter ulnar-minus variance, but was otherwise normal.

Radiographs of the left wrist revealed flattening of the proximal pole of the left lunate with associated mild sclerosis and a small two-millimeter subchondral fracture within the proximal pole of the left lunate. The left proximal carpal arc was distorted with indentation of the arc at the proximal pole of the lunate. A three-millimeter negative ulnar variant was also present on the left (See Figure 1). A magnetic resonance imaging (MRI) study of the left wrist revealed abnormal signal intensity of the entire left lunate, mild loss of height of the medial aspect of the lunate bone, and a three-millimeter ulnar-minus variant. Additionally, a 4x3 mm lobulated collection of fluid was present posterior to the distal aspect of the ulna (See Figure 2). The final diagnosis was Kienbock's disease, three-millimeter ulnar-minus variant, and a ganglion cyst overlying the dorsal aspect of the distal ulna.

The patient was referred to three different orthopaedic surgeons that specialized in the hand and wrist. All three surgeons recommended that a left radial osteotomy (radial shortening procedure) be performed after a trial of conservative therapy. After consultation with the orthopaedic surgeons, the patient's left wrist and forearm was placed in a fibreglass cast for three months in duration for immobilization and revascularization purposes. Radiographic series of the left wrist occurred after a trial of immobilization for three months. The radiographs revealed no osseous changes or progression through the stages of Kienbock's disease. The patient was still symptomatic after three months of immobilization. A radial shortening procedure was recommended.

Prior to the radial shortening procedure the patient consented to experimental extracorporeal shockwave therapy (ESWT) as a conservative measure to avoid sur-

gery. The patient received three treatments of ESWT at the chiropractic clinic for three weeks in duration. The extracorporeal shockwave head was directed specifically to the dorsal aspect of the lunate bone. The patient received 1500 shocks at 8 Hz with no improvements in pain or function. The chiropractor continued with Active Release Technique® (ART®) to the extensor carpi radialis brevis (ECRB), extensor carpi radialis longus, extensor digitorum, extensor pollicis brevis, abductor pollicis longus and the dorsal radiocarpal ligaments. The patient also received acupuncture with intramuscular stimulation as a neuromodulator of pain. The specific acupuncture points targeted were LI-10, LI-11, TH-5, LI-4 and an inline technique to ECRB. Concurrently, the patient was prescribed Ruta Graveolens and Aurum Arsenic from a homeopathic doctor to decrease muscle pain and help regenerate the periosteum and cartilage. He took 5 pills of these homeopathic medications, 3 times per day for 12 weeks with no improvement in pain or function of the left wrist.

A left radial shortening procedure of three-millimeters was performed six months after initial diagnosis by the chiropractor. After the left radial shortening was performed, the patient was placed in a hard fibreglass cast for the next six weeks to allow for boney healing. After six weeks of immobilization a radiographic series of the left wrist was performed and confirmed healing of the left radius. Over the course of the next 12 weeks the patient was given simple range of motion and strengthening exercises to be performed daily. Initially, the patient was instructed to perform all active ranges of motion of the left wrist and progress to passive range of motion. At 8 weeks post-surgery, concentric and eccentric wrist curls and radial and ulnar deviation movements with a two-pound dumbbell were prescribed daily. Three sets of ten repetitions were completed.

The majority of post-surgical pain was located on the dorsal aspect of the left wrist distal to the ulnar styloid process. Supination and pronation of the affected wrist was significantly diminished compared to the contralateral wrist. A twister splint was prescribed to passively force the affected wrist into supination or pronation to increase range of motion and decrease pain. The splint was worn for three times per day for twenty minutes each session for a duration of eight weeks. The patient was also instructed to execute 'the hammer' exercise. The patient was told to grab the handle of a regular hammer in his left hand and slowly supinate and pronate his forearm. This exercise was designed to strengthen pronator quadratus, pronator teres and supinator. It was also used as a passive stretch into supination or pronation due to the weight of the hammer. Finally, the patient received laser therapy using the Bioflex laser. Five laser treatments were provided for two weeks that ranged from 409.32 J/cm^2 to 457.9 J/cm^2 for 1710 seconds.

At 22-months post surgery, the patient has returned to sport including basketball, ball hockey, flag football, golf, baseball and weight lifting. He is no longer participating in varsity football. During resistance training, he performs pain-free exercises and limits activities of the wrist in terminal extension or axial loading (ex. push ups or bench press). Follow-up radiographs were taken and no progression of Kienbock's disease had occurred.

Discussion

In 1910, Kienbock theorized that lunatomalacia was a result of traumatic rupture of the ligaments surrounding the lunate with associated disruption of the internal blood supply to the lunate.[1,4,5,6] Subsequently resulting in a disturbance of the bony nutrition, aseptic necrosis, softening, and progressive collapse of the bone.[1,4,5,6] The etiology of the condition after 100 years is still very controversial in the literature.[3] The prevalence of Kienbock's disease can be a challenge to diagnose as a result of its rarity and overlapping clinical presentation with general wrist sprains. Consequently, delayed diagnosis can compromise prognosis due to the progression in staging and further deterioration.

Kienbock's disease most commonly affects adult males between the ages of 20 and 40 years of age, who are predominantly manual workers.[2,3,7] Manual workers tend to have more repetitive trauma to the carpal bones leading to degenerative changes and subsequent loss of lunate blood supply. It can be theorized that athletes using their hands may also have an increase in repetitive trauma to the wrist leading to an increased risk of developing Kienbock's disease. There is a predilection in males compared to females with a male to female ratio of up to 9:1.[6] It affects the dominant and non-dominant hand equally and is usually a unilateral condition but bilateral involvement may occur.[2,3,7] A history of a traumatic event is common and may have occurred prior to signs and symptoms of wrist pain and dysfunction.[3]

Kienbock's disease is characterized as insidious, progressive dorsal wrist pain surrounding the lunate with or without localized swelling. Patients may report generalized or focal wrist pain, pain aggravated with wrist flexion and/or extension, weak gripping abilities, and an inability to load the affected wrist into extension.[6,8,9] Frequently, the patient will report tenderness with bony palpation of the lunate. The final result of Kienbock's disease is lunate sclerosis, fragmentation, and collapse secondary to avascular necrosis.[3]

The precise etiology of Kienbock's disease is unknown and remains controversial within the current literature.[2,3] Currently, there are many etiological theories of Kienbock's disease. According to the mechanical theory, avascular necrosis of the lunate bone is a consequence of a slow progressive collapse of the lunate under excessive bony stresses and excessive loading. Excessive load causes small repeated microfractures of the lunate trabecula leading to subsequent bony collapse.[3] One of the most common mechanical etiological factors is an ulnar-minus variant. A short ulna cannot share axial loads with the radius thus increasing force transmission through the lunate. This creates a "nutcracker effect" with increased compressive forces through the lunate from the radius and the capitate leading to repeated microfractures.[3,10,11,12] Trapezoidal shaped lunate bones may also increase the mechanical loading from the radial-ulnocarpal joint to the lunate causing an increase in compressive loading.[2] An increase in compressive loads can cause sclerosis, fragmentation, and collapse of the lunate.[3]

The vascular supply theory states that the etiology of Kienbock's disease may be from a limited arterial supply to the dorsal or palmer aspect of the lunate. The proximal pole of the lunate is likely to become osteonecrotic due to its limited anastomosis of arterial supplying and the presence of only terminal arterial branches supply the proximal pole. Also, impairment in the venous outflow can lead to an increase in intraosseous pressure in the lunate causing avascular changes and osteonecrosis.[2,3,6,7,13]

The traumatic theory of Keinbock's disease states that ligamentous rupture of ligaments attaching to the lunate may disrupt nutrient arteries supplying the internal aspect of the lunate causing avascular changes. Small microfractures in the lunate may also lead to sclerosis, fragmentation, and collapse of the bone.[2,3,6,7,13]

Kienbock's disease is fundamentally an avascular process of the lunate. Therefore, it is important to highlight the vascular supply to the carpals, more importantly, the lunate. It receives blood supply from the palmar and dorsal wrist vascular sources including the radial artery and the ulnar artery. The vessels on the dorsal aspect of the wrist originate from the dorsal intercarpal arch, and occasionally from the dorsal branch of the interosseous artery.[14,15] On the palmer surface, the lunate bone gets blood supply from the palmer intercarpal arch, the palmer radiocarpal arch, and communicating branches from the anterior interosseous artery and the ulnar recurrent artery.[14,15] The vascular supply to the dorsal aspect of the lunate is smaller than those vessels entering on the palmar surface. The vessels entering from the dorsal aspect and the palmar aspect anastomose intraosseously distal to the mid portion of the lunate.[14] The proximal aspect of the lunate has relatively less vascularity compared to the rest of the bone.[14,15] Due to the limited blood supply of the lunate, especially the proximal pole, it is the main carpal bone vulnerable to post-traumatic avascular necrosis.[16] It has been theorized that vascular disturbances to the lunate, either congenital or acquired, may result in insufficient bony nutrition leading to an increased risk of developing avascular necrosis.

Systemic factors that may be implicated in the development of avascular necrosis are hypercoagulability conditions, arterial problems, and conditions causing an increase in venous congestion.[6] Systemic corticosteroids use, sickle cell anemia, cerebral palsy, septic emboli, and lupus have all be associated with osteonecrosis in general.[6] However, these systemic conditions have not necessarily been associated with osteonecrosis of the lunate bone specifically.[6]

Appropriate diagnosis requires assessment of the degree of osseous changes within the lunate and the chronicity of the disease.[6] Radiographs may demonstrate sclerosis, cystic changes, fragmentation, articular surface collapse of the lunate, and perilunate arthritic changes. No significant radiographic changes are present in the early stages. Thus, it is imperative to perform an MRI in the early stages due to its sensitivity in distinguishing between vital (lipid-rich) and necrotic (lipid-poor) bone.[17,18,19] A loss of signal intensity of the entire lunate bone on a T1-weighted MRI represents a loss of lipids within in the bone and therefore represents necrotic tissue.[19] If the change in signal intensity is only located within a small region of the

Table 1
Lichtman Classification[3,6,20,21]

Stage	Radiographical Findings
Stage I	Normal architecture and density on radiographs, signal intensity changes are present on MRI
Stage II	Lunate density changes present on radiographs, size and anatomic relationship to carpal bones affected, fractures lines may be present
Stage III	Collapse of the lunate articular surface
Stage IIIA	Lunate sclerosis, loss of lunate height, and normal carpal alignment
Stage IIIB	Fixed scaphoid rotation, proximal capitate migration, and loss of carpal height, and carpal instability
Stage IV	Lunate collapse with associated radiocarpal or midcarpal arthritis

lunate other pathologies need to be considered.[6] MRI may be clinically relevant in the early stages of the disease.

The staging of Kienbock's disease from the Lichtman classification is critical in the evaluation of the condition as it dictates the specific management options.[6,20,21] (see Table 1) In stage 1, plain film radiographs reveal normal density and articular surfaces of the lunate, MRI reveals decreased signal intensity in the distal portion of the lunate.[3,6,20,21,22] In stage 2, the lunate appears sclerotic and a fracture line may be visible with preservation of the articular surface of the lunate.[3,6,20,21,22] Stage 3 is divided in stage 3A and stage 3B. Stage 3A is the most common stage at initial presentation due to a painful collapse of the articular surface of the lunate. In stage 3A there is normal carpal alignment and height. In stage 3B there is fixed scaphoid rotation, proximal capitate migration, and loss of carpal height.[3,6,20,21,22] Finally, stage 4 is defined by lunate collapse associated with significant perilunate arthritic changes in the carpals.[3,6,20,21,22] According to Geommine et al. the Lichtman classification is a valid and reliable tool for detecting and staging Kienbock's disease.[20] This case reports a patient with stage 3A on the Lichtman classification system, presenting with lunate sclerosis, loss of lunate height, and with normal carpal alignment.[3,6,20,21,22]

The management of Kienbock's disease is highly dependent on the stage of the disease based on the Lichtman classification system, symptomatology, and functional deficits.[3,6] The methods for treatment of Kienbock's disease ranges from conservative care to operative care and focuses on the alleviation of subjective pain, improving function, and limiting progression of the disease.[3,6,23,24] The most commonly used conservative therapies include immobilization with splinting or casting and activity modification.[3,6,7,8,9,21] Innes and Strauch performed a systematic review in 2010 on the treatment of Kienbock's disease in its early and late stages.[23] Interventions included radial osteotomy, vascularized bone graft, carpectomy, and total wrist athrodesis. The results of this review suggest that subjective pain scores in Kienbock's disease improves regardless of the treatment type provided.[23] Further, objective scores in grip strength and range of motion were significantly changed in all surgical groups. Therefore, all surgical interventions tend to be associated with improved range of motion and improved grip strength.[23]

There are multiple surgical procedures for the management of Kienbock's disease and literature casts little evidence-based information to determine which management strategy is superior or to indicate its superiority over conservative measures.[6] In stage 1 of the Lichtman classification a trial of conservative therapy including immobilizaton is warranted with the use of analgesics and nonsteriodal anti-inflammatories for pain relief.[6] In stages 2 and 3A with an ulnar-minus variant a trial of conservative management for three months is warranted. If conservative therapy is unsuccessful in abating the symptoms a joint leveling technique is considered.[23,24,25] The primary goal is to revascularize the lunate in order to prevent progression.[6] A positive ulnar variant dictates surgical management involving vascularized bone graphs and external fixations to increase vascular supply to the lunate.[6] These procedures do not reverse or halt Kienbock's disease but

Table 2
Management of Kienbock's disease according to Lichtman classification[3,6]

Stage	Radiographical Findings
Stage I	Cast immobilization for 3 months with use of analgesics and NSAIDS
Stage II and IIIA with ulnar negative variance	3 month trial therapy of immobilization with use of analgesics and NSAIDS Joint leveling technique (radial shortening, ulnar lengthening, capitate shortening)
Stage II and IIIA with ulnar positive variance	3 month trial therapy of immobilization with use of analgesics and NSAIDS Vascularized bone graft and external fixation; radial wedge or dome osteotomy; capitate shortening
Stage IIIB	Internal carpal artheodesis; lunate excision with or without replacement; proximal row carpectomy; joint leveling
Stage IV	Proximal row carpectomy; carpal arthrodesis; wrist denervation

slows down the rate of degenerative change in the affected wrist.[26,27] Joint leveling procedures are performed when there is an ulnar-variance because the ulna is unable to share the axial loads with the radius resulting in increased force transmission through the lunate.[3,25,26,27] In advanced stages of the disease intercarpal athrodesis, lunate excision with or without replacement, carpectomies, and wrist denervations are considered primary management options.[6,28] (See Table 2)

Extracorporeal shockwave therapy may be a viable conservative treatment option for osteonecrosis because of an increase in osteogenesis, vascular endothelial growth factor, and angiogenesis allowing for revascularization of the affected bone.[29,30,31,32]

The prognosis of Kienbock's disease is highly dependent on the stage of the disease at presentation. There seems to be variability between individuals in the prognosis of the disease. The natural history of the condition is to slowly progress through each of the Lichtman stages through time and eventually it will result in degenerative changes within in the lunate and the surrounding carpal joints.[8] Surgical management has not shown to halt the condition but does decrease the rate of progression through the Lichtman stages.[26] Taniguchi et al., in 2002 performed a retrospective case series on 16 patients treated conservatively for late stage Kienbock's disease with associated scaphoid rotation.[33] The results show that there was no progression to scapholunate advanced collapse. Thus, the conclusion was that there is no need to perform a wrist arthrodesis and that wrist immobilization for three months may be sufficient in the late stages as well as the early stages of Kienbock's disease.[33] The natural history of the condition is to slowly progress through each of the Lichtman stages resulting in degenerative changes within in the lunate and surrounding carpal joints.[8,33]

Summary

Kienbock's disease is a rare condition with an unknown etiology. This case highlights the necessity for all primary healthcare practitioners responsible for diagnosing and managing wrist injuries to be aware of avascular necrosis. Kienbock's disease should be considered a differential diagnosis in patients with insidious, progressive wrist pain, especially in males between 20-40 years of age and those with a history of repetitive wrist trauma. Our case presented with dorsal and ventral pain on the ulnar aspect of the left wrist just distal to the ulnar styloid process that was insidious over three years and progressive in nature. There was pain on palpation of the lunate and surrounding ligaments. Further, this patient presented with pain at terminal wrist flexion and extension resulting in a weak grip and an inability to fully load the wrist. Considering Kienbock's disease may have a vascular origin, we hypothesize that due to the repetitive wrist motion in football the normal intralunate vascular anastomosis may have been disrupted leading to bony ischemia, necrosis, and ultimately collapse of the proximal pole.

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