Imaging of Kienböck Disease

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OBJECTIVE. The purpose of this article is to provide an overview of Kienböck disease focusing on imaging findings, including the relevant anatomy, pathophysiology, and treatment of Kienböck disease and the differential diagnoses (pseudo-Kienböck lesions).

CONCLUSION. Kienböck disease is a condition marked by avascular necrosis of the lunate bone. MRI is useful in diagnosis and staging and should be considered, after conventional radiography, for patients with suspected Kienböck disease.

Kienböck disease is a condition characterized by avascular necrosis of the lunate bone. It is also known as osteonecrosis, lunatomalacia, and aseptic or ischemic necrosis of the lunate. Although the mechanisms by which this disorder develops are not fully understood, compromise of the bone vasculature is the most commonly proposed cause. This leads to bone infarction and ultimately mechanical failure. Kienböck disease is often progressive, resulting in joint destruction within 3–5 years if left untreated.

The Austrian radiologist Robert Kienböck described this entity in 1910 [1]. He described the disease process and clinical features of lunatomalacia and proposed a disturbance of the nutrient vessel supply to the lunate as the cause. Kienböck noted various radiographic changes, including sclerosis and collapse of lunate bone [1]. Ståhl [2] in 1947 first described a classification system based on the radiographic findings of Kienböck disease. He defined the following five stages of lunate osteomalacia: I, radiodense fracture; II, secondary resorption with development of a rarefaction line; III, sclerosis; IV, secondary fractures with fragmentation; V, arthrosis.

Lichtman et al. [3] in 1977 modified Ståhl's original radiologic classification system to help select the most appropriate treatment of each patient. This classification has shown itself to be simple to use even by inexperienced observers. It has low interobserver variability, good reproducibility, and appears to accurately reflect the natural history of the disorder [4]. In 2010, Lichtman et al. [5] introduced a new stage IIIC, which corresponds to chronic coronal lunate fracture. In 1997 Schmitt et al. [6] used contrast-enhanced MRI to describe three patterns of Kienböck disease in a functional classification system with which one could also determine the most appropriate treatment. The patterns Schmitt et al. described reflect the degree of bone necrosis present.

Many surgical procedures have been described, but it has not been proved whether surgery improves the natural history of Kienböck disease [5, 7]. Treatments include mechanical lunate unloading to encourage spontaneous revascularization; direct lunate bone graft procedures; and more aggressive options, such as proximal row carpectomy, joint replacement, and carpal bone fusion [5, 7].

In this article we review the anatomy of the lunate bone and the pathophysiologic features of Kienböck disease, the imaging findings and classification of Kienböck disease, the differential diagnoses, and treatment.

Anatomy and Vascularization

Anatomic Features

The lunate bone (semilunar bone) is a bone of the carpus. The name derives from the Latin *luna*, which means "moon," because the shape of the bone resembles a crescent moon. The lunate bone is situated in the center of the proximal row of the carpus and articulates with the radius and the scaphoid, triquetrum, and capitate bones and in 45% of cases with the hamate bone. The proximal

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and distal surfaces of the lunate are completely covered with articular cartilage and have no vascular foramina or sites for ligament attachment. The radial and ulnar aspects are covered by articular cartilage, except at the insertion of the scapholunate interosseous ligament and the lunotriquetral interosseous ligament.

According to the Antuña Zapico classification, there are three morphologic types of lunate based on the angle between the lateral scaphoid and proximal radial sides of the lunate [8] (Fig. 1). In type I, the angle is more than 130°; in type II, the angle is less than 130° (≈ 100°); and in type III, there are two distinct facets on the proximal surface. One articulates with the radius and the other with the triangular fibrocartilage.

Vascularization

The lunate bone has extraosseous and intraosseous components to its blood supply. The extraosseous blood supply comprises vessels that enter the lunate through the dorsal and volar poles of the bone [9]. The volar vessels normally provide a greater proportion of the blood supply, having more foraminal vessels, from zero to five, compared with the dorsal supply, which has zero to three [9–11]. Volar lunate vascularity consists of the palmar radiocarpal and intercarpal arches. Vessels enter the volar pole through various ligament insertions, such as the radioscapholunate ligament, radiolunate triquetral ligament, and ulnar lunate triquetral ligament [9]. Dorsal lunate vascularity consists of the dorsal intercarpal and radiocarpal arches, which arise from the plexus of vessels located directly over the dorsal pole of the lunate. For the intraosseous blood supply, there are different patterns of intraosseous anastomosis between the volar and dorsal vessels, which enter the lunate through the bone foramina [9].

Pathophysiologic and Predisposing Risk Factors

The pathophysiologic mechanism of Kienböck disease is multifactorial [5], and a uniform cause has not been determined. Anatomic factors are associated with an increase in shear forces on the lunate bone that may contribute to the development of Kienböck disease. This includes a negative ulnar variance, which has been found in 78% of cases [12], and rectangular or square geometry of the lunate, as in Antuña Zapico types I and II, in which an angle greater than 110° also presents higher risk of Kienböck disease [8].

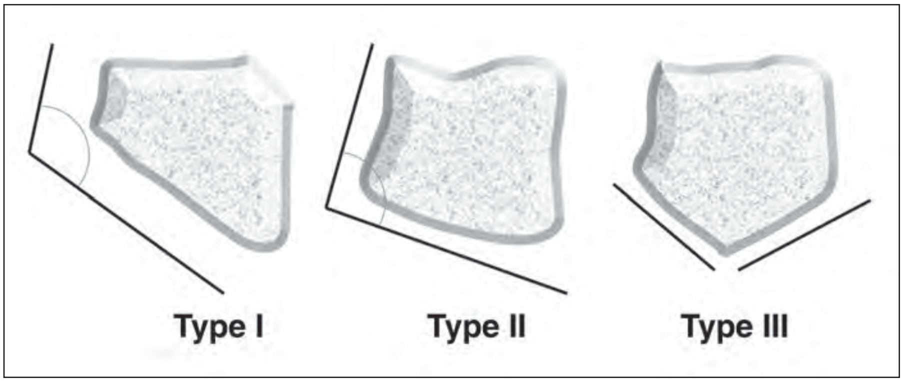
Type I
Type II
Type III

Fig. 1—Diagram shows Antuña Zapico morphologic classification of lunate bone.

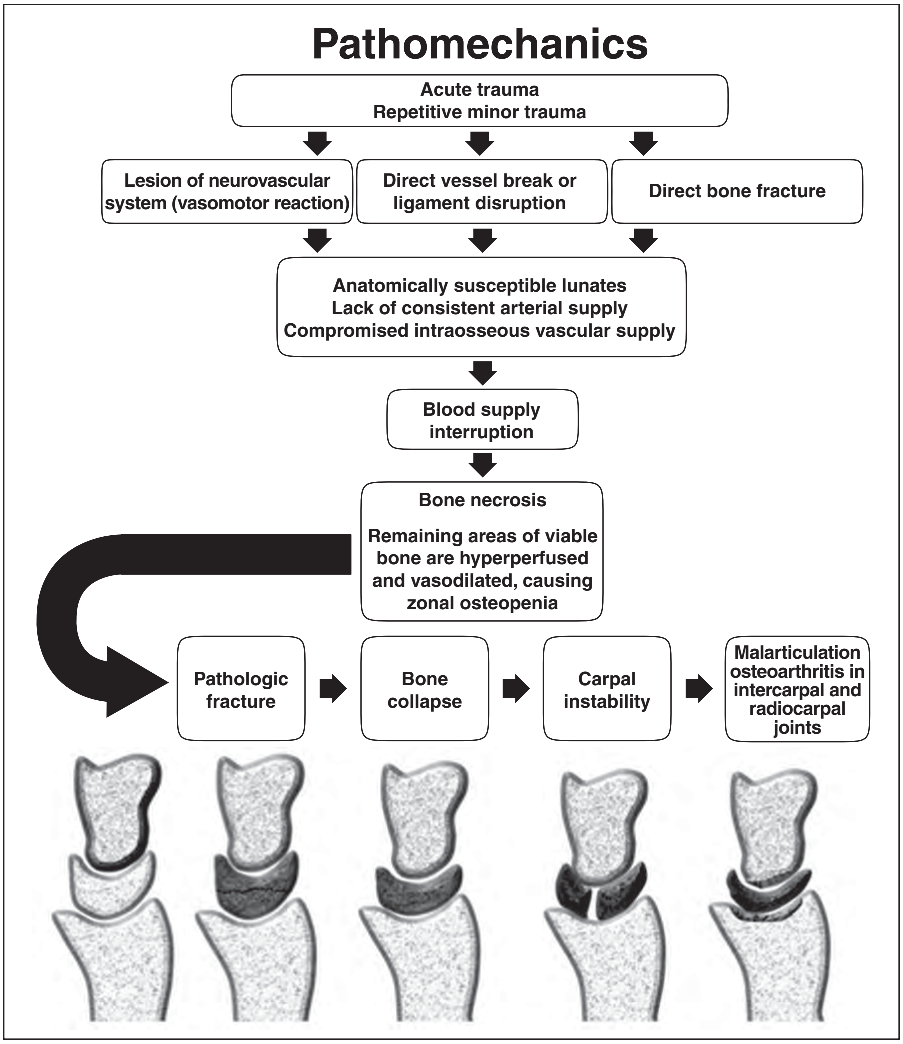
Pathomechanics
Acute trauma
Repetitive minor trauma
Lesion of neurovascular system (vasomotor reaction)
Direct vessel break or ligament disruption
Direct bone fracture
Anatomically susceptible lunates
Lack of consistent arterial supply
Compromised intraosseous vascular supply
Blood supply interruption
Bone necrosis
Remaining areas of viable bone are hyperperfused and vasodilated, causing zonal osteopenia
Pathologic fracture
Bone collapse
Carpal instability
Malarticulation osteoarthritis in intercarpal and radiocarpal joints

Fig. 2—Chart shows pathomechanics of Kienböck disease.

Patients with Kienböck disease may have a distinctly unique vascular pattern that is susceptible to excessive or unusual forces. Areas supplied by terminal arterial branches without sufficient collateral vessels and those with a single palmar vessel are expected to have a predisposition to Kienböck disease. Venous congestion has been discussed as another cause of Kienböck disease. Intraosseous measurements have higher pres-

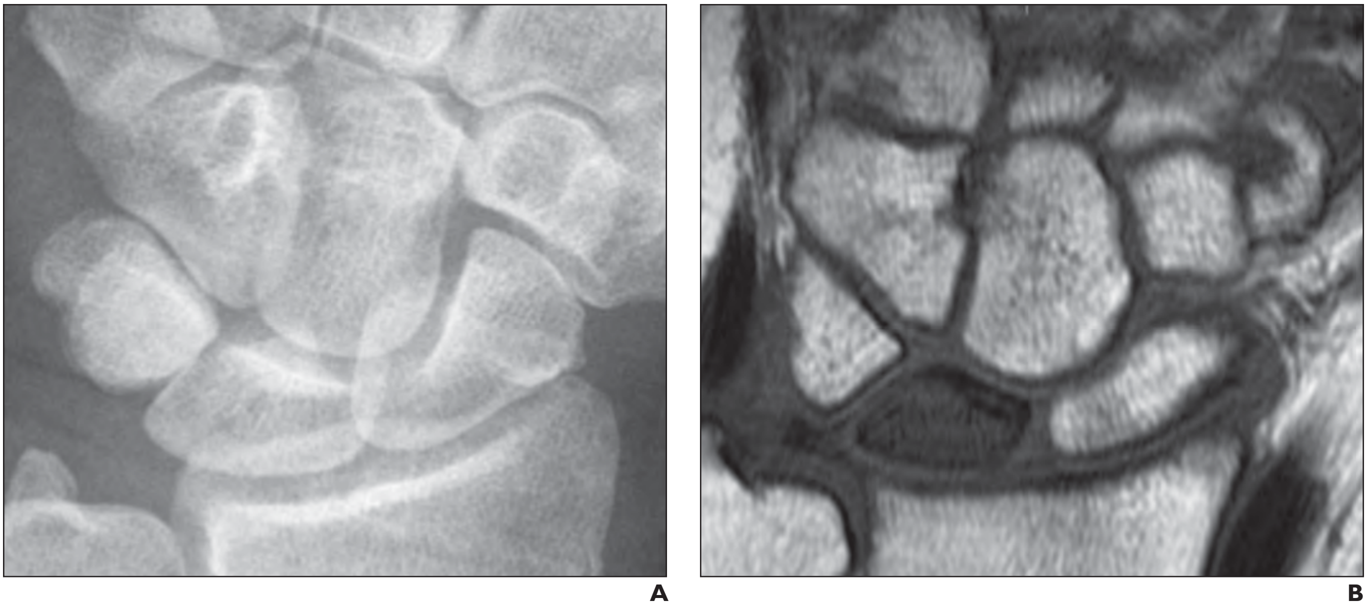

Fig. 3—40-year-old woman with wrist pain and tenderness due to stage I Kienböck disease.
A, Radiograph shows normal lunate bone.
B, Coronal T1-weighted MR image shows hypointensity of lunate bone.

sures in extension in necrotic than normal lunate bones [13].

Acute trauma or repetitive minor trauma over anatomically susceptible lunate bones, which have a suboptimal arterial supply, can lead to neurovascular injury (with subsequent vasomotor reaction), direct vessel breakage, ligament disruption, or direct fracture that eventually interrupts the blood supply and ultimately causes bone necrosis. The remaining areas of viable bone become hyperperfused, and vessels vasodilate, causing zonal osteopenia. The bone necrosis and zonal osteopenia can lead to pathologic fracture, bone collapse, and eventually carpal instability with subsequent malarticulation and degenerative osteoarthrosis in the intercarpal and radiocarpal joints (Fig. 2).

Clinical Features and Epidemiology

The symptoms of Kienböck disease typically begin between the ages of 20 and 40 years with a 2:1 ratio of men to women. The disease commonly affects the dominant wrist, but bilateral disease can occur. The clinical onset can be abrupt or insidious, and the symptoms are nonspecific and can mimic other conditions, so it is important to ask about any history of trauma. The most common symptoms are dorsally located central wrist pain and tenderness around the lunate. Synovitis and inflammation can lead to weakness, limited motion, decreased grip strength, and in some cases, carpal tunnel syndrome [14].

Imaging

Diagnosis

Kienböck disease can be diagnosed with radiography, CT, or MRI. The imaging criteria for Kienböck disease are based on bone hyperdensity and hyperattenuation (radiography and CT) and the degree of collapse of

TABLE 1: Lichtman Staging of Kienböck Disease

Stage	Radiographic and CT Findings	MRI Findings
I	Normal morphologic findings	Morphologic preservation
		Edema pattern in bone marrow
II	Normal morphologic findings	Morphologic preservation
	Sclerosis in bone marrow	Signal intensity low on T1-weighted images, variable on T2-weighted images
IIIA	Collapse of lunate bone	Morphologic collapse of lunate bone
	Radioscaphoid angle < 60°	Signal intensity low on T1-weighted images, variable on T2-weighted images
IIIB	Collapse of lunate bone	Morphologic collapse of lunate bone
	Radioscaphoid angle > 60°	Signal intensity low on T1-weighted images, variable on T2-weighted images
IIIC	Collapse of lunate bone	Morphologic collapse of lunate bone
	Coronal lunate fracture (chronic)	Signal intensity low on T1-weighted images, variable on T2-weighted images
		Coronal lunate fracture (chronic)
IV	Radiocarpal or midcarpal degenerative arthritis	Radiocarpal or midcarpal degenerative arthritis
		Signal intensity low on T1-weighted images, variable on T2-weighted images

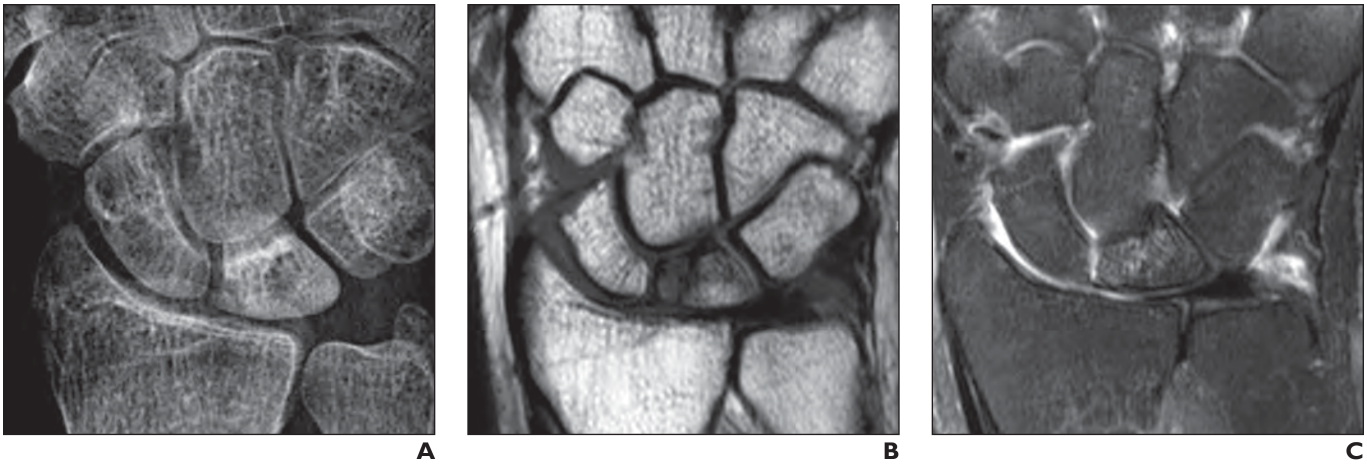

Fig. 4—25-year-old woman with chronic wrist pain due to stage II Kienböck disease.
A, Radiograph shows increase in bone density of lunate compared with other carpal bones.
B, Coronal T1-weighted MR image shows decreased signal intensity on radial side of lunate bone.
C, Coronal T2-weighted fat-suppressed MR image shows mild increase on signal intensity in lunate bone. Size, shape, and anatomic relations of lunate are preserved.

the lunate bone (radiography, CT, and MRI). At MRI, changes in bone marrow signal intensity are commonly seen as areas of hyperintensity on T2-weighted images and areas of hypointensity on T1-weighted images.

Assessment and Classification

Kienböck disease can be assessed morphologically and functionally. This is important in evaluating progression of the disease and choosing the most appropriate treatment. Morphologic evaluation is made with the Lichtman classification (Table 1), which entails radiographic, CT, and unenhanced MRI findings. In the Lichtman classification the disease is categorized according to the morphologic features and density of the bone (bone density and attenuation at radiography and CT and bone marrow signal intensity at MRI). It was traditionally based on radiographic findings, but CT and MRI are more accurate for staging this disease with the same imaging criteria. This classification is highly reliable and reproducible and has the most clinical relevance because it helps in determining the most appropriate treatment [4].

In stage I, the patient presents with clinical symptoms of Kienböck disease. The lunate has normal architecture and density or attenuation at radiography or CT (Fig. 3A). At MRI, uniformly decreased signal intensity on T1-weighted images (Fig. 3B) and increased signal intensity on T2-weighted images are typically found. These imaging findings reflect the edemalike changes associated with the early stages of Kienböck disease. Before MRI became available, the disease was diagnosed clinically or with scintigraphy.

Stage II Kienböck disease is characterized by sclerosis evidenced by increased bone density and attenuation of the lunate compared with the other carpal bones on radiographs and CT scans (Fig. 4A). The size, shape, and anatomic relations of the bones are preserved. At MRI, areas of decreased signal intensity on T1-weighted images (Fig. 4B) and variable signal intensity on T2-weighted images are found, mostly on the radial side of the lunate (Fig. 4C). Areas of decreased signal intensity on T2-weighted images reflect bone sclerosis and can sug-

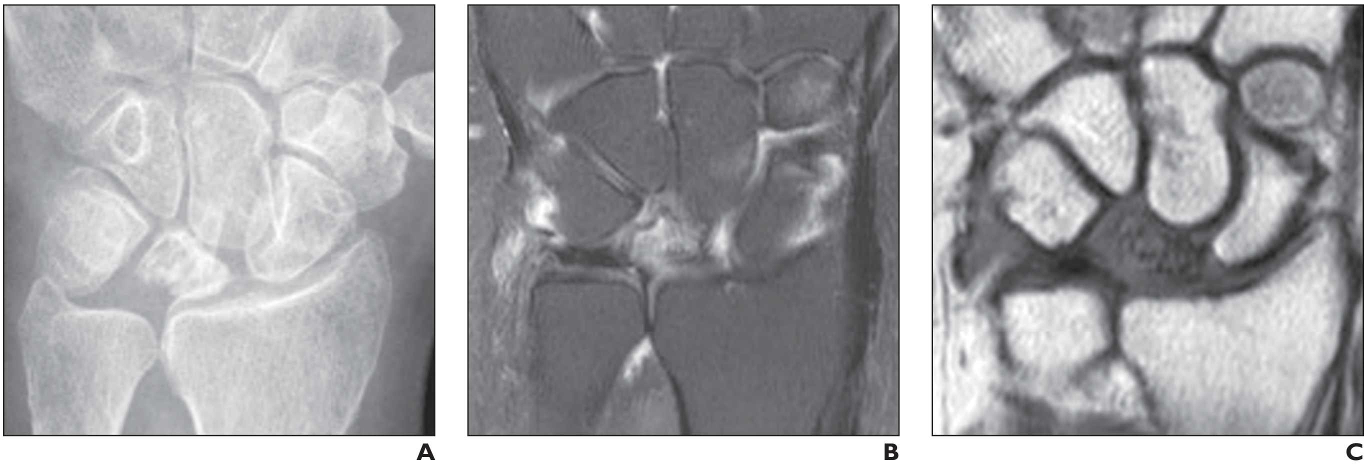

Fig. 5—45-year-old man with wrist pain due to stage III Kienböck disease.
A, Radiograph shows lunate collapse and sclerosis.
B, Coronal T2-weighted fat-suppressed MR image shows diffuse hyperintensity and collapse of lunate bone.
C, Coronal T1-weighted MR image shows diffuse hypointensity of lunate bone.

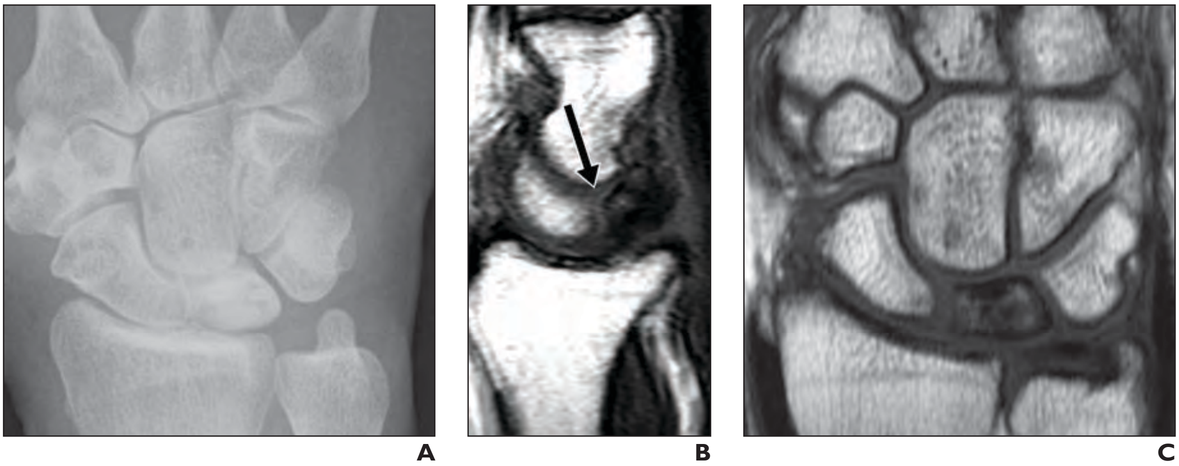

Fig. 6—32-year-old woman with wrist pain due to stage IIIC Kienböck disease.
A, Radiograph shows mild lunate sclerosis and collapse. Negative ulnar variance is evident.
B, Sagittal T1-weighted MR image shows coronal fracture of lunate bone (*arrow*).
C, Coronal T1-weighted MR image shows patchy hypointensity of lunate bone.

gest stage II Kienböck disease. If there is no decrease in signal intensity on T2-weighted images, MRI alone cannot be used to differentiate stage I and stage II disease, thus radiography or CT is needed to determine the bone density or attenuation and to accurately classify a lunate bone that has signal-intensity alteration and preserved shape at MRI.

Stage III Kienböck disease is characterized by collapse of the entire lunate bone from distal to proximal in the coronal plane and elongation in the sagittal plane. No radiocarpal or midcarpal associated degenerative arthrosis is found (Fig. 5A). This stage can be diagnosed with radiography, CT, or MRI. At MRI, variable nonspecific areas of increased or decreased signal intensity on T2-weighted images (Fig. 5B) and decreased signal intensity on T1-weighted images (Fig. 5C) may be found. Stage III is divided in three subcategories: IIIA, absence of changes in the carpal alignment and a radioscaphoid angle less than 60°; IIIB, carpal instability and a radioscaphoid angle greater than 60° with rotatory scaphoid subluxation; and IIIC, chronic coronal lunate fracture (Fig. 6).

Stage IV is characterized by lunate collapse with associated radiocarpal or midcarpal degenerative arthritis, also known as Kienböck disease advanced collapse [5]. These degenerative changes at the radiocarpal and midcarpal joints are similar to the advanced collapse found in chronic scapholunate dissociation and scaphoid nonunion (Fig. 7A). Splaying of the volar and dorsal poles of the lunate is also found (Fig. 7B). At MRI, areas of increased or decreased signal intensity on T2-weighted images and decreased signal intensity on T1-weighted images (Fig. 7C) are present. Adjacent reactive synovitis and joint effusion may be associated with bone necrosis.

Functional evaluation, as proposed by Schmitt et al. in 1997 [6], should be performed with contrast-enhanced MRI and is complementary to the morphologic evaluation (Table 2). The contrast-enhanced MRI findings help to guide the treatment algorithm in some stages of the disease [5] and provide functional information about the degree of bone necrosis. This is important for planning re-

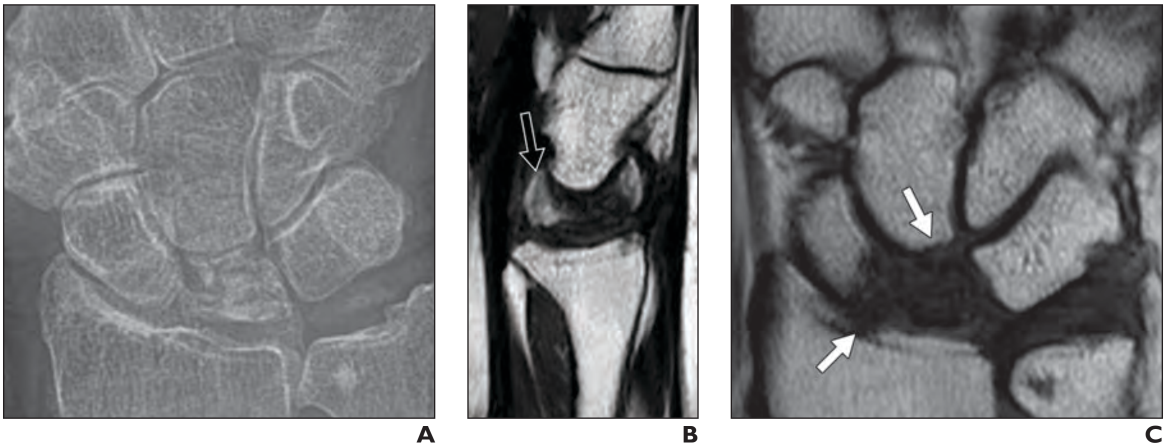

Fig. 7—32-year-old woman with wrist pain, diffuse swelling, and stiffness due to stage IV Kienböck disease.
A, Radiograph shows collapse and osteoarthritic changes in radiolunate and midcarpal joints.
B, Sagittal T1-weighted MR image shows coronal fracture of lunate bone and elongation (*arrow*).
C, Coronal T1-weighted MR image shows marked hypointensity of lunate bone and collapse and osteoarthritic changes in radiolunate and midcarpal joints (*arrows*).

TABLE 2: Functional Evaluation of Kienböck Disease With Contrast-Enhanced MRI

MRI Pattern	T1-Weighted Images	T2-Weighted Images	Contrast Enhancement
Edema, ischemia, nonnecrotic	Hypointensity	Variable (generally hyperintensity)	Homogeneous
Partial necrosis	Hypointensity	Variable	Patchy inhomogeneous
Complete necrosis	Hypointensity	Variable	Absent

vascularization techniques in cases of Lichtman stage II and IIIA disease. There are three MRI patterns of Kienböck disease, as follows.

In the edema pattern the lunate bone has uniformly decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images with intact perfusion (homogeneous contrast enhancement) (Fig. 8A). This pattern reflects edemalike changes associated with the early stages of Kienböck disease.

The partial necrosis pattern is areas of necrosis and repair with nonspecific patchy or homogeneous areas that are hypointense on T1-weighted images and have variable signal intensity on T2-weighted images. Partial enhancement of the lunate is seen on contrast-enhanced images (Fig. 8B). The partial areas of nonenhancing necrotic tissue are usually proximal.

The complete necrosis pattern is complete absence of contrast enhancement of the lunate (Fig. 8C). This pattern also consists of nonspecific patchy or homogeneous areas of variable signal intensity on T2-weighted images and hypointensity on T1-weighted images. It indicates complete necrosis of the bone and is most likely found in the late stages of the disease.

Imaging Approach to Kienböck Disease

Radiography is the initial imaging technique for assessing Kienböck disease. No further studies are necessary in cases of stages IIIB and IV disease because radiography is sensitive, and both late stages necessitate salvage surgical procedures. Radiographic findings also can be used to rule out other pathologic conditions, such as arthrosis and fractures.

CT best depicts the bone anatomy and is better than radiography for determining the exact stage of disease. It usually confirms a more advanced stage of Kienböck disease, particularly in stage II. To our knowledge no studies have compared the diagnostic accuracy of CT, MRI, and radiography for Kienböck disease staging.

MRI is likely to be the next best imaging examination after routine radiography. MRI can depict the bone anatomy and, like radiography and CT, can be used to determine the Lichtman stage of disease. MRI is accurate for appreciating the morphologic changes in the lunate bone and differentiating all disease stages. An exception is that stage I cannot be differentiated from stage II in cases that lack T2 hypointensity. In these specific cases, radiography or CT can be useful.

MRI is also essential for diagnosing stage I Kienböck disease, defined by clinical symptoms of the wrist with normal radiographic findings. In addition, MRI facilitates not only assessment of the lunate but also ruling out of other disorders (pseudo-Kienböck lesions), such as ganglion cysts and osteoarthritis among others. Furthermore, MRI is useful for longitudinal assessment of the postoperative response to direct and indirect revascularization procedures. Contrast-enhanced MRI is important for determining the degree of necrotic tissue and the most appropriate treatment of stage II and IIIA disease. Contrast-enhanced MRI is not necessary in stages I, IIIB, IIIC, or IV because the degree of necrosis does not change treatment in these stages.

Before MRI became available, three-phase ^{99m}Tc scintigraphy was used to diagnose stage I Kienböck disease. The scintigraphic findings typically included an abnormal increase in uptake of ^{99m}Tc in both the blood pool and delayed phases. In rare cases a decrease in uptake was found. Bone scintigraphy has high sensitivity but low specificity. Therefore, it is not currently recommended for routine investigation for Kienböck disease [15].

Treatment

Despite the array of options there remains no definitive treatment of Kienböck

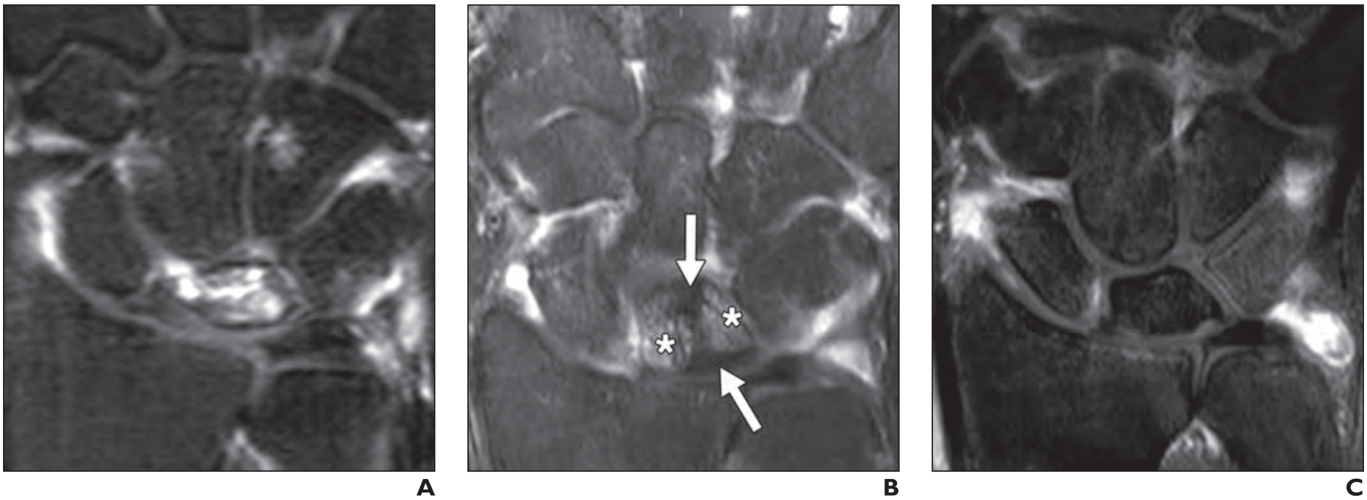

Fig. 8—Functional evaluation of Kienböck disease.
A, 33-year-old woman with stage IIIA Kienböck disease. Coronal fat-suppressed T1-weighted MR image shows intact perfusion (homogeneous contrast enhancement).
B, 25-year-old woman with stage II Kienböck disease. Partial enhancement of lunate (*asterisks*). Coronal fat-suppressed T1-weighted MR image shows partial areas of nonenhancing necrotic tissue (normally proximal) (*arrows*).
C, 21-year-old man with stage II Kienböck disease. Coronal fat-suppressed T1-weighted MR image shows no enhancement after contrast injection.

TABLE 3: Staging and Treatment of Kienböck Disease

Lichtman Stage	MRI Pattern	Ulnar Negative	Ulnar Neutral or Positive
I	Edema	Immobilization; capitate-shortening osteotomy or revascularization if not healing	Immobilization; capitate-shortening osteotomy or revascularization if not healing
II	Partial necrosis	3 mo of immobilization; joint leveling if not healing	3 mo of immobilization; capitate-shortening osteotomy or revascularization if not healing
	Complete necrosis	Joint leveling	Capitate-shortening osteotomy or revascularization
IIIA	Partial necrosis	3 mo of immobilization; joint leveling if not healing	3 mo of immobilization; capitate-shortening osteotomy or revascularization if not healing
	Complete necrosis	Joint leveling	Capitate-shortening osteotomy or revascularization
IIIB	Any	Arthrodesis[a] or proximal row carpectomy	Arthrodesis[a] or proximal row carpectomy
IIIC	Any	Lunate excision and arthrodesis[a] or proximal row carpectomy	Lunate excision and arthrodesis[a] or proximal row carpectomy
IV	Any	Proximal row carpectomy, total wrist arthroplasty, or total wrist fusion	Proximal row carpectomy, total wrist arthroplasty, or total wrist fusion

[a] Arthrodesis is scaphoid-capitate or scaphoid-trapezium-trapezoid intercarpal fusion.

disease. Many surgical procedures have been recommended, but it is not known whether these provide any improvement in the course of the disease [5]. The three main surgical options are mechanical unloading procedures, revascularization procedures, and salvage procedures.

Mechanical unloading procedures are aimed at encouraging spontaneous revascularization of the lunate bone. The most common technique is to level the joint by either radial shortening or ulnar lengthening. Capitate-shortening osteotomy is an alternative option for patients with positive variance [5]. Direct lunate revascularization procedures involve bringing a new source of blood supply to the lunate. In most cases this is done with a vascularized bone graft from a nearby bone [5]. Salvage procedures include proximal row carpectomy, carpal bone fusion (radioscapholunate fusion, total wrist fusion), and total wrist arthroplasty.

Lichtman stage I disease is usually managed conservatively with immobilization and nonsteroidal antiinflammatory drugs, but mechanical unloading procedures can be performed. Lichtman stages II and III are usually managed by either mechanical unloading or revascularization procedures. Lichtman stage IV is usually managed with salvage procedures.

Two other techniques are indicated in special cases. The first, for Lichtman stage IIIB disease, is aimed at repositioning the scaphoid bone into a neutral (45°) position (scaphoid-capitate or scaphoid-trapezium-trapezoid intercarpal fusion). The other technique, for Lichtman stage IIIC, is aimed at excising the collapsed lunate bone. The staging and treatment of Kienböck disease are summarized in Table 3.

Prognosis

Kienböck disease is often a progressive disorder resulting in joint destruction within 3–5 years if untreated.

Pseudo-Kienböck Lesions: Differential Diagnosis and Other Pathologic Conditions Affecting the Lunate Bone

Kienböck disease has radiologic findings common to other pathologic conditions affecting the lunate bone. Therefore, the diagnosis of Kienböck disease can be misinterpreted. The following are guidelines to the differential diagnosis of Kienböck disease (pseudo-Kienböck lesions).

Acute Fracture or Bone Contusion of the Lunate

Acute fracture or bone contusion of the lunate can be difficult to differentiate from stage I Kienböck disease at imaging. Often a wrist radiograph does not depict the fracture line, and in these cases wrist MRI or CT can be useful. At MRI the acute fracture or bone contusion can usually be seen as diffuse hyperintensity on T2-weighted images (Fig. 9A) and as a hypointense line on T1-weighted images. However, the key differentiating feature between Kienböck disease and acute fracture or contusion of the lunate is that acute fracture or contusion usually presents with a history of an acute episode of severe trauma to the hand.

Ulnar-Side Wrist Impaction Syndrome

Ulnar-side wrist impaction syndrome is a degenerative condition that results from chronic impaction between the ulnar head, the triangular fibrocartilage complex (TFCC), and the ulnar carpus. Radiographic findings include positive ulnar variance, subchondral sclerosis, and cystic changes in the ulnar head. In advanced cases these changes can also be found in the ulnar aspect of the proximal lunate and proximal radial aspect of the triquetrum. MRI findings include joint cartilage abnormality of the ulnar head, lunate, and triquetrum and TFCC degeneration (Fig. 9B). Bone marrow edema associated with cartilage abnormalities on images obtained with water-sensitive sequences can lead to misdiagnosis of this entity as Kienböck disease. The key differentiating features between these two entities are the distribution of bone edema in the ulnar aspect of the lunate, positive ulnar variance, and accompanying degenerative lesions in the TFCC and ulnar head that occur in ulnar-side wrist impaction syndrome [16, 17].

Infantile and Juvenile Lunatomalacia

Infantile and juvenile lunatomalacia is considered the Kienböck disease of children. Lunatomalacia and Kienböck disease, however, differ in treatment and prognosis, which has been a reason for their differentiation [18]. Lunatomalacia is a self-limiting condition that can usually be treated with immobilization and has a good prognosis. The cause is unknown but has been thought to be related to an autoimmune process [19]. Differentiation between infantile lunatomalacia, juvenile lunatomalacia, and Kienböck disease is based on patient age. Infantile lunatomalacia affects children 12 years old and younger, whereas juvenile lunatomalacia affects those 13 years old until the end of skel-

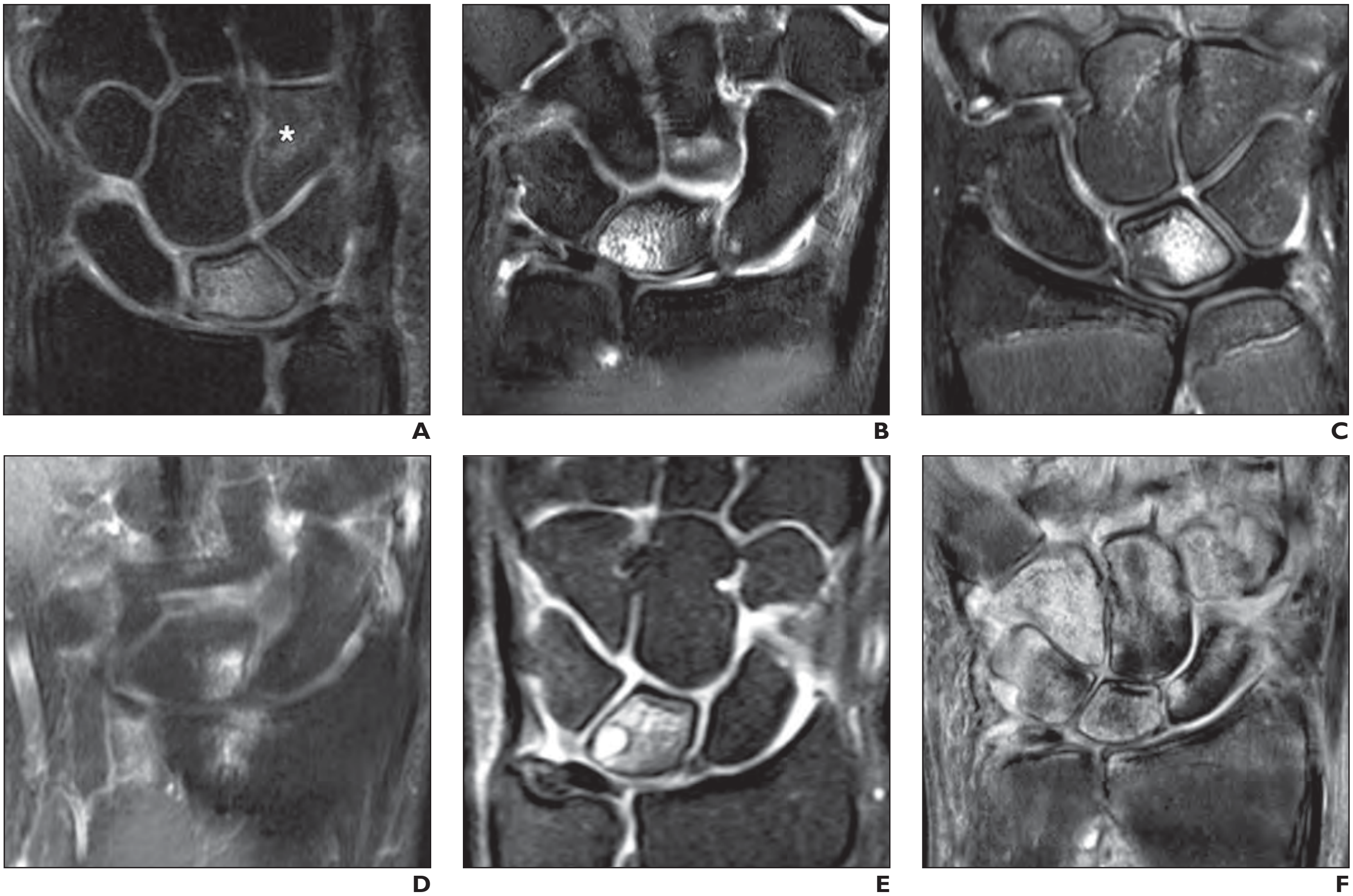

Fig. 9—Mimics of Kienböck disease.
A, 38-year-old man with wrist pain and swelling and history of trauma 1 week ago. Example of bone contusion of lunate. Coronal proton density–weighted fat-suppressed MR image shows diffuse hyperintensity in lunate and mild hyperintensity in hamate, signifying bone edema (*asterisk*).
B, 38-year-old man with intense medial wrist pain due to ulnar-sided wrist impaction syndrome. Coronal proton density–weighted fat-suppressed MR image shows diffuse lunate bone marrow edema.
C, 13-year-old girl with wrist pain due to juvenile lunatomalacia. Coronal T2-weighted fat-suppressed MR image shows focal hyperintensity within lunate bone (edema).
D, 60-year-old woman with wrist pain and stiffness due to degenerative arthritis. Coronal T2-weighted fat-suppressed MR image shows focal hyperintensity in lunate bone and radius (edema).
E, 32-year-old woman with dorsal ulnar wrist pain due to intraosseous ganglia. Coronal proton density–weighted fat-suppressed MR image shows radial-side intraosseous cyst and diffuse lunate marrow edema.
F, 42-year-old man with referred chronic wrist pain after wrist immobilization with cast. Example of complex regional pain syndrome. Coronal T2-weighted fat-suppressed MR image shows diffuse patchy edema in carpal bones and adjacent soft tissues.

etal maturity [18]. Thereafter patients are considered to have Kienböck disease. The radiographic findings range from no abnormality of the lunate to increased bone density and collapse of the lunate. At MRI focal hypointensity within the lunate bone may be seen on T1-weighted images and focal hyperintensity within the lunate on T2-weighted images [18] (Fig. 9C).

Arthritis

Rheumatoid arthritis, gout, and degenerative or posttraumatic arthritis can impair the normal bone marrow intensity pattern of the lunate and lead to misinterpretation of the imaging findings as Kienböck disease. The key differentiating features between arthritis and Kienböck disease is that patients with arthritis often have a different clinical presentation and demographic characteristics and more widespread imaging findings and usually lack negative ulnar variance (Fig. 9D).

Lunate Intraosseous Ganglia

Lunate intraosseous ganglia is a relatively uncommon condition that represents a benign, cystlike lesion that results from mucoid degeneration of the intraosseous connective tissue or synovial herniation into the underlying bone [20]. The intraosseous ganglia are seen on radiographs as a radial-side area of hyperlucency that communicates with the scapholunate joint space. Alternatively, they may appear as a hyperlucent area within the distal ulnar aspect of the lunate bone that communicates with the lunotriquetral joint space. At MRI intraosseous ganglia have a typical cystic appearance (low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, similar to water) (Fig. 9E). Furthermore, areas of bone marrow edema adjacent to the cyst can be seen as diffuse hyperintensity on T2-weighted images.

Bone Island

Large radial-side bone islands can mimic Kienböck disease on radiographs and T1-weighted MR images. They are seen as a focal sclerotic density on radiographs and as hypointense lesions on T1-weighted images. The key feature for diagnosis of a bone island or enostosis is a combination of low signal intensity on T2-weighted images and the presence of well-defined spiculation representing trabeculae entering the bone island.

Complex Regional Pain Syndrome

Complex regional pain syndrome is a chronic pain disorder of the sympathetic nervous system, usually as a result of trauma, casting, or infection. It is commonly a diagnosis of exclusion and is characterized by patchy and diffuse edema of the soft tissue and bone, usually not restricted to the lunate. The imaging findings are diffuse foci of hyperintensity on T2-weighted MR images (Fig. 9F).

Conclusion

Kienböck disease is a condition marked by avascular necrosis of the lunate bone. MRI can help in visualizing of the bone anatomy, the staging of Kienböck disease, and ruling out alternative diagnoses that mimic Kienböck disease (pseudo-Kienböck lesions). MRI therefore should be considered after conventional radiography in the care of patients with suspected Kienböck disease.

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