Kienbock’s Disease: Osteonecrosis of the Lunate

Kaleigh Petekavich
Abstract

Kienbock’s disease is a rare disease that affects the lunate of the wrist. It is a progressive disease by nature eventually causing a necrosis of the lunate, which will lead to a complete collapse of the lunate, as well as a malalignment of the carpal bones. Though the disease was discovered in 1910 by Robert Kienbock, its specific etiology is still unknown. There are many different factors that are believed to contribute to the development of the disease, but none have given a precise reasoning behind the actual origin. Kienbock’s disease is primarily diagnosed through the use of the imaging modalities: diagnostic radiography, magnetic resonance imaging (MRI), and computed tomography (CT). Each imaging modality plays a specific role in the diagnosis and staging of the disease. Initial treatment of Kienbock’s disease is typically nonsurgical by immobilization. If immobilization is not effective and the disease continues to progress and cause discomfort, there are many different treatments available depending on the stage of the disease. The treatments appear to be highly effective resulting in an overall satisfying prognosis for the patient.
In 1910 an Australian radiologist by the name of Robert Kienbock discovered numerous cases of people that suffered from malacia of the lunate, or osteonecrosis of the lunate, which is now known today as Kienbock’s disease.\(^1\) Kienbock’s disease is a slowly progressive disorder that will eventually lead to a collapse of the lunate, causing an alteration of the carpal bones surrounding it; it can also be referred to as lunatomalacia or osteochondrosis of the lunate bone.\(^2\) Kienbock’s disease is so rare that it affects less than 5 in 10,000 people.\(^3\) Even though it was discovered over 100 years ago, it still has an unknown etiology. Over the years some possible theories have developed regarding the origin. There are many different treatments depending on the stage of the disease; this disease has the possibility of being debilitating, but many of the treatments can be very successful. The way this disease is diagnosed is primarily through the use of the imaging sciences: diagnostic radiography, magnetic resonance imaging and computed tomography.

Methods

A report was conducted on the causality, imaging and treatment of Kienbock’s disease. For this study there were four electronic databases used: Cinahl, Medline-EBSCO, Medline-Pubmed and Google Scholar. Terms associated to Kienbock’s disease were posted into each of the databases. The terms included “Kienbock’s disease”, “Kienbock’s disease imaging”, and “Kienbock”. Only resources that were within the past 10 years were utilized and articles that had free access from the databases. Twenty resources were obtained for this study.
Etiology

The etiology for Kienbock’s disease appears to be multifactorial. Researchers believe there are some predisposing factors that may put a person at risk for developing the disease, these include: ulnar variances, morphology, vascularization, trauma, trabeculae and systemic conditions. Ulnar variance refers to the length of the radius and ulna at the distal end of the bones on articulating surfaces (see Figure 1).

**Figure 1** *(on the left) Normal Wrist, (on the right) Wrist with Kienbock’s disease, ulna is shorter than the radius.*

The lengths can be measured using radiographs and what is known as the perpendicular method. The perpendicular method utilizes a posteroanterior radiograph of the wrist, then at the ulnar point of the distal articular surface of the radius, a line is drawn that is perpendicular to the long axis of the bone. The distance is measured between this point and the ulnar dome to determine the ulnar variance in millimeters. Positive ulnar variance demonstrates the articular surface of the ulna protruding more distally, while a neutral ulnar variance will have both of the articular surfaces of the radius and ulna at an equal level. A negative ulnar variance exhibits the surface of the ulna more proximally and occurs in 78% of all Kienbock’s disease cases. The
morphology of the lunate may influence the chances of developing Kienbock’s disease, they tend to be square or rectangular in shape, such as type one or two of the Antuna Zapico classification of the lunate (see Figure 2).

**Figure 2:** Antuna Zapico’s classification of the lunate bone.

In addition to the shape of the lunate, variations of the capitate morphology relates to a change in the lunate anatomy. The lunate is supplied with nutrient rich blood through the volar and dorsal poles. There are a few scenarios in which a decreased flow of blood in these areas could result in this disease, such as a lack of collateral vessels, existence of only one palmar vessel or issues with venous outflow. When there is suboptimal arterial supply, trauma can impede on this more, causing a higher risk of developing the disease. Trauma by itself will not cause avascular necrosis of the lunate, it’s always in conjunction with other factors.

A recently explored predisposing factor is the trabeculae of the bone. The trabeculae of lunates were 2.67 times denser and 1.84 thicker in patients with Kienbock’s disease than unaffected lunates. Trabeculae are not randomly arranged, they are carefully constructed by the body to support the areas that experience the most stress. As a person moves, they create stress on the trabeculae and it can grow and change shape and direction in order to give them the support they need to have an active life. So loss of
blood supply that will occur with this disease will create the fragmentation, or fracture, in the lunate; it will then have a subchondral collapse with loss of radially arranged trabeculae, which becomes thick as a remodeling to withstand the stress. Finally there is a slight correlation between Kienbock’s disease and systemic conditions such as autoimmune disease and sickle cell anemia.

**Symptoms**

Some cases of Kienbock’s disease can actually be asymptomatic, but most exhibit symptoms. It is often difficult to diagnose, especially the beginning stages, because the symptoms mimic other conditions. Some of the more frequent symptoms observed with this disease is dorsally located central wrist pain and tenderness, especially around the lunate. As well as inflammation of the synovial membrane that can lead to weakness, limited mobility and a loss of grip strength, and even at times the development of carpal tunnel syndrome. Flexion and extension also tend to be restricted. See Table I below for a demonstration of the changes in a patient’s range of movement and strength in his affected right wrist.

<table>
<thead>
<tr>
<th>Table I. Functional evaluation</th>
<th>Right</th>
<th>Left</th>
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<tbody>
<tr>
<td><strong>Range of movement</strong></td>
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<tr>
<td>Flexion</td>
<td>30</td>
<td>85</td>
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<tr>
<td>Extension</td>
<td>10</td>
<td>55</td>
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<tr>
<td>Radial deviation</td>
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<td>Ulnar deviation</td>
<td>20</td>
<td>55</td>
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<tr>
<td>Pronation</td>
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<td>80</td>
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<tr>
<td>Supination</td>
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<td>75</td>
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<tr>
<td><strong>Strength (kg)</strong></td>
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</tr>
<tr>
<td>Grip</td>
<td>9</td>
<td>16</td>
</tr>
<tr>
<td>Pinch</td>
<td>9.5</td>
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Kienbock’s disease has 4 stages, with stage 3 being subdivided. In Stage 1 of the disease, activities may aggravate it, causing minor pain especially when in extension under an axial weight; this stage tends to mimic symptoms of a wrist sprain. Stage 2 shows signs of swelling from synovitis, inflammation of the synovial membrane, and pain becomes continual often even at night. Stage 3 the pain becomes more constant and symptoms mimic that of early degenerative arthritis. Stage 4 the wrist becomes stiff and mimic the symptoms of advanced degenerative arthritis. For morphological evaluation of the lunate to be made, use the Lichtman classification to determine the extent of the disease. The Lichtman classification uses findings of CT, diagnostic radiography and MRI to help stage the disease, as well as using the shape of the lunate and the density of the bone itself. The most common stage at initial appearance for Kienbock’s disease is stage III. The symptoms of the disease may not also correspond with the radiographic severity, which makes it even more difficult to evaluate the natural history of the ailment.

Imaging

Diagnostic Radiography

Diagnostic radiography is the first imaging modality utilized to diagnose Kienbock’s disease in an effort to assess the extent of the disease. Diagnostic radiography by itself is not sufficient in confirming later stages IIIB and IV. It is also used to assess pertinent anatomy of the wrist useful in diagnosing and staging – ulnar variance, radial inclination, carpal height, radioscaphoid angle, and lunate size and morphology. Stage I will appear normal in shape on a radiograph with no sign of
disease. Stage II will appear normal in shape, but with slight sclerosis. Sclerosis can be seen on an image as an increase in bone density in comparison with the other carpals. Stage III-A, the lunate collapses entirely but the carpal alignment remains stable and the radioscaphoid angle is less than 60 degrees. Stage III-B, the lunate collapses entirely and there is instability in the carpal bones and the radioscaphoid angle is greater than 60 degrees. This stage can be demonstrated by a cortical ‘ring sign’ on a posteroanterior radiograph. Stage III-C, the lunate collapses entirely and often a coronal lunate fracture is present. Stage IV will show radiocarpal and midcarpal degenerative arthritis as well as widening of volar and dorsal end of lunate. See Figure 3 for visualization of the different stages of Kienbock’s disease on radiographs.

Figure 3: Lichtman classification Stage 1: normal x-rays, Stage 2: the lunate appears to be homogenously or heterogeneously denser (A); Stage IIA: lunate collapsed with non-fixed adaptive severance of the carpus (B); Stage IIB: lunate collapsed with fixed adaptive severance of the carpus (C); Stage IV: osteoarthritis (D and E).

Magnetic Resonance Imaging

MRI is the next best imaging modality to utilize after diagnostic radiography whenever there is a suspicion of Kienbock’s disease. MRI is necessary in order to properly diagnose stage I, since it cannot be diagnosed by diagnostic radiography or CT alone. Contrast-induced MRI is also useful for helping to figure out the degree of necrotic tissue in stages II and IIIA, because these stages tend to be the ones where there
could be changes in necrosis. Normally, on both T1 and T2 weighted images, a consistently decreased signal intensity implies a marrow pathology that is typical of avascular necrosis. Stage I the shape and density is the same, but there is edema formation detected in the bone marrow, demonstrated by a decreased signal on the T1 weight image and a increased signal on the T2 weighted image (See Figure 4).²

![Figure 4: MRI Images of Stage I (A) Lunate bone appears normal on radiograph (B) Coronal T1-weighted image allows visualization of the hypointensity occurring in the lunate bone.]

Stage II will still appear to have a normal shape, but the lunate will become denser. T1 weighted images will have a low signal intensity. T2 weighted images will have a variable signal intensity, mainly on the side of the lunate closest to the radius. Stage III-A and stage III-B demonstrate the collapse of the lunate. Stage III-C also demonstrates the collapse of the lunate, as well as a coronal lunate fracture. Stage IV demonstrates a complete collapse of the lunate and osteoarthritic changes to the joint spaces (See Figure 5).⁵
Figure 5: MRI Images of Stage IV (A) A collapse of the lunate and osteoarthritic changes are visualized in the radiolunate and midcarpal bones in the radiograph (B) Sagittal T1-weighted MR image demonstrates a coronal fracture of lunate bone as well as elongation (arrow) (C) Coronal T1-weighted MR image demonstrates a hypointensity of the lunate bone as well as collapse and osteoarthritic changes in the radiolunate and midcarpal joints (arrows).\(^5\)

Computed Tomography

The best imaging modality to depict bone anatomy in Kienbock’s disease is computed tomography (CT); it is also utilized to help stage more advanced stages of disease. CT is good to utilize if the lunate has become fragmented or to detect any fractures within the lunate. A common fracture demonstrated is a coronal lunate fracture, which would result in volar and dorsal fragments. Stage I will appear normal in shape. Stage II will also appear normal in shape, but have slight sclerosis. Sclerosis is demonstrated in the image by the increase in bone density in comparison with other carpal bones. Stage III-A the lunate collapses entirely, but carpal alignment still remains the same and the radioscaphoid angle is less than 60 degrees. Stage III-B the lunate collapses entirely and there is instability in the carpal bones and the radioscaphoid angle is greater than 60 degrees. Stage III-C the lunate collapses and a coronal lunate fracture can be seen on the image (See Figure 6). Stage IV will demonstrate radiocarpal or midcarpal degenerative arthritis and widening of the volar and dorsal ends of lunate.\(^{15}\)
Figure 6: (A) A fracture in the midportion of the lunate bone ranging from both of the articular surfaces can be seen on this initial sagittal view (arrow). (B) 1 month later another sagittal view was done which revealed comminution and a further corrosion of the lunate (arrow).16

Treatment

Treatments may vary during different stages based off the patient and their ulnar variance. Extended immobilization is the first step, normally the length of 3 months, in an effort to boost spontaneous revascularization as well as non-steroidal inflammatory medications. Some of the immobilization techniques utilized are external fixation, cast, or temporary intercarpal pinning. If revascularization is not successful or if it was diagnosed late, it will require a more aggressive treatment; surgery is offered after six months if their pain persists. Treatments often will either mechanically unload the lunate or reestablish vascularity. There are many different treatments, the key to picking the right one for that particular patient is to stage their disease and evaluate their anatomy at the time of the diagnosis.2

Stage I Treatment

For stage I, no matter the ulnar deviance, the treatment is the same. First step is to immobilize, but if it is not healing then capitate-shortening osteotomy or revascularization of the lunate may be necessary. Dorsal distal radius pedicled
vascularized bone grafting (VBGs) is one of the procedures suggested for this stage. VBGs allow for primary bone healing as well as new bone formation by providing the embedding of sustainable osteoclasts and osteoblasts. Radial shortening or ulnar lengthening can also be performed to help to take some of the load off of the lunate (See Figure 7).

![Figure 7: Illustration demonstrating ulnar lengthening.](image)

**Stage II Treatment**

For stage II with partial necrosis the first step is to try 3 months of immobilization. If immobilization is still not helping it to heal, the next step is to perform joint leveling if the patient has ulnar negative deviance or revascularization of the lunate if the patient has ulnar positive or neutral deviance. For stage II with complete necrosis, the immobilization step is skipped and either the joint leveling for the ulnar negative deviation patient or the radial-shortening osteotomy procedure for the ulnar positive or neutral patient will be performed (See Figure 8). Dorsal distal radius pedicled vascularized bone grafting (VBGs) are also recommended for this stage of the disease as well.
Stage III Treatment

For stage III-A with partial necrosis the first step is to try 3 months of immobilization for all types of ulnar deviances. If the patient is not healing, it is suggested to perform joint leveling if the patient has ulnar negative deviance or revascularization of the lunate if the patient has ulnar positive or neutral deviance. For stage III-A with complete necrosis, skip the immobilization step and perform either the joint leveling for the ulnar negative deviance patient and the capitate-shortening osteotomy procedure or revascularization for the ulnar positive or neutral patient. Stage III-A is the final stage that a dorsal distal radius pedicled vascularized bone grafting (VBGs) is recommended.

For stage III-B no matter the ulnar deviance, performing arthrodesis or a proximal row carpectomy is the typical treatment route. For proximal row carpectomy to be successful, the capitate head can’t have any degenerative changes yet. The purpose of intercarpal arthrodesis is to relocate the scaphoid into a neutral (45 degree) position. By
correcting this malalignment, it will slow or possible prevent the advancement of arthritis within the midcarpal and radiocarpal joints.²

For stage III-C no matter the ulnar deviance, one of the following will be performed: proximal row carpectomy or arthrodesis and excision of the lunate (See Figure 9).

![Illustration of proximal row carpectomy](figure9.png)

**Figure 9:** *Illustration of proximal row carpectomy* ¹⁸

*Stage IV Treatment*

For stage IV no matter the ulnar deviance, one of the following will be performed: proximal row carpectomy, total wrist arthroplasty or total wrist fusion. Intercarpal fusion tries to keep the scaphoid in its correct position as well as trying to preserve the carpal height, to thwart the possibility of degenerative arthritis; if the lunate can be conserved it will try to take some of the load off of it (See Figure 10). There is a significant loss of movement in fusions as well as it may have complications such as nonunion or late arthritis.¹⁹ Wrist denervation can also be performed in conjunction with other treatments or by itself.
Figure 10: Illustration of a wrist fusion

Prognosis

Ultimately all the treatments aim to relieve the pain, increase function and stop the progression of the disease. Since Kienbock’s disease is a progressive disorder, if left untreated, it could result in joint destruction. In a recent survey, patients who were treated nonoperatively reported an improvement in their symptoms by 63% and patients who were treated surgically reported between 72% and 90% no matter what procedure or stage of disease. Below are some graphical representations of patients who have undergone different treatments and their level of pain relief or absence (See Figure 11).

Figure 11: Graphical illustration of pain relief in the early stages (on the left). Graphical illustration of pain relief in the late stages (on the right).
Conclusion

This paper describes the different theories proposed on the origin of this rare disease, typical symptoms of the four stages, its presentation in different imaging modalities and the many different treatments available for patients. Diagnostic radiography, MRI and CT all play an important role in diagnosing, staging and monitoring the disease. It is important for an imaging professional to be educated on Kienbock’s disease so they know what to look for in order to give the patient the proper care. Though it is difficult to catch in the beginning stages, improvements in medical imaging, as well as continued research, help to increase the likelihood of a diagnosis at an earlier stage, therefore stopping the progression of the disease by earlier intervention.
AMA References


18. Houstonmethodist.org. Houston Methodist. 2015. Available at:
