Kienböck Disease: Moving Forward

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Over the past decade, a plethora of new information has been reported regarding etiology, natural history, classification, and treatment options for lunate osteonecrosis. New disease classifications have been described based on advanced imaging determination of lunate viability as well as a cartilage-based arthroscopic classification. Here we review the newest literature regarding Kienböck disease and present a new treatment algorithm that incorporates the traditional osseous classification system with a perfusion/viability classification and an articular cartilage-based classification. (J Hand Surg Am. 2016; - ( - ): - e. Copyright © 2016 by the American Society for Surgery of the Hand. All rights reserved.)

Key words Kienböck, lunate, osteonecrosis, avascular, lunatomalacia.

I t has been over 100 years since Robert Kienböck1 published his observations on osteomalacia of the lunate, yet many aspects of the disorder remain an enigma. Although there is general consensus that the pathogenesis of Kienböck disease is related to non-physiological stress transmission across the lunate, acting in concert with one of several possible vascular abnormalities, it is often difficult to determine the precise etiology. Furthermore, the treatment options are many, but few have been subjected to high-level, statistically valid outcome studies.

The natural history of untreated Kienböck disease has been well described in terms of progression of clinical findings and serial changes on standard x-rays.2–5 The classification scheme devised in 19776 and modified in 19937 is widely used as a guide for treatment at various stages of the disease (Table 1).

In 2010, the classification system was expanded to include a hypothetical stage 0 and a well-represented stage IIIC disease.8 Stage 0 disease is analogous to stage 0 of the Association Research Circulation Osseous International classification9 of hip osteonecrosis in which patients present with intermittent pain but standard radiographs and magnetic resonance imaging (MRI) studies are normal. The advantage of diagnosis at this stage would be the potential to abort true osteonecrosis onset by correction of etiological factors associated with later stages of the disease. In stage IIIC disease, a coronal fracture or fragmentation of the lunate exists. In the authors’ personal experience, when this occurs, the prognosis for lunate revascularization is poor. Treatment recommendations are lunate excision with prosthetic replacement, proximal row carpectomy, or intercarpal arthrodesis.

Despite the usefulness of existing classification systems, recent advances in diagnostic imaging (particularly gadolinium perfusion MRI) and arthroscopy have provided information that has taken our understanding of the natural history and prognosis to a higher level.10,11 In addition, new studies on the biomechanical effects of local anatomical variations have enhanced our understanding of the pathogenesis and prognosis. Finally, we have learned that Kienböck disease in children, adolescents, and the elderly behaves differently from the disease in the typical young adult. As a result, we now have the ability to gather much more information to provide useful treatment choices for each individual patient.

Many promising new treatment options for Kienböck disease have been introduced, including minimally invasive techniques, revascularization procedures, lunate replacement arthroplasty, and late-stage reconstruction.
The goal of this manuscript is to review these recent advances to see how this varied information can be integrated into a more clinically reliable treatment algorithm.

**PATHOANATOMY OF KIENBÖCK DISEASE**

Ulnar variance has long been suggested as a factor in the development of Kienböck disease. Afshar et al. noted a significant ulnar-negative variance in wrists with Kienböck disease compared with unaffected controls. Goeminne et al. identified a significant difference in ulnar variance between early- and late-stage Kienböck disease, with a more negative variance noted in advanced disease. Although ulnar variance has been observed to have an association with the development of Kienböck disease, no direct causality was found in a meta-analysis performed by Stahl et al in 2013.

Lunate morphology itself has also been identified as a potential etiological and prognostic factor in Kienböck disease. Thienpont et al. found significant smaller lunate diameter and height, more radially inclined lunate tilt, and a flatter radial inclination in patients with Kienböck disease compared with uninvolved contralateral control wrists. Antuno-Zapico described 3 geometrically different lunate morphologies based on the convergence of the proximal and medial borders into a crest. In type I (trapezoidal) lunates, the angulated trabecular bone pattern was noted to be weakest and most susceptible to fatigue. In addition, the type I pattern had the highest incidence of ulnar minus variance.

In 1990, Viegas et al. categorized lunate morphology based on the absence or presence of a distal medial facet (type I vs type II, respectively). In a 2015 report, Rhee et al. performed a retrospective review of 105 wrists with Kienböck disease and observed more advanced disease in type I compared with type II lunates at presentation. Coronal lunate fractures were more prevalent in type I lunates. In lunates without a coronal fracture, radioscapoid angles were greater in type I lunates than in type II (53° vs. 45°). Rhee et al. suggest that Viegas type II lunates are protective against coronal fractures and scaphoid flexion deformity and, thus, deter progression to advanced disease stages.

In 2014, Low et al. published a study using 3-dimensional micro-computed tomography to compare the osseous microanatomy of normal and Kienböck lunates. The normal lunate has trabeculae spanning from proximal to distal subchondral bone plates acting as a scaffold to maintain lunate height. The subchondral plate of the Kienböck lunate is fractured and sometimes includes fragmentation and reabsorption, especially of the proximal cortex. The medullary bone often has significant reabsorption.

Researchers have continued to look for variations in vascular anatomy as a cause of Kienböck disease. Pichler and Putz reported on a previously undescribed venous plexus on the palmar and dorsal surfaces of the lunate. This plexus is connected to the palmar and dorsal connective tissue. Pichler and Putz hypothesize it may be a point of obstruction in venous drainage, which could lead to increased intraosseous pressure and subsequent osteonecrosis.

Kawanishi et al. evaluated the kinematics of the wrist in advanced Kienböck disease and observed...
excessive scaphoid flexion in stage IIIB Kienböck disease without the same dorsal scaphoid shift and radioscaphoid incongruity noted in scapholunate dissociation. This distinction may explain why patients with advanced Kienböck disease may remain asymptomatic, and why there are fewer radioscaphoid degenerative changes than commonly noted in advanced scapholunate advanced collapse wrists.

### IMAGING AND LUNATE VIABILITY

Plain radiographs have been traditionally used to diagnose and monitor the progression of Kienböck disease and is the imaging modality behind the traditional osseous classification.\(^6\) Once MRI became widely available, the Lichtman classification was modified to incorporate T1-weighted signal changes without radiographic abnormalities (stage I disease).\(^7\)

In recent years, both T1- and T2-weighted images have been used to evaluate the viability of bone marrow.\(^22\) On T1-weighted images, normal viable marrow has homogeneous high signal intensity. Loss of this signal can be caused by anything that replaces viable marrow (edema, neovascularization, space-occupying lesions, bone necrosis, or sequestrum). T2-weighted images have traditionally been used in Kienböck disease to distinguish between fluid-containing tissue (high T2 signal) and nonvascular or desiccated tissue, such as a sequestrum. However, T2-weighted images cannot distinguish between edema and neovascularization, which have different prognostic values.

Schmitt et al\(^{22}\) reported gadolinium perfusion techniques to enhance fat-suppressed T1-weighted sequences. They distinguished low-signal edema from enhanced high-signal neovascular repair tissue. Using these techniques, they were able to identify 3 zones or patterns of enhancement of the Kienböck lunate: the proximal necrotic bone with no enhancement, the intermediate hypervascular repair zone, and the distal normal lunate. The hyperenhancement in the reparative zone indicates areas with a good healing prognosis, whereas low signal indicates a poor prognosis owing to nonviable marrow.

Based upon these findings, Schmitt et al\(^{23}\) described a classification that depends on lunate signal changes after administration of intravenous gadolinium contrast (Table 2). In MRI stage I, homogenous lunate enhancement indicates marrow edema and intact perfusion. Magnetic resonance imaging stage II shows inhomogeneous signal with contrast enhancement of the reparative zone and viable distal bone with a necrotic proximal lunate. Magnetic resonance imaging stage III has no enhancement, corresponding to complete lunate osteonecrosis. Schmitt et al\(^{23}\) assume that these stages correlate with lunate revascularization potential.

### ARTHROSCOPY IN THE ASSESSMENT AND MANAGEMENT OF KIENBÖCK DISEASE

In 2006, Bain and Begg\(^{24}\) described an arthroscopic method of assessment and classification of Kienböck disease. They defined an articular surface as “nonfunctional” if there was extensive fibrillation, fissuring, extensive articular loss, floating articular surface, fracture, or arthritis. The grading system is based on the number of nonfunctional articular surfaces ranging from grade 0 to grade 4 (Fig. 1). Note there is a grade 2a (proximal lunate and lunate fossa) and a grade 2b (proximal and distal lunate). Bain and Begg observed that preoperative imaging often underestimated the severity of articular involvement.

Bain and Begg\(^{24}\) further described a treatment algorithm based on arthroscopic grading. The principle underpinning the recommendations is to excise, fuse, or bypass the “nonfunctional” articulations.\(^{21–26}\) For example, scaphocapitate fusion bypasses the diseased central column and loads the wrist through the radial column.\(^{21}\) This is particularly useful in grades 2b, 3, and 4 disease. Because patients in all groups had synovitis, synovectomy may be considered in all stages.

In 2011, Bain and Durrant\(^{25}\) again stressed the importance of arthroscopic evaluation of articular cartilage but also noted other modalities, including MRI and computed tomography, are useful for assessing the subchondral plate and the articular cartilage.

### TABLE 2. Schmitt Classification of Lunate Vascularity/Viability

<table>
<thead>
<tr>
<th>Pattern</th>
<th>MRI Findings</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>Normal signal</td>
<td>NA</td>
</tr>
<tr>
<td>A</td>
<td>Homogenous enhancement of lunate with marrow edema; <strong>intact lunate perfusion</strong></td>
<td>Good</td>
</tr>
<tr>
<td>B</td>
<td>Inhomogeneous signal with enhancement of the reparative zone and viable distal bone; necrotic proximal lunate (<strong>partial osteonecrosis</strong></td>
<td>Intermediate</td>
</tr>
<tr>
<td>C</td>
<td>No contrast enhancement; <strong>complete osteonecrosis</strong></td>
<td>Poor</td>
</tr>
</tbody>
</table>
NATURAL HISTORY IN CHILDREN AND THE ELDERLY

The osseous classification system described previously was based primarily on findings in young adults. However, lunate osteonecrosis has also been observed in pediatric and elderly patients, and presentation and prognosis within these populations appears to vary considerably from that in the typical 20 to 40 year old. Because the disease behaves differently, it is important to take age into account when considering treatment options for these patients.

The presentation of Kienböck disease in pediatric patients is similar to that in adults, with dorsal tenderness, synovitis, and swelling, with a decreased range of motion and grip strength.8,27 However, progression to advanced-stage disease with lunate collapse is much less predictable.8

Irisarri et al28 subdivided pediatric Kienböck disease into infantile (12 years and younger) and juvenile (13 years to skeletal maturity) types. The infantile group had nonsurgical treatment, and at final follow-up, all had excellent outcomes, with lunate revascularization evident on MRI. However, in juveniles, 30% of patients treated with immobilization had progression and required a joint-leveling procedure. These observations led Irisarri et al to recommend immobilization for patients younger than 15 years and to reserve surgical management for the older patient with disease progression.

Ferlic et al29 observed improvement in the carpal height ratio in pediatric Kienböck treated nonsurgically, but the disease progressed.

FIGURE 1: Articular-based approach to Kienböck disease. The Bain/Begg arthroscopic classification system is derived from the number and location of nonfunctional articular surfaces in the central column. Treatment algorithm is based on the principle to excise, fuse, or bypass the nonfunctional articular surfaces. (Modified from Bain GI, Begg M. Arthroscopic assessment and classification of Kienböck’s disease. Tech Hand Up Extrem Surg. 2006;10[1]:8–13.)
with a normalized appearance of the lunate on radiographs. Kim et al\textsuperscript{30} observed improved lunate height with immobilization alone in patients younger than 12 years of age. This suggests that, in pediatric patients, there is less progression and normal lunate architecture may reestablish itself without surgical intervention.

Radial epiphysiodesis has been suggested as an approach to radial shortening for pediatric patients.\textsuperscript{31} Epiphysiodesis restricts radial overgrowth while allowing a short ulna to grow preferentially. Because long-term outcome data are unavailable, further investigation is required before a recommendation for routine use of radial epiphysiodesis can be made.

Kienböck disease also appears to present differently in the elderly than in pediatric or adult cohorts.\textsuperscript{32–36} Taniguchi et al\textsuperscript{33} followed a cohort of 15 patients older than 70 (3 stage II, 6 stage IIIA, and 6 stage IIIB). Negative ulnar variance was less frequently associated and there was a higher prevalence among women, with more diffuse or advanced disease at presentation. At mean follow-up of 5.6 years, all patients had progressed to stage IV disease. Despite radiographic progression, all patients had good to excellent clinical outcomes without surgical intervention.

Separate studies have noted an association between osteoporosis and Kienböck disease in elderly patients.\textsuperscript{35,36} With decreased bone density, microfractures within the osteoporotic lunate may serve as a catalyst for osteonecrosis. Further investigation of a causal relationship between osteoporosis and Kienböck disease is warranted.

**RECENT THERAPEUTIC ADVANCES**

Higgins et al\textsuperscript{37,38} have described the use of a vascularized medial femoral trochlea graft to reconstruct an avascular lunate and restore carpal height in stage III (A or B) disease. The convexity of the medial femoral trochlea is similar to that of the proximal lunate and the vascularized osteocartilaginous graft provides its own blood supply when reanastomosed. Because the graft replaces the proximal and central necrotic lunate with viable bone and cartilage, this technique provides an alternative to excision or fusion in advanced cases with extensive proximal lunate cartilage damage and collapse.(See links to surgical technique for pedicle revascularization that can be used when the articular surfaces are not damaged—either as bone graft or arterial implantation.\textsuperscript{a,b})

Lunate excision and replacement with silicone,\textsuperscript{1} autogenous tendon, metallic spheres, or even pisiform bone\textsuperscript{29} has been performed for many years with poor or inconsistent outcomes.\textsuperscript{40} (This link provides information on a variety of vascularized bone grafts including pisiform.\textsuperscript{c}) Newer attempts at lunate arthroplasty using pyrocarbon implants have shown promising early results with respect to patient satisfaction and range of motion.\textsuperscript{31,42} Further investigation into pyrocarbon lunate replacement is warranted.

Hierner and Wilhelm\textsuperscript{43} described lunate excision with capitate lengthening for patients with preserved lunate fossae and capitate head articular surfaces but nonfunctional articular surfaces on the lunate. Through a capitate osteotomy and distraction osteogenesis via an external fixator, the capitate is lengthened to articulate with the radius through the lunate facet. The procedure preserves hand strength while restoring carpal height and appears to be promising for stage III Kienböck with a functional lunate facet and capitate head.

As in other areas of reconstructive hand surgery, there has been a recent movement toward the development of minimally invasive surgical treatments. Temporary pinning of the scaphotrapeziotrapezoid (STT) joint was first described by Yajima et al\textsuperscript{44} as an adjuvant to surgical revascularization of the lunate. If the scaphoid is pinned in the extended position, it unloads the lunate long enough to allow the revascularization to become established. Yajima et al\textsuperscript{44} used the second or third dorsal metacarpal artery and vein in addition to STT pinning and noted complete pain relief in 80% of patients after surgery. Others have reported the use of the procedure without revascularization and noted spontaneous lunate revascularization on MRI with improved wrist motion and grip strength.\textsuperscript{44–48} Temporary pinning of the STT joint is now recommended both as an adjunct to revascularization and as an attractive option for primary treatment of pediatric Kienböck disease given the high likelihood of spontaneous lunate revascularization.

The use of a balloon to perform “lunatoplasty” to restore lunate morphology has recently been described.\textsuperscript{49} A major concern during lunatoplasty is cement extravasation into the carpus, which, if unrecognized, may be devastating. Limited information is available regarding the biomechanical effects of increased lunate density after lunatoplasty using cement. Given the limited long-term outcomes of this procedure, it is not currently recommended as a routine treatment option for Kienböck disease.

Core decompression of the distal radius has been observed to provide pain relief as well as potentially halt the progression of Kienböck disease.\textsuperscript{50} The exact mechanics of this response is unclear. Sherman et al\textsuperscript{51} identified a decrease in stiffness of the distal radius after core decompression but no change in force distribution across the radiocarpal joint. Illarramendi
et al.\textsuperscript{50} suggest a regional vascular response from the procedure may lead to symptomatic improvement.

Advances in wrist arthroscopy have furthered the trend toward minimally invasive approaches to decrease patient morbidity.\textsuperscript{26} Arthroscopic synovectomy can be considered in all disease stages.\textsuperscript{24,52} Arthroscopic-assisted and open lunate decompression has also been described.\textsuperscript{52–54} Lunate decompression may be a reasonable option if there is an intact lunate (Lichtman stage I or II) with functional articular surfaces (Bain 0). However, it is less likely to be successful once lunate collapse has occurred (stage III) or if the articular cartilages are compromised (Bain 1, 2, 3, 4). Arthroscopic techniques within the wrist have also advanced to the

### Table 3: Expanded Algorithm for the Treatment of Kienböck Disease

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Osseous (Lichtman)</th>
<th>Vascular (Schmitt)</th>
<th>Cartilage (Bain)</th>
<th>Description</th>
<th>Principle</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lunate Intact</strong></td>
<td></td>
<td></td>
<td></td>
<td>Early, infantile, juvenile, old</td>
<td>Immobilization</td>
<td>Orthosis (cast)</td>
</tr>
<tr>
<td>0, I, II Teen-bock</td>
<td>A</td>
<td></td>
<td>0</td>
<td>Intact lunate</td>
<td>Lunate decompression</td>
<td>Lunate forage ± bone graft</td>
</tr>
<tr>
<td>I, II</td>
<td>A, B</td>
<td>0</td>
<td>Intact lunate Negative ulnar variance</td>
<td>Unload lunate</td>
<td>Radial shortening or epiphysiodesis (ulnar lengthening)</td>
<td></td>
</tr>
<tr>
<td>I, II, IIIA</td>
<td>A, B</td>
<td>0</td>
<td>Intact lunate Positive ulnar variance</td>
<td>Unload lunate</td>
<td>STT pinning, capitate shortening (radial wedge osteotomy, external fixation)</td>
<td></td>
</tr>
<tr>
<td><strong>Lunate Compromised</strong></td>
<td></td>
<td></td>
<td></td>
<td>Necrotic lunate, intact cortex, cartilage</td>
<td>Lunate revascularization</td>
<td>Vascularized bone graft (nonvascularized graft)</td>
</tr>
<tr>
<td>0, I, II, IIIA</td>
<td>B, C</td>
<td>0</td>
<td>Proximal lunate collapse</td>
<td>Lunate reconstruction or excision ± replacement</td>
<td>MFC osteochondral reconstruction</td>
<td></td>
</tr>
<tr>
<td>IIIA, IIIB</td>
<td>B, C</td>
<td>1</td>
<td>Entire lunate collapse</td>
<td>Lunate excision ± replacement or bypass central column</td>
<td>Lunate replacement, (lunatoplasty)</td>
<td></td>
</tr>
<tr>
<td>IIC</td>
<td>B, C</td>
<td>2b</td>
<td>Radiolunate joint compromised</td>
<td>Fuse radiolunate</td>
<td>PRC, capitate lengthening, SC fusion, pyrocarbon replacement</td>
<td></td>
</tr>
<tr>
<td><strong>Wrist Compromised</strong></td>
<td></td>
<td></td>
<td></td>
<td>Hemiarthroplasty or bypass central column</td>
<td>SC fusion (STT fusion)</td>
<td></td>
</tr>
<tr>
<td>IIB</td>
<td>B, C</td>
<td>2a</td>
<td>Only capitale surface intact</td>
<td>Hemiarthroplasty or bypass central column</td>
<td>SC fusion (STT fusion)</td>
<td></td>
</tr>
<tr>
<td>IIB, IIIC</td>
<td>B, C</td>
<td>3</td>
<td>Only radial column intact</td>
<td>Bypass central column</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV, (KDAC)</td>
<td>B, C</td>
<td>4</td>
<td>Pan - OA</td>
<td>Salvage</td>
<td>Total wrist fusion or arthroplasty</td>
<td></td>
</tr>
<tr>
<td><strong>Wrist Salvage</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KDAC, Kienböck disease advanced collapse; MFC, medial femoral condyle; PA, osteoarthritis; PRC, proximal row carpectomy; RSL, radioscapohulate fusion; SC, scaphocapitate.</td>
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There are some surgical options that can be considered as part of almost any surgical procedure of the wrist for Kienböck disease. These include synovectomy, STT pinning, neurectomy and radial forage.

The treatment options in parentheses are reported in the literature but are not our preferred option. The adjunctive procedures are techniques that can be used for many cases, but are not specific to any particular stage of the condition. Note: In the teen-bock and the elderly patient, Kienböck disease has a better prognosis. Therefore, these patients are often offered nonsurgical treatment, or joint leveling procedures if they have persistent pain. Radial shortening, ulnar lengthening, and capitate shortening are all osteotomies.

point that intercarpal fusions and proximal row carpectomy are now being performed arthroscopically, and there will continue to be a trend toward a more minimally invasive approach to Kienböck disease.

**PUTTING IT ALL TOGETHER: A NEW(ER) TREATMENT ALGORITHM**

The development of the osseous classification system provided a treatment algorithm based on radiographic findings. With the addition of information on osseous viability gained from advanced MRI techniques and cartilage damage from arthroscopy and MRI, we now have more sophisticated tools to guide decision making. In addition, we now know that the age at presentation is an important prognostic factor and should also be taken into account. In an attempt to demonstrate the synergy of the 3 separate classification systems, Table 3 places them side by side to see how their amalgamation can lead to a more nuanced treatment protocol.

A disease algorithm is good for as long as it can provide the best treatment solutions for a large variety of clinical circumstances. The algorithm presented here will not be the last iteration. Future discoveries regarding the etiology, pathoanatomy, biomechanics, demographics, imaging, natural history, and treatment of Kienböck disease will have to be taken into account. There is obviously much more work to be done before we can all agree on the definitive management of this fascinating disorder. (Two prior publications are worth reading. An evidence-based clinical scenario shows what advancements in arthroscopic diagnostics might now be added to *evidence-based treatment* of specific clinical scenarios. A *current concepts* article is an excellent review and also shows how our treatment algorithms are evolving.

**REFERENCES**


EDITOR’S SUGGESTIONS FOR MORE INFORMATION


