Kienböck Disease: A New Algorithm for the 21st Century

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Abstract

Background It has been over 100 years since the initial description of avascular necrosis of the lunate. Over the last two decades, there has been the introduction of advanced information regarding the etiology, natural history, classification, and treatment options for lunate osteonecrosis. There have been new classifications developed based on advanced imaging, perfusion studies of lunate viability, and arthroscopic assessment of the articular cartilage.

Purpose This article brings together a new treatment algorithm, incorporating the traditional osseous classification system (Lichtman) with the perfusion/viability classification (Schmitt) and the articular cartilage classification (Bain).

Methods We have developed a new algorithm to manage Kienböck avascular necrosis of the lunate. This new algorithm incorporates the current concepts of the diseased lunate and its effects on the remainder of the wrist.

Conclusion For patients with a good prognosis and in the earliest stages, the “intact lunate” is initially protected utilizing nonoperative measures. If this fails, then appropriate lunate unloading procedures should be considered. If the lunate is “compromised” then it can be reconstructed with a medial femoral condyle graft or proximal row carpectomy (PRC). With the further collapse of the lunate, the wrist is then also compromised, with the development of secondary degeneration of the central column articulation. The “compromised wrist” will have functional articulations, which allows motion-preserving procedures to be utilized to maintain a functional wrist. With advanced disease (Kienböck disease advanced collapse), the wrist is not reconstructable, so only a salvage procedure can be performed. Other than these objective pathoanatomical factors, the final decision must accommodate the various patient factors (e.g., age, general health, lifestyle, financial constraints, and future demands on the wrist) and surgeon factors (skill set, equipment, and work environment).

Keywords
- Kienböck
- lunate
- osteonecrosis
- avascular
- classification
The knowledge of Kienböck disease has evolved over more than 100 years since Robert Kienböck published his article on osteomalacia of the lunate in 1910. The purpose of this article is to amalgamate much of this information, particularly the newer advances in diagnosis and treatment, into a practical, yet highly granular algorithm for the management of this disorder. The natural history of Kienböck disease has been reported in terms of the progression of clinical findings and serial changes on standard X-rays. The Lichtman classification (1977) identifies these changes. The clinical presentation of pediatric Kienböck disease is similar to the adults, with dorsal tenderness, swelling, reduced wrist motion, and grip strength. However, the prognosis is better in the pediatric and elderly patients, than the typical 20- to 40-year-old patient.

Irisarri et al divided pediatric Kienböck disease into infantile (12 years and younger) and juvenile (13 years to skeletal maturity). All patients in the infantile group were treated nonoperatively, with excellent outcomes, including lunate revascularization on magnetic resonance imaging (MRI). The juvenile group was also treated with immobilization, however, 30% had progression and required a joint leveling procedure. Irisarri et al recommended immobilization for patients under 15 years, and joint leveling procedures in the older patient, if there was a disease progression despite immobilization.

In the elderly patient, Kienböck disease behaves differently in comparison to the pediatric and adult cohorts. In Kienböck patients older than 70 years, there is less negative ulnar variance, a higher prevalence in women, who had more advanced radiological changes. At a mean follow-up of 5.6 years, all 15 patients progressed to stage IV disease but had good-to-excellent clinical outcomes without surgery.

**Clinical Assessment**

**Plain Radiographs**

Plain radiographs have been used to diagnose and follow the progression of Kienböck disease, using the osseous Lichtman classification for almost 40 years. It has evolved with time, and now includes stages 0 and IIIC.

**Magnetic Resonance Imaging**

Recently, both T1-weighted fast spin-echo (FSE) and T2-weighted FSE fat-saturated MR images have been used to evaluate viability of bone marrow.

T1-weighted FSE: Normal marrow will have a high signal with T1 due to the fat in the marrow. The T1 signal will be of low intensity if the fat is replaced with edema, neovascularization, space-occupying lesions, bone necrosis, or sequestrum.

T2-weighted FSE: Normal marrow will have low signal T2, due to its low water content. Ischemic marrow is edematous, so has a high T2 signal. Necrotic marrow has a low signal as it has low water content.

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**Fig. 1** The stages of the Lichtman osseous classification of Kienbock disease.
Schmitt et al demonstrated how gadolinium perfusion enhanced T1-weighted FSE fat-saturated sequences so that they are now seen as a high signal. This distinguished low-signal edema from the enhanced high signal of neovascular repair tissue. He identified three zones or patterns of enhancement of the necrotic lunate: The proximal necrotic lunate without enhancement, intermediate hypervascular repair zone, and normal distal lunate. The hyperenhancement reparative zone indicates a good-healing prognosis, and low signal indicates a poor prognosis due to the nonviable marrow.

Schmitt et al classified the lunate signal changes after administration of intravenous gadolinium contrast (Fig. 2).

1. **MRI stage N—Normal**: Normal lunate signal with no enhancement.
2. **MRI stage A—Ischemic (viability maintained)**: Proximal lunate is edematous but well perfused (enhancement with gadolinium).
3. **MRI stage B—Partially necrotic (viability partially lost)**: Proximal necrotic lunate (no enhancement), with an adjacent reparative zone with enhancement, and viable distal lunate.
4. **MRI stage C—Completely necrotic**: Total necrosis, no enhancement corresponding to complete lunate osteonecrosis.

### Arthroscopic Assessment of Kienböck Disease

In 2006, Bain et al reported an arthroscopic assessment and classification of Kienböck disease, which is based on the number of nonfunctional articular surfaces of the lunate (Fig. 3). A functional articular surface has a normal smooth arthroscopic appearance and is firm to palpation without significant softening.

A nonfunctional articular surface has at least one of the following: extensive fibrillation, fissuring, localized, or extensive loss, a floating articular surface or fracture. Synovitis was

![Fig. 2 Schmitt schematic diagram of bone-marrow viability in Gadolinium-enhanced MRI normal marrow (viability unaffected). MRI, magnetic resonance imaging.](image-url)
identified in all of our cases; therefore, it was not used for grading.

He reported that plain radiographs often underestimate the severity of articular changes, and arthroscopic findings commonly change the recommended treatment. Also, 82% of cases had at least one nonfunctional articulation, and 61% had at least two nonfunctional articulations. From these findings, an articular-based approach to treatment was developed based on the functional articular surfaces. The nonfunctional (compromised) articulations are identified and either excised, fused, or bypassed. The wrist is then mobilized with the remaining functional articulations.

A New Treatment Algorithm

Having reviewed these important classifications, we have put together a new treatment algorithm. There are several important concepts that determine the outcome of the patient and need to be considered.

In demonstrating the synergy of the three classifications, we have placed them side-by-side in • Table 1.

The new classification system extends these concepts further: It respects the importance of the patient’s age, the lunate revascularization potential, the pathoanatomical aspects of the lunate disease and the secondary effects that the disorder has had on the wrist. The treatment recommendations are based on these factors.

The five questions underpin the management of Kienböck disease. The first three questions are the essential components of the new combined classification and the treatment recommendations (• Fig. 4). The last two concern the capabilities of the surgeon and the needs of the patient and assist in guiding the final decision.

Patient’s Age

The age at presentation is important, with “Teenbock” and elderly patients having a better prognosis. As these are different prognostic groups, we have separated them from the start. The treatment recommendations for these populations are:

A1 < 15 years: Treat nonoperatively. Consider minimally invasive procedure for symptoms > 6 months.

A2 16–20 years: Trial of nonoperative management. Consider unloading procedure for symptoms > 3 months.

A3 > 70 years: Usually responds to nonoperative management. Consider synovectomy or proceed according to the protocol described below for symptoms > 6 months.

For patients, 21 to 69 years, proceed to sections B or C, as appropriate.

Lunate Stage: How Does the Disease Affect the Lunate?

The lunate consists of osseous, vascular, and cartilaginous components, which are each affected in different ways. (Osseous—Lichtman, vascular—Schmitt, cartilage—Bain).

The lunate is “intact” if it is not fractured and the articular surfaces are “functional.” Lichtman stages 0, 1, and II, Schmitt stage A, and Bain grade 0 all represent an intact lunate. A “compromised” lunate has localized areas of collapse or degeneration, but other areas that can be used to reconstruct and maintain a functional lunate. This coincides with the Lichtman stage IIIA, Schmitt stage B, and Bain grade 1. A lunate is “not reconstructable” if it is fragmented, collapsed, or has completely lost its vascular supply. (e.g., Lichtman stage IIIC, Schmitt stage C, and Bain grade 2b).

Lunate Intact—Lunate Protection

By definition, the lunate that has not collapsed (Lichtman stage 0, I, and II), and has functional articular surfaces (Bain 0), with a positive prognosis for revascularization (Schmitt stage A). The three aims of treatment are:

1. Decrease the patient’s pain and improve function.
2. Protect the vulnerable lunate before collapses.
3. Set the stage for spontaneous revascularization.
Nonoperative Management
In the earliest stages, patients are managed nonoperatively for at least 3 months (e.g., short arm cast or splint), however, minimally invasive techniques may be appropriate as well.
Patients are advised to avoid strenuous activities or lifting over 5 kg (10 lb). Medical causes of osteonecrosis should be treated. If the patient is symptomatic after 3 months, or imaging demonstrates disease progression, a lunate unloading or revascularization procedure is considered.

Lunate Unloading Procedures
Lunate unloading is often used for patients with intact lunates who fail nonoperative treatment. They protect the lunate and set the stage for spontaneous revascularization. It is usually a radial shortening osteotomy, which is effective for the patient with a negative ulnar variance and/or abnormal radial inclination. If the physis is still intact, a radial epiphysiodesis is an alternative. If there is a neutral or positive ulnar variance, a capitate shortening osteotomy can be performed.

Lunate Decompression
Lunate forage is another option, which involves drilling the lunate to decompress venous hypertension. It can be performed arthroscopically, in conjunction with a synovectomy and cancellous bone grafting.

Revascularization Procedures
Core decompression of the distal radius provides an indirect revascularization of the lunate, due to the increased regional vascularity. Direct revascularization of the lunate can be performed, with either a pedicle graft or free vascularized bone graft. Pedicle graft procedures are appropriate for patients with an intact, partially viable lunate (Schmitt grade B). It is also used as an alternative to unloading in patients with a positive ulnar variance.

Lunate Compromised: Lunate Reconstruction (Lichtman Stage IIIA, Schmitt Stage B, and Bain Grade 1)
The lunate has localized disease, with areas of nonfunctioning proximal articular cartilage, but other parts are intact. The ideal treatment would be localized lunate reconstruction.

Vascularized Medial Femoral Trochlea Graft
This is indicated for reconstruction of the proximal lunate, and also to restore carpal height. This is a demanding procedure, so other treatment options are required. These include lunate...
salvage (PRC or lunate replacement), radioscapholunate (RSL) fusion, or scaphocapitate fusion. Note these alternatives are more destructive than an isolated lunate reconstruction.29,30

Lunate Not Reconstructable: Lunate Salvage (Lichtman Stage III C, Schmitt Stage C, and Bain Grade 2b)
The lunate is not reconstructable if the lunate has collapsed, has no revascularization potential or the lunate articular surfaces are nonfunctional. Then a salvage procedure of the lunate is required. Once the lunate has collapsed, it is common for the adjacent articulations to be degenerated and the carpus to collapse. In this case proceed, to the section C of the algorithm.

Lunate Replacement
If the lunate is not reconstructable, then it needs to be excised. The lunate has been replaced with silicone,6,31 autogenous tendon, metallic spheres, and the pisiform,32 but often with poor results.32 If the capitate and lunate fossa articular surfaces are functional, then a lunate excision and capitate lengthening with interposition graft is a possibility. Recently, pyrocarbon implant replacement has been advocated. Unfortunately, instability is an issue, and modifications are required to achieve reliable clinical outcomes.33,34

Proximal Row Carpectomy
Proximal row carpectomy is a time tested technique that is indicated for the unreconstructable lunate if the articular surfaces of the lunate facet and the capitate are functional (Bain grade 2b).

Wrist Stage: How Does the Disease Affect the Wrist?
The secondary effects of the collapsing lunate are a “compromised” wrist, which include:

### A. Patient’s age?
- **A1. < 15 years** – Non-operative
- **A2. 16 - 20 years** – Non-operative first. Consider unloading procedure.
- **A3. > 70 years** – Non-operative first. Consider synovectomy and/or follow algorithm below.

### B. Stage of the lunate?
- **B1. Lunate intact** (Cortex and cartilage intact – Lichtman 0, I, II, Schmitt A, Bain 0)
  - Protect / unload the lunate:
    - Orthosis or cast first (trial for 2–3 months)
    - Radial shortening osteotomy, capitate shortening for ulnar +ve (radial epiphysiodesis*)
  - (Alternatives – Lunate decompression, vascularized bone graft*, radius forage*)
  - Lunate reconstruction: MFT*, lunate replacement*, PRC (RSL fusion, SC [or STT] fusion)
- **B3. Lunate not reconstructable** (Advanced lunate disease – Lichtman III C, Schmitt C, Bain 2b)
  - Lunate salvage (excision): Lunate replacement*, capitate lengthening, PRC (SC fusion)

### C. State of the wrist?
- **C1. Central column articulations compromised** (Lichtman III A or C, Schmitt B, Bain 2a, 3, or 4)
  - **C1a. Radiolunate articulation compromised** (Lichtman III A, Schmitt B, Bain 2a)
    - Fuse or Bypass radiolunate joint: RSL fusion, SC fusion, Lunate Prosthesis, MFT graft
  - **C1b. Radiolunate and midcarpal articulations compromised** (Lichtman III A or C, Schmitt B, Bain 3 or 4)
    - Bypass central column: SC fusion
- **C2. Carpal collapse with intact radioscaphoid articulation** (Lichtman III B or C, Schmitt B, Bain 2 - 4)
  - Stabilize radial column: SC fusion
- **C3. Wrist not reconstructable** (Advanced wrist disease – Lichtman IV, Schmitt C, Bain 4)
  - Wrist salvage: Total wrist arthrodesis, total wrist arthroplasty*
1. Degeneration of the central column at the radiolunate and midcarpal articulations.
2. The collapse of the central column.
3. Proximal row instability and radial column collapse.
4. Finally, degeneration of the radial column.

Fractures of the lunate produce irregularity of the lunate articular surfaces. If the fracture heals in a stable configuration then the prognosis is good. However, with secondary “kissing lesions” of the lunate facet and capitate, there is likely to be degeneration of the central column will occur. With fracture propagation, there will be a loss of lunate height, which affects the kinematics of the perilunate ligaments and the central column. Commination, disruption of the spanning trabeculi, and/or a coronal lunate fracture produces the collapse of the lunate, which allows proximal migration of the capitate, and then the collapse of the entire central column. Scapholunate ligament laxity contributes to proximal migration and collapse of the central column.

With lunate comminution and interposition of the capitate between the lunate fragments, it appears on sagittal X-rays as a widening of the entire lunate. If the major fragments are pushed dorsally, the lunate will appear to be in flexion (volar intercalated segment instability). A dorsal intercalated segment instability deformity occurs if the capitate migrates into a gap due to scapholunate ligament instability or if the main fragments are pushed anteriorly. In each case, the proximal migration of the capitate (and distal row) cause excessive flexion of the scaphoid (Lichtman stage IIIB), as manifested by volar subluxation of the scaphoid articulation often remains functional, so a scaphocapitate fusion can be performed. A STT fusion is an alternative.

Prior to carpal collapse, the wrist with nonfunctional surfaces can be reconstructed with motion preserving procedures by excising, fusing, or bypassing the nonfunctional articulations and maintaining the functional articulations. The recommended procedure will depend upon the extent of osseous collapse, but more importantly the remaining functional articular surfaces. The surgeon matches the remaining functional articular surfaces with the prerequisites.

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primary treatment in the “Teenbock” patient and to protect the lunate in conjunction with a vascularized bone graft.\textsuperscript{37} Some authors recommend posterior interosseous neurectomy for relief for carpal pathology.\textsuperscript{42} However, we are concerned that it will negatively affect function and protection of the wrist.\textsuperscript{36}

What Can the Surgeon Offer?

Surgeons have different abilities, training, and experience. Some surgical options require advanced skills in microsurgery or arthroscopy, which are beyond the capabilities of some surgeons or their facilities. We have identified these procedures within the algorithm. With technical advances, some of these procedures will become more mainstream.

What Does the Patient Want?

The final decision comes down to the needs and desires of the informed, competent patient. The patient’s health, lifestyle, and demands on the wrist need careful consideration. Each patient is different, and their personal considerations will affect all levels of the proposed algorithm. Unfortunately, constraints outside of the control of the doctor and patient may affect the final decision (e.g., financial restrictions, the insurer, governing bodies).

No treatment algorithm will cover all contingency, especially when addressing disease condition such as Kienböck disease. However, when armed with this information, we believe the clinician can make an evidence-based decision that is tailored to the unique needs of the patient.

Conflict of Interest

None.

References


