Kienböck disease is a disorder of undetermined etiology that results in osteonecrosis of the lunate. It most commonly affects the dominant hand in men aged 20 to 40 years. The natural history of the disorder is uncertain. Treatment ranges from immobilization to revascularization of the lunate, but no single treatment has been proven superior. In many cases, the radiographic results do not correlate with the clinical outcome.

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In 1910, Robert Kienböck, a radiologist in Vienna, described the radiographic and clinical characteristics of a disease he termed “lunatomalacia.” He believed that the underlying cause of this condition was rupture of the ligaments and vessels surrounding the lunate, which resulted in fracture and subsequent collapse. Although this condition would eventually bear his name, the initial description of lunate collapse secondary to suspected trauma was presented in 1843 by Peste. The treatment of Kienböck disease was significantly advanced by Hultén, who in 1928 noted an association between the disease and the presence of negative ulnar variance. He was the first to present radial shortening as a possible form of treatment, and this was followed shortly thereafter by Persson, who advocated lengthening the ulna. Several decades later, intercarpal fusions and revascularization procedures were introduced for the treatment of Kienböck disease. The myriad number of treatments that have been described for this disorder is evidence that we are far from solving its riddle. Our treatment is hampered because a clearly defined etiology and natural history have not been determined.

Etiology

Although the underlying etiology of Kienböck disease is still unclear, it is generally agreed that the endpoint of fragmentation and collapse is secondary to osteonecrosis. Both extrinsic and intrinsic factors have been offered as causes of the disease. Extrinsic factors include the relationship between the radius of curvature of the capitate head and distal lunate, concentration of axial loads preferentially through the lunate, and ulnar variance; intrinsic factors include the vascular supply and trabecular arrangement of the lunate.

The radius of curvature of the capitate is less than that of its articulating surface with the lunate. Compressive forces are therefore concentrated on the distal articular surface of the lunate. Late in the disease, as the capitate migrates proximally, it can act as a wedge, splitting the lunate into dorsal and volar portions.

Conventional wisdom has suggested that repetitive microtrauma plays a role in the disease, but there are no specific data to support this contention. The absence of a single traumatic event is common. Curiously, Kienböck disease is not seen after perilunate
dislocations. Although early signs of osteonecrosis such as increased radiodensity are often seen, progression to collapse has not been reported.

Much has been written about the relationship between Kienböck disease and negative ulnar variance. Hultén found that 23% of the general population have negative ulnar variance, whereas 74% of his patients with Kienböck disease were ulnar negative. Although a causal relationship cannot be proven, a statistical relationship appears evident. The ulnar-negative variant produces a situation in which loading across the radiocarpal joint is not shared normally between the ulna and radius. The lunate bears the brunt of this abnormal loading. The triangular fibrocartilage complex is thicker in these patients, and the difference in resistance to loading between it and the ulnar edge of the radius is accentuated.

The vascular supply of the lunate has been extensively studied. Most lunates have a dual blood supply with both dorsal and palmar inflow and a rich anastomotic network. Given this finding, it is hard to ascribe the osteonecrosis to a major vessel disruption in most cases. Increased intraosseous pressure has been shown in femoral heads with osteonecrosis, and this finding has been duplicated in lunates with Kienböck disease. This suggests that impairment of venous drainage may contribute to bone necrosis.

**Natural History**

The natural history of Kienböck disease is not well understood because there are few studies that focus primarily on nonoperative treatment. Kristensen et al reviewed 49 patients who were managed nonoperatively with an average follow-up time of 20.5 years. They found that 80% of these patients had no pain or experienced pain only with hard work. The majority reported a gradual decrease in symptoms over time, and Kristensen described this as a benign course. The radiographic course of the disease, however, did not mirror the clinical course. All 49 lunates were deformed, and two thirds of patients showed evidence of osteoarthritis in the radiocarpal or carpal joints. Mikkelson and Gelineck also found that 80% of their 25 patients, followed up for 8 years, had no pain or experienced pain only with hard work. However, their conclusion was that nonoperative treatment is unsuccessful. Nearly 60% of the patients had daily problems with their wrists, and 7 required a change in occupation.

**Diagnosis**

The most common clinical presentation is a man aged 20 to 40 years who complains of intermittent wrist pain, decreased wrist motion, and a weak grip in the dominant hand. Many of the patients are manual laborers. There may be a long history of symptoms before presentation. Usually, the pain is activity related and abates with rest. A history of trauma is variable and may be in the distant past. The wrist may be swollen and tender to palpation over the radiocarpal joint. Motion is predictably diminished in the flexion/extension arc, and grip strength is commonly decreased to 50% of the unaffected side.

Evaluation of plain radiographs forms the basis for the staging and treatment of patients with Kienböck disease. Lichtman et al modified Stahl’s classification of Kienböck disease by dividing the disease into 4 stages (Table 1, Figs 1A-D). Initially, the lunate appears to be more radiodense, which is consistent with the radiologic appearance of osteonecrosis. A coronal fracture that splits the lunate into dorsal and volar fragments may be noted. If the lunate does not revascularize, the fractures will not heal and the lunate will collapse. This is especially evident on the lateral view, where the capitate may be imagined to be the pestle in the mortar of the lunate, splitting it in half. Scaphoid flexion occurs secondary to the proximal descent of the capitate into the area of lunate collapse. Finally, the altered carpal mechanics result in radiographically visible degenerative disease around the lunate.

Other imaging modalities can be helpful. Bone scintigraphy may be useful to exclude Kienböck dis-

<table>
<thead>
<tr>
<th>Stage</th>
<th>Radiographic Findings</th>
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<tbody>
<tr>
<td>I</td>
<td>Normal (MRI shows decreased signal intensity on T1 image)</td>
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<tr>
<td>II</td>
<td>Increased radiodensity, possible mild decrease in lunate bone height on radial side</td>
</tr>
<tr>
<td>IIIA</td>
<td>Lunate bone collapse, no scaphoid bone rotation</td>
</tr>
<tr>
<td>IIIB</td>
<td>Lunate bone collapse, fixed scaphoid bone flexion</td>
</tr>
<tr>
<td>IV</td>
<td>Degenerative changes around the lunate bone</td>
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ease, but is not sensitive enough to eliminate other more common causes of wrist pain. It may be helpful in patients with known disease in one wrist who develop mild symptoms on the contralateral side. In a patient whose diagnosis is unclear, the examination most commonly used is magnetic resonance imaging (MRI). This is an extremely sensitive test for detecting changes in marrow fat that are consistent with, but not diagnostic of, osteonecrosis. On the T1-weighted image, a lunate with Kienböck disease will show low to medium signal intensity instead of high signal intensity (Fig 2). This decreased signal may occupy the entire bone or only focal portions. MRI provides a method for diagnosing this disorder in its earliest stage, giving the opportunity for early intervention. High-quality tomograms have been shown to enhance the staging of Kienböck disease by better showing early collapse, thus upgrading many stage II patients to stage III.

**TREATMENT**

Treatment is based on the stage of disease and the measurement of ulnar variance. There is no agreement on the optimal way to treat Kienböck disease. The options range from immobilization to revascularization procedures. Although the natural history of progressive collapse leading to perilunate arthritis is unquestioned, these changes do not universally lead to a poor clinical outcome. Although comparison of techniques is difficult because most studies have small numbers and inadequate follow-up periods, the results

**FIGURE 1A.** Stage II disease.
do not vary significantly between the options for treatment.

**Immobilization.** Several researchers, in long-term follow-ups of patients treated conservatively versus those treated surgically, noted no significant difference in pain and function between the 2 groups.\(^{14,19}\) Caution should be used in interpreting these results, because the patients were not randomized and surgery was reserved for patients who failed nonoperative care.

**Revascularization.** The intuitive answer for the treatment of avascular necrosis is to provide a new source of arterial blood. This is a well documented approach to femoral head osteonecrosis, and it has been used for Kienböck disease as well. In 1979, Hori et al.\(^{20}\) presented the initial results of a novel revascularization technique that implanted a dorsal metacarpal vascular pedicle directly into the lunate. Results of longer-term follow-up studies of this technique\(^{21}\) showed disappearance or reduction of pain in 50 of 51 patients and increased strength in all 51. These improvements in clinical symptoms were not necessarily associated with radiographic improvement, however, and increased degenerative changes were noted in almost 20% and fragmentation in nearly 10% of patients. In a number of these cases, additional procedures such as bone grafting, scaphotrapeziotrapezoidal (STT) fusion, or external fixation were performed as an adjunct. Newer methods for providing a blood supply use vascularized bone pedicles that can be rotated and implanted into the lunate. Sheetz et al.\(^{22}\) elegantly showed the blood supply of the distal radius and ulna and identified several vascular pedicles that provide a wide arc of rotation and can be harvested with relative ease (Figs 3 and 4). As with other bones affected with osteonecrosis, there is a concern that

**FIGURE 1B.** Stage IIIA disease. (Courtesy Joseph Imbriglia, MD.)
collapse may follow revascularization unless additional support is provided to decrease the load across the lunate during the healing phase. This has been accomplished with the use of external fixation spanning the wrist.

Biomechanical studies on various options of treatment include both cadaver and computer modeling experiments. Iwasaki et al, in a 3-dimensional modeling study of intercarpal fusions, found that although both scaphocapitate (SC) and STT fusions significantly reduced the load across the lunate, this load was preferentially shifted to the unfused articulations around the scaphoid, including the radioscaphoid joint. A cadaveric experiment comparing intercarpal fusions with joint-leveling procedures noted a 70% strain reduction at the lunate with STT fusion, ulnar lengthening, and radial shortening. Only the STT fusion, however, resulted in motion loss. Ninety percent of the strain reduction after joint-leveling procedures occurs within the first 2 mm of shortening or lengthening. Peak pressure at the lunate, both in the midcarpal and radiocarpal joints, is reduced with a radial closing-wedge osteotomy as well. Kinematic studies comparing the SC and STT fusions in a cadaver model showed no superiority in motion of one over the other. They did note that although the range of motion lost was modest, the kinematic relationships between the scaphoid and lunate are dramatically altered. Capitohamate (CH) fusion without shortening was unsuccessful in reducing load at the lunate in both studies.

Joint-leveling procedures. The most commonly used procedures for the treatment of Kienböck disease

FIGURE 1C. Stage IIIB disease. Note scaphoid signet ring sign on AP film indicating scaphoid flexion. (Courtesy Joseph Imbriglia, MD.)
have focused on leveling the distal radioulnar joint. Both radial shortening and ulnar lengthening have been described. The goal in either procedure is to obtain approximately neutral ulnar variance. In 2 of the largest studies of radial shortening,\textsuperscript{28,29} approximately 90% of the patients had decreased pain (Figs 5 and 6). Improvement in wrist motion and grip strength were also noted at 3- to 4-year follow-up examinations. In both studies, there was a low rate of complication when the osteotomy was performed with a 3.5-mm dynamic compression plate. The radiographic appearance of the lunate, however, showed no significant improvement despite a good clinical outcome. This has also been supported in a study of patients who had undergone radial osteotomy, in whom follow-up wrist arthroscopy showed progression of degenerative changes around the lunate in two thirds of the cases, despite clinical outcomes.\textsuperscript{30} In ulnar-neutral or ulnar-positive patients, successful outcomes have also been obtained by performing lateral radial closing-wedge osteotomies to decrease radial inclination.\textsuperscript{31} Ulnar lengthening with intercalary

**FIGURE 1D.** Stage IV disease.
bone graft and plate fixation has also resulted in pain relief and improvements in grip strength and range of motion. \textsuperscript{32} Studies comparing the 2 leveling techniques have shown essentially equivalent clinical outcomes, but more nonunions and delayed unions result from ulnar lengthening. \textsuperscript{33,34} The rates of nonunion and delayed union for ulnar lengthening and radial shortening range from 8\% to 19\% and 0\% to 6\%, respectively. \textsuperscript{28,32-34}

**Intercarpal procedures.** Intercarpal fusions are used to treat a variety of conditions in the wrist. They were applied to Kienböck disease for their ability to control carpal malalignment and decrease the load experienced by the lunate. They may be more useful in ulnar-neutral or ulnar-positive patients who are not amenable to joint leveling. Several different variations have been used for stage II and stage III disease.

**STT fusion.** As the lunate collapses into stage III disease, the capitate migrates proximally and the
scaphoid begins to palmar flex. STT fusion was applied to this disorder because of concern that this progressive rotatory subluxation of the scaphoid would lead to continued pain and degenerative changes (Fig 7). Watson et al\(^3\) reviewed their experience with 28 STT fusions for Kienböck disease at an average follow-up time of 51 months and found that 78% of patients had good or excellent pain relief. Range of motion and grip strength were also improved. They emphasized that the scaphoid should be fused at 55° to 60° of flexion, because a position of relative extension compromises motion and accentuates the load across the radioscapoid joint. Unfortunately, 43% of these patients required additional operative procedures (lunate excision, proximal row carpectomy, or wrist arthrodesis) to obtain this result. Watson believes a collapsed lunate is a contraindication to a joint-leveling procedure because it fails to address scaphoid rotation. This concern has not been supported in the radial shortening literature, however, as 20 of 28 patients in Weiss’s study\(^2\) were in stage III (16 stage IIIA, 4 stage IIIB).

**SC fusion.** SC fusion in Kienböck disease\(^6,7\) provided good pain relief but somewhat less motion as compared with STT fusion. The advantage of this fusion is that it is technically easier to perform and requires only 1 fusion site (Fig 8).

**Capitate shortening with CH fusion.** Originally described as a CH fusion alone,\(^3\) this variant has now been modified to include capitate shortening.\(^5\) This approach reduces load on the lunate by prevent-
ing the proximal migration of the capitate. There are no long-term studies to document the success of this technique (Fig 9).

**Salvage procedures.** Proximal row carpectomy (PRC) and wrist fusion are salvage procedures that are reserved for advanced stages of Kienböck disease and failures with other treatments. A number of researchers\(^{40-42}\) have reported relatively good results when PRC was used to treat various conditions including Kienböck disease. One can expect to achieve approximately 75\(^\circ\) of motion in the flexion/extension arc and a grip strength of 75\% of the unaffected side. Pain relief has been achieved in more than 80\% of the patients. These results were confirmed in a study that specifically addressed PRC in patients with stage III Kienböck disease.\(^{43}\) The obvious advantage of PRC over arthrodesis is preservation of motion. Some researchers\(^{40,41}\) also recommend the addition of a radial styloidectomy if impingement of the triquetrum on the radial styloid in radial deviation is noted intraoperatively. A common dilemma is determining whether the articular damage to the capitate head or lunate fossa is too severe to perform a PRC. The appearance may range from a small partial-thickness defect to complete eburnation. The maximal amount of damage with which a PRC can be successfully performed is unknown, and there are no good data to aid in this...
decision. Imbriglia et al\textsuperscript{40} recommend adding a capsular imbrication if there is full-thickness damage less than 3 mm in size. Larger lesions may necessitate wrist fusion. A failed PRC can successfully be converted to arthrodesis.

**Other options.** Despite encouraging early results, silicone implants into the lunate space\textsuperscript{15} were not effective because the implants were found to deteriorate secondary to particulate synovitis.\textsuperscript{44} These implants can no longer be considered a primary treatment modality. Zelouf and Ruby\textsuperscript{45} performed cancellous bone grafting with external fixation and found that it provided good pain relief in 80\% of patients and contributed to improvement in both grip strength and range of motion. They also detected evidence of revascularization on postoperative MRI scans in a third of their patients. Lunate excision has generally been avoided because of concerns about subsequent carpal collapse. The addition of a fascial interposition graft after lunate excision has been advocated and has yielded good long-term results in a small series.\textsuperscript{46} Arthroscopic débridement in patients with stage II disease was successful in reducing pain, increasing range of motion, and providing complete relief of mechanical symptoms.\textsuperscript{47} Although this study had a small number of patients and a relatively short follow-up period, it does show that this simple procedure may provide relief of symptoms. Finally, in the European community, wrist denervation has been advocated both as an adjunct and as a primary procedure.\textsuperscript{48}
TREATMENT BY STAGE

Because there is no literature that discusses the management of stage I disease, it seems prudent to begin with immobilization in treating these cases. Most patients, however, will not present this early in the disease course. Stage II disease with negative ulnar variance can be effectively managed with radial shortening. Vascularized bone grafting is particularly attractive for young patients with stage II or stage IIIA disease who manifest neutral or positive ulnar variance. A lateral closing-wedge osteotomy of the radius is also an option for the patient with an ulnar-positive wrist. Stage IIIA disease with negative ulnar variance can also be effectively treated with radial shortening, and although good results have been obtained in a few patients with stage IIIB disease, many surgeons now favor PRC. Stage IV wrists can also be managed with PRC, provided the damage to the capitale head or lunar fossa is not too severe. The exact role for capsular interposition in addition to PRC is unknown. Total wrist arthrodesis is reserved for cases of failed PRC or if significant cartilage damage is noted on the head of the capitale or in the lunar fossa.

CONCLUSION

A number of issues are unresolved with regard to Kienböck disease. The etiology and natural history of the disorder are not clearly defined. This makes it difficult to determine the value of operative intervention. The role of intercarpal fusions, with their requisite loss of motion and transfer of loads to the radioscaploid joint, is debatable. Further work needs to focus on the role of revascularization procedures and whether they provide added benefit over joint-leveling procedures. Radial shortening and proximal row carpectomy remain the mainstays of treatment of this disease.

REFERENCES

22. Sheetz KK, Bishop AT, Berger RA. The arterial blood supply of the distal radius and ulna and its potential use in vascu-


