Kienböck's disease: current concepts

Pablo De Carli, Ezequiel Zaidenberg
*Reconstructive and Hand Surgery Sector, Department of Orthopedics and Traumatology,
Hospital Italiano de Buenos Aires (Buenos Aires, Argentina)*

ABSTRACT
Osteonecrosis of the lunate or Kienböck's disease is a progressive, debilitating condition that can lead to chronic pain and function loss. Despite it was identification almost 100 years ago, the etiology remains unknown, although mechanical, vascular, and traumatic factors have been associated. The natural history of Kienböck's disease is poorly defined, and the radiographic findings do not always correlate with the clinical findings. There has been some progress in identifying and understanding the progression of the avascular process and its deleterious effects on wrist mechanics. Initial treatment is conservative. Improvement in surgical techniques with vascularized bone grafts from the distal radius may lead to an outcome improvement for patients in the early stages of disease. However, more research is still needed to determine whether this surgical treatment represents an improvement over conventional treatment alternatives. Recent reports of long-term outcomes for radial shortening osteotomy in osteonecrosis early stage patients and for proximal row carpectomy (PRC) in advanced Kienböck's disease patients reveal that these procedures provide reliable options for the long-term management of this challenging condition.
Key words: Kienböck's disease

Enfermedad de Kienböck: conceptos actuales

RESUMEN
La osteonecrosis del semilunar o enfermedad de Kienböck es un cuadro progresivo y debilitante que puede llevar al dolor crónico y la pérdida de la función. Pese a que se conoce hace más de 100 años, la etiología todavía es incierta, si bien se la ha relacionado con factores mecánicos, vasculares y traumáticos. Su evolución natural está pobremente definida y los hallazgos radiográficos no siempre se correlacionan con los hallazgos clínicos. Se han logrado avances para identificar y entender la progresión del proceso avascular y sus efectos nocivos sobre la mecánica de la muñeca. El tratamiento inicial es no quirúrgico. El perfeccionamiento de la técnica quirúrgica utilizando injertos óseos vascularizados de radio distal puede mejorar los resultados en los pacientes que cursan etapas tempranas de la enfermedad, aunque resta mucho trabajo para determinar si este tratamiento quirúrgico representa una mejor opción que los tratamientos tradicionales. Reportes recientes de resultados a largo plazo de la osteotomía de acortamiento del radio para estadios tempranos de la enfermedad y la resección de la primera fila del carpo para estadios avanzados revelan que estos procedimientos son una opción válida para el tratamiento a largo plazo de este cuadro de difícil manejo.
Palabras clave: Enfermedad de Kienböck.

INTRODUCTION

Osteonecrosis of the lunate or Kienböck's disease is a progressive, debilitating condition that can lead to chronic pain and function loss. Despite it was identification almost 100 years ago, the etiology remains unknown, although mechanical, vascular, and traumatic factors have been associated. The natural history of Kienböck's disease is poorly defined, and the radiographic findings do not always correlate with the clinical findings. There has been some progress in identifying and understanding the progression of the avascular process and its deleterious effects on wrist mechanics. Initial treatment is conservative. Improvement in surgical techniques with vascular-

Received on July 30th, 2020. Accepted after evaluation on July 31st, 2019 • PABLO DE CARLI, MD • pablo.decarli@gmail.com ID http://orcid.org/0000-0002-9474-8129
How to cite this paper: De Carli P, Zaidenberg E. Kienböck's disease: current concepts. *Rev Asoc Argent Ortop Traumatol* 2020;85(3):26-35. https://doi.org/10.15417/issn.1852-7434.2020.85.4S.1170

ized bone grafts from the distal radius may lead to an outcome improvement for patients in the early stages of disease. However, more research is still needed to determine whether this surgical treatment represents an improvement over conventional treatment alternatives. Recent reports of long-term outcomes for radial shortening osteotomy in osteonecrosis early stage patients and for proximal row carpectomy (PRC) in advanced Kienböck's disease patients reveal that these procedures provide reliable options for the long-term management of this challenging condition.

HISTORICAL OUTLOOK

Robert Kienböck, an Austrian radiologist, was the first to describe the avascular necrosis of the lunate.[1] His article "Concerning Traumatic Malacia of the Lunate and Its Consequences" was published in 1910. The article described the condition as an isolated disease of the lunate that was associated with secondary changes in the other carpal bones. Clinical findings included pain, range of motion (ROM) loss, and and prominence in the wrist area. Radiographic findings included isolated changes in the proximal lunate, with occasional collapse and fragmentation of the bone. Kienböck believed the cause of this condition to be a "disturbance in the nutrition" of the lunate due to repetitive trauma.

In 1928, Hultén noted that 78% of his study group comprised of 23 Swedish patients with Kienböck's disease showed negative ulnar variance while the same parameter had a 23% prevalence in the general population.[2] This study led to the association between ulnar-minus variance and avascular necrosis of the lunate, and the constitution of groups that supported a mechanical etiology of the disease. More recent studies on patients of Asian ethnicity have shown that Kienböck's disease can occur in patients with ulnar neutral and ulnar positive variance.[3]

ETIOLOGY

The lunate is the central bone of the carpal proximal row, and it articulates proximally with the lunate fossa of the radius and the triangular fibrocartilage complex, and distally with the capitate and the hamate bones. The lunate and the lunate fossa of the radius have an hemispherical shape and a high level of congruence between their articular aspects. This anatomical characteristic is believed to play a major role in the joint between the radius and the lunate being one of the last joints to develop degenerative changes,

As the Kienböck's disease is mainly an avascular process, the vascular patterns of the lunate have been studied in depth in order to identify the development of osteonecrosis.[4,5] Several vascular patterns have been identified in cadaveric specimens; however, most nutrient vessels belong to the dorsal and palmar vessels.[5] These vessels enter through the bone non-articular surfaces. In one study, between 7% and 23% of the lunate were supplied by a single dorsal artery.[4,5]

The vessel or vessels that enter the bone may result in several intra-osseous branching patterns, including "I", "X" and "Y" patterns. Lunate with a single vessel or with a limited intra-osseous branching may be at increased risk of osteonecrosis associated with acute or chronic repetitive trauma. A palmar vascular supply may explain why patients with perilunate instability or dislocations rarely develop avascular changes in the lunate, since the capsules volar to the lunate remain intact.[6]

Disruption of venous outflow leading to increased intra-osseous venous pressure may also cause avascular changes and osteonecrosis. *In vitro* intra-osseous pressure measurements of necrotic lunates showed marked increases in pressure when compared to normal lunates. The study describes dense plexus of small venous vessels that were found at the palmar and dorsal periosteal aspect of the bones.[7] The authors suggest the plexus as a potential site of venous outflow disruption, secondary to systemic and local compression factors.

Mechanical factors have also been studied as contributing factors to osteonecrosis development.[8,9]As already mentioned, the height ratio between the radius and the ulna at the distal radioulnar joint and the radiocarpal joint (ulnar variance) has long been believed to play a major role in the development of Kienböck's disease. Other mechanical factors, such as the radial inclination, may also lead to Kienböck's disease, especially in patients with ulnar-neutral and ulnar-positive variance who would not develop an avascular process as a result from an increase of radiolunate pressure due to ulnar-negative variance. Patients with decreased radial inclination are more likely to have a smaller lunate and to develop Kienböck's disease.[8]

There have been also systemic factors associated with lunate osteonecrosis. Conditions associated with hypercoagulability, decreased arterial inflow or increased venous congestion may affect this process.[6] Osteonecrosis has been association with systemic corticosteroid use, sickle cell disease, cerebral palsy and septic embolism, although there is no defined correlation between theses systemic conditions and Kienböck's disease. To this day, over 100 years after Kienböck study, the condition etiology remains to be defined. Although mechanical, anatomic, and systemic mechanisms have all been implicated in the development of the Kienböck's disease process, no specific etiologic mechanism has been identified. Our understanding is that a complex combination of all the above factors is likely to be responsible for the Kienböck's disease process.[6,8,9]

CLINICAL AND RADIOGRAPHIC FINDINGS

Kienböck's disease patients commonly present with wrist pain and weakness, often without history of acute trauma. Symptoms duration before consultation vary, although patients commonly report progressive symptoms over long periods of time. For this reason there is no clear understanding of the natural history of the disease. Pain ranges from mild and occasional to severe and debilitating. Kienböck's disease is most common in young adults (between 20 and 40 years) and occurs infrequently in children, although there have been reported cases. Bilateral Kienböck's disease is rare.

Physical examination reveals swelling and tenderness over the dorsal aspect of the wrist (over the lunate), probably due to synovitis. Typical presentation includes a decreased ROM, with associated loss of flexion and extension; pain at the end of the physiological ROM, especially in extension; and forearm rotation is usually normal. In addition, grip strength may be markedly reduced in comparison to the contralateral wrist.

Kienböck's disease can be diagnosed by radiographic studies. Depending on the disease stage, radiographs may evidence diffuse sclerosis of the lunate, cystic change, fragmentation, articular surface collapse of the lunate, and perilunate arthritic changes. Radiographic findings in the early stages of Kienböck's disease commonly include a fracture in the lateral view; however, it is not clear whether the lunate fracture line represents a primary event or whether it occurs later as a consequence of the structural weakness of the necrotic bone. Radiographic studies also play a key role in establishing the affected wrist anatomical and mechanical characteristics, including ulnar variance, radial inclination, carpal height, radioscaphoid angle, and lunate size.

MRI is a key imaging study, particularly instrumental in the early stages of Kienböck's disease when radiographic imaging is less reliable. On T1-weighted images, bone marrow fat loss results in low signal intensity. Accordingly, T2-weighted sequence images also demonstrate low signal intensity. In order to establish a Kienböck's disease diagnosis, imaging evidence must include diffuse signal changes that involve the entirety of the lunate. Confinement of signal changes within the lunate should lead to considering other differential diagnoses. For example, if signal changes are confined to the proximal ulnar region of lunate, ulnar carpal impaction should be considered. Other studies may also prove instrumental. Before MRI, bone scintigraphy was the next diagnostic study recommended in cases of non-diagnostic initial radiographs. Finally, computed tomography proves useful in assessing articular surface collapse and fractures.

Cases where plain radiographs fail to provide clear evidence of radiocarpal or midcarpal joint involvement may benefit from the use of arthroscopic diagnosis in staging Kienböck's disease. Bain and Begg have developed an arthroscopic staging system based on the number of articular surfaces involved.[10] The system classifies lunate articular surfaces (proximal lunate, distal lunate, lunate facet of the radius) into functional and non-functional, and provides a treatment algorithm based on arthroscopic findings. However, performing a diagnostic arthroscopic procedure should be carefully considered in this setting as plain radiography and other imaging studies commonly provide diagnostic findings when assessing articular surfaces.

STAGES

Kienböck's disease staging is made mainly on radiographic findings and is a critical step in the assessment and management of Kienböck's disease patients as it establishes the different treatment options for each case. There are several staging systems, but the one described by Lichtman *et al*. is the most widely used and has been shown to have adequate reproducibility and reliability.[6]

Stage-1 plain radiographs evidence normal density and articular surfaces of the lunate, while stage-1 MRIs reveal diffuse signal intensity changes. Stage-2 plain radiographs show a diffuse sclerotic appearance of the lunate, and fracture lines may be visible although there is preservation of the articular surface. Stage 3 is the most common stage at initial presentation and is characterized by the collapse of the articular surface of the lunate. This stage is divided into stages 3A and 3B. Stage 3A is characterized by articular collapse with no changes in the carpal alignment and height. Contrarily, stage 3B is defined by lunate articular collapse and its secondary scaphoid rotation, proximal capitate migration, and loss of carpal height. Lichtman later added a new stage, 3C, similar to 3A but with a lateral radiographic view of the lunate evidencing two opposed triangular fragments associated to a coronal fracture. Finally, stage 4 is defined by lunate collapse associated with radiocarpal or midcarpal osteoarthritis (Table 1).

Table 1. Lichtman Classification for Kienböck's Disease

Stage 1	Normal radiographs, signal intensity changes are present on MRI
Stage 2	Radiographs evidence lunate sclerosis; fractures lines may be present
Stage 3	Collapse of the articular surface of the lunate.
Stage 3A	Normal carpal alignment and height
Stage 3B	Scaphoid rotation, proximal capitate migration, and loss of carpal height
Stage 3C	Lunate coronal fracture; lunate divided into two (palmar and dorsal) triangular fragments
Stage 4	Lunate collapse with associated radiocarpal or midcarpal osteoarthritis

Goldfarb *et al*. conducted a study to assess Lichtman classification reliability and found that its interobserver reliability was substantial.[11] However, they also found the differences set for stages 3A and 3B not to be particularly reliable- Therefore, they proposed a new modification the the staging system: patients with a radioscaphoid angle of greater than 60° belonged to stage 3B. This modification resulted in an interobserver reliability increase both for general staging (kappa coefficient from 0.63 to 0.81) and for stage 3A (kappa coefficient from 0.38 to 0.75). The authors concluded that the use of the radioscaphoid angle increases the interobserver reliability of the Lichtman classification and allows for a better distinction between stage 3A and 3B disease.

TREATMENT

Kienböck's disease treatment is based on the patient's symptoms and functional impairment and should observe the previously mentioned staging system. Most longitudinal studies on the conservative treatment for Kienböck's disease have reported a radiographic progression of the disease through the previously mentioned stages.[12-14] Keith *et al*. studied 33 patients treated solely by conservative means.[14] They identified ROM reduction (flexion) and function deterioration, which was evidenced by a reduction in the Disabilities of the Arm, Shoulder and Hand (DASH) scores. Although the radiographic longitudinal study was limited to their patient series, they authors concluded that Kienböck's disease is a progressive condition.

There is no consensus on which approach, whether surgical or conservative, yields better outcomes. Delaere *et al*. compared management with night splinting versus surgery in patients with stage 1, 2, or 3 Kienböck's disease.[15] Their results with immobilization were equivalent to those obtained with surgical treatment. However, these results should be considered with reservations as the average stage in the splinted group was lower than that in the surgery group. Other studies have reported surgical treatment to be superior to conservative management. Salmon *et al*. provided evidence showing that radial shortening osteotomy may be superior to conservative management.

Despite Kienböck's disease progression, many patients, even in advanced stages of the disease, still have adequate wrist function and bearable pain. Hence, conservative therapy using a splint or plaster cast and analgesic or anti-inflammatory drugs may be considered a valid approach for all patients, irrespectively of the disease stage at presentation. Immobilization should be maintained for 3 months.[6] Surgery should only be considered for patients for whom conservative treatment and remain symptomatic.

There are multiple surgical procedures for Kienböck's disease; however, we failed to find enough literature data to establish superiority among surgical options. Despite the plurality of surgical procedures, none has been proven superior to the others. Following the previously mentioned staging system, surgeons opt for different procedures according to the different stages. Ulnar variance, radial inclination and other relevant anatomical variables play a key role in establishing which procedure is adequate for each individual patient (Table 2).

Table 2. Kienböck's disease treatment according to its stage

Stage 1	Cast immobilization for 3 months
Stages 2 and 3A, negative ulnar variance	Radius shortening; ulnar lengthening; capitate shortening
Stages 2 and 3A, positive ulnar variance	Vascularized bone grafting and external fixation; radial-wedge or dome osteotomy; capitate shortening
Stage 3B	Scaphotrapeziotrapezoid fusion; lunate excision; radius shortening; proximal-row carpectomy
Stage 4	Proximal-row carpectomy; wrist fusion; wrist denervation

Stage 1

Splint or plaster cast immobilization remains the first treatment option. External fixation has been used both for immobilization as well as lunate unloading procedures, although this technique data is still limited.[16] Despite there are patients who show improvement with the conservative treatment, most of them are likely to progress to stage 2.[6]

Stages 2 and 3A

Stages 2 and 3A are often considered together in terms of treatment options, with the main goal being lunate revascularization in order to prevent the disease progression. The only exceptions to this pairing are the direct revascularization procedures, which are mainly used during stage 2, where no articular collapse has taken place. However, many authors describe the use of this procedures in patients with some degree of collapse and loss of lunate height, a scenario referred to early 3A stage.

Several revascularization procedures have been described for the treatment during stage 2, such as vascularized grafts of the pisiform bone, vascularized pedicled grafts from the distal radius (including the pronator quadratus), direct implantation of the metacarpal arteries and free vascularized grafts, among others.[17-22] In addition to the vascularized grafts, unloading procedures to decrease mechanical stress on the lunate, such as external fixation, STT or scaphocapitate fusion, are commonly performed.[20,21]

Distal radius vascularized bone grafts have become popular.[19] Several types of grafts have been described based on their location with respect to the wrist extensor compartment and also based on whether the donor artery is located within or outside the extensor compartment. The most used graft for Kienböck's disease is based on the 4+5 extracompartmental artery (ECA). Retrograde flow of the fifth ECA is directed in an antegrade direction into the fourth ECA after ligation of the posterior branch of the anterior interosseous artery. This grafting allows for a longer and larger-diameter pedicle of ulnar location. Moran *et al*. have studied 4+5 ECA bone grafts in a retrospective analysis including 26 patients with a 31-month average follow-up.[22] Most patients experienced pain relief and grip strength improvement. From the 26 patients, 71% evidenced revascularization of the lunate.

Despite the overall success of the procedure, 23% of patients had radiographic evidence of disease progression at last follow-up.

Vascularized grafts of the pisiform bone are performed using the ipsilateral pisiform bone as donor site, along with its blood supply from small dorsal and radial branches of the ulnar artery. The pedicled bone graft is then implanted to the bone defect surgically created in the dorsal surface of the lunate. Daecke *et al*. described this procedure in 23 patients with stage 2 or 3A Kienböck's disease, with a 12-year mean follow-up.[18] Pain and function improvement were recorded in 20 out of the 23 patients. Out of 20 patients with preoperative radiographic studies, 6 (33%) evidenced disease progression.

Free vascularized grafts have also been used in the treatment of Kienböck's disease. Gabl *et al*. studied 18 patients with stage 3 Kienböck's disease that were treated with implantation of a free vascularized corticocancellous iliac bone.[23] The wrist was stabilized using external fixation during the healing period. In a 5-year mean follow-up, the graft became incorporated in the lunate in 16 patients with no carpal collapse. The graft did not integrate and was resorbed in the remaining two patients. In their cohort study, Arora *et al*. reported that after a 10-year follow-up those 16 patients still showed no evidence of Kienböck's disease progression or carpal collapse.

Other treatment options for patients with these Kienböck's disease stages include "joint-leveling" procedures.[6,8,9] These procedures are believe to produce a mechanical unload of the lunate, resulting in revascularization and preventing progression to collapse of the articular surface. Establishing the adequate procedure for each individual patient is primarily based on the height relationship between the radius and the ulna at wrist level.

The goal in negative ulnar variance is to level the distal radioulnar joint to result in neutral to 1-mm positive ulnar variance. This result can be achieved by shortening the radius or lengthening the ulna. Ulnar lengthening requires bone graft and has a higher rate of nonunion, which explains why is not commonly performed.[8,9]

Radius shortening osteotomies are technically easier procedures and have low nonunion rates. Several authors have published on long-term follow-up studies, describing this procedure outcomes. Raven *et al*. studied 12 patients who underwent radius shortening osteotomies, with a 22-year mean follow-up period.[25] One patient had stage 3B Kienböck's disease and the rest were either stage 2 or 3A. Only 3 patients evidenced mild disease progression at last follow-up while most patients experienced excellent outcomes as regards pain relief and function. In a study of similar characteristics, Watanabe *et al*. studied 12 patients who underwent radius shortening osteotomies, with a 21-year mean follow-up period.[26] Most patients had mild wrist pain and good ROM and function, but radiographs revealed that the Kienböck's disease had progressed in 50% of the patients.

Radius shortening osteotomy in stage 3B Kienböck's disease remains controversial. Altay *et al*. compared the procedure effectiveness in stage 3A (n = 13) and 3B (n = 10) patients during a 85-month follow-up period.[27] They found no significant differences between the two groups with regard to pain and ROM between the different stages and concluded that the procedure produces similar clinical outcomes irrespective of the disease stage, despite no radiological healing.

Joint leveling options for patients with neutral or positive ulnar variance include radial closing-wedge osteotomy, radial-dome osteotomy, and capitate shortening osteotomy with or without capitate-hamate fusion.[28] Radial osteotomies are used to decrease radial inclination in an attempt to decrease the contact pressure between the radius and the lunate. Leveling is achieved by increasing the area of contact between the radius and the lunate and decreasing the force transmitted through the radiolunate joint and the lunocapitate joint.[28] Koh *et al*. reported improvements in pain, ROM and function in their 10-year minimum follow-up study.[29] However, osteoarthritic changes were observed in almost 75% of patients at the final follow-up evaluation, suggesting that Kienböck's disease progression did not stopped. The goal of these procedures is to unload the lunate articular surfaces by distributing its load across the adjacent joints. These procedures commonly achieve similar outcomes to those of radial shortening osteotomy, providing good pain relief, motion and function restoration, and potentially stopping or slowing disease progression.

Metaphyseal decompression of the radius is a novel concept that has been described for patients with stages 1 to 3A. Illaramendi *et al*. originally described the procedure in their report on their experience with this procedure in 22 patients with a 10-year mean follow-up.[30] This procedure is performed by decompressing the distal metaphysis of the radius through small cortical windows, using a curette or other blunt instrument to produce cancellous bone impaction against the medullary canal, leaving cortical bone without cancellous bone through the 3cm distal to the radius (Figure 1).

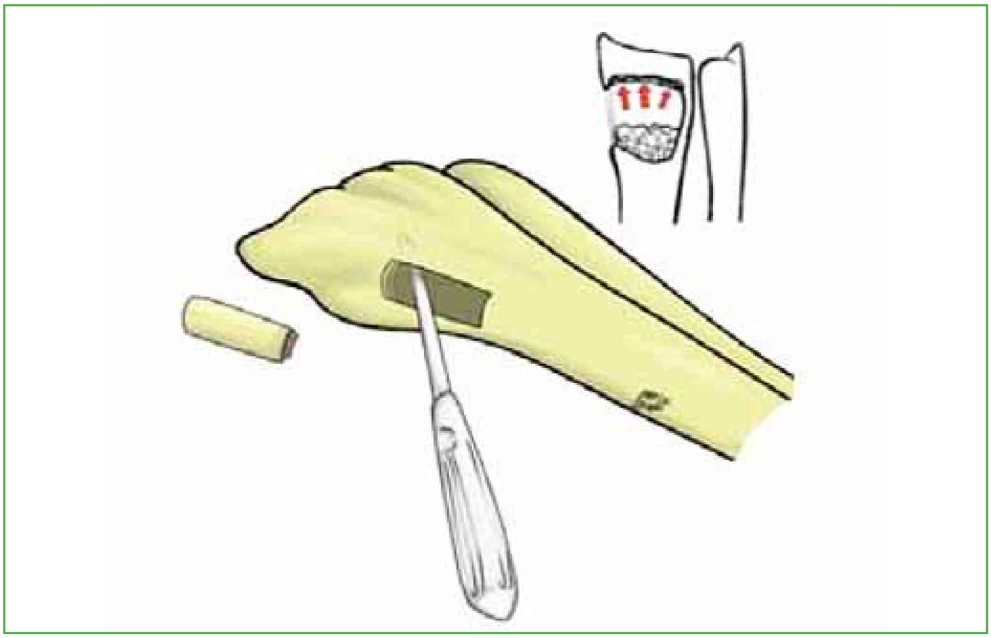

Figure 1. Lateral window for metaphyseal decompression of the radius.

The authors reported good motion restoration and pain relief, with only 4 patients with complaints of mild pain. Most of the patients (17 out of 22) did not show disease progression at last follow-up, 2 showed radiographical improvements and 3 showed radiographical disease progression. In 2017, De Carli *et al*. published a series of 15 stage 3A Kienböck's disease patients, with a 13-year mean follow-up. Based on the Mayo Wrist Score, outcomes were excellent in 6 patients, good in 8 patients, and poor in 1 patient (Figure 2).[31]

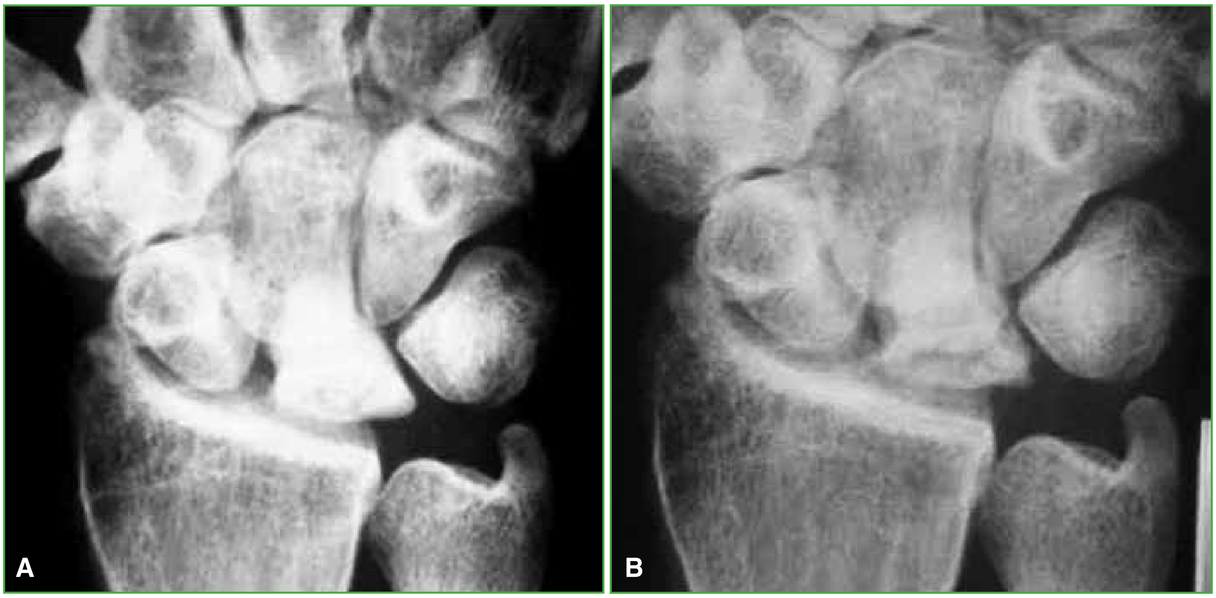

Figure 2. A 45-year old woman with stage 3A Kienböck's disease. **A.** Initial X-rays. **B.** Radiographic control at 12-year follow-up.

Stage 3B

In this stage, there is loss of carpal height due to flexion of the scaphoid as well as articular collapse of the lunate articular surface. Correction of the flexed scaphoid coupled with intercarpal fusion (STT or scaphocapitate) can decrease lunate load, which facilitates midcarpal joint stabilization, and can prevent further carpal collapse. In addition to fusion, the lunate can be removed and replaced with a tendon or a lunate implant to reduce joint irritation. Van den Dungen *et al*. compared the outcomes of patients treated conservatively (n = 19) and with STT fusion (n = 11), with a 13-year mean follow-up period. The authors state that the groups were statistically comparable and found that STT fusion caused an increased loss of mobility and pain and a longer rehabilitation period as compared to conservative treatment.[32] Tambe *et al*. compared outcomes of patients treated with partial intercarpal fusion and radiocarpal fusion for advanced Kienböck's disease, and found that patients treated with radiocarpal fusion had better outcomes at last follow-up.[33]

PRC is a successful salvage procedure, but it requires the capitate head to be free of degenerative changes. In presence of mild arthritic changes are present on the capitate head, an interpositional arthroplasty of dorsal wrist capsule in addition to the PRC may prove effective.[34]

Several studies present detailed long-term PRC outcomes for the treatment of Kienböck's disease. Croog and Stern described a series of 21 patients treated for stages 3 and 4, with a 10-year mean follow-up.[35] Three patients required radiocapitate fusion due to chronic pain, from which 2 had stage 4 disease at the time of the index procedure. The authors concluded PRC to be a reliable procedure in this setting, but advise caution in performing PRC in advanced Kienböck's disease patients. In a similar study, DiDonna *et al*. detailed their experience with CFP in their 10-year minimum follow-up study for patients with degenerative conditions of the wrist, including Kienböck's disease.[36] Again, the authors concluded PRC to be a reliable procedure in this setting, but advise caution in performing PRC in Kienböck's disease patients younger than 35 years of age. De Smet *et al*. reported on 21 patients with advanced Kienböck's disease, with a 67-month mean follow-up period.[37] Lumsden *et al*. report on 13 patients, with a 15-year mean follow-up period.[38] Like the previous authors, these authors found PRC to be a reliable longterm procedure for the treatment of advanced Kienböck's disease.

Stage 4

The treatment pillars for this stage include PRC and wrist fusion. As mentioned above, mild arthritic changes on the capitate head can be addressed at the time of PRC with an interpositional arthroplasty of dorsal wrist capsule. However, arthritic changes in this region may cause chronic pain and PRC failure, resulting in further intervention, this time a fusion.[35]

Wrist denervation alone or in combination with the aforementioned procedures can help with symptom relief. Schweizer *et al*. reported their long-term experience with complete wrist denervation in a 70-patient series, of which 11 patients had Kienböck's disease.[39] The average follow-up was 9.6 years. Complete wrist denervation led to subjective improvement in approximately 66% of patients, and approximately 50% reported complete or significant pain relief.

Kienböck's disease remains a clinical challenge for hand surgeons. The etiologic factors behind the disease process remain to be identified. Surgical advances involving local vascularized grafts from the distal radius have the potential to improve its treatment. Traditional procedures for the treatment of advanced Kienböck's disease, such as radial shortening osteotomy and PRC, have shown in long-term studies to provide adequate symptom control and functional restoration.

Conflict of interests: Authors claim they do not have any conflict of interest.

E. Zaidenberg ORCID: https://orcid.org/0000-0002-1535-0586

REFERENCES

1. Wagner JP, Chung KC. A historical report on Robert Kienböck (1871-1953) and Kienböck's disease. *J Hand Surg Am* 2005;30:1117-21. https://doi.org/10.1016/j.jhsa.2005.08.002
2. Hultén O. Uber anatomische variationen der handgelenkknochen. *Acta Radiol Scand* 1928;9:155-68. https://doi.org/10.3109/00016922809176658
3. Nakamura R, Tsuge S, Watanabe K, Tsunoda K. Radial wedge osteotomy for Kienböck's disease. *J Bone Joint Surg Am* 1991;73:1391-6. PMID: 1918122
4. Gelberman RH, Bauman TD, Menon J, Akeson WH. The vascularity of the lunate bone and Kienböck's disease. *J Hand Surg Am* 1980;5(3):272-8. https://doi.org/10.1016/s0363-5023(80)80013-x
5. Panagis JS, Gelberman RH, Taleisnik J, Baumgaertner M. The arterial anatomy of the human carpus. Part II: the intraosseous vascularity. *J Hand Surg Am* 1983;8(4):375-82. https://doi.org/10.1016/s0363-5023(83)80195-6
6. Allan CH, Joshi A, Lichtman DM. Kienbock's disease: diagnosis and treatment. *J Am Acad Orthop Surg* 2001;9:128-36. https://doi.org/10.5435/00124635-200103000-00006
7. Pichler M, Putz R. The venous drainage of the lunate bone. *Surg Radiol Anat* 2003;24:372-6. https://doi.org/10.1007/s00276-002-0065-y
8. Luo J, Diao E. Kienböck's disease: an approach to treatment. *Hand Clin* 2006;22:465-73. https://doi.org/10.1016/j.hcl.2006.07.003
9. Schuind F, Eslami S, Ledoux P. Kienböck's disease. *J Bone Joint Surg Br* 2008;90:133-9. https://doi.org/10.1302/0301-620X.90B2.20112
10. Bain GI, Begg M. Arthroscopic assessment and classification of Kienbock's disease. *Tech Hand Up Extrem Surg* 2006;10:8-13. https://doi.org/10.1097/00130911-200603000-00003
11. Goldfarb CA, Hsu J, Gelberman RH, Boyer MI. The Lichtman classification for Kienböck's disease: an assessment of reliability. *J Hand Surg Am* 2003;28:74-80. https://doi.org/ https://doi.org/10.1053/jhsu.2003.50035
12. Salmon J, Stanley JK, Trail IA. Kienböck's disease: conservative management versus radial shortening. *J Bone Joint Surg Br* 2000;82:820-3. https://doi.org/10.1302/0301-620x.82b6.10570
13. Beckenbaugh RD, Shives TC, Dobyns JH, Linscheid RL. Kienböck's disease: the natural history of Kienböck's disease and considerations of lunate fractures. *Clin Orthop* 1980;149:98-106. PMID: 7408323
14. Keith PP, Nuttall D, Trail I. Long-term outcome of nonsurgically managed Kienböck's disease. *J Hand Surg Am* 2004;29:63-7. https://doi.org/10.1016/j.jhsa.2003.10.016
15. Delaere O, Dury M, Molderez A, Foucher G. Conservative versus operative treatment for Kienböck's disease: a retrospective study. *J Hand Surg Br* 1998;23:33-6. https://doi.org/10.1016/s0266-7681(98)80214-3
16. Lichtman DM, Roure AR. External fixation for the treatment of Kienböck's disease. *Hand Clin* 1993;9:691-7. PMID: 8300738
17. Moran SL, Shin AY. Vascularized bone grafting for the treatment of carpal pathology. *Orthop Clin North Am* 2007;38:73-85. https://doi.org/10.1016/j.ocl.2006.10.010
18. Daecke W, Lorenz S, Wieloch P, Jung M, Martini AK. Vascularized os pisiform for reinforcement of the lunate in Kienböck's disease: an average of 12 years of follow-up study. *J Hand Surg Am* 2005;30:915-22. https://doi.org/10.1016/j.jhsa.2005.03.019
19. Sheetz KK, Bishop AT, Berger RA. The arterial blood supply of the distal radius and ulna and its potential use in vascularized pedicled bone grafts. *J Hand Surg Am* 1995;20:902-14. https://doi.org/10.1016/S0363-5023(05)80136-4
20. Bochud RC, Büchler U. Kienböck's disease, early stage 3: height reconstruction and core revascularization of the lunate. *J Hand Surg Br* 1994;19:466-78. https://doi.org/10.1016/0266-7681(94)90212-7
21. Tamai S, Yajima H, Ono H. Revascularization procedures in the treatment of Kienböck's disease. *Hand Clin* 1993;9:455-66. PMID: 8408255
22. Moran SL, Cooney WP, Berger RA, Bishop AT, Shin AY. The use of the 4-5 extensor compartmental vascularized bone graft for the treatment of Kienbock's disease. *J Hand Surg Am* 2005;30:50-8. https://doi.org/10.1016/j.jhsa.2004.10.002
23. Gabl M, Lutz M, Reinhart C, Zimmerman R, Pechlaner S, Hussl H, et al. Stage 3 Kienböck's disease: reconstruction of the fractured lunate using a free vascularized iliac bone graft and external fixation. *J Hand Surg Br* 2002;27:369-73. https://doi.org/10.1054/jhsb.2002.0766

24. Arora R, Lutz M, Deml C, Krappinger D, Zimmermann R, Gabl M. Long-term subjective and radiological outcome after reconstruction of Kienböck's disease stage 3 treated by a free vascularized iliac bone graft. *J Hand Surg Am* 2008;33:175-81. https://doi.org/10.1016/j.jhsa.2007.11.005
25. Raven EE, Haverkamp D, Marti RK. Outcome of Kienböck's disease 22 years after distal radius shortening osteotomy. *Clin Orthop Relat Res* 2007;460:137-41. https://doi.org/10.1097/BLO.0b013e318041d309
26. Watanabe T, Takahara M, Tsuchida H, Yamahara S, Kikuchi N, Ogino T. Long-term follow-up of radial shortening osteotomy for Kienböck disease. *J Bone Joint Surg Am* 2008;90(8):1705-11. https://doi.org/10.2106/JBJS.G.00421
27. Altay T, Kaya A, Karapinar L, Ozturk H, Kayali C. Is radial shortening useful for Litchman stage 3B Kienbock's disease? *Int Orthop* 2008;32(6):747-52. https://doi.org/10.1007/s00264-007-0428-4
28. Watanabe K, Nakamura R, Horii E, Miura T. Biomechanical analysis of radial wedge osteotomy for the treatment of Kienböck's disease. *J Hand Surg Am* 1993;18:686-90. https://doi.org/10.1016/0363-5023(93)90319-X
29. Koh S, Nakamura R, Horii E, Nakao E, Inagaki H, Yajima H. Surgical outcome of radial osteotomy for Kienböck's disease—minimum 10 years of follow-up. *J Hand Surg Am* 2003;28:910-6. https://doi.org/10.1016/s0363-5023(03)00490-8
30. Illarramendi AA, Schulz C, De Carli P. The surgical treatment of Kienböck's disease by radius and ulna metaphyseal core decompression. *J Hand Surg Am* 2001;26:252-60. https://doi.org/10.1053/jhsu.2001.22928
31. De Carli P, Zaidenberg EE, Alfie V, Donndorff A, Boretto JG, Gallucci GL. Radius core decompression for Kienböck disease stage IIIA: outcomes at 13 years follow-up. *J Hand Surg Am* 2017;42(9):752.e1-752.e6. https://doi.org/10.1016/j.jhsa.2017.05.017
32. Van den Dungen S, Dury M, Foucher G, Marin Braun F, Loréa P. Conservative treatment versus scaphotrapeziotrapezoid arthrodesis for Kienböck's disease. A retrospective study. *Chir Main* 2006;25:141-5. https://doi.org/10.1016/j.main.2006.07.030
33. Tambe AD, Trail IA, Stanley JK. Wrist fusion versus limited carpal fusion in advanced Kienbock's disease. *Int Orthop* 2005;29:355-8. https://doi.org/10.1007/s00264-005-0013-7
34. Salomon GD, Eaton RG. Proximal row carpectomy with partial capitate resection. *J Hand Surg Am* 1996;21:2-8. https://doi.org/10.1016/S0363-5023(96)80147-X
35. Croog AS, Stern PJ. Proximal row carpectomy for advanced Kienböck's disease: average 10-year follow-up. *J Hand Surg Am* 2008;33:1122-30. https://doi.org/10.1016/j.jhsa.2008.02.031
36. DiDonna ML, Kiefhaber TR, Stern PJ. Proximal row carpectomy: study with a minimum of ten years of follow-up. *J Bone Joint Surg Am* 2004;86:2359-65. PMID: 15523004
37. De Smet L, Robijns P, Degreef I. Proximal row carpectomy in advanced Kienböck's disease. *J Hand Surg Br* 2005;30:585-7. https://doi.org/10.1016/j.jhsb.2005.06.024
38. Lumsden BC, Stone A, Engber WD. Treatment of advanced-stage Kienböck's disease with proximal row carpectomy: an average 15- year follow-up. *J Hand Surg Am* 2008;33:493-502. https://doi.org/10.1016/j.jhsa.2007.12.010
39. Schweizer A, von Känel O, Kammer E, Meuli-Simmen C. Long-term follow-up evaluation of denervation of the wrist. *J Hand Surg Am* 2006;31:559-64. https://doi.org/10.1016/j.jhsa.2005.12.012