Introduction to Kienböck's Disease

Basic Science, Diagnosis and Treatment

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Part I

Introduction

Introduction

1

Norimasa Iwasaki

Kienböck's disease is a well-known wrist disorder that presents as wrist pain and stiffness and most commonly affects 20–40 year-old men who undertake manual labor. The reported male-to-female ratio of Kienböck's disease is two to one. [1, 2] The prevalence of bilateral Kienböck's disease is extremely low. Although patients commonly describe an insidious onset of wrist pain, some have a specific inciting trauma or accident. Physical examination frequently reveals swelling of the dorsal wrist due to synovitis of the radiocarpal joint, with tenderness over the dorsal aspect of the lunate. The range of motion of the wrist and grip strength is decreased compared with the contralateral side. However, these subjective and objective findings are not specific to Kienböck's disease. Therefore, the diagnosis of Kienböck's disease is based on radiographic findings, including a change in bone density, collapse, and fragmentation of the lunate. If these findings are not observed on plain radiographs, magnetic resonance imaging (MRI) is recommended. Magnetic resonance imaging is the most sensitive method for detecting early-stage Kienböck's disease without any specific radiographic changes in the lunate. Kienböck's disease is indicated by a decrease in the signal intensity of the entire lunate on MRI. Bone scintigraphy also shows an increase in uptake in the early stage of Kienböck's disease.

Kienböck's disease has been referred to by a variety of names based on the possible etiology, including lunatemalacia, aseptic necrosis, traumatic osteoporosis, osteochondritis, and osteitis. The etiology of Kienböck's disease is currently thought to be aseptic necrosis of the lunate. Ueba et al. [3] morphologically and histologically demonstrated a major fracture and multiple minor fractures in the collapsed lunate, with a mosaic pattern of viable and necrotic areas in the fragmented lunate (Fig. 1.1). However, the etiology of Kienböck's disease is still debatable.

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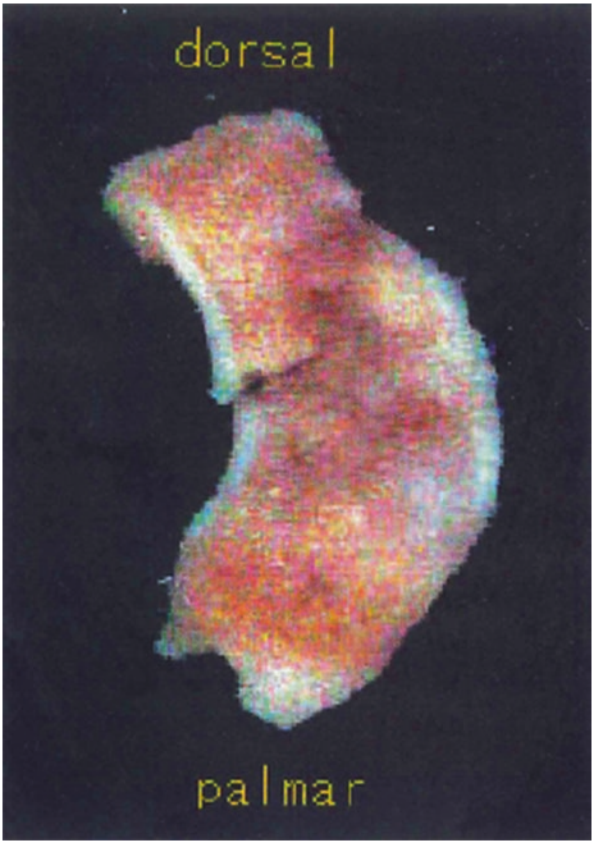


Fig. 1.1 Collapsed lunate specimen showing a major fracture and multiple minor fractures, with a mosaic pattern of pinkish viable and yellowish necrotic areas. From Ueba Y, et al.: Hand Surg 18 (2):141–149, 2013, Fig. 5 (A) with permission

Numerous authors have discussed the pathogenesis, etiology, diagnosis, and treatments of Kienböck's disease in textbooks and journal articles. However, Kienböck's disease is still challenging for hand surgeons to treat. In 1843, Peste [4] first described the collapse of the lunate in certain cadaver dissections. Approximately seven decades later, Robert Kienböck [5, 6], an Austrian radiologist, described a trauma-induced lesion resulting in loss of blood supply to the lunate. Since then, lunate collapse, referred to as Kienböck's disease, has been considered to result from avascular necrosis. However, as mentioned above, it remains difficult to determine the exact etiology. This has led to difficulty in establishing the optimal treatment strategy for Kienböck's disease.

Understanding the natural history of Kienböck's disease plays a crucial role in choosing the best treatment option for each patient. To describe disease progression, Lichtman and coworkers [1, 2] established a radiographic staging system based on Stahl's classification [7]. This system consists of four stages: stage I, normal except for the possibility of either a linear or a compression fracture; stage II, lunate sclerosis; stage III, lunate collapse; and stage IV, pancarpal arthritis. Stage III is further divided into IIIA (lunate collapse without fixed scaphoid rotation) and IIIB (lunate and carpal collapse with fixed scaphoid rotation). This radiographic staging system is widely accepted as a guide for determining the appropriate treatment. In 2010, Lichtman et al. [8] expanded the staging system to include stage 0 and stage IIIC. Stage 0 presents as intermittent pain without abnormal findings on radiographs and MRI. Stage IIIC presents as a coronal fracture or fragmentation of the lunate on radiographs, suggesting a poor likelihood of lunate revascularization. Therefore, Lichtman et al. [9] recommended lunate excision with prosthetic replacement, proximal row carpectomy, or intercarpal arthrodesis for stage IIIC Kienböck's disease.

Recent developments in imaging and arthroscopic techniques have furthered our understanding of the natural history and prognosis of Kienböck's disease [9–11]. Magnetic resonance imaging can evaluate the viability of bone marrow. On T1-weighted images, the loss of a homogeneous high signal intensity indicates a pathological condition such as edema, necrosis, sequestrum, revascularization, or tumor. T2-weighted images are used to distinguish between fluid-containing tissue and nonvascular or desiccated tissue. In 2016, Lichtman et al. [9] described the relationship between radiographic findings of the staging system and MRI findings (Table 1.1). The T1 signal intensity decreases from stage I to IV, while the T2 signal is variable, except for a consistently decreased signal intensity in stage IV. Currently, both radiographic and MRI examinations are recommended to correctly diagnose the disease stage. In 2010, Schmitt et al. [12] used gadolinium perfusion techniques to enhance T1-weighted sequences, leading to the development of a classification system that correlates with the lunate revascularization potential or predicts the disease prognosis [9, 13] (Table 1.2).

In 2006, Bain and Begg [14] described an arthroscopic classification of Kienböck's disease that they focused on the condition of the articular surface, with an articular surface with pathological conditions (including extensive fibrillation, fissuring, extensive articular loss, floating articular surface, fracture, and arthritis) defined as nonfunctional. The grading system consists of five grades ranging from 0 to 4 (grade 2a, proximal lunate and lunate fossa; 2b, proximal and distal lunate)

Table 1.1 Relationship between X-ray findings of the classification system and MRI findings [9]

Disease stage	X-ray findings	MRI findings
Stage I	Normal	T1 signal: Decreased T2 signal: Variable
Stage II	Lunate sclerosis	T1 signal: Decreased T2 signal: Variable
Stage IIIA	Lunate collapse	T1 signal: Decreased T2 signal: Variable
Stage IIIB	Lunate and carpal collapse Scaphoid rotation	T1 signal: Decreased T2 signal: Usually decreased
Stage IV	Pancarpal arthritis KDAC	T1 signal: Decreased T2 signal: Decreased

KDAC Kienöck's disease advanced collapse

Modified from Lichtman DM, et al.: J Hand Surg Am 41 (5): 630–638, 2016, Table 1.1 with permission

Table 1.2 Schmitt classification of Lunate vascularity/viability for predicting disease prognosis [9, 13]

MRI findings	Prognosis
Normal signal	NA
Homogenous enhancement of lunate with marrow edema; **intact lunate perfusion**	Good
Inhomogeneous signal with enhancement of the reparative zone and viable distal bone; necrotic proximal lunate (**partial osteonecrosis**)	Intermediate
No contrast enhancement; **complete osteonecrosis**	Poor

Modified from Lichtman DM, et al.: J Hand Surg Am 41 (5): 630–638, 2016, Table 1.2 with permission

based on the number of nonfunctional articular surfaces (Fig. 1.2) [9, 14]. This classification emphasizes the importance of arthroscopic findings of the articular surfaces to determine the treatment strategy for Kienböck's disease. To understand the disease severity in each case, surgeons should use the abovementioned diagnostic modalities to assess the bone morphology and the conditions of the subchondral bone, articular cartilage, and synovium; this information enables the selection of the best treatment option for each case.

In 1928, Húlten [15] reported a positive relationship between Kienböck's disease and a short ulna relative to the distal articular surface of the radius, also referred to as the ulnar minus variant or negative ulnar variance. Since this first description, the ulnar minus variant has been considered a characteristic radiographic finding of Kienböck's disease. This radiographic finding motivated researchers to clarify the role of mechanical stress in the etiology of Kienböck's disease. Werner et al. [16] evaluated the biomechanical effect of ulnar variance in a cadaveric model and demonstrated that changes in ulnar variance dramatically alter the force distribution through the wrist. Their results suggest that an increase in force transmission through the distal radius occurs in tandem with a decrease in the ulnar length, such as in the ulnar minus variant. Based on these experimental data, joint leveling procedures such as radial shortening or ulnar lengthening have been performed in patients with negative ulnar variance. Basic studies suggest that leveling procedures unload the lunate by ulnarly shifting the force transmission through the wrist [16–19]. Numerous studies have demonstrated satisfactory clinical outcomes after ulnar shortening [20–27]. This indicates the surgical efficacy of lunate decompression for the treatment of Kienböck's disease.

Tamai et al. [28] stated that there seem to be some racial differences in the ulnar variance. The prevalence of the ulnar minus variant among patients with Kienböck's disease is reported to be 78% [15] and 65% [2]; in contrast, studies from Japanese institutes report a prevalence of only 20%–26% [28]. There is no rationale for the joint leveling procedure in patients with neutral or positive ulnar variance. Kojima et al. [29] and Tsumura et al. [30, 31] proposed radial wedge osteotomy with reduction of the radial inclination for the treatment of Kienböck's disease with either negative or positive ulnar variance, as this procedure unloads the lunate by shifting the load toward the radioscaphoid joint. Nakamura et al. [32] reported that radial

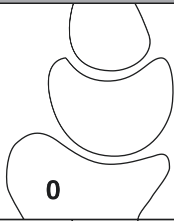
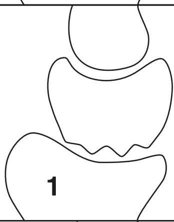
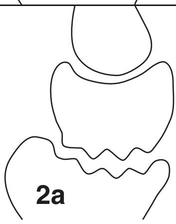
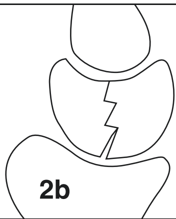
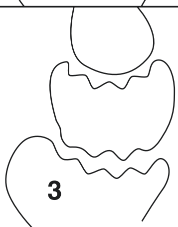
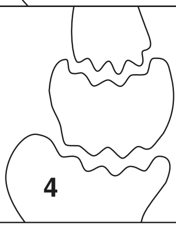
Grade	Description
 0	**Grade 0** 0 Nonfunctional surface
 1	**Grade 1** 1 Nonfunctional surface - Proximal lunate
 2a	**Grade 2a** 2 Nonfunctional surface - Proximal lunate and lunate facet of radius
 2b	**Grade 2b** 2 Nonfunctional surface - Proximal and distal lunate
 3	**Grade 3** 3 Nonfunctional surface - Capitate surface usually preserved
 4	**Grade 4** All 4 articular surfaces are nonfunctional

Fig. 1.2 Arthroscopic classification system by Bain and Begg [9, 14]. Modified from Lichtman DM, et al.: J Hand Surg Am 41 (5): 630–638, 2016, Fig. 1.1. with permission

wedge osteotomy achieved satisfactory clinical outcomes at a minimum 2-year follow-up in 25 of 27 patients, most of whom had neutral or positive ulnar variance. Similarly, Wada et al. [33] reported good long-term clinical and radiographic results after radial wedge osteotomy in 13 patients with Kienböck's disease with a minimum 10-year follow-up. However, Iwasaki et al. [34] reported that 2 of 11 patients who underwent radial wedge osteotomy had progression of degenerative changes at the radioscaphoid joint. Although biomechanical and clinical studies indicate that radial wedge osteotomy is suitable for treating Kienböck's disease with neutral and positive ulnar variance [29–37], radial closing osteotomy seems to result in a high probability of progressive degenerative changes in the joints adjacent to the scaphoid.

Partial or complete capitate shortening is also considered to decompress the lunate by transferring the load to the radial and ulnar intercarpal joints [38–40]. A study reporting the clinical and radiographical results of 23 patients with more than 2 years of follow-up demonstrated that partial capitate shortening is effective in treating stage III Kienböck's disease [41]; the authors suggested that this procedure markedly reduces compressive forces only on the lunate, thereby facilitating better lunate revascularization. Other studies have also shown that capitate shortening achieves successful functional and radiological results for stages II and IIIA Kienböck's disease [42–44].

The rational approach to the treatment of necrotic bone is to re-establish new blood supply to the bone. Based on animal experimental results, Hori et al. [45] directly implanted the dorsal metacarpal vascular pedicle into the diseased lunate of nine patients with Kienböck's disease; all but one patient achieved satisfactory clinical and radiographic outcomes, with the longest follow-up of 3 years. Tamai et al. [28] reported that vascular bundle implantation improved pain and grip strength in 50 of 51 patients with Kienböck's disease; however, 84.3% of patients showed a decreased or unchanged roentgenologic carpal vertical height postoperatively. These results suggest the necessity of mechanical support to relieve the vertical stress on the lunate.

Vascularized bone grafting is a direct revascularization technique that provides revascularization and mechanical support for the diseased lunate. Direct revascularization techniques have been developed for the early stages of Kienböck's disease and are theoretically best indicated for stage II Kienböck's disease without obvious collapse of the lunate. Direct revascularization techniques mainly include pedicled or vascularized bone grafts. The commonly selected donor site is the 4 + 5 extensor compartmental artery bone graft harvested from the distal radius [46]. Moran et al. [46] reported that 85% of patients achieved satisfactory results based on the Lichtman outcome score at a mean of 31 months after surgery. Postsurgical radiographs showed no further collapse of the lunate in 65% of patients, while T2 and/or T1 MRI findings suggested improvement of lunate vascularization. Recent studies have reported satisfactory outcomes of vascularized medial femoral trochlea (MFT) osteochondral lunate reconstruction for advanced Kienböck's disease [47, 48]. The MFT flap allows reconstruction of the proximal portion of the collapsed lunate, including the radiolunate articular surface. Pet et al. [48] evaluated 18 patients with stage IIIA and IIIB Kienböck's disease and found that MFT reconstruction of the proximal lunate provides acceptable clinical and radiographic outcomes, suggesting a cessation of radiocarpal collapse, with acceptable postoperative donor knee function. Another study showed fibrocartilage filling of the defect at the donor site of the knee, without pathological MRI or radiographic changes [49]. This surgical technique may provide anatomical reconstruction of the diseased lunate to prevent further carpal collapse.

As mentioned above, Kienböck's disease typically occurs in men aged 20–40 years who engage in manual labor. However, this disease has also been observed in pediatric and elderly patients. Because of the variations in the disease characteristics and prognosis, it is important to consider the patient's age when selecting treatment options. Irisarri et al. [50] classified pediatric Kienböck's disease into two groups, infantile (12 years and younger) and juvenile (13 years to the end of skeletal maturity). They stated that Kienböck's disease in pediatric patients is unlikely to be caused by mechanical factors, such as negative ulnar variance and repetitive minor trauma related to occupation. Regarding the disease prognosis, they consider infantile and early juvenile (13 or 14 years) Kienböck's disease to be self-limiting, with a normal or near normal outcome, because of the great potential for remodeling of the lunate. In contrast, patients older than 15 years with advanced-stage Kienböck's disease are frequently unresponsive to conservative treatments and need surgical treatment, usually radial shortening; however, the prognosis of these patients seems better than that of adults [50]. Iwasaki et al. [25] demonstrated that radial osteotomies, including radial shortening and lateral closing wedge osteotomy, are effective in improving not only clinical outcomes but also radiographic findings in teenagers with stage II and III Kienböck's disease. These results indicate that lunate revascularization and remodeling occur after radial osteotomies in teenagers.

Kienböck's disease is relatively rare in the elderly. Taniguchi et al. [51] showed that Kienböck's disease in a cohort of 14 patients older than 60 years commonly affected the dominant hand of manual laborers but was more common in women than men; radiographically, the frequency of negative ulnar variance was low. Yoshida et al. [52] demonstrated obvious radiographic osteoporosis in the affected wrist of elderly patients and suggested that osteoporosis may be an etiologic factor of Kienböck's disease in the elderly. Taniguchi et al. [51] reported that all elderly patients who received nonsurgical treatment had good or excellent clinical outcomes, despite radiographic progression. Iwasaki et al. [24] found that patients older than 54 years had a significantly lower clinical score after radial osteotomy than younger patients, suggesting that older age is a negative predictor of postoperative clinical outcomes. The etiologic factors and prognosis may differ between elderly patients versus younger patients with Kienböck's disease. These issues warrant investigation in a study with a large population.

Novel surgical procedures have been advocated for the treatment of Kienböck's disease, such as lunate excision with capitate lengthening [53], lunatoplasty using a balloon [54], core decompression of the distal radius [55], arthroscopically assisted -intercarpal fusion [56], and -proximal row carpectomy [57]. The ideal procedure is a minimally invasive one that improves the clinical symptoms and regenerates the diseased lunate.

Unfortunately, the current widely performed procedures do not achieve the treatment goal. Ogawa et al. [58, 59] developed a new treatment strategy for regenerating the necrotic lunate using a combination of local bone marrow transfusion, low-intensive pulsed ultrasound therapy, and external fixation and demonstrated improvements in clinical symptoms and MRI findings. These favorable results suggest that this procedure may be useful as a less-invasive surgical alternative for stage II and III Kienböck's disease. Future studies are expected to develop a less-invasive surgical strategy based on cell therapy or tissue engineering techniques for regeneration or remodeling of the diseased lunate.

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Part II

Pathogenesis and Basic Science

2 Wrist Anatomy and Vascularity

Yuichiro Matsui

Abstract

The wrist joint comprises the two forearm bones (radius and ulna) and eight carpal bones (scaphoid, lunate, triquetrum, pisiform, trapezium, trapezoid, capitate, and hamate). The four proximal carpal bones (scaphoid, lunate, triquetrum, and pisiform) comprise the proximal carpal row, and the four distal bones (trapezium, trapezoid, capitate, and hamate) make up the distal carpal row. The wrist joint forms the midcarpal joint between the proximal and distal carpal rows, the radiocarpal joint between the radius and proximal carpal rows, and the distal radioulnar joint between the radius and ulna. The triangular fibrocartilage, radioulnar ligament, meniscus homologue, sheath of the extensor carpi ulnaris tendon, and other tissues present on the ulnar side of the wrist are called the triangular fibrocartilage complex (TFCC). The lunate is considered to be the keystone of the entire carpal bone anatomical structure. It has four articular surfaces: proximal, distal, radial, and ulnar, with the volar and dorsal surfaces being nonarticular and attached to the ligaments. The lunate is the most proximal of the carpal bones and is the most susceptible to direct impact with the radius and, because of its relatively weak ligamentous connections with the other carpal bones, it is thought to be the most commonly dislocated of the carpal bones. In 2007, Lamas et al. injected latex into the brachial arteries of 27 cadaveric upper limbs and investigated the arterial distribution and anastomosis in the lunate. The number of blood vessels in the lunate was greater on the palmar side than on the dorsal side.

Keywords

Wrist joint · Radius · Ulna · Carpal bones

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The wrist joint comprises the two forearm bones (radius and ulna) and eight carpal bones (scaphoid, lunate, triquetrum, pisiform, trapezium, trapezoid, capitate, and hamate). The four proximal carpal bones (scaphoid, lunate, triquetrum, and pisiform) comprise the proximal carpal row, and the four distal bones (trapezium, trapezoid, capitate, and hamate) make up the distal carpal row. The pisiform is not exactly a carpal bone constituting the wrist joint, as it lies palmar to the surface formed by the other three bones and is anatomically the sesamoid bone of the flexor carpi ulnaris tendon.

The wrist joint forms the midcarpal joint between the proximal and distal carpal rows, the radiocarpal joint between the radius and proximal carpal rows, and the distal radioulnar joint between the radius and ulna. The midcarpal joint has relatively strong bony support because of the structure of the capitate and hamate fitting into the proximal carpal row. In contrast, the radiocarpal joint has strong ligamentous support. In other words, the radiolunate ligament and the dorsal radiocarpal ligament mainly control the slippage of the carpal bones caused by the palmar–ulnar tilt of the distal radial joint surface. The distal radioulnar joint is formed by the sigmoid notch on the distal ulnar side of the radius and the ulnar head, and it is involved in forearm rotation together with the proximal radioulnar joint. The triangular fibrocartilage, radioulnar ligament, meniscus homologue, sheath of the extensor carpi ulnaris tendon, and other tissues present on the ulnar side of the wrist are called the triangular fibrocartilage complex (TFCC).

The lunate is considered to be the keystone of the entire carpal bone anatomical structure. It has four articular surfaces: proximal, distal, radial, and ulnar, with the volar and dorsal surfaces being nonarticular and attached to the ligaments [1] (Fig. 2.1). The distal articular surface is concave and articulates with the head of the capitate and the proximal pole of the hamate. In about 65% of people, there is a distinct articular surface opposite each bone. The radial articular surface of the lunate articulates with the scaphoid, and the ulnar articular surface articulates with the triquetrum. The proximal articular surface of the lunate is a convex surface with a larger curvature radius than that of the scaphoid. In the neutral wrist position, approximately two-thirds of the radial articular surface of the lunate articulates with the radius, and one-third of the ulnar articular surface articulates with the articular disc. The lunate is the most proximal of the carpal bones and is the most susceptible to direct impact with the radius and, because of its relatively weak ligamentous connections with the other carpal bones, it is thought to be the most commonly dislocated of the carpal bones.

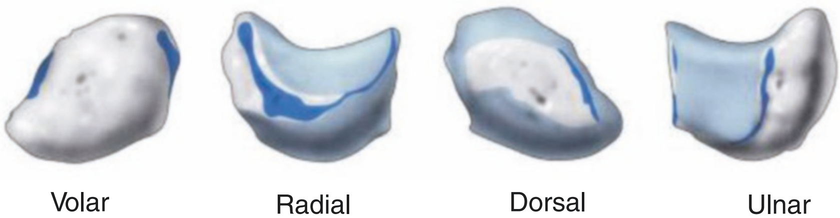

Fig. 2.1 Lunate morphology. The light blue and dark blue areas indicate joint surface and ligament attachment, respectively. Reproduced with permission [5]

The normal range of motion of the wrist joint is 80° in flexion, 70° in extension, 20° in radial deviation, and 30° in ulnar deviation. As mentioned earlier, with the tilt of the distal radial joint surface toward the palmar–ulnar side, the ranges of motion of flexion and ulnar deviation become greater than those of extension and radial deviation, respectively. These movements are mainly performed at the midcarpal and radiocarpal joints. During extension of the wrist, 66.5% of the motion is performed at the radiocarpal joint and 33.5% at the midcarpal joint. During flexion of the wrist, 40% of the motion is performed at the radiocarpal joint and 60% at the midcarpal joint [2]. The distal carpal row moves with the metacarpus radially during radial deviation of the wrist and ulnarly during ulnar deviation, while the proximal carpal row not only moves in the opposite direction to the distal carpal row but also rotates in the sagittal plane. In other words, the proximal carpal row flexes palmarly while moving ulnarly when the wrist is deviated radially and dorsiflexes radially when the wrist deviates ulnarly. The movement of the wrist from radial extension to ulnar flexion is called the dart-throwing motion, but this is mainly performed at the midcarpal joint, with less movement at the radiocarpal joint [3, 4].

The nutritive vessels of the wrist joint are the radial artery, the ulnar artery, and the anterior and posterior interosseous arteries [5] (Fig. 2.2). The radial artery provides the most stable blood flow to the carpal bones. The radial artery runs distally on the radial side of the flexor carpi radialis tendon between the two layers of the forearm fascia and divides proximally at the wrist joint into the palmar and dorsal branches of the radial artery. The scaphoid is supplied with blood flow by the radial artery, 80% of which flows from the dorsal ridge of the scaphoid waist [6]. The ulnar artery runs distally with the ulnar nerve through the dorsal portion of the flexor carpi ulnaris muscle, exits the radial side of the pisiform, enters the Guyon canal, and divides into the superficial and deep branches of the ulnar artery. The palmar and dorsal branches of the radial artery anastomose with the superficial and deep branches of the ulnar artery in the palm to form the superficial palmar arch and deep palmar arch, respectively, which send branches to the fingers. The anterior interosseous artery runs along the anterior surface of the interosseous membrane of the forearm and divides into the palmar dorsal branches at the distal forearm. The two branches are located at the wrist joints. The posterior interosseous artery runs peripherally along the posterior aspect of the interosseous membrane of the forearm and anastomoses with the dorsal branch of the anterior interosseous artery at the distal part of the forearm. The branches of these arteries anastomose with each other to form a network of arteries surrounding the wrist joint [7]. There are three arterial arches on the dorsal side of the wrist. The dorsal radiocarpal arch is located under the extensor tendons at the radiocarpal joint and supplies blood to the lunate and triquetrum. The dorsal intercarpal arch has the largest and most stable blood flow and supplies the distal carpal row. The basal metacarpal arch is the thinnest and supplies blood to the distal carpal row. On the palmar side, the palmar radiocarpal arch travels along the articular capsule of the wrist and provides blood to the palmar side of the lunate and triquetrum. In addition, the deep palmar arch resides at the base of the metacarpal bone and connects to the terminal branch of the radial artery, providing blood to the distal carpal row.

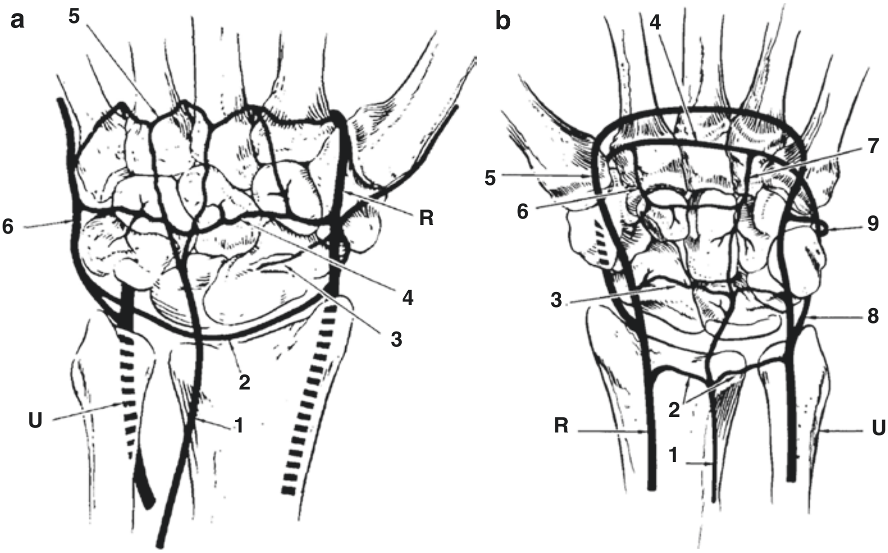

Fig. 2.2 Schematic drawings of the arterial supply. Reproduced from [5] with permission. (**a**) The arteries to the dorsal side of the wrist. *R*, Radial artery; *U*, ulnar artery; *1*, dorsal branch, anterior interosseous artery; *2*, dorsal radiocarpal arch; *3*, branch to the dorsal ridge of the scaphoid; *4*, dorsal intercarpal arch; *5*, basal metacarpal arch; *6*, medial branch of the ulnar artery. (**b**) The arteries to the volar side of the wrist. *R*, Radial artery; *U*, ulnar artery; *1*, palmar branch, anterior interosseous artery; *2*, palmar radiocarpal arch; *3*, palmar intercarpal arch; *4*, deep palmar arch; *5*, superficial palmar arch; *6*, radial recurrent artery; *7*, ulnar recurrent artery; *8*, medial branch, ulnar artery; *9*, branch off ulnar artery contributing to the dorsal intercarpal arch

In 2007, Lamas et al. [8] injected latex into the brachial arteries of 27 cadaveric upper limbs and investigated the arterial distribution and anastomoses in the lunate using the Spalteholz technique [9] The results showed that the lunate consistently had dorsal and palmar arteries entering the bone in all specimens. The number of blood vessels in the lunate was greater on the palmar side than on the dorsal side. In 2011, Dubey et al. [10] investigated the number of nutrient foramina in the lunate. They found that 91.33% of the lunates had two or more nutrient foramina. From the palmar side, 100% of the radial artery branches, 72.22% of the anterior interosseous artery branches, and 69.4% of the branches from the arterial intercarpal arch entered the lunate. From the dorsal side, 100% of the radial artery branches, 85.71% of the anterior interosseous artery branches, and 50% of the branches from the dorsal intercarpal arch entered the lunate.

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3 Wrist Biomechanics of Kienböck's Disease

Hisao Moritomo

Abstract

Negative ulnar variance, a high lunate uncovering index, and angulated lunate trabeculae are anatomic variants that predispose to Kienböck's disease. These conditions induce abnormal internal lunate stress, encourage the progression of incomplete fractures, and cause progressive lunate collapse and localized trabecular osteonecrosis. Conversely, type II lunates appear to be protective against coronal fractures and scaphoid flexion deformities. Lichtman stage IIIb Kienböck's disease and scapholunate dissociation appear to have different patterns of carpal collapse, particularly with respect to radioscaphoid joint congruity. Unlike scapholunate dissociation, stage IIIb Kienböck's disease does not involve dorsal subluxation of the proximal scaphoid proximal pole, and congruity of the radioscaphoid joint is retained. Radial shortening or ulnar lengthening of 2.5 mm decreases radiolunate pressure by one-half. Radial wedge osteotomy or lateral closing wedge osteotomy does not actually decrease the total radiolunate load; however, it enlarges the radiolunate joint contact by allowing better coverage of the lunate, thereby decreasing its deleterious peak pressures. Among the different types of surgical decompression procedures, the largest reduction in compressive forces on the lunate is obtained by capitate shortening. However, capitate shortening causes the scaphoid–trapezium joint to be greatly overloaded, and under such forces, the scaphoid progressively adopts an abnormal palmar flexed position, and carpal collapse occurs. Carpal collapse following capitate shortening can be prevented by partial capitate shortening, which retains articular contact between the capitate and the scaphoid.

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Keywords

Kienböck's disease · Lunate · Kinematics · Kinetics · Biomechanics · Radial shortening · Partial capitate shortening · Type II lunate · Contact area · Carpal deformity · Carpal collapse · Lunate morphology

3.1 Introduction

Normal carpal mechanics depend on a complex interplay between a sophisticated arrangement of carpal ligament and carpal bone geometry [1]. Although the origin and natural history of Kienböck's disease remain unclear, treatment of Kienböck's disease has been designed to decrease compressive loading of the lunate to prevent lunate collapse and to allow lunate revascularization. Information regarding the load transfer characteristics, contact area, and pressures on the lunate has offered a basis to better understand the pathomechanics of Kienböck's disease. It has also served as a means to assess the possible efficacy of different types of surgical decompression procedures. This chapter first reviews the kinetics and kinematics of the normal wrist, followed by the morphological influence on and kinetics of Kienböck's disease, and finally, the kinetic efficacy of different types of surgical decompression procedures.

3.2 Kinetics and Kinematics of the Normal Lunate

3.2.1 Midcarpal Joint

The midcarpal joint consists of the scaphoid–trapezoid–trapezoid joint, scaphoid–capitate joint, lunate–capitate joint, and triquetrum–hamate joint. The distal row has been considered a relatively fixed or constrained group of bones, and the proximal carpal row is, by comparison, much less constrained [1]. The normal load mechanics of the midcarpal joint were studied using pressure-sensitive film, and load distribution through the midcarpal joint was reported to be as follows: scaphoid–trapezium–trapezoid, 23%; scaphoid–capitate, 28%; lunate–capitate, 29%; and triquetrum–hamate, 20% [1] (Fig. 3.1a). When the scaphoid flexes in normal wrists, the flexion moment is constrained by the extension moment of the triquetrum, and stable equilibrium is achieved via the lunate as a bony link [2]. If the link is broken [e.g., due to scapholunate dissociation (SLD), scaphoid nonunion, or advanced Kienböck's disease], the scaphoid flexes, and the capitate translates proximally, i.e., carpal collapse occurs.

In Viegas et al.'s cadaveric study, in 35% of wrists, the lunate articulates only with the capitate (type I lunate); in the other 65% of wrists, a second distal joint surface is presently articulating with the hamate (type II lunate) [3]. When present,

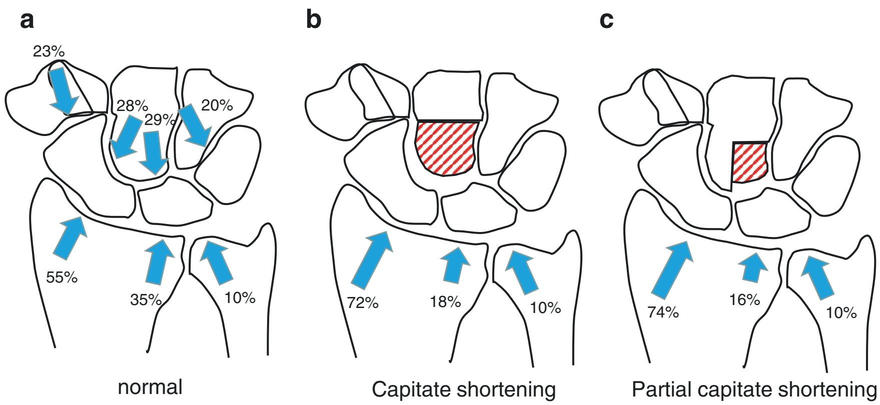


Fig. 3.1 Load distribution through the midcarpal joint and the radiocarpal joint. Partial capitate shortening showed a greater decompression effect than capitate shortening. (**a**) Normal wrist. (**b**) Capitate shortening. (**c**), Partial capitate shortening

the medial facet of the lunate articulates with the hamate during midcarpal motion, which can result in degenerative change at the proximal pole of the hamate in 44% of type II lunate wrists [3]. One, however, should take into account that this degenerative change is typically asymptomatic [4]. In a magnetic resonance imaging study in six positions of radial-ulnar deviation, the wrists with a type I lunate did not have contact between the lunate and hamate in any position; the wrists with a type II lunate did have contact between the hamate and the lunate but only in ulnar deviation [5]. We investigated contact between the type II lunate and the hamate using computed tomographies in nine wrist positions (neutral, radial extension, pure extension, ulnar extension, radial deviation, ulnar deviation, radial flexion, pure flexion, and ulnar flexion) [6]. We found that the type II lunate contacts the hamate only at wrist ulnar extension, ulnar deviation, and ulnar flexion and does not contact other wrist positions. These results suggest that resection of the hamate pole may not be necessary when applying a partial capitate shortening procedure.

3.2.2 Radiocarpal Joint

The lunate articulates proximally with the radius and with the triangular fibrocartilage. The investigation of force transmission through the normal wrist using discrete element analysis models revealed that, on average, 90.3% of the total radioulnocarpal force was transmitted to the radius, with 61.0% (55% of total) transmitted through the radioscaphoid joint and 39.0% (35% of total) transmitted through the radiolunate joint [7]. A mean of 9.7% was dissipated through the triangular fibrocartilage (Fig. 3.1a).

3.3 Morphological Influence on and Kinetics of Kienböck's Disease

3.3.1 Morphological Influence

Morphological variations, such as negative ulnar variance, high uncovering of the lunate, abnormal radial inclination and/or a trapezoidal shape of the lunate, and the particular vascular pattern, may be predisposing factors [8]. Hultén noted in 1928 that a short ulna (ulnar negative variance) was present in 78% of his patients with Kienböck's disease, whereas only 23% of normal patients had a short ulna [9]. However, D'Hoore et al. found no statistically significant relationship between ulnar variance and Kienböck's disease [10]. In addition, Nakamura et al. noted that most Japanese patients with Kienböck's disease had positive variance [11].

Ledoux et al. analyzed the influence of morphological variations on Kienböck's disease using a finite element analysis model [12]. The ulnar variance had a strong influence on the ratios of radiolunate/ulnolunate total load and peak pressures. The distribution of internal stresses was markedly affected by the lunate uncovering index. The evolution of a simulated incomplete fracture was dramatically influenced by morphological parameters: with positive ulnar variance, the fracture did not progress, but in the presence of three associated conditions—negative ulnar variance, a high lunate uncovering index, and angulated trabeculae of the lunate—the fracture progressed, and the proximal part of the lunate collapsed. This study supports the concept that some lunates are predisposed to Kienböck's disease because their anatomy induces abnormal internal stresses, which allow an incomplete fracture to progress under heavy loading conditions and cause progressive collapse and localized trabecular osteonecrosis.

Rhee et al. studied the incidence of type I and type II lunates in patients presenting with Kienböck's disease [13]. They reported a 29% prevalence of type II lunates in the affected wrist of Kienböck patients. There was significantly more advanced disease (stage IIIA or greater) upon initial presentation in the wrist with a type I compared with a type II lunate. Overall, the radioscaphoid angle was not significantly different between type I and type II lunates. Coronal plane fractures were significantly more common in type I compared with type II lunates. In the absence of a coronal plane fracture, type II lunates exhibited significantly smaller radioscaphoid angles than type I lunates. There were no significant differences between the two types of lunates for ulnar variance, ulnar translocation of the carpus, or carpal collapse. They concluded that type II lunates appear to be protective against coronal fractures and scaphoid flexion deformities.

3.3.2 Kinetics

According to the Lichtman classification scheme, carpal collapse and osteoarthritis gradually develop as Kienböck's disease progresses [14]. Iwasaki et al. [15] studied the effects of lunate collapse and demonstrated that in the early stages of the disease (stages II and IIIa), the normal position of the scaphoid prevented, to some extent, transmission of excessive forces to the lunate. However, as the scaphoid assumed its flexed position (in stage IIIb), the loads across the lunate were increased, thereby accelerating the process of collapse and fragmentation. The proximal articular cartilage facing the radius is generally more affected than the distal articular cartilage facing the capitate [16].

Clinically, however, some patients with advanced Kienböck's disease can be asymptomatic, and the symptoms do not correlate well with the changes in the shape of the lunate and the degree of carpal collapse [17]. Taniguchi et al. reported patients with asymptomatic stage IV Kienböck's disease that was associated with marked carpal collapse, and their radioscaphoid joint (RSJ) space was retained [18]. In contrast, it was revealed that wrists with an SLD had RSJ incongruity caused by dorsal subluxation of the scaphoid proximal pole as well as carpal collapse, and the incongruity caused osteoarthritis in the RSJ [19, 20]. These facts suggest that the deformity pattern of SLD would be different from that of stage IIIb Kienböck's disease. We examined three-dimensional carpal alignment in stage IIIa and IIIb Kienböck's disease to determine whether RSJ incongruity involving dorsal subluxation of the scaphoid proximal pole was present [21]. These results were compared with those for normal wrists and wrists with SLD and RSJ incongruity involving scaphoid rotatory subluxation. We found that normal RSJ congruity of both stage IIIa and IIIb Kienböck's disease was retained (Figs. 3.2a and 3.3a–c). Conversely, in the SLD, the scaphoid flexed to the same extent as in stage IIIb Kienböck's disease but also shifted dorsodistally, producing RSJ incongruity (Fig. 3.2b and 3.3d). The centroid of the capitate in stages IIIa and IIIb Kienböck's disease was not significantly different from the capitate in the normal wrists. Compared with the capitate in stage IIIb Kienböck's disease, the centroid of the capitate with SLD significantly translated 5.2 mm in the dorsal direction, where dorsiflexion of the lunate was associated (Fig. 3.4). We concluded that the patterns of carpal collapse differed between stage IIIb Kienböck's disease and SLD in terms of RSJ congruity. Our results suggest that carpal collapse in Kienböck's disease is less commonly associated with RSJ incongruity, which may explain why there are many asymptomatic patients with advanced Kienböck's disease.

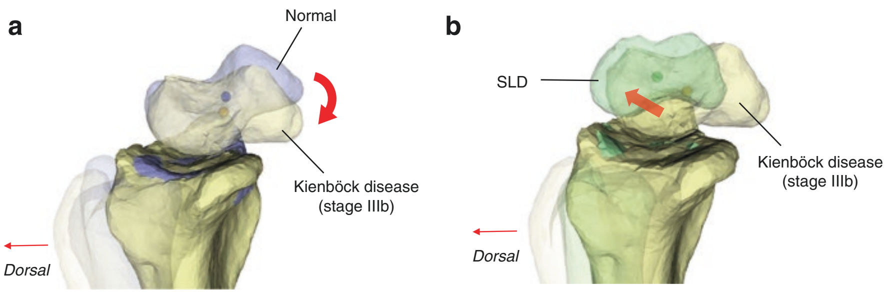


Fig. 3.2 Bone models of the scaphoid and radius in the right wrist with normal, stage IIIb Kienböck's disease, and scapholunate dissociation (SLD) superimposed and viewed from the ulnar side. The similar color points represent the centroids of each scaphoid. Compared with a normal scaphoid, the scaphoid in stage IIIb Kienböck disease is flexed, and the centroids are located proximally (**a**). Compared with stage IIIb Kienböck's disease, the centroid and the proximal pole of the scaphoid with SLD show significant translation in the dorsal and distal directions (**b**)

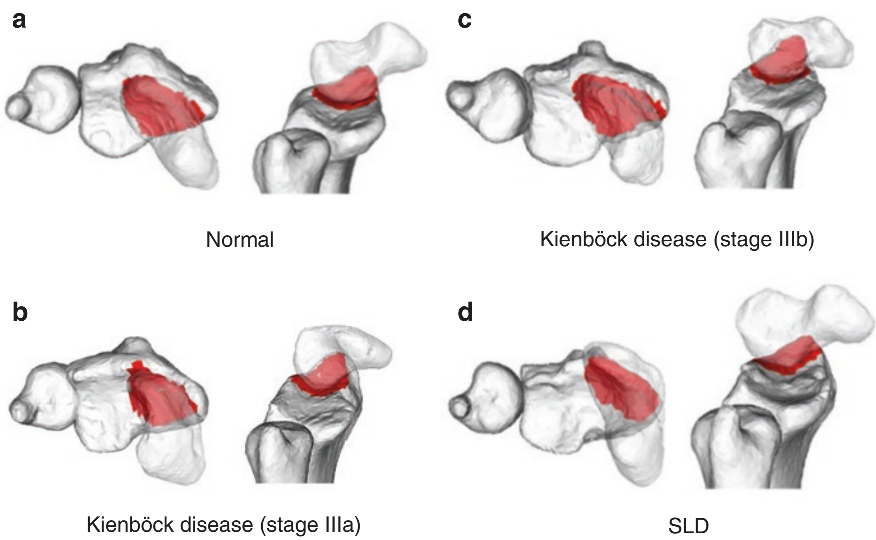
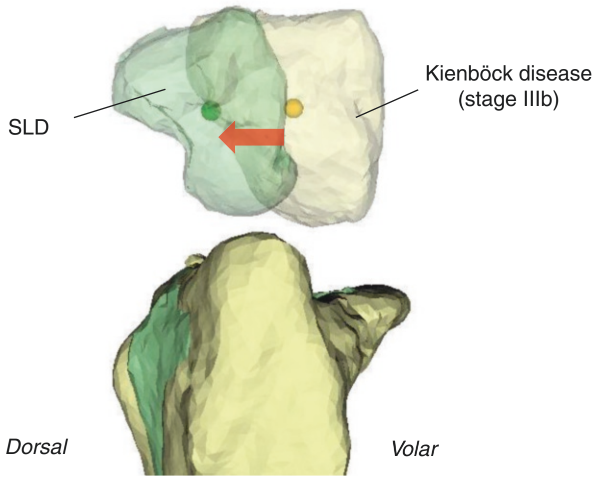


Fig. 3.3 Inferred contact areas of the articular surface of the radioscaphoid joint (RSJ). A typical example of each joint is shown. The left illustration of each shows a distal view, and the right illustration shows the ulnar view. The contact areas in the RSJ of normal, stage IIIa and stage IIIb Kienböck's disease wrists are similar, and congruency is retained (**a**–**c**). In RSJ with scapholunate dissociation (SLD), the contact area is located on the dorsal side, and there is a joint incongruity for dorsal subluxation of the scaphoid proximal pole (**d**)

Fig. 3.4 Bone models of the capitate and radius in the right wrist with stage IIIb Kienböck's disease and SLD superimposed. Compared with stage IIIb Kienböck's disease, the centroid of the capitate with SLD shows significant dorsal translation



3.4 Kinetic Efficacy of Different Types of Surgical Decompression Procedures

3.4.1 Radial Shortening Osteotomy

As Kienböck's disease could be the result of excessive bone stress, studies have been conducted to understand how it might be possible to unload the lunate [8]. The most common procedure for unloading the lunate in patients with ulnar negative variance is radial shortening osteotomy [22–24]. Radial shortening or ulnar lengthening appears to redistribute some of the radiolunate load to the radioscaphoid joint and the ulnocarpal joint [25–27]. Werner et al. examined the effect of ulnar lengthening in cadavers and found that the radiolunate pressure was halved with a 2.5-mm ulnar lengthening [27]. On the other hand, radial shortening or ulnar lengthening increases the ulnocarpal pressure, which potentially produces postoperative ulnocarpal impaction syndrome, which requires ulnar shortening osteotomy (Fig. 3.5).

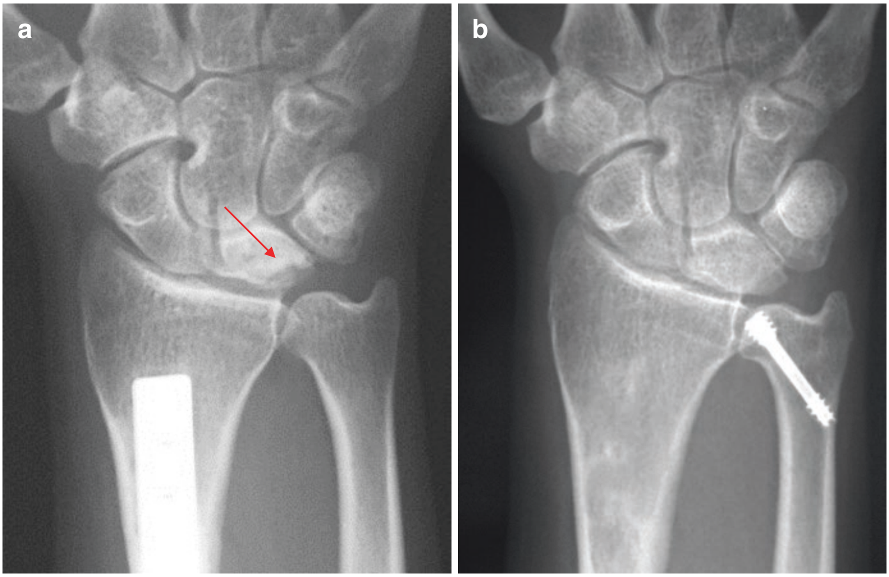


Fig. 3.5 Ulnocarpal impaction syndrome that occurred 1 year after radial shortening was performed for a 19-year-old girl with stage II Kienböck's disease (**a**). Note the cystic changes at the proximal and ulnar aspects of the lunate facing the ulnar head (arrow). Pain was resolved by distal metaphyseal ulnar shortening osteotomy (modified Kitano procedure) (**b**)

3.4.2 Radial Wedge Osteotomy

Radial wedge osteotomy, or lateral closing wedge osteotomy, which decreases radial inclination, has been proposed for patients with neutral or positive ulnar variance [28]. Conflicting results have been published regarding the unloading effects of changing the radial inclination [27, 29–31]. It is likely that lateral closing wedge osteotomy does not actually decrease the total radiolunate load; however, by allowing better covering of the lunate by the distal radius, it enlarges radiolunate joint contact, thereby decreasing its deleterious peak pressures [8, 30].

3.4.3 Capitate Shortening Osteotomy

Decompression procedures for the treatment of Kienböck's disease include the capitate shortening procedure [32, 33]. In the classical capitate shortening procedure, a transverse osteotomy is located at the capitate waist, and both the lunate and the scaphoid facets of the capitate are shortened. Capitate shortening is particularly useful in positive or neutral ulnar variance because its operative indication does not depend on ulnar variance, whereas the radial shortening procedure is only appropriate for negative ulnar variance. Biomechanically, Werber et al. noted that significant load reduction on the lunate was evident in all specimens after capitate shortening [34]. An average decrease of 49% (18% of the total, Fig. 3.1b) was observed under a 9.8-N load, and a decrease of 56% was observed under a 19.6-N load. Horii et al., however, suggested that this operation causes the scaphoid–trapezium joint to be tremendously overloaded and, under such forces, the scaphoid progressively adopts an abnormal palmar flexed position [26]. Along with this collapse, the distal carpal row migrates proximally until capitate–lunate contact is reestablished (Figs. 3.6 and 3.7).

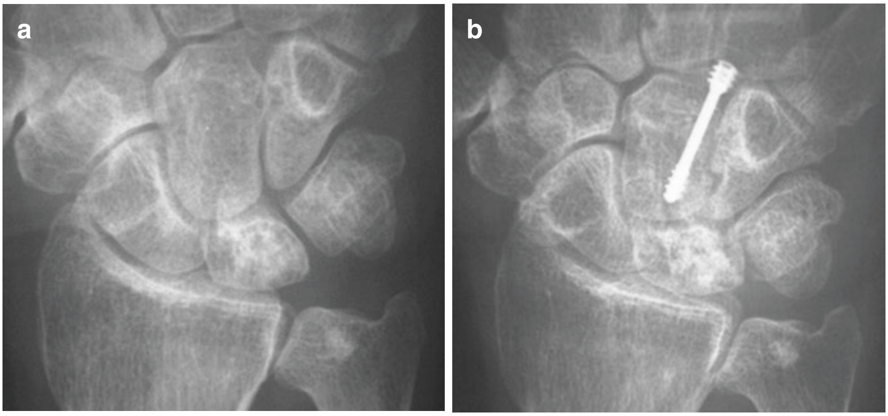


Fig. 3.6 Preoperative (**a**) and 1.5-year postoperative (**b**) X-rays of classical capitate shortening procedure performed for a 25-year-old man with stage II Kienböck's disease. Note that the scaphoid flexed and carpal collapse progressed postoperatively until capitate–lunate contact was reestablished, decreasing carpal height

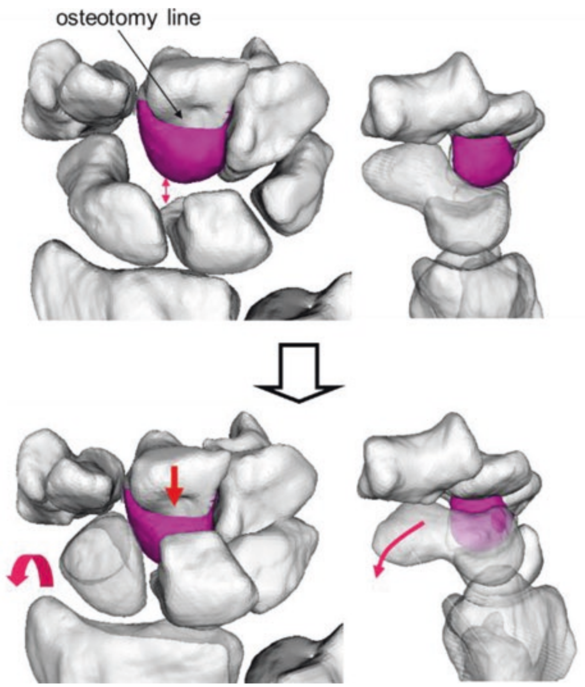


Fig. 3.7 After classical capitate shortening, the scaphoid progressively adopts an abnormally flexed position. With this collapse, the distal carpal row migrates proximally until capitate–lunate contact is reestablished

3.4.4 Partial Capitate Shortening

To preserve scaphoid–capitate and midcarpal dynamics, in 2001, we developed a modified capitate shortening procedure known as "partial capitate shortening" [35, 36]. Only the lunate facet of the capitate is osteotomized and shortened, leaving the scaphoid–capitate joint intact (Fig. 3.8). In our experience, carpal collapse did not progress postoperatively, with the lunate–capitate joint space maintained in most cases (Fig. 3.9) [36, 37]. Using fresh frozen cadavers, we measured the intra-articular pressure with axial load in the radioscaphoid fossa, radiolunate fossa, and ulnocarpal fossa before and after partial capitate shortening [38]. The radioscaphoid mean pressure significantly increased by an average of 39%, and the radiolunate mean pressure significantly decreased by an average of 53% (16% of the total, Fig. 3.1c), which showed a greater decompression effect than capitate shortening. The ulnocarpal mean pressure did not significantly change. We also investigated whether the effect of lunate decompression is related to the presence or absence of the lunate–hamate articulation (type I or type II lunate) after partial capitate shortening. Despite the presence or absence of lunate–hamate articulation, the radiolunate mean pressure significantly decreased, and the ulnocarpal mean pressure was unchanged. The early clinical results combined with our biomechanical data demonstrate decreased radiolunate joint loading after partial capitate shortening and suggest a role for partial capitate shortening in the treatment of Kienböck's disease. Clinically, Citlak et al. reported that partial capitate shortening appears to be an

Fig. 3.8 In partial capitate shortening, only the lunate facet of the capitate is osteotomized and shortened, and the scaphoid–capitate joint (arrowhead) remains intact

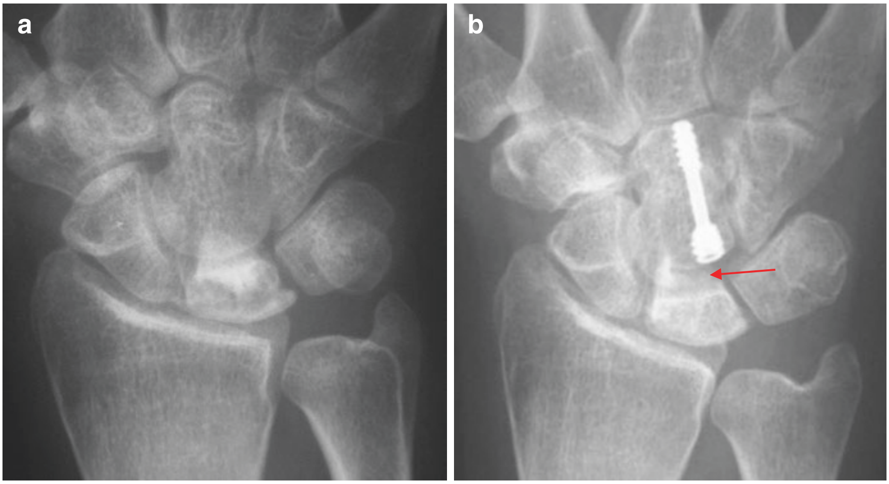


Fig. 3.9 Preoperative (**a**) and 1-year postoperative (**b**) X-rays of partial capitate shortening procedure performed for a 29-year-old woman with stage IIIA Kienböck's disease. Note that carpal collapse did not progress postoperatively with the lunate–capitate joint space maintained (arrow)

effective treatment for Lichtman stage II and IIIA patients [39]. Carpal collapse following the capitate shortening procedure seldom occurs if the articular contact between the capitate and the scaphoid is retained.

3.4.5 Intercarpal Fusions

Some intercarpal fusions also decrease the loading across the radiolunate joint. Two examples are scaphoid–trapezium–trapezoid joint fusion [25, 27, 40, 41] and scaphoid–capitate fusion [26, 41], but Werner et al. did not support the use of capitate–hamate fusion for unloading the radiolunate joint [27].

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Part III

Etiology

Etiology

4

Yuichiro Matsui

Abstract

Kienböck's disease is relatively more common in young males with occupations that require heavy use of the hands, but it sometimes occurs in younger or older individuals. Because the lunate is centrally located in the carpal bones and the inflow vessels are limited to the palmar and dorsal sides of the wrist joint, it is thought that once osteonecrosis occurs, revascularization is difficult to achieve. The disease is considered to be due to a complex combination of many factors, including repetitive minor trauma, stress concentrated on the lunate because of imbalance in radial and ulnar length, morphological abnormalities of the lunate, abnormal local vascularization, and genetic abnormalities. In this chapter, we describe in detail the representative theories about the etiology of Kienböck's disease; the arterial injury theory, fracture theory, and ulnar variance theory. The arterial injury theory remains inconclusive. The fracture theory is now considered incorrect. The ulnar variance theory has gradually declined. However, investigations of the theory led to more mechanical studies of the wrist, and it is continued to have had great influence for many years as a supporting theory for joint levelling procedures for Kienböck's disease.

Keywords

Arterial injury · Fracture · Ulnar variance

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Kienböck's disease is more common in relatively young males with occupations that require heavy use of the hands, but it sometimes occurs in younger or older individuals. Because the lunate is centrally located in the carpal bones and the inflow vessels are limited to the palmar and dorsal sides of the wrist joint, it is thought that once osteonecrosis occurs, revascularization is difficult to achieve. The disease is considered to be due to a complex combination of many factors, including repetitive minor trauma, stress concentrated on the lunate because of imbalance in radial and ulnar length, morphological abnormalities of the lunate, abnormal local vascularization, and genetic abnormalities. Representative theories [1] are discussed in the following section.

4.1 Arterial Injury Theory

In 1955, Grettve et al. reported on the distribution of arteries within the carpal bones [2]. By contrast injections into seven cadaver forearm arteries, they confirmed that nutrient arteries entering the lunate penetrate into the bone mainly from non-cartilaginous areas such as the dorsal and volar aspects.

In 1980, Gelberman et al. reported on arterial travel within and outside the lunate [3]. They injected latex into the arteries of 35 fresh-frozen limbs and showed that two to three arteries entered from the dorsal side of the lunate and three to four arteries entered from the palmar side and anastomosed within the lunate. They classified them into three types: type Y (59%), type X (10%), and type I (31%) (Fig. 4.1).

Panagis et al. investigated the intrabony vascular anatomy of the carpal bones using 25 fresh cadaver limbs [4]. Their study showed that if there was only one artery flowing into the lunate (20%), there was a possibility of avascular necrosis of the bone. However, in 2007, Lamas et al. injected latex into 27 cadaver upper limbs to investigate the distribution and anastomosis of arteries in the lunate [5]. Moreover, they examined the number and size of the feeding holes through which these vessels entered. In all specimens, the lunate was consistently supplied by both dorsal and palmar arteries. The nutrient vessels to the lunate were more numerous on the palmar side than on the dorsal side, and they were observed to enter the bone from the

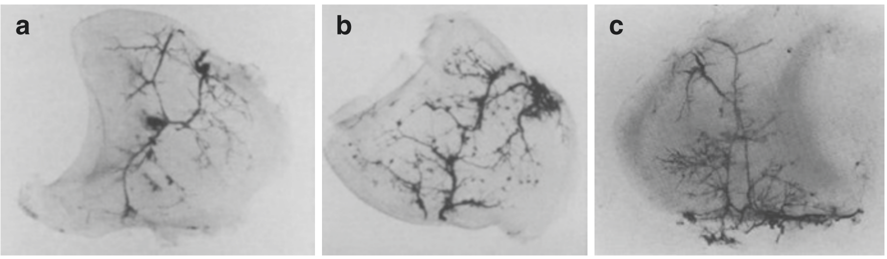

Fig. 4.1 Arterial pathways within the lunate. Reproduced from [2] with permission. (**a**) The most common intraosseous vascular pattern-the "Y" pattern. (**b**) The "I" interosseous vascular pattern is formed by single major dorsal and volar vessels. (**c**) The "X" interosseous vascular pattern formed by two dorsal and two volar vessels anastomosing in the midportion of the lunate

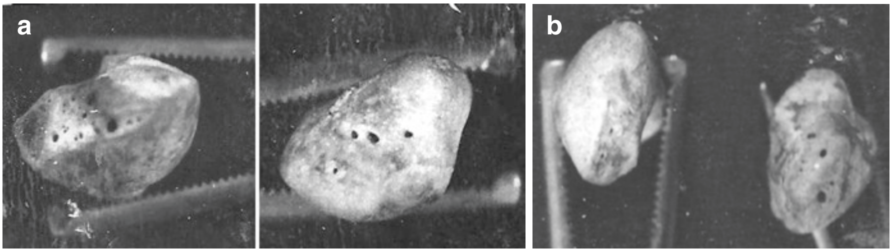

Fig. 4.2 Foramina on surface of the lunate. Reproduced from [5] with permission. (**a**) Multiple foramina on palmar surface of the lunate. (**b**) Foramina on dorsal surface of the lunate

insertions of various ligaments, including the ligament of Testut–Kuentz (radioscapholunate ligament), the dorsal carpal ligament, and the ulnar lunate triangular ligament. In the pathogenesis of Kienböck's disease, acute or chronic, traumatic or nontraumatic injury of the ligaments supporting the vessels may play an important role in the appearance of lunate necrosis.

In 2011, Dubey et al. [6] reported the number of arteries entering the lunate. More than two vascular foramina were found in 91.33% of cadaveric specimens examined (Fig. 4.2). From the palmar side, 100% of the radial artery branches, 72.22% of the anterior interosseous artery branches, and 69.4% of the branches from the arterial intercarpal arch entered the lunate. From the dorsal side, 100% of the radial artery branches, 85.71% of the anterior interosseous artery branches, and 50% of the branches from the dorsal intercarpal arch entered the lunate. They considered that there was abundant arterial flow within the lunate and that it was unlikely that arterial occlusion would cause osteonecrosis of the bone. The blood supply of the lunate, along with various ligaments, can be disrupted by trauma or strain, leading to avascular necrosis. The group concluded that the blood supply of the lunate was higher than in other studies and may be the reason for the low incidence of Kienböck's disease in the North Indian cohort they studied. Thus, the arterial injury theory remains inconclusive.

4.2 Fracture Theory

The fracture theory suggests that a lunate fracture causes hemorrhage and thrombus formation in the bone, resulting in osteonecrosis. Because osteosclerosis appears on radiographs, it was thought—at least until the 1970s—that the aseptic necrosis of the lunate was caused by impaired blood flow associated with the fracture. In 1988, Teisan et al. reported on the radiographic images of 17 patients with lunate fractures [7]. They classified the fractures into five types based on the location of the fracture, and the number of cases belonging to each type was investigated (Fig. 4.3, Table 4.1). Most of the fractures were chip fractures, which occurred near the palmar or dorsal aspects. Based on the results, they demonstrated that diagnosed and treated lunate chip fractures did not lead to Kienböck's disease. With the development of magnetic resonance imaging [8, 9], the diagnosis of early-stage Kienböck's disease became

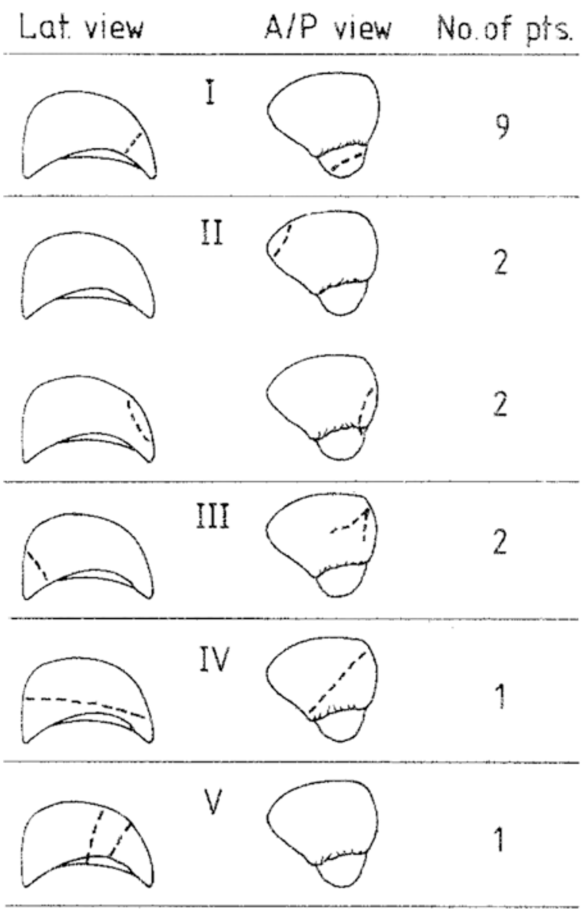

Fig. 4.3 Drawing of the five patterns of lunate fracture, with the number of patients affected (Reproduced from [6] with permission)

Table 4.1 The follow-up radiological examination revealed that none of these 11 patients had developed Kienbock's disease (Reproduced from [6] with permission)

Group I: Fracture of the volar pole of the lunate, possibly affecting the volar nutrient artery (Fig. 4.2).
Group II: Chip fracture which does not affect the main blood supply (Fig. 4.3).
Group III: Fracture of the dorsal pole of the lunate, possibly affecting the dorsal nutrient artery (Fig. 4).
Group IV: Sagittal fracture through the body of the lunate (Fig. 5).
Group V: Transverse fracture through the body of the lunate (Fig. 6).
The follow-up radiological examination revealed that none of these 11 patients had developed Kienbock's disease.

more accurate, and it was found that there were no fracture findings on radiographs, computed tomography images, or magnetic resonance images in Stage I disease. Therefore, it is now recognized that fracture of the lunate does not cause Kienböck's disease.

4.3 Ulnar Variance Theory

In 1928, Hulten reported that ulnar variance, the difference in length between the radius and ulna in the wrist joint, was closely related to the occurrence of Kienböck's disease [10]. Normal hand radiographs of 400 patients showed neutral ulnar variance in 61%, negative ulnar variance in 23%, and positive ulnar variance in 16%. In contrast, the radiographs of patients with wrists affected by Kienböck's disease showed a neutral ulnar variance of 26%, a negative variance of 74%, and a positive variance of 0%. A worldwide survey was conducted to verify the results of this study, and there was a tendency for Kienböck's disease to occur more frequently in wrists with negative variance. It was thought that anatomical predisposition and external forces were involved in the development or progression of this disease. However, there are many patients without Kienböck's disease that display negative variance of the wrist because of thickening of the triangular fibrocartilage complex [11]. If negative ulnar variance were, by itself, sufficient to result in lunatomalacia, the condition would be more common because about 25% of normal wrists in the general population are reported to have varying degrees of negative variance [10, 12]. Moreover, Kienböck's disease does not occur frequently in patients who have undergone the Darrach procedure or ulnar shortening osteotomy [13]. From these reports, support for ulnar variance as the cause of Kienböck's disease has gradually declined. However, investigations of the theory led to more mechanical studies of the wrist [14, 15], and it is continued to have had great influence for many years as a supporting theory for joint levelling procedures such as radial shortening osteotomy [16, 17] and ulnar lengthening osteotomy for Kienböck's disease.

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Part IV

Diagnosis

History and Presentation

5

Daisuke Kawamura

Abstract

Kienböck's disease usually affects adults between 20 and 40 years of age, who are predominantly male manual workers. The condition is typically unilateral. Both sides are equally affected. The history of a specific traumatic event is frequent, months or years before the diagnosis. Symptoms of Kienböck's disease are wrist pain, swelling, and limited range of motion. These symptoms usually occur at the time of carpal collapse, probably related to the progressive alteration in carpal architecture and function rather than to bone necrosis.

In some cases, Kienböck's disease is associated with extensor or flexor tendon ruptures. Unlike rheumatoid arthritis or osteoarthritis of the distal radioulnar joint, extensor tendons of the radial digits tend to rupture in Kienböck's disease. Carpal tunnel syndrome may also occur in advanced stages.

Kienböck's disease is found not only in young adults but also in pediatric and elderly patients. The presentation and prognosis of these populations are different from those of young adult patients.

The prognosis for Kienböck's disease in childhood is good, and many cases improve with conservative treatment. It is presumed that pediatric patients are more likely to undergo revascularization and remodeling.

The exact timing of disease onset is often unclear in elderly patients. The radiographs show severe stages of Kienböck's disease at the first visit. In such patients, avascular necrosis of the lunate may have been present for decades but with a pain level insufficient to seek medical attention.

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Keywords

Symptoms · Medical history · Patient age

Symptoms of Kienböck's disease are wrist pain, swelling, and limited range of motion. Patients often feel muscle weakness due to pain.

Patients with Kienböck's disease become aware of wrist pain without high-energy trauma that causes a fracture of the lunate, as described by Robert Kienböck in 1910. Acute trauma is absent or mild, and patients often do not remember when symptoms begin. The duration of a patient's symptoms before visiting the hospital varies, and symptom intensity also varies from mild to severe or intermittent. Patients can be affected at all ages, but the typical patient is a young adult male aged 20–40 years who is often engaged in hard work. In general, the condition is unilateral, although bilateral cases have been reported [1, 2], and the sides are affected equally.

Patients experience exacerbation of pain due to activities, particularly extension and axial loading of the wrist. Patients often appear symptomatic when the lunate collapses. Patients frequently described an incident that triggered wrist pain. The incident was presumed to have triggered the collapse of the lunate in such cases. Physical examination often reveals swelling on the dorsal side of the wrist joint and tenderness consistent with the site of the lunate bone. The grip strength of the affected side is usually lower than that of the contralateral side. Both flexion and extension of the wrist joint tend to be restricted, and the pain becomes stronger when the wrist joint is fully extended. Forearm rotation is not restricted. In cases of lunate collapse, dorsal wrist swelling is more pronounced and may be accompanied by capsulitis and extensor tendon tenosynovitis [3]. Additionally, carpal tunnel syndrome may occur in advanced stages [4, 5].

The original literature by Kienböck describes that tapping on the head of the third metacarpal induces pain and that crepitus can be palpated on the lunate [6]. In recent years, articles and textbooks on Kienböck's disease often do not have such detailed descriptions. Advances in diagnostic imaging technology, such as magnetic resonance imaging (MRI), may have reduced the dependence on physical findings in the diagnosis of Kienböck's disease.

Kienböck's disease is a progressive disease that, except in very early stages, is thought to lead to collapse and fragmentation of the lunate, followed by carpal collapse (Fig. 5.1). Kieth et al. investigated the outcome of 33 patients who received conservative treatment and reported that Kienböck's disease is both clinically and radiologically progressive [7]. Lichtman et al. described the symptoms at each stage of the Lichtman classification [8]. In stage I, the clinical presentation resembles a wrist sprain, and patients complain of mild pain during their activities. Patients in stage II exhibit swelling of the wrist as a result of synovitis; these patients experience more persistent pain, which is often nocturnal. Patients in stage III complain of more constant pain as the mechanical symptoms become more apparent with the progression of carpal instability. The clinical picture is similar to that of early

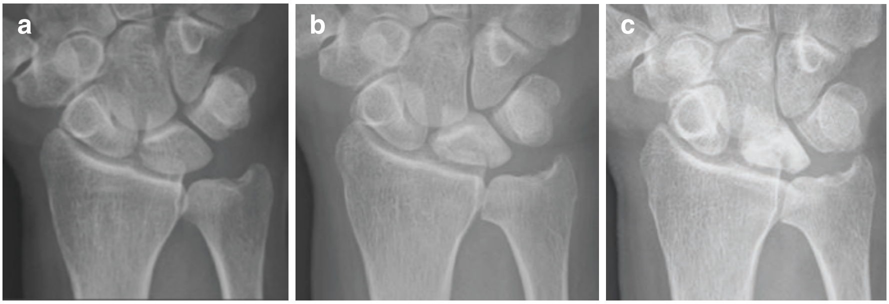

Fig. 5.1 Progression of Kienböck's disease. (**a**) A 32-year-old woman. A radiograph was taken by chance 4 years before onset. (**b**) The patient complained of mild wrist pain without acute trauma. Plain radiograph represents lunate collapse without carpal collapse. (**c**) Eight months later, the lunate further collapsed, and the capitate migrated proximally

degenerative arthritis. Patients in stage IV present wrist stiffness with constant pain and swelling. However, others have claimed that the duration of symptoms and Lichtman's stage did not correlate with pain or weakness [7]. In other words, the severity observed in radiographs does not always correlate with the severity of symptoms. As an extreme example, Taniguchi et al. reported 14 cases in which Kienböck's disease was incidentally diagnosed on radiographic examination [9]. Although the radiographic findings revealed advanced Kienböck's disease, none of the patients had symptoms that required treatment for Kienböck's disease or problems in activities of daily living or at work.

In some cases, Kienböck's disease is associated with extensor or flexor tendon ruptures (Fig. 5.2) [10–15]. Unlike rheumatoid arthritis or osteoarthritis of the distal radioulnar joint (so-called Vaughan-Jackson syndrome [16]), extensor tendons of the radial digits tend to rupture in Kienböck's disease, while ruptures can also occur in the ulnar digits [13, 14]. There have also been reports of flexor pollicis longus rupture and flexor digitorum profundus rupture of the little finger, as well as other fingers [15]. Patients with Kienböck's disease with tendon rupture tend to be older, with more advanced stages of Kienböck's disease, but often do not present with pain. As a result, these patients are not diagnosed or treated for Kienböck's disease and seek medical assistance only when they lose normal movement of the fingers [12].

Kienböck's disease is found not only in young adults but also in pediatric and elderly patients. The presentation and prognosis of these populations are different from those of young adult patients.

Elderly women are more likely to have Kienböck's disease. Taniguchi et al. compared 15 patients (two stage I, three stage II, nine stage III, and one stage IV) who developed Kienböck's disease after 50 years of age, with 30 patients under 40 years of age, and age-matched controls. The radiological findings demonstrated that the negative ulnar variance on the affected side was less than that on the unaffected side; however, the degree of negative ulnar variance in elderly patients was not as great as that in young patients [17].

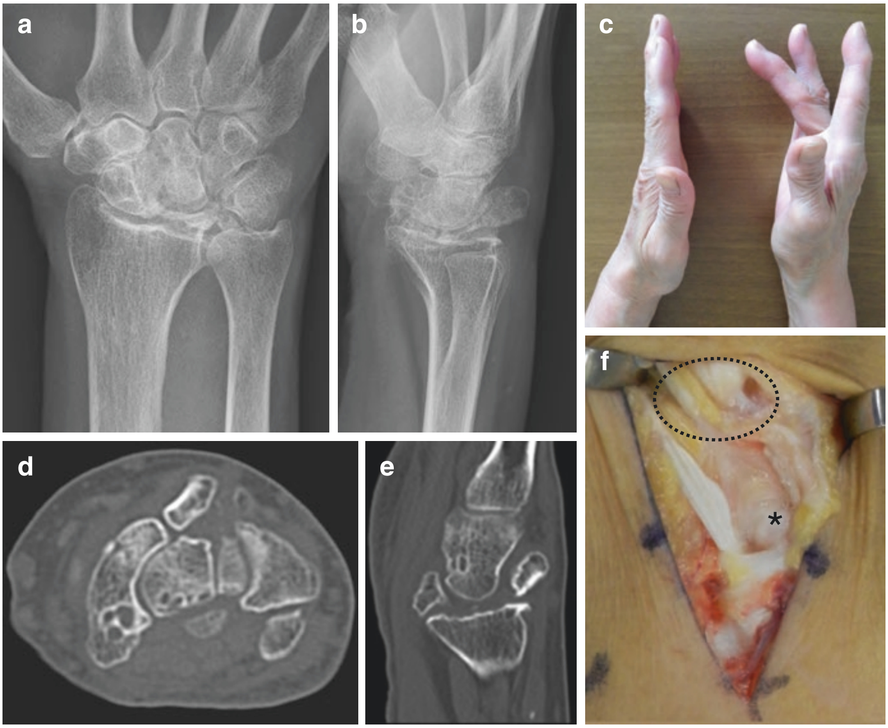

Fig. 5.2 Kienböck's disease with extensor tendon rupture. (**a**) A 69-year-old woman visited our hospital complaining of difficulty extending her right middle finger. (**b**, **c**) Plain radiograph exhibited fragmentation of the lunate. (**d**, **e**) Computed tomography images revealed one of the fragments of the lunate protruding dorsally. (**f**) Extensor indicis proprius and extensor digitorum communis of the middle finger were ruptured (dotted circle). The dorsal capsule was ruptured by the dorsal fragment of the lunate (asterisk)

Several reports have suggested an association between Kienböck's disease in the elderly and osteoporosis, but no clear association has yet been clarified [17, 18].

The exact timing of disease onset is often unclear in older patients with radiographic findings of severe stages of Kienböck's disease at the first visit. In such patients, avascular necrosis of the lunate may have been present for decades but with a pain level insufficient to seek medical attention [18]. During the medical interview, it was important to confirm the profession of the patients when they were young and to ask them if they had ever experienced severe wrist pain.

Kienböck's disease has also been reported in pediatric patients. The youngest report of Kienböck's disease was in a 6-year-old boy [19]. Kienböck's disease in pediatric patients presents as wrist pain, dorsal tenderness, synovitis, limited range of motion, and reduced grip strength, similar to the presentation in young adult cases. Because ossification of the ulnar head is often incomplete in pediatric patients, it is difficult to accurately measure ulnar variance. Therefore, the importance of negative ulnar variance in the development of Kienböck's disease in pediatric patients is unclear.

There are also cases of Kienböck's disease that are considered to be related to sports in young patients [20]. Therefore, sports activities or repetitive forceful exercises may contribute to the development of Kienböck disease in pediatric patients.

The prognosis for Kienböck's disease in childhood is good, and many cases improve with conservative treatment (splinting or discontinuation of sports activity). Moreover, revascularization of the lunate has been confirmed by radiographs or MRI images [21]. It is presumed that pediatric patients are more likely to undergo revascularization and remodeling. If conservative treatment fails, even in pediatric patients, surgical treatment is selected, and the clinical results are generally good (Fig. 5.3).

Irisarri et al. subdivided pediatric Kienböck's disease into infantile (12 years and younger) and juvenile (13 years to skeletal maturity) types. While the infantile group showed excellent outcomes with nonoperative treatment, 30% of the patients in the juvenile group had disease progression and required surgery [21]. In many articles, conservative treatment has been successful in patients under 12 years of age, while surgical treatment tends to be selected in patients over 13 years of age.

Kienböck's disease may be associated with other medical and anatomical variations. Indeed, Lanzer et al. reported a case of Kienböck's disease with sickle cell anemia, suggesting that bone infarction may have occurred [22]. Other studies have reported patients with cerebral palsy presenting with Kienböck's disease; patients with cerebral palsy have abnormally flexed wrists, which result in high pressure against the lunate. Therefore, repeated minor trauma caused by the wrist position is thought to be a developmental cause of Kienböck's disease in cerebral palsy [23, 24]. Kienböck's disease is also observed in patients with systemic sclerosis, which is speculated to be associated with arterial vasculopathy [25]. Other diseases, such as systemic lupus erythematosus or Crohn's disease, have been reported in patients with Kienböck's disease, and it is speculated that the use of

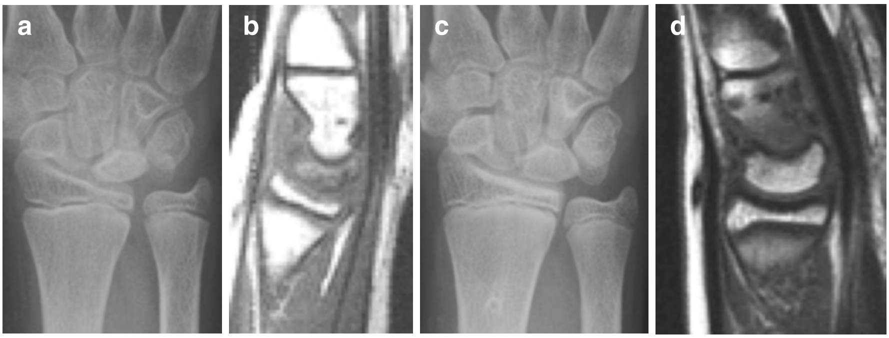

Fig. 5.3 Kienböck's disease in an 11-year-old patient. (**a**) Plain radiograph representing sclerotic changes in the lunate. (**b**) Magnetic resonance imaging (MRI) of the diseased wrist. On a T1-weighted image, the lunate demonstrated diffuse low signal intensity. The patient underwent radial shortening osteotomy. Two years after the surgery, the lunate showed its normal contour radiographically (**c**). (**d**) MRI showed a normal lunate signal (diffuse high signal on a T1-weighted image)

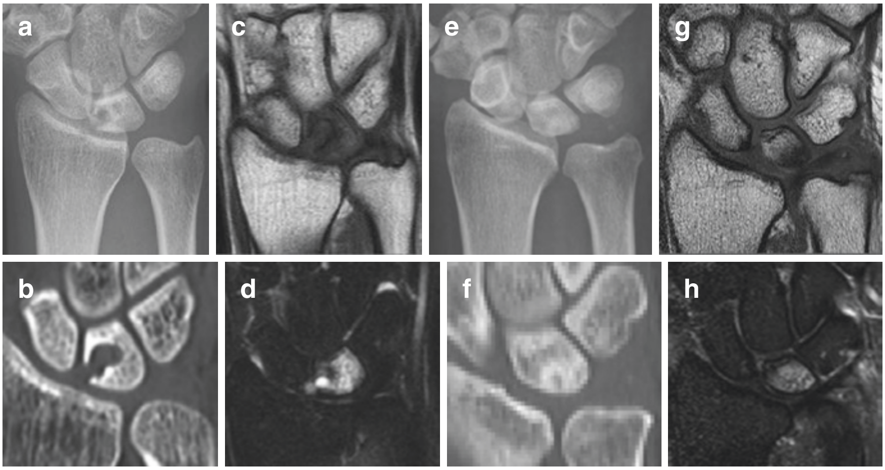

Fig. 5.4 Differential diagnosis for Kienböck's disease. (**a**–**d**) A 27-year-old woman with an intraosseous ganglion of the lunate. A, B: A plain radiograph and computed tomography (CT) revealed an intraosseous cystic lesion of the lunate. (**c**) On a T1-weighted image, the lunate demonstrated diffuse low signal intensity. (**d**) On short-tau inversion recovery (STIR) MRI, the lunate demonstrated diffuse high signal intensity. (**e**–**h**) A 43-year-old woman with ulnocarpal abutment. (**e**, **f**) The ulnar side of the proximal surface of the lunate exhibited sclerotic changes and subchondral cysts. (**g**) On a T1-weighted image, the lunate demonstrated partially low signal intensity. (**h**) On STIR MRI, the lunate ultimately demonstrated high signal intensity. The pathology is limited to the proximal ulnar aspect of the lunate in the ulnocarpal abutment

steroids is involved in the onset of Kienböck's disease. Kienböck's disease has also been reported in dialysis treated patients, and bone fragility caused by dialysis treatment or arteriovenous fistula placement is speculated to be associated with its pathology [26, 27]. Moreover, carpal coalitions (capitate–hamate, lunate–triquetrum), which are anatomical variations, have been reported to coexist in patients with Kienböck's disease [28–30].

The differential diagnosis for Kienböck's disease includes occult dorsal wrist ganglia [31], inflammation or degenerative arthritis [32], and osteoid osteoma of the lunate (Fig. 5.4) [33, 34].

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Imaging

6

Daisuke Kawamura

Abstract

Since the first report by Robert Kienböck himself, the diagnosis of Kienböck's disease has usually been made on plain radiographs. Radiographs show different lunate findings as the disease progresses. In early lesions, the lunate shows a normal architecture and bone density, whereas as the disease progresses, it shows osteosclerosis, fragmentation of the lunate, and abnormal carpal arrangement. Radiographs are also important in determining the associated anatomic and mechanical properties of the involved wrist, such as ulnar variance, radial inclination, carpal height, radioscaphoid angle, and lunate size or shape.

Magnetic resonance imaging (MRI) is helpful early in the disease when plain radiographs may not reveal any abnormalities. On T1-weighted images, the lunate demonstrates diffuse low signal intensity as a result of decreased vascularity. T2-weighted images may reveal high or low signal intensity, depending on the extent of the disease process. It is critical to consider that this diffuse signal change within the entirety of the lunate is necessary to establish the diagnosis of Kienböck disease.

Computed tomography (CT), especially sagittal reconstruction views, is helpful in assessing the extent of articular surface collapse and the presence of fractures. CT sagittal views are also helpful in detecting the presence of carpal instability as well as early osteoarthritis of the radiocarpal and midcarpal joints.

Staging of Kienböck disease depends primarily on the radiographic findings. Staging is an important step for planning the treatment by the stage of disease. The classification system described by Lichtman et al. is used most commonly.

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Keywords

Imaging · Radiography · Magnetic resonance imaging · Staging

6.1 Standard Radiographs

Robert Kienböck, a radiologist, published his renowned article "Über traumatische Malazie des Mondbeines und ihre Folgezustände: Entartungsformen und Kompressionsfrakturen," which appeared in Fortschritte auf dem Gebiet der Roentgenstrahlen in 1910 [1]. The article presented 16 cases showing "traumatic malacia of the lunate," including clinical and radiographic findings. This article was published only 13 years after the discovery of X-rays by Conrad Roentgen, and since then, the diagnosis of Kienböck's disease has usually been made on plain radiographs.

Radiographs show different lunate findings as the disease progresses. In early lesions, the lunate shows a normal architecture and bone density, whereas as the disease progresses, it shows osteosclerosis, fragmentation of the lunate, and abnormal carpal arrangement.

A diligent search should be performed for evidence of compression fracture or changes in the relative density of the lunate in patients with acute or chronic wrist pain. Radiographs are also important in determining the associated anatomic and mechanical properties of the involved wrist, such as ulnar variance, radial inclination, carpal height, radioscaphoid angle, and lunate size or shape. First, obtaining adequate posteroanterior (PA) radiographs of the wrist is important for accurate and reproducible measurements. Lichtman et al. emphasized the importance of taking adequate radiographs in their article describing the use of the Lichtman classification [2] in the measurement of ulnar variance changes with forearm rotation, wrist deviation, and the X-ray beam incidence angle [3–6].

A standard PA radiograph is taken with standard positioning of the arm; the wrist and the forearm are in the neutral position, the elbow flexed 90°, and the shoulder abducted to 90° [3]. Typical measurements and their clinical significance are described below.

6.1.1 Ulnar Variance

Since Hulten's report in 1928, the association between negative ulnar variance and Kienböck's disease has been investigated. Several methods are used to measure ulnar variance (Fig. 6.1).

Gelberman et al. measured ulnar variance using the so-called "project a line technique [7], in which a line is drawn from the ulnar side of the articular surface of the distal radius toward the ulna (Fig. 6.1a). The variance is defined as the distance between the line and the distal end of the ulna. Although several authors have followed this technique [8], anatomical landmarks to draw the line were not described in the original article by Gelberman.

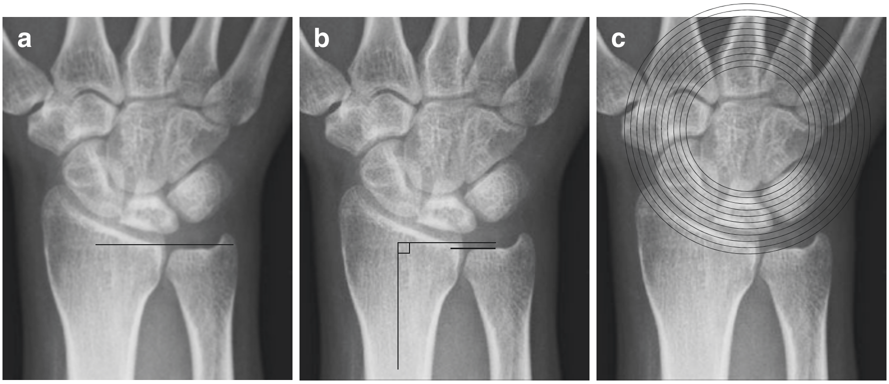

Fig. 6.1 Methods of ulnar variance measurements. (**a**) The project a line technique, (**b**) Method of perpendiculars, (**c**) Concentric circle technique

Coleman et al. reported another method in [9]; this method involves drawing a line perpendicular to the longitudinal axis of the radius through the distal ulnar aspect of the radius, following which the distance between the line and cortical rim of the ulnar head is measured (Fig. 6.1b).

Palmer et al. reported ulnar variance measurement with a transparent plastic template marked with concentric semicircles of varying radii [10]. The template had a selection of radii ranging from 20 to 50 mm in 1-mm increments. The template curve that most closely approximated the concavity of the distal radial sclerotic line was chosen. The number of radii separating this template from the cortical rim of the ulnar head represents the amount of ulnar variance in millimeters.

Yoshida et al. modified Palmer's measurement [11] by using the circumferential template to the lunate fossa of the radius and not to the whole distal joint surface of the radius (Fig. 6.1c).

Steyers et al. compared the three methods of measuring ulnar variance [12] and found all of them to be highly reliable. These findings suggest that the technique may be selected at the discretion of the clinician when measuring ulnar variance.

Several authors have highlighted that ulnar variance differs between males and females and increases with advancing age [5, 13]. Studies have also reported an association between negative ulnar variance and Kienböck's disease [7, 14, 15], although several other studies have rejected this hypothesis [4, 5, 16, 17].

6.1.2 Radial Inclination

The radial inclination was measured as the angle between a line from the ulnar side of the carpal surface of the radius to the tip of the radial styloid and a line perpendicular to the axis of the radius or the ulna [8, 18].

Some authors claim that a flatter radial inclination may predispose individuals to Kienböck's disease [8, 18]. Mirabello et al. reported a correlation between the slope of the distal radial articular surface and the age of onset [19].

6.1.3 Carpal Height Ratio

The Carpal Height Ratio (CHR) is calculated as the distance from the proximal end of the third metacarpal to the radial articular surface divided by the length of the third metacarpal [20]. The CHR declines in advanced stages after Lichtman IIIb, suggesting the presence of carpal instability.

6.1.4 Radioscaphoid Angle

The long axis of the scaphoid is determined with a line tangential to the palmar outline of the scaphoid using the technique of Gilula and Weeks from the lateral view of the wrist radiographs [21]. The axis of the radius is determined by a dorsal cortex line through the radius or drawn through the center of the radius at 2 and 5 cm proximal to the joint [18, 22]. Condit et al. found that the preoperative radioscaphoid angle correlated best with clinical outcomes [23].

6.1.5 Size or Shape of the Lunate

Together with ulnar variance and radial inclination, lunate morphology is also considered to be a mechanical factor of Kienböck's disease. Several reports have described that the height and diameter of the lunate are smaller in patients with Kienöck's disease than in normal controls [8, 18]. It is presumed that these anatomical features may result in greater load transmission onto the lunate, which may lead to avascular necrosis.

Antuno-Zapico classified lunates into three types according to the angle between the lateral scaphoid and the proximal radial sides of the lunate (Fig. 6.2) [24]. In type I, the angle is more than 130° (Fig. 6.2a); in type II, the angle is approximately 100° (Fig. 6.2b); and in type III, there are two distinct facets on the proximal surface that articulate with the radius and the triangular fibrocartilage (Fig. 6.2c). Antuno-Zapico described the association of ulnar negative variance in wrists with type I lunate and ulnar neutral or positive variance in type II and III lunates and theorized that type I lunates are the weakest to compressive stress.

Viegas et al. classified lunate morphology into two types based on the absence (type I) (Fig. 6.2d) or presence (type II) of a medial hamate facet (Fig. 6.2e) [25]. Ulnar variance, age, sex, and side were not correlated with the presence or absence of a medial hamate facet on the lunate.

Tatebe et al. reported that the prevalence of type 2 lunate in Kienböck's disease was much lower (28%) than that described in the report by Viegas et al. (65.5%) [26]. They presented data implying a relationship between the lunate type and Kienböck's disease. In contrast, Tsuge et al. reported that the medial facet was

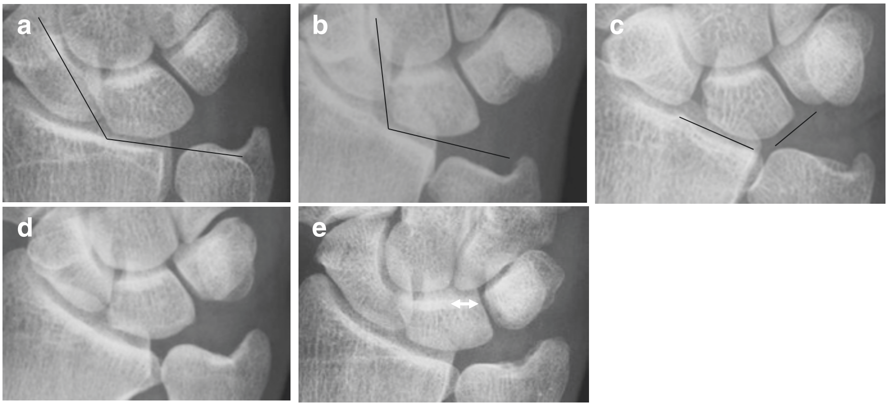

Fig. 6.2 Classification of lunate morphology. (**a**–**c**) Morphologic classification by Antuno-Zapico, (**a**). Type I, (**b**) Type II, (**c**) Type III. (**d**, **e**) Morphologic classification by Viegas, (**a**) Type I, (**b**) Type II (white arrow: medial facet)

present in 37% of the Kienböck group and 42% of the normal controls; however [18], there was no significant difference between the two groups in terms of the incidence of this medial facet.

Rhee et al. studied the effect of lunate type on the radiographic characteristics of patients with Kienböck's disease [27]. They concluded that lunate morphology may affect the severity of Kienböck's disease at the time of initial presentation. Type II lunates appear to be protective against coronal and scaphoid flexion deformities.

6.2 Magnetic Resonance Imaging

Magnetic Resonance Imaging (MRI) is helpful early in the disease when plain radiographs may not reveal any abnormalities. On T1-weighted images, the lunate demonstrates diffuse low signal intensity as a result of decreased vascularity. T2-weighted images may reveal high or low signal intensities depending on the extent of the disease process. It is critical to consider that this diffuse signal change within the entirety of the lunate is necessary to establish a diagnosis of Kienböck's disease (Fig. 6.3c, f, i). Other pathologic conditions, such as ulnocarpal abutment (in which the pathology is limited to the proximal ulnar aspect of the lunate), fractures, and tumors, demonstrate abnormal signals localized to a specific area.

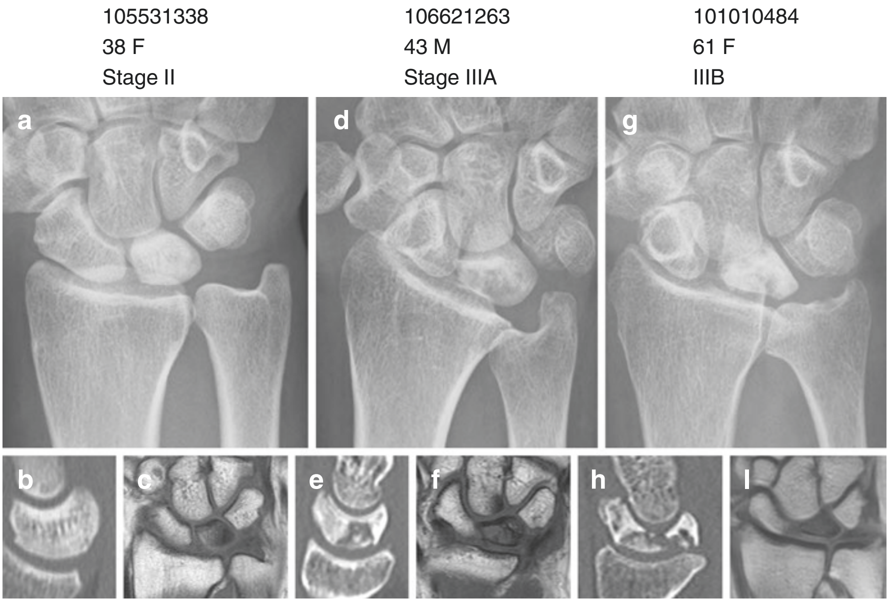

Fig. 6.3 Typical imaging findings of Kienböck's disease. (**a**–**c**) A 38-year-old woman with stage II disease. (**d**–**f**) A 43-year-old man with stage IIIA disease. (**g**–**i**) A 61-year-old woman with stage IIIB disease. (**a**, **d**, **g**) plain radiographs. (**b**, **e**, **h**) Sagittal reconstruction views of computed tomography, (**c**, **f**, **i**) T1-weighted images of magnetic resonance imaging)

6.3 Computed Tomography

Computed Tomography (CT), especially sagittal reconstruction views, is helpful in assessing the extent of articular surface collapse and the presence of fractures (Fig. 6.3b, e, h). CT sagittal views are also helpful in detecting the presence of carpal instability as well as early osteoarthritis of the radiocarpal and midcarpal joints.

Friedman et al. reported that direct coronal CT of the wrist is more sensitive than plain radiographs [28].

Schmitt et al. found that 24 out of 37 patients had a higher stage on CT than on radiographs. CT enables earlier and more extensive detection of cystic lesions in the lunate or shell-formed fractures at the proximal pole of the lunate [29].

Mohan et al. measured the width and height ratio of the lunate in the sagittal planes on a CT scan [30]. They reported that the width/height ratio correlates with carpal height and found that lunate collapse precedes the reduction in the carpal height ratio in some patients and concluded that this ratio is a better measure of lunate collapse.

6.4 Staging

Staging or classification is an important step in the evaluation of patients with Kienböck's disease because structural and kinematic alterations appear to have a significant effect on treatment outcomes. Furthermore, staging is important for accurately comparing the clinical results of different treatment options. The surgical management options for Kienböck's disease are dictated by the disease stage.

Ståhl described a radiographic and pathologic classification system for Kienböck's disease in 1947 [31]. Decoulx et al. also proposed his renowned classification system in 1957 [32]. Lichtman et al. modified Stahl's original classification in 1977 [2]. The classification scheme described by Lichman et al. is most widely used and has evolved with time [33–35]. This classification system is based on radiographic and clinical findings rather than on surgical or pathologic examination of the lunate (Table 6.1).

Lichtman's first classification in 1977 was composed of four stages according to the structural alteration of the lunate and the surrounding carpal bones. Stage I indicates normal architecture and density of the lunate on plain radiographs. A linear fracture line in the lunate may be present, but lunate collapse is not observed. In an original article in 1977, Lichtman stated that a bone scan may be abnormal in stage I disease. However, it has fallen out of favor since the introduction of MRI. MRI is an ideal imaging technique for stage I. Uniformly decreased signal intensity in both T1- and T2-weighted images in comparison with the neighboring bones reflects the loss of vascularity. An increased signal on T2 may represent revascularization [35].

In stage II, the lunate demonstrates definite sclerotic changes on plain radiographs, with no alteration in the size or shape of the lunate. Additionally, one or more fracture lines may be present.

In stage III, the entire lunate collapses. Later, stage III is divided into stages IIIA and B [33]. In stage IIIA, lunate collapse occurs, but carpal height and alignment remain normal. Stage IIIB has lunate collapse with fixed scaphoid rotation, proximal capitate migration, and loss of carpal height. As the carpal height ratio decreases, the lunate collapses, and the capitate migrate proximally. Scaphoid rotation produces a dorsal intercalated segment instability (DISI) pattern of carpal instability. In stage IV, continued carpal collapse is related to arthritic changes in the radiocarpal and midcarpal joints. Radiographs show subchondral sclerosis with joint space narrowing, osteophyte formation, and degenerative cysts.

Table 6.1 Lichtman osseous classification of Kienböck's disease [36] (引用番号)

Stage	Radiographic findings	MRI findings
I	Normal	T1: decreased, T2: variable
II	Lunate sclerosis	T1: decreased, T2: variable
IIIA	Lunate collapse + normal carpal alignment	T1: decreased, T2: variable
IIIB	Lunate and carpal collapse +	T1: decreased, T2: usually decreased
IIIC	Lunate collapse + a coronal fracture/ fragmentation of the lunate	T1: decreased, T2: variable
IV	Lunate collapse + radiocarpal/midcarpal arthritis	T1: decreased, T2: decreased

Stages 0 and IIIC were included in the latest version of the classification [34]. Stage 0 represents the onset of lunate ischemia; patients in stage 0 may have a normal MRI. Lichtman himself described that stage 0 is hypothetical. Therefore, there is no specific test to diagnose stage 0 objectively. Stage IIIC is defined as a complete coronal plane split, regardless of the lunate or wrist morphology. This modification was made because patients with a complete coronal split of the lunate tend to have a poor prognosis. Lichtman himself described the classification as the Lichtman osseous classification in his latest review and did not include stage 0 [36].

The reliability or the reproducibility of the Lichtman classification has been studied in several reports [37, 38]. Jensen et al. investigated the reliability of the unmodified four-stage Lichtman classification with four observers for 76 radiographs [37]. They reported reliability kappa values ranging from 0.45 to 0.52 and reproducible kappa values ranging from 0.26 to 0.63. Jafarnia et al. also investigated the reliability of a nonmodified four-stage Lichtman classification with four observers [38]. They reported reliability kappa values ranging from 0.66 to 0.74 and reproducible kappa values ranging from 0.72 to 0.82. There was no statistically significant difference in reliability or reproducibility among the observers with different amounts of experience in hand surgery. Goldfarb et al. proposed a modification to the classification system in which stage IIIB was defined as a radioscaphoid angle greater than 60° [22]. With this modification, interobserver reliability increased from a kappa value of 0.63–0.75. Goeminne et al. also reported good reliability and reproducibility of the classification [39]. They reported reliability kappa values of 0.52–0.77 and reproducible kappa values of 0.72–0.90 among four observers who reviewed radiographs from 70 patients. Contrary to the above three studies, Aydemir et al. reported poor reliability among 10 residents, 10 orthopedic surgeons, and 10 hand surgeons [40]. They reported a reliability kappa value of 0.203 within all of the observers. They concluded that the Lichtman classification by plain radiographs alone is insufficient and should be supported by other imaging and measurement techniques.

Schmitt et al. reported how gadolinium perfusion enhanced T1-weighted fast spin–echo (FSE) fat-saturated sequences [29]. Gadolinium enhancement assists with distinguishing necrotic parts from neovascular repair tissue with high signal. They classified the lunate signal alterations after administration of intravenous gadolinium contrast into four stages: MRI stage N, normal; MRI stage A, ischemic (viability maintained); MRI stage B, partially necrotic (viability partially lost); and MRI stage C, completely necrotic.

Bain et al. reported an arthroscopic assessment and classification of Kienöck’s disease [41], in which articular surfaces of the lunate were diagnosed as functional or nonfunctional arthroscopically. A functional articular surface has a smooth appearance and is firm to palpation, while a nonfunctional articular surface has at least one of the following findings: extensive fibrillation, fissuring, localized or extensive loss, a floating articular surface, or fracture.

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Part V

Treatment and Outcome

7 Treatment Strategy

Norimasa Iwasaki

Abstract

Although the etiology of Kienböck's disease is not fully understood, it has been associated with potential risk factors, including anatomic, mechanical, and traumatic causes. The principal purpose of treatments for Kienböck's disease is to reduce or eliminate these risk factors to achieve direct or indirect revascularization of the diseased lunate. A variety of treatment options have been introduced in the clinical field. Direct revascularization techniques mainly include pedicled or vascularized bone grafting. After sequestrectomy of the diseased lunate, the defect is filled with viable bone with a native blood supply. Direct revascularization techniques are generally applied to treat Lichtman's stage II and IIIA Kienböck's disease. To unload the lunate, radial osteotomy or capitate shortening is often performed simultaneously. Indirect techniques aim to achieve revascularization by reducing excessive force on the lunate. The indirect techniques include radial shortening or ulnar lengthening, open or closing radial wedge osteotomy, capitate shortening, limited intercarpal fusion, and core decompression. Direct or indirect revascularization techniques are not indicated for patients with lunate fragmentation or a severely collapsed lunate. Such patients generally undergo lunate excision with or without replacement arthroplasty for symptomatic relief as an alternative to revascularization techniques. Salvage procedures, such as proximal row carpectomy or total wrist fusion, are also proposed for patients with unsuccessful revascularization or for those with advanced disease with osteoarthritic changes in the wrist. The treatment target for stage IV is not only the diseased lunate but also radiocarpal or midcarpal osteoarthritis. Salvage procedures for symptomatic relief are indicated in stage IV.

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Treatment strategy · Direct revascularization technique · Indirect revascularization technique · Lichtman's stage

Kienböck's disease is defined as avascular necrosis of the lunate. The etiology of this disorder is not fully understood but has been associated with potential risk factors, including anatomic, mechanical, and traumatic causes. The principal purpose of treatments for Kienböck's disease is to reduce or eliminate these risk factors to achieve direct or indirect revascularization of the diseased lunate. A variety of treatment options have been introduced in the clinical field.

Direct revascularization techniques mainly include pedicled or vascularized bone grafting. After sequestrectomy of the necrotic lunate, the defect is filled with viable bone with a native blood supply. The distal radius is often chosen as a donor site [1, 2]. The bone graft is usually harvested from the dorsal radius 1 cm proximal to the radiocarpal joint. The pedicle is supplied by the fourth and fifth extracompartmental arteries, which are branches of the posterior division of the anterior interosseous artery. Recently, Bürger et al. [3] recommended a vascularized osteochondral graft from the medial femoral trochlea, mainly for stages II and IIIA. The periosteal vascular branches of the descending geniculate artery or superomedial genicular arteries supply blood to the bone graft. The considerable advantages of this procedure are the restoration of the vascularity of the lunate and replacement of the articular cartilage of the radiocarpal articulation. Direct revascularization techniques are generally applied to treat stage II and IIIA Kienböck's disease. To unload the lunate, radial osteotomy [2, 4–6] or capitate shortening [2, 6, 7] is often performed simultaneously.

Indirect techniques aim to achieve revascularization by reducing excessive force on the lunate. The indirect techniques include radial shortening or ulnar lengthening (referred to as joint leveling procedures), open or closing radial wedge osteotomy, capitate shortening, limited intercarpal fusion, and core decompression. Radial shortening is often performed for negative ulnar variance, while capitate shortening is often performed for positive or neutral ulnar variance. Regarding the effects of radial shortening on the diseased lunate, one study found that performing distal radius osteotomy without modifying the radial length or inclination for Kienböck's disease decreased pain and improved grip strength and wrist motion at 10 or more years postoperatively [8]. This suggests that radial osteotomy near the lunate may induce an increase in regional blood flow, improving the clinical symptoms. It seems reasonable to conclude that radial osteotomies have biomechanical [9–12] and biological [8] effects on revascularization of the diseased lunate.

Direct or indirect revascularization techniques are not indicated for patients with lunate fragmentation or a severely collapsed lunate. Such patients generally undergo lunate excision with or without replacement arthroplasty for symptomatic relief as an alternative to revascularization techniques [13–24]. Salvage procedures, such as proximal row carpectomy [25–30] or total wrist fusion [17, 31, 32], are also proposed for patients with unsuccessful revascularization or for those with more advanced disease with osteoarthritic changes in the wrist.

Kienböck's disease is widely staged in accordance with Lichtman's radiologic classification [16, 17, 32]. This classification basically consists of four stages (Table 7.1). Stage III is further divided into IIIA (lunate collapse without fixed scaphoid rotation) and IIIB (lunate collapse with fixed scaphoid rotation and other secondary derangements). In addition, Lichtman et al. [33] proposed stage IIIC, which is defined as lunate collapse with a coronally oriented lunate fracture. Bain and Begg [34] advocated an alternate classification system for Kienböck's disease focusing on the number of nonfunctional articular surfaces of the lunate at arthroscopy, which is useful for surgeons. Although the treatment strategy for Kienböck's disease is still controversial, the choice of treatment offered to each patient depends mainly on the Lichtman's stage and ulnar variance.

Table 7.2 presents the treatment options for Kienböck's disease in accordance with Lichtman's stage. Plain radiographs of stage I show normal architecture and density of the lunate, except for either a linear or a compression fracture. Stage I Kienböck's disease is typically treated with immobilization to unload the lunate, encouraging revascularization. This can be achieved using simple cast fixation for approximately 3 months. Children or teenagers have a much better prognosis for revascularization than adults [35].

In stage II, plain radiographs demonstrate definite density changes in the lunate relative to other carpal bones. The size, shape, and anatomic relationships of the bones are not apparently altered, which indicates the possibility of lunate revascularization. Therefore, treatment options for stage II aim to achieve direct or indirect revascularization of the lunate. In patients with ulnar minus variance, the author mainly performs radial shortening as a joint leveling procedure. When neutral or ulnar positive variance is found, the author prefers to perform capitate shortening to unload the lunate while preventing ulnocarpal abutment. Alternatively, direct revascularization procedures can be performed for stage II Kienböck's disease. The author considers direct revascularization for patients with ulnar neutral or positive variance.

Since the first description by Lichtman, the definition of stage III has been altered. The original definition of stage III includes the entire lunate collapse with secondary proximal migration of the capitate and disruption of the carpal architecture [16]. As mentioned above, Lichitman et al. [17] divided stage III into IIIA and IIIB. Based on the radiographic parameters, stages IIIA and IIIB are defined as the presence of lunate collapse with radioscaphoid angles of less than 60° and greater

Table 7.1 Lichtman radiographic classification of Kienböck's disease [16, 17, 32]

Stage	X-ray findings
Stage I	Normal except for the possibility of either a linear or a compression fracture
Stage II	Definite density changes
Stage III	Entire lunate collapse
Stage IV	Pancarpal arthritis

Table 7.2 Author's recommended treatment options for stages of Kienböck's disease

Stage	Treatment option
Stage I	Immobilization
Stage II	Radial shortening for ulnar minus variance Capitate shortening or radial wedge osteotomy for neutral or ulnar positive variance Direct revascularization
Stage IIIA	Same as Stage II
Stage IIIB	STT or SC fusion ± lunate excision Radial shortening or radial wedge osteotomy
Stage IIIC	Simple lunate excision or excisional arthroplasty
Stage IV	Salvage (PRC, total wrist fusion, TWA)

STT scaphotrapezium-trapezoid, *SC* scaphocapitate, *PRC* proximal row carpectomy, *TWA* total wrist arthroplasty

than 60°, respectively. Additionally, in 2010, Lichtman et al. [33] defined stage IIIC as a complete coronal plane split regardless of the lunate or wrist morphology. Although the staging system that includes stage IIIC may not be universally accepted, the author prefers to use this system to select surgical options. Stage IIIA comprises lunate collapse without carpal malalignment, so the surgical options are identical to those for stage II. The choice of surgical options for stage IIIB remains debatable. Wrists with stage IIIB Kienböck's disease have lunate collapse with fixed rotation of the scaphoid. Previous biomechanical studies have suggested that correction of scaphoid malrotation is critical in unloading the lunate [36, 37]. Consequently, radial osteotomies or capitate shortening, which cannot correct scaphoid rotation, might be ineffective for stage IIIB. Lichtman et al. [38] recommended either limited wrist fusion or proximal row carpectomy (PRC) for stage IIIB. Scaphoid malalignment can be corrected via limited wrist fusions, such as scaphotrapezium-trapezoid (STT) or scaphocapitate (SC) fusion, which reduce the excessive load on the lunate. However, Weiss et al. [39] demonstrated that radial shortening achieved excellent clinical and functional results for stage IIIB Kienböck's disease, despite the lack of radiological improvement. Regardless of radiological progression after radial osteotomies, radial shortening in patients with Kienböck's disease with ulnar minus variance and lateral closing osteotomy for those with neutral or ulnar plus variance successfully reduces wrist pain and improve wrist function [40, 41]. These studies suggest that radial osteotomy might be indicated for stage IIIB. The characteristic finding of stage IIIC is a coronal lunate fracture. In wrists with stage IIIC disease, healing or revascularization of the lunate is not expected. Therefore, simple lunate excision or excisional arthroplasty using autologous tissues or prostheses is indicated for stage IIIC. Simultaneously, STT or SC fusions may be performed to prevent proximal migration of the capitate.

The treatment target for stage IV is not only the diseased lunate but also radiocarpal or midcarpal osteoarthritis. Salvage procedures such as PRC or total wrist fusion for symptomatic relief are indicated in stage IV. Recently, total wrist arthroplasty has been performed to achieve both pain relief and wrist motion [38, 42].

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Conservative Treatments

8

Makoto Motomiya

Abstract

Although a conservative treatment is commonly applied for Kienböck's disease by most surgeons, the detailed procedures and immobilization period of conservative treatment remain uncertain. Patients less than 20 years old should be firstly treated by rigorous conservative treatment because younger generations have great potential for revascularization of the lunate. On the other hand, elderly patients have less capability of revascularization of lunate unlike younger patients, and conservative treatment for elderly patients is mainly intermittent immobilization using a simple brace during pain and/or manual handling tasks. In adult patients with early-stage Kienböck's disease, conservative treatment of immobilization is recommended for about 3 months to obtain revascularization of the lunate. However, it remains unclear what kinds of patients' characteristics have effects on good or deteriorated clinical course of Kienböck's disease treated by conservative treatment. Because any salvage surgery often leaves functional impairment such as a loss of mobility of the wrist joint, surgical intervention should be considered when initial conservative treatment is not effective.

Keywords

Kienböck's disease · Conservative treatment · Immobilization

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8.1 Introduction

Although a conservative treatment is commonly applied for Kienböck's disease by most surgeons [1], the detailed procedures and immobilization period of conservative treatment remain uncertain. As mechanical stress on the lunate and circular disturbance in the lunate have been considered to attribute to the occurrence of Kienböck's disease [2], rigorous immobilization is preferable to decrease mechanical force and to improve blood circulation, which is considered to accelerate revascularization of lunate and/or prevent disease progression [3]. On the other hand, simple intermittent immobilization is often applied only to relieve symptoms [4]. The conservative treatment also includes general lifestyle guidance including avoidance of repetitive forceful activity and lifting heavy loads [1, 5]. In this chapter, a conservative treatment and timing of surgical treatment are summarized in each age group according to the previous reports.

8.2 Conservative Treatments for Young Patients

Patients less than 20 years old should be firstly treated by rigorous conservative treatment because younger generations have great potential for revascularization of the lunate [5]. Skeletally immature patients with Kienböck's disease have been reported better clinical outcomes in conservative treatment compared to adult patients, even in case with advanced stage of disease [5, 6]. Although various immobilization methods and period have been reported based on a few experiences, rigorous prolonged immobilization of wrist using cast have been generally recommended for 3–6 months in skeletally immature patients because of less risk for joint contracture than in adults.

Kienböck's disease in skeletally immature patients is divided into infantile (less than 12 years old) and juvenile (from 13 years old to the end of skeletal maturity) [7]. Irissari [7] reported that good pain relief and lunate revascularization could be obtained in all four infantile patients who were treated by immobilization alone although asymptomatic deformity and sclerotic change remained only in one 9-year-old girl. On the other hand, some patients have been reported to continue the symptom in spite of prolonged conservative treatment [8], and Foster [9] reported the youngest, 8-year-old patient who had taken a radial shortening osteotomy. In juvenile patients with Kienböck's disease, 30% of patients who took conservative treatment had disease progression, and those patients need to take any surgical treatment such as radial shortening osteotomy [5, 7]. Among skeletally immature patietns with Kienbock's disease, there were many cases who resistant to the conservative treatment in patients more than 15 years old. Juvenile patients between 13 and 14 years old were expected to good outcomes but need longer immobilization than infantile patients.

8.3 Conservative Treatments for Elderly Patients

In elderly patients with Kienböck's disease, conservative treatment is also effective regardless of clinical stage of Kienböck's disease [5]. Because elderly patients have less capability of revascularization of lunate unlike younger patients, conservative treatment for elderly patients is mainly intermittent immobilization using a simple brace during pain and/or manual handling tasks. In elderly patients, Kienböck's disease is characterized by the dominant hand of manual labor similar to the common Kienböck's disease. While there have been reported to be less incidence of ulnar negative variance and to be mostly female patients in elderly patients, unlike common adult Kienböck's disease. Taniguchi et al. [10] reported the clinical outcomes of 15 hands of 14 patients more than 60 years old who were treated with either treatment of observation-only management, intermittent immobilization using a splint, or surgical treatment of excisional arthroplasty. In their series with mean 5.6 years follow-up, all patients regardless of the type of treatments obtained good or excellent outcomes although disease progression and development of carpal collapse were found in all wrists. That is, conservative treatment should be firstly considered for elderly patients with Kienböck's disease and salvage surgery after progression of the carpal collapse should be secondly considered in case of patients who continue symptoms because of good outcome of salvage surgery in elderly patients. On the other hand, recently some surgeons reported complications of subcutaneous flexor and extensor tendon ruptures caused by collapsed fragment of lunate [11, 12]. Although most elderly Kienböck's disease without symptoms are likely to be left, we should inform elderly patients with advanced Kienböck's disease of the possibility of these complications.

8.4 Conservative Treatments for Adults (Aged 21–70 Years)

In adult patients with early-stage Kienböck's disease, conservative treatment of immobilization is recommended for about 3 months to obtain revascularization of the lunate [1, 5], however, it remains uncertain whether rigorous immobilization is needed or not [4]. Conservative treatment is also applied for adult patients with advanced stage Kienböck's disease if the patients do not hope for any surgical treatment. In the long-term outcome of more than 10 years, Fujisawa et al. [13] reported relatively favorable outcomes in 17 hands of 17 patients with conservative treatment in more than 10 years follow-up. In their series, there were no change of clinical stage in 5 hands and improvement in 4 hands although there was disease progression in 8 hands. Viljakka et al. [14] reported the clinical outcomes in 9 hands of 8 adult patients for more than 10 years follow-up, and there was no disease progression in 5 hands. They described that two patients obtained morphological improvement in X-ray evaluation in their series and some patients treated by conservative treatment had spontaneous potential of remodeling and revascularization in adult

Kienböck's disease. On the other hand, Keith et al. [15] described that Kienböck's disease is mostly progressive in clinically and radiologically from their long-term outcome of nonsurgical treatment and 6 out of the 16 patients with conservative treatment resulted in poor outcomes. Mikkelsen et al. [16] reported poor functional outcomes treated by conservative treatment. In their 25 hands of 24 patients with a mean 8 years follow-up, functional disability in daily life remained in 14 patients and salvage surgical procedures of arthrodesis were needed in 5 hands. They concluded immobilization for Kienböck's disease was not satisfactory and resulted in disease progression, especially in patients with ulnar minus variance.

There have been some comparative studies between conservative treatment and surgical treatments for adult Kienböck's disease. We should evaluate the outcomes by paying attention to the following two points: First, the group of surgical treatments tended to include patients with more advanced stages of Kienböck's disease than that of conservative treatment in most studies. Second, the group of surgical treatments include various types of surgical procedures, not only revascularization and joint leveling procedures but also salvage procedures. In comparative studies between conservative treatment and salvage surgical procedures such as excisional arthroplasty, silicon replacement, and partial carpal arthrodesis, most surgeons showed better outcomes in conservative treatments than those in surgical procedures [17–19].

Surgical procedures such as radial shortening osteotomy and various vascularized bone graft procedures have been considered to be able to slow down disease progression as it could not improve the clinical stage in adult patients with Kienböck's disease [20, 21]. Because any salvage surgery often leaves functional impairment such as a loss of mobility of the wrist joint [17, 22], early surgical intervention should be considered when initial conservative treatment is not effective [1, 5]. Salmon et al. [21] reported 15 hands with radial shortening osteotomy had less pain and better functional outcomes than 18 hands with conservative treatments in the comparative outcomes between conservative treatments and radial shortening osteotomy for Kienböck's disease. They stressed that surgical intervention should be considered to avoid carpal collapse, especially in patients with stage III of Kienböck's disease because conservative treatment mostly resulted in disease progression in advanced stage Kienböck's disease.

It remains unclear what kinds of patients' characteristics have effects on good or deteriorated clinical course of Kienböck's disease treated by conservative treatment. Compared to surgical treatments such as various types of joint leveling procedures and vascularized bone graft procedures, there have been a few studies to evaluate the effectiveness of conservative treatment for Kienböck's disease. We should gather high-quality data on the long-term follow-up in patients with nonsurgical treatment, and recognize the risk factor of poor outcomes in conservative treatment [23]. For patients who are resistant to conservative therapy and who are at high risk of disease progression, a treatment strategy involving early surgical intervention is desired in the future.

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Surgical Treatments: Revascularization

9

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Abstract

Vascularized bone graft (VBG) for Kienböck's disease is an attractive strategy that can be expected to revascularize the lunate bone by transplanting bone with blood flow to the lunate. The VBG is generally applied for patients with ulnar neutral or plus variance in stage II/III of Lichtman's classification, but there have been no clear criteria related to the indication of VBG. Because the VBG can maintain cell viability through the whole operative procedure, bone necrosis and resorption seem less likely occur in the VBG compared to the conventional non-vascularized bone graft. However, when VBG is implanted into the lunate bone, it gradually loses its mechanical strength during revascularization, and if proper procedures are not taken to relieve excessive stress on the implanted VBG, the lunate bone will collapse. Therefore, it is very important to add any procedures, including temporary pinning between carpal bones and external fixator, for reducing force on the grafted VBG and lunate until achieving revascularization of the lunate and union of the VBG into the recipient bed. The VBG is a technical dependent procedure that needs appropriate insertion of the harvested VBG into the lunate and achievement of the integration of the implanted VBG to obtain good clinical and radiological outcomes.

The VBG can be also used as a joint spacer after completely resecting the lunate and as an osteochondral graft for reconstruction of the radiocarpal joint in Kienböck's disease.

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Keywords

Kienböck's disease · Revascularization · Vascularized bone graft · Surgical treatment

9.1 Introduction

Joint leveling procedure such as radial shortening osteotomy is the most commonly performed surgery for Kienböck's disease to reduce the load shearing force to the lunate, and vascularized bone graft (VBG) for this disease is another important procedure to directly get the lunate revascularization. The VBG is generally applied for patients with ulnar neutral or plus variance in stage II/III of Lichtman's classification [1, 2], but there have been no clear criteria related to the indication of VBG. The VBG is generally indicated for patients with an intact shell of cartilage of the lunate and without osteoarthritic change in the radiocarpal and midcarpal joint [3–5]. Because the VBG can maintain cell viability through whole operative procedure, bone necrosis and resorption seem less likely to occur in the VBG compared to the conventional non-vascularized bone graft. However, the grafted VBG into the lunate gradually decreases the mechanical strength during revascularization, followed by collapse of the lunate unless the proper procedure is used to reduce the excessive load on the grafted VBG [6, 7]. Therefore, it is very important to add any procedures, including temporary pinning between carpal bones and external fixator, for reducing force on the grafted VBG and lunate until achieving revascularization of the lunate and union of the VBG into the recipient bed.

In the treatment using the VBG for revascularization of the lunate, we should first check the patient's background and clinical stage classified by radiological findings. Then, the optimal VBG is selected based on the following contents: approach to the lunate (dorsal or volar side), the harvest site, reliability of vascular pedicle, quality and quantity of the VBG, donor site morbidity, and technical simplicity and difficulty (Fig. 9.1: What is the optimal VBG for revascularization?)

When we evaluate the clinical outcome using the VBG in Kienböck's disease, we should take into account the accompanying procedure including core decompression of the necrotic lunate and temporary fixation for reducing the force on the lunate. In addition, some surgeons like to perform a joint leveling procedure, including radial shortening osteotomy and capitate shortening osteotomy, and carpal fusion in combination with the VBG graft. Clinical improvement may attribute to the accompanying procedures as well as the VBG graft itself.

Although the VBG for revascularization is generally grafted into the lunate after curettage of necrotic tissue, the VBG can be also used as a joint spacer after completely resecting the lunate and as an osteochondral graft for reconstruction of the radiocarpal joint in Kienböck's disease (Fig. 9.2: Surgical strategy for Kienböck's disease with vascularized tissue transfer). In this chapter, various VBG procedures and each clinical outcome are summarized according to the previous reports.

What is the optimal VBG for revascularization?

- **Approach to lunate** Volar or Dorsal
- **Vascular reliability** Pedicle length, Vascular diameter size
- **Harvested bone** Quality & Quantity
- **Technical difficulties** Microsurgery

Type	Nutrient	Approach	Comments
os pisiform	Dorsal branch of ulnar a.	Volar	Application for revascularization & replacement
Distal radius	Transverse volar carpal a.	Volar	Easy access in RSO
	PQ muscle	Volar	Strong pedicle, Easy handling Easy access in RSO
	1,2 ICSRA	Dorsal	Thin & Short pedicle Popular application for scaphoid nonunion
	4+5 ECA	Dorsal	Long, reliable pedicle
Phalangeal base/ Metacarpal neck	Metacarpal a.	Dorsal	Limited harvesting volume Less invasive access in CSO
Free iliac bone	Deep iliac circumflex a.	Both	Enough bone Volume Flexibly setting VBG Need for microsurgical anastomosis

VBG: vascularized bone graft; PQ: pronator quadratus; ICSRA: the intercompartmental supraretinacular arteries; ECA: the extensor compartmental arteries; RSO: radial shortening osteotomy; CSO: capitate shortening osteotomy

Fig. 9.1 What is the optimal VBG for revascularization in Kienböck's disease?

a

Revascularization

- Vascular bundle implantation
- Pedicled vascularized bone graft
- Free vascularized bone graft

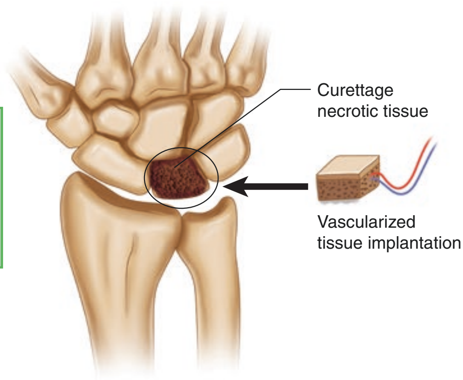

Fig. 9.2 Surgical strategy for Kienböck's disease with vascularized tissue transfer. (**a**) Revascularization. (**b**) Replacement. (**c**) Reconstruction

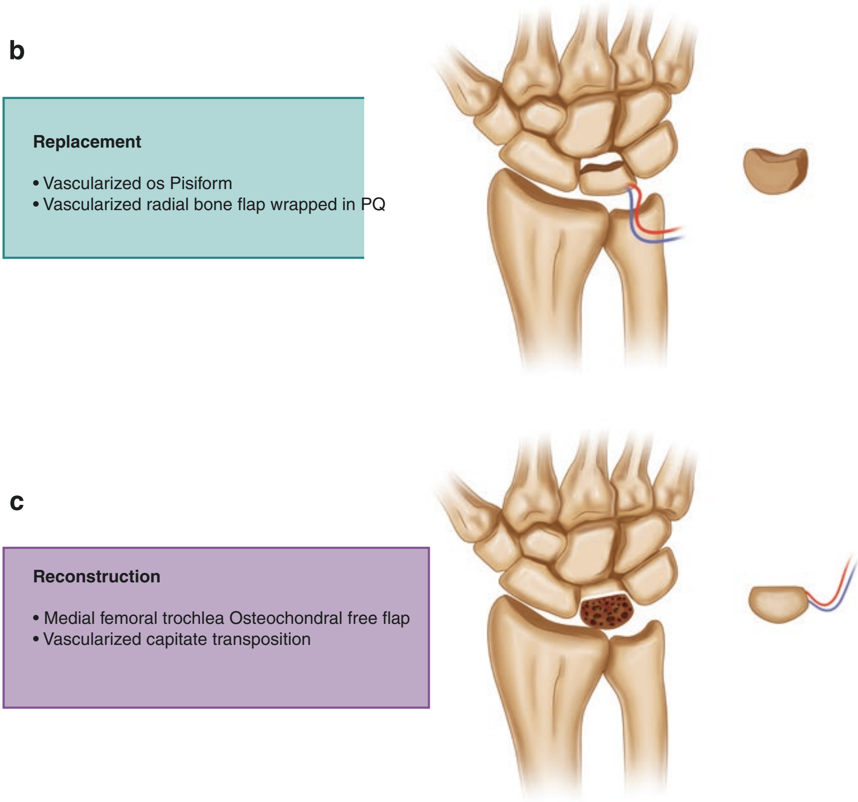

Fig. 9.2 (continued)

9.2 Harvest from Dorsal Distal Radius

Sheets et al. [8] reported a detailed anatomical study related to the vascular network around the distal radius. They described that the distal radius is one of the most suitable sites as a donor for the VBG because of the abundant vascular network and good quality cortico-cancellous bone. The VBG with a vascular pedicle of the fourth plus fifth extensor compartmental artery (ECA) has been one of the most commonly performed surgeries among the VBG harvested from the dorsal distal radius [9, 10]. This VBG has a great advantage in the sufficient diameter size and vascular pedicle length. In addition, the whole procedure can be achieved through the dorsal approach, which has less risk of injury to the volar radiocarpal ligament that contributes to the stability of the lunate. After implantation of the VBG, temporal fixation between the scaphoid and capitate is usually performed using Kirschner wires to reduce axial mechanical force on the lunate. In cases without the fifth ECA, there have been reported alternative procedures of the VBG harvested from the dorsal distal radius, such as the first and second intercompartmental supraretinacular artery (ICSRA) [11] which is popular in the scaphoid nonunion, the second and third ICSRA [5, 6], and the fourth ECA [12].

Moran et al. [9] reported the clinical outcomes of the fourth plus fifth ECA bone graft for 26 patients with a mean duration of 31 months. In their series, 92% of patients had significant pain relief and 77% of patients did not show radiological progression of collapse after surgery, although most of the patients had Lichtman stage II or IIIA. The revascularization could be confirmed by an MRI in 71% of patients, and those with revascularized lunate obtained significantly good clinical outcomes. They indicated that achieving revascularization depends on the delicate surgical technique, such as putting the VBG in the appropriate position while preserving vascularity. Kirkeby et al. [13] have also reported on middle-term results of the fourth plus fifth ECA for five patients. All patients had good outcomes and complete revascularization was confirmed in 50% of patients and partial revascularization in 50% of patients. Havulinna et al. [14] reported a case with stage IIIC with coronal plane fracture of the lunate treated by the fourth plus fifth ECA. In this patient, bony union was obtained in the separated lunate without progression of lunate collapse using the VBG in the shape of a bone-peg harvested from the dorsal distal radius.

9.3 Harvest from Volar Distal Radius

In 1983, Braun [15] has reported the VBG procedure harvested from the volar distal radius, which was nourished by the pronator quadratus (PQ). Shin et al. [16] implanted the PQ pedicled VBG in 27 patients with a mean age of 42 years. In spite of most patients with advanced stages of IIIA and IIIB, two-thirds of patients obtained successful outcomes and radiological improvement in their series. The PQ pedicled VBG has advantages, including a strong and thick pedicle without fear of pedicle kinking and easy handling without meticulous pedicle dissection. They reported that the PQ pedicled VBG was a useful treatment to improve lunate morphology although it could not restore the alignment in radiological findings.

On the contrary, in cases of patients with radial shortening osteotomy through the volar approach, the VBG from the volar distal radius is a useful option because both procedures can be done in the same volar incision. Mathoulin et al. [17] reported the clinical outcome using the VBG harvested from the volar distal radius nourished by the transverse carpal artery with the radial osteotomy for 22 patients with various staged patients. Most patients obtained good to excellent clinical outcomes in more than 5 years of follow-up and 77% of patients achieved radiological improvement at mean 8 months after surgery. They concluded that good functional outcome was significantly associated with early stage, younger age (less than 40 years), sedentary occupation, and early intervention from diagnosis.

9.4 Dorsal Metacarpal Artery Pedicled Vascularized Bone Graft

The dorsal metacarpal artery is also a commonly available pedicle for the VBG around the wrist joint, and there have been reports of the metacarpal vascularized bone nourished by the dorsal metacarpal artery, such as the base of the second and third metacarpal bone graft [18] and the neck of the second metacarpal bone [19]. The dorsal metacarpal artery pedicled VBG can be harvested through the same dorsal incision as the exposure of the lunate and is an easy and reliable option for the treatment of Kienböck's disease.

Waitayawinyu et al. [20] reported the clinical outcome using the base of the second and third metacarpal bone graft accompanied by capitate shortening osteotomy for 14 patients with Lichtman stage II or IIIA which had a neutral or positive ulnar variance in X-ray. In their series with mean 41 months follow-up, all patients obtained good clinical outcomes and preservation of the wrist range of motion without progression of the radiological collapse of the lunate.

9.5 Vascularized Pisiform Bone Graft

Although Beck [21] firstly reported the procedure of vascularized pisiform nourished by the ulnar artery itself transferred to the carpal bone in 1971, the modified technique by Kuhlmann et al. [22] was more popular, which was nourished by the dorsal branch of the ulnar artery. Daecke et al. [23] reported the long-term results with a mean follow-up of 12 years in 23 patients treated with the vascularized pisiform bone graft. Although 11 patients underwent radial shortening osteotomy at the same time, 87% of the patients obtained pain relief and functional improvement, and only 32% of the patients had the progression of arthrosis in radiological findings. They stressed the importance of the vascularity of the implanted graft bone because they found a correlation between poor clinical outcomes and radiological sclerotic change of the graft in their series.

Saffar et al. [24] reported the vascularized pisiform bone graft for the replacement after complete resection of the lunate. Anatomically, the anteroposterior width of the pisiform bone has been reported to be similar to the height of the lunate, and the space height after lunate excision could be maintained by the implantation of the vascularized pisiform bone graft with a 90-degree rotation. However, problematically, the implanted pisiform was gradually dislocating because of a small volume of the pisiform [25]. Daecke et al. [26] used the vascularized pisiform bone graft also for the replacement after resection of the lunate in patients with advanced stage. In their series of 21 patients with a mean 9.9 years follow-up, most patients were satisfied with clinical outcomes including maintaining wrist function, but the carpal alignment could not maintain radiological findings and arthritic changes occurred in half of the patients. Tan et al. [27] also reported that 82% of the patients treated with this technique accompanied with any carpal fusion obtained good or excellent outcomes in the long term with more than 15 years. Partial carpal fusion such as scapho-trapezio-trapezoid arthrodesis or scaphocapitate arthrodesis has been recommended to be added in patients with advanced stage to reduce the mechanical axial force of the implanted pisiform [28].

In regard to the use of the VBG for the replacement, the vascularized radial bone flap wrapped in the pronator quadratus was also used in patients with advanced stage and satisfactory short to middle-term outcomes were reported [29, 30].

9.6 Free Vascularized Iliac Crest Bone Graft

In regard to the free VBG for Kienböck's disease, the free iliac bone graft nourished by the deep circumflex iliac vascular bundle has been reported. Arora et al. [3] reported good long-term results of the free iliac VBG for 18 patients with stage IIIA and IIIB accompanied with temporally external fixation and lunotriquetrum pinning through volar approach. As the same group had reported good clinical outcomes in the mid-term outcome with a mean 5 years follow-up [31], good alignment without collapse of the lunate bone could be maintained in the long-term follow-up of more than 10 years in the patients with osteointegration of the grafted bone. Revascularization of the lunate could be confirmed in the MRI examination. Resorption of the grafted bone and the progression of collapse were found in two patients, who had poor functional outcomes. They concluded that because enough volume of cortico-cancellous bone could be harvested from the iliac crest which has good mechanical property compared to the other VBG, the free iliac VBG could make the entire necrotic lunate revascularized even in the advanced cases with the lunate fracture. Because of the complicated procedure of microvascular anastomosis, there have been few reports of the free VBG for the treatment of Kienböck's disease in the era with many simple and reliable pedicled VBG.

9.7 Vascularized Pedicle Implantation

Hori et al. [32] reported that the procedure consisted of a free non-vascularized cancellous bone graft accompanied with vascular bundles into the defect of the lunate after debridement of the necrotic bone to aim at revascularization of the lunate. The dorsal metacarpal artery and anterior and posterior interosseous artery with its venae comitantes were used as donor pedicles, and the indication of this technique was considered to be for cases with ulnar plus and/or neutral variance with stage II [33]. Bochud et al. [34] reported the clinical outcome of the vascularized pedicle implantation combined with other procedures. In their series, only 43% of patients obtained excellent or good outcomes and patients with advanced stages resulted in unfavorable outcomes. As there are currently many reliable pedicled VBG around the wrist joint, the procedure of vascular pedicle implantation seems to be less popular in the treatment of Kienböck's disease. Kakar et al. [35] showed a good clinical case using the fourth dorsal metacarpal pedicle implantation in a 14-year-old case with stage IIIA, and this procedure may be applied for younger patients with open growth plate to avoid the invasive procedure to the donor site.

9.8 Reconstruction of Lunate Using Free Medial Femoral Trochlea Osteochondral Free Flap

Burger et al. [36] reported an innovative strategy for reconstruction of the whole proximal part of the necrotic lunate using the medial femoral trochlea osteochondral free flap. In this method, both volar and dorsal approaches can be chosen depending on the condition. At first, the necrotic proximal part of the lunate is completely resected while carefully preserving the distal portion of the lunate including the scapholunate and lunotriquetrum ligament and the distal cartilage surface of the lunate. Then the osteochondral free flap is harvested from the medial femoral trochlea with pedicle vessels of the descending genicular artery, and is transferred into the space of the proximal part of the lunate. The osteochondral flap is fixed with the distal part of the native lunate using a miniplate, screw, or Kirschner wires, and scapholunate and lunotriquetrum are temporally fixed with K-wires for approximately 3 months.

Pet et al. [37] reported the clinical outcome of the free osteochondral flap for patients with stage IIIA and IIIB. All 18 patients with 1.4 years of radiological follow-up obtained bony union with maintaining carpal alignment and functional improvement without donor site morbidities although the ROM was not improved. In their series, this technique could be applied even for 14 patients with coronal split fracture using suture anchor repair. Although this technique requires a high degree of skill and the short-term outcome has been reported from the limited surgeons, the free osteochondral flap is expected, especially in younger patients because of poor clinical outcomes by any salvage procedures.

9.9 Partial Reconstruction of Radiolunate Joint Using Vascularized Capitate Transposition Technique

There have been some techniques for reconstruction of the radiocarpal joint using transposition of the capitate head [38, 39], but it is susceptible to necrosis of the transposed capitate head. Lu et al. [40] reported the technique of the vascularized capitate transposition for advanced Kienböck's disease. This technique consists of resection of the necrotic whole lunate, transposition of the divided capitate head with vascularity by the dorsal branch of the anterior interosseous artery, and conventional iliac bone graft into the gap between the capitate base and transposed capitate head. Li et al. [41] reported the good clinical and radiological outcomes of the vascularized capitate transposition in 17 patients with stage IIIA and IIIB, except for one patient who failed bony union. Li et al. [42] reported the mid-term clinical outcomes of this technique in 26 patients with all stage IIIB. In their series, functional outcome and radiological alignment were significantly improved, but radioscaphoid angle could not be reduced enough because of the disruption of the scapholunate and lunotriquetral interosseous ligaments, possibly followed by the progression of wrist arthritis.

On the contrary, Ruttermann [43] described a negative opinion of the transposition of capitate head because of poor outcomes in the long-term follow-up.

9.10 Combination of VBG with Joint Leveling Procedure

Because it is considered to take 6–8 months to achieve the revascularization and integration of the VBG into the lunate in a case with an advanced stage, some surgeons stressed the importance of decreasing mechanical force on the lunate after the surgery of the VBG during healing process [6, 7, 44]. Although most VBG procedures are generally performed with temporal fixation for 2–3 months, they stressed that it was insufficient periods for lunate unloading in patients with the VBG surgery, possibly leading to poor outcomes. There have been several reports about the VBG surgeries combined with permanent decompression procedures, including joint leveling procedures and partial carpal fusions [20, 23, 45]. Fujiwara et al. [5] reported an interesting outcome of the pedicled VBG in patients with stage IIIA and IIIB with more than 10 years follow-up. In their series, all patients with stage IIIB undertook the joint leveling procedures in addition to the VBG surgery, while patients with stage IIIA undertook only the VBG surgery. Although all patients obtained good clinical outcomes, the patients with stage IIIB were superior to those with stage IIIA both in clinical and radiological measurement, which supports the usefulness of the combination procedures of the VBG surgery with permanent lunate unloading.

9.11 Comparison of Clinical Outcome Between VBG and Joint Leveling Procedures

It remains to be seen as to which treatment is better for Kienböck’s disease, the VBG or the joint leveling procedure. Afsher et al. [46] reported a comparison study between radial shortening osteotomy and the VBG nourished by the fourth plus fifth ECA in patients with the same background except for the ulnar variance. In their series with a mean 6.5-year follow-up, there was no significant difference in pain, range of motion, grip strength, and radiological assessment. Although the VBG was superior to radial shortening osteotomy only in the Cooney’s score, they could not make a conclusion with regard to the superiority of either treatment. On the other hand, Quenzer et al. [47] reported an interesting study about radial shortening osteotomy with or without additional procedures including the VBG and the vascularized pedicle implantation for Kienböck’s disease. In their series, 55% of patients with any additional revascularization procedures achieved good radiological improvement in the appearance of the lunate, while only 20% with radial shortening osteotomy alone were favorable. They considered that the addition of any revascularization procedures improved the clinical and radiological outcome of radial shortening osteotomy in Kienböck’s treatment.

9.12 Summary

The integration of the implanted VBG is considered to be important in the VBG treatment, although many surgeons reported the discrepancy between lunate morphology and clinical outcome. Arora et al. [3] and Moran et al. [9] reported that some cases without integration of the implanted VBG resulted in the collapse of the lunate and poor clinical outcome, while most cases with completion of integration of the implanted VBG would achieve not only clinical outcomes but also radiological appearance. Regardless of the type of the VBG, good clinical and radiological outcomes should maintain for more than 10 years in patients with integration of the implanted VBG [3, 5, 23]. Nakagawa et al. [48] reported that there has been no significant difference between three different VBG from the dorsal wrist and hand area in functional outcomes, and stressed the importance of not only vascularity but also appropriate insertion of the VBG to obtain a morphological improvement of the lunate. They recommended that the VBG should be correctly inserted as the cortex aligned longitudinally and should be chosen the type of the VBG depending on the lesion of the necrosis and fragmentation.

In summary, the VBG treatment for Kienböck's disease seems to be a technical dependent procedure that needs appropriate insertion of the harvested VBG into the lunate and achievement of the integration of the implanted VBG to obtain good clinical and radiological outcomes. When we choose the VBG treatment for Kienböck's disease, we also should consider accompanying procedures, including temporally pinning, external fixator, and permanent decompression by any joint leveling procedure and partial wrist fusion, to decrease mechanical force on the lunate during healing process of the lunate. The VBG treatment always requires a larger sacrifice related to the VBG elevation compared to the other procedures, so if we choose the VBG treatment, the patients have to get an equivalent amount of satisfaction. We should make more reliable evidences about the long-term efficacy of the VBG treatment for Kienböck's disease.

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10 Surgical Treatments: Radial Osteotomy

Michiro Yamamoto

Abstract

Radial osteotomy is indicated for Kienböck's disease. In this chapter, we describe our surgical technique for radial osteotomy using a volar locking plate (VLP) and present the results of a clinical study. We retrospectively compared younger and older patients with Kienböck's disease stages II–IIIB who had radial osteotomies fixed with volar locking plates, examining their clinical and radiological outcomes.

We find that radial osteotomy is an appropriate treatment option for symptomatic Kienböck's disease Lichtman stages II to IIIB, not only for younger patients but also for those 40 years of age and older. While younger patients showed greater radiological and clinical improvements in both frequency and degree, the older group had postoperative clinical improvements significant enough to recommend the technique.

Keywords

Kienböck's disease · Radial osteotomy · Volar locking plate · Younger · Older

10.1 Introduction

Radial osteotomy or other joint leveling procedures to offload the lunate by reducing the mechanical forces on it are indicated for Lichtman stages II and IIIB. Treatment goals include pain relief, motion preservation, and maintaining hand strength and function. Outcomes are usually positive, particularly in young

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patients [1–3]. The good revascularization potential and remodeling capacity of the lunate in skeletally immature patients preclude operative management as the first line of treatment [4]. It seems that the natural history of Kienböck's disease in skeletally immature patients differs from adults. However, there are few comparative studies that assess outcomes by age.

An earlier study of patients 14–29 years of age had better outcomes than a comparatively older group of 30–54 years did, although the differences were not statistically significant [3]. Whether or not radial osteotomy is beneficial for older patients is not well understood. Recently, we reported that both radiological and clinical improvements were more frequently observed in younger patients. However, even the older group had significant clinical improvement. Our evidence suggests that radial osteotomy is a viable option for the treatment of symptomatic Kienböck's disease stages II–IIIB, even in older patients [5].

In this chapter, we describe our surgical technique for radial osteotomy using a volar locking plate (VLP) and present the results of a clinical study in which it is used to treat Kienböck's disease stages II–IIIB. This builds upon our earlier comparative work of younger and older patients, with a larger sample size.

10.2 Surgical Technique

Arthroscopy of the wrist is performed under general or regional anesthesia via a radiocarpal and midcarpal portal with the upper limb positioned in a traction tower. The proximal and distal sides of the lunate are inspected to assess its condition. The functional surface is evaluated using the Bain and Begg articular-based classification [6]. The degree of synovitis is determined to be none, mild, moderate, or severe. Subsequent to the arthroscopic procedure, a tourniquet is applied for the radial osteotomy. The type of radial osteotomy—radial shortening, wedge, or a combination of both—is chosen based on the ulnar variance (UV). The distal radius is exposed with a modified Henry approach using the plane between the flexor carpi radialis tendon and radial artery [7]. The pronator quadratus muscle is detached from the radial insertion. For a patient with a negative UV, the radius is shortened by means of two parallel transverse osteotomies, aligning the length of the distal ends of the radius and ulna. In the case of a neutral or positive UV, a radial closing wedge osteotomy is employed to attain a 15° radial inclination angle. For a patient with a UV close to zero, shortening and closing wedge osteotomies are often combined. While cutting the radius with a bone saw, care is taken not to injure the extensor tendon. Usually, the extensor tendons are protected by placing elevators between the radius and tendons.

After the osteotomy is completed, the radius is fixed with the surgeon's preferred volar locking plate (VLP), immobilized for 2 weeks in a short arm cast, and then supported in an orthosis for 1 month.

10.3 Clinical Study

Among the numerous treatment options for Kienböck's disease, a joint leveling procedure such as a radial osteotomy has been indicated for stages II–IIIB. Successful outcomes are typically reported, especially in young patients [1–3]. However, there are few studies that compare the outcomes of radial osteotomy according to age. In other words, there is relatively little evidence regarding the efficacy of radial osteotomy for older patients with Kienböck's disease. Nakamura et al. showed better results in younger patients (14–29 years old) than in older ones (30–54 years old), but this difference was not significant [3]. The extent to which radial osteotomy is indicated for older patients is not well understood.

We retrospectively compared younger and older patients with Kienböck's disease stages II–IIIB who had radial osteotomies fixed with volar locking plates, examining their clinical and radiological outcomes.

10.4 Patients and Methods

10.4.1 Patients

Patients treated by radial osteotomy for Kienböck's disease between 2007 and 2019 who had postoperative follow-ups for no less than 1 year were included in this study. Twenty-four consecutive patients were enrolled, 14 females and 10 males ranging from 12 to 74 (mean 38) years old at the index procedure. Two groups were formed, those younger than age 40 years and those 40 years and older, to make an age-based comparison. Four patients were Lichtman stage II, 9 stage IIIA, and 11 stage IIIB. The surgical technique and postoperative care were administered according to the method *previously described* [5].

10.4.2 Outcome Measures

Radiological and functional assessments were performed pre- and postoperatively. Radiographs to ascertain the carpal height radio (CHR), Stahl index, ulnar variance (UV), and radial inclination (RI) were taken. Where applicable, measurements were determined digitally using Rapideye Core (Canon Medical Systems, Tochigi, Japan). In all patients, the fractures of the lunate were evaluated by preoperative computed tomography (CT) and wrist arthroscopy. An assessment of how the fracture healed was done at the final follow-up. Pre- and postoperative magnetic resonance images (MRI) were available for 11 patients (8 in the younger group and 3 in the older) for whom coronal T1- and T2-weighted signal intensity changes were evaluated.

Functional assessments were made based on the wrist range of motion, grip strength, numeric rating scale for pain (NRS pain; 11-point scale from 0 [no pain] to 10 [worst pain imaginable]), and Hand20 (a self-rated questionnaire used to measure upper extremity disability, scored from 0 to 100 with higher values indicating poorer function) [8].

10.4.3 Statistical Analysis

The *t*-test was used for the comparison between younger and older groups preoperatively and at final follow-up. The mean including standard deviation was used to present continuous data, while categorical data were presented, where appropriate, by frequencies and percentages. Statistical significance was set at a two-tailed *p*-value of <0.05.

10.5 Results

There were no postoperative complications and bony union with proper alignment was confirmed by 3 months at the site of all osteotomies. Within several months, each patient was able to resume their occupation or daily activities unimpeded. The mean follow-up period was 4.1 years for the younger group and 3.5 years for the older group.

Patients were separated into two groups based on age, with 40 years serving as the point of division, and further identified by *the Lichtman disease stage and Bain and Begg arthroscopic classification* [6, 9]. One wrist from each patient was included (Table 10.1).

Table 10.1 Patient demographics and outcomes

Group	Younger (n = 13)	Older (n = 11)	p-value
Mean age (range)	24 (12–37)	56 (40–74)	<0.01
Male patients (n)	7	3	0.19
Female patients (n)	6	8	
Mean follow-up period, year (range)	4.1 (1–10)	3.5 (1–11)	0.6
Lichtman stage II (n)	2	2	0.98
IIIA (n)	5	4	
IIIB (n)	6	5	
Bain and Begg classification 1 (n)	5	4	0.92
2a (n)	0	0	
2b (n)	8	7	

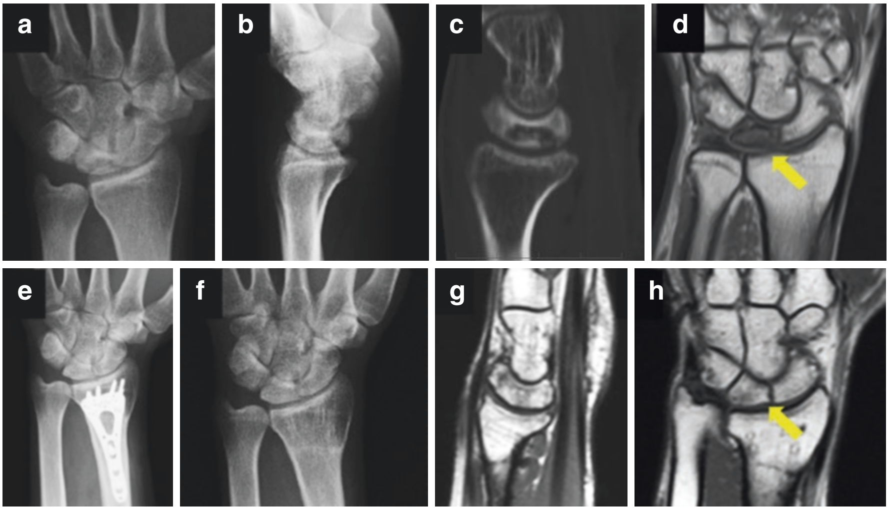

Fig. 10.1 A representative case from the younger group. An 18-year-old male with severe right wrist pain was admitted to our hospital. Preoperative X-ray revealed Kienböck's disease, Lichtman's stage IIIB (**a** and **b**). Preoperative computed tomography (CT) showed a fracture of the lunate (**c**). Preoperative T1WI magnetic resonance imaging (MRI) showed low-intensity signals of the lunate (yellow arrow) (**d**). A radial shortening osteotomy was performed using a volar locking plate (Variable Angle LCP Two-Column Volar Distal Radius Plate 2.4 mm, DePuy Synthes, Oberdorf, Switzerland) (**e**). Radiographic examination at 2 years (**f**) and T1WI MRI showed significant signal improvement at the lunate (yellow arrow) (**g** and **h**)

10.5.1 Younger Group (39 Years and Under)

The younger group was composed of 13 patients ranging in age from 12 to 37 years (mean, 24). A Lichtman stage and Bain and Begg grade were recorded for each patient. The Lichtman stages were II (2 wrists), IIIA (5), and IIIB (6) while the Bain and Begg grades were 1 (5 wrists) and 2b (8). Of 13 wrists, 11 had a degree of synovitis greater than moderate and 8 had a fractured lunate bone revealed by preoperative CT and intraoperative arthroscopy (Fig. 10.1).

10.5.2 Older Group (40 Years and Over)

The older group was composed of 11 patients ranging in age from 40 to 74 years (mean, 56 years). The Lichtman stages were II (2 wrists), IIIA (4), and IIIB (5) while the Bain and Begg grades were 1 (4) and 2b (7). Of 11 wrists, 7 had a degree of synovitis greater than moderate and 7 had a fractured lunate revealed by preoperative CT and intraoperative arthroscopy (Fig. 10.2).

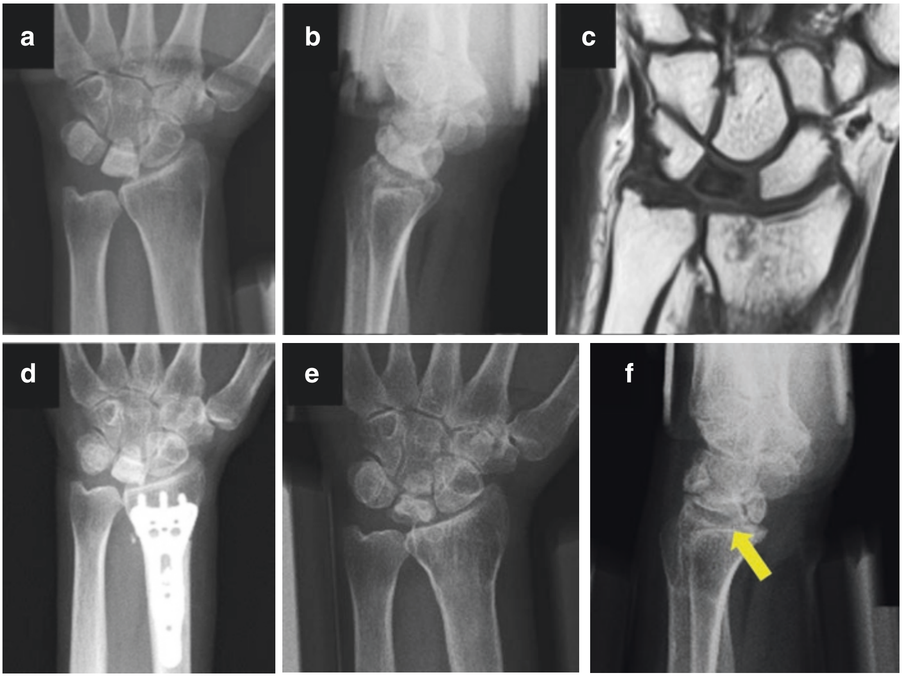

Fig. 10.2 A representative case from the older group. A 64-year-old female patient with left wrist pain was admitted to our hospital. Preoperative X-ray revealed Kienböck's disease, Lichtman's stage IIIA (**a** and **b**). Preoperative T1WI magnetic resonance imaging (MRI) showed a low-intensity signal of the lunate (**c**). A combination of radial shortening and wedging was performed using a volar locking plate (Matrix-SmartLock, Stryker Leibinger, Kalamazoo, MI, USA) (**d**). Progression of lunate segmentation with collapse (yellow arrow) was shown by radiograph 4 years postoperatively (**e**, **f**). However, a reduction was achieved in self-reported measures of pain: NRS-pain decreased from 6 to 3 and Hand20 improved from 62 to 36

10.5.3 Inter-group Comparison

The groups proved relatively well balanced on most parameters. No significant differences were observed in either group preoperatively, including in either the Lichtman stages or Bain and Begg grades. Further, no significant differences were shown in the CHR, Stahl index, UV, and RI. Postoperatively, the mean CHR of the younger group significantly increased and the mean Stahl index of the older group significantly decreased.

Both the younger and older groups demonstrated significant improvement postoperatively in objective assessments of wrist range of motion and grip strength as well as subjective NRS-pain and Hand20 measures (Table 10.2).

Bony union of the lunate was confirmed postoperatively in 4 of the 8 patients in the younger group who had sustained a fracture and only 1 of the 7 in the older group. MRI signal intensity improved in all eight patients in the younger group and in 1 of the 3 patients in the older group for whom images were available.

Table 10.2 Radiological and clinical outcomes

Group	Younger, *n* = 13 (mean ± SD)	Older, *n* = 11 (mean ± SD)	*p*-value
CHR *pre-op*	0.48 ± 0.03	0.5 ± 0.03	0.17
post-op	0.50 ± 0.04#	0.48 ± 0.04	0.39
Stahl index *pre-op*	0.34 ± 0.09	0.34 ± 0.05	0.9
post-op	0.35 ± 0.09	0.3 ± 0.07#	0.2
UV (mm) *pre-op*	-1 ± 1	−0.6 ± 1.7	0.43
post-op	0.3 ± 0.5##	0.7 ± 1.4##	0.38
RI (degree) *pre-op*	26 ± 2.3	28 ± 3.1	0.12
post-op	22 ± 4.7##	22 ± 2.4##	0.99
GP (%) *pre-op*	58 ± 21	55 ± 26	0.75
post-op	96 ± 26##	85 ± 23#	0.29
Arc (degree) *pre-op*	95 ± 26	92 ± 22	0.7
post-op	133 ± 32##	114 ± 18#	0.12
NRS pain *pre-op*	5.4 ± 1.9	6 ± 1.7	0.56
post-op	1.2 ± 1.2##	2.3 ± 1.1##	0.11
Hand20 score *pre-op*	53 ± 15	40 ± 13	0.1
post-op	17 ± 16#	20 ± 11#	0.76

CHR carpal height ratio, *SD* standard deviation, *UV* ulnar variance, *RI* radial inclination, *GP* grip power (% of the healthy contralateral side); *##p* < 0.01, *#p* < 0.05 comparison between pre- and postoperative values

10.6 Discussion

While the outcomes of radial osteotomies in younger patients are generally reported to be good, its effectiveness in older patients is uncertain [1, 2]. The main findings of our study are that younger patients typically showed both radiological (CHR, Stahl index, and union of fractured lunate) and clinical improvement (range of motion of the wrist, grip power, pain, and Hand20) while older patients also had significant clinical improvement, although postoperative radiological improvements were not observed.

None of the patients had intra- or postoperative complications. Our results suggest that radial osteotomy is a viable treatment for symptomatic Kienböck's disease stages II–IIIB in all patients.

There are several milestones in the history of treating Kienböck's disease. Substantial progress was made by Hultén, who proposed radial shortening osteotomy as a treatment [10]. Persson subsequently introduced the concept of ulnar lengthening [11]. Both observed that a disproportionate number of patients with Kienböck's disease appeared to have negative UVs.

Radial osteotomies have numerous theoretical advantages. In a radial shortening osteotomy, the axial load is transferred from the lunate to the ulnocarpal joint [2]. In a radial wedge osteotomy, the axial load shifts from the lunate to the radioscaphoid joint, with the lunate brought toward the radial side, subsequently increasing the radiolunate contact area [3, 12]. Pressure on the radiolunate decreases as the contact surface area increases. The leading biomechanical rationale for radial shortening and radial wedge osteotomies or a combination of both is to reduce the axial load on the lunate per unit. A countervailing perspective is put forth by Irisarri et al. who remind us that Kienböck's disease is not caused solely by mechanical factors [13]. In fact, there are reports of successful outcomes for radial osteotomies without shortening for Kienböck's disease stages II–IIIB [14]. Other than biomechanical mechanisms, radial osteotomies may suppress the progression of Kienböck's disease biologically, such as by altering the supply of blood in the area of the lunate.

To fix a shortened radius, Nakamura et al. used screws and Weiss et al. introduced the six-hole dynamic compression plate [2, 3]. Since the VLP was developed for the treatment of distal radius fracture, we have used it in all radial osteotomies. Fixation with VLPs has distinct advantages for both surgeons and patients. A VLP affords the surgeon flexibility to choose the kind of osteotomy to perform based on individual wrist morphology. VLPs are versatile and can be used in radial shortening osteotomies for wrists with negative UVs and in radial wedge osteotomies for wrists with neutral or positive UVs. All will avoid the emergence of postoperative ulnocarpal abutment syndrome. Due to proper fixation, VLPs afford an early return to daily activities for patients.

While treating stages II–IIIA Kienböck's disease by radial osteotomy is widely accepted, its use for stage IIIB is controversial. A limited number of studies report good outcomes following radial osteotomy for stage IIIB disease [9, 15, 16]. Alternatively, a carpal bony fusion or proximal row carpectomy is suggested in some studies [9]. Consensus is lacking as to the best approach to treat Kienböck's disease and systematic reviews have proven inconclusive [17]. Therefore, a randomized controlled study is needed to better direct evidence-based medicine approaches.

Both the Lichtman stage and Bain and Begg grade were employed for each patient in our study. The latter arthroscopic classification yielded nine patients in grade 1 (nonfunctional articular surface in the proximal lunate) and 15 in grade 2b (nonfunctional proximal and distal lunate). For grade 2b disease, Bain and Begg suggest a proximal row carpectomy as a salvage procedure [6]. But our data indicates that radial osteotomy remains effective to treat grade 2b, especially for younger patients. Our evidence is that the fractured lunate united successfully in 50% (4 of 8) of patients in the younger group.

The presence of synovitis was identified during the arthroscopic procedure. It proved to be more severe in the younger group. An association between synovitis and progression of Kienböck's disease is possible, but more data is necessary before inferring from such results.

10.7 Conclusion

We find that radial osteotomy is an appropriate treatment option for symptomatic Kienböck's disease Lichtman stages II–IIIB, not only for younger patients but also for those 40 years of age and older. While younger patients showed greater radiological and clinical improvement in both frequency and degree, the older group had postoperative clinical improvements significant enough to recommend the technique.

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Surgical Treatments: Capitate Shortening Osteotomy

11

Yuichiro Matsui

Abstract

Although radial shortening osteotomy for Kienböck's disease with ulnar negative variance has shown good results in patients with this disease, there have been many reports of ulnar impaction syndrome because of radial shortening in patients with ulnar neutral or positive variance. In 1993, Almquist et al. introduced a technique of capitohamate fusion with capitate shortening osteotomy for Kienböck's disease with ulnar neutral or positive variance. The purpose of capitate shortening osteotomy is to decompress the lunate by transverse cutting and shortening of the waist of the capitate and distributing the stress to the scaphotrapeziotrapezoid (STT) joint and the triquetrohamate joint. Capitate shortening osteotomy can be expected to have a sufficient lunate decompression effect to prevent osteonecrosis. Other advantages are that the procedure is relatively easy and does not block blood flow to the lunate.

In 2004, Moritomo et al. first devised and reported partial shortening osteotomy of the capitate. The purpose of this procedure was to reduce the effect on the carpal bone arrangement by partially shortening the capitate only on the articular surface with the lunate, preserving the scaphocapitate articular surface. When a long-axis load is applied to the hand, the force applied to the scaphoid is distributed to these three surfaces, and the scaphoid moves while all three joints are adjusted. Based on three-dimensional research on the anatomy and kinematics of the midcarpal joint, when the STT joint, scaphocapitate joint, and triquetrohamate joint remain, they considered that normal carpal movements and carpal arrangements could be maintained without articular contact between the capitate and the lunate. Considering these concepts, they performed partial capitate shortening osteotomy to preserve the scaphocapitate joint.

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Keywords

Capitate shortening osteotomy · Ulnar neutral or positive variance · Lunate decompression · Partial shortening osteotomy of the capitate

The capitate, the largest and most central of the carpal bones, has three parts: the head, neck, and body [1]. The volar and dorsal surfaces are non-articulated and possess many vascular foramina. The proximal third (head) has three articular aspects with different curvature radii adjoining the scaphoid on the radial side, the lunate on the proximal side, and the hamate on the ulnar side. The distal part of the capitate articulates with the trapezoid on the radial side. On the ulnar side, there is an articular surface with the hamate, but the palmar and distal sides are non-articular and are the attachment points of the strong capitohamate ligament. There are usually two articular surfaces distal to the capitate. There is a slightly concave articular surface with the third metacarpal in the middle, and a small articular surface with the second metacarpal on the radial side. On the ulnar side, a small articular surface with the fourth metacarpal may be present.

In an anatomical study, Vander Grend et al. [2] reported that the blood to the capitate is supplied predominantly by the palmar arteries. The palmar carpal branches supply vessels at the palmar ulnar border of the capitate neck. In addition, another large consistent branch from the deep palmar arch enters the distal to the capitate. Dorsally, the branches enter the capitate at the midwaist portion. They reported three types of intraosseous blood supply to the capitate. In the first type, the arterial supply is exclusively from the palmar vessels. In the second, both palmar and dorsal arteries supply the waist and distal capitate, but the head is supplied predominantly from the palmar vessels. In the third, the proximal pole is supplied equally by the palmar and dorsal vessels.

Although radial shortening osteotomy for Kienböck's disease with ulnar negative variance has shown good results in patients with this disease [3–9], there have been some reports of ulnar impaction syndrome because of radial shortening in patients with ulnar neutral or positive variance [10–12]. In 1993, Almquist et al. [13] introduced a technique of capitohamate fusion with capitate shortening osteotomy for Kienböck's disease with ulnar neutral or positive variance. The purpose of capitate shortening osteotomy is to decompress the lunate by transverse cutting and shortening of the waist of the capitate and distributing the stress to the scaphotrapeziotrapezoid (STT) joint and the triquetrohamate joint. Viola et al. [14] demonstrated a 25% decrease after a 2.9-mm capitate shortening. Horii et al. [15] reported that a 4-mm capitate shortening with capitate–hamate fusion decreased the radiolunate pressure by 66%. Judging from these reports, capitate shortening osteotomy can be expected to have a sufficient lunate decompression effect to prevent osteonecrosis. Other advantages are that the procedure is relatively easy and does not block blood flow to the lunate. In 2009, Gay et al. [16] performed capitate shortening osteotomy in 11 patients with Lichtman stages I–IIIA of Kienböck's disease (ulnar neutral variance cases) and reported good clinical outcomes (follow-up period: mean 67.4 months). In capitate shortening osteotomy (Fig. 11.1), a longitudinal

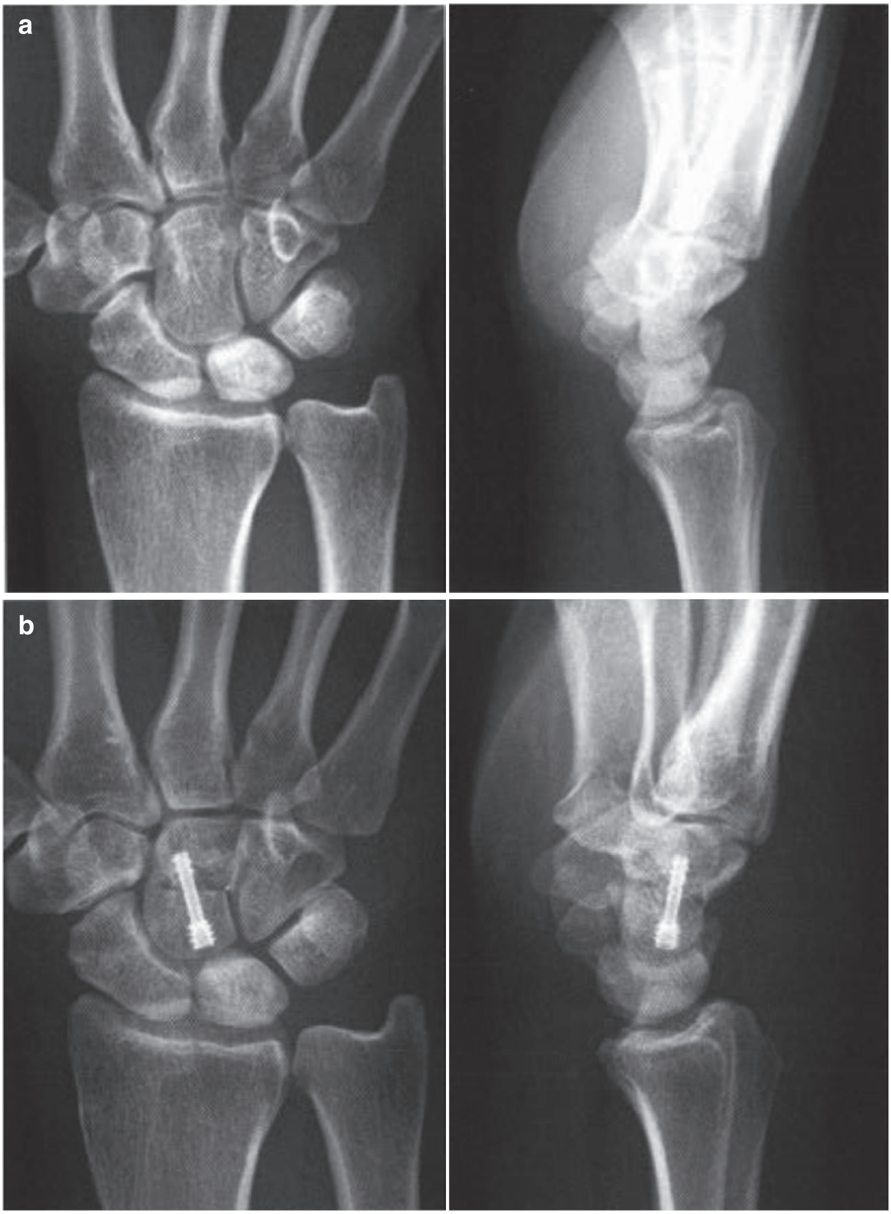

Fig. 11.1 Capitate shortening osteotomy (38-year-old woman, Lichtman stage II) (**a**) Preoperatively. (**b**) Immediately after surgery. (**c**) 2 years postoperatively (Reproduced with permission Matsui Y, Iwasaki N. MB Orthop. 27(4): 59–65, 2014)

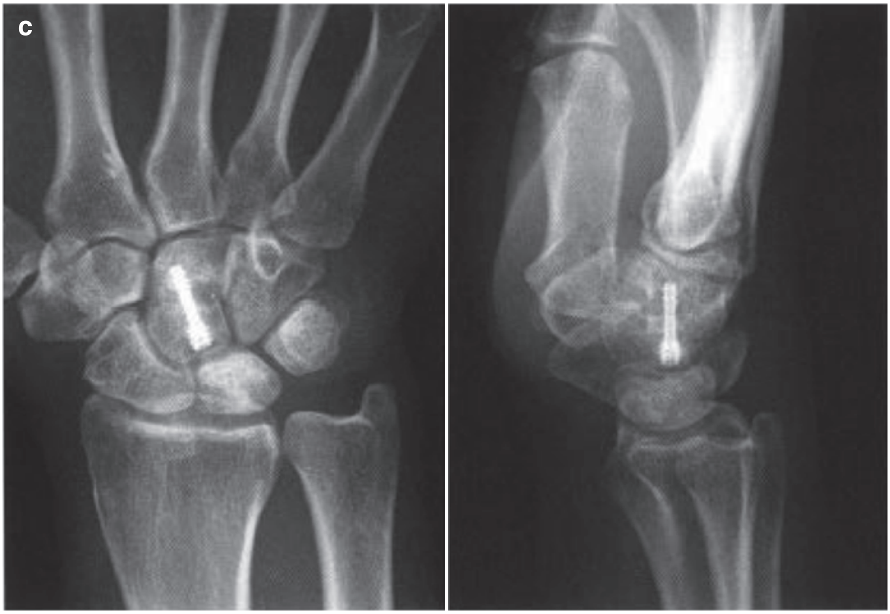

Fig. 11.1 (continued)

skin incision of about 3 cm is added to the dorsal side of the capitate, the extensor tendons are separated, and the wrist joint capsule and the dorsal intercarpal (DIC) ligament are longitudinally cut. The capitate is exposed, excised, and shortened by about 2 mm perpendicular to the long axis in the center, and fixed with one or two headless screws from the proximal side. The DIC ligament is repaired and the wrist is immobilized with a cast for 2–4 weeks after surgery. A method of inserting a screw from the distal side has also been reported [17]. In 2018, Li et al. [18] reported capitate osteotomy and transposition combined with an autologous iliac bone graft for 17 patients with stage III Kienböck's disease. Bone union failure occurred in one patient, but the clinical results were good in the other patients (follow-up period: mean 68 months), and they concluded that it was an effective surgical procedure for patients with stage III disease. In 2021, Hunter et al. [19] reported good short- to medium-term outcomes (follow-up period: mean 40 months) for seven patients with ulnar positive or neutral wrists (stages II–IIIB). who were treated by capitate shortening osteotomy combined with a 4 + 5 extensor compartmental artery vascularized bone graft placed in the lunate.

In 2004, Moritomo et al. [20] first devised and reported partial shortening osteotomy of the capitate. The purpose of this procedure was to reduce the effect on the carpal bone arrangement by partially shortening the capitate only on the articular surface with the lunate, preserving the scaphocapitate articular surface. In 2015, Citlak et al. [21] showed good clinical outcomes (follow-up period: mean 38 months) for seven patients with Lichtman stages II and IIIA who were treated by partial capitate

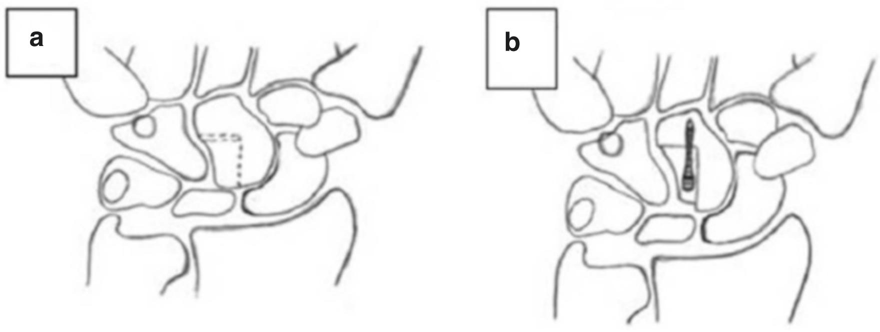

Fig. 11.2 Diagram of the partial capitate shortening osteotomy. (**a**) Osteotomy lines. (**b**) After shortening and fixation of the osteotomy with a screw. Reproduced from [21] with permission

shortening osteotomy (Fig. 11.2). Recently, Yıldırım et al. [22] performed the same operation for a total of 19 patients (12 with Lichtman stage II, 7 with stage IIIA), and reported good clinical results (follow-up period: 16 months on average). Among the midcarpal joints, the scaphoid has three articular surfaces: the trapezium, the trapezoid, and the capitate. When a long-axis load is applied to the hand, the force applied to the scaphoid is distributed to these three surfaces, and the scaphoid moves while all three joints are adjusted. Based on three-dimensional research on the anatomy and kinematics of the midcarpal joint [23], when the STT joint, scaphocapitate joint, and triquetrohamate joint remain, they considered that normal carpal movements and carpal arrangements could be maintained without articular contact between the capitate and the lunate. Considering these concepts, they performed partial capitate shortening osteotomy to preserve the scaphocapitate joint. However, because of the possibility of postoperative osteoarthritis of the radioscaphoid and lunocapitate joints, caution should be exercised when osteoarthritis is present preoperatively in the proximal capitate articulation or radiolunate joint. As in the capitate shortening osteotomy, a 3-cm longitudinal skin incision is made on the dorsal side, and the capitate is approached. The capitate has separate articular surfaces for the scaphoid and lunate bones, and there is a clear cartilaginous ridge at the border of the articular surfaces. An F-shaped osteotomy is added so that the vertical osteotomy line is on this ridge, shortening the bone by about 2 mm. The proximal fragment is pushed distally and fixed with a single screw, and the cartilage at the step-off is trimmed with a scalpel. Postoperative immobilization of the wrist is maintained for 4 weeks after surgery.

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Surgical Treatments: Limited Intercarpal Fusion

12

Norimasa Iwasaki

Abstract

The aims of limited intercarpal fusions for the treatment of Kienböck's disease are to reduce the load on the diseased lunate, correct the rotational deformity of the scaphoid, and restore carpal height. These surgical effects result in a decrease in wrist pain and increase in grip strength. Although several procedures have been used to achieve these aims, the surgical efficacy of each procedure is still debatable. Biomechanically, it is reasonable to conclude that fusions across both carpal rows, such as triscaphe (scaphotrapezium-trapezoid) fusion (STT fusion) or scaphocapitate (SC) fusion (SC fusion), effectively decompress the lunate by preventing capitate migration toward the lunate. Therefore, biomechanical studies suggest that capitohamate (CH) fusion (CH fusion) does not unload the lunate. Lunate decompression by STT and SC fusions is achieved by shifting the load from the radiolunate to the radioscaphoid joint. The excessive load at the radioscaphoid joint after these fusions is considered to result in progression of degenerative changes at this joint. To achieve favorable effects regarding unloading the lunate, the author recommends STT and SC fusions for advanced Kienböck's disease. However, the increase in the force transmission through the radioscaphoid joint may lead to osteoarthritic changes postoperatively.

Keywords

Limited intercarpal fusion · Capitohamate fusion · Scaphocapitate fusion · Triscaphe (scaphotrapezium-trapezoid) fusion · Advanced Kienböck's disease

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In the advanced stages of Kienböck's disease, the scaphoid rotates palmarly and the capitate migrates proximally into the space occupied by the lunate. The purposes of limited intercarpal fusions for the treatment of Kienböck's disease are to reduce the load on the lunate, correct the rotational deformity of the scaphoid, and restore carpal height. These surgical effects lead to a reduction in wrist pain and increase in grip strength while preserving wrist motion. Although several procedures have been used to achieve these purposes, the surgical efficacy of each procedure is still debatable.

Chuinard first described capitohamate (CH) fusion for the treatment of Kienböck's disease [1, 2], and showed that CH fusion was successful in 41 of 43 patients. Oishi et al. [3] reported the postoperative outcomes of CH fusion in 45 wrists with stage I, II, and III Kienböck's disease. At a mean follow-up of 32 months, 93% of patients had either no pain or less pain than preoperatively, with preservation of wrist range of motion (ROM) and improved grip strength. Therefore, they concluded that CH fusion is effective in patients with stage I to III Kienböck's disease.

In 1946, Sutro [4] introduced scaphocapitate (SC) fusion to treat scaphoid nonunion in four patients. Since this first description, other studies have demonstrated favorable clinical outcomes of SC fusion in the treatment of Kienböck's disease [5–8]. In 1995, Sennwald and Ufenast [5] reported the clinical outcomes of SC fusion in 11 patients (one with Lichtman stage II, four with stage IIIA, and six with stage IIIB). At a mean follow-up of 36 months, all patients except one had complete pain relief, and nine patients had returned to their previous vocations. Luegmair and Saffar [6] reviewed 10 patients who underwent SC fusion for stage IIIB and IV Kienböck's disease with a mean follow-up of 8.75 years. The collapsed lunates were excised in four patients. Scaphocapitate fusion resulted in substantial pain relief, functional wrist ROM, and good grip strength. Radiographic analysis demonstrated maintenance of carpal height with a corrected radioscaphoid angle and no evidence of ulnar translation. However, mild-to-moderate secondary osteoarthritic changes of the radioscaphoid joint were often found in patients with a follow-up of more than 10 years. The authors suggested that excision of diseased lunate did not affect the clinical and radiological outcomes. Rhee et al. [7] reported the outcomes of SC fusion in 27 patients with stage III (10 with stage IIIA and six with stage IIIB) and IV (11 patients). Subtotal lunatectomy was performed on 12 wrists. At a mean follow-up of 60 months, postoperative wrist motion was significantly decreased in flexion (14°), extension (11°), and ulnar deviation (9°). However, grip strength was significantly improved postoperatively (+7 kg). Although the modified Mayo wrist scores were mostly fair to poor and Lichtman scores were satisfactory in 32% of patients, 74% of patients returned to regular employment. Radiographic progression of carpal collapse and ulnar translation of the carpus were found, particularly in patients who underwent subtotal lunatectomy. However, the authors stated that lunate management, with or without lunate excision, did not appear to affect clinical outcomes. Collon et al. [8] evaluated the outcomes of SC fusion (without lunate excision) for advanced Kienböck's disease (4 stage IIIA wrists and 13 stage IIIB wrists). After a mean follow-up of 5.8 years, there was a significant reduction in wrist pain and an improvement in grip strength (+10 kg). In contrast to other studies, there was also a significant postoperative improvement in wrist flexion (+10°) and extension (+10°). The average Disabilities of the Arm, Shoulder, and Hand (DASH) and Patient-Related Wrist Evaluation (PRWE) scores were 19 points (range, 2–61) and 23 points (range, 0–77), respectively. The outcome was rated as very good in eight patients, good in seven, and fair in two. While the radiographic carpal height ratio significantly decreased, the radioscaphoid and scapholunate angles were restored to within normal ranges. These studies suggest that SC fusion has clinical benefits in the treatment of advanced-stage Kienböck's disease. Radiographically, there is possible development or progression of secondary osteoarthritic changes at the radioscaphoid joint. However, these radiographic changes do not contribute to the clinical outcomes.

In 1986, Watson et al. [9] described triscaphe (scaphotrapezium-trapezoid) fusion (STT fusion) for the treatment of Kienböck's disease. This procedure reestablishes the radioscaphoid relationship and protects the diseased lunate from the excessive load. Watson et al. [9] evaluated the outcomes of STT fusion for stage III Kienböck's disease; eight patients underwent STT fusion with silicone rubber lunate replacement, while another eight were treated with STT fusion alone. At a mean follow-up of 20.5 months, all patients achieved relief of wrist pain. The mean grip strength on the affected side was 86.3% of that on the unaffected side. The average postoperative extension of the affected wrist was 42.4°, which was 143.2% of the average preoperative extension. The average postoperative wrist flexion was 46°, which was 120.7% of the average preoperative flexion. The average radial deviation/ulnar deviation values of the affected and unaffected wrists at the final follow-up were 19.3°/25.5° and 31.5°/36.6°, respectively. The postoperative outcomes were rated as satisfactory based on criteria of Lichtman et al. [10] in 15 of 16 patients. Radiographic examination showed no degenerative changes in the remaining joints of all patients. Meier et al. [11] reviewed the outcomes of STT fusion in 59 patients with Kienböck's disease (one with stage II, 14 with stage IIIA, 35 with stage IIIB, and nine with stage IV). At an average follow-up of 4 years, the pain scores were significantly improved. The average total arc of wrist extension/flexion was 67° (81% of the preoperative arc) and that of ulnar/radial deviation was 31° (56% of the preoperative arc). The average grip strength significantly improved from 45 kPa preoperatively to 52 kPa postoperatively. The average modified Mayo wrist score was 63 points. Radiographically, radioscaphoid degeneration was found in 13 of 59 patients. Acar et al. [12] reported the results of STT fusion using a limited wrist fusion plate in nine patients with stage IIIB Kienböck's disease. At a mean follow-up of 18.4 months, all patients were free of night pain. The wrist motion was limited compared with the contralateral wrist in both extension/flexion and ulnar/radial deviation. The grip strength was significantly improved postoperatively. Based on these studies, we may reasonably conclude that STT fusion provides relief of wrist pain, improves grip strength, and preserves acceptable wrist motion. However, osteoarthritic changes occur or progress at the radioscaphoid joint after STT fusion.

Previous biomechanical studies have clarified the biomechanical effects of limited wrist fusions on lunate decompression. In a cadaveric study using a strain gauge, Trumble et al. [13] demonstrated significant lunate decompression after STT fusion, but not after CH fusion. Horii et al. [14], using a two-dimensional rigid body spring model (RBSM), showed that STT and SC fusions unloaded the lunate by 5% and 12%, respectively. In contrast, CH fusion did not unload the lunate. Iwasaki et al. [15] developed a three-dimensional (3D) theoretical model using the discrete element analysis technique to simulate limited wrist fusion. In the theoretical analysis, a significant decrease in the lunate compressive force was observed following SC and STT fusions but not following CH fusion (Fig. 12.1). Florenz et al. [16] also, using a 3D RBSM, clarified the biomechanical efficacy of SC and STT fusions for lunate decompression. Their 3D theoretical analysis demonstrated that STT and SC fusions increased the load through the radioscaphoid joint. A cadaveric study by Short et al. [17] supported the results of these theoretical analyses of STT fusion. Based on the studies, it is reasonable to conclude that fusions across both carpal rows, such as STT or SC fusions, effectively decompress the lunate by preventing capitate migration toward the lunate. Lunate decompression is achieved by shifting the load from the radiolunate to the radioscaphoid joint.

The excessive load at the radioscaphoid joint after STT and SC fusions is considered to result in early progression of degenerative changes at this joint. Fortin and Louis [18] demonstrated that 6 of 14 patients had progressive degeneration after

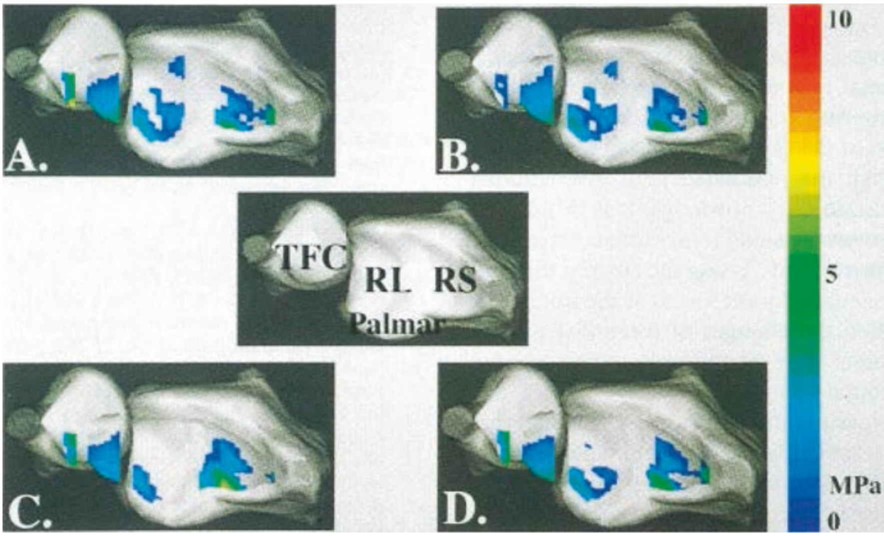

Fig. 12.1 Force distribution at the radiocarpal joint and ulnocarpal space. *RS* radioscaphoid joint, *RL* radiolunate joint, *TFCC* triangular fibrocartilage. (**A**) Normal; (**B**) capitohamate (CH) fusion, (**C**) scaphocapitate (SC) fusion; (**D**) scaphotrapezium-trapezoid (STT) fusion. A significant decrease in the lunate compressive force is observed following SC and STT fusions but not following CH fusion. From Iwasaki N, et al.: J Orthop Res 16(2): 256-263, 1998, Fig. 6

STT fusion. Regarding the scaphoid position, Short et al. [17] demonstrated that STT fusion with the scaphoid in a neutral or extended position unloaded the lunate fossa in a simulated cadaveric model of Kienböck's disease, while STT fusion with the scaphoid in flexion did not change the load on the lunate. Minami et al. [19] reported that the progression of osteoarthritis after STT fusion was the main negative factor influencing clinical outcomes. Therefore, they recommended a radioscaphoid angle of less than 60° flexion to prevent progressive degenerative changes at the radioscaphoid joint. In performing STT fusion, overcorrection may be a risk factor contributing to the development of osteoarthritis at the radioscaphoid joint by excessive load distribution through this joint.

A considerable complication of limited intercarpal fusion is nonunion at the fusion site. The reported incidences of nonunion after limited intercarpal fusion are 15% [11] and 4% [20] after STT fusion, and 12% [21] and 10% [6] after SC fusion. Minami et al. [22] reported that nonunion results from inadequate removal of the articular cartilage at the fusion site. However, some studies report excellent union rates after STT and SC fusions [5, 9, 19, 23–25]. One of the most critical factors in achieving solid union is stability at the fusion site. Limited intercarpal fusions are performed using various fixation implants and techniques. Acar et al. [12] suggested that limited wrist fusion plates that provide biomechanical stability effectively increase the union rate at the fusion site (Fig. 12.2).

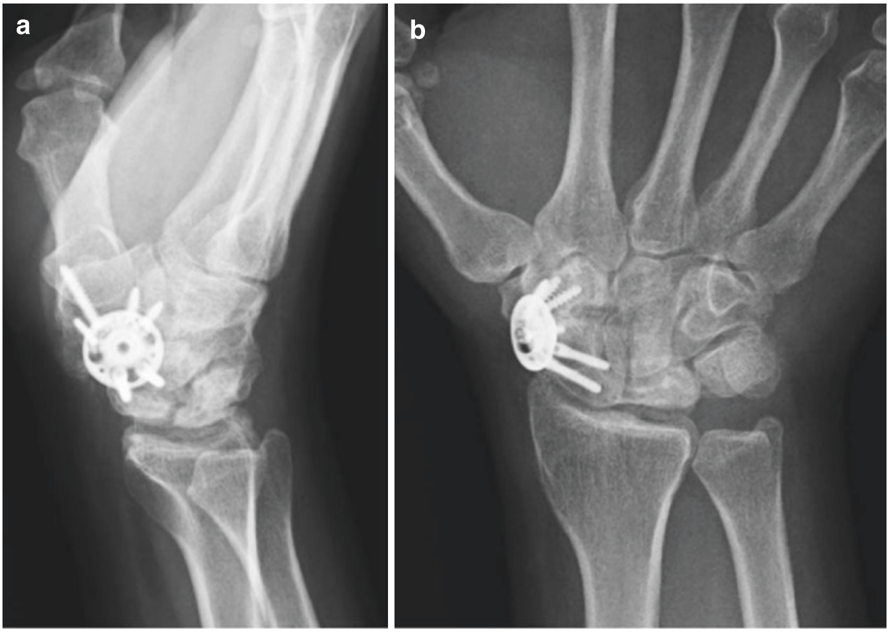

Fig. 12.2 Lateral (**a**) and anteroposterior (**b**) wrist radiographs at 18 months after STT fusion using a limited wrist fusion plate limited. From Acar B, et al.: Cureus 11(2):e4025, 2019, Fig. 2(a), (b)

To achieve favorable effects regarding unloading the lunate, the author recommends STT and SC fusions for advanced Kienböck's disease. However, the increase in the force transmission through the radioscaphoid joint may result in osteoarthritic changes after these fusions.

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Surgical Treatments: Lunate Excision

13

Norimasa Iwasaki

Abstract

Lunate excision arthroplasty is one of the first surgical procedures considered for the treatment of Kienböck's disease. This procedure is generally performed for patients with lunate fragmentation or a severely collapsed lunate without pan-osteoarthritic changes throughout the wrist. Based on the radiographic staging system by Lichtman, lunate excision arthroplasty is considered suitable for patients with stage III (especially stage IIIB and IIIC) and stage IV Kienböck's disease, especially for those with osteoarthritis localized at the radioscaphoid joint. Some studies report good clinical outcomes after simple lunate excision without interposing any biological or synthetic material in the remaining space for Kienböck's disease. In contrast, other studies also show that lunate excision without replacement arthroplasty accelerates carpal collapse. To avoid such anatomical disruption, lunate excision with replacement arthroplasty has been developed. The procedures are classified into two categories: replacement arthroplasty using a prosthesis, and replacement arthroplasty using biological tissue. Historically, the most common type of prosthesis is a silicone rubber implant. However, some reports showed radiographic findings indicating wear-particle synovitis in response to the silicone rubber prosthesis postoperatively. Therefore, silicone replacement arthroplasty for Kienböck's disease is discouraged. Regarding replacement arthroplasty using biological tissue, the most common procedure seems to be palmaris longus tendon replacement. Previous clinical studies suggest that lunate excision arthroplasty using the palmaris longus tendon provides acceptable clinical outcomes for advanced Kienböck's disease,

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despite the radiographic progression of pathological changes, such as carpal collapse or osteoarthritic changes. To prevent these pathological changes, the author recommends lunate excision arthroplasty using a palmaris longus tendon ball with an osseous core for advanced Kienböck's disease.

Keywords

Lunate excision arthroplasty · Synthetic material · Biological tissue material · Silicone implant · Palmaris longus tendon · Advanced Kienböck's disease

Many types of surgery have been advocated for symptomatic Kienböck's disease. However, there is no consensus about the appropriate procedure for each stage of the disease. Lunate excision arthroplasty is one of the first surgical procedures considered for the treatment of Kienböck's disease. Although several reports have shown acceptable outcomes of lunate excision arthroplasty for stage IV Kienböck's disease [1–3], this procedure is generally indicated for patients with lunate fragmentation or a severely collapsed lunate without pan-osteoarthritic changes throughout the wrist.

Some studies report good clinical outcomes after simple lunate excision without interposing any biological or synthetic material in the remaining space for Kienböck's disease [4–7]. Blanco [7] demonstrated favorable results regarding wrist pain and stiffness in 11 of 13 patients at more than 5 years after simple lunate excision; postoperative radiographs show palmar flexion of the scaphoid and proximal migration of the capitate. Other studies also report that lunate excision without replacement arthroplasty accelerates carpal collapse, leading to disruption of the carpal architecture [8, 9]. Although simple lunate excision provides acceptable clinical outcomes by decreasing painful synovitis, the proximal migration of the capitate progresses postoperatively. To avoid such anatomical disruption, lunate excision with replacement arthroplasty was developed. Since the application of vitallium prostheses by Lippman in 1949 [10], several lunate excision and replacement procedures have evolved. The procedures are classified into two categories: replacement arthroplasty using a prosthesis, and replacement arthroplasty using biological tissue.

Historically, the most common type of prosthesis is a silicone rubber implant. In 1977, Lichtman et al. [11] introduced the clinical results reported by Ståhl that over 50% of patients with Kienböck's disease are improved or cured after wrist immobilization for more than 2 months; however, 19 of 22 patients treated with immobilization had unsatisfactory results, and 17 of the 22 patients had progressive collapse of the diseased lunate during immobilization. Alternatively, Lichtman et al. performed a silicone replacement arthroplasty. In 1982, they reported satisfactory outcomes in 15 of 16 patients treated with silicone replacement arthroplasty for stages II and III Kienböck's disease at an average of 18 months postoperatively [12]. Kato et al. [1] also reported outcomes of 19 patients at 8 years 2 months after excisional arthroplasty with a silicone implant. Patients with mild stages of the preoperative collapse (carpal height index (CHI) $\geq$ 0.93) achieved good clinical outcomes, while patients with advanced stages (CHI $<$ 0.93) had unsatisfactory outcomes because of the postoperative progression of osteoarthritic changes or subluxation of the prosthesis. In 1990, Alexander et al. [13] presented a 5-year follow-up of 10 patients who underwent lunate silicone arthroplasty for stages II and III Kienböck's disease. Contrary to the previous reports [11, 12], only 5 of 10 patients achieved satisfactory results. Three of the five patients had radiographic evidence of wear-particle synovitis in response to the silicone rubber prosthesis at follow-up [14–17]. Other surgical procedures, such as leveling procedures or direct revascularization techniques, provide the promise of good results while preserving the lunate. Therefore, silicone replacement arthroplasty (without intercarpal fusion) for Kienböck's disease is discouraged.

Several studies have reported prosthesis alternatives such as pyrocarbon or titanium materials [10, 18–20]. Ma et al. [20] recently developed a varisized three-dimensional (3-D) printed lunate prosthesis for advanced (stage III/IV) Kienböck's disease. Based on a preoperative surgical simulation using the 3D-printed photosensitive resin model, the lunate prosthesis precisely matches the wrist of each patient. The prosthesis is made from titanium alloy (Ti-6Al-4 V9) using electron beam melting technology. Lunate excision followed by 3D printing prosthetic arthroplasty was performed in five patients with advanced Kienböck's disease (one with stage IIIA, two with stage IIIB, one with stage IIIC, and one with stage IV). All except one patient achieved either very satisfactory (three patients) or satisfactory (one patient) outcomes at a mean of 19.4 months (range, 11–33 months) postoperatively. At follow-up, imaging showed that the implant prosthesis remained in anatomical position, with no dislocation or subluxation. No considerable complications occurred during follow-up.

Regarding replacement arthroplasty using biological tissue, Caroll [21] reported the application of fascial replacement after excision of the lunate to treat Kienböck's disease. The material for replacement was fascia lata from the lateral femoral site, palmaris longus tendon, long toe extensors, and half of the flexor carpi radialis. The long-term (10 years or more) outcomes of 10 patients showed that this procedure was very successful in resolving pain and increasing the wrist range of motion (ROM). Radiographs of the wrist showed no carpal collapse. Currently, the most common procedure seems to be palmaris longus tendon replacement [1, 2, 22]. In 1986, Kato et al. [1] reported that 13 patients with mild carpal collapse (CHI $\geq$ 0.93) who underwent coiled palmaris longus tendon replacement achieved good long-term (3–11 years) clinical results; however, significant carpal collapse was radiographically observed, indicating that a coiled palmaris longus tendon is a weak supportive spacer in patients with mild preoperative carpal collapse. In 1999, Ueba et al. [2] reported the results of lunate excision and subsequent replacement with a tendon-ball implant for advanced Kienböck's disease. The tendon-ball was created from the palmaris longus tendon and the proximal half of the plantaris tendon. The long-term (average, 16 years and 3 months) results of 15 patients (1 with stage II, 10 with stage III, and 4 with stage IV) were excellent in nine patients and good in six patients using Dorman's classification [23]. Radiographs showed postoperative calcification at the site of the implanted tendon in several patients. However, the presence of calcification was not relevant to the clinical results. Ueba et al. [2] emphasized that both the palmaris longus and plantaris tendons should be utilized to create a tendon-ball implant sufficient to fill the space left by the removed lunate. In 2011, Matsuhashi et al. [22] reported excellent results of lunate excision arthroplasty using a palmaris longus tendon ball with osseous core in 12 patients with advanced Kienböck's disease (6 with stage IIIA, 5 with stage IIIB, and 1 with stage IV). Although the carpal height ratio showed no significant progression postoperatively, two patients had osteoarthritic progression in the radiocarpal joint.

As mentioned above, lunate excision arthroplasty is considered suitable for patients with stage III (especially stage IIIB and IIIC) and stage IV Kienböck's disease, especially for those with osteoarthritis localized at the radioscaphoid joint. The abovementioned clinical studies suggest that lunate excision arthroplasty using soft tissue (mainly the palmaris longus or/and plantaris tendon) provides acceptable clinical results for advanced Kienböck's disease, despite the radiographic progression of pathological changes, such as carpal collapse or osteoarthritic changes. Regarding replacement arthroplasty using a prosthesis, silicone replacement arthroplasty is not currently considered appropriate for advanced Kienböck's disease. Other artificial materials, such as titanium alloy, are expected to provide better clinical outcomes and prevent the disruption of carpal architecture. Based on our experience and previous studies, we recommend lunate excision arthroplasty using a palmaris longus tendon ball with or without an osseous core for advanced Kienböck's disease.

In younger or active patients with advanced Kienböck's disease, the ideal treatment strategy is to preserve the carpal anatomy after resection of the diseased lunate. For patients with an intact capitate and lunate facet, Pet et al. [24] reconstructed the proximal lunate using a vascularized medial femoral trochlear (MFT) flap after resecting the proximal part of the lunate in 18 patients with Lichtman stage IIIA (11 patients) and IIIB (seven patients) between 2011 and 2017. The mean age at the time of surgery was 28.4 years. Eight of these patients were directly examined. The mean follow-up periods for physical and radiographic examinations were 2.2 and 1.4 years, respectively. At final follow-up, the patients demonstrated acceptable levels of function, pain, and wrist ROM. Radiographic findings showed a cessation of radiocarpal collapse. Although the conclusions of this study are based on data from a small sample size and short follow-up, MFT reconstruction of the proximal lunate is an alternative to lunate excision arthroplasties [24, 25].

13.1 Author's Preferred Technique [1, 22]

The author has carried out lunate excision arthroplasty using a palmaris longus tendon ball with an osseous core for advanced Kienböck's disease [22]. The operation is mainly performed under general anesthesia. A 5-cm longitudinal skin incision is made on the dorsal center of the wrist joint. The extensor retinaculum is incised longitudinally between the third and fourth extensor compartments. The posterior interosseous nerve is segmentally resected at the level of the radiocarpal joint. After incising the capsule, the lunate is removed piecemeal with great care to prevent injury of the palmar wrist ligaments. Synovectomy is usually performed for the surrounding synovial tissue. The removed lunate is trimmed into the shape of the osseous core. In patients with severe lunate collapse, the osseous core is harvested from the ipsilateral iliac crest. The ipsilateral palmaris longus tendon is then harvested using a tendon stripper. A tendon ball is created by wrapping the tendon around the osseous core and is then inserted into the empty space left by the excised lunate (Fig. 13.1). To avoid dorsal migration of the tendon ball, the capsule is closed tightly. The extensor retinaculum is then repaired. Finally, the subcutaneous tissue and skin are closed. A below-elbow splint is applied for 2 weeks postoperatively.

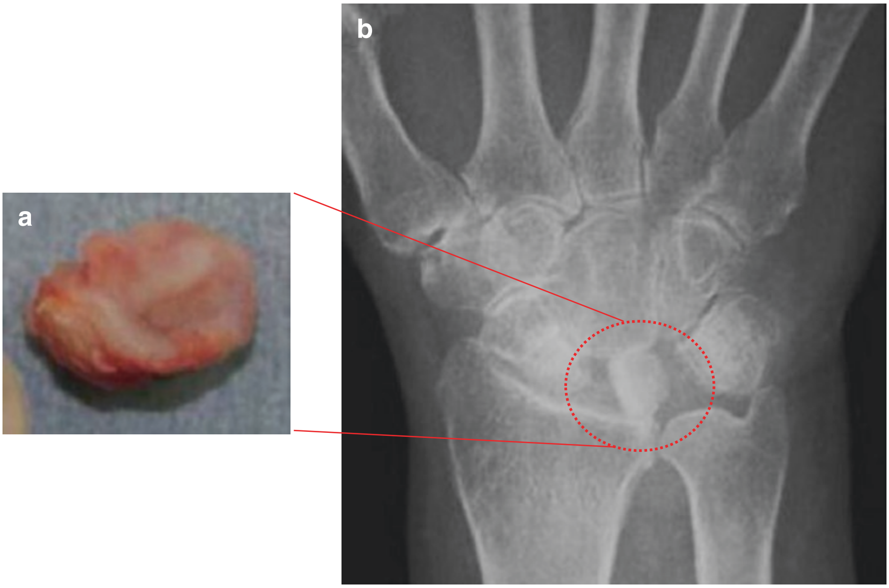

Fig. 13.1 A tendon ball is created by wrapping the tendon around the osseous core (**a**) and is then inserted into the empty space left by the excised lunate (**b**). Modified from Matsuhashi T, et al.: Hand Surg 16:277-282, 2011, Fig. 1

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Surgical Treatments: Salvage Operations

14

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Abstract

Severe Kienböck's disease is treated with a salvage operation, such as total wrist fusion, proximal row carpectomy (PRC), or wrist denervation. Although total wrist fusion results in pain relief and stability of the wrist, the range of wrist motion is completely lost. Wrist denervation is successful in relieving wrist pain. However, this procedure cannot improve wrist function. Consequently, the indication of these two procedures for severe Kienböck's disease is extremely limited. Proximal row carpectomy involves excision of the proximal carpal row, including the lunate, scaphoid, and triquetrum. The candidates for PRC are patients having a painful wrist with a limited wrist motion and decreased grip strength. Although the occurrence or progression of secondary osteoarthritis at the radiocapitate articulation is frequently found after performing PCR, this procedure mostly leads to satisfactory clinical outcomes. The current consensus on PRC as a salvage operation for Kienböck's disease is that this procedure provides acceptable clinical outcomes for patients with advanced stages, including stage IIIB and IV. On the other hand, most patients develop radiocapitate osteoarthritic changes postoperatively. If severe complications occur, total wrist fusion or total wrist arthroplasty are viable options.

Keywords

Salvage operation · Total wrist fusion · Proximal row carpectomy · Wrist denervation · Severe Kienböck's disease

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Severe Kienböck's disease is managed with a salvage operation, such as total wrist fusion, proximal row carpectomy (PRC), or wrist denervation. Although total wrist fusion may provide pain relief and stability of the wrist [1–3], the range of wrist motion is completely lost. Furthermore, although wrist denervation is successful in relieving wrist pain [3–6], this procedure cannot improve wrist function. Therefore, the indication of these two procedures for advanced Kienböck's disease is extremely limited.

In 1944, Stamm [7] reported the excision of the proximal carpal row as a motion-sparing procedure for the treatment of wrist arthritis. The candidates for PRC are patients having a painful wrist with a limited wrist motion and decreased grip strength. However, some studies have reported that PRC results in loss of wrist motion, weakening of grip strength, and poor functional outcomes [8, 9]. In contrast, recent clinical studies have demonstrated pain relief and functional improvement following PRC [10–12]. This procedure involves excision of the proximal carpal row, including the lunate, scaphoid, and triquetrum. To achieve satisfactory results, the proximal articular surface of the capitate and lunate fossa of the distal radius must be preserved to create a novel radiocapitate articulation [13–15]. The occurrence or progression of secondary osteoarthritis at the radiocapitate articulation is a considerable postoperative complication. Therefore, PRC is relatively contraindicated for patients with radiographic degenerative changes at the proximal articular surface of the capitate or lunate fossa.

Several studies have reported the long-term outcomes of PRC for advanced Kienböck's disease [11, 12, 14, 16, 17]. Lumsden et al. [11] presented the long-term clinical results of PRC with an average 15-year follow-up for Lichtman stage IIIA and IIIB Kienböck's disease. Using the clinical grading scale of Glickkel and Millender [18], PRC achieved excellent or good results in 12 of 13 patients. Compared with preoperative values, postoperative wrist range of motion (ROM) and grip strength improved by an average of 16% and 129%, respectively. Although all patients demonstrated radiographic degenerative changes (usually localized to the radiocapitate articulation in the lunate fossa), there was no correlation between the clinical results and radiographic degeneration. There were no early or late complications and no patient required wrist arthrodesis during follow-up. Croog and Stern [16] presented the clinical and radiographic results after PRC for Lichtman stage III and IV Kienböck's disease. At an average 10-year follow-up, 7 of 18 wrists were pain-free, nine were mildly painful, and two were moderately painful. The average wrist ROM and maximal grip strength were 78% and 87% of the contralateral wrist, respectively. The average modified Mayo wrist score was 84 points, composed of nine excellent, four good, three fair, and two poor results. Radiographic findings at final follow-up demonstrated degeneration of the radiocapitate articulation in 16 of 18 patients. There was no significant association between the radiographic findings and clinical outcomes.

Although most studies reporting the outcomes of PRC for Kienböck's disease were based on a small population [11, 19, 20], this procedure mostly leads to satisfactory clinical outcomes. Biomechanically, the axial load following PRC is transmitted through the novel radiocapitate articulation, resulting in rapid development of degenerative changes at this articulation. Radiographs obtained at a median of 2 years after surgery (range, 9 months to 4 years) revealed a reduced radiocapitate joint space in 5 of 12 wrists (41.7%) and complete loss of the space in one (8.3%) [20]. Croog and Stern [16] emphasized the risk of early symptomatic radiocapitate degeneration in stage IV cases. A systematic review of studies with 10 or more years of follow-up showed that patients who engaged in heavy manual labor were likely to have poorer outcomes after PRC for wrist arthritis [21]. Based on a large cohort of 144 patients with a mean follow-up of 13.4 years, Wagner et al. [17] clarified that improved outcomes after PRC for wrist arthritis were associated with a patient age older than 40 years, nonmanual laborers, and Kienböck's disease. In performing PRC for patients with Kienböck's disease, surgeons should consider these factors to achieve better clinical outcomes.

The current consensus on PRC as a salvage procedure for Kienböck's disease is that this procedure provides acceptable clinical outcomes for patients with advanced stages. However, most patients develop radiocapitate arthrosis postoperatively (Fig. 14.1). Van Heest and House [22] stated that PRC is indicated for patients with stage II and III Kienböck's disease who are not candidates for joint leveling procedures. The articular surfaces of the capitate and the lunate fossa of the distal radius in these stages are intact, theoretically leading to greater longevity after PRC. However, as mentioned above, the degenerative changes at the radiocapitate joint are not correlated with clinical outcomes [22]. Therefore, PRC is suitable for advanced stage Kienböck's disease, including stages IIIB and IV. If severe complications occur, total wrist fusion or total wrist arthroplasty are viable options.

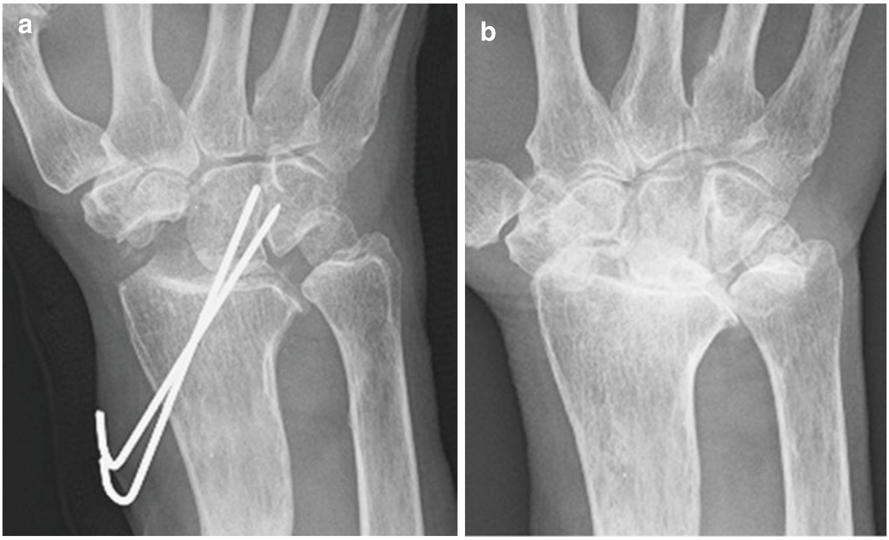

Fig. 14.1 (**a**) Immediately after performing PRC. (**b**) 8 years postoperatively. At 8 years after performing PRC, an X-ray shows the development of radiocapitate arthrosis

14.1 Author's Preferred Technique

The operation is mainly performed under general anesthesia. An oblique longitudinal skin incision is made on the dorsal center of the wrist joint from the second or third carpometacarpal joint distally to the radial side of the ulna proximally. The extensor retinaculum is incised longitudinally between the third and fourth extensor compartments. The posterior interosseous nerve is identified in the floor of the fourth extensor compartment and then segmentally resected at the level of radiocarpal joint. A T-capsulotomy is performed from just distal to the lunocapitate joint to just proximal to the distal end of the radius, with a proximal limb over the scapholunate joint. The diseased lunate, scaphoid, and triquetrum are removed piecemeal with great care to preserve the proximal articular surface of the capitate and the lunate fossa of the radius. Synovectomy is usually performed for the surrounding synovial tissue. The author routinely performs a radial styloidectomy through the same skin incision. The radial styloid is exposed by dissection between the first and second extensor compartments. The styloidectomy is done at the proximal 6–8 mm of the distal end of the radial styloid. The capitate then settles into the lunate fossa of the radius. The author prefers to perform Kirschner wire fixation to immobilize the radiocapitate articulation for 4 weeks (Fig. 14.2). However, some studies have reported that wire fixation is unnecessity [22, 23]. The capsule is closed tightly and then the retinaculum is repaired. A volar plaster splint is applied to maintain the wrist in slight extension for 3 or 4 weeks.

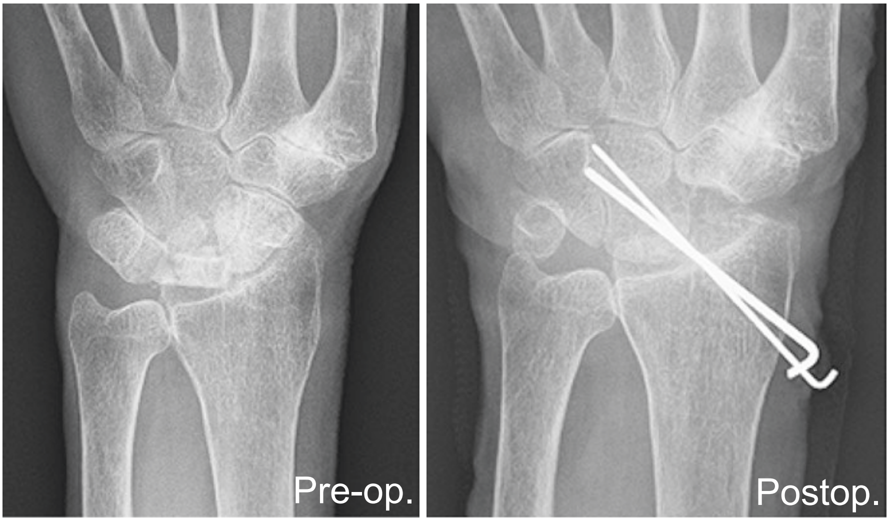

Fig. 14.2 Kirschner wire fixation to immobilize the radiocapitate articulation

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