Wrist Swelling in Kienböck's Disease

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Abstract

Background and Purpose Wrist swelling is a frequent clinical manifestation of Kienböck's disease, but no study has reported the site and pathology of wrist swelling in this disease. The aim of this study is to elucidate the site and pathology of wrist swelling in Kienböck's disease.

Materials and Methods Dorsal and palmar soft tissue thicknesses of the wrist were measured on standard lateral radiographs of the wrist in 26 patients with Kienböck's disease and 30 subjects without intra-articular lesion. Axial magnetic resonance imaging (MRI) views were examined to detect the site of swelling. The dorsal capsular ligament in three patients with Kienböck's disease underwent histological examination.

Results Radiographic study confirmed dorsal wrist swelling in 24 of 26 (92%) patients examined compared with the contralateral unaffected wrists. MRI demonstrated thickening of the dorsal capsular ligament and extensor layer with synovial proliferation. Histological examination revealed nonspecific chronic inflammation.

Conclusion Dorsal wrist swelling in Kienböck's disease is a common manifestation and constitutes a part of pathology of Kienböck's disease, although further study is required to clarify the relation between wrist swelling and etiology of Kienböck's disease.

Level of Evidence This is a Level III study.

Keywords

- wrist swelling
- Kienböck's disease
- inflammation
- capsulitis
- synovitis

Although wrist swelling is a frequent clinical manifestation of Kienböck's disease, no study has reported the site and pathology of wrist swelling in this disease. Empirically, wrist swelling is observed in patients with increasing wrist pain and progressive lunate flattening. Thus, wrist swelling is suspected to play a role in the etiological process or at least in the pathological process of Kienböck's disease. The aim of this study is to elucidate the pathology of wrist swelling in Kienböck's disease, utilizing X-ray and magnetic resonance imaging (MRI).

Materials and Methods

There were three main goals to this study. First, to determine the extent of laterality of the soft tissue thickness in normal wrists, the soft tissue thickness of 30 bilateral wrists without

intra-articular lesions and without soft tissue swelling was measured using standard lateral X-ray images projected with support.¹ From a series of 54 patients, 30 pairs of wrist X-ray images with less than a 3-mm difference in the horizontal distance between the palmar end of the scaphoid and pisiform compared with the contralateral wrist² were selected to minimize the influence of forearm rotation and were included in the study. Second, to determine whether a wrist affected with Kienböck's disease has a thicker soft tissue than that of an unaffected wrist, the soft tissue thickness of the bilateral wrists of 26 patients with unilateral Kienböck's disease was measured using standard lateral wrist X-rays which satisfied the criteria that each pair showed less than a 3-mm difference in horizontal distance between the palmar end of the scaphoid and pisiform. Finally, to determine which site of the wrist

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structure is swollen in Kienböck's disease (i.e., intra-articular, capsular ligament, tendon and sheath, or skin and subcutaneous tissue), measurements in layers were performed on T2-weighted fat-saturated axial view images through the capitate head in 26 wrists with Kienböck's disease and in 30 control wrists without intra-articular lesions and without soft tissue swelling around the lunate (13 patients complained of ulnar wrist pain but did not show remarkable lesions, 11 had de Quervain's disease, 4 had extra-articular palmar wrist ganglion, and 2 had contralateral Kienböck's disease).

Twenty-six patients with Kienböck's disease in stage I, II, or IIIA were selected from a consecutive series of 75 patients. Twenty-three of 75 patients were excluded due to lack of MRI or poor quality of X-rays or MRI. Kienböck's disease in stage IIIB or IV³ was found in 23 patients, and they were excluded from the study. Three patients were excluded due to complications (pseudogout, distal radius fracture in the affected side, and ganglion just dorsal to the lunate, respectively). Of the included 26 patients with Kienböck's disease, 7 had an ulnar variance of >1 mm, 10 had a variance of 0 mm, and 9 had a variance of <1 mm. The time interval between the onset of wrist pain and X-ray examination ranged from 3 to 60 months (average 13 months). Sixteen patients (62%) were examined less than 12 months from the onset of the wrist pain. According to Lichtman and Degnan's classification,³ 4 patients had stage II of the disease and 22 had stage IIIA. There were no patients with stage I Kienböck's disease.

Wrist swelling was estimated by soft tissue thickness from the capitate head to the skin in standard lateral X-rays of the wrist and T2-weighted fat-saturated axial MRI views across the capitate head (**→Figs. 1 and 2**). As the lunate among patients with Kienböck's disease is flattened with an

Fig. 1 Method of measurement of soft tissue thickness on X-rays. (A) Dorsal soft tissue thickness (length of perpendicular line from the capitate head to dorsal skin surface). (B) Palmar soft tissue thickness (length of perpendicular line from the capitate head to palmar skin surface).

Fig. 2 Measurement in layers on magnetic resonance imaging (T2 fat-saturated axial view thorough the capitate head) of a patient with Kienböck's disease. (A) Joint space. (B) Joint capsule to gliding floor of extensors or flexors. (C) Gliding floor to extensor retinaculum, or transverse carpal ligament. (D) Skin and subcutaneous tissue.

increasing diameter of varying degrees from the dorsal to palmar direction, the capitate head level is considered more preferable for the measurement of wrist swelling. The degree of lunate flattening and deformation does not directly affect the measurement at the capitate head level. In fact, the correlation coefficient between the increase of the soft tissue thickness in affected wrists with Kienböck's disease compared with unaffected wrists and the increase of lunate diameter in the affected wrists was 0.121, showing that lunate flattening did not affect the measurement of soft tissue thickness at the capitate head level. Furthermore, a sagittal MRI view of the wrist showed the most remarkable swelling at the capitate head level on preliminary examination (**→Fig. 3**). At the capitate head level, an axial MRI view is sufficient to detect the joint space, the capsular ligament with gliding floor of extensors and flexors, extensors with the extensor retinaculum and flexors with the transverse carpal ligaments, as well as skin with subcutaneous tissue.

MRI studies were performed using a 1.5T MRI scanner (General Electric, Signa Excite, Fairfield, CT) with a slice thickness of 2 mm. All measurements were performed on a high-resolution screen under two- or threefold magnification using software supplied by the 3Z Corporation (Tokyo, Japan). In addition, three specimens intraoperatively obtained from the dorsal capsule of wrists with Kienböck's disease were used for histological examinations.

Statistical Analysis

The obtained data were statistically analyzed using Student's *t*-test or the chi-square test. Differences in averaged value of measurements with a probability (*p*) value of less than 0.05 were considered statistically significant. Interrater reliability between the first and second authors was judged by correlation coefficient in soft tissue measurement on X-rays and MRI.

Fig. 3 T2 fat-saturated axial view and sagittal view in a patient with Kienböck's disease, showing prominent swelling of the capsular ligament with high signal (left, arrows) and showing that the swelling extend from the lunate dorsal to the capitate dorsal.

Results

There were no significant differences in age and sex between patients with Kienböck's disease and controls. Although the number of females and the elderly were higher among patients with Kienböck's disease than among controls, these differences were not statistically significant (►Table 1).

The length of the third metacarpal was measured in both wrists affected with Kienböck's disease and control wrist. The average of the third metacarpal length was similar between the affected and control wrists, with an average difference of 0.7 mm, suggesting similar average hand and wrist sizes between patients with Kienböck's disease and controls (►Table 1).

X-ray measurements could not detect a difference in the average thickness of the dorsal and palmar soft tissue between the right and left wrists in the control group (►Table 2). The average dorsal soft tissue thickness of wrists with Kienböck's disease was greater than that of contralateral unaffected wrists. The dorsal soft tissue was thicker in 24 of 26 patients by more than 1 mm in affected wrists versus that in the unaffected wrists. None showed thinner dorsal soft tissue thickness by more than 1 mm in affected wrists compared with unaffected wrists. However, the average palmar soft tissue thickness of affected wrists was similar to that of unaffected wrists (►Table 3). MRI zonal measurements revealed significantly more swelling of the dorsal capsule and extensor layer in wrists with Kienböck's disease (►Table 4, $p < 0.001$). Radiologist reports available for all 26

patients with Kienböck's disease showed thickening of the dorsal intercarpal ligament or dorsal radiocarpal ligament and surrounding synovia, which extend from the scaphoid to the triquetrum in 24 of 26 patients (►Figs. 2 and 3). There were no significant differences in soft tissue thickness in any other sites.

Interrater reliability between the first and second authors calculated by correlation coefficient was 0.98 in X-ray measurement and 0.80 to 0.94 in MRI zonal measurement. These results showed satisfactory interrater reliability.

Histological examination of the dorsal capsule and gliding floor of the extensors revealed nonspecific chronic

Table 2 Comparison of X-ray soft tissue thickness on between the normal right wrist and left

	Right wrist (mm)	Left wrist (mm)	p-Value
Dorsal	14.1 ± 2.0	14.1 ± 2.0	>0.5
Palmar	18.8 ± 2.2	19.2 ± 2.2	>0.1

Table 3 Comparison of X-ray soft tissue thickness between the affected wrist with Kienböck's disease and contralateral unaffected wrist

	Affected wrist (mm)	Unaffected wrist (mm)	p-Value
Dorsal thickness	15.4 ± 2.0	12.9 ± 2.0	<0.001
Palmar thickness	19.7 ± 2.7	20.0 ± 2.4	>0.5

Table 1 Demographics of patients with Kienböck's disease and controls

	Patients with Kienböck's disease	Controls	p-Value
Gender	Man 8, woman 18	Man 13, woman 17	>0.1 (chi-square test)
Averaged age ± SD	46.4 ± 19.6 y	43.9 ± 14.5 y	>0.5
Averaged third metacarpal length (SD)	60.6 ± 3.9 mm	61.2 ± 3.8 mm	>0.5

Abbreviation: SD, standard deviation.

Table 4 MRI measurements in layers (mm)

	Articular Bone to capsule	Capsule Capsule to gliding floor	Tendon Gliding floor to carpal ligament	Skin Carpal ligament to skin	Total
Kienböck's dorsal	1.2 ± 0.5	5.8 ± 1.2	4.0 ± 0.9	3.6 ± 1.1	14.5 ± 2.0
Control	1.0 ± 0.3	4.4 ± 0.9	2.8 ± 0.9	3.5 ± 1.2	11.7 ± 1.6
p-Value	>0.05	<0.001	<0.001	>0.5	<0.001
Kienböck's palmar	1.0 ± 0.6	2.2 ± 0.7	9.1 ± 1.5	6.7 ± 2.1	19.0 ± 2.6
Control	0.8 ± 0.2	2.6 ± 0.9	9.0 ± 1.7	6.7 ± 1.7	19.1 ± 1.8
p-Value	>0.05	>0.05	>0.5	>0.5	>0.5

Abbreviation: MRI, magnetic resonance imaging.

inflammation with lymphocyte infiltration and angiogenesis consistently in all three collected specimens (►Fig. 4).

Discussion

Most cases of Kienböck's disease have been considered idiopathic and the etiology has not been identified. On the contrary, many cases of femoral head necrosis are primarily caused by femoral neck fracture, corticosteroid administration, or alcoholism. Therefore, it is more difficult to clarify a primary etiology and to follow the pathological process in Kienböck's disease than in femoral head necrosis. We believe that detailed examination of clinical findings is important to find a clue to the etiology and detailed pathology.

The results of this study revealed that soft tissue thickness at the capitate head level was similar between the normal right wrist and the normal left wrist, possibly because of a minimum amount of muscular structure at the capitate head level.

Measurements using X-ray images confirmed that the average dorsal soft tissue thickness was greater in wrists affected with Kienböck's disease than in contralateral unaffected wrists. However, the palmar soft tissue thicknesses were similar between the affected and contralateral unaffected wrists. As the dorsal soft tissue thickness in most

affected wrists was at least 1 mm greater than that in contralateral normal wrists, dorsal wrist swelling may be a common feature of Kienböck's disease. Furthermore, MRI zonal measurements showed swelling of both the dorsal wrist capsule and extensor layer. Therefore, wrist swelling in Kienböck's disease may not be caused by intra-articular lesions but by periarticular lesions.

Histological analysis confirmed chronic inflammation around the dorsal intercarpal ligament with synovitis, together with MRI findings which showed the site of the swelling, and the inflammation is believed to be dorsal capsulitis and extensor tenosynovitis (►Figs. 2 and 3).

Limitations of this study are the lack of patients with stage I, although detection of patients with stage I of the disease is extremely difficult, and that most control patients complained of radial wrist pain or ulnar wrist pain, but there are no remarkable changes around the lunate. Another limitation is the small number of histological examinations. Three-phase bone scans of wrists with Kienböck's disease had been reported to show unexpected isotope accumulation not only in the lunate but also the surrounding carpus on pool images⁴ and rarely shows a cold spot in the lunate.^{5,6} The dorsal capsulitis and synovitis are suspected to collect extensive accumulations of isotopes.

As extra-articular lesions are known causes of dorsal wrist swelling, the development of such lesions may represent a peculiar pathology of Kienböck's disease. It is difficult to determine whether this inflammatory process is etiologically a primary or an associated change. Empirically, considerable wrist swelling associated with increasing wrist pain and progressive flattening of the lunate are hallmarks of Kienböck's disease. Therefore, dorsal wrist capsulitis and tenosynovitis are suspected to be at least an aggravating factor of lunate necrosis. Lunate osteonecrosis identical to Kienböck's disease has been reported in patients with sustained lunate dislocations.⁷ Moreover, flattening of the lunate, possibly due to inflammation around the lunate, is sometimes noted in rheumatoid arthritis of the wrist.⁸⁻¹⁰ Therefore, vascular insufficiency caused by trauma or inflammation around the lunate could cause lunate necrosis. These findings suggest that dorsal capsulitis is possibly engaged in the etiological process of Kienböck's disease.

Fig. 4 Specimen obtained from dorsal capsular ligaments, showing proliferated synovia with lymphocyte infiltration (hematoxylin-eosin, ×40).

On the contrary, Gelberman et al found dorsal and palmar arterial blood supply in 80% of the lunates with Kienböck's disease and 20% had nutrient vessels entering only from the palmar surfaces.^{11–13} Their findings indicate that isolated loss of the dorsal arterial blood supply to the lunate does not necessarily yield bone necrosis if the loss of arterial blood supply is the primary cause of Kienböck's disease. Most authors considered that Kienböck's disease is caused by the loss of the arterial blood supply to the lunate until the last decade of the 20th century. However, studies investigating the intraosseous pressure of the lunate with Kienböck's disease found higher intraosseous pressure of the lunate than in the surrounding bones,^{14,15} bringing into question the concept of whether Kienböck's disease is indeed caused by arterial insufficiency. They advocated that venous congestion caused lunate necrosis analogous to venous congestion in osteonecrosis of the femoral head.^{16,17} Furthermore, an anatomical study found a dense plexus of small venous vessels at the dorsal and palmar sides of the lunate which is woven into the capsular ligament.¹⁸ They speculated that disruption of venous drainage plays a role in the origin of aseptic necrosis. The site of capsulitis is believed to coincide with the site of the dorsal venous plexus. Therefore, at present, we speculate that an unknown primary etiology including repetitive stress^{19,20} to the wrist and overuse of the wrist induce venous insufficiency and cause Kienböck's disease. We cannot deny that dorsal wrist swelling has etiological significance, although we are unable to present more positive data about the etiology.

Note

This work was performed at the Nagoya Hand Center, Chunchi Hospital, Nagoya, Japan.

Ethical Approval

This study was approved by the ethical committee of Chunchi Hospital.

Conflict of Interest

None.

References

- 1 Nakamura R, Hori M, Imamura T, Horii E, Miura T. Method for measurement and evaluation of carpal bone angles. *J Hand Surg Am* 1989;14(2 Pt 2):412–416
- 2 Nakamura R, Horii E, Imaeda T, Tsunoda K, Nakao E. Distal radioulnar joint subluxation and dislocation diagnosed by standard roentgenography. *Skeletal Radiol* 1995;24(02):91–94
- 3 Lichtman DM, Degnan GG. Staging and its use in the determination of treatment modalities for Kienböck's disease. *Hand Clin* 1993;9(03):409–416
- 4 Makino N, Ishigaki T, Sakuma S, Takamatsu K, Tsunoda K, Nakamura R. [Three-phase bone scintigraphy of Kienböck disease]. *Kaku Igaku* 1992;29(12):1419–1427
- 5 Jacobsson H, Svartengren G. The development of Kienböck's disease as followed up by bone scintigraphy and radiography. *RoFo Fortschr Geb Röntgenstr Nuklearmed* 1988;149(05):541–543
- 6 Stuckey SL, Kalff V, Hoy G. Bone scan findings in Kienböck's disease. A case report with atypical findings and literature review. *Clin Nucl Med* 1997;22(07):481–483
- 7 Lesire MR, Allieu Y, Bonnel F, et al. Etiologie traumatique de la maladie de Kienböck. (Luxations péri-lunaires et nécrose du semi-lunaire). *Ann Chir Main* 1982;1(03):242–246
- 8 Mok CC, Wong RW, Lau CS. Kienböck's disease in rheumatoid arthritis. *Br J Rheumatol* 1998;37(07):796–797
- 9 Desy NM, Bernstein M, Harvey EJ, Hazel H. Kienböck's disease and juvenile idiopathic arthritis. *McGill J Med* 2011;13(02):8
- 10 Nakamura R, Niwa S, Takahashi S, Nakao E, Tatebe M. Lunate flattening in rheumatoid wrists. *Ann Orthop Rheumatol* 2014;2:1020
- 11 Gelberman RH, Panagis JS, Taleisnik J, Baumgaertner M. The arterial anatomy of the human carpus. Part I: the extraosseous vascularity. *J Hand Surg Am* 1983;8(04):367–375
- 12 Panagis JS, Gelberman RH, Taleisnik J, Baumgaertner M. The arterial anatomy of the human carpus. Part II: the intraosseous vascularity. *J Hand Surg Am* 1983;8(04):375–382
- 13 Freedman DM, Botte MJ, Gelberman RH. Vascularity of the carpus. *Clin Orthop Relat Res* 2001;(383):47–59
- 14 Jensen CH. Intraosseous pressure in Kienböck's disease. *J Hand Surg Am* 1993;18(02):355–359
- 15 Schiltenswolf M, Martini AK, Mau HC, Eversheim S, Brocai DRC, Jensen CH. Further investigations of the intraosseous pressure characteristics in necrotic lunates (Kienböck's disease). *J Hand Surg Am* 1996;21(05):754–758
- 16 Ficat RP. Idiopathic bone necrosis of the femoral head. Early diagnosis and treatment. *J Bone Joint Surg Br* 1985;67(01):3–9
- 17 Liu SL, Ho TC. The role of venous hypertension in the pathogenesis of Legg-Perthes disease. A clinical and experimental study. *J Bone Joint Surg Am* 1991;73(02):194–200
- 18 Pichler M, Putz R. The venous drainage of the lunate bone. *Surg Radiol Anat* 2003;24(06):372–376
- 19 Müller W. Über die Erweichung und Verdichtung des Os Lunatum, eine typische Erkrankung des Handgelenks. *Beitr Z Klin Chir* 1920;119:664
- 20 Bain GI, MacLean SB, Yeo CJ, Perilli E, Lichtman DM. The etiology and pathogenesis of Kienböck's disease. *J Wrist Surg* 2016;5(04):248–254