Journal of Hand Surgery Global Online 6 (2024) 567–570

Contents lists available at ScienceDirect

Journal of Hand Surgery Global Online

journal homepage: www.JHSGO.org

Original Research

Treatment Selection and Characteristics of Patients With Preiser Disease

Keisuke Ishizaka, MD, * Koji Moriya, MD, * Takuma Kuroda, MD, * Hisao Koda, MD, * Naoto Tsubokawa, MD, * Yutaka Maki, MD *

* Department of Orthopaedic Surgery, Niigata Hand Surgery Foundation, Kitakambara, Japan

ARTICLE INFO

Article history:
Received for publication November 29, 2023
Accepted in revised form April 21, 2024
Available online May 22, 2024

Key words:
Idiopathic avascular osteonecrosis
Preiser disease
Scaphoid

Purpose: Preiser disease is difficult to diagnose and treat because of its unclear pathophysiology. Although both nonsurgical treatment and surgical treatment for Preiser disease have been reported, there is no consensus on the optimal treatment because of its rarity. The purpose of this study was to investigate the relationship between treatment selection and characteristics of patients with Preiser disease.
Methods: This single-institution retrospective chart review included nine patients (two men and seven women) with Preiser disease who were treated at our hospital. We divided patients into two groups consisting of elderly (older than 65 years of age) and nonelderly patients. Herbert-Lanzetta classification, presence of dorsal intercalated segment instability (DISI), Watson classification based on plain radiography, Kalainov classification based on magnetic resonance imaging, and treatment modalities were investigated in both groups.
Results: In the elderly group, three of five cases were in advanced stages of Preiser disease according to the Herbert-Lanzetta classification. Three wrists had a DISI deformity. Three patients underwent conservative treatment. The two remaining cases classified as Herbert-Lanzetta stage II underwent closing radial wedge osteotomy. In the nonelderly group, three of four cases were in the early stages of Preiser disease according to the Herbert-Lanzetta classification. One wrist had a DISI deformity. Two patients were treated conservatively. The other two patients were surgically treated using closing radial wedge osteotomy in one case and vascularized bone graft from the second metacarpal base in another case, both classified as Herbert-Lanzetta stage II.
Conclusions: Most elderly patients with Preiser disease showed concurrent DISI at the time of initial presentation and advanced stage. Most elderly patients underwent nonsurgical treatment. Even when surgical treatment is implemented, our study suggests that the less invasive and optimal treatment is closing radial wedge osteotomy.
Type of study/level of evidence: Therapeutic IV.

Preiser disease is difficult to diagnose and treat because of its unclear pathophysiology.[1] Although both nonsurgical treatment and surgical treatment for Preiser disease have been reported, there is no consensus on its optimal treatment because of the rarity of the disease.[2]

When a trial of nonsurgical treatment for Preiser disease fails, there are three main surgical treatment strategies: revascularization surgery, salvage surgery, and joint-leveling surgery.[3] A vascularized bone graft (VBG) is usually performed for revascularization in early stages of the disease.[4] In contrast, salvage surgery including proximal row carpectomy and partial/total arthrodesis is used for advanced disease, and joint-leveling surgery such as closing radial wedge osteotomy (CRWO) is performed regardless of disease stage.[3,5–7] The optimal surgical procedure for Preiser disease still remains unknown.

This study investigated the relationship between treatment selection and characteristics of patients with Preiser disease. We hypothesized that treatments performed for Preiser disease are influenced by patient conditions (eg, age and disease stage).

Corresponding author: Keisuke Ishizaka, MD, Department of Orthopaedic Surgery, Niigata Hand Surgery Foundation, 997 Suwayama Seiro, Kitakambara, Niigata, 957-0117 Japan.
E-mail address: ishizaka.ms@gmail.com (K. Ishizaka).

https://doi.org/10.1016/j.jhsg.2024.04.014

Materials and Methods

This single-institution retrospective chart review extracted patient demographic characteristics, medical history, imaging findings, and follow-up data from medical records between July 2012 and July 2022. Patients who had radiographically confirmed vascular impairment of the scaphoid and underwent a treatment plan at our institution were included in this study. We excluded patients who had a vascular impairment of the scaphoid with acute fracture or fracture nonunion and visited our institution after being treated at other hospitals. Informed consent was obtained from all patients, and the study protocol was approved by the Niigata Hand Surgery Foundation Institutional Review Board according to the ethical guidelines of the 1975 Declaration of Helsinki. This clinical trial was registered in the University Hospital Medical Infomation Network Clinical Trials Registry (UMIN-CTR number: UMIN000052323).

Imaging modalities

All patients were examined with conventional plain radiography on anteroposterior and lateral views. Degenerative changes as well as the density and fragmentation of the proximal pole were observed on anteroposterior radiographs. The scapholunate angle (SLA) was measured on lateral radiographs. Magnetic resonance imaging (MRI) examinations were performed to document bone ischemia and necrosis. Osteonecrosis was defined as a region of bone marrow with a low-intensity signal on both T1-weighted imaging and T2-weighted imaging. Ischemic changes or bone edema were identified as areas of bone marrow with patchy, decreased, or absent signal on T1-weighted imaging when compared with the surrounding carpals and with patchy, isointense, or high signal on T2-weighted imaging.

Imaging evaluation

Herbert-Lanzetta classification, presence of dorsal intercalated segment instability (DISI) deformity, Watson classification, Kalainov classification, and treatment modalities were evaluated.[8–10] Herbert-Lanzetta classification was based on anteroposterior radiographs and categorized according to the following staging: stage I, normal x-ray and positive bone scan; stage II, increased density of proximal pole and generalized osteoporosis; stage III, fragmentation of proximal pole ± pathological fracture; and stage IV, carpal collapse pattern and/or osteoarthritis. Dorsal intercalated segment instability was evaluated according to the SLA (>70°). Watson classification was based on anteroposterior radiographs. Degenerative changes beginning at the radial styloid were defined as stage I, progression to the proximal scaphoid as stage II, and progression to the capitolunate joint as stage III. Kalainov classification was based on contrast MRI examinations and categorized into the following two types: type I, diffuse ischemia and necrosis of the scaphoid, and type II, vascular changes present in only a section of bone.

All imaging evaluations were performed by one of the authors (K.I).

Results

Patients

A total of nine wrists of nine consecutive patients with Preiser disease were included in this analysis with no excluded patients. The patients comprised two men and seven women with a mean age of 62 (range: 37–84) years at the time of the first visit. We divided these patients into two groups consisting of elderly patients (≥65 years of age) and nonelderly patients for statistical analysis. Five of nine Preiser disease patients were elderly patients ≥ 65 years of age (five women). The remaining four patients were nonelderly patients (two men and two women). Eight patients complained of wrist pain, and one, of numbness in the fingers at the time of initial presentation. All patients had no history of steroid use; however, one patient underwent surgery for de Quervain disease, one patient experienced repeated minor trauma because of snowboarding, and one patient underwent extensor indicis proprius tendon transfer for the rupture of extensor pollicis longus tendon after distal radius fracture surgery. Other characteristics of the patients are shown in Tables 1 and 2.

Table 1
Data of Patients at Initial Visit to our Hospital

Case	Age (y)	Sex	R/L	Patient History	Symptoms
1	37	M	L	Repeated minor trauma	Pain
2	44	F	R	de Quervain disease surgery	Pain
3	53	M	R	-	Pain
4	60	F	L	-	Pain
5	65	F	R	-	Pain
6	66	F	L	-	Pain
7	70	F	L	EIP tendon transfer for the rupture of EPL after DRF	Pain
8	79	F	R	-	Numbness
9	84	F	L	-	Pain

DRF, distal radius fracture; EIP, extensor indicis proprius; EPL, extensor pollicis longus.

Table 2
Additional Data of Patients at Initial Visit to our Hospital

Case	Age (y)	Stage			Radiographic Measurement	
		Herbert-Lanzetta	Watson	Kalainov	SLA	DISI (Yes/No)
1	37	2	0	2	66	No
2	44	2	0	2	55	No
3	53	4	1	-	79	Yes
4	60	1	0	-	56	No
5	65	2	0	2	58	No
6	66	2	0	2	63	No
7	70	3	0	2	71	Yes
8	79	3	0	-	98	Yes
9	84	4	1	-	75	Yes

DISI, dorsal intercalated segment instability; SLA, scapholunate angle.

Image evaluation

According to the Herbert-Lanzetta classification based on plain radiography, the elderly group consisted of stage II in two patients, stage III in two, and stage IV in one. The nonelderly group consisted of stage I in one patient, stage II in two, and stage IV in one. The overall median SLA was 69° (range: 55°–88°), which indicated that four wrists had DISI. In the elderly group, the median SLA was 73° (range: 58°–98°), which indicated that three wrists had DISI. In the nonelderly group, the median SLA was 64° (range: 55°–79°), which indicated that one wrist had DISI. One patient each from the elderly group and the nonelderly group showed stage I osteoarthritis of the radioscaphoid joint according to the Watson classification. Five patients underwent MRI examinations, which showed that five wrists were of type 2 according to the Kalainov classification, of which three were in the elderly group and two in the nonelderly group.

Table 3
Data of Patients at End point

Case	Age (y)	Follow-Up Periods, Age (y)	Treatment	Stage		Radiographic Measurement	
				Herbert-Lanzetta	Watson	SLA	DISI (Yes/No)
1	37	39	CRWO	2	0	66	No
2	44	45	VBG	3	0	65	No
3	53	-	Nonsurgical	NYA	NYA	NYA	NYA
4	60	67	Nonsurgical	4	1	82	Yes
5	65	68	CRWO	4	1	76	Yes
6	66	68	CRWO	4	1	70	Yes
7	70	74	CRWO	4	0	81	Yes
8	79	-	Nonsurgical	NYA	NYA	NYA	NYA
9	84	-	Nonsurgical	NYA	NYA	NYA	NYA

CRWO, closing radial wedge osteotomy; DISI, dorsal intercalated segment instability; NYA, data not yet available; SLA, scapholunate angle; VBG, vascularized bone graft.

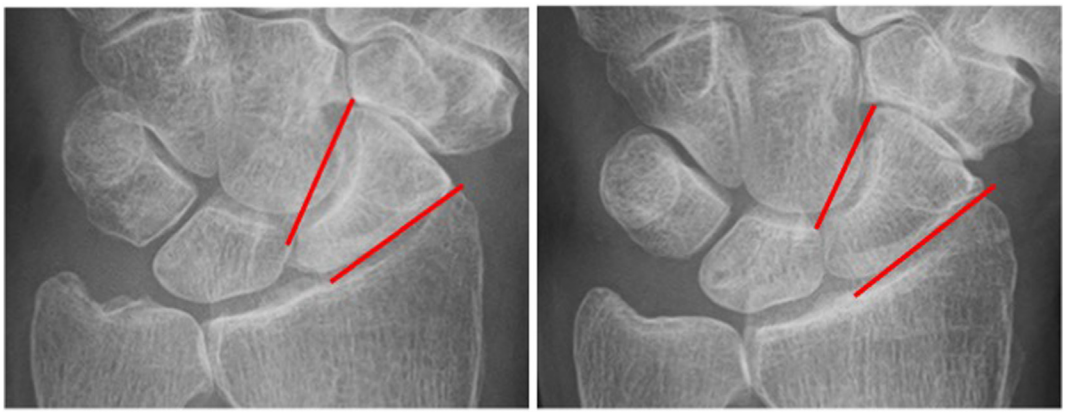

Figure. Comparison of the affected side compared with the healthy side. The proximal pole showed narrowing with moderate volume loss, the so-called "nipple sign."

Treatment

Five patients underwent conservative treatment and four underwent surgery. In the elderly group, three patients underwent conservative treatment and two underwent surgery. In nonsurgical treatment, a combination of pain medication (including nonsteroid anti-inflammatory drugs), immobilization with a splint or orthosis, and rest was employed for various periods. The remaining two relatively young elderly patients (65 and 66 years old) classified as Herbert-Lanzetta stage II underwent CRWO. In the nonelderly group, two patients underwent conservative treatment. The other two patients underwent CRWO in one case and a VBG from the second metacarpal base in another case, both classified as Herbert-Lanzetta stage II. Three of nine patients only exhibited mild symptoms and opted not to pursue follow-up (Table 3).

Discussion

This study showed that more than half of the nine cases of Preiser disease were elderly patients. Most elderly patients were accompanied by DISI deformity at the time of initial presentation and exhibited advanced stage Preiser disease according to the Herbert-Lanzetta classification. Three of the five cases underwent nonsurgical treatment. In terms of surgical treatment, three of four cases in the early stages of Preiser disease (Herbert-Lanzetta stage II) underwent CRWO, of which two cases were in the elderly group and one case in the nonelderly group. The one remaining case underwent a VBG. We investigated treatments according to patient conditions (eg, age and disease stage). Including patients who "became" elderly by age from 60 to 67 years during the study period, there were four elderly patients who received nonsurgical treatment and two who underwent surgery, and nonsurgical treatment tended to be implemented in elderly patients, whereas CRWO tended to be the surgical procedure of choice. However, we believe that causality cannot be determined because of the retrospective nature and small number of patients.

Nonsurgical treatment for patients with Preiser disease tends to be performed before surgical treatment.[11] Although favorable outcomes have been reported with nonsurgical treatment of Preiser disease, the results described in the literature have not accounted for age.[12] In contrast, reports on Kienböck disease have demonstrated that elderly patients respond well to conservative treatment with favorable clinical results.[13] Taniguchi et al[14] noted that some patients have no problems without treatment for many years in terms of carrying out activities of daily living or performing work. We believe that nonsurgical treatment tended to be chosen for elderly patients with Preiser disease in this study because it was expected to have the same effect as Kienböck disease. In this study, two patients in the nonelderly group who were treated conservatively did not wish to undergo surgery because their symptoms were mild.

Tomori et al[3] noted that CRWO addresses the following three idiopathic avascular necrosis-related issues: (1) CRWO may decompress venous hypertension, similar to osteotomy for idiopathic avascular necrosis of the femoral head. Normalization of the intraosseous pressure may reduce pain and consequently improve range of motion. (2) Closing radial wedge osteotomy changes the contact area of the carpals with the radius. Kojima et al[15] and Tsumura et al[16] demonstrated that CRWO reduces the axial load to the lunate and scaphoid, and the procedure decompresses joint pressure between the radius and the lunate in an experimental model. (3) Closing radial wedge osteotomy could induce revascularization of the carpals following radial osteotomy. The revascularization effects of CRWO in Kienböck disease have been previously reported. CRWO has been used regardless of the stage of

Preiser disease. Even in the advanced stage of Preiser disease, pain relief and cessation of ischemia progression were the main positive effects.[7] In contrast, a VBG should be strictly reserved for early stages of Preiser disease (Herbert-Lanzetta stage I or II) without intracarpal instability or signs of incipient osteoarthritis. Vascularized bone graft cannot prevent the collapse of the advanced stage of Preiser disease (Herbert-Lanzetta stage III or IV) because of the technical difficulty involved in removing all necrotic or sclerotic bone—particularly from the proximal pole—without perforating the cartilaginous shell.[4] All four of our cases that underwent surgery were classified as Herbert-Lanzetta stage II. In these cases, the proximal pole showed narrowing with moderate volume loss, referred to as the so-called "nipple sign" (Fig.).[17] Since Herbert and Lanzetta[8] noted that idiopathic avascular necrosis affecting the whole scaphoid always starts in the proximal pole of the bone, we believe that this finding may be the beginning of the collapse of the scaphoid. If the collapse of the scaphoid has already begun with Herbert-Lanzetta stage II, a VBG does not prevent collapse in stage II and may only be able to prevent collapse for early stages of Preiser disease classified as Herbert-Lanzetta stage I. Therefore, in early stages of the disease, we tended to choose the less invasive CRWO, and the choice of CRWO or a VBG was determined by the surgeon's preference.

A major limitation of this study was its retrospective design with a small number of patients. Statistical analyses for image evaluation and treatment could not be performed because of the small sample size. Considering the rarity of the condition, it might be difficult to collect a larger sample in a single center. Future studies are warranted to obtain a larger number of cases through a multicenter collaboration. Another limitation was the lack of standardization in the imaging evaluation. Not all cases underwent MRI examinations, and a single surgeon performed all imaging evaluations.

In conclusion, more than half of the patients with Preiser disease in this study were elderly, and nonsurgical treatment was implemented in most elderly patients. In surgical treatment, the less invasive CRWO tended to be the surgical procedure of choice.

Conflicts of Interest

No benefits in any form have been received or will be received related directly to this article.

References

1. Bergman S, Petit A, Rabarin F, Raimbeau G, Bigorre N. Preiser's disease or avascular osteonecrosis of the scaphoid: an updated literature review. *Hand Surg Rehabil*. 2021;40(4):359–368.
2. Lin JD, Strauch RJ. Preiser disease. *J Hand Surg Am*. 2013;38(9):1833–1834.
3. Tomori Y, Sawaizumi T, Nanno M, Takai S. Closing radial wedge osteotomy for Preiser Disease: clinical outcomes. *J Hand Surg Am*. 2019;44(10):896.e1–896.e10.
4. Moran SL, Cooney WP, Shin AY. The use of vascularized grafts from the distal radius for the treatment of Preiser's disease. *J Hand Surg Am*. 2006;31(5):705–710.
5. Bayley J, Simmons B. Avascular necrosis of the proximal carpal row. A case report. *Ann Chir Main*. 1987;6(3):210–215.
6. Lenoir H, Coulet B, Lazerges C, Mares O, Croutzet P, Chammas M. Idiopathic avascular necrosis of the scaphoid: 10 new cases and a review of the literature. Indications for Preiser's disease. *Orthop Traumatol Surg Res*. 2012;98(4):390–397.
7. Hayashi O, Sawaizumi T, Ito H. Closed radial wedge osteotomy for Preiser's disease: a report of four cases. *Hand Surg*. 2011;16(3):347–352.
8. Herbert TJ, Lanzetta M. Idiopathic avascular necrosis of the scaphoid. *J Hand Surg Br*. 1994;19(2):174–182.
9. Watson HK, Ballet FL. The SLAC wrist: scapholunate advanced collapse pattern of degenerative arthritis. *J Hand Surg Am*. 1984;9(3):358–365.
10. Kalainov DM, Cohen MS, Hendrix RW, Sweet S, Culp RW, Osterman AL. Preiser's disease: identification of two patterns. *J Hand Surg Am*. 2003;28(5):767–778.
11. Tomori Y, Nanno M, Takai S. Clinical outcomes of nonsurgical treatment for Preiser disease. *Med (Baltim)*. 2020;99(4):e18883.
12. Vidal MA, Linscheid RL, Amadio PC, Dobyns JH. Preiser's disease. *Ann Chir Main Memb Super*. 1991;10(3):227–235.
13. Geutjens GG. Kienböck's disease in an elderly patient. *J Hand Surg Am*. 1995;20(1):42–43.
14. Taniguchi Y, Nakao S, Tamaki T. Incidentally diagnosed Kienböck's disease. *Clin Orthop Relat Res*. 2002;395:121–127.
15. Kojima T, Kido M, Tsumura H, Himeno S, Shinohara N. Wedge osteotomy of radius for Kienböck's disease. *J Jpn Soc Surg Hand*. 1984;1:431–434.
16. Tsumura H, Kojima T, Himeno S, Kido M. The optimal correcting angle of the wedge osteotomy of radius for Kienböck disease. *J Jpn Soc Surg Hand*. 1984;1:435–439.
17. Schmitt R, Fröhner S, van Schoonhoven J, Lanz U, Gölles A. Idiopathic osteonecrosis of the scaphoid (Preiser's disease)—MRI gives new insights into etiology and pathology. *Eur J Radiol*. 2011;77(2):228–234.