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Clinical outcomes of nonsurgical treatment for Preiser disease

Yuji Tomori, MD, PhD*, Mitsuhiko Nanno, MD, PhD, Shinro Takai, MD, PhD

Abstract

To elucidate whether nonsurgical treatment for Preiser disease is effective.

Eight patients with Preiser disease (median age 59 [47–69] years) underwent nonsurgical treatment (median symptom-onset-to-treatment interval 8 [9–180] months). At presentation, 7 patients complained of constant pain and 1 of motion-related pain. Pain restricted wrist range of motion (median modified Mayo wrist score [MMWS] 17.5 [range 10–30]). Radiography revealed stages 1 to 3 disease (Herbert–Lanzetta classification). Median scapholunate angle was 62° (54°–75°), with 3 wrists suffering dorsal intercalated segment instability (DISI). Magnetic resonance imaging showed (Kalainov criteria) 4 stage 1 wrists (complete necrosis) and 4 stage 2 (incomplete necrosis). Two had concomitant Kienböck disease. All patients underwent nonsurgical treatment (ie, oral pain killer, immobilization, rest) and were monitored via radiographic and clinical evaluations. Scapholunate angles and the scaphoid area reduction ratio were calculated using radiography. Response criteria were the patients' subjective and objective status. Endpoint was the time from start of non-surgical to surgical treatment.

Immobilization lasting 0 to 24 months (median 1.8 months) did not relieve their symptoms. Follow-up radiography showed that the disease stage had progressed in 5 of 8 wrists, with 5 wrists having DISI. The median area reduction ratio of the scaphoid was 11% (4%–52%) on anteroposterior views and 4% (−23% to 17%) on lateral views. Compared with the contralateral wrist, the median wrist flexion-extension arc was 61% (50%–79%) and the median grip strength 39%. Median MMWS score was 17.5 (10–25) – poor in 6 of 8 patients. Surgery was thus necessary in all patients.

Nonsurgical treatment for Preiser disease did not improve subjective or objective outcomes and did not prevent deterioration of radiographic findings.

Type of study/level of evidence: Therapeutic, Level V.

Abbreviations: ADL = activities of daily life, AVN = idiopathic avascular necrosis, DISI = dorsal intercalated segment instability, MEPS = Mayo elbow performance score, MRI = magnetic resonance imaging, ROM = range of motion, SLA = scapholunate angle, VBG = vascularized bone graft.

Keywords: idiopathic avascular osteonecrosis, nonsurgical treatment, Preiser disease, scaphoid

1. Introduction

Preiser disease – idiopathic avascular necrosis (AVN) of the scaphoid – is an uncommon disorder that was first reported by Preiser in 1910.[1] AVN of the scaphoid has been reported in patients with no significant history of trauma but who complained of increasing, severe radial wrist pain with spontaneous onset. Physical examination showed tenderness around the dorsoradial aspect of the wrist and swelling around the wrist joint. To date, although the etiology and pathology of this disease have not been established, idiopathic vascular insufficiency of the carpals and repetitive mechanical stress on the wrist are regarded as its cause.[1–4] Most cases have involved systemic steroid use[5,6] for a systemic illness, such as autoimmune hemolytic anemia,[7] systemic lupus erythematosus,[8] or renal transplantation.[9] Another etiology could be topical steroid use for a local illness such as de Quervain disease.[10]. In addition, not a few cases have involved risk factors such as smoking, alcoholism, and/or infection.[11–14]

Although both nonsurgical and surgical treatment for Preiser disease have been reported, there is no consensus on the optimal treatment because of the rarity of the disease.[15] Some researchers reported favorable clinical outcomes of nonsurgical treatment for Preiser disease in a few cases.[2] Others; however, reported that nonsurgical treatment (eg, immobilization, oral analgesics, corticosteroid injections, electrical stimulation) have not provided satisfactory outcomes.[2,4,16,17] Thus, whether nonsurgical treatment for Preiser disease is effective is a highly controversial issue. The purpose of this study was to investigate the clinical outcomes of 8 patients with Preiser disease treated nonsurgically to elucidate whether such an effort is effective.

Editor: Qing Yao.

All procedures performed in studies involving human participants were in accordance with the 1975 Helsinki declaration and its later amendments or comparable ethical standards after obtaining approval from our institutional review board (No. 30-12-1048, 450-30-21).

Informed consent for the treatment and publication was obtained from all patients in this study.

The authors have no conflicts of interest to disclose.

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How to cite this article: Tomori Y, Nanno M, Takai S. Clinical outcomes of nonsurgical treatment for Preiser disease. Medicine 2020;99:4(e18883).

Received: 14 June 2019 / Received in final form: 29 November 2019 / Accepted: 19 December 2019

http://dx.doi.org/10.1097/MD.0000000000018883

Table 1

Patients' demographic data at the initial visit to our hospital.

Case	Age, yr*	Sex	R/L†	Disease	Steroid use	Alcohol habit	Pack-year	History	Job
1	69	F	L	Preiser	–	–	–	Emphysema	Housewife
2	59	F	L	Preiser	–	Whisky 100 mL/d	0.35 pack × 40 yr	Graves' disease	Clerical work
3	49	F	(R)	Preiserand Kienböck	–	Sometimes	pack × 30 yr	Breast cancer	Post-office clerk
4	58	F	(R)	Preiser	Repeated steroid injections in left shoulder bursa	Beer 500 mL/d	0.5 pack × 39 yr	Angina pectoris, myocardial infarction	Housewife
5	63	F	(R)	Preiser	–	–	–	–	Clerical work
6	59	F	(R)	Preiserand Kienböck	–	Distilled spirits 30 mL/wk	1 pack × 30 yr	Gastric ulcer	Nursing care staff
7	47	M	(R)	Preiser	–	Distilled spirits 180 mL/d	1 pack × 28 yr	Chronic pancreatitis	Cook, restaurant business
8	54	F	(R)	Preiser	Repeated steroid injections around right wrist joint	–	–	Hepatitis C	Clerical work

BI = Brinkman index.

* Age at the beginning of treatment.

† Parentheses indicate that the affected hand was the dominant hand.

2. Materials and methods

This retrospective single-centered consecutive case series was performed at our institution. This study was conducted in conformity with the ethical guidelines of the 1975 Declaration of Helsinki after approval from our institutional review board, the ethic committee of Nippon Medical School Hospital (No. 30-07-970). For this retrospective investigation, patients' demographic characteristics, medical history, imaging findings, and follow-up data were extracted from their clinical records and medical linkage systems.

2.1. Patients

From April 1998 to March 2015, 8 patients (1 man, 7 women) with Preiser disease were treated in our hospital. The demographic data for the patients at the first visit in our hospital are shown in Table 1. All patients, with no significant history of trauma, had complained of increasing, intolerable dorsoradial wrist pain of spontaneous onset. There was no history of any steroid use in 6 of the patients. One patient had been given a total of 8 steroid injections (2 mg; betamethasone sodium phosphate; Rinderon) in the contralateral subacromial bursa to treat periarthritis of the left shoulder during the 5 to 8 years before the definitive diagnosis of AVN of the scaphoid in the right wrist. Another patient had 5 injections of betamethasone phosphate (2 mg; Rinderon), and 4 injections of triamcinolone acetonide (10 mg) around a painful wrist joint during the 6 years before the current definitive diagnosis.[10] The physical examination showed tenderness around the dorsoradial aspect of the wrist and swelling around the wrist joint.

All patients were examined with plain radiography, and 3 patients underwent computed tomography as well. AVN of the scaphoid was diagnosed by radiographic evidence (ie, sclerosis, fragmentation, erosion, collapse of the scaphoid in the absence of a significant history of trauma). Magnetic resonance imaging (MRI) was performed in all patients, and evidence of AVN on MRI was confirmed by radiologists and an orthopedic surgeon. Osteonecrosis was defined as a region of bone marrow with low-intensity signal on both T1-weighted imaging (T1WI) and T2WI. Ischemic changes or bone edema were identified as areas of bone marrow with patchy, decreased, or absent signal on T1WI when compared with the surrounding carpal bones but with patchy, isointense, or high signal on T2WI. Contrast-enhanced MRI was performed to gain a definitive diagnosis in a patient, and showed the decrease in bone marrow perfusion.

Eight patients with Preiser disease who underwent non-surgical treatment were a median age of 59 years (range 47–69 years) at the time of their first visit. The period from the onset of symptoms to the start of nonsurgical treatment was a median 8 months (range 9–180 months). At the time of the first visit, 7 patients complained of constant pain, and 1 complained of motion-related pain. The radiographic and MRI results, the wrists' range of motion (ROM), and grip strength at the first visit to our hospital are shown in Table 2.

Table 2

Data for the patients at the initial visit to our hospital.

	Stage at first medical examination				Radiographic measurement		ROM and grasp strength on wrist (affected/unaffected)		
Case	Herbert class	Watson's class	Kalainov's criteria	Lichtman class*	DISI	SLA, °	Extension, °	Flexion, °	GS, kg
1	3	1 RCJ	2	–	No	39	45/85	50/60	5/12
2	2	–	1	–	No	60	20/48	35/50	0/10
3	2	–	1	2	No	59	45/85	70/70	19/22
4	2	–	1	–	No	63	60/57	50/58	10/24
5	3	1 DRUJ, RCJ	1	–	Yes	73	40/70	40/70	2/17
6	2	–	2	2	Yes	75	37/64	34/62	8/25
7	3	1	2	–	Yes	54	39/75	38/65	25/37
8	1	–	2	–	No	64	10/42	30/60	17/21

Class = classification, DISI = dorsal intercalated segment instability, DRUJ = distal radioulnar joint disorder, GS = grasp strength, RCJ = radiocarpal joint, ROM = range of motion, SLA = scapholunate angle.

* Concomitant Kienböck disease.

Table 3

Further data of the patients at the initial visit to our hospital.

Case	Duration of pain, mo	Pain	Pain score	Function	Function score	ROM (%)* (ext-flex arc)	ROM score	GS† (%)	GS score	Initial-MMWS
1	24	At rest	0	Unable to work	0	66	10	42	5	15
2	2	At rest	0	Unable to work	0	56	10	0	0	10
3	11	At rest	0	Unable to work	0	74	10	86	15	25
4	7	At rest, at night pain	0	Unable to work	0	96	15	36	5	20
5	5	At rest	0	Unable to work	0	57	10	10	0	10
6	9	Moderate with ADLs	5	Working but restricted due to pain	10	56	10	28	5	30
7	180	At rest	0	Unable to work	0	55	10	59	10	20
8	10	At rest	0	Unable to work	0	39	5	70	10	15

ADL = activities of daily living, ext-flex = extension–flexion, GS = grasp strength, MMWS = modified Mayo wrist score, ROM = range of motion.

* Ratio of wrist flexion-extension arc of the affected wrist compared that of the unaffected wrist.

† Ratio of grasp strength of the affected wrist compared that of the unaffected wrist with a correction for limb dominance.

The data for pain duration and the modified Mayo wrist score (MMWS)[18] are shown in Table 3. Staging classification of the disease and clinical outcomes (MMWS) are shown in Table 4 and Table 5, respectively. One wrist had stage 1 disease, 4 had stage 2, 3 had stage 3 (Herbert–Lanzetta classification). The median scapholunate angle (SLA) was 62° (range 54°–75°), which indicated that 3 wrists had dorsal intercalated segment instability (DISI). Plain radiography and/or computed tomography showed that 3 patients had stage 1 osteoarthritis of the radioscaphoid joint according to the Watson classification.[19] MRI showed that 4 wrists had stage 1 (Kalainov criteria) (complete necrosis), and 4 had stage 2 (incomplete necrosis). Two wrists had concomitant Kienböck disease. The ROMs of the wrists were restricted by pain, and the median MMWS was 17.5 points (range 10–30 points).

The response criteria were defined as the subjective and objective status at the clinical and radiological evaluations, respectively, and the endpoint was defined as the time from starting nonsurgical treatment to the surgical intervention to mitigate the intolerable wrist pain. All patients underwent a follow-up examination that included radiographic evaluation, interviews regarding wrist pain, and evaluation of ROM, grip strength, and MMWS.

Informed consent was obtained in writing from all patients for the nonsurgical treatment and publication of the cases at the first visit our hospital.

Table 4

Staging classification of avascular necrosis of the scaphoid and lunate and classification of osteoarthritis on the wrist joint according to radiography and MRI.

Herbert and Lanzetta grading scale[8] for Preiser disease	
Stage 1	Normal radiograph
	MRI signal change
	abnormal sign on bone scan
Stage 2	Increased density of the proximal pole
Stage 3	Fragmentation of the proximal pole without pathological fracture
Stage 4	Carpal collapse with osteoarthritis
Kalainov criteria[9] for Preiser disease (magnetic resonance imaging)	
Type 1	Complete necrosis
Type 2	Incomplete necrosis
Lichtman grading scale[10] for Kienböck disease	
Stage 1	Normal radiograph
	MRI signal change
	abnormal sign on bone scan
Stage 2	Lunate sclerosis without collapse
Stage 3A	Lunate fragmentation and collapse without scaphoid rotation
3B	Lunate fragmentation and collapse with fixed scaphoid rotation
Stage 4	Perilunate arthritic change
Watson staging for the wrist joint	
Stage 1	osteoarthritis of the articulation between the radial styloid and the scaphoid
Stage 2	osteoarthritis involving the whole radioscaphoid articulation
Stage 3	ostoarthritis of the radioscaphoid and capitolunate articulations
Stage 4	ostoarthritis of the radiocarpal and intercarpal articulations +/-
	distal radioulnar joint (DRUJ)

Table 5

Modified Mayo wrist score.

Category and score	Findings
Pain (25 points)	
25	No pain
20	Mild pain with vigorous activities
	Pain only with weather changes
15	Moderate pain with vigorous activities
10	Mild pain with activities of daily living
0	Pain at rest
Satisfaction (25 points)	
25	Very satisfied
20	Moderately satisfied
10	Not satisfied but working
0	Not satisfied, unable to work
Range of motion (25 points) (% of normal)	
25	100%
15	75%–99%
10	50%–74%
5	25%–49%
0	0%–24%
Grip strength (25 points) (% of normal)	
25	100%
15	75%–99%
10	50%–74%
5	25%–49%
0	0%–24%

The final results are graded as follows: excellent 100, good 80–89, fair 65–79, poor < 65.

Table 6

Data of the patients at the endpoint.

Case	Nonsurgical treatment	Follow-up periods, mo	Period of immobilization, mo	Stage		Radiographic measurement		Area reduction ratio of the scaphoid (%)		ROM and GS on wrist (affected/unaffected)		
				Herbert class.	Watson's class.	DISI	SLA, °	AP view (%)	Lateral view (%)	Ext, °	Flex, °	GS, kg
1	Cast, orthosis, p/o NSAIDs	12	24 (on & off)	4	1	No	52	14	−23	30/80	40/60	3/12
2	Splinting, p/o NSAIDs	2	1.5	2	0	Yes	74	18	4	20/50	35/50	0/10
3	Rest, p/o NSAIDs	5	None	2	0	No	53	12	2	35/85	60/70	17/20
4	Splinting, p/o NSAIDs	11	2 (on & off)	3	0	No	66	38	17	28/57	42/58	10/24
5	Casting, p/o NSAIDs	3	1.5	4	1	Yes	84	52	4	*	*	*
6	Orthosis, splinting, p/o NSAIDs	3	9 (on & off)	3	0	Yes	74	9	8	*	*	*
7	Rest, p/o NSAIDs	3	None	3	0	Yes	71	6	3	60/75	50/65	25/37
8	Orthosis, splinting, p/o NSAIDs	14	2	3	1	Yes	55	4	7	22/42	41/60	15/21

AP = anteroposterior, class = classification, DISI = dorsal intercalated segment instability, Ext = extension, Flex = flexion, GS = grasp strength, NSAIDs = nonsteroidal anti-inflammatory drugs, SLA = scapholunate angle.

*No data.

2.2. Nonsurgical treatment

Non-surgical treatment – including oral administration of nonsteroidal anti-inflammatory drugs, immobilization with a splint or orthosis, and rest – was undertaken for various periods.

2.3. Radiographic evaluation

The SLA was calculated on lateral radiographs. The DISI deformity was evaluated according to the SLA (>70°). To measure the whole area of the scaphoid, the area of the scaphoid was traced and calculated using image analysis software Leafareacounter Plus (https://www.vector.co.jp/download/file/win95/business/fh625135.html) on anteroposterior and lateral radiographs. The area reduction ratio of the scaphoid was presented as a percentile – that is, dividing the area of the scaphoid at the endpoint by that on the initial radiographs, subtract from 1, and multiply by 100.

2.4. Clinical evaluation

The clinical evaluation included ROM, the wrist flexion-extension arc, grip strength measured with a dynamometer (Jamar; Baran/Tec, Clifton, NJ), and calculation of the MMWS. We then calculated the ratio of the wrist flexion–extension arc of the affected wrist compared that of the unaffected wrist. Also, the ratio of the grasp strength of the affected wrist compared that of the unaffected wrist with a correction for limb dominance was also evaluated. This correction was always conducted when the dominant hand was the affected hand. The grip strength of the dominant hand was assumed to be 15% stronger than the nondominant hand. A correction was not conducted for the nondominant hand.

3. Results

The results of the nonsurgical treatment are shown in Table 6 and Table 7. The median duration of immobilization was 1.8 months (range 0–24 months). There were no complications (eg, neuropathy, contracture of fingers). Our patients were initially treated nonsurgically (ie, wrist immobilization with a splint or orthosis) for various periods. To mitigate the pain, all patients took painkillers, including loxoprofen (180 mg), lornoxicam (24 mg), or mofezolac (225 mg) per a day. The drugs were administered in the full therapeutic dose. The wrist pain; however, was not alleviated in any of the patients, indicating that the nonsurgical treatment was ineffective. Surgery was; therefore, ultimately required for all patients.

3.1. Radiographic evaluations

The relationship between Herbert–Lanzetta classification and the follow-up periods are shown in Figure 1. Follow-up radiographs and the Herbert–Lanzetta classification[4] at the endpoint showed that 2 wrists had stage 2 disease, 4 had stage 3, and 2 had stage 4. Thus, 2 of the wrists had progressed to stage 4, and 3 had progressed to stage 3 – that is, the disease stage had progressed in 5 of the 8 wrists while under nonsurgical treatment. At the endpoint, the median SLA was 68.5° (range 52°–84°), which indicated that 5 wrists had DISI. Radiographs from a representative case (case 4) at the initial visit and at the endpoint are shown in Figures 2 and 3. The median area reduction ratio of the scaphoid was 11% (range 4%–52%) on anteroposterior

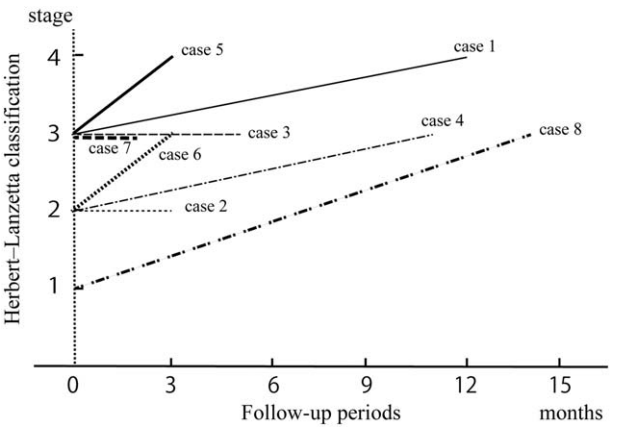

Figure 1. The relationship between Herbert–Lanzetta classification and the follow-up periods, which depicted Herbert–Lanzetta classification on the *Y*-axis and duration of follow-up on the *X*-axis. Thin line (solid line: case 1, dotted line: case 2, dashed line: case 3, dash-dot line: case 4), thick line (solid line: case 5, dotted line: case 6, dashed line: case 7, dash-dot line: case 8).

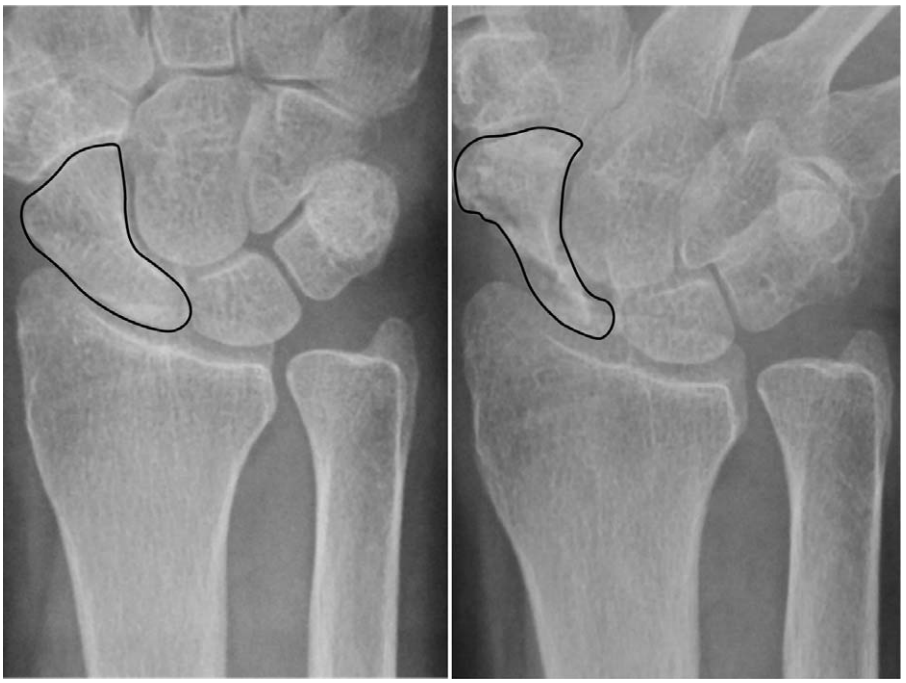

Figure 2. Measurement of the area reduction ratio of the scaphoid on anteroposterior radiographs. The area of the scaphoid was traced and calculated at the time of their first visit (A) and at the endpoint (B). (A) Anteroposterior radiograph of the right wrist from a representative case (case 4) at the initial visit showing the increased density of the proximal pole scaphoid bone. The area of the scaphoid was traced and calculated using image analysis software (the area: 272 mm^2). (B) Radiograph at the endpoint showing marked collapse of the proximal pole of the scaphoid bone without pathological fracture. The area of the scaphoid was traced and calculated using image analysis software (the area: 170 mm^2). The area reduction ratio was 38%.

views and 4% (range −23% to 17%) on lateral-view radiographs, respectively.

3.2. Clinical results

At the endpoint, compared with the findings in the contralateral wrist, the median wrist flexion–extension arc was 61% (range 50%–79%), and the median grip strength was 39%. The median MMWS was 17.5 points (range 10–25) – that is, poor in 6 patients (the flexion–extension arc and grip strength were not investigated in the other 2 patients).

4. Discussion

AVN of the scaphoid (Preiser disease) has been reported in only a few case series in the English-language literature.[2,4,6,12,14,16,20] Also, AVN of the scaphoid and lunate (Preiser disease with concomitant Kienböck disease) is rare, having been reported in only 6 patients, including our 2.[5,11,21–23] Although the etiology of AVN of the wrist is incompletely understood, most cases have involved steroid use[5–7] and involved risk factors such as smoking, alcoholism, and/or infection.[11,14] Moreover, undeveloped vascular networks and mechanical stress (eg, repetitive strain, overloading), which induce joint swelling and interruption of the intraosseous and extraosseous blood supply, could be

Table 7

Further data of the patients at the endpoint.

Case	Pain	Pain score	Function	Function score	ROM (%)* (ext-flex arc)	ROM score	GS† (%)	GS score	Endpoint MMWS
1	At rest	0	Unable to work	0	50	10	42	5	15
2	At rest	0	Unable to work	0	55	10	0	0	10
3	At rest	0	Unable to work	0	61	10	74	10	20
4	At rest and at night	0	Unable to work	0	61	10	36	5	15
5	At rest	0	Unable to work	0	*	*	*	*	*
6	Moderate with activities of daily living	5	Working but restricted due to pain	10	*	*	*	*	*
7	At rest	0	Unable to work	0	79	15	59	10	25
8	At rest	0	Unable to work	0	62	10	62	10	20

Ext-flex = extension-flexion, GS = grasp strength, MMWS = modified Mayo wrist score, ROM = range of motion.

* The ratio of wrist flexion-extension arc of the affected wrist compared that of the unaffected wrist.

† The ratio of grasp strength (GS) of the affected wrist compared that of the unaffected wrist with a correction for limb dominance.

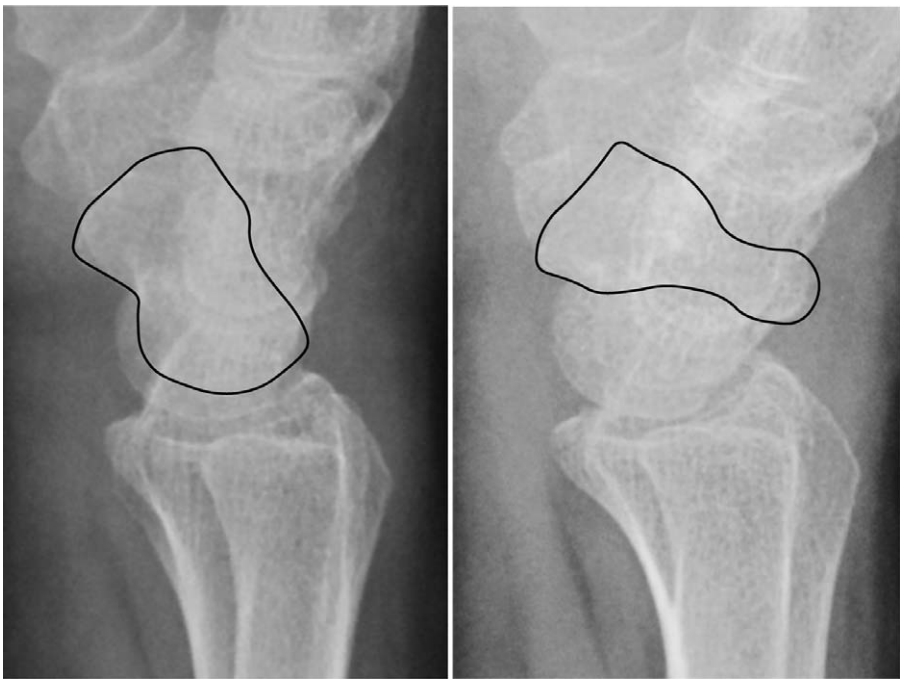

Figure 3. Measurement of the area reduction ratio of the scaphoid on lateral-view radiographs. The area of the scaphoid was traced and calculated at the time of their first visit (A) and at the endpoint (B). (A) Lateral radiographs of the right wrist from a representative case (case 4) at the initial visit. The area of the scaphoid was traced and calculated using image analysis software (the area: 200 mm^2). (B) Radiograph at the endpoint showing marked collapse of the proximal pole of the scaphoid bone. The area of the scaphoid was traced and calculated using image analysis software (the area: 166 mm^2). The area reduction ratio was 17%.

predisposing factors.[1–4,12,24] Although 2 of our patients had a history of repeated steroid injection of the contralateral shoulder or around the affected wrist, respectively, the other 6 did not have steroid use. However, 5 of the 8 had a smoking habit and a history of daily alcohol intake. Moreover, nearly all of our patients had a job that entailed repetitive mechanical stress on the wrist (Table 1). These factors may have contributed to the onset of Preiser disease in our patients.

Although radiographic staging guides treatment, early radiographic changes of AVN of the wrist are mostly undetectable, resulting in initial nonsurgical treatment. Previously, some researchers reported favorable outcomes of nonsurgical treatment for Preiser disease in a few cases.[2] Others, however, reported that nonsurgical treatment – including immobilization, oral analgesics, corticosteroid injections, and electrical stimulation, among others – have not provided satisfactory outcomes.[2,4,16,17] Moreover, the natural history of Preiser disease would presage unfavorable results, suggesting residual wrist pain and radiographic evidence of progressive deterioration. Although our patients were initially treated nonsurgically (ie, wrist immobilization with a splint or orthosis, rest) for various periods, it was ineffective.

Radiographically, the SLA was increased in most patients, and DISI was observed in 5 of our 8 patients. The area reduction ratio of the scaphoid was increased on anteroposterior radiographs, suggesting that almost all patients with AVN of the wrist exhibit radiographic progression and collapse of the scaphoid. Although the area reduction ratio of the scaphoid on lateral radiographs showed an increase in some patients, it could indicate that the shape of the scaphoid was collapsed and flattened. The scaphoid changes may initially occur as a cavitation due to AVN and result in flattening, collapse, and fragmentation caused by compression from the axial load from the radial portion of the articular surface of the distal radius. Moreover, all patients with AVN of the wrist exhibited persistent wrist pain. In addition, there was no obvious improvement of the wrist flexion–extension arc or grip strength compared with that of the contralateral wrist.

Based on our results, we concluded that nonsurgical treatment did not prevent deterioration of the radiographic findings and did not alleviate the wrist pain, ROM, grip strength, or MMWS. Because nonsurgical treatment had no positive effect on radiological or clinical improvement, and the scaphoid was at immediate risk of collapsing, prompt surgical intervention was indicated after arriving at the definitive diagnosis of AVN of the scaphoid. Nevertheless, there is still no consensus on the optimal surgical procedure for Preiser disease (with or without concomitant Kienböck disease) because of the disease's rarity.

Among various surgical procedures, 3 main protocols have been proposed for Preiser disease: revascularization surgery; salvage surgery; joint-leveling surgery. According to the Herbert and Lanzetta staging system, or depending on the arthritic location in the wrist described by Watson and Ballet,[19] different procedures are indicated for different stages.[12–15,25,26]

Among these procedures, however, only revascularization surgery using a vascularized bone graft (VBG), mainly performed for early (stage 1–2) disease (Herbert–Lanzetta classification), could be a possible solution to simultaneously prevent scaphoid collapse and provide revascularization and augmentation of the bone strength of the scaphoid. Moran et al[13] performed VBG from the distal radius in 8 patients with Preiser disease and reported good clinical outcomes for 2 patients with stage 1 to 2 disease. Also, as for the patient with Kienböck disease, several researchers reported that VBG provides an avascular lunate along with revascularization, increasing the bone strength of the

lunate and possibly preventing its collapse.[27–29] Because VBG allows preservation and survival of osteocytes,[30] it provides accelerated graft consolidation in necrotic areas.[31,32] Moreover, several studies have shown that VBGs provide more favorable mechanical and biological features than nonviable bone grafts.[33–35] Thus, VBG might a good option for the avascular scaphoid to prevent carpal collapse in patients with early-stage disease.

To date, several authors reported the effectiveness of metaphyseal core decompression or radial osteotomy with or without radius shortening for Kienböck disease.[36,37] Our previous study; however, showed that radial osteotomy for Preiser disease did not prevent scaphoid collapse.[14] Thus, radial osteotomy might not be a solution for preventing collapse of the scaphoid. Further investigation regarding the effectiveness and outcomes of radial osteotomy for AVN of the carpal bone is required.

The major limitations of this study are the small sample size and its retrospective nature. Moreover, the study provides no comparison between treatment groups. Although our case series revealed that nonsurgical treatment for Preiser disease did not have a positive effect on the clinical and radiologic outcomes, further investigation regarding the effectiveness and outcomes of nonsurgical treatment for AVN of the carpal bone is needed.

5. Conclusion

Nonsurgical treatment for Preiser disease did not improve subjective or objective outcomes and did not prevent deterioration of radiographic findings.

Acknowledgment

We thank Nancy Schatken, BS, MT(ASCP), from Edanz Group (www.edanzediting.com/ac), for editing a draft of this manuscript.

Author contributions

Conceptualization: Yuji Tomori.
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Writing – original draft: Yuji Tomori.
Writing – review & editing: Yuji Tomori, Mitsuhiko Nanno.

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