

Case Report

Idiopathic Preiser disease managed with cyst excision, distal radius bone grafting, and headless compression screw fixation: a case report

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ABSTRACT

Preiser disease is an uncommon disorder characterized by idiopathic osteonecrosis of the scaphoid without preceding fracture. Owing to its rarity and nonspecific symptoms, diagnosis is frequently delayed, and the optimal management remains challenging. A 47-year-old right-hand-dominant male presented with a three-year history of progressively worsening left wrist pain, reduced grip strength, and functional limitation without previous trauma. Clinical examination demonstrated localized scaphoid tenderness with restricted wrist motion. Plain radiographs showed cystic changes without fracture, while magnetic resonance imaging (MRI) demonstrated diffuse marrow signal alteration consistent with idiopathic scaphoid osteonecrosis without carpal collapse or degenerative arthritis. The patient underwent excision of the scaphoid cyst, autologous cancellous bone grafting from the distal radius, and fixation with a 2.4-mm headless compression screw. The postoperative period was uneventful. At one-month follow-up, radiographs demonstrated satisfactory screw position, maintained scaphoid alignment, stable graft incorporation, and no evidence of hardware failure or carpal collapse. Clinical recovery progressed as expected with immobilization followed by supervised rehabilitation. This report highlights the importance of considering Preiser disease in patients with persistent wrist pain despite the absence of trauma. MRI plays a crucial role in early diagnosis, and joint-preserving reconstruction using cyst excision, distal radius cancellous bone grafting, and headless compression screw fixation may provide encouraging early clinical and radiographic outcomes in appropriately selected patients.

Keywords: Preiser disease, Idiopathic scaphoid osteonecrosis, Avascular necrosis of scaphoid, Distal radius bone graft, Headless compression screw, Bone grafting

INTRODUCTION

Preiser disease refers to idiopathic avascular necrosis (AVN) of the carpal scaphoid occurring in the absence of an antecedent fracture. It was first described by Georg Preiser in 1910, who termed it a "rarefying osteitis" of the scaphoid and distinguished it from AVN secondary to scaphoid fracture, likening its pathogenesis to Kienböck disease of the lunate.^{1,2}

The scaphoid's vulnerability to ischemic injury stems from its precarious, largely retrograde blood supply. Vascularization is derived mainly from branches of the

radial artery, with the dorsal scaphoid branch supplying approximately 70-80% of intraosseous flow and the palmar branch supplying the remainder.^{1,3} This anatomical pattern leaves the proximal pole particularly susceptible to devascularization once flow is interrupted.

The etiology of Preiser disease remains incompletely understood and is frequently idiopathic. Proposed contributing factors include chronic corticosteroid therapy, alcoholism, connective tissue disease, coagulation fibrinolytic abnormalities, congenital scaphoid hypoplasia, smoking, and iatrogenic vascular injury—including cases reported after transradial arterial catheterization.^{2,4,5}

Clinically, patients typically present with insidious, progressive radial-sided wrist pain, swelling, and tenderness over the anatomical snuffbox, often without a history of fracture or significant trauma, evolving over months to years.⁴ Plain radiographs are frequently normal in early disease, which delays diagnosis; magnetic resonance imaging (MRI) is considered the gold standard, demonstrating altered marrow signal within the scaphoid before radiographic changes become evident.^{3,5}

Two classification systems commonly guide staging and management. Herbert and Lanzetta's four-stage radiographic classification (1994) categorize disease from normal radiographs with a positive bone scan (stage I), through increased proximal pole density (stage II) and proximal pole fragmentation (stage III), to carpal collapse (stage IV).^{3,5,6} Kalainov et al later proposed a complementary MRI-based classification distinguishing diffuse scaphoid involvement (type 1, associated with worse outcomes) from limited proximal pole involvement (type 2, more favourable prognosis).^{3,6}

Given the rarity of Preiser disease and the absence of prospective comparative trials, treatment remains controversial and is largely guided by surgeon preference and disease stage.^{5,6}

Options range from conservative management (immobilization, NSAIDs, electrical stimulation) in early-stage disease, to scaphoid-preserving procedures such as vascularized bone grafting, curettage with bone grafting, closing wedge radial osteotomy, arthroscopic debridement, and internal fixation with bone grafting, to salvage procedures-including proximal row carpectomy, four-corner fusion, silicone replacement, or wrist arthrodesis-reserved for advanced-stage disease with degenerative change.^{3,5,6}

Open reduction and internal fixation of the scaphoid using a 2.4 mm Meril headless compression screw, combined with cyst excision from the scaphoid and autologous bone grafting from the distal end of the radius (DER), was the procedure performed in our patient.

This joint-preserving approach provides structural support to the scaphoid, facilitates incorporation of the bone graft, promotes revascularization and osseous healing, and aims to preserve wrist function while preventing progression to carpal collapse.^{1,5}

Given the rarity of this condition, with a limited number of cases reported in the literature to date, we present this case of Preiser disease managed with open reduction and internal fixation using a 2.4 mm Meril headless compression screw, cyst excision from the scaphoid, and autologous bone grafting from DER at our institution to contribute to the existing body of evidence and highlight considerations relevant to diagnosis and surgical decision-making.

CASE REPORT

Patient presentation

A 47-year-old right-hand-dominant male presented with a three-year history of progressively worsening left wrist pain, weakness, reduced grip strength, and functional limitation affecting activities of daily living, driving, and load-bearing activities. The patient denied any history of previous wrist trauma or scaphoid fracture. Conservative treatment, including analgesics, activity modification, and wrist immobilization, failed to provide symptomatic relief, and symptoms progressively worsened, prompting definitive orthopedic evaluation.

Clinical examination demonstrated localized tenderness over the scaphoid on the radial aspect of the wrist, reduced grip strength, and restricted terminal wrist flexion and extension (Figures 1 and 2). No neurovascular deficit was identified.



Figure 1: Pre-operative clinical photograph showing restricted wrist extension.



Figure 2: Pre-operative clinical photograph showing restricted wrist flexion.

Plain radiographs of the left wrist did not demonstrate a fracture line but showed cystic changes within the scaphoid. MRI (Figures 3-5) demonstrated diffuse marrow signal alteration involving the scaphoid, characterized by low signal intensity on T1-weighted images and marrow edema on proton-density fat-saturated sequences, consistent with idiopathic avascular necrosis of the scaphoid. There was no evidence of carpal collapse, scaphoid fragmentation, radiocarpal arthritis, or midcarpal degenerative changes. Based on the clinical presentation

and MRI findings, a diagnosis of idiopathic Preiser disease was established.

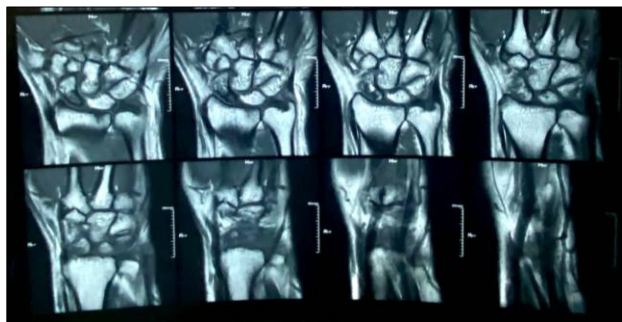


Figure 3: Coronal T1-weighted MRI of the left wrist showing altered marrow signal intensity within the scaphoid.

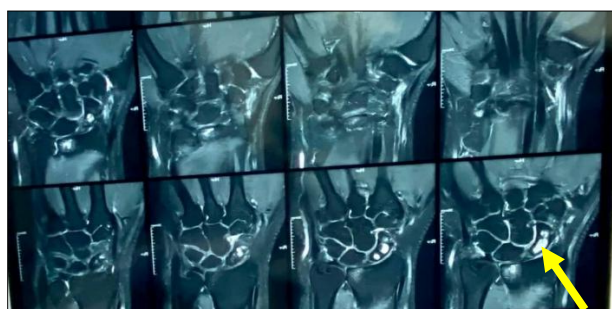


Figure 4: Coronal proton-density fat-saturated (PDFS) demonstrating diffuse marrow edema and associated intraosseous cystic lesion (yellow arrow).

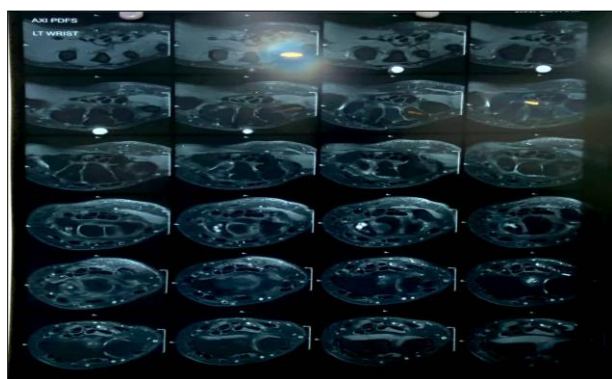


Figure 5: Axial PDFS MRI of the left wrist demonstrating scaphoid marrow signal change.

Surgical technique

Considering the chronicity of symptoms, the presence of a cystic lesion within the scaphoid, radiological evidence of avascular necrosis, and preservation of the carpal architecture without collapse or degenerative arthritis, a joint-preserving surgical procedure was planned. The patient underwent scaphoid cyst excision, autologous cancellous bone grafting from the distal radius, and

internal fixation using a 2.4-mm Meril headless compression screw.

Under brachial block anesthesia, the patient was positioned supine with a pneumatic tourniquet applied to the upper arm. A standard volar approach to the wrist was used to expose the scaphoid. The cystic and necrotic bone was thoroughly curetted and excised, creating a healthy bleeding bony bed suitable for graft incorporation. Autologous cancellous bone graft harvested from the distal radius was combined with bone graft substitute and packed into the scaphoid defect to restore structural integrity and promote osteogenesis. The distal radius was selected as the donor site because it provides adequate cancellous bone through the same surgical incision while avoiding the donor-site morbidity associated with iliac crest bone graft harvesting.

Following graft placement, internal fixation was achieved using a 2.4×18 mm Meril headless compression screw to provide compression, maintain scaphoid alignment, prevent collapse during graft incorporation, and enhance construct stability. The superficial branch of the radial nerve was carefully protected throughout the procedure. Implant position, screw length, restoration of scaphoid alignment, and overall fixation were confirmed intraoperatively using fluoroscopy before wound closure.

Postoperative management

The patient was discharged on the first postoperative day with oral analgesics, antibiotics, and gastric protection. Wound inspections were performed on postoperative days 2 and 4, and sutures were removed after two weeks. The wrist was immobilized in a below-elbow slab for six weeks to facilitate graft incorporation and protect the reconstruction. Following immobilization, a structured hand rehabilitation programme consisting of supervised physiotherapy and splint-assisted mobilization was initiated to progressively restore wrist motion and grip strength. The patient was advised to avoid heavy manual activities for two months and driving for three months following surgery.



Figure 6: Intraoperative photograph demonstrating the volar approach to the scaphoid. The flexor carpi radialis (FCR) tendon has been retracted to expose the scaphoid.



Figure 7: At 1 month X-ray AP view.

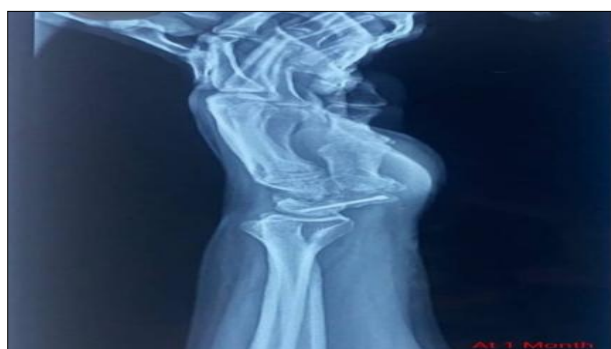


Figure 8: At 1 month X-ray LAT view.

DISCUSSION

Preiser disease is a rare form of AVN of the carpal scaphoid, first described by Preiser in 1910, and is defined specifically by its occurrence in the absence of an antecedent fracture.^{1,2} This distinguishes it from the far more common scenario of scaphoid AVN secondary to an ununited fracture, in which vascular compromise follows directly from disruption of the bone's already precarious blood supply. Establishing this distinction is clinically important, as the two entities, though similar in presentation, differ in underlying etiology and carry different implications for prognosis and treatment planning.^{3,4}

The scaphoid receives its blood supply predominantly from branches of the radial artery, with the dorsal carpal branch supplying 70-80% of intraosseous flow in a retrograde fashion, leaving the bone inherently vulnerable to ischemic insult even without a fracture.¹ In Preiser disease, this vulnerability manifests spontaneously, and the precise trigger for vascular interruption in a given patient often remains unclear. Proposed contributing factors described in the literature include corticosteroid therapy, alcoholism, connective tissue disorders, coagulation and fibrinolytic abnormalities, congenital scaphoid hypoplasia, smoking, and, in some reports, iatrogenic vascular injury such as that following transradial arterial catheterization.^{2,4,5} In a substantial proportion of cases, however, no clear precipitating factor

is identified, and the condition is considered truly idiopathic.^{3,6}

Clinically, Preiser disease typically presents with insidious, progressive radial-sided wrist pain, tenderness over the anatomical snuffbox, reduced grip strength, and diminished range of motion, often evolving gradually over months to years, as demonstrated in the present case, where the patient experienced a three-year history of progressive symptoms before definitive diagnosis. Because plain radiographs are frequently unremarkable in early disease, the diagnosis is often delayed or mistaken for other causes of radial-sided wrist pain, including occult scaphoid fracture. MRI is considered the gold standard for early diagnosis, as it can detect marrow signal changes within the scaphoid well before radiographic abnormalities become apparent and, importantly, helps exclude a fracture line, thereby supporting the diagnosis of idiopathic Preiser disease.

Correct identification of Preiser disease as distinct from a scaphoid fracture is essential, since the two conditions are managed differently: a fracture (with or without evolving AVN) is generally addressed with fixation and, where needed, bone grafting to achieve union, whereas idiopathic Preiser disease may be managed with joint-preserving procedures such as cyst excision from the scaphoid, screw fixation using a 2.4 mm Meril headless compression screw, and autologous bone grafting from the DER or other joint-preserving procedures depending on disease stage and cartilage integrity.^{3,4,6} Misattributing this presentation to fracture risks inappropriate management directed at bony union rather than addressing the underlying vascular pathology.

The present case demonstrates that early recognition of Preiser disease using MRI allows timely intervention before the development of carpal collapse or degenerative arthritis. In appropriately selected patients, scaphoid cyst excision, autologous distal radius cancellous bone grafting, and headless compression screw fixation may preserve the native scaphoid and provide encouraging early clinical and radiographic outcomes. However, further studies with larger patient cohorts and longer follow-up are required to determine the long-term durability of this joint-preserving technique.

CONCLUSION

Preiser disease should be considered in patients presenting with persistent radial-sided wrist pain without a history of preceding trauma. Magnetic resonance imaging is essential for early diagnosis and for differentiating idiopathic scaphoid osteonecrosis from occult scaphoid fracture. In patients with preserved carpal alignment and no evidence of carpal collapse or degenerative arthritis, scaphoid cyst excision, autologous distal radius cancellous bone grafting, and headless compression screw fixation can serve as an effective joint-preserving surgical option,

providing early pain relief, improved wrist function, and satisfactory radiographic stability.

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