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Case Report

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MRI-Confirmed Resolution of Persistent Radiocarpal Synovitis and Ulnar Styloid Bone Marrow Oedema Following Single High-Concentration Platelet-Rich Plasma Injection: A Case Report

Hassan Mubark

Rheumatologist, Auckland Regenerative Clinic, Ormiston Specialist Centre, 125 Ormiston Road / Flat Bush, Auckland 2019, New Zealand

Corresponding author

Hassan Mubark, Rheumatologist, Auckland Regenerative Clinic, Ormiston Specialist Centre, 125 Ormiston Road / Flat Bush, Auckland 2019, New Zealand.

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ABSTRACT

Persistent monoarticular radiocarpal synovitis with associated ulnar styloid bone marrow oedema (BMO) presents a significant diagnostic and therapeutic challenge, particularly when systemic inflammatory disease cannot be definitively established. Differentiating early inflammatory arthritis from localized osteochondral pathology is essential to prevent unnecessary long-term immunomodulatory therapy and to ensure appropriate targeted management.

Magnetic resonance imaging (MRI) is central to evaluation. Bone marrow oedema is increasingly recognized as an active subchondral pathological entity characterized by trabecular microinjury, increased vascularity, inflammatory cell infiltration, and altered bone remodelling rather than simple fluid accumulation. The concurrent presence of synovitis and BMO reflects biological interaction within the osteochondral unit, potentially perpetuating pain, inflammatory signalling, and structural dysfunction at the radiocarpal joint.

We report the case of a 40-year-old female with a two-year history of persistent radiocarpal synovitis and progressive ulnar styloid BMO. Hydroxychloroquine was initiated following a single positive antinuclear antibody titre (1:320); however, repeat autoimmune serology was negative, inflammatory markers remained normal, and established classification criteria for systemic lupus erythematosus were not fulfilled. Symptoms persisted despite multiple corticosteroid injections.

After discontinuation of systemic therapy, a single ultrasound-guided injection of high-concentration, leukocyte-poor platelet-rich plasma (PRP) was administered. was administered targeting the radiocarpal joint and ulnar styloid region. Four-month follow-up MRI demonstrated complete resolution of radiocarpal synovitis and ulnar styloid BMO, with interval healing of the previously torn triangular fibrocartilage complex (TFCC).

This case underscores the importance of diagnostic precision in isolated wrist synovitis and suggests that localized biologic modulation of the synovium–subchondral bone interface may represent a promising therapeutic strategy. Prospective controlled studies are warranted to confirm reproducibility and further elucidate the underlying biological mechanisms.

Keywords: Radiocarpal Synovitis, Bone Marrow Oedema, Wrist Monoarthritis, Subchondral Bone Remodeling, Platelet-Rich Plasma, Osteochondral Unit, Triangular Fibrocartilage Complex

Introduction

Chronic monoarticular wrist synovitis requires careful diagnostic stratification. Infection, crystal arthropathy, mechanical injury, early inflammatory arthritis, and localized osteochondral pathology must be systematically excluded prior to escalation to systemic disease-modifying therapy [1]. Misclassification may expose patients to unnecessary immunomodulatory treatment while failing to address localized drivers of pathology.

Bone marrow oedema (BMO) detected on MRI sequences is now understood as a dynamic subchondral lesion. Histologic correlation studies demonstrate trabecular microfracture, increased vascularity, inflammatory cell infiltration, and fibrosis rather than simple fluid deposition [2]. These lesions are metabolically active and biomechanically significant.

In large joints such as the knee, BMO lesions strongly correlate with pain severity, cartilage loss, and structural progression, suggesting that subchondral bone participates actively in symptom generation and disease evolution [3]. Contemporary osteochondral unit models describe bidirectional signaling between synovium and subchondral bone mediated

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by inflammatory cytokines, angiogenic factors, and matrix-degrading enzymes [4]. Cytokines such as IL-1β and TNF-α amplify osteoclast activation and synovial inflammation, perpetuating a cycle of synovitis and bone remodeling.

Platelet-rich plasma (PRP) contains supraphysiologic concentrations of platelets that release growth factors including PDGF, TGF-β, VEGF, and IGF-1. These bioactive mediators can modulate NF-κB signaling, alter macrophage polarization, and influence synoviocyte behavior [5]. Clinical evidence is most robust in knee osteoarthritis, where PRP has demonstrated reductions in synovial inflammation and improvements in imaging biomarkers compared with hyaluronic acid in several studies [6]. However, evidence in small joints such as the wrist remains limited. We report and interesting case of using high concentration PRP to resolve wrist synovitis and BMO.

Case Presentation

A 40-year-old woman presented with atraumatic right wrist pain beginning in December 2023. She reported progressive swelling, stiffness, reduced grip strength, and increasing limitation in daily activities. There was no history of preceding trauma, infection, or systemic symptoms.

She was initially reviewed by an orthopaedic surgeon and underwent MRI in April 2024, which demonstrated radiocarpal joint effusion, synovitis, and focal triquetral bone marrow oedema. Treatment with oral anti-inflammatory medication and a corticosteroid injection provided no sustained relief.

A repeat MRI in March 2025 revealed persistent diffuse radiocarpal synovitis with interval progression to ulnar styloid bone marrow oedema, without cortical erosions or cartilage collapse, together with a tear of the triangular fibrocartilage complex (TFCC) (Figure 1).

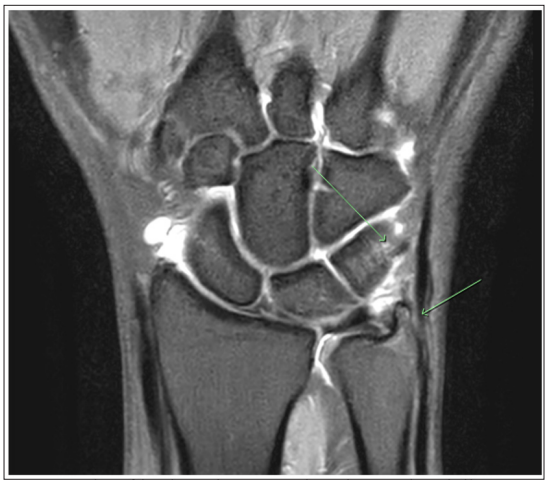

Figure 1: Coronal MRI of the right wrist demonstrating TFCC capsular synovitis, ulnar styloid bone marrow edema, and a triangular fibrocartilage complex (TFCC)

She was referred to a rheumatologist, where initial assessment identified a single positive antinuclear antibody (ANA) titre of 1:320, leading to commencement of hydroxychloroquine therapy. However, three subsequent ANA tests performed at a different laboratory were negative. Anti–double-stranded DNA, rheumatoid factor, and anti–cyclic citrullinated peptide antibodies were also negative, and inflammatory markers (CRP and ESR) remained within normal limits.

Prior to our involvement, she had received three ultrasound-guided intra-articular corticosteroid injections, which provided only transient symptomatic relief. She subsequently sought a second opinion. On reassessment, she did not meet classification criteria for systemic lupus erythematosus or rheumatoid arthritis. Hydroxychloroquine was discontinued, and a working diagnosis of chronic inflammatory monoarthritic of the right wrist was established.

A repeat MRI performed on 1 August 2025 demonstrated persistent radiocarpal synovitis with new ulnar styloid bone marrow oedema and possible early erosion.

Given the isolated monoarticular presentation in the absence of systemic inflammatory disease, a trial of localized biologic modulation was proposed as a safe investigative strategy. This approach was u

Intervention

In September 2025, following informed consent, 40 mL of autologous venous blood was collected and processed using a standardized single-spin protocol (5 minutes), yielding 7 mls of high-concentration, leukocyte-poor platelet-rich plasma (PRP). Although the exact platelet fold-increase was not quantified, the preparation aimed to achieve a leukocyte-poor, high-platelet formulation to minimise pro-inflammatory cytokine burden.

Under ultrasound guidance and after local anaesthesia with 0.2% ropivacaine, 4 mL of PRP was injected intra-articularly into the radiocarpal joint, and 3 mL was delivered directly into and around the ulnar styloid region, including the ulnocarpal joint and triangular fibrocartilage complex (TFCC) (Figure 2), targeting areas of active synovitis and subchondral pathology.

Post-procedure, non-steroidal anti-inflammatory drugs (NSAIDs) were avoided to preserve platelet-mediated biological signalling.

Clinical and Imaging Outcome

Four months following PRP therapy, the patient reported a marked reduction in pain, with near-complete resolution of symptoms, and complete disappearance of swelling. Wrist range of motion was fully restored, grip strength improved substantially, and daily function returned to normal. Notably, she progressed from experiencing persistent daily pain despite anti-inflammatory therapy prior to PRP treatment to being able to perform a handstand using the right wrist.

Repeat MRI at four months post-injection demonstrated:

- Complete resolution of ulnar styloid bone marrow oedema (BMO)
- Absence of radiocarpal synovitis
- Restoration of normal marrow signal intensity
- Structural normalization of the previously disrupted triangular fibrocartilage complex (TFCC)

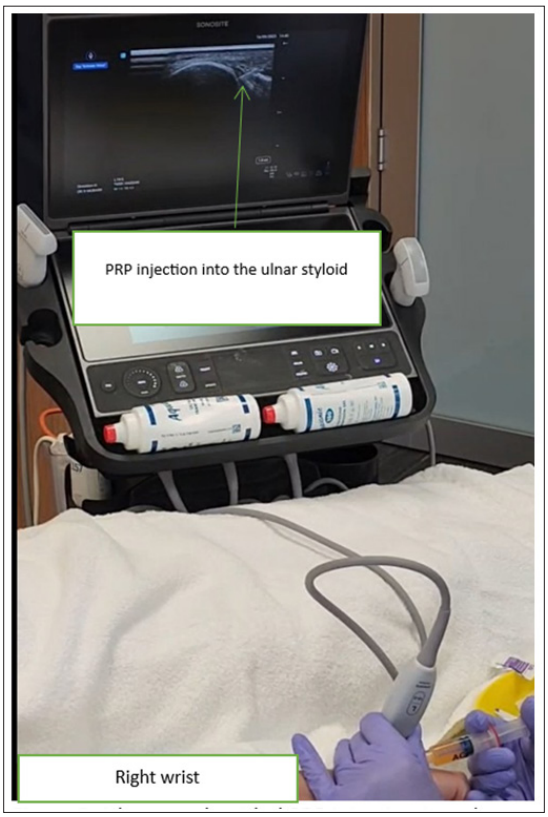

Figure 2: Ultrasound-guided PRP injection into the right ulnar styloid

No erosions or degenerative progression were identified. Compared with prior MRI studies dating back to April 2024, there was complete resolution of the previously documented ulnar styloid BMO and no recurrence of radiocarpal synovitis. A mild pisotriquetral joint effusion persisted without associated synovitis, unchanged from the previous scan. There was no evidence of new bone marrow oedema, joint synovitis, or tenosynovitis elsewhere in the right wrist, with imaging confirming interval healing of the TFCC (Figure 3).

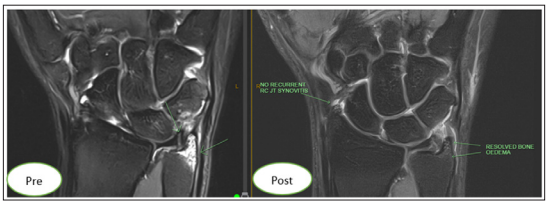

Figure 3: Coronal MRI of the right wrist pre- and post-PRP showing resolution of ulnar styloid bone marrow edema, absence of radiocarpal synovitis or erosions, and healed TFCC

Discussion

Bone marrow oedema (BMO) represents a biologically active osteochondral lesion rather than a passive imaging phenomenon. Histopathologic correlation studies demonstrate that MRI-defined BMO corresponds to trabecular microfracture, fibrovascular marrow replacement, increased intraosseous pressure, and inflammatory infiltration within subchondral bone [7]. These findings confirm that BMO reflects active bone remodelling rather than simple interstitial fluid accumulation.

Advanced MRI biomarker studies further show that BMO lesions correlate with pain severity and structural progression in osteoarthritis, and that reduction or resolution of BMO is associated with clinical improvement and stabilization of joint pathology [8]. Although these observations are primarily derived from large joints, the biological mechanisms governing subchondral remodelling are not joint-specific and are likely applicable to the wrist.

At the molecular level, inflammatory cytokines such as IL-1β and TNF-α play a central role in sustaining osteoclast activation and synovial inflammation. These mediators promote RANKL-driven osteoclast genesis, perpetuating bone resorption and inflammatory amplification within the osteochondral unit [9]. Persistent cytokine signalling therefore provides a mechanistic explanation for the coexistence of synovitis and BMO in chronic monoarthritic.

Platelet-rich plasma (PRP) contains concentrated growth factors and bioactive mediators capable of influencing synovial cell behaviour. Experimental evidence demonstrates that PRP can modulate synoviocyte inflammatory signalling pathways, suppress NF-κB activation, and reduce production of pro-inflammatory cytokines [10]. Through attenuation of these pathways, PRP may indirectly downregulate osteoclast-mediated bone remodelling and interrupt the feed-forward cycle between synovium and subchondral bone.

Clinical meta-analyses in knee osteoarthritis indicate that PRP provides superior symptomatic improvement compared with hyaluronic acid and may exert favourable effects on structural imaging parameters [11]. While extrapolation to small joints must be undertaken cautiously, these findings support the broader concept of biologic modulation of joint homeostasis rather than purely symptomatic therapy.

Contemporary orthopaedic literature increasingly emphasizes biologic strategies aimed at influencing cartilage and subchondral bone remodelling as part of joint preservation paradigms [12]. In the present case, the simultaneous MRI-confirmed resolution of synovitis, normalization of ulnar styloid BMO, and restoration of TFCC integrity suggest coordinated modulation of inflammatory signalling, osteoclast activity, and matrix repair within the osteochondral unit.

Prospective controlled studies are required to confirm reproducibility and to further define optimal biologic preparation parameters in persistent wrist monoarthritic.

Conclusion

This case demonstrates MRI-confirmed resolution of persistent radiocarpal synovitis and ulnar styloid bone marrow oedema following a single high-concentration, leukocyte-poor platelet-rich plasma, with concurrent restoration of triangular fibrocartilage complex integrity. The simultaneous normalization of synovial inflammation, subchondral marrow signal, and fibrocartilaginous structure suggests biologic modulation of the osteochondral unit. Controlled prospective studies are required to confirm reproducibility and clarify underlying mechanisms.

Patient Consent

Written informed consent was obtained for publication.

Acknowledgement
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