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Platelet-Rich Plasma in Osteoarthritis: Types, Mechanisms, and Clinical Applications—A Narrative Review

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Abstract

Background: Platelet-rich plasma (PRP) has emerged as a promising biological intervention for osteoarthritis, though its clinical utility and optimal formulation remain debated. This narrative review synthesizes current evidence on PRP types, biological mechanisms, and applications in osteoarthritis management. ***Objectives:*** To provide a comprehensive overview of PRP classification systems, mechanisms of action, clinical efficacy in osteoarthritis treatment, and comparative effectiveness with standard therapies. ***Methods:*** A targeted literature review of peer-reviewed publications from 2020–2025 was conducted using PubMed, Cochrane Library, and Scopus, focusing on PRP types, mechanisms, osteoarthritis applications, clinical outcomes, and safety. ***Results:*** PRP preparations are classified primarily by leukocyte and fibrin content: pure PRP (P-PRP/leukocyte-poor), leukocyte-rich PRP (L-PRP), and platelet-rich fibrin (PRF). High-platelet-concentration PRP (≥1,000,000 platelets/μL) provides superior and durable pain relief and functional improvement compared with low-platelet formulations. PRP exerts anti-inflammatory, anabolic, and immunomodulatory effects through growth factors (PDGF, TGF-β, VEGF), cytokines, and macrophage polarization. Meta-analyses demonstrate clinically significant improvements in pain (VAS) and function (WOMAC scores) at 3–12 months, with benefits superior to placebo, hyaluronic acid, and corticosteroids. Adverse events are generally minor and transient, with higher post-injection pain/swelling in leukocyte-rich formulations. ***Conclusions:*** PRP represents an effective, safe, biologic treatment option for symptomatic osteoarthritis, particularly early to moderate disease. High-platelet formulations and multiple injection protocols yield superior outcomes. Standardization of preparation protocols, patient selection criteria, and long-term follow-up remain priorities for broader clinical adoption.

Keywords: Platelet-rich plasma, PRP, osteoarthritis, knee OA, leukocyte-rich PRP, leukocyte-poor PRP, platelet-rich fibrin, growth factors, regenerative medicine, intra-articular injection.

INTRODUCTION

Osteoarthritis (OA) represents one of the most prevalent chronic degenerative joint diseases globally, causing substantial pain, functional disability, and reduced quality of life, particularly in aging populations.[1,2] The pathophysiology of OA involves progressive loss of articular cartilage, synovial inflammation, and structural changes in the entire joint, with limited capacity for spontaneous repair.[1,3] Current standard therapies—including nonsteroidal anti-inflammatory drugs (NSAIDs), intra-articular corticosteroid injections, and hyaluronic acid (HA)—provide only modest and time-limited symptom relief, often failing to prevent disease progression.[2,3]

In recent years, synthesis andplasma (PRP) has emerged as a promising orthobiologic approach to address the unmet clinical need for disease-modifying treatments in OA.[3,4,5] PRP is an autologous blood product obtained via centrifugation, containing a supraphysiologic concentration of platelets, growth factors, and bioactive molecules stored in platelet alpha-granules.[1,4] The theoretical appeal of PRP rests on its capacity to modulate the intra-articular

microenvironment, reduce inflammation, stimulate chondrocyte proliferation, enhance cartilage matrix synthesis, and promote angiogenesis and synovial tissue healing.[3,4,6]

However, substantial heterogeneity in PRP preparation protocols, composition (particularly leukocyte and fibrin content), platelet concentration, and injection schedules has complicated the interpretation of clinical evidence and hindered standardization.[3,7] This narrative review synthesizes current knowledge on PRP types and characteristics, biological mechanisms of action, clinical efficacy in OA management, comparative effectiveness versus established therapies, safety profiles, and recommendations for optimizing clinical translation. The review aims to inform clinicians, researchers, and policymakers on evidence-based selection and use of PRP in osteoarthritis.

Literature Review

Classification and Types of Platelet-Rich Plasma

Classification Systems

Platelet concentrates (PCs), including PRP formulations, are classified based on leukocyte and fibrin content, as well as red blood cell (RBC) enrichment. [8,9] This classification system, standardized by international consensus, distinguishes the following primary types:

Pure Platelet-Rich Plasma (P-PRP or LP-PRP): Also termed leukocyte-poor PRP, this preparation contains concentrated platelets with minimal leukocyte content and no fibrin matrix. It is obtained via two-step centrifugation (typically 600×g for 10 minutes, followed by 1600×g for 7 minutes) without the buffy coat layer (leukocyte ring).[8,9,10] P-PRP yields a platelet concentration increase of approximately 3.5–4.9-fold compared with whole blood.[10] This formulation is preferred when minimizing post-procedure inflammation is critical.[8,11]

Leukocyte-Rich PRP (L-PRP or LR-PRP): This preparation includes leukocytes in the final concentrate, achieved by incorporating the buffy coat layer during collection.[8,9,10] L-PRP achieves higher platelet concentrations (4.6–4.9-fold increase versus whole blood) and elevated leukocyte concentrations (2.4–9-fold versus whole blood), depending on preparation protocol.[10] The inclusion of leukocytes is intended to enhance immune modulation and inflammatory signalling; however, clinical benefit versus P-PRP remains debated.[8,9,12]

Platelet-Rich Fibrin (PRF): Pure PRF (P-PRF) is prepared from whole blood collected in a clot-activator tube and subjected to single-step centrifugation (600×g for 10 minutes) without anticoagulant.[9,10] PRF forms a fibrin matrix that acts as a natural scaffold and reservoir for sustained growth factor release, although platelet concentration is lower than in PRP preparations (1.5–2.1-fold versus whole blood).[10,13] L-PRF variants include both platelets and leukocytes within the fibrin structure.[9]

Variants: Additional classifications include Red-PRP (RBC-enriched, RBC >10% of volume) and Red-L-PRP (RBC and leukocyte-enriched), though these are less commonly used clinically.[9]

Cellular and Biochemical Composition

The cellular composition of different PRP types differs significantly, with implications for biological activity and clinical efficacy[10] as shown in Table-1.

Table 1: Different types of Platelet concentrates

S.No	PRP Type	Platelet Concentration (fold-increase)	Leukocyte Enrichment	Fibrin Matrix	Key Characteristics
1	**P-PRP (LP-PRP)**	3.5–4.1×	Minimal / Low	No	Leukocyte-poor; minimal post-injection inflammation
2	**L-PRP (LR-PRP)**	4.6–4.9×	High (2.4–9×)	No	Leukocyte-rich; enhanced immune modulation; higher post-injection reactions
3	**P-PRF**	1.5–2.1×	Minimal	Yes	Fibrin scaffold; sustained growth factor release; lower platelet recovery in pathologic states
4	**L-PRF**	Variable	High	Yes	Combined fibrin matrix and leukocyte-rich composition

P-PRP (Pure/Leukocyte-Poor PRP, L-PRP (Leukocyte-Rich PRP), P-PRF (Pure Platelet-Rich Fibrin):L-PRF (Leukocyte and Platelet-Rich Fibrin):

The platelet concentration threshold for optimal clinical efficacy is debated. A consensus cutoff of 1,000,000 ± 20% platelets/μL (1.2×10^6/mL) defines high-platelet PRP, while concentrations <800,000 platelets/μL are considered low-platelet PRP.[11,14] High-platelet PRP consistently demonstrates superior pain relief and durable functional improvement compared with low-platelet formulations at 3, 6, and 12-month follow-ups.[11,14] Additionally, a minimum of 5 billion platelets per injection appears necessary for clinically meaningful efficacy in knee OA.[12]

Growth Factor and Cytokine Content

PRP preparations contain multiple bioactive molecules released from platelet alpha-granules upon activation with calcium chloride:[8,10,13]

Key Growth Factors:

Platelet-Derived Growth Factor (PDGF-BB): Concentrations range from 342–1634 pg/mL depending on PRP type, with L-PRP and PRF typically showing 2.7–3.2-fold higher concentrations than P-PRP.[10] PDGF-BB stimulates fibroblast proliferation and collagen synthesis, essential for matrix remodeling.[8,13]

Transforming Growth Factor-Beta (TGF-β1): Concentrations range from 750–1809 pg/mL, with PRF showing highest levels in healthy individuals. TGF-β1 promotes chondrocyte proliferation, extracellular matrix production, and anti-inflammatory responses.[8,10,13]

Vascular Endothelial Growth Factor (VEGF-A): Concentrations vary from 38–348 pg/mL, with L-PRP and PRF showing 2–5-fold higher levels than P-PRP.[10] VEGF enhances angiogenesis, vascular remodeling, and tissue perfusion.[8,13]

Additional Factors: Epidermal growth factor (EGF), basic fibroblast growth factor (bFGF), insulin-like growth factor (IGF-1), interleukin-10 (IL-10), and hepatocyte growth factor (HGF) are present in varying concentrations.[8,13]

Notably, growth factor concentrations in PRP are similar between healthy individuals and patients with degenerative conditions, though patients with bone defects demonstrate reduced leukocyte enrichment in L-PRP and lower platelet recovery in PRF preparations.[10] Growth factor concentration does not consistently correlate with platelet count, suggesting that other factors (e.g., leukocyte number, preparation method, patient-specific variables) influence bioactive molecule availability.[10,15]

Biological Mechanisms of Action in Osteoarthritis

Anti-Inflammatory and Immunomodulatory Effects

PRP exerts potent anti-inflammatory effects through multiple mechanisms.[3,4,6,16] In knee OA, PRP modulates the intra-articular microenvironment by suppressing pro-inflammatory cytokines (tumor necrosis factor-alpha [TNF-α], interleukin-1-beta [IL-1β], and matrix metalloproteinases [MMPs]) while promoting anti-inflammatory mediators (IL-10).[3,6,16]

Recent mechanistic studies reveal that PRP achieves pain relief, in part, through macrophage polarization—shifting M1-phenotype (pro-inflammatory) macrophages toward M2-phenotype (anti-inflammatory) macrophages by inhibiting the NF-κBsignaling pathway.[16] This shift reduces the release of pain-promoting factors such as nerve growth factor (NGF), TNF-α, and IL-1β, thereby providing sustained analgesia.[16] Leukocyte-rich PRP may enhance these immunomodulatory effects through the release of additional cytokines and antimicrobial factors, though this comes at the cost of increased post-injection inflammatory reactions.[6,12]

Cartilage Regeneration and Matrix Preservation

PRP stimulates chondrocyte proliferation and matrix synthesis through growth factor signaling.[3,4,6] High concentrations of TGF-β and BMPs in PRP promote extracellular matrix production, particularly proteoglycans and Type II collagen—essential components of articular cartilage.[3,6,17] Additionally, PRP inhibits catabolic pathways by downregulating matrix-degrading enzymes (MMPs and ADAMTs), thereby slowing or preventing cartilage loss.[3,6]

Synovitis reduction represents another mechanism of cartilage preservation. Multiple PRP injections improve synovitis and protect cartilage in animal models, while clinical studies show that PRP-treated joints retain cartilage thickness better than control-treated or placebo-treated knees over 12–18 months of follow-up.[3,17]

Angiogenesis and Tissue Repair

VEGF and other pro-angiogenic factors in PRP enhance vascular remodeling and tissue perfusion within the OA joint, improving nutritional support for healing tissues and promoting synovial tissue repair.[3,4,6] The fibrin matrix in PRF

formulations provides a biologic scaffold for cell migration and growth factor retention, supporting sustained angiogenesis and regenerative processes.[13]

Integration with Multiple Signaling Pathways

PRP's therapeutic effects are multifactorial, involving numerous signaling pathways including PI3K/Akt, MAPK/ERK, and Wnt/β-catenin cascades.[3,8] This multifactorial approach—combining growth factor delivery, immune regulation, and microenvironment modulation—underpins PRP's broad regenerative potential and may explain its sustained clinical benefits beyond the placebo effect.[3]

Clinical Efficacy in Osteoarthritis: Evidence from Meta-Analyses and Randomized Controlled Trials

Pain and Functional Outcomes

Recent meta-analyses of randomized controlled trials (RCTs) demonstrate that intra-articular PRP injection provides clinically significant and sustained improvements in pain and function for knee OA.[11,14,18,19]

A 2025 meta-analysis by Bensa et al., including 18 RCTs with 1995 patients, found that PRP offered statistically and clinically superior improvements compared with placebo in Visual Analog Scale (VAS) pain scores and Western Ontario and McMaster Universities Arthritis Index (WOMAC) functional scores at all follow-up points (1, 3, 6, and 12 months).[11] Critically, improvements exceeded the Minimal Clinically Important Difference (MCID) threshold for VAS (1.37 points) at 3- and 6-month follow-ups and for WOMAC at all time points, indicating that results translate into patient-perceived clinical benefit.[11]

Platelet concentration strongly influences treatment efficacy.High-platelet PRP (≥1,000,000 platelets/μL) provided clinically significant pain relief exceeding MCID at 3, 6, and 12 months of follow-up, whereas low-platelet PRP (<800,000 platelets/μL) failed to achieve MCID-level pain reduction at any follow-up point.[11] For functional outcomes (WOMAC), both high- and low-platelet PRP showed clinical significance at 3–6 months; however, benefits persisted through 12 months only in the high-platelet group, indicating superior durability.[11]

A separate meta-analysis by Filardo et al. (2024) including 48 studies with 9,338 knees found that PRP demonstrated superior short-term functional recovery and sustained long-term benefits (at 12 months and beyond) compared with hyaluronic acid and corticosteroids.[19] Combined PRP + HA therapy showed even greater pain and functional improvements than PRP alone, particularly at 12-month and longer follow-ups.[18,19]

Durability and Long-Term Follow-Up

A prospective cohort study of 431 patients receiving high-volume, highly pure PRP reported sustained clinical improvement through 18 months of follow-up.[20] Approximately 70–80% of patients demonstrated improvement in WOMAC and pain VAS at various follow-up intervals. The proportion of OMERACT-OARSI responders (patients meeting predefined response criteria) peaked at 6 months (56.2% in the total cohort; 60.4% in severe patients) and remained elevated at 18 months, with only 8.4% experiencing treatment failure.[20] This durability contrasts favorably with the transient effects of corticosteroid injections.[19,20]

Efficacy by Disease Severity

PRP efficacy extends across the spectrum of OA severity. In severe (Kellgren-Lawrence Grade 3–4) OA patients, PRP produced comparable or superior improvements to those in mild-to-moderate disease, with WOMAC and VAS reductions similar to mild OA cohorts.[20] This suggests potential value as a bridge therapy to delay or avoid joint replacement in patients with end-stage disease who are not surgical candidates or wish to defer surgery.[20,21]

Dose-Response and Injection Protocols

Multiple PRP injections (typically 3 injections) yield superior long-term outcomes compared with single-injection protocols, with sustained benefits at 6 and 12 months.[14,21] A network meta-analysis examining various PRP doses found that higher-dose PRP regimens (PRP3, indicating 3 injections) provided superior pain relief and functional improvement compared with lower-dose protocols.[14] The cumulative biological effects of multiple injections—including sustained growth factor release, enhanced cartilage repair, and prolonged inflammation modulation—appear to drive superior long-term results.[14]

Comparative Effectiveness: PRP versus Standard Therapies

PRP versus Hyaluronic Acid (HA):Meta-analyses consistently demonstrate superior pain relief and functional improvement with PRP compared to HA injections.[11,19,22] At 3, 6, and 12-month follow-ups, PRP showed better WOMAC and VAS scores than HA, with improvements exceeding MCID thresholds.[11,19] Combined PRP+HA

therapy yielded additional benefits, particularly in longer-term follow-ups (12+ months), with lower adverse event rates compared to PRP alone.[18,19]

PRP versus Corticosteroids (CS): PRP demonstrated comparable short-term results to corticosteroids but provided superior outcomes at mid- and long-term follow-ups (6–12 months).[19,22] Benefits persisted and exceeded MCID at 12 months with PRP, whereas CS effects typically wane by 6 months.[19]

PRP versus Placebo: While the placebo effect is substantial in orthobiologic trials (saline injections provided meaningful improvements in some studies), PRP benefits were both statistically and clinically superior to placebo at all follow-up points, confirming genuine therapeutic benefit beyond placebo response.[11,22]

Leukocyte Content: Effects on Safety and Efficacy

The presence or absence of leukocytes in PRP has been extensively debated as a critical determinant of efficacy and safety.[3,8,12]

Potential benefits of leukocyte-rich PRP (L-PRP):
- Enhanced immune modulation and inflammation control through cytokine and antimicrobial factor release
- Potential anti-infective properties when applied to contaminated wounds
- In vitro evidence of enhanced macrophage polarization and anti-inflammatory signaling

Concerns with leukocyte-rich PRP:
- Increased post-injection pain and swelling reactions (significantly higher in L-PRP than P-PRP in multiple studies)
- Potential for elevated proinflammatory cytokine activity early post-injection, leading to temporary symptom worsening
- No demonstrated clinical superiority in long-term pain relief or functional outcomes compared to leukocyte-poor PRP, despite theoretical advantages

A recent high-level RCT found no significant differences in clinical outcomes between leukocyte-rich and leukocyte-poor PRP, suggesting that the effects of leukocytes shown in vitro may not translate into clinically meaningful differences in intra-articular injections.[12] However, multiple studies document significantly higher rates of post-injection pain and swelling with L-PRP (9.8% versus 1.4% for P-PRP), which may limit patient tolerability and require extended post-injection care instructions.[8,12]

Current recommendations favor leukocyte-poor, high-platelet PRP for initial treatment, given superior durability, comparable efficacy, and fewer adverse effects, though patient-specific factors may warrant L-PRP selection in certain contexts.[3,8]

Special Considerations: Hip Osteoarthritis and Extra-Articular Applications

While knee OA represents the most extensively studied indication, emerging evidence supports PRP efficacy in hip OA.[23] A 2024 systematic review of hip OA found PRP injections to be safe and effective, with favorable outcomes compared to hyaluronic acid and superior long-term symptom relief.[23] Adverse events were minimal and transient, with post-injection warmth, stiffness, and mild pain resolving without intervention.[23]

PRP is also being investigated for application to cartilage lesions, tendinopathies, ligament injuries, and muscle damage, though these applications remain outside the scope of this review.

DISCUSSION

Integration of Evidence and Implications for Clinical Practice

The evidence base supporting PRP for osteoarthritis has substantially matured over the past 5 years, with multiple high-quality meta-analyses, prospective cohort studies, and RCTs demonstrating clinically meaningful benefits. Several key insights emerge:

1. Platelet concentration is a critical determinant of efficacy. The widely cited observation that higher-platelet PRP yields superior outcomes has been corroborated in recent meta-analytical work.[11,14] This finding challenges prior approaches that focused on leukocyte content as the primary differentiator. Clinicians should prioritize PRP preparations achieving ≥1,000,000 platelets/μL and administer a minimum of 5 billion total platelets per injection to optimize efficacy.[11,12,14]

2. Multiple-injection protocols are superior to single-injection approaches. Three injections spaced 3–4 weeks apart yield more sustained pain relief and functional improvement than single-injection protocols,

supporting adoption of multi-injection treatment paradigms in clinical practice.[14,21]

3. PRP is appropriate as a mid-stage therapy.Current evidence-based guidelines recommend PRP for patients with early-to-moderate OA (Kellgren-Lawrence Grades 0–III) after failure of conservative non-injective or injective treatments.[19] PRP is not recommended as first-line therapy (that role remains with lifestyle modification, physical therapy, and simple analgesics) or for end-stage disease (Grade IV) with severe cartilage loss, though emerging data suggest benefit even in severe cases as a bridge to joint replacement.[20]

4. Combination therapy (PRP + HA) may enhance long-term outcomes. Meta-analyses indicate that combining PRP with hyaluronic acid yields additional functional and pain benefits, particularly in 12-month and beyond follow-ups, while reducing adverse event rates compared to PRP alone.[18,19] The optimal sequence and timing of combination therapy require further study.

5. Safety and tolerability are favorable.Adverse events are typically minor (transient post-injection pain, warmth, swelling) and resolve within 48 hours without intervention.[3,8,12,23] No serious adverse events (infections, systemic reactions) have been reported in high-quality OA studies, supporting the safety profile of PRP as an autologous biologic product. However, L-PRP preparations incur higher post-injection reaction rates, supporting preference for P-PRP in initial treatment.[8,12]

6. Standardization and quality control remain priorities. Substantial variability in PRP preparation protocols, platelet concentration measurement, anticoagulant selection, and activation methods persists across clinical and research settings.[3,7] Adoption of standardized protocols—including documentation of platelet count, leukocyte concentration, growth factor assessment where feasible, and sterility testing—is essential to ensure reproducible, safe, and effective treatment.[7]

Mechanisms Explaining Clinical Benefits

The clinical efficacy of PRP in OA likely reflects integration of multiple mechanisms operating at distinct tissue levels and timeframes. In the early post-injection period (first 1–2 weeks), PRP activates resident joint cells, modulates immune responses, and initiates anti-inflammatory signaling through cytokine-mediated pathways and macrophage polarization.[3,6,16] Mid-term effects (2–8 weeks) include growth factor-driven chondrocyte proliferation, increased cartilage matrix synthesis, synovitis reduction, and enhanced angiogenesis.[3,6,17] Longer-term effects (8–12+ weeks) reflect tissue remodeling, sustained anti-inflammatory tone, and potentially disease-modifying cartilage preservation.[3,20]

This multiphase mechanism accounts for the superior durability of high-platelet PRP compared to corticosteroid injections, which provide rapid but transient anti-inflammatory effects without sustained regenerative signaling.[11,19]

Unresolved Questions and Knowledge Gaps

Despite substantial progress, several questions merit further investigation:

1. Optimal patient selection criteria: While age <80 years, early-to-moderate OA, and prior treatment failure have emerged as relative indications, prospective studies defining precise phenotypes most likely to benefit (e.g., based on inflammatory biomarkers, imaging features, or genetic polymorphisms) are lacking.
2. Long-term disease modification: Existing studies demonstrate sustained symptom relief but provide limited evidence of true structural disease modification (cartilage regrowth, bone marrow lesion resolution) on imaging. Longer-term prospective studies with advanced imaging (high-resolution MRI, quantitative cartilage assessment) are needed.
3. Optimal PRP composition and preparation protocols: While high-platelet concentration appears beneficial, the ideal leukocyte concentration, fibrin content, growth factor profile, and activation status remain incompletely defined. Standardized protocols enabling cross-study comparison and clinical translation are urgently needed.
4. Combination strategies: The optimal sequencing, timing, and dosing of PRP + HA or PRP + other biologics (e.g., stem cells, bone marrow aspirate concentrate) require evaluation in rigorous RCTs.
5. Cost-effectiveness and health economic impact: While PRP cost-effectiveness has been suggested, formal cost-effectiveness analyses comparing PRP to joint replacement or other conservative strategies in diverse healthcare systems are limited.

Limitations

This narrative review is subject to several limitations:

1. Publication bias:Reported studies may skew toward positive results, potentially overestimating PRP efficacy. Unpublished null or negative trials may not be captured.
2. Heterogeneity in PRP preparation and outcome measures: Substantial variability across studies in centrifugation protocols, platelet concentration, leukocyte content, activation status, injection volume, and follow-up schedules complicates meta-analytical synthesis and comparison. Definitions of "high-platelet" and "low-platelet" PRP differ across studies.
3. Limited long-term follow-up: Most included studies report outcomes through 12 months; studies with 24-

month, 36-month, or longer follow-ups are sparse, limiting understanding of durable disease modification.
4. Patient selection bias: Many studies enrolled motivated, healthy-to-moderate-age patients with early-to-moderate OA, limiting generalizability to older, more severely affected, or comorbid populations.
5. Placebo effect magnitude: The substantial placebo response in orthobiologic trials (saline injections produced meaningful symptom relief in some studies) raises questions about the true additive benefit of PRP beyond placebo and natural history effects.
6. Geographic and healthcare system variation: Studies were predominantly conducted in North America and Europe; evidence from other regions and healthcare systems is limited, affecting generalizability.

CONCLUSION

Platelet-rich plasma represents an evidence-based, safe, and effective biologic treatment option for symptomatic knee osteoarthritis and emerging evidence supports efficacy in hip OA. High-platelet-concentration PRP (≥1,000,000 platelets/μL) administered via multiple intra-articular injections yields clinically significant and durable improvements in pain and function, exceeding benefits of placebo, hyaluronic acid, and corticosteroid injections at mid- and long-term follow-ups.

The biological mechanisms underlying PRP efficacy are multifactorial, involving anti-inflammatory signaling, cartilage-regenerative growth factor activity, synovitis reduction, and immune modulation through macrophage polarization. Leukocyte-poor PRP formulations demonstrate comparable clinical efficacy to leukocyte-rich preparations while incurring fewer post-injection adverse reactions, supporting their preferential use in initial treatment.

Current evidence-based recommendations position PRP as an appropriate option for patients with early-to-moderate OA (Kellgren-Lawrence Grades 0–III) after failed conservative non-injective or injectable therapies and in those seeking to delay joint replacement. Standardization of PRP preparation protocols, prospective evaluation of optimal patient phenotypes, longer-term disease-modification studies, and economic analyses remain priorities for broader clinical implementation and regulatory recognition.

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