

Ultrasound-Guided PRP for Tendinopathy: a Practical, Multimodal Framework from Biology to Rehabilitation

Ultrazvukem navigovaná PRP terapie tendinopatií: praktický, multimodální přehled od biologie po rehabilitaci

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SUMMARY

Tendinopathies are common and heterogeneous, and recovery depends on both the local biological milieu and appropriately progressed mechanical

loading. Platelet-rich plasma (PRP) is a biologically plausible adjunct, yet reported clinical effects are inconsistent, likely due to variation in phenotype, PRP formulation and dosing, delivery technique, and rehabilitation protocols. Ultrasound guidance improves injection accuracy and safety, but its incremental clinical value may be context dependent. This review outlines

tendon-healing biology, summarizes evidence for conservative management, and appraises PRP – its putative mechanisms, sources of heterogeneity, and its integration into individualized, multimodal care.

Key words: platelet-rich plasma, tendinitis, tendinosis, tendon healing, tendinopathy, ultrasound guidance.

INTRODUCTION

Tendinopathy encompasses a spectrum of common, pain-predominant tendon disorders that impair function and limit daily, occupational, and sporting activities. (36). Its pathophysiology is heterogeneous and shaped by interacting mechanical and systemic influences, resulting in variable clinical trajectories and treatment responses (2). Accordingly, contemporary management relies on individualized strategies centred on progressive loading and rehabilitation, with selected adjunctive modalities used when symptoms persist or limit effective training progression. (3, 37, 43).

Among biologic options, platelet-rich plasma (PRP) is widely used as an adjunct to modulate the local microenvironment in chronic degenerative conditions. Ultrasonographic (USG) guidance may enhance procedural accuracy and safety by enabling lesion-targeted delivery and reducing technique-related variability, yet clinical outcomes remain inconsistent across studies (20). This review summarizes conservative management principles and critically appraises the biological

rationale, clinical evidence, and practical implementation of USG-guided PRP within multimodal, criterion-based innovative rehabilitation pathways for tendinopathy.

Biology of tendinopathy

Tendinopathy is an umbrella term for painful tendon disorders that reflect a maladaptive response to repetitive mechanical loading, accumulated microdamage, and systemic metabolic stressors. When loading repeatedly exceeds the tendon's capacity for recovery, homeostasis becomes unstable and repair shifts from efficient remodelling toward a state of failed or exhausted healing. (11, 36). Histologically, chronic tendinopathy is characterized by collagen disorganization with increased type III collagen, altered extracellular matrix turnover, hypercellularity, and neovascularization – features that align more with degeneration and dysregulated remodelling rather than with persistent acute inflammation (1, 48).

At the cellular level, tenocyte dysfunction is central. Altered mechanotransduction, mitochondrial dysfunction,

metabolic exhaustion, and senescence-like changes impair resident cells' ability to maintain extracellular matrix architecture. Dysregulated matrix remodelling, driven by an imbalance in matrix metalloproteinases and their inhibitors, further accelerates collagen breakdown and perpetuates structural disorganization. These processes are amplified by ageing and common comorbidities such as diabetes, dyslipidaemia, and obesity, which contribute to low-grade inflammation, oxidative stress, and impaired collagen turnover (28, 54, 55).

As degeneration progresses, aberrant angiogenesis and sensory nerve ingrowth – typically absent in healthy tendon – emerge and correlate with pain and progressive matrix disruption. (53). Mechanobiological signalling pathways, particularly along integrin–Focal adhesion kinase (FAK) and Yes-associated protein (YAP) and transcriptional coactivator with *Post* synaptic density protein (PSD95), *Drosophila* disc large tumor suppressor (Dlg1), and *Zonula* occludens-1 protein (zo-1) (PDZ)-binding motif (YAP/TAZ) axes, translate repetitive tensile strain into maladaptive transcriptional programs that drive the transition from reactive change to disrepair and structural degeneration. (26, 31, 49). Collectively, tendinopathy reflects failed tissue adaptation across mechanical, metabolic, immune, and neurobiological dimensions, resulting in a biologically depleted microenvironment with limited intrinsic regenerative capacity – thereby providing a rationale for biologic adjuncts such as PRP.

CONSERVATIVE MANAGEMENT AND MULTIMODAL CARE

Conservative treatment is the cornerstone of tendinopathy management (Fig. 1) (37, 43). Its primary goals are to reduce pain, restore function, and progressively rebuild tendon load tolerance while addressing contributory biomechanical and contextual factors. Across tendon sites, structured exercise therapy – particularly progressive loading programs incorporating isometric, eccentric exercise – remains the most consistently supported first-line intervention (12, 46). Load modification and staged return to activity are essential for controlling tendon irritability and enabling meaningful training adaptation. Where appropriate, biomechanical optimisation (e.g., addressing kinetic-chain imbalances, restricting the range of movement of the body segment, and optimal postural-pattern retraining) and patient education support symptom control and adherence to therapy (8, 14).

Adjunctive modalities may be considered in selected patients, typically when pain limits tendon loading progression or when symptoms persist despite individual tailoring of the loading protocol. Extracorporeal shockwave therapy and other physical modalities may provide additional benefit in specific scenarios, but their effects are generally modest and context-dependent (16, 32, 44). Importantly, clinical outcomes are rarely driven by a single intervention; rather, they

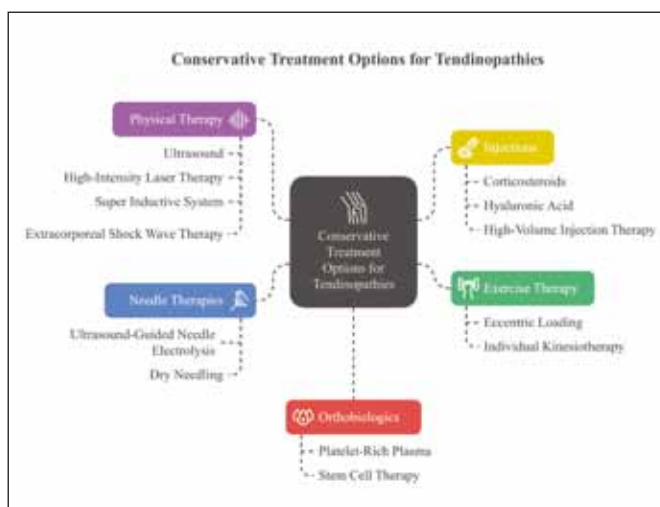


Fig. 1. Overview of conservative management in tendinopathy, including progressive exercise-based rehabilitation, adjunct physical modalities, and orthobiologic interventions. The figure highlights a multimodal framework in which treatment components are combined and tailored to the patient's phenotype, symptom irritability, and disease stage.

reflect the alignment of biological state, mechanical loading, and patient-specific factors (35, 37).

Within this multimodal framework, biologic and regenerative injectables – most notably PRP – are increasingly used as adjuncts for chronic, degenerative tendinopathies with features of failed healing in physical medicine and rehabilitation (PM&R) settings (20). Their aim is to modulate the local microenvironment to support matrix remodelling and enhance tenocyte activity, while individualised kinesiotherapy provides the mechanical stimulus required for functional recovery. Clinical experience and emerging evidence suggest that PRP is more likely to be beneficial when integrated into a structured, criterion-based rehabilitation program than when delivered as a stand-alone procedure, further supporting its use in the PM&R setting (13, 33). This integration requires careful timing, individualized kinesiotherapy with optimal load progression, and a clear definition of treatment targets, supported by USG imaging (40–42), to enable lesion-focused delivery and reduce procedural variability.

Current evidence for PRP in tendinopathy

PRP is an autologous blood product that delivers a concentrated pool of platelets and bioactive mediators to a region of failed tendon healing. Growth factors such as Platelet-derived growth factor (PDGF), Transforming growth factor-beta (TGF- β), and Vascular endothelial growth factor (VEGF) can support tenocyte proliferation, collagen synthesis, and aspects of vascular and matrix remodelling. PRP may also modulate the local inflammatory milieu by influencing macrophage

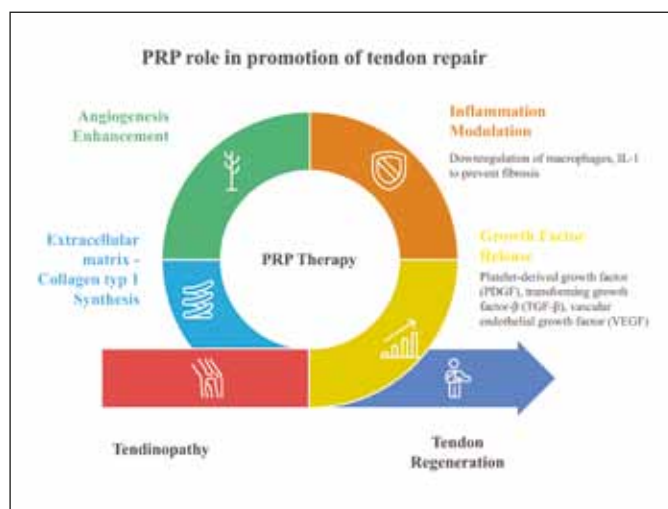


Fig. 2. Proposed mechanisms by which platelet-rich plasma (PRP) may support tendon repair. PRP, prepared from autologous blood, concentrates platelets and associated mediators (e.g., platelet-derived growth factor (PDGF), transforming growth factor-beta (TGF-β), Vascular endothelial growth factor (VEGF)) that can influence tenocyte activity, extracellular matrix remodelling, and angiogenic responses. PRP may also modulate the local inflammatory milieu, including macrophage-related pathways and pro-inflammatory cytokine signalling.

polarization and reducing pro-inflammatory signalling (e.g., Interleukin-1 (IL-1)-related pathways), thereby limiting maladaptive fibrosis (Fig. 2) (52, 56).

In vitro studies are broadly consistent with these mechanisms, reporting upregulation of genes linked to collagen type I production and extracellular matrix organization in PRP-stimulated human tenocytes (27).

Preclinical data further support biological activity, with models of Achilles and patellar tendon injury showing improvements in structural healing, angiogenesis, and collagen deposition following PRP exposure (9, 27). Collectively, mechanistic and preclinical findings suggest that PRP can promote a pro-regenerative microenvironment that supports tenocyte activity and matrix reorganization and may interact with tendon-derived stem/progenitor cell responses. USG guidance can enhance procedural accuracy by targeting the pathologic tendon/enthesis region, improving intralesional delivery, and reducing off-target infiltration (20, 40–42).

Clinical evidence across tendinopathy sites is substantial but heterogeneous. Effect sizes vary and are sensitive to the comparator and the quality of rehabilitation. Randomized trials and meta-analyses report small-to-moderate improvements in pain and function in selected chronic degenerative presentations, while other studies show limited or no added benefit over high-quality rehabilitation alone – differences likely driven by variation in patient phenotype and chronicity, PRP formulation (including leukocyte and platelet content), dose and injection strategy, comparator, and rehabilitation

protocols. Where benefit is observed, it is most often reported in imaging-confirmed degenerative lesions and in protocols emphasizing lesion-targeted delivery and structured post-injection loading (4, 7, 18–20, 29).

Across sites, trials of lateral epicondylitis commonly show better long-term outcomes with PRP than corticosteroid injection, which tends to provide short-term relief but inferior durability (22, 39, 45). In patellar tendinopathy, studies using leukocyte-poor preparations suggest an advantage over dry needling in pain/function scores (including Victorian Institute of Sport Assessment Patella Questionnaire (VISA-P)) and time to return to sport in some cohorts (15). For Achilles and gluteal tendinopathies, pooled analyses suggest a potential benefit in chronic or refractory cases, although effect estimates vary across protocols and study quality (18, 19, 29, 38).

Interestingly, PRP also performs better in the treatment of osteoarthritis, for example, compared to the use of hyaluronic acid (5, 51). Hyaluronic acid products for intra-articular and intra-tendinous administration differ, so this conclusion cannot automatically be applied to a comparison of PRP with the latest hyaluronic acid products in the treatment of tendinopathies (21).

Overall, PRP is best framed as a bioregenerative adjunct for rehabilitation rather than a stand-alone solution. Its clinical value appears most plausible when (I) the lesion phenotype is consistent with tendinosis – chronic degenerative failed healing, (II) delivery is lesion-targeted via USG guidance, and (III) PRP is integrated within a criterion-based, progressive loading physiotherapeutic program.

Ultrasound-guided PRP injection: rationale, technique, and standardization

USG guidance is a logical extension of lesion-focused treatment in tendinopathy. Because PRP is intended to act within a biologically depleted, structurally compromised region of the tendon or enthesis, accurate targeting is critical. Real-time USG enables identification of intratendinous, enthesal, or peritendinous pathology and supports controlled placement of the injectate within the intended tissue plane (Fig. 3).

USG guidance also improves procedural safety by visualizing needle trajectory, avoiding neurovascular structures, and monitoring injectate dispersion.

In practice, USG-guided PRP offers pathology-specific targeting with structural and vascular assessment (including microvascularization non-Doppler imaging), which optimizes biologic exposure of the degenerative segment to promote rehabilitation. (34). Paired with a favourable safety profile and overall low complication rates when performed with appropriate technique by an experienced USG-interventionist and optimal post-procedural management.

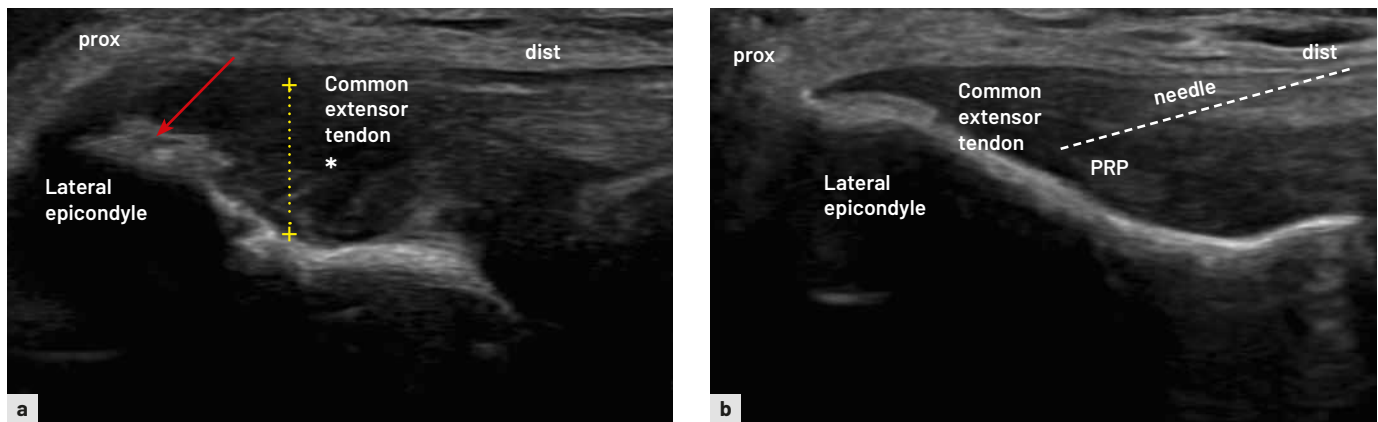


Fig. 3. Ultrasound features and ultrasound-guided injection in lateral epicondylitis. (a) Long-axis view showing tendon thickening (yellow dashed line), loss of the normal fibrillar pattern, and intratendinous hypoechoic regions (asterisk). An enthesophyte (red arrow) is visible, consistent with chronic changes. (b) Ultrasound-guided needle placement for lesion-targeted injection; injectate distribution is visualized in real time. Percutaneous needle fenestration/tenotomy can be performed under ultrasound guidance before or during PRP delivery.

Key technical decisions

In clinical practice, the first consideration in the treatment of tendinopathy is the evaluation of chronic changes in the tendon, and the decision whether to perform fenestration of the degenerative segment. US-guided fenestration with simultaneous PRP application provides microstimulation that may significantly enhance the healing response and facilitate intratendinous dispersion of PRP (17). It is most appropriate for focal, well-delineated, chronic degenerative defects that have not responded to conservative management with a tailored rehabilitation protocol. However, structural integrity must be assessed, and the risk of partial/complete tearing considered, especially when using USG imaging with dynamic assessment, as there is an increased risk of tendon structural failure with USG-guided needle fenestration. The use of USG-guidance is mandatory to precisely target the fenestration and PRP dispersion, since blind, anatomically guided tendon “peppering” should be avoided (6).

A second decision is the choice between leukocyte-poor (LP-PRP) and leukocyte-rich (LR-PRP) formulations. LP-PRP is generally favoured for chronic intratendinous degenerative lesions and intraarticular applications, where minimizing excessive inflammatory amplification may reduce post-injection irritability and support an anabolic remodelling response. In contrast, LR-PRP induces a stronger inflammatory response and may be considered in carefully selected insertional or peritendinous presentations with a substantial inflammatory component, in which a transient inflammatory stimulus is believed to contribute to remodelling. In practice, formulation choice should be phenotype-driven by lesion type, clinical presentation, and USG findings (30).

Physician variability and the need for standardization

Despite the intrinsic advantages of USG guidance, clinical outcomes can be influenced by operator-dependent variability. Differences in transducer handling, needle-tip visualization, interpretation of subtle structural changes, and lesion-targeting precision can materially affect the site of injectate deposition, thus reducing its therapeutic potential. Standardization can reduce this variability: consistent pre-injection assessment, explicit definition of the target, a standardized needle approach, and documentation of injectate distribution form the basis of a clinically relevant, reproducible procedure. Competency-based training and protocolized workflows are therefore essential to improve external validity across studies and ensure safe, accurate delivery in routine practice (10, 47).

Post-procedural management and rehabilitation after PRP

Post-procedural care is integral to PRP effectiveness, as the biologic stimulus requires a structured rehabilitation program aligned with tendon healing and load-tolerance kinetics (50). A short period of relative unloading (typically 48–72 hours) is usually sufficient to control post-injection discomfort while allowing early biological activity to proceed (23). Prolonged immobilization is discouraged because it may impair collagen organization and delay return to functional loading (50).

Rehabilitation is commonly initiated 7–14 days after the procedure, starting with range-of-motion work and low-load isotonic exercises to restore neuromuscular control without excessive tendon strain. As symptoms permit, loading is progressed in a criterion-based manner, typically introducing structured isometric exercises, progressively isolating the

Table 1. Minimal reporting checklist for ultrasound-guided PRP in tendinopathy to improve reproducibility and interpretability across studies and clinical reports

1.	Lesion localization (central/peripheral, extension of the lesion), phenotype (reactive/degenerative changes of the tendon based on echogenicity and microvascularization of the tendon), and targeting (needle trajectory selection to avoid neurovascular structures)
2.	US-guided intervention strategy (single-point/needle fenestration with PRP application, volume planning intratendinously/peritendinously)
3.	PRP specifics (Leukocyte-rich vs. leukocyte-poor PRP, single vs serial injections, volume of blood drawn, preparation – number of centrifugation cycles, duration of cycles, relative centrifugal force, post-process analysis – automatic cell counters, activation of platelets)
4.	US-guided intervention technique (approach in-plane/out-plane, report on a spread of the injectate)
5.	Post-intervention rehabilitation (Nonsteroidal anti-inflammatory drugs [NSAID] withdrawal, resting period, need for immobilization, optimization of timing and key elements [isokinetic, isometric and eccentric contraction] of criterion-based loading)
6.	Documentation/safety (image documentation, targeting and injection spreading, adverse events, post-intervention imaging).

affected muscle group, and tailoring the program to the involved tendon and functional demands (14, 50).

By 3–6 weeks, patients progress to eccentric loading, incorporating the gradual integration of kinetic-chain retraining and movement-pattern optimization to restore tendon load capacity during complex movements rather than isolated strength of the affected muscle group. Return to plyometric and sport-specific activities is generally considered to occur between 6 and 12 weeks, depending on the tendon site, baseline degeneration, and clinical response to physiotherapy (24).

Across studies, the most consistent outcomes are reported when PRP is embedded in a supervised, criterion-based rehabilitation program rather than in unsupervised home exercise. Physiotherapists are therefore key to monitoring symptom trajectories, adjusting load progression, and addressing contributory factors such as flexibility deficits, impaired postural control, and altered limb-loading strategies.

FUTURE DEVELOPMENTS

Future progress in the care of tendinopathy will likely be driven by four converging directions.

First, refined phenotyping. The field is moving beyond broad anatomical labels toward mechanism-informed phenotypes defined by load response/irritability, matrix organization, vascularity, and inflammatory-metabolic features. More granular classification should improve patient stratification and help identify who is most likely to benefit from biologic adjuncts, such as PRP (optimal treatment candidacy), and when to apply them in the disease course.

Second, personalization of multimodal care. Outcomes depend on aligning biologic intervention, mechanical loading, and integration to complex movement patterns to the individual tissue state. Criterion-based, adaptive rehabilitation – potentially supported by objective monitoring of symptoms, load exposure, and adherence – may reduce variability and improve durability of response.

Third, the development of complex regenerative biologic systems. Progress will require standardized, purpose-tailored

biologics, including well-defined PRP preparation frameworks (platelet count, leukocyte profile, activation, and optimal delivery), as well as next-generation combinations that integrate bioactive fractions with scaffolding or cell-mediated mechanisms (25). These “profiled” regenerative systems aim to match the biologic composition to the lesion phenotype rather than to apply uniform formulations across heterogeneous tendinopathies.

Fourth, advances in USG and imaging biomarkers. Quantitative ultrasonography, non-Doppler visualization of microvascularization, elastography, and improved needle-visualization workflows are poised to enhance lesion characterization, procedural reproducibility, and response monitoring. As the USG becomes more quantitative and operator-independent, it may function not only as a guidance tool but also as a practical biomarker platform to support stratified, image-informed intervention pathways. In parallel, more rigorous study design and standardized reporting will strengthen the evidence base for US-guided PRP. To facilitate comparability across trials and accelerate evidence synthesis, we propose a minimal reporting set for US-guided PRP (Table 1).

CONCLUSIONS

Tendinopathies are heterogeneous disorders in which mechanical overload interacts with cellular dysfunction, metabolic factors, and neurogenic mechanisms to drive chronic failed healing. This complexity supports a phenotype-oriented, individualized approach rather than a uniform rehabilitation strategy. Exercise-based rehabilitation remains the foundation of care. In chronic degenerative lesions with limited intrinsic repair, PRP is a biologically plausible adjunct, most likely effective when delivered precisely under USG and integrated into a structured, progressive loading program. Key uncertainties remain regarding optimal formulation, timing, and patient selection. With optimal protocols for preparation, delivery, and post-procedural kinesiotherapy, PRP presents as a valuable therapeutic tool in the PM&R setting. ■

Abbreviations

PRP	Platelet-rich plasma
USG	Ultrasonography
FAK	Focal adhesion kinase
YAP	Yes-associated protein
PSD95	Post synaptic density protein
Dlg1	Drosophila disc large tumor suppressor
Zo-1	Zonula occludens-1 protein
PDZ	Post synaptic density protein, Drosophila disc large tumor suppressor, Zonula occludens-1 protein
YAP/TAZ	Yes-associated protein (YAP) and transcriptional coactivator with PDZ-binding motif
PM&R	Physical medicine and rehabilitation
PDGF	Platelet-derived growth factor
TGF- β	Transforming growth factor-beta
VEGF	Vascular endothelial growth factor
IL-1	Interleukin-1
VISA-P	Victorian Institute of Sport Assessment Patella Questionnaire
LP-PRP	Leukocyte-poor platelet-rich plasma
LR-PRP	Leukocyte-rich platelet-rich plasma
NSAID	Nonsteroidal anti-inflammatory drugs

Statement on the use of AI tools

The authors confirm that all scientific ideas, interpretation of evidence, conceptual frameworks, and conclusions in this manuscript were developed entirely by the authors. ChatGPT (OpenAI) was used only for minor language refinement and reduction of redundancies in text already drafted by the authors. No part of the scientific analysis, argumentation, or conclusions was generated or altered by AI. All content was reviewed and approved by the authors, who take full responsibility for the manuscript.

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