RESEARCH PAPER

Platelet-Rich Fibrin as a Biological Augment in Meniscal Repair: Current Evidence and Future Directions

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ABSTRACT

Meniscal preservation has replaced meniscectomy as the preferred treatment for many meniscal tears, but repair still fails in roughly one in four isolated cases, largely because the inner meniscus has little blood supply and few reparative cells. Biological augmentation aims to improve this healing environment. Platelet-rich fibrin (PRF) is a second-generation autologous platelet concentrate prepared from the patient's own blood without anticoagulants or activators. It forms a dense fibrin matrix that traps platelets, leukocytes and growth factors and releases them gradually over about a week, while also acting as a scaffold for cell migration.

This narrative review summarises the biology of meniscal healing, the classification and preparation of PRF, its proposed mechanisms of action, and the arthroscopic techniques used to apply it. It then examines the clinical evidence on functional and structural outcomes after PRF-augmented meniscal surgery, set against the larger body of evidence for platelet-rich plasma (PRP). Laboratory studies show that PRF stimulates meniscal cell migration, proliferation and matrix synthesis. Clinical studies report clear improvements in Lysholm, IKDC and related scores after PRF-augmented repair, but they are small, mostly non-randomised, use varied preparation protocols, and have not consistently shown superiority over repair alone.

PRF is safe, simple and inexpensive, which makes it attractive in resource-limited settings. Adequately powered randomised controlled trials with standardised preparation and long-term follow-up are needed before routine use can be recommended.

Keywords: meniscus; meniscal repair; platelet-rich fibrin; orthobiologics; arthroscopy; knee

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1. Introduction

The menisci are semilunar fibrocartilaginous structures that transmit load across the tibiofemoral joint, absorb shock, contribute to stability, and aid joint lubrication and proprioception [1,2]. Meniscal tears are among the most common knee injuries in

both athletes and the general population, and they are a frequent indication for arthroscopic surgery [2]. Because the meniscus is so important to joint mechanics, its loss has lasting consequences. In a long-term cohort, nearly three out of four patients had developed radiographic tibiofemoral osteoarthritis 20 to 30 years after meniscectomy [3]. These observations have shifted practice towards meniscal preservation. A systematic review by Paxton et al. showed that although meniscal repair carries a higher reoperation rate than partial meniscectomy, it is associated with better long-term function and less radiological degeneration [4]. Advances in suture devices and repair techniques have since widened the range of tears considered repairable, including radial, horizontal and complex patterns [5,6].

Healing, however, remains the weak link. Meta-analyses report an overall failure rate of around 25% after isolated meniscal repair, with failure concentrated in tears that extend into the poorly vascularised inner zones [7,8]. This has driven interest in biological augmentation, meaning strategies that improve the local healing environment at the repair site.

Platelet-rich fibrin (PRF) is one such strategy. First described by Choukroun and colleagues for oral and maxillofacial surgery, PRF is a second-generation platelet concentrate obtained by centrifuging whole blood without anticoagulant or bovine thrombin [9]. The result is a solid, autologous fibrin clot rich in platelets, leukocytes and growth factors, which can be handled, sutured and placed directly into a meniscal tear.

The aim of this review is to summarise the biological rationale for PRF in meniscal repair, describe its preparation and arthroscopic application, critically appraise the current clinical evidence on functional and structural outcomes, and identify the gaps that future research must address.

2. Meniscal Anatomy, Vascularity and Healing Biology

The meniscus is composed mainly of water and an extracellular matrix dominated by type I collagen, with smaller amounts of proteoglycans and other proteins [1]. Collagen bundles in the deeper layers run circumferentially, converting axial load into hoop stresses, while radial fibres tie these bundles together and resist longitudinal splitting [1,2]. The cell population is heterogeneous. Cells in the outer region are fibroblast-like, whereas those in the inner region are rounder and more chondrocyte-like, reflecting the greater compressive load the inner meniscus bears [2].

Vascularity is the key determinant of healing. In their classic cadaveric study, Arnoczky and Warren showed that a perimeniscal capillary plexus arising from the genicular arteries penetrates only the peripheral 10% to 25% of the meniscus [10]. This finding underlies the zonal description used in clinical practice: the vascular red-red zone at the periphery, the transitional red-white zone, and the avascular white-white zone of the inner third.

Meniscal healing follows the general sequence of inflammation, proliferation and remodelling. In the vascular zone, a haematoma and fibrin clot form at the tear, delivering platelets, inflammatory cells and growth factors that recruit reparative cells and support the formation of fibrovascular scar, which gradually remodels into fibrocartilage-like tissue [1]. In the avascular zone this sequence stalls. There is no bleeding to form a fibrin scaffold, resident cell density is low, the dense matrix restricts cell migration, and the joint environment after injury is rich in catabolic cytokines [11,12]. Interleukin-1 and related mediators promote matrix breakdown and inhibit repair [12], and angiogenesis within the inner zone is now viewed as a critical, and largely missing, step in healing [13].

These deficits define what a biological augment should supply: a provisional matrix that bridges the tear, signals that recruit and stimulate reparative cells, and modulation of the hostile intra-articular environment [11]. PRF is attractive because it is designed to provide all three.

3. Evolution of Meniscal Management

The management of meniscal tears has moved from open total meniscectomy, through arthroscopic partial meniscectomy, to meniscal repair whenever the tear pattern and tissue quality allow [5,6]. Longitudinal vertical tears in the peripheral third remain the classic indication for repair, but improved instruments now allow repair of radial, horizontal, root and complex tears [5]. The inside-out technique is still regarded as the reference standard for many body tears because it permits multiple, perpendicular sutures, while modern all-inside devices avoid additional incisions and the need for an assistant to retrieve needles [14].

The benefits of repair come at a cost. In Paxton and colleagues' systematic review, reoperation rates after isolated repair were 16.5% at short-term and 20.7% at long-term follow-up, compared with 1.4% and 3.9% after partial meniscectomy [4]. Patients may also recover more slowly. In a Danish cohort of young adults, those undergoing repair improved less in patient-reported outcomes at 1 year and 5 years than those undergoing partial meniscectomy [15]. These findings make the case for strategies that increase the reliability of healing after repair.

Several factors influence the success of repair: tear location and vascularity, tear pattern, chronicity, patient age, and the presence of ligament instability [6]. Concomitant anterior cruciate ligament (ACL) reconstruction is particularly important. Repairs performed alongside ACL reconstruction consistently fail less often than isolated repairs [4]. In a cohort of 550 patients, Everhart et al. reported 3-year failure rates of 20.3% for isolated repairs and 14.1% for repairs with ACL reconstruction [16]. The

usual explanation is that tunnel drilling releases marrow elements, including blood, growth factors and progenitor cells, into the joint, creating a favourable biological environment around the repair [14]. Biological augmentation of isolated repair is, in essence, an attempt to recreate that environment.

4. Biological Augmentation Strategies: A Brief Overview

A range of techniques has been developed to stimulate healing at the repair site [12,17]. Mechanical methods such as rasping of the parameniscal synovium and trephination of the meniscal rim aim to create vascular access channels and provoke a local healing response. The exogenous fibrin clot, introduced experimentally by Arnoczky et al., was shown in a canine model to act as a chemotactic and mitogenic stimulus and as a scaffold for reparative cells in the avascular zone [18]. It remains in use because it is cheap and easy to prepare from the patient's own blood [19].

Marrow venting procedures, in which the intercondylar notch is perforated to release marrow contents, attempt to reproduce the effect of ACL tunnel drilling. In a double-blind randomised trial, Kaminski et al. found a meniscal healing rate of 100% with marrow venting compared with 76% with repair alone [20]. Platelet-rich plasma (PRP) is the most studied blood-derived biologic. The same group reported significantly higher healing with PRP injected at the repair site in a randomised trial of bucket-handle tears [21]. More advanced options include bone marrow aspirate concentrate, mesenchymal stromal cells, meniscal wrapping and scaffolds [12,17].

In practice, surgeons favour simple methods. In an international survey of expert knee surgeons, 90% used at least one augmentation technique for isolated meniscal repair, but rasping, trephination and marrow venting were far more common than PRP, which was used by only 2% to 19% depending on the scenario [22]. A recent Delphi consensus likewise supported low-cost techniques for higher-risk tear patterns and judged the evidence for routine use of autologous blood products to be inconclusive [23].

PRF occupies a middle ground. Like the fibrin clot, it is autologous, cheap and solid enough to be placed into a tear. Like PRP, it concentrates platelets and growth factors. Whether this combination translates into a clinical advantage is the central question of this review.

5. Platelet-Rich Fibrin: Concept, Preparation and Types

Platelet concentrates developed from fibrin glues in the 1970s and 1980s, followed by first-generation PRP, which requires anticoagulant during collection and is usually activated with thrombin or calcium chloride before use. PRF was introduced in the early 2000s as a second-generation concentrate that avoids any biochemical handling of blood [9]. Venous blood is drawn into plain tubes without anticoagulant and centrifuged immediately. During the spin, coagulation is activated by contact with the tube wall, and fibrinogen polymerises slowly into a fibrin network in the middle layer of the tube, between the red cell base and the acellular plasma above [9]. Dohan and colleagues showed that this slow, natural polymerisation produces a flexible fibrin architecture similar to a physiological clot, in which platelet cytokines and glycan chains become trapped within the fibrin mesh [9,24]. Speed matters: because clotting begins as soon as blood contacts the tube, a delay before centrifugation produces a small, poorly formed clot.

To bring order to a confusing literature, Dohan Ehrenfest et al. proposed a classification of platelet concentrates into four families based on leukocyte content and fibrin architecture [25]. These are pure PRP (P-PRP), leukocyte- and platelet-rich plasma (L-PRP), pure platelet-rich fibrin (P-PRF), and leukocyte- and platelet-rich fibrin (L-PRF). Choukroun's original PRF belongs to the L-PRF family, while products such as Fibrinet, often described in the orthopaedic literature as platelet-rich fibrin matrix (PRFM), belong to the P-PRF family [25]. This classification was later endorsed for orthopaedic and sports medicine use [26]. The distinction is clinically relevant, because studies labelled "PRF" or "PRFM" may be using biologically different products.

Several PRF variants have since been developed by modifying centrifugation settings. Under the low-speed centrifugation concept, reducing relative centrifugal force from 710 g towards 44 g increased the number of platelets and leukocytes and the concentration of growth factors retained in the PRF matrix [27]. This principle underlies advanced PRF (A-PRF), a softer and more porous clot, and injectable PRF (i-PRF). i-PRF is prepared with a very short, low-speed spin (700 rpm, about 60 g, for 3 minutes) that yields a liquid which clots within minutes after injection [28].

Compared with PRP, PRF is simpler and cheaper. It needs only a standard tabletop centrifuge and plain tubes, with no anticoagulant, activator or commercial kit. Its physical form also differs. PRP is a liquid that tends to wash away under arthroscopic irrigation, whereas L-PRF and A-PRF form a solid clot or membrane that can be sutured into the tear. Finally, PRF releases growth factors gradually rather than in an early burst [29], a property discussed in the next section.

6. Mechanism of Action in Meniscal Healing

PRF is thought to act through three complementary mechanisms: delivery of growth factors, provision of a fibrin scaffold, and modulation of inflammation by its cellular content.

Growth factors. Activated platelets within PRF release platelet-derived growth factor (PDGF), transforming growth factor-β1 (TGF-β1), vascular

endothelial growth factor (VEGF), insulin-like growth factor-1 (IGF-1) and others [24,29]. In meniscal biology, PDGF and IGF-1 stimulate fibrochondrocyte proliferation, TGF-β1 promotes collagen and proteoglycan synthesis, and VEGF promotes angiogenesis, which is lacking in the inner zone [12,13]. A defining feature of L-PRF is the timing of release. Dohan Ehrenfest et al. showed that a PRF membrane continued to release TGF-β1, PDGF-AB, VEGF and thrombospondin-1 over 7 days, and that trapped leukocytes kept producing TGF-β1 and VEGF throughout this period [29]. In a direct comparison relevant to meniscal surgery, Nakanishi et al. found that TGF-β1 had the highest cumulative release from both fibrin clot and L-PRF over 2 weeks, followed by PDGF-AB, VEGF and basic fibroblast growth factor, with no significant difference in amount or pattern between the two preparations [30].

Fibrin scaffold. The fibrin network of PRF provides a three-dimensional matrix that fills the gap between torn surfaces and supports cell migration and attachment [9]. Arnoczky's canine experiments showed that a fibrin clot placed in an avascular meniscal defect supported proliferation of fibrous tissue that gradually modulated into fibrocartilage, whereas untreated defects remained empty [18]. PRF combines this scaffold function with a higher platelet concentration.

Cellular and anti-inflammatory effects. Leukocytes in L-PRF contribute cytokines and antimicrobial activity, and may help regulate the local immune response [29]. Modulating the post-injury inflammatory environment is now considered a legitimate target of meniscal augmentation in its own right [11].

Preclinical evidence. Wong et al. cultured rabbit meniscal cells with PRF-conditioned medium and found that PRF induced cell migration, promoted proliferation and increased extracellular matrix and cartilaginous matrix synthesis [31]. In the same study, PRF improved histological healing of meniscal defects in rabbits at 3 months [31]. Not all animal data are favourable. In a rabbit model of avascular-zone vertical tears, neither PRP nor PRFM added a clear benefit to suture repair alone [32]. Another rabbit study found that PRP increased matrix metalloproteinase-1 and -3 and promoted meniscal cell dedifferentiation, with defects filling with fibrous tissue rather than meniscal tissue [33]. These conflicting findings suggest that the composition of the platelet product, particularly its leukocyte content and release profile, may determine whether the net effect is anabolic or catabolic.

7. Surgical Techniques of PRF Application

Several arthroscopic techniques for delivering PRF to a meniscal repair have been described, mostly as technical notes.

PRF clot sutured into the tear. Narayanaswamy and Sha, working in India, described a reproducible method for harvesting PRF, forming a clot, and integrating it with an arthroscopic meniscal repair [34]. The clot is passed into the joint and positioned within the tear, and the repair sutures are tied over it so that the clot is held between the torn surfaces. Kowalski and Gallo described a similar PRF clot-augmented repair for horizontal cleavage tears, a pattern traditionally treated by resection because of its poor vascularity [35].

PRF membrane. Screpis et al. described the use of an autologous platelet-rich fibrin matrix membrane to augment meniscal repair, combined with excision of a parameniscal cyst [36]. The membrane is introduced into the tear or cyst cavity before the sutures are tightened.

Injectable delivery. Kemmochi et al. developed a device to inject PRF and PRP into the joint at the repair site during arthroscopy [37]. Liquid i-PRF can also be injected into the tear after suturing, where it clots in situ.

Root repair. Meng et al. combined a reversed suture anchor technique for medial meniscus posterior root repair with a PRF clot placed at the root attachment, an area with poor blood supply [38].

Practical considerations. Common principles from fibrin clot and PRF techniques include [19,34]:

- Preparing the tear bed by rasping or trephination before placement
- Draining the joint of irrigation fluid (dry arthroscopy) while the clot is delivered, to avoid it being washed away
- Delivering the clot through a cannula, often loaded onto a suture limb, so it can be guided into the tear
- Tying the repair sutures over the clot so that it is held mechanically in place
- Harvesting blood and starting centrifugation close to the time of use, because PRF begins to clot as soon as blood contacts the tube

8. Clinical Evidence

Clinical evidence specific to PRF in meniscal surgery is limited to a small number of cohort studies and case series, together with systematic reviews that pool PRF with other fibrin-based or platelet-based products. No adequately powered randomised controlled trial of PRF alone was identified. The key studies are summarised in Table 1.

8.1 Approach to the Literature

PubMed was searched up to October 2026 using combinations of the terms "platelet-rich fibrin", "L-PRF", "PRFM", "fibrin clot", "platelet-rich plasma", "meniscus", "meniscal repair" and "meniscectomy". Clinical studies, systematic reviews and technical notes were included, and reference lists of relevant reviews were screened. Because PRF-specific data are scarce, key PRP trials were also reviewed as indirect evidence and are presented separately.

8.2 PRF in Isolated Meniscal Repair

Kemmochi et al. performed arthroscopic meniscal repair with PRF and PRP in 17 patients, delivering the products with a purpose-built injection device, and compared them with 5 patients repaired without augmentation [37]. Lysholm and IKDC scores improved in all patients, but there was no significant difference between groups at 6 months, and follow-up MRI did not clearly show improved healing [37]. The very small control group means the study was not powered to detect a difference.

The same author later reported a 2-year prospective follow-up of 35 patients who underwent all-inside meniscal repair with PRP and PRF, of whom 31 were evaluated [39]. Lysholm, IKDC and Japanese Orthopaedic Association scores improved significantly over time, while Tegner activity levels did not change [39]. MRI showed no regeneration of the repaired meniscus, but none of the repairs had deteriorated. Notably, this study characterised its platelet products using a standardised reporting framework, with platelet and leukocyte concentrations of about 5.5 and 3.6 times baseline in the PRP component [39].

8.3 PRF in Complex and Avascular-Zone Tears

Screpis et al. retrospectively reviewed 15 patients with lateral parameniscal cysts treated by cyst excision, meniscal repair and an autologous platelet-rich fibrin matrix membrane, with a minimum follow-up of 2 years [40]. The Lysholm score improved from 41.3 to 92.3, the subjective IKDC score from 37.6 to 89.4, and pain on the numeric rating scale fell from 6.8 to 1.3, with no cyst recurrence [40]. The absence of a control group limits interpretation, as cyst excision and repair alone may produce similar results.

The most comprehensive synthesis to date is a 2026 systematic review by Skaik et al. of fibrin-based scaffold augmentation, including 15 studies and 438 patients with tears mainly in avascular zones [41]. Four fibrin-based methods were identified: fibrin clot from peripheral venous blood (252 patients), fibrin clot from bone marrow aspirate concentrate (30 patients), PRF (15 patients), and PRF combined with PRP (48 patients). With fibrin clot, 77.5% of repairs healed completely, 12.2% partially, and 10.3% failed. All fibrin-based methods were associated with improved outcome scores, and augmentation as a whole yielded better outcomes than non-augmented repair [41]. However, PRF-specific data in this review came from only 63 patients in total, and no separate pooled healing rate for PRF could be derived.

8.4 PRF with Concomitant ACL Reconstruction

No clinical study isolating the effect of PRF on meniscal repair performed with ACL reconstruction was identified. Indirect evidence comes from PRP. In Everhart et al.'s cohort of 550 patients, PRP was independently associated with a substantially lower risk of failure for isolated repairs (adjusted hazard ratio 0.18) but had no effect on failure for repairs performed with ACL reconstruction [16]. This suggests that the added value of platelet concentrates is likely to be greatest when there is no other biological stimulus in the joint, and that PRF trials should analyse isolated and ACL-associated repairs separately.

8.5 PRF after Partial Meniscectomy

No comparative clinical study of PRF after arthroscopic partial meniscectomy was identified in this search. Whether PRF can reduce postoperative pain and effusion or speed return to activity after meniscectomy remains an open question.

8.6 Functional Outcomes

Across the PRF studies, patients consistently showed large improvements from baseline in Lysholm and IKDC scores after augmented repair [37,39,40]. The critical question is whether these improvements exceed those achieved by repair alone, and here the evidence is weak. The only PRF study with a control group found no between-group difference [37]. In a meta-analysis of nine studies involving 1,164 patients, Li and Weng found that platelet augmentation lowered failure rates and improved pain and KOOS symptom scores, but did not improve IKDC or Lysholm scores [42]. In their subgroup analysis, PRP was favoured over PRFM for IKDC score, and PRFM showed no functional benefit [42]. Broader systematic reviews of biological augmentation reach similar conclusions. Keller et al. found that only 3 of 10 comparative studies showed a functional advantage over standard repair [43], and Blough et al. concluded that platelet augmentation may reduce failure without improving symptom scores [44].

8.7 Structural Healing Outcomes

Structural evidence for PRF is particularly limited. In Kemmochi's studies, MRI did not demonstrate regeneration of the repaired meniscus, although no repair deteriorated over 2 years [37,39]. No PRF study used second-look arthroscopy as a primary endpoint. Most data on healing rates therefore come from fibrin clot and PRP studies [41,21].

8.8 Comparison with PRP Evidence

The PRP literature is larger and includes randomised trials. Kaminski et al. randomised 37 patients with complete vertical (bucket-handle) tears in the red-white zone to PRP or saline injection at the repair site [21]. At 18 weeks, healing was 85% with PRP versus 47% with saline, and IKDC, KOOS and WOMAC scores were significantly better in the PRP group at 42 months [21]. In a second randomised trial of 72 patients with chronic degenerative tears treated by percutaneous trephination, failure was 48% with PRP versus 70% without, and fewer PRP-treated patients required subsequent arthroscopy (8% versus 28%) [45]. Other studies are less positive. Griffin et al. found reoperation rates of 27% with PRP and 25% without, with no difference in function [46], while Pujol et al. reported modestly

better KOOS pain and sport scores with PRP after open repair of horizontal tears [47]. A meta-analysis by Zaffagnini et al. found a failure rate of 9.9% with PRP versus 25.7% in controls [8].

These results cannot be transferred directly to PRF. PRP and PRF differ in leukocyte content, physical form, and release kinetics, and the subgroup analysis by Li and Weng suggests that different platelet products may not perform equally [42]. They do, however, establish that platelet-derived augmentation can improve healing in well-designed trials, which strengthens the rationale for testing PRF in the same way.

8.9 Synthesis of Evidence

Taken together, the evidence shows that PRF-augmented meniscal repair is safe and is followed by substantial functional improvement, but there is not yet convincing evidence that PRF improves function or healing beyond what repair alone achieves. Recent reviews describe PRF as promising but inconsistent [48], judge the overall evidence for biological augmentation to be of low quality [6], and do not recommend routine use of autologous blood products outside clinical studies [23]. The Minimum Information for Studies Evaluating Biologics in Orthopaedics (MIBO) checklist provides a framework for reporting preparation details that would make future PRF studies comparable [49].

Table 1. Summary of clinical studies on PRF and related platelet concentrates in meniscal surgery

Study	Design (LoE)	Patients	Tear / setting	Product and application	Follow-up	Outcomes measured	Key findings
PRF and fibrin-based studies							
Kemmochi et al., 2018 [37]	Comparative cohort (III)	17 PRF+PRP vs 5 controls	Arthroscopic meniscal repair	PRF + PRP injected with custom device	6 months	Tegner, Lysholm, IKDC, MRI	Scores improved in all; no significant difference between groups; MRI not clearly improved
Kemmochi, 2024 [39]	Prospective cohort (III)	35 treated, 31 evaluated	All-inside meniscal repair	PRF + PRP; products characterised by MIBO	24 months	Lysholm, IKDC, JOA, Tegner, MRI, second-look in some	Lysholm, IKDC, JOA improved; Tegner unchanged; no meniscal regeneration on MRI, no deterioration
Screpis et al., 2021 [40]	Retrospective case series (III)	15	Lateral parameniscal cyst with horizontal tear	Cyst excision + repair + PRF matrix membrane	Minimum 2 years	Lysholm, IKDC, NRS, recurrence	Lysholm 41.3 to 92.3; IKDC 37.6 to 89.4; NRS 6.8 to 1.3; no recurrence
Skaik et al., 2026 [41]	Systematic review (IV)	15 studies, 438 patients (PRF 15; PRF+PRP 48)	Mostly avascular-zone tears	Fibrin clot, BMAC clot, PRF, PRF+PRP	Varied	Healing, complications, IKDC, Lysholm, Tegner	Fibrin clot: 77.5% complete healing; fibrin-based augmentation better than non-augmented repair
Li and Weng, 2022 [42]	Meta-analysis (2 RCTs, 7 non-RCTs)	1,164	Meniscal repair	PRP and PRFM subgroups	Varied	Failure, IKDC, Lysholm, VAS, KOOS	Lower failure with platelet augmentation (OR

Study	Design (LoE)	Patients	Tear / setting	Product and application	Follow-up	Outcomes measured	Key findings
							0.64); no IKDC or Lysholm benefit; PRFM showed no functional benefit
PRP studies (for comparison)							
Kaminski et al., 2018 [21]	Double-blind RCT (I)	37	Complete vertical tears, red-white zone	PRP vs saline at repair site	42 months	Healing, IKDC, KOOS, WOMAC, VAS	Healing 85% vs 47% at 18 weeks; better IKDC, KOOS, WOMAC with PRP
Kaminski et al., 2019 [45]	Double-blind RCT (I)	72	Chronic degenerative tears	Trephination ± intrameniscal PRP	Short-term	Failure on MR arthrography or arthroscopy, KOOS, VAS	Failure 48% vs 70%; later arthroscopy 8% vs 28%
Griffin et al., 2015 [46]	Retrospective cohort (III)	35	Isolated repair	PRP vs no PRP	Mean 4 years	Reoperation, IKDC, Lysholm	Reoperation 27% vs 25%; no functional difference
Pujol et al., 2015 [47]	Case-control (III)	34	Horizontal tears, open repair	PRP vs no PRP	Mean 32 months	KOOS, IKDC, MRI	Better KOOS pain and sport with PRP; more complete MRI signal resolution
Everhart et al., 2019 [16]	Cohort (III)	550	Isolated and ACL-associated repair	PRP vs no PRP	3 years	Failure	Lower failure for isolated repair (aHR 0.18); no benefit with ACL reconstruction

Study	Design (LoE)	Patients	Tear / setting	Product and application	Follow-up	Outcomes measured	Key findings
Zaffagnini et al., 2021 [8]	Meta-analysis	286 in pooled analysis	Isolated repair	PRP vs no PRP	Varied	Failure, adverse events	Failure 9.9% vs 25.7%; one septic arthritis with PRP

aHR, adjusted hazard ratio; BMAC, bone marrow aspirate concentrate; IKDC, International Knee Documentation Committee; JOA, Japanese Orthopaedic Association; KOOS, Knee Injury and Osteoarthritis Outcome Score; LoE, level of evidence; MIBO, Minimum Information for Studies Evaluating Biologics in Orthopaedics; NRS, numeric rating scale; OR, odds ratio; PRFM, platelet-rich fibrin matrix; RCT, randomised controlled trial; VAS, visual analogue scale; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

9. Safety, Cost-Effectiveness and Practical Considerations

PRF is fully autologous and contains no anticoagulant, bovine thrombin or other additives, so the risks of immune reaction and disease transmission are negligible [9]. No PRF-specific complications were reported in the clinical studies reviewed [37,39,40]. In the broader fibrin-based literature, complications occurred in 8.2% of patients receiving fibrin clot augmentation and consisted mainly of paraesthesia and re-injury, which are more plausibly related to the repair itself than to the biologic [41]. One case of septic arthritis has been reported after PRP augmentation [8], a reminder that any additional handling of blood products requires strict asepsis.

Cost is a major practical advantage. PRF requires only a venous blood draw, plain tubes and a standard tabletop centrifuge, with no commercial kit. Preparation adds a few minutes to the operation and can be done by theatre staff while the surgeon prepares the tear. The recent Delphi consensus explicitly favoured simple, low-cost augmentation strategies for meniscal repair [23], and PRF fits that description. This makes it particularly suitable for resource-limited settings such as India, where commercial PRP systems and cell-based therapies may be unaffordable for many patients, and where the technique has already been described by Indian surgeons [34].

The main practical drawbacks are the need to coordinate blood harvest with the timing of surgery, the learning curve of delivering a clot arthroscopically, and the lack of a validated, standard protocol.

10. Limitations of Current Evidence

The evidence base for PRF in meniscal repair has several important weaknesses.

Lack of standardisation. PRF is not a single product. Centrifuge model, speed, duration, tube material and the delay between blood draw and centrifugation all change the platelet, leukocyte and growth factor content of the final clot. Few studies report these details, and products described as PRF, L-PRF, PRFM or PRF combined with PRP are often grouped together despite being biologically different.

Small samples and weak designs. The PRF-specific clinical literature consists largely of case series and small cohorts, some with very small or no control groups. None is an adequately powered randomised controlled trial, so a moderate but clinically meaningful benefit could easily be missed, and positive results from uncontrolled series cannot be attributed to PRF rather than to the repair itself.

Short follow-up. Most studies report outcomes at 6 months to 2 years. The main justification for meniscal repair is long-term protection against osteoarthritis, and no study has followed PRF-augmented repairs long enough to address this.

Heterogeneous populations. Tear pattern, zone, chronicity, patient age and concomitant procedures vary widely within and between studies. Augmentation may be valuable for isolated avascular-zone tears but add little to peripheral tears or repairs with ACL reconstruction, and pooled analyses can obscure such differences.

Inconsistent outcome measures. Studies use different combinations of Lysholm, IKDC, KOOS, Tegner and visual analogue scales, and different definitions of healing and failure. Structural assessment by MRI or second-look arthroscopy is inconsistent, which makes it difficult to compare or pool results.

Risk of bias. Blinding of patients and assessors is rarely described, and publication bias towards positive results is likely in a small and enthusiastic literature.

Uncertain role of leukocytes. Leukocytes in L-PRF may support healing through cytokine production and antimicrobial activity, but they may also increase catabolic activity in meniscal tissue. Whether leukocyte-rich or leukocyte-poor preparations are preferable for the meniscus remains unknown.

11. Future Directions

The most pressing need is for adequately powered, prospective randomised controlled trials comparing meniscal repair with and without PRF. Such trials should be stratified by tear zone, tear pattern and the presence of ACL reconstruction, and should blind patients and outcome assessors where feasible. Primary endpoints should combine validated patient-reported outcomes with an objective measure of healing, such as MRI or second-look arthroscopy, and follow-up should extend beyond 5 years to capture repair survival and the development of osteoarthritis.

Preparation protocols need to be standardised and reported in full, including blood volume, tube type, centrifuge model, relative centrifugal force, spin time, the interval between blood draw and centrifugation, and the platelet and leukocyte content of the final product. Head-to-head comparisons of L-PRF, A-PRF, i-PRF and PRP would clarify which product, if any, is best suited to the meniscus.

PRF could also serve as a carrier. Combining it with mesenchymal stromal cells, collagen or synthetic scaffolds, or meniscal wrapping may enhance healing of complex or avascular tears. Injectable PRF could be explored as a postoperative intra-articular treatment to support healing and reduce effusion and pain. Separate trials are needed to test whether PRF benefits patients undergoing partial meniscectomy.

Finally, formal cost-effectiveness analyses, particularly in low- and middle-income countries, would show whether the modest cost of PRF is justified by fewer failures and reoperations. Clear

patient selection criteria, identifying which patients and tears are most likely to benefit, would allow PRF to be used where it adds the most value.

12. Conclusion

Platelet-rich fibrin is a biologically plausible, safe, simple and inexpensive augment for meniscal repair. It supplies a natural fibrin scaffold together with a sustained release of growth factors, and laboratory studies show that it stimulates meniscal cell migration, proliferation and matrix synthesis. Clinical studies report marked improvement in function after PRF-augmented repair, but they are small, mostly non-randomised and methodologically heterogeneous, and they have not yet shown a clear advantage over repair alone. Until well-designed randomised controlled trials with standardised preparation, objective healing assessment and long-term follow-up are available, PRF should be regarded as a promising adjunct best used within structured clinical protocols, particularly for isolated repairs of tears at higher risk of failure.

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