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The Effect of Platelet-Rich Fibrin on Block, Crushed and Diced Cartilage

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Abstract

Background In nasal surgery, autologous cartilage grafts are routinely used to correct structural weakness, contour deformities, and irregularities, yet current techniques still face challenges related to long-term cartilage graft viability and resorption. Platelet-rich fibrin (PRF) is a second-generation platelet concentrate that may improve graft survival through its sustained release of bioactive molecules within a natural fibrin matrix.

Objective To evaluate the biochemical and histopathological effects of combining PRF with autologous cartilage grafts of different forms (block, crushed, and diced) in a rabbit model and to determine which combination provides optimal cartilage viability and matrix stability.

Methods Twenty-four New Zealand rabbits divided into six groups based on their form: block, crushed, and diced. PRF was applied to three groups, while the remaining three served as controls. The grafts were placed in subcutaneous pockets on the rabbits' backs and harvested after 8 weeks for histopathological and biochemical analyses. Cartilage tissues, growth factors, matrix components, angiogenesis, oxidative stress, and apoptosis markers were evaluated.

Results PRF-treated groups demonstrated significantly higher growth factor levels, antioxidant status, lower oxidative stress and apoptosis markers compared with controls ($p < 0.05$). Histological analyses showed better cellular proliferation and extracellular matrix preservation, particularly in PRF-combined diced and block cartilage grafts, whereas crushed cartilage exhibited marked degeneration.

Conclusion PRF enhanced the viability, proliferation, and angiogenic response of autologous cartilage grafts through

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its sustained release of growth factors and scaffold-like fibrin matrix. These findings suggest that PRF may improve graft stability and reduce postoperative complications in nasal surgery.

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Keywords Platelet-rich fibrin · PRF · Autologous cartilage graft · Rhinoplasty

Introduction

In rhinoplasty, weakness of the nasal framework and contour irregularities can lead to functional impairment and aesthetic dissatisfaction [1]. Autologous cartilage grafts remain the preferred material for structural support and contour restoration in such cases. The principal indications for cartilage grafting include concealing surface irregularities, reinforcing the nasal skeleton, and refining nasal shape. Cartilage can be used in several configurations—block, diced, minced, sliced, or crushed—depending on the surgical indication [1–3]. Numerous techniques have been described to enhance cartilage graft viability and long-term stability, reduce resorption, and minimize graft displacement or dispersion. These include employing cartilage in different physical forms, wrapping it with various biological or synthetic materials, and applying specific modification techniques to optimize graft integration and maintain the desired contour and volume [4–6].

Recently, platelet-rich fibrin (PRF), an advanced platelet concentrate rich in growth factors, has emerged as a promising biological agent for enhancing tissue regeneration. PRF provides a sustained release of bioactive molecules within a natural fibrin matrix, promoting angiogenesis, cellular proliferation, and extracellular matrix remodeling.

The present experimental study aimed to evaluate the effects of PRF on biochemical composition and histological architecture of autologous cartilage grafts in different forms (block, crushed, and diced) using a rabbit model. By comparing PRF-treated and control grafts, this study sought to elucidate the biological and physiological mechanisms through which PRF may enhance cartilage viability, matrix preservation, and overall graft stability.

Materials and Methods

The study was conducted at the Experimental Animal Laboratory of our University Research Center and was approved by the University Animal Experiments Ethics Committee on 27.05.2022 (Approval No: E.63385).

Subjects

A total of 24 New Zealand rabbits, aged 4–6 months and weighing an average of 3000 g (2500–3500 g), were used in the study.

Preparation of Cartilage Grafts

A 3 x 2 cm cartilage graft without perichondrium was harvested through an incision made 4 cm distal to the external auditory canal of animal subjects. From the harvested cartilage graft, six grafts measuring 1 x 1 cm were obtained. Two 1 x 1 cm block cartilage grafts, diced cartilage grafts (1 mm^3 each), and moderately crushed cartilage grafts were prepared. Dicing was performed using an 11-blade scalpel, and crushing was achieved with a Cottle cartilage crusher [7].

Preparation of PRF and Combination with Cartilage Grafts

Blood samples were collected from each animal into sterile venous blood tubes and centrifuged at 3000 rpm for 10 min using the Choukroun method. Following centrifugation, the platelet-rich fibrin (PRF) layer obtained from the middle fraction of the tube (Fig. 1) was carefully separated and combined with autologous cartilage grafts under standardized conditions [8].

Placement of Cartilage Grafts

Six subcutaneous pockets measuring 1.5 cm in length were created in the paraspinal region of the back, positioned 3 cm from the midline and spaced 5 cm apart (Fig. 2). Block, crushed, and diced cartilage grafts were placed into the upper, middle, and lower pockets, respectively. On one side, the grafts were combined with platelet-rich fibrin (PRF), while the contralateral grafts were left untreated to serve as controls (Fig. 3).

Harvesting of Cartilage Grafts

The grafts were carefully harvested from the subcutaneous pockets eight weeks after the implantation.

Biochemical Analysis

Half of the harvested cartilage grafts were homogenized, and the resulting supernatants were collected for analysis. Total protein concentration was determined using the Bradford method [9]. The following biochemical markers were quantified using commercial ELISA kits: hydroxyproline (HPR), glycosaminoglycan (GAG), type II collagen alpha-1 chain (COL2A1), transforming growth factor beta-1 (TGF-β1), basic fibroblast growth factor (bFGF), epidermal growth factor (EGF), vascular endothelial growth factor (VEGF), hypoxia-inducible factor-1 alpha (HIF-1α), platelet-derived growth factor (PDGF), cartilage oligomeric matrix protein (COMP), and cytokeratin-18 M30 (STK-18 M30). Total antioxidant status (TAS) and total oxidant status (TOS) were determined using the Erel method [10], and the oxidative stress index (OSI) was subsequently calculated as the TOS/TAS ratio.

Histopathological Analysis

The remaining half of the tissue samples were processed for histopathological evaluation. Sections were stained with hematoxylin–eosin (H&E), Masson's trichrome, orcein, and Safranin O. H&E staining was used for general morphological assessment of cartilage tissue, identifying cellular integrity and degenerative changes. Masson's trichrome facilitated evaluation of collagen content, fibrosis, and extracellular matrix (ECM) integrity. Orcein staining was employed to differentiate elastic fibers, while Safranin O was essential for assessing proteoglycan and glycosaminoglycan (GAG) content within the cartilage matrix.

Statistical Analysis

Intergroup differences were evaluated using one-way ANOVA. Variance homogeneity was checked using Levene's test, and post hoc analyses were performed using Tukey and Games-Howell tests. Statistical analyses were performed using SPSS 26.0 with a significance level of $p < 0.05$.

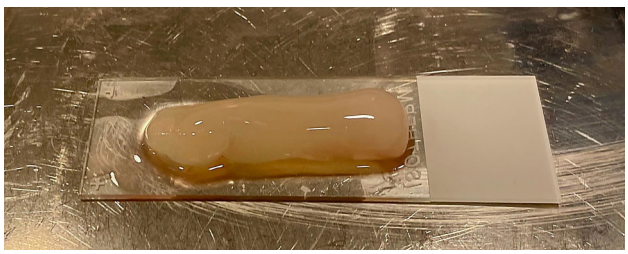

Fig. 1 Platelet-rich fibrin (PRF) layer obtained after centrifugation, demonstrating clear separation from the underlying red blood cell fraction

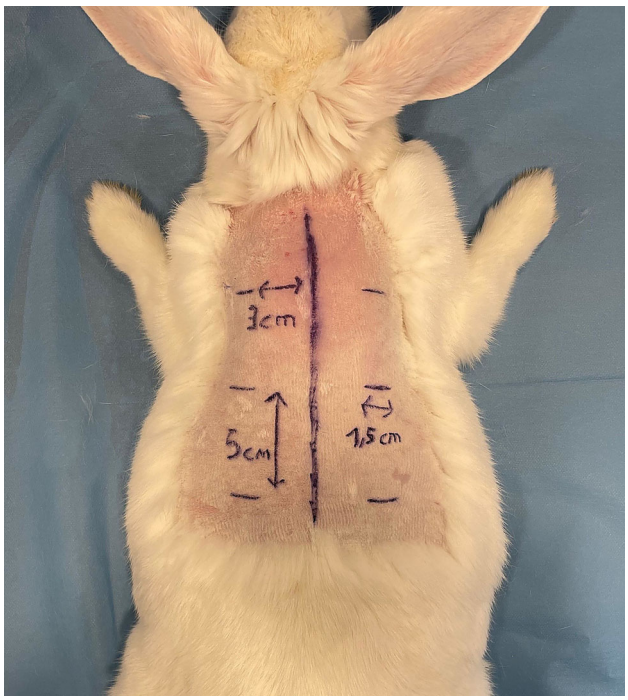

Fig. 2 Markings for subcutaneous pockets to be created in the paraspinal region on the backs of the experimental animals

Results

Biochemical Findings

Biochemical analyses demonstrated that COL2A1, HPR, VEGF, GAG, bFGF, HIF-1α, PDGF, COMP, TGF-β1, and EGF levels were significantly higher in the PRF-treated groups compared with controls ($p < 0.05$). Intragroup comparisons revealed that these biomarkers were markedly elevated in the diced and block cartilage groups relative to the crushed cartilage groups ($p < 0.05$). Assessment of oxidative stress and apoptosis markers showed that TAS was significantly higher in PRF-treated groups, whereas TOS, OSI, and STK-18 values were greater in the control groups ($p < 0.05$). Among all groups, the crushed cartilage samples exhibited the highest TOS, OSI, and STK-18 levels ($p < 0.05$).

The results of the one-way analysis of variance (ANOVA) for the biochemical parameters among the study groups are summarized in Table 1.

Histopathological Findings

H&E Staining

In the block cartilage group, approximately half of the cartilage specimens remained intact, while the remainder exhibited mild to severe degenerative changes. Areas of

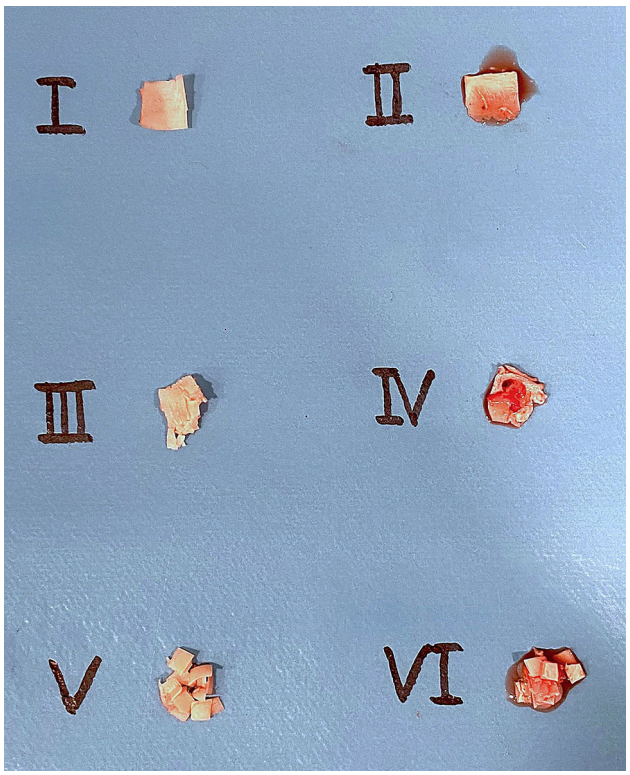

Fig. 3 Control block (I), crushed (III), and diced (V) autologous cartilage grafts alongside their corresponding PRF-combined counterparts: block (II), crushed (IV), and diced (VI)

cellular proliferation extending into the adjacent connective tissue were also observed. In contrast, the PRF-block group demonstrated predominantly intact cartilage with peripheral proliferation extending minimally into the connective tissue.

In the crushed cartilage group, marked degeneration was evident, characterized by the replacement of cartilage with spongy bone tissue (Fig. 4). The PRF-crushed group also showed evidence of degeneration; however, the extent of cellular proliferation was greater than in the untreated crushed cartilage group.

In the diced cartilage group, approximately half of the grafts retained normal cartilage structure, with peripheral proliferative zones observed adjacent to viable cartilage. The PRF-diced group demonstrated largely preserved cartilage architecture, accompanied by widespread peripheral proliferation (Fig. 5).

Overall, PRF-treated diced and block cartilage grafts exhibited better cartilage viability, reduced fibrosis, and less degeneration compared with both their respective control groups and the crushed cartilage groups.

Masson's Trichrome Staining

Denser matrix staining was observed in the block and diced cartilage groups compared with the crushed cartilage group. In the PRF-block and PRF-diced groups, the cartilage matrix exhibited more intense staining than in their respective controls, indicating that platelet-rich fibrin (PRF) contributed to the preservation of collagen fiber content (Figs. 6 and 7).

Orcein Staining

The block and diced cartilage groups exhibited a greater number of orcein-stained regions compared with the crushed cartilage group. The PRF-treated groups demonstrated more prominent elastic fiber formation and new cartilage development within the peripheral proliferative zones, indicating enhanced matrix remodeling and cartilage regeneration relative to the control groups (Fig. 8).

Safranin O Staining

The proteoglycan content of the cartilage matrix was higher in the block and diced cartilage groups compared with the crushed cartilage group. The PRF-block and PRF-diced groups demonstrated intense Safranin O staining, indicative of greater chondrocyte viability and higher proteoglycan concentration relative to the other groups. In contrast, the crushed cartilage grafts exhibited increased bone tissue formation, reflecting more extensive cartilage degeneration (Figs. 9 and 10).

Discussion

In the early 20th century, the use of block cartilage in nasal surgery was limited due to issues of bending and warping, the mechanisms of which were poorly understood at the time. Advances in the understanding of cartilage biomechanics and refinements in incision and shaping techniques subsequently led to broader clinical adoption of block, diced, and crushed autologous cartilage grafts [6]. Numerous adjunctive methods have since been developed to enhance cartilage viability, prevent resorption, and minimize graft displacement. These include the use or wrapping of grafts with oxidized regenerated cellulose, autologous fascia, acellular dermal matrix (ADM), fibrin glue, perichondrium, Tutopatch, esterified hyaluronic acid, fat grafts, blood clots, and platelet-derived products [4–6, 11–16].

Despite these advancements, a definitive consensus on the optimal material for autologous cartilage grafting has yet to be established. Currently, no alternative technique or

Table 1 Biochemical biomarker comparisons between control and PRF-treated cartilage graft groups

Biomarker	CBC	CCC	CDC	PRF-BC	PRF-CC	PRF-DC	*p* value
COL2A1	7.81	7.66	8.08	10.43	8.93	14.72	< 0.05
COMP	4.99	4.51	5.03	5.95	5.78	7.80	< 0.05
EGF	2.65	2.07	2.69	3.63	2.75	4.53	< 0.05
GAG	43.48	40.84	48.83	65.09	56.49	76.42	< 0.05
HIF-1α	5.41	3.94	5.98	7.11	6.24	7.85	< 0.05
HPR	573.68	513.91	615.87	1020.39	985.03	1223.87	< 0.05
OSI	15.81	21.37	12.04	6.48	8.97	4.27	< 0.05
PDGF	53.41	41.80	58.33	71.76	66.27	83.12	< 0.05
STK-18 M30	31.85	37.45	29.47	24.83	27.81	21.24	< 0.05
TAS	0.76	0.64	0.89	1.31	1.07	1.58	< 0.05
TGF-β1	26.25	21.35	31.23	38.77	35.63	42.76	< 0.05
TOS	11.72	13.16	10.52	8.33	9.57	6.68	< 0.05
VEGF	40.76	39.93	48.56	71.90	62.70	86.68	< 0.05
bFGF	171.48	143.83	213.77	258.03	225.44	309.33	< 0.05

Values represent group means. Statistical significance tested using one-way ANOVA. Variance homogeneity was checked using Levene's test, and post hoc analyses were performed using Tukey and Games-Howell tests, significance level $p < 0.05$

Mean biochemical biomarker values among the groups. PRF-treated groups demonstrated significantly higher chondrogenesis, extracellular matrix preservation, angiogenesis, and antioxidant markers compared with controls ($p < 0.05$). Oxidative stress and apoptosis markers were lowest in the PRF-diced cartilage group, indicating enhanced cartilage viability and reduced degeneration. *CBC* control block cartilage, *CCC* control crushed cartilage, *CDC* control diced cartilage, *PRF-BC* PRF-block cartilage, *PRF-CC* PRF-crushed cartilage, *PRF-DC* PRF-diced cartilage

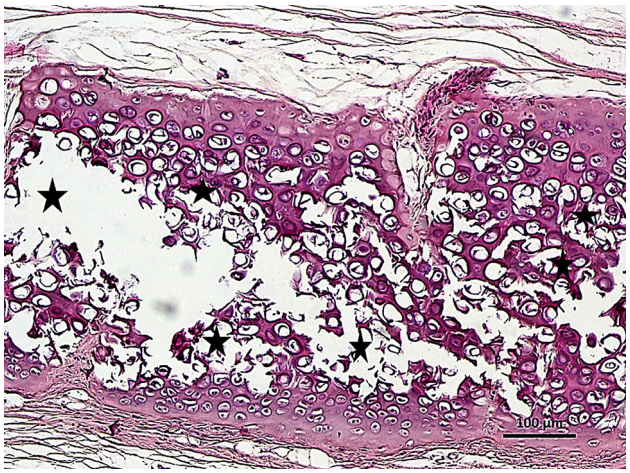

Fig. 4 Histological section of the transferred cartilage tissue in the control crushed cartilage group, demonstrating marked cartilage degeneration (black stars) (H&E, × 100)

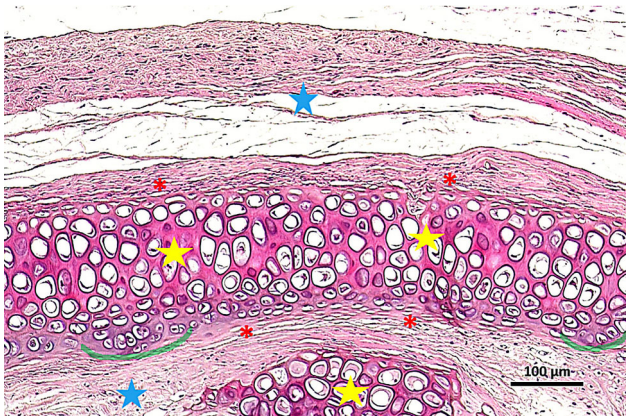

Fig. 5 Histological section of the transferred cartilage tissue in the PRF-diced cartilage group (PRF-DC). Intact cartilage areas (yellow stars) and prominent peripheral proliferation zones (outlined in green) are evident (H&E, × 100)

adjunctive material qualifies as a gold standard for consistently enhancing graft stability, integration, or long-term viability.

Current tissue engineering strategies rely on three fundamental elements: a cellular component, a biocompatible and mechanically stable scaffold, and bioactive molecules that promote regeneration. In the context of cartilage grafting, the ideal scaffold should provide structural support and moldability, minimize inflammation, prevent infection or immune reaction, and promote cartilage viability, integration, and long-term survival.

Platelet-rich fibrin (PRF), a second-generation platelet concentrate obtained by centrifuging whole blood without additives, contains multiple growth factors that modulate cellular activity through specific receptor-mediated pathways. PRF forms a natural fibrin scaffold that stabilizes the clot and serves as a reservoir for sustained growth factor

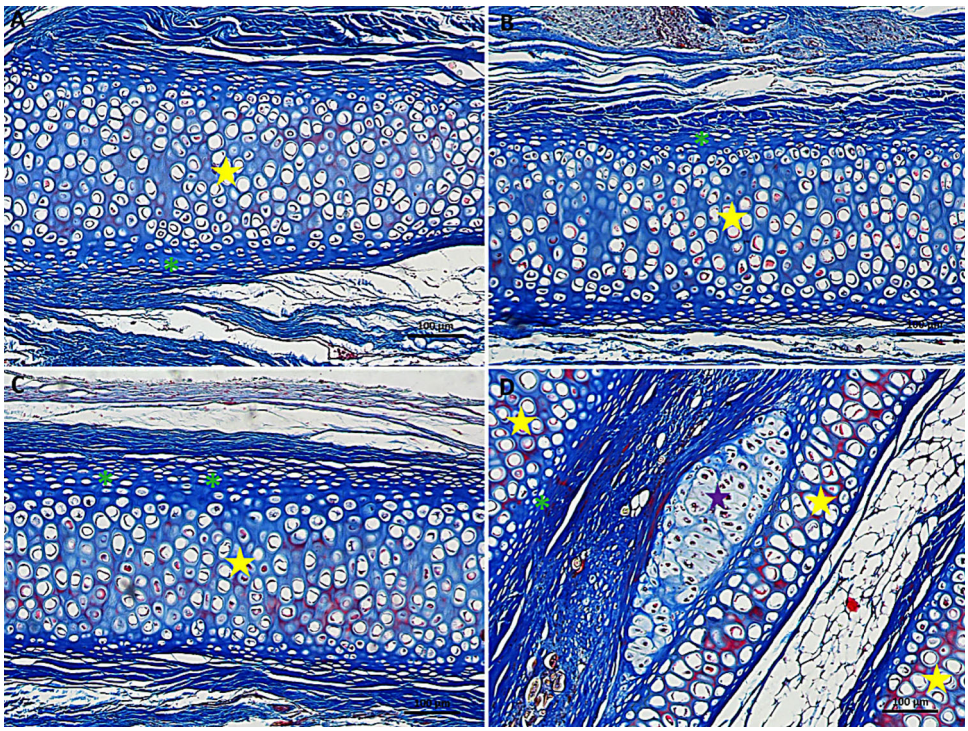

Fig. 6 Masson's trichrome-stained sections of transferred cartilage tissues. **A** Control block cartilage group (CBC), **B** PRF-block cartilage group (PRF-BC), **C** Control diced cartilage group (CDC), **D** PRF-diced cartilage group (PRF-DC). Cartilage tissue (yellow stars) in the PRF-BC and PRF-DC groups appears darker compared to the CBC and CDC groups. Green asterisks indicate peripheral proliferation areas, and purple stars indicate locally proliferated cartilage areas (Masson's Trichrome, × 100)

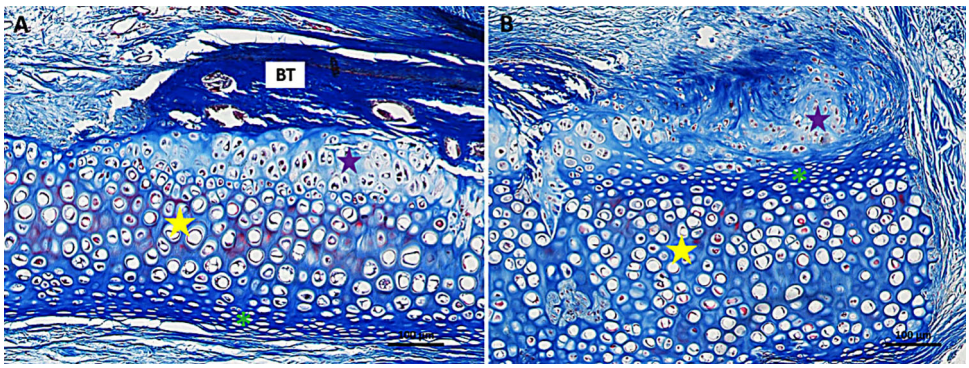

Fig. 7 Masson's trichrome-stained sections of transferred cartilage tissues. **A** Crushed cartilage group (CCC), **B** PRF-crushed cartilage group (PRF-CC). Cartilage tissue (yellow stars) in the CCC and PRF-CC groups appears paler compared to the block and diced cartilage groups shown in Fig. 6. Green asterisks indicate peripheral proliferation areas, and purple stars indicate locally proliferated cartilage areas. *BT* Cancellous bone tissue (Masson's Trichrome, × 100)

release. During the healing process, platelet activation stimulates mesenchymal stem cell (MSC) migration and differentiation, while the fibrin matrix sequesters growth factors at the application site. Concurrently, fibroblasts remodel the matrix and initiate collagen synthesis, thereby facilitating tissue repair and regeneration [17].

As PRF is entirely autologous, it carries a minimal risk of immunogenic reaction or disease transmission. In comparison, platelet-rich plasma (PRP) requires the addition of anticoagulants, activators, or gel agents, making PRF a simpler and more cost-effective option for both clinicians and patients [17]. Unlike PRP and other first-generation platelet concentrates, which release growth factors rapidly and transiently, PRF provides a sustained release, with most factors secreted over approximately 7 days, and some persisting for up to 23–28 days [18]. This prolonged activity results from PRF's dense fibrin architecture, which resists rapid proteolysis and allows gradual remodeling of the matrix [19].

Dohan et al. demonstrated that PRF functions similarly to living tissue, entrapping platelets and leukocytes that actively contribute to the healing cascade [20]. Beyond its regenerative capacity, PRF also serves as an effective hemostatic agent, releasing coagulation-related factors that aid in clot stabilization. Compared with PRP, PRF contains higher concentrations of growth factors, promotes

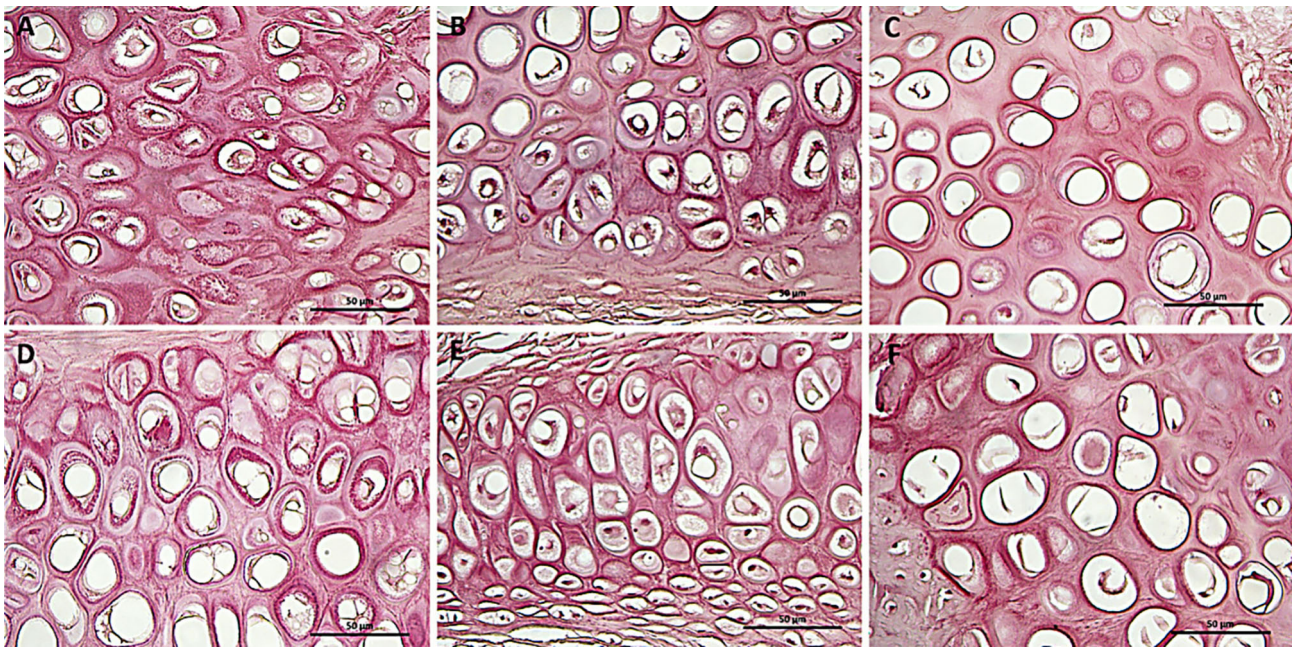

Fig. 8 Orcein-stained sections of the cartilage graft tissues are shown. **A** Control block cartilage (CBC), **B** Control diced cartilage (CDC), **C** Control crushed cartilage (CCC), **D** PRF-block cartilage (PRF-BC), **E** PRF-diced cartilage (PRF-DC), **F** PRF-crushed cartilage (PRF-CC) (Orcein, × 400)

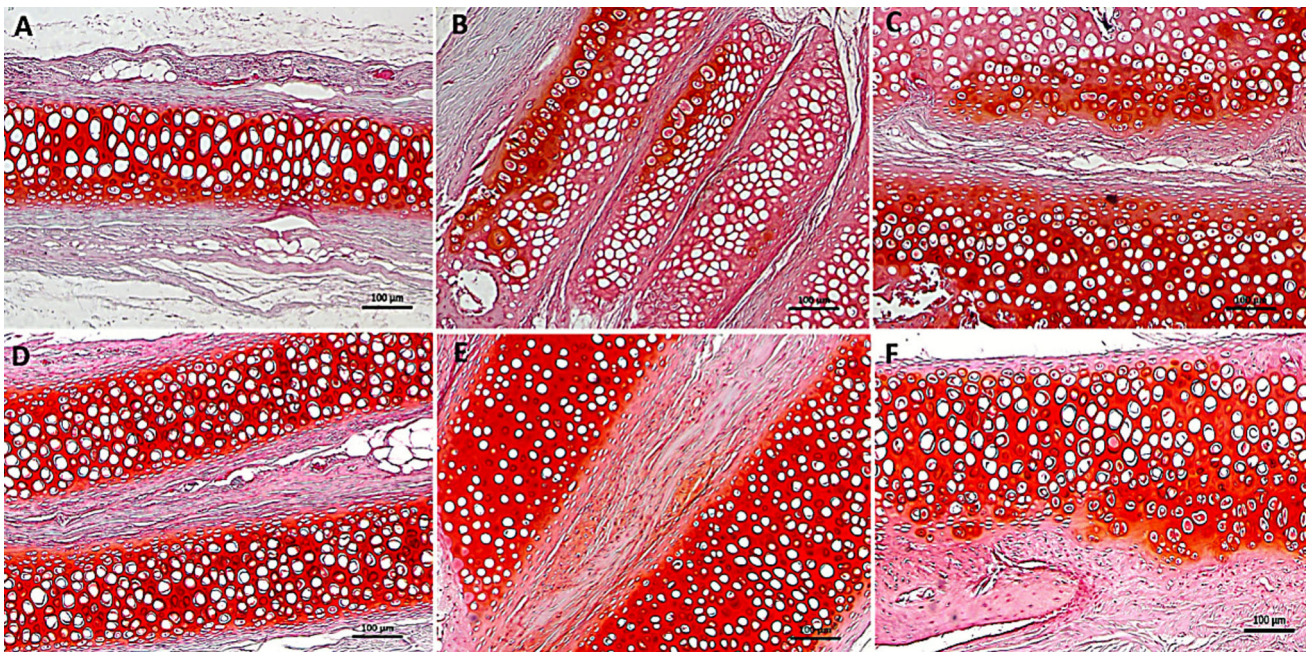

Fig. 9 Safranin O-stained sections of the cartilage graft tissues. **A** Control block cartilage (CBC), **B** Control diced cartilage (CDC), **C** Control crushed cartilage (CCC), **D** PRF-block cartilage (PRF-BC), **E** PRF-diced cartilage (PRF-DC), **F** PRF-crushed cartilage (PRF-CC) (Safranin O, × 100)

angiogenesis, enhances tissue repair, and more effectively stimulates MSC migration and differentiation [21].

Previous studies have demonstrated that platelet-rich fibrin (PRF) enhances chondrocyte proliferation, viability, and differentiation, supports cartilage matrix mineralization, and increases type II collagen synthesis. PRF treatment has also been shown to improve cartilage integration and promote matrix organization in experimental models [22]. Furthermore, studies involving diced cartilage grafts have reported that PRF, particularly in its

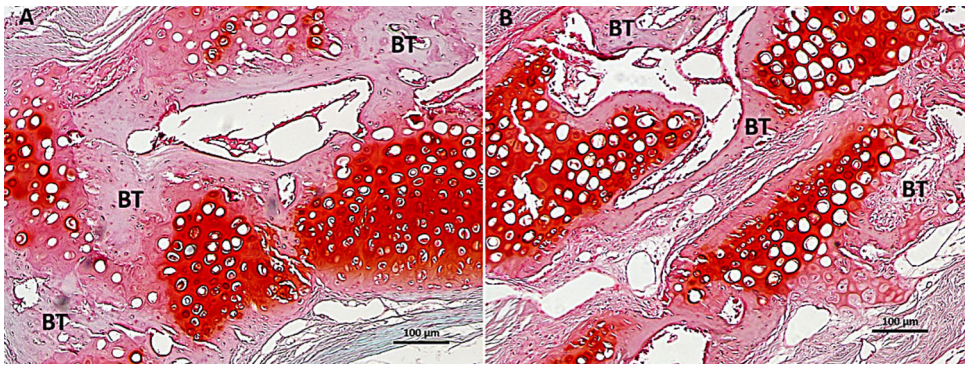

Fig. 10 Safranin O-stained sections of crushed cartilage grafts. **A** Control crushed cartilage (CCC) and **B** PRF-crushed cartilage (PRF-CC). The cartilage matrix adjacent to the bone tissue (BT) shows weak Safranin O staining, while adjacent regions exhibit minimal to no staining (Safranin O, × 100)

gel form, serves effectively as a biological carrier, facilitating graft handling and stabilization [23].

In the present study, we assessed key determinants of cartilage graft behavior—including cartilage viability, cellular proliferation, angiogenesis, oxidative stress, and apoptotic activity—across different graft morphologies and systematically compared the effects of PRF using both biochemical and histopathological outcomes.

ELISA analyses revealed that type-II collagen and chondrogenesis biomarker COL2A1, intracellular proteoglycan accumulation biomarker GAG, collagen turnover indicator and a wound healing biomarker HPR, proteoglycan synthesis-related FGF, and COMP which plays a role in the formation and regulation of the extracellular matrix and contributes to tissue homeostasis were significantly higher in all PRF-combined groups compared to controls. These biochemical findings align with previous literature and support the chondrogenic, proliferative, chondroprotective, and cartilage-stabilizing effects attributed to PRF [24].

PDGF, which plays a role in mesenchymal cell migration, proliferation, macrophage activation, and angiogenesis; TGF-β1, which triggers fibroblast chemotaxis, collagen synthesis, immune cell angiogenesis, cellular differentiation, and cell proliferation; VEGF, a key regulator of angiogenesis and cell survival; HIF-1α, which increases the production of angiogenic factors like VEGF in response to hypoxia; and EGF, an important growth factor that promotes angiogenesis, accelerates granulation tissue formation, and supports wound healing, were all significantly higher in PRF-combined groups compared to controls. The upregulation of these factors in the PRF groups further reinforces PRF's role in chondrogenesis, cellular proliferation, angiogenesis, and regulation of tissue repair [20, 25–27].

PRF-treated groups demonstrated reduced oxidative stress, reflected by lower TOS and OSI scores and higher TAS levels compared with controls. The PRF-diced group showed the lowest oxidative stress, whereas the control crushed group showed the highest. STK-18 M30 apoptosis levels were also elevated in controls relative to PRF groups. These findings, consistent with prior reports, support the antioxidant and antiapoptotic effects of PRF, indicating its capacity to limit free radical-induced injury and protect cartilage from oxidative and DNA damage through sustained growth-factor release [28–36].

The biochemical findings of this study were corroborated by the histopathological results, reinforcing the internal consistency of the data and strengthening the translational relevance of these outcomes for potential clinical application.

Biochemical and histopathological analyses demonstrated that diced and block cartilage grafts exhibited higher chondrocyte viability, less degeneration, and richer extracellular matrix (ECM) composition compared with crushed cartilage grafts. PRF treatment improved collagen content, proteoglycan density, and chondrocyte viability across all graft types; however, these beneficial effects were less pronounced in the crushed cartilage group, which showed greater degenerative changes and evidence of ossification.

Recent evidence reinforces PRF's relevance in both experimental and clinical rhinoplasty. When used as a gel carrier or wrapping material, PRF has been shown to enhance diced cartilage viability, reduce degeneration and inflammation, limit fibrosis, and provide a stable scaffold for graft handling and contouring. Clinical studies report improved dorsal graft stability, lower resorption rates, and better extracellular-matrix organization [8, 37–42]. PRF has also been associated with reduced nasal skin thickness, improved postoperative tissue quality, and decreased edema, ecchymosis, and pain [43, 44]. In augmentation rhinoplasty, combining PRF with high-density autologous fat improves fat retention and long-term stability [45]. These findings parallel the present results, supporting PRF as a biologically active scaffold that enhances cartilage integrity and viability. Collectively, current evidence underscores the translational relevance of this study and highlights PRF's multifunctional role in tissue integration and postoperative healing in clinical practice.

Our findings align with existing literature and confirm that PRF not only enhances cartilage graft viability but also establishes a biologically favorable microenvironment that supports long-term matrix remodeling and tissue integration, potentially improving the durability and predictability of outcomes in both aesthetic and reconstructive rhinoplasty. PRF-supported diced or block cartilage grafts may be especially advantageous in procedures requiring nasal tip or dorsal augmentation, contour refinement, or camouflage of surface irregularities. In structurally demanding cases, such as crooked-nose deformities, block cartilage continues to provide reliable skeletal support. Conversely, in open-roof deformities or situations requiring camouflage over the bony dorsum, crushed cartilage—which demonstrated a tendency toward osseous transformation—may serve as a practical and effective option. This osteogenic potential can be viewed as a favorable clinical attribute and may be intentionally leveraged in routine surgical practice.

Compared with the existing literature, our study advances current understanding by simultaneously evaluating PRF's biochemical, histopathological and antioxidant effects across three distinct cartilage morphologies within a single controlled animal model—a comprehensive approach that has not been previously integrated into one study design.

An 8-week follow-up period is widely used in experimental cartilage studies and was therefore selected to capture the early and intermediate phases of cartilage healing, as structural alterations have been reported as early as 6 weeks [46]. However, cartilage remodeling and resorption may continue beyond this interval, indicating that longer-term observation could provide additional insight into the durability and progression of graft changes.

The absence of weight and volume measurements represents a methodological limitation. Although these parameters are clinically relevant for assessing resorption, reliable quantification was difficult—particularly in crushed and diced grafts, where variability in hydration and PRF absorption could significantly affect measurements. In addition, PRF exerts its effects primarily at the cellular and extracellular matrix levels rather than on gross tissue mass [47, 48], meaning early gains in chondrocyte viability, matrix preservation, and reduced oxidative stress may not yield measurable macroscopic changes within an 8-week period. Previous studies similarly report that PRF improves chondrocyte viability and matrix synthesis in large-animal models [23, 47], while attempts to measure graft mass or volume have produced inconsistent and technically limited results [49, 50]. Accordingly, this study prioritized histological and biochemical outcomes, which provide greater sensitivity for detecting early resorption, viability, and matrix integrity than macroscopic measurements.

Within-animal design was selected in line with ethical committee guidance to reduce animal use. Both control and PRF-treated grafts were placed in separate subcutaneous pockets within the same animal under the assumption that PRF acts primarily locally rather than systemically. However, a small degree of systemic crossover cannot be entirely excluded. Studies on PRP have shown that locally administered platelet concentrates may induce transient systemic anti-inflammatory effects [51], and given the shared growth-factor profile, a limited systemic influence from PRF remains theoretically possible. Nonetheless, PRF's dense fibrin matrix supports a slow, localized release of bioactive molecules, making systemic dissemination unlikely. Importantly, several referenced studies have utilized the same design to limit biological variability and adhere to ethical standards [6, 8, 23, 52]. Therefore, this approach aligns with established experimental practice and supports the validity of the study design.

The generalizability of this study is limited by the follow-up duration and the use of an experimental rabbit model, as species-specific differences in cartilage biology and healing may not fully reflect human responses.

Conclusion

In this experimental model, we demonstrated that platelet-rich fibrin (PRF)—a second-generation platelet concentrate—enhances cartilage viability across different graft morphologies. PRF exerted its beneficial effects through its fibrin matrix architecture, scaffold-like mechanical properties, and sustained release of regenerative growth factors, collectively supporting graft survival, matrix preservation, and tissue homeostasis. These findings suggest that PRF can be effectively combined with autologous cartilage grafts to reduce complications and achieve more predictable outcomes in both primary and secondary rhinoplasty.

By integrating the principles of autologous tissue repair with a biologically active scaffold, this study contributes valuable evidence to the fields of tissue engineering and cartilage regeneration. However, the molecular complexity of PRF and the precise bioactive components responsible for its regenerative effects remain incompletely understood. Further experimental and clinical investigations are warranted to elucidate these mechanisms and to clarify PRF's full therapeutic potential in nasal surgery and beyond.

Author Contributions All authors made substantial contributions to conception and design, and/or acquisition of data, and/or analysis and interpretation of data; participated in drafting the article or revising it

critically for important intellectual content; and gave final approval of the version to be submitted.

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Declarations

Conflict of interest Authors declare no conflict of interest relating to this research.

Ethical Approval All applicable international, national, and/or institutional guidelines for the care and use of animals were followed. The current study was performed in the Plastic and Reconstructive Surgery Department of our tertiary care center with an approval from the local Institutional Review Board.

Informed Consent For this type of study, informed consent is not required.

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