

REVIEW ARTICLE

Platelet-rich plasma as a regenerative adjunct in breast reconstruction following mastectomy or lumpectomy for invasive ductal carcinoma

Hurricane Thierry Cabaron^{1†}, Cyra Mae Lazarito^{1†}, Onikka Bernice E. Jara¹,
Jomar Adams Ganding^{1*}, Rebecca Shin-Yee Wong^{2,3}, and Nancy Choon-Si
Ng^{2,4}

¹Department of Medical Technology, School of Health Sciences, Mapua University, Manila, Philippines

²Department of Medical Education, Sir Jeffrey Cheah Sunway Medical School, Faculty of Medical and Life Sciences, Sunway University, Bandar Sunway, Selangor, Malaysia

³Faculty of Medicine, University of Cyberjaya, Cyberjaya, Selangor, Malaysia

⁴Faculty of Education and Liberal Arts, INTI International University, Nilai, Negeri Sembilan, Malaysia

Abstract

†These authors contributed equally to this work.

***Corresponding author:**

Jomar Adams Ganding
(jaganding@mapua.edu.ph)

Citation: Cabaron HT, Lazarito CM, Jara OBE, Ganding JA, Wong RSY, Ng NCS. Platelet-rich plasma as a regenerative adjunct in breast reconstruction following mastectomy or lumpectomy for invasive ductal carcinoma. *Eurasian J Med Oncol*.
doi: 10.36922/EJMO026040044

Received: January 23, 2026

Revised: March 18, 2026

Accepted: March 25, 2026

Published online: July 8, 2026

Copyright: © 2026 Author(s). This is an Open-Access article distributed under the terms of the Creative Commons Attribution License, permitting distribution, and reproduction in any medium, provided the original work is properly cited.

Publisher's Note: AccScience Publishing remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Breast cancer remains a major global health burden, accounting for an estimated 2.3 million new cases, 685,000 deaths, and approximately 20 million disability-adjusted life years annually. Invasive ductal carcinoma (IDC), the most common invasive subtype, is biologically heterogeneous and frequently managed with lumpectomy or mastectomy in non-metastatic disease. Although these procedures are essential for oncologic control, they often result in breast deformity, compromised soft tissue integrity, delayed healing, and substantial psychosocial impact, thereby increasing the need for effective reconstructive strategies. This narrative review synthesises current evidence on platelet-rich plasma (PRP) as a regenerative adjunct in breast reconstruction following IDC surgery. Current evidence suggests that PRP may improve graft viability, vascularisation, tissue integration, wound healing, aesthetic outcomes, and patient satisfaction, particularly when used in conjunction with autologous fat grafting. PRP has also shown potential utility in complex reconstructive settings, including radiated or compromised tissue beds, although supporting evidence remains limited. Importantly, available clinical data do not indicate increased rates of locoregional recurrence or metastasis, while emerging preclinical findings suggest that PRP does not promote tumour progression and may exert inhibitory effects under certain experimental conditions. Nevertheless, the evidence base remains heterogeneous, with substantial variation in PRP preparation protocols, leukocyte content, activation methods, study design, and outcome assessment, and long-term oncologic surveillance data are still lacking. Overall, PRP appears to be a promising but evolving adjunctive strategy in post-oncologic breast reconstruction after mastectomy or lumpectomy for IDC. Standardised protocols, larger comparative studies, and long-term follow-up are needed to define its optimal clinical role, durability, and oncologic safety.

Keywords: Breast reconstruction; Invasive ductal carcinoma; Platelet-rich plasma; Regenerative medicine; Soft tissue healing

1. Introduction

Breast cancer remains the leading cause of cancer burden among women worldwide, with an estimated 2.3 million new cases and 685,000 deaths reported globally, and projections indicating a potential 54.7% increase in incidence by 2050.¹ It is recognised as a biologically complex and highly heterogeneous disease comprising multiple subtypes, largely classified based on hormone receptor expression and molecular characteristics.² In addition to its impact on mortality, breast cancer contributes substantially to global disability, accounting for approximately 20 million disability-adjusted life years annually, reflecting both years of life lost and years lived with disease-related morbidity.³ Furthermore, the global disability-adjusted life years burden associated with breast cancer has increased markedly over recent decades, highlighting its growing public health impact and the need for effective treatment and supportive care strategies.³

Invasive ductal carcinoma (IDC) is the most common invasive breast cancer subtype, representing approximately 60–75% of cases, and is characterised by stromal invasion following loss of myoepithelial and basement membrane integrity, reflecting a biologically heterogeneous endpoint arising through multiple evolutionary pathways.⁴ IDC develops when abnormal epithelial cells originating within the mammary ducts infiltrate the surrounding breast stroma, establishing invasive disease. Grossly, IDC typically presents as a firm, irregular mass, often accompanied by a desmoplastic stromal reaction characterised by increased extracellular matrix deposition and collagen synthesis.⁵ This invasive phenotype facilitates subsequent dissemination to regional lymph nodes and distant organs, thereby contributing to metastatic progression.

For non-metastatic IDC, primary surgical management commonly involves lumpectomy (tumour excision) or mastectomy (complete breast tissue removal).⁶ Although these procedures are essential for oncologic control, they frequently result in breast deformity and loss of natural contour, which may impose significant psychological, emotional, social, and cultural challenges for affected patients.⁷ These consequences highlight the importance of reconstructive strategies that not only restore breast form but also enhance tissue healing and long-term functional and aesthetic outcomes. In response to these clinical needs, regenerative medicine has increasingly gained attention as a complementary approach to improve reconstructive results beyond conventional techniques.⁸

Within this evolving landscape, this review explores the potential and clinical success of platelet-rich plasma

(PRP) as a regenerative adjunct for IDC patients who have undergone mastectomy or lumpectomy, with emphasis on its reconstructive and healing benefits rather than as a replacement for primary cancer therapies.

2. Methodology

This review adopted a narrative approach to synthesise current knowledge on the application of PRP as a regenerative therapy in patients with IDC who have undergone mastectomy or lumpectomy. A structured yet non-systematic literature search was conducted using PubMed/MEDLINE, Scopus, and Web of Science, covering publications from January 2020 to August 2025, supplemented by manual screening of reference lists to identify additional relevant studies. The search strategy incorporated both Medical Subject Headings and free-text terms related to breast cancer, PRP, and reconstructive surgery, including “invasive ductal carcinoma,” “breast cancer,” “mastectomy,” “lumpectomy,” “breast reconstruction,” “platelet-rich plasma,” and “regenerative medicine,” with terminology adapted appropriately across databases.

Studies were considered eligible if they discussed the clinical or experimental use of PRP in breast reconstruction, wound healing, soft tissue repair, or fat grafting in the context of breast cancer surgery. The review primarily focused on adult female populations but also included relevant laboratory and translational research where clinically applicable to IDC-associated reconstructive outcomes. Articles not published in English, conference abstracts without full manuscripts, and publications unrelated to PRP use in breast or oncologic reconstructive settings were excluded. Although formal systematic review protocols and quantitative risk-of-bias tools were not employed due to the narrative nature of this review, considerations such as study design, methodological clarity, sample size, duration of follow-up, and reported limitations were taken into account when interpreting evidence quality.

The included literature comprised randomised and non-randomised clinical trials, observational cohort studies, case series, laboratory-based investigations, and scholarly reviews. Findings were synthesised thematically, focusing on the biological rationale and preparation of PRP, its clinical efficacy in breast reconstruction, and its safety profile, including oncologic implications. This narrative approach enables meaningful clinical integration of emerging evidence while acknowledging existing heterogeneity across studies and the evolving landscape of regenerative medicine in breast oncology.

3. Invasive ductal carcinoma: Pathology and treatment

3.1. Genomic and aetiological factors driving invasive ductal carcinoma

3.1.1. Hereditary genetic predisposition

The strongest and most reliable indicators of inherited breast cancer risk are germline mutations in the high-penetrance tumour suppressor genes, *BRCA1* and *BRCA2*. These genes are fundamentally responsible for DNA repair, playing a critical protective role in maintaining genomic integrity. When these genes acquire loss-of-function mutations, this protective mechanism is disabled, directly leading to uncontrolled cell growth and tumourigenesis.⁹ The clinical impact of these mutations is substantial: mutations in *BRCA1* are associated with a 57–65% lifetime risk, while *BRCA2* mutations carry a 45–49% risk.¹⁰ Furthermore, these high-penetrance genetic defects influence the penetrance of other, lower-risk hereditary genes, including *PALB2*, *P53*, *PTEN*, and *CHEK2*.

3.1.2. Somatic drivers and mutational heterogeneity

Beyond inherited susceptibility, the malignant progression of IDC is also driven by acquired somatic mutations affecting key oncogenic and tumour suppressor pathways. Alterations in classical driver genes such as *MAP3K1*, *PTEN*, *TP53*, *GATA3*, and *PIK3CA* play a central role in regulating cellular proliferation, survival signalling, and tumour development, thereby shaping the aggressive biological behaviour of the disease. More recent genomic investigations have further expanded this molecular landscape by identifying additional, less frequently mutated genes, including *CBFB*, *MAP2K4*, *NCOA3*, *RB1*, and *ZNF384*, which contribute to the heterogeneity of IDC. Although these novel alterations occur at lower frequencies, they enhance the current understanding of tumour evolution and represent emerging avenues for future targeted therapeutic strategies.¹¹

3.1.3. The progression from ductal carcinoma in situ to invasion

Invasive ductal carcinoma is often preceded by ductal carcinoma *in situ* (DCIS), its non-invasive precursor, and the transition from DCIS to invasive disease is largely driven by specific genomic alterations that promote malignant progression. A number of key driver mutations, including *TP53*, *PIK3CA*, *CDH1*, and *RAD50*, are shared between both DCIS and IDC, indicating that they play a fundamental role in tumour initiation and remain relevant across the continuum of disease. In addition to these shared alterations, the shift toward invasive behaviour is also characterised by more pronounced genomic instability,

particularly amplification of the *ERBB2* locus and copy number gains across chromosomal regions such as 1q, 8q, 16p, and 17q, along with genomic losses involving regions such as 11q and 16q. Collectively, these molecular events facilitate the progression from localised *in situ* lesions to invasive carcinoma, contributing to the increased aggressiveness and metastatic potential observed in IDC.¹²

3.1.4. External and lifestyle risk factors

In addition to the defined genetic and genomic drivers, the overall risk profile for developing IDC is shaped by numerous non-genetic and environmental factors. Obeagu *et al.*¹³ reviewed multiple risk factors, including a history of the disease (personal and family history), reproductive and hormonal factors, and modifiable lifestyle exposures such as smoking, obesity, and exposure to ionising radiation. An overview of the key genomic alterations, mutations, and factors driving IDC, grouped by classification, is provided in Table 1.

Collectively, these genomic and molecular features are not only central to tumour initiation and progression but also have important implications for reconstructive planning and the safe application of regenerative therapies such as PRP. Tumour biology influences treatment, likelihood of recurrence, and the composition of the post-oncologic tissue microenvironment, all of which are critical considerations when introducing biologically active adjuncts.^{4,12} In particular, genomic instability, oncogenic pathway activation, and tumour–stroma interactions shape the local microenvironment that PRP is intended to modulate through angiogenesis, extracellular matrix remodelling, and growth factor signalling.^{4,11,14} As a result, understanding the genomic landscape of IDC is essential not only for oncologic management but also for informing the timing, appropriateness, and potential biological interactions of PRP-assisted reconstruction.^{8,15,16} This integrative perspective reinforces the need to align regenerative interventions with oncologic stability and multidisciplinary treatment planning.

3.2. Molecular subtypes and surgical management

Surgical procedures (lumpectomy or mastectomy) are primarily indicated in the non-metastatic stages of IDC (Stages I to III). Lumpectomy is often offered with radiation if cosmetically acceptable, while mastectomy may be chosen for larger tumours, family history, or lack of access to radiation therapy.⁶ Once IDC reaches Stage IV, treatment focuses on systemic therapy and palliation; surgery is not recommended except for symptom control.¹⁷

Molecular subtype also matters when determining reconstruction timing because it influences the anticipated

Table 1. Genomic alterations and factors in invasive ductal carcinoma

Mechanistic grouping	Key genes/mutations/factors	Role in IDC pathogenesis	Clinical relevance in IDC management	Implications for PRP and reconstruction	References
High-penetrance hereditary risk	<i>BRCA1</i> <i>BRCA2</i>	Germline loss-of-function mutations disrupt DNA repair, leading to genomic instability and markedly elevated lifetime IDC risk	Identifies high-risk individuals; guides genetic counselling, screening intensity, and prophylactic/surgical decisions	Patients are more likely to undergo bilateral or risk-reducing surgery; PRP-assisted reconstruction may support tissue regeneration and aesthetic recovery in this high-risk population	9,10
Moderate/low-penetrance hereditary risk	<i>PALB2</i> <i>P53</i> <i>PTEN</i> <i>CHEK2</i>	Contributes to hereditary susceptibility through additive mutational burden and compromised tumour-suppressor function	Supports genetic risk stratification and personalised surveillance strategies	These patients may present earlier and undergo reconstructive procedures sooner; PRP may assist soft tissue healing post-intervention	9
Key driver mutations shared in DCIS and IDC	<i>TP53</i> <i>PIK3CA</i> <i>CDH1</i> <i>RAD50</i>	Central regulators of proliferation, DNA repair, and cell adhesion persist from pre-invasive to invasive transition	Highlights early molecular events; informs prognostic understanding and potential targeted therapy direction	Understanding tumour biology supports safe timing of reconstruction and reinforces the importance of oncologic clearance before PRP use	12
Classical IDC development drivers	<i>MAP3K1</i> <i>PTEN</i> <i>TP53</i> <i>GATA3</i> <i>PIK3CA</i>	Promote malignant transformation, survival signalling, and tumour progression	Associated with tumour behaviour, treatment planning, and therapeutic response expectations	Reconstruction decisions may consider tumour biology; PRP use should align with oncologic stability and multidisciplinary clearance	11
Progression-specific genomic alterations (DCIS to IDC transition)	- Amplification: <i>ERBB2</i> (17q21), 1q, 8q, 16p, 17q - Chromosomal loss: 11q, 16q	Represents genomic instability driving invasive transition and aggressive phenotypic change	Supports staging, prognostication, human epidermal growth factor receptor 2-targeted therapy decisions, and treatment planning	Highlights the importance of ensuring oncologic treatment completion and surveillance before applying regenerative interventions such as PRP	12
Novel cancer-associated genes	<i>CBFB</i> <i>MAP2K4</i> <i>NCOA3</i> <i>RB1</i> <i>ZNF384</i>	Recently identified low-frequency alterations contributing to IDC heterogeneity and tumour evolution	Expands the potential future biomarker and therapeutic target landscape	As personalised oncology advances, PRP reconstruction may increasingly integrate with tailored oncologic care pathways	11

Abbreviations: DCIS: Ductal carcinoma *in situ*; IDC: Invasive ductal carcinoma; PRP: Platelet-rich plasma.

sequence of multimodal cancer treatment. While stage and resectability remain the principal determinants of surgical management, subtype helps predict the need for neoadjuvant therapy and the likelihood of postoperative radiotherapy, both of which can affect reconstructive planning. Human epidermal growth factor receptor 2-positive and triple-negative tumours more commonly undergo neoadjuvant systemic treatment because of their aggressive biology and treatment responsiveness, whereas some hormone receptor-positive/human epidermal growth factor receptor 2-negative cancers may be managed with more direct progression to surgery in selected cases. These distinctions are important because immediate reconstruction may be less suitable when adjuvant radiotherapy is highly likely or when surgical timing depends on response to preoperative therapy. In this context, molecular subtype indirectly influences whether immediate, delayed-immediate, or delayed reconstruction is the most oncological and surgically appropriate strategy.^{4,6,17}

3.3. Emerging diagnostic and prognostic tools for early detection of breast cancer

Early detection of breast cancer remains critical for improving survival outcomes, enabling less extensive surgical intervention, and facilitating more effective reconstructive planning. Recent advances in imaging, nanoscale diagnostics, and computational technologies have significantly enhanced the sensitivity and specificity of early cancer detection, while also providing deeper insight into tumour biology.

Magnetic resonance imaging (MRI) is among the most sensitive modalities for breast cancer detection, particularly in high-risk populations. Contrast-enhanced MRI enables functional assessment of tumour angiogenesis and vascularity, allowing detection of lesions that may be occult on conventional imaging.¹⁸ MRI has demonstrated sensitivity exceeding 90% in screening settings and is particularly effective in identifying early-stage and node-negative cancers.¹⁹ Furthermore, MRI can detect additional multifocal or contralateral disease not visible on mammography or ultrasound, thereby improving preoperative planning.²⁰ However, limitations include high cost, limited accessibility, and the potential for overestimation of tumour size, which may lead to overtreatment or unnecessary surgical intervention.²⁰

Beyond conventional imaging, emerging diagnostic approaches have highlighted the importance of tumour biomechanics and microenvironmental properties in cancer progression. Techniques such as atomic force microscopy and fluorescence lifetime imaging microscopy

have demonstrated that malignant breast cells exhibit altered viscoelasticity, cytoskeletal organisation, and membrane properties compared with normal cells.^{21,22}

These biomechanical changes are increasingly recognised as key features of tumour biology, influencing processes such as cell migration, invasion, and interaction with the extracellular matrix.

Artificial intelligence and machine learning approaches have been further applied to facilitate analysis of complex nanoscale and biomechanical datasets, supporting the identification of diagnostic and prognostic biomarkers.²³ However, despite their mechanistic and diagnostic potential, these technologies remain largely confined to research settings and have limited direct application in routine clinical practice.

In the context of post-oncologic breast reconstruction, these advances are relevant primarily in illustrating how tumour biology and tissue microenvironment may influence reconstructive planning and healing dynamics. Nevertheless, detailed discussion of these diagnostic modalities lies beyond the principal scope of this review, which focuses on regenerative strategies such as PRP.

In addition to these advanced modalities, integrated diagnostic approaches combining imaging, biosensors, and wearable technologies are emerging to improve early detection and patient monitoring. These multimodal systems aim to enhance accessibility, enable continuous monitoring, and support personalised screening strategies.²⁴ Nevertheless, limitations such as variable sensitivity, false positive rates, and challenges in clinical validation continue to constrain widespread adoption.

Collectively, these emerging diagnostic and prognostic tools reflect a shift toward more precise, biologically informed, and technology-driven approaches to breast cancer detection. Importantly, earlier and more accurate identification of malignancy not only improves oncologic outcomes but also facilitates optimised surgical planning and reconstructive strategies. In this context, improved characterisation of tumour biology and microenvironment may further inform the safe and effective integration of regenerative adjuncts such as PRP in post-oncologic breast reconstruction. The multistep interplay between genetic susceptibility, somatic genomic alterations, tumour evolution, and microenvironmental reprogramming that underpins the initiation and progression of IDC is illustrated in [Figure 1](#).

4. Platelet-rich plasma in breast reconstruction

To provide an overview of the biological rationale, clinical

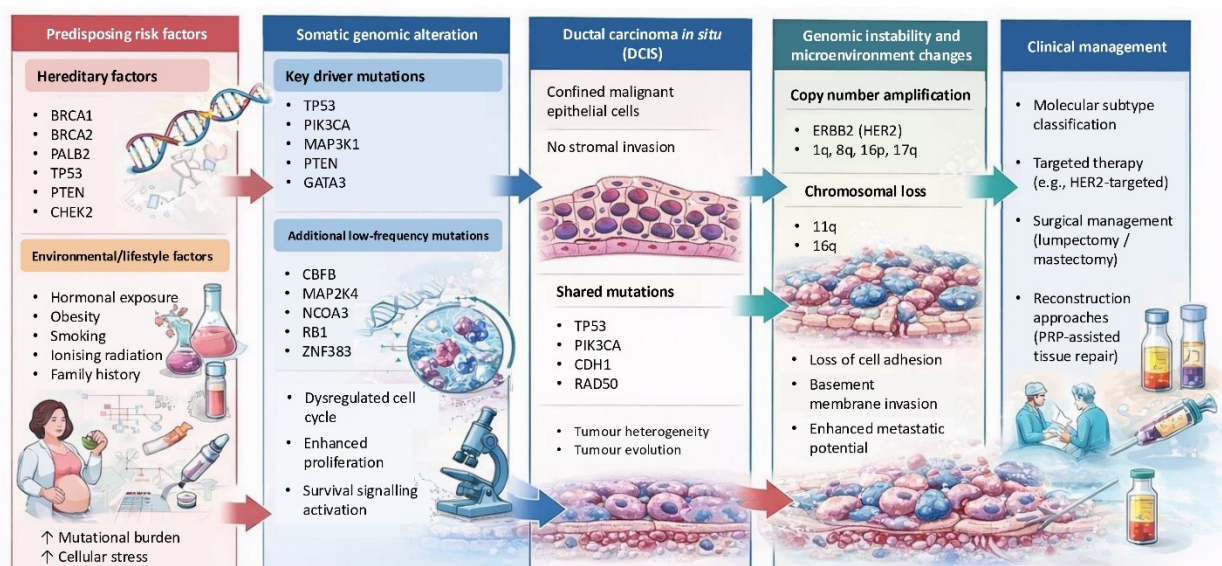


Figure 1. Schematic overview of the genetic, molecular, and microenvironmental mechanisms underlying the initiation and progression of invasive ductal carcinoma (IDC). The figure illustrates the progression from predisposing risk factors, including hereditary (e.g., *BRCA1*, *BRCA2*, *TP53*, *PTEN*) and environmental influences, to somatic genomic alterations characterised by key driver mutations (e.g., *TP53*, *PIK3CA*, *PTEN*, *GATA3*) and dysregulated proliferation and survival signalling. These changes contribute to the development of ductal carcinoma *in situ* (DCIS), which is characterised by confined malignant epithelial cells without stromal invasion but with emerging tumour heterogeneity and clonal evolution. The transition to invasive disease is mediated by epithelial–mesenchymal transition (EMT), extracellular matrix degradation, and tumour–stroma interactions. Progressive genomic instability, including copy number alterations (e.g., *ERBB2*) and chromosomal loss, together with tumour microenvironment changes such as loss of cell adhesion, basement membrane invasion, and enhanced metastatic potential, further drive disease progression. These biological processes inform clinical management strategies, including molecular subtype classification, targeted therapy, surgical intervention, and reconstructive approaches such as platelet-rich plasma (PRP)-mediated microenvironment modulation and tissue repair. Figure created using Adobe Illustrator (Adobe Inc., USA).

outcomes, and safety considerations underpinning the use of PRP in post-oncologic breast reconstruction, [Figure 2](#) summarises the key mechanisms of PRP preparation and action, its reported reconstructive benefits, and current evidence regarding oncologic safety. This conceptual framework situates PRP as a regenerative adjunct rather than a replacement for established reconstructive modalities.

4.1. Mechanism and preparation

Platelet-rich plasma treatment is a regenerative medical therapy that utilises a concentrated autologous blood product enriched with platelets, growth factors, cytokines, and chemokines to support tissue repair and healing.¹⁶ PRP is derived from peripheral blood through centrifugation-based separation, producing a platelet-rich fraction with platelet concentrations typically three- to five-fold higher than baseline whole blood levels, depending on the preparation protocol employed.²⁵ This biologically active concentrate is subsequently injected into surgical sites or combined with graft materials to promote angiogenesis, extracellular matrix remodelling, and soft tissue regeneration, while potentially reducing complications

such as oil cyst formation, calcification, and fat necrosis.^{16,25}

From a mechanistic perspective, the regenerative effects of PRP are primarily mediated through platelet activation and degranulation, leading to the localised release of bioactive mediators stored within platelet α -granules and extracellular vesicles. These include platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), transforming growth factor- β (TGF- β), fibroblast growth factors, and epidermal growth factor, which collectively regulate chemotaxis, fibroblast proliferation, collagen synthesis, angiogenesis, and tissue remodelling. In addition to growth factor signalling, PRP has been shown to exert immunomodulatory effects by influencing macrophage polarisation and inflammatory cytokine balance, thereby creating a regenerative microenvironment conducive to wound healing and graft integration.¹⁴

Platelet-rich plasma preparation relies on differential centrifugation techniques that separate blood components according to density. Commonly used methods include double-spin protocols and buffy coat-based techniques, each yielding PRP products with differing platelet concentrations, leukocyte content, and plasma composition.¹⁴ Following centrifugation, the platelet-rich

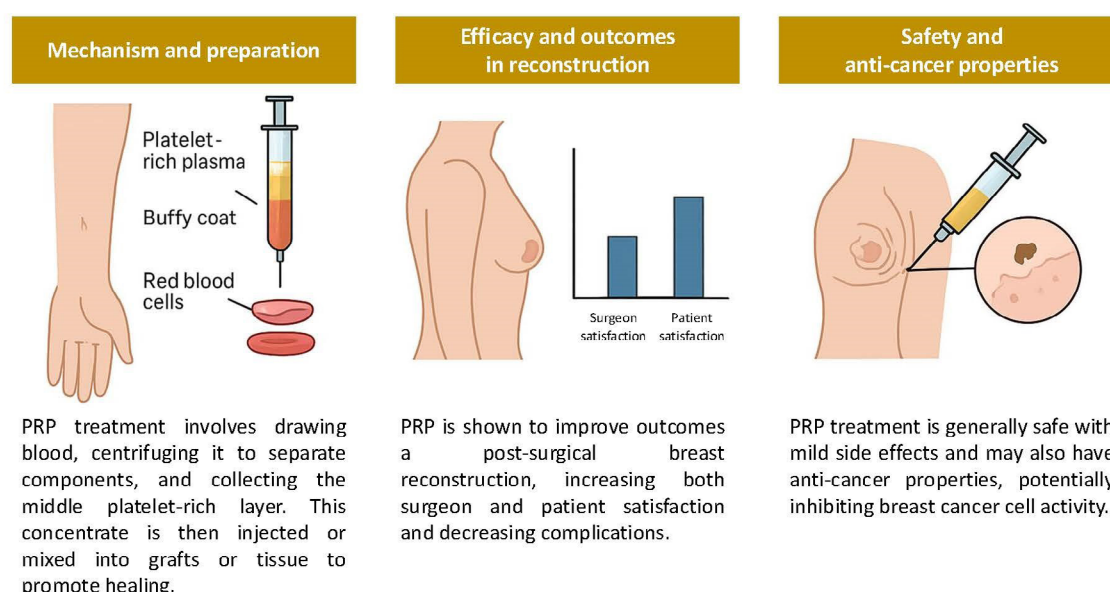


Figure 2. Platelet-rich plasma (PRP) in post-oncologic breast reconstruction. Schematic overview illustrating PRP preparation through blood centrifugation and platelet concentration, reported clinical benefits of PRP-assisted reconstruction, including improved tissue healing and patient satisfaction, safety considerations, and emerging evidence regarding oncologic neutrality and potential anti-cancer effects. PRP is positioned as a regenerative adjunct to established reconstructive techniques following mastectomy or lumpectomy. Figure created using Adobe Illustrator (Adobe Inc., USA).

layer, which is typically located between platelet-poor plasma and erythrocytes, is aspirated and prepared for clinical application. PRP may be administered in liquid form or activated exogenously using calcium chloride or thrombin to induce immediate platelet degranulation, although endogenous activation upon tissue contact is also widely reported.¹⁵

A major source of variability in PRP therapy arises from differences in cellular composition, particularly leukocyte concentration. PRP is commonly categorised into leukocyte-poor PRP and leukocyte-rich PRP, each exhibiting distinct biological profiles. Experimental and clinical evidence suggest that leukocyte-rich PRP may elicit a stronger early inflammatory response and promote angiogenesis, whereas leukocyte-poor PRP may reduce excessive inflammation while preserving growth factor-mediated regenerative effects.^{26,27} These differences are clinically relevant, as leukocyte content may influence both healing kinetics and postoperative tissue response, underscoring the importance of tailoring PRP formulation to specific reconstructive contexts.

Despite increasing clinical adoption, there remains no universally accepted standard for PRP preparation or classification. Multiple classification systems have been proposed to account for platelet concentration, leukocyte content, red blood cell contamination, and activation status; however, heterogeneity in preparation protocols

continues to limit direct comparison across studies. This lack of standardisation has been consistently highlighted in plastic and reconstructive surgery literature as a key challenge in interpreting PRP efficacy and reproducibility.¹⁵

Overall, PRP preparation aims to concentrate platelets and their associated bioactive mediators while modulating cellular composition to optimise regenerative potential. In post-oncologic breast reconstruction, these mechanistic and preparative characteristics position PRP as a biologically active adjunct capable of enhancing tissue healing and graft survival, while emphasising the need for careful protocol selection and standardised reporting in future clinical studies.

4.2. Efficacy and outcomes in reconstruction

Current evidence suggests that PRP may meaningfully enhance outcomes in breast reconstruction following lumpectomy or mastectomy; however, the magnitude and consistency of this benefit must be interpreted with caution. PRP has been most extensively evaluated as an adjunct to autologous fat grafting, where it appears to enhance graft viability, vascularisation, and integration into recipient tissue. Shaaban *et al.*²⁸ demonstrated that patients receiving PRP-enriched fat grafts following breast-conserving surgery experienced significantly higher surgeon- and patient-reported satisfaction, along with reduced complication rates such as fat necrosis. These findings align with broader

reconstructive surgery literature reporting improved wound healing, decreased postoperative discomfort, and favourable cosmetic outcomes when PRP is incorporated into clinical practice.^{15,16,25} As illustrated in Figure 2, these reconstructive benefits are reflected in improved tissue integration and higher surgeon- and patient-reported satisfaction, positioning PRP as a biological enhancer rather than a standalone reconstructive modality.

Despite these promising findings, much of the current evidence is derived from relatively small, single-centre studies with short- to intermediate-term follow-up periods, limiting the ability to draw firm conclusions regarding long-term reconstructive durability. A major challenge in PRP research is methodological heterogeneity, including variation in platelet concentration, leukocyte content, activation techniques, delivery methods, and timing of application.^{15,25,29} This variability complicates direct comparison across studies and likely contributes to differences in reported outcomes. Furthermore, although many studies report favourable results, only a subset utilise standardised aesthetic assessment tools or validated patient-reported outcome measures, which restricts robust comparative evaluation. Gentile and Garcovich³⁰ further emphasise that the clinical effects of PRP are highly dependent on preparation characteristics, reinforcing the need for standardised protocols.

Outcomes reported across studies also vary according to study design and sample size. For example, the randomised controlled study by Shaaban *et al.*²⁸ demonstrated significantly improved graft retention, reduced complication rates, and higher patient satisfaction when PRP was combined with autologous fat grafting compared with conventional lipoinjection alone. In contrast, several observational and review-based analyses have reported more modest or heterogeneous clinical improvements, often limited by smaller patient cohorts and inconsistent outcome measurements. Systematic reviews in plastic and reconstructive surgery similarly highlight that while PRP appears to enhance angiogenesis and tissue regeneration, the magnitude of clinical benefit remains difficult to quantify due to differences in platelet concentration, leukocyte content, activation methods, and delivery techniques across studies. These methodological variations likely contribute to discrepancies in reported outcomes and underscore the need for standardised PRP preparation protocols and larger controlled clinical trials.

Taken together, the literature supports PRP as a promising reconstructive adjunct, particularly in fat grafting applications following IDC surgery. However, the field remains characterised by evolving and heterogeneous evidence. Larger, multicentre, methodologically rigorous

trials with standardised PRP preparation and outcome assessment frameworks are needed to confirm clinical benefit, define optimal protocols, and determine whether improvements are sustained over the long term. To contextualise current evidence and highlight practical use cases, Table 2 summarises the principal clinical applications of PRP in post-oncologic breast reconstruction, including methods of application, intended regenerative benefits, reported outcomes, and key limitations.

An additional consideration when interpreting current PRP evidence is the potential for sex- and age-related bias in the available breast reconstruction literature. Most studies in this field are conducted in adult women, reflecting the epidemiology of breast cancer and reconstructive practice, whereas male breast cancer patients are rarely represented. As a result, current conclusions regarding PRP efficacy and safety may not be readily generalisable across sexes. Age is also a potentially important source of bias, as reconstructive outcomes may be influenced by age-related differences in wound-healing capacity, tissue vascularity, and collagen remodelling. Older patients may have more fragile tissue quality and slower healing, whereas younger patients may demonstrate different regenerative responses and cosmetic expectations. These biological differences in skin aging and repair mechanisms have been widely documented in dermatologic and wound-healing literature.³¹ However, most currently available PRP studies in breast reconstruction involve relatively small cohorts and predominantly female populations,^{28,32} making it difficult to determine whether PRP confers differential benefit across demographic subgroups. Future trials should therefore incorporate subgroup analyses and standardised demographic reporting to better define which patients are most likely to benefit from PRP-assisted reconstruction.

4.3. Comparative and contextual considerations in breast reconstruction

When evaluating the role of PRP in breast reconstruction after mastectomy or lumpectomy, it is essential to position it within the broader reconstructive landscape. Conventional reconstructive approaches, such as implant-based reconstruction, autologous tissue flaps, acellular dermal matrix (ADM) reinforcement, and standard fat grafting, remain widely utilised and clinically validated. However, each carries inherent limitations. Implant-based reconstruction may be associated with complications, including capsular contracture, implant loss, infection, and the need for revision surgery, particularly in complex or radiated fields.^{33,34} Fat grafting, although increasingly employed for contour correction and volume enhancement, can be limited by graft resorption, volume unpredictability, and fat necrosis.³⁵ ADM-supported reconstruction

Table 2. Clinical applications of platelet-rich plasma in post-oncologic breast reconstruction

Clinical context	Study design	PRP application method	Intended regenerative benefits	Reported clinical outcomes	Key limitations	References
Autologous fat grafting following lumpectomy or mastectomy	Randomised controlled clinical trial	PRP mixed with harvested autologous fat prior to injection	Enhance angiogenesis, improve graft survival, and reduce fat necrosis	Improved graft retention, fewer complications, and higher surgeon- and patient-reported satisfaction	Single-centre study; moderate sample size	28
Correction of post-lumpectomy contour deformities	Prospective clinical study	Local PRP injection alone or combined with fat grafting	Improve soft tissue quality and wound healing	Improved breast symmetry, faster healing, and favourable cosmetic outcomes	Limited standardised aesthetic assessment tools	28
Reconstruction in radiated breast tissue	Narrative review/literature synthesis	PRP injected into fibrotic or hypovascular tissue beds	Mitigate radiation-induced fibrosis and enhance tissue pliability	Improved tissue elasticity and wound healing are suggested in clinical reports	Sparse oncologic-specific studies; evidence largely extrapolated	14,30
Implant-based breast reconstruction	Systematic review of clinical studies	PRP is applied to the implant pocket or surgical incision	Reduce wound complications and enhance local healing	Reported reduction in seroma formation and improved incision healing in some studies	Heterogeneous study protocols; limited randomised trials	15,29
Delayed breast reconstruction after completion of oncologic therapy	Narrative literature review	PRP used as tissue pre-conditioning prior to reconstruction	Enhance vascular bed and soft tissue regeneration	Potential improvement in graft viability and tissue integration	Limited direct clinical validation in breast oncology populations	14,16

Notes: Study designs include randomised controlled trials, prospective clinical studies, systematic reviews, and narrative reviews. Variability in PRP preparation protocols, leukocyte content, platelet concentration, and activation methods contributes to heterogeneity across reported outcomes. Abbreviation: PRP: Platelet-rich plasma.

contributes structural support and improved implant coverage, but it also introduces considerations related to cost, risk of seroma, and potential for infection.³⁶

Within this reconstructive context, PRP does not act as a replacement for established reconstructive modalities but rather as a biological adjunct intended to enhance their outcomes. Unlike structural interventions such as implants or ADM, PRP introduces a regenerative, growth factor-rich microenvironment that may support angiogenesis,

improve soft tissue healing, and enhance fat graft viability. Compared to fat grafting alone, PRP-enriched grafting has the potential to improve graft survival and reduce complication rates, particularly in patients with compromised tissue quality or reduced vascularity.^{15,16,28} PRP's autologous nature also contributes to favourable biocompatibility and generally good tolerability.

However, unlike standardised implant systems or ADM protocols, PRP currently lacks universally accepted

preparation and application guidelines. Variability in platelet concentration, leukocyte content, activation technique, delivery timing, and operator experience contributes to heterogeneity in reported outcomes.^{25,29,30} Furthermore, while evidence supporting implants, flaps, and ADM is extensive and conducted long-term, PRP literature remains comparatively new, with many studies characterised by smaller cohorts and shorter follow-up durations.

Accordingly, PRP is best understood as an evolving adjunctive regenerative strategy positioned within an established reconstructive framework rather than a replacement modality. Its use is most appropriate where biological enhancement may meaningfully improve soft tissue healing, graft survival, aesthetic contouring, or patient satisfaction. Optimal integration requires individualised clinical decision-making, multidisciplinary collaboration, and continued accumulation of comparative evidence to determine where PRP offers clear advantages over conventional approaches and which patient populations are most likely to benefit.

4.4. Safety and anti-cancer properties

Platelet-rich plasma generally demonstrates a favourable safety profile in breast reconstruction, with most reported adverse effects being mild, transient, and primarily related to localised injection reactions such as swelling, bruising, or discomfort. Serious complications remain uncommon when appropriate sterile technique and careful patient selection are applied.¹⁵ Importantly, available clinical literature does not indicate an increased risk of local recurrence or distant metastasis associated with PRP use in post-oncologic breast reconstruction settings, providing preliminary reassurance regarding oncologic safety. These safety considerations, summarised in [Figure 2](#), reflect current clinical observations and ongoing investigation into the biological interaction between PRP and the post-oncologic breast microenvironment.

Beyond procedural safety, an equally critical consideration is whether PRP may biologically influence tumour behaviour. The theoretical concern arises from the presence of multiple growth factors within PRP, including PDGF, VEGF, TGF- β , and insulin growth factor 1, which are essential for tissue regeneration and angiogenesis but could, in principle, interact with oncogenic pathways. However, current evidence does not substantiate this theoretical risk in clinical practice. Conversely, emerging laboratory research has reported potentially protective effects; Han *et al.*³⁷ demonstrated that PRP inhibited proliferation, migration, and invasion in MDA-MB-231 and 4T1 breast cancer cell lines, suggesting that PRP does

not necessarily promote tumourigenesis and may, under certain biological conditions, exert inhibitory influences.

To better contextualise this uncertainty, it is important to consider the potential interaction between PRP-derived growth factors and tumour microenvironmental regulatory systems. PRP contains bioactive mediators such as TGF- β , VEGF, PDGF, and insulin growth factor 1, which are known to influence angiogenesis, extracellular matrix remodelling, and stromal signalling. These processes overlap with pathways that regulate cancer stem cell (CSC) behaviour within the tumour microenvironment.

Cancer stem cells are maintained within specialised niches composed of stromal cells, extracellular matrix components, and soluble cytokines, where signalling pathways such as TGF- β , inflammatory cytokine cascades (e.g., interleukin 6/signal transducer and activator of transcription 3), chemokine axes (e.g., C-X-C motif chemokine ligand 12/C-X-C motif chemokine receptor 4), and hypoxia collectively regulate stemness, survival, and metastatic potential.³⁸⁻⁴¹

While these pathways are well-established regulators of CSC biology, their modulation in the context of PRP exposure remains largely theoretical.

Accordingly, it has been hypothesised that a regenerative microenvironment could, in principle, influence residual tumour cell behaviour through niche-level signalling interactions. However, there is currently no direct clinical or translational evidence demonstrating that PRP promotes CSC expansion, niche reconstitution, or increased oncologic risk in breast reconstruction. In contrast, some preclinical data suggest neutral or even inhibitory effects of PRP on tumour cell proliferation and invasion.³⁷ Therefore, any potential interaction between PRP and CSC-related pathways should be interpreted as biologically plausible but unproven and warrants further investigation rather than definitive concern.

Despite these encouraging signals, caution remains warranted. Much of the anti-cancer evidence is derived from *in vitro* or preclinical models that do not fully replicate the complex stromal, immune, and microenvironmental interactions present in human breast cancer. Moreover, PRP preparations are inherently heterogeneous, with variations in platelet concentration, leukocyte content, activation methods, and delivery protocols potentially influencing biological behaviour.^{25,29} This lack of standardisation introduces uncertainty, particularly when considering long-term oncologic implications. Additionally, longitudinal surveillance data following PRP-assisted reconstruction are still limited, and while existing evidence is reassuring, the absence of demonstrated harm

cannot yet be equated to definitive proof of long-term oncologic neutrality.

Accordingly, PRP should be regarded as a promising yet biologically active adjunct whose oncologic neutrality has not been definitively established in post-oncologic breast reconstruction rather than an oncologically validated standard of care. Its application is best guided by multidisciplinary decision-making, explicit patient counselling regarding benefits and uncertainties, and careful follow-up. Continued prospective research, ideally incorporating standardised PRP protocols and long-term oncologic monitoring, remains essential to conclusively establish safety and clarify whether PRP confers any additional protective biological effects beyond its reconstructive benefits.

4.4.1. Sex- and age-related considerations

Interpretation of PRP outcomes in post-oncologic breast reconstruction should also take into account potential demographic bias, particularly with respect to sex and age. The available literature is overwhelmingly derived from adult female populations, which is understandable given the higher incidence of breast cancer in women, while male breast cancer accounts for only a small proportion of all cases. Consequently, male patients remain largely underrepresented in reconstructive and regenerative breast surgery studies, including investigations of PRP-assisted reconstruction, which limits the generalisability of current findings across sexes.¹

Age may likewise influence both the biological effect of PRP and the clinical success of reconstruction. Differences in platelet activity, angiogenic capacity, tissue vascularity, wound-healing kinetics, hormonal milieu, and the prevalence of comorbidities may affect regenerative responses following reconstructive procedures. Aging is known to alter skin structure, collagen turnover, and fibroblast activity, which can impair tissue repair and prolong wound-healing processes.³¹ In addition, older patients are more likely to experience treatment-related fibrosis, reduced tissue elasticity, and other factors that may influence graft retention and postoperative recovery.

Despite these plausible influences, most published studies evaluating PRP-assisted fat grafting or reconstructive outcomes involve relatively small cohorts and rarely report results stratified by demographic variables such as age or sex. For example, recent analyses of PRP-assisted breast reconstruction include limited sample sizes and predominantly female patient populations, restricting the ability to perform subgroup analyses.^{28,32} This represents an important limitation of the current evidence base and highlights the need for future studies to

report age-disaggregated and sex-inclusive outcomes more systematically.

4.5. Clinical indications and workflow for platelet-rich plasma use in post-oncologic breast reconstruction

The clinical application of PRP in post-oncologic breast reconstruction should be guided by clearly defined indications, appropriate patient selection, and structured integration within established reconstructive workflows. Rather than serving as a standalone intervention, PRP functions as a biological adjunct intended to enhance tissue healing, graft survival, and aesthetic outcomes following mastectomy or lumpectomy for IDC, as supported by current regenerative and reconstructive surgery literature.^{14,17}

4.5.1. Patient selection and indications

Platelet-rich plasma-assisted reconstruction is most appropriately considered in patients who have completed definitive oncologic treatment and demonstrate no evidence of active disease. Ideal candidates include individuals undergoing autologous fat grafting for contour correction, volume restoration, or symmetry enhancement following breast-conserving surgery or mastectomy. Patients with compromised soft tissue quality, such as those with prior radiotherapy exposure, thin mastectomy flaps, delayed wound healing, or previous reconstructive complications, may derive particular benefit from the angiogenic and regenerative properties of PRP.^{16,28}

Additional clinical indications include correction of post-lumpectomy contour deformities, optimisation of implant-based reconstruction through enhanced incision healing, and tissue pre-conditioning in delayed reconstruction settings. In these contexts, PRP may contribute to improved vascularisation, reduced inflammatory sequelae, and enhanced integration of grafted or reconstructed tissues. However, PRP use should be avoided in patients with unresolved oncologic treatment plans, active locoregional disease, or uncertain disease status, reinforcing the importance of multidisciplinary clearance prior to application.¹⁷

4.5.2. Timing and mode of application

Platelet-rich plasma may be incorporated at different stages of the reconstructive pathway depending on clinical objectives. In immediate reconstruction, PRP is commonly applied intraoperatively, either through combination with autologous fat grafts or localised delivery to surgical beds and incision sites. In delayed reconstruction, PRP may be administered during secondary contouring procedures or used as a tissue pre-conditioning strategy to improve

local vascularity and tissue quality prior to definitive reconstruction.^{15,16}

The mode of PRP delivery varies according to the reconstructive context. For fat grafting, PRP is typically mixed with processed adipose tissue to enhance graft viability and reduce fat necrosis. In implant-based reconstruction, PRP may be applied to the implant pocket or incision margins to support wound healing. In radiated or fibrotic tissues, localised PRP injection may be used to improve tissue pliability and microvascular perfusion, although supporting evidence in oncologic breast reconstruction remains limited and largely extrapolated from broader reconstructive applications.^{15,25,30}

4.5.3. Workflow integration and multidisciplinary oversight

Effective integration of PRP into post-oncologic breast reconstruction requires a structured workflow supported by multidisciplinary collaboration. Preoperative evaluation should include confirmation of oncologic clearance, assessment of tissue quality, and alignment of reconstructive goals with cancer surveillance strategies. Coordination between breast surgeons, plastic and reconstructive surgeons, and oncologists is essential to ensure appropriate timing and oncologic prudence.^{17,34}

Platelet-rich plasma preparation should follow standardised institutional protocols with explicit documentation of platelet concentration, leukocyte content, activation method, and volume administered. Intraoperative handling must adhere to strict sterile technique, and postoperative follow-up should assess both reconstructive outcomes and routine oncologic surveillance parameters, including wound healing, graft retention, complication rates, and patient-reported satisfaction.^{15,25}

4.5.4. Patient counselling and ethical considerations

Given the evolving nature of evidence, comprehensive patient counselling is a critical component of PRP-assisted reconstruction. Patients should be informed that PRP is an adjunctive regenerative strategy intended to enhance, but not replace, conventional reconstructive techniques. Counselling should address anticipated benefits, procedural uncertainties, variability in preparation protocols, and the current state of oncologic safety evidence. Explicit informed consent is particularly important in post-cancer settings, where theoretical concerns regarding growth factor signalling may arise despite reassuring clinical and experimental data.^{15,37}

4.5.5. Clinical positioning of platelet-rich plasma in reconstruction pathways

Overall, PRP is best positioned as a selective adjunct within established breast reconstruction pathways, offering biological enhancement in clinical scenarios where tissue healing, vascularity, or graft integration may be compromised. Its most consistent utility has been reported in autologous fat grafting and complex reconstructive settings, while broader applications require further validation. This framework represents a narrative synthesis of available clinical and experimental evidence summarised in preceding sections and [Table 2](#), rather than formal guideline recommendations.

5. Future recommendations

Although current evidence supports the potential regenerative benefits of PRP in post-mastectomy and post-lumpectomy reconstruction, several priority areas require attention to advance its clinical integration. Future research should prioritise the design of large, multicentre, methodologically rigorous clinical trials with standardised protocols to overcome the heterogeneity that characterises current literature. This includes uniform definitions of PRP composition, standardised platelet thresholds, leukocyte concentration reporting, activation protocols, delivery techniques, and timing of administration. Establishing internationally accepted preparation frameworks would significantly improve comparability between studies and enhance reproducibility and clinical confidence.

Longitudinal follow-up is equally critical. Most available evidence reflects short- to intermediate-term outcomes; therefore, long-term studies are needed to determine the durability of aesthetic and functional benefits, volume retention in fat grafting, complication reduction, and sustained patient satisfaction. Crucially, prospective surveillance data evaluating local recurrence, disease progression, and overall oncologic outcomes following PRP-assisted reconstruction are needed to definitively establish oncologic safety. Integration of oncologic endpoints into PRP clinical trials would strengthen the evidence base guiding safe clinical use.

Comparative effectiveness research is also needed. Future studies should compare PRP-assisted reconstruction against conventional fat grafting, implant-based reconstruction, ADM-reinforced techniques, and emerging regenerative strategies to identify clinical scenarios in which PRP provides clear additive value. Parallel evaluation of patient-reported outcome measures,

quality-of-life indices, and psychosocial recovery should accompany surgical metrics to ensure patient-centred evaluation of benefit. Cost-effectiveness analyses will further support health-system decision-making, particularly as PRP availability and accessibility vary across healthcare environments.

Finally, future translational research should continue exploring PRP's biological interaction with the post-oncologic breast microenvironment, including its influence on tissue healing, immune modulation, and angiogenic signalling. In particular, further studies may evaluate whether PRP modulates pathways associated with CSC regulation within the surgical niche, such as cytokine-mediated signalling, chemokine axes, stromal activation, and cellular plasticity.^{38,40,42} As personalised oncology continues to expand, understanding whether PRP responsiveness varies by molecular subtype, prior radiation exposure, tissue vascularity, or genetic profile may allow patient-specific optimisation. Overall, a coordinated multidisciplinary research agenda integrating oncologic, reconstructive, biomedical, and patient-centred perspectives is essential to fully realise the regenerative potential of PRP while ensuring long-term safety and clinically meaningful benefit.

6. Conclusion

Invasive ductal carcinoma remains a major global health burden. While mastectomy and lumpectomy are essential for disease control, they frequently result in physical and psychosocial consequences that necessitate effective reconstruction. Current evidence suggests that PRP is a promising adjunctive regenerative therapy, particularly in enhancing fat graft viability, supporting soft tissue healing, and improving aesthetic satisfaction in selected post-oncologic breast reconstruction settings.

Although clinical outcomes are encouraging and early safety data, including emerging anti-tumour laboratory findings, are reassuring, evidence remains heterogeneous, and long-term oncologic surveillance is still limited. At present, PRP should therefore be viewed as a valuable but evolving adjunct rather than a replacement for established reconstructive modalities. Its use is best guided by multidisciplinary decision-making and appropriate patient selection, while future rigorously designed studies are needed to standardise protocols, define long-term outcomes, and clarify its definitive role in post-IDC breast reconstruction.

Acknowledgments

None.

Funding

None.

Conflict of interest

The authors declare that they have no competing interests.

Author contributions

Conceptualization: Hurrycane Thierry Cabaron, Cyra Mae Lazarito

Writing-original draft: Hurrycane Thierry Cabaron, Cyra Mae Lazarito, Nancy Choon-Si Ng

Writing-review & editing: Rebecca Shin-Yee Wong, Nancy Choon-Si Ng, Onikka Bernice E. Jara, Jomar Adams Ganding

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data

Not applicable.

References

1. Zhang Y, Ji Y, Liu S, *et al.* Global burden of female breast cancer: new estimates in 2022, temporal trend and future projections up to 2050 based on the latest release from GLOBOCAN. *J Natl Cancer Cent.* 2025;5(3):287-296.
doi: 10.1016/j.jncc.2025.02.002
2. Orrantia-Borunda E, Anchondo-Núñez P, Acuña-Aguilar LE, Gómez-Valles FO, Ramírez-Valdespino CA. Subtypes of Breast Cancer. In: *Breast Cancer*. Exon Publications; 2022:31-42.
doi: 10.36255/exon-publications-breast-cancer-subtypes
3. Lan T, Lu Y, He J, *et al.* Global, regional, national burden and risk factors in female breast cancer from 1990 to 2021. *iScience.* 2024;27(10):111045.
doi: 10.1016/j.isci.2024.111045
4. Wang J, Li B, Luo M, *et al.* Progression from ductal carcinoma in situ to invasive breast cancer: molecular features and clinical significance. *Signal Transduct Target Ther.* 2024;9(1):81.
doi: 10.1038/s41392-024-01779-3
5. Scholl AR, Flanagan MB. Educational Case: Invasive Ductal Carcinoma of the Breast. *Acad Pathol.* 2020;7:2374289519897390.
doi: 10.1177/2374289519897390

6. Trayes KP, Cokenakes SEH. Breast Cancer Treatment. *Am Fam Physician*. 2021;104(2):171-178.
7. Mishra A, Nair J, Sharan AM. Coping in Post-Mastectomy Breast Cancer Survivors and Need for Intervention: Systematic Review. *Breast Cancer (Auckl)*. 2023;17:11782234231209126.
doi: 10.1177/11782234231209126
8. Halim NSSA, Wisasi M, Rungsisiwut R, *et al*. Revolutionizing Breast Cancer Treatment: The Role of Regenerative Medicine. *Biomed Res Ther*. 2025;12(6):7489-7498.
doi: 10.15419/tjcsrg93
9. Casaubon JT, Kashyap S, Regan JP. BRCA1 and BRCA2 Mutations. In: *StatPearls*. StatPearls Publishing; 2025. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK470239/> [Last accessed on January 2, 2026].
10. Zafar M, Krishnakumar M, Reddy A. Impact of Early Testing and Analysis of Germline Genetic Mutation in Patients with Breast Cancer: A Single Institution Experience. *J Cancer Prev*. 2025;30(1):41-46.
doi: 10.15430/JCP.25.001
11. Yu T, Choi KP, Chen ES, Zhang L. Stage-specific protein-domain mutational profile of invasive ductal breast cancer. *BMC Med Genomics*. 2020;13(S10):149.
doi: 10.1186/s12920-020-00777-y
12. Trinh A, Gil Del Alcazar CR, Shukla SA, *et al*. Genomic Alterations during the In Situ to Invasive Ductal Breast Carcinoma Transition Shaped by the Immune System. *Mol Cancer Res*. 2021;19(4):623-635.
doi: 10.1158/1541-7786.MCR-20-0949
13. Obeagu EI, Obeagu GU. Breast cancer: A review of risk factors and diagnosis. *Medicine (Baltimore)*. 2024;103(3):e36905.
doi: 10.1097/MD.00000000000036905
14. Wu WS, Chen LR, Chen KH. Platelet-Rich Plasma (PRP): Molecular Mechanisms, Actions and Clinical Applications in Human Body. *Int J Mol Sci*. 2025;26(21):10804.
doi: 10.3390/ijms262110804
15. Hasiba-Pappas SK, Tuca AC, Luze H, *et al*. Platelet-Rich Plasma in Plastic Surgery: A Systematic Review. *Transfus Med Hemother*. 2022;49(3):129-142.
doi: 10.1159/000524353
16. Giannakaki AG, Stylianaki A, Giannakaki MN, *et al*. Stem-Cell-Assisted Lipotransfer and Platelet-Rich Plasma in Breast Reconstruction: A Literature Review. *Aesthetic Plast Surg*. 2025;49(20):5764-5773.
doi: 10.1007/s00266-025-04921-w
17. Menon G, Alkabban FM, Ferguson T. Breast Cancer. In: *StatPearls*. StatPearls Publishing; 2025. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK482286/> [Last accessed on January 2, 2026].
18. Gao Y, Reig B, Heacock L, Bennett DL, Heller SL, Moy L. Magnetic Resonance Imaging in Screening of Breast Cancer. *Radiol Clin North Am*. 2021;59(1):85-98.
doi: 10.1016/j.rcl.2020.09.004
19. Fields BKK, Joe BN. Screening Breast MRI Effectively Detects Early-Stage Breast Cancer in High-Risk Patients without Prior History of Breast Cancer. *Radiol Imaging Cancer*. 2024;6(2):e249005.
doi: 10.1148/rycan.249005
20. García Ruiz C, Saidi LZ, Zambrana Aguilar L, *et al*. Role of MRI in the Diagnosis of Ductal Carcinoma In Situ: A Retrospective Study. *J Clin Med*. 2025;14(8):2842.
doi: 10.3390/jcm14082842
21. Marcuello C, Lim K, Nisini G, Pokrovsky VS, Conde J, Ruggeri FS. Nanoscale Analysis beyond Imaging by Atomic Force Microscopy: Molecular Perspectives on Oncology and Neurodegeneration. *Small Sci*. 2025;5(11):2500351.
doi: 10.1002/ssmc.202500351
22. Shimolina L, Efremov YM, Gulin A, *et al*. Comparison of viscoelastic properties of breast cancer and normal cells using AFM, FLIM and ToF-SIMS techniques. *Sci Rep*. 2025;15:16978.
doi: 10.1038/s41598-025-31162-3
23. O'Dowling AT, Rodriguez BJ, Gallagher TK, Thorpe SD. Machine learning and artificial intelligence: Enabling the clinical translation of atomic force microscopy-based biomarkers for cancer diagnosis. *Comput Struct Biotechnol J*. 2024;24:661-671.
doi: 10.1016/j.csbj.2024.10.006
24. Khan AQ, Touseeq M, Rehman S, *et al*. Advances in breast cancer diagnosis: a comprehensive review of imaging, biosensors, and emerging wearable technologies. *Front Oncol*. 2025;15:1587517.
doi: 10.3389/fonc.2025.1587517
25. Gupta S, Paliczak A, Delgado D. Evidence-based indications of platelet-rich plasma therapy. *Expert Rev Hematol*. 2021;14(1):97-108.
doi: 10.1080/17474086.2021.1860002
26. Tang P, Rahmati M, Xiao W, *et al*. Differences in the effectiveness of leukocyte-rich platelet-rich plasma compared with leukocyte-poor platelet-rich plasma in the treatment of rotator cuff surgery: an umbrella review of meta-analyses. *J Orthop Traumatol*. 2024;25(1):65.
doi: 10.1186/s10195-024-00791-1
27. Yuan Z, Wang Y, Li Y, *et al*. Comparison of Leukocyte-Rich and Leukocyte-Poor Platelet-Rich Plasma on Pressure Ulcer in a Rat Model. *J Burn Care Res*. 2023;44(4):860-868.

- doi: 10.1093/jbcr/irac191
28. Shaaban A, Anwar M, Ramadan R. The role of platelet rich plasma enriched fat graft for correction of deformities after conservative breast surgery. *Breast Dis.* 2024;43(1):111-118.
doi: 10.3233/BD-230057
 29. Everts P, Onishi K, Jayaram P, Lana JF, Mautner K. Platelet-Rich Plasma: New Performance Understandings and Therapeutic Considerations in 2020. *Int J Mol Sci.* 2020;21(20):7794.
doi: 10.3390/ijms21207794
 30. Gentile P, Garcovich S. Systematic Review: Adipose-Derived Mesenchymal Stem Cells, Platelet-Rich Plasma and Biomaterials as New Regenerative Strategies in Chronic Skin Wounds and Soft Tissue Defects. *Int J Mol Sci.* 2021;22(4):1538.
doi: 10.3390/ijms22041538
 31. Khalid KA, Nawi AFM, Zulkifli N, Barkat MA, Hadi H. Aging and Wound Healing of the Skin: A Review of Clinical and Pathophysiological Hallmarks. *Life.* 2022;12(12):2142.
doi: 10.3390/life12122142
 32. Hakami AHM, Mobarki OA, Muthaffar AHI, *et al.* Comparison of clinical outcomes between autologous fat grafting and platelet-rich plasma-enhanced fat grafting in breast reconstruction. *Eurasian J Med Oncol.* 2025;9(4):84.
doi: 10.36922/EJMO025220216
 33. Spear SL, Jespersen MR. Breast Implants: Saline or Silicone? *Aesthet Surg J.* 2010;30(4):557-570.
doi: 10.1177/1090820X10380401
 34. Cordeiro PG. Breast Reconstruction after Surgery for Breast Cancer. *N Engl J Med.* 2008;359(15):1590-1601.
doi: 10.1056/NEJMct0802899
 35. Coleman SR, Saboeiro AP. Fat Grafting to the Breast Revisited: Safety and Efficacy. *Plast Reconstr Surg.* 2007;119(3):775-785.
doi: 10.1097/01.prs.0000252001.59162.c9
 36. Breuing KH, Warren SM. Immediate Bilateral Breast Reconstruction With Implants and Inferolateral AlloDerm Slings. *Ann Plast Surg.* 2005;55(3):232-239.
doi: 10.1097/01.sap.0000168527.52472.3c
 37. Han C, Chen C, Lu N, *et al.* Platelet-Rich Plasma Inhibits Breast Cancer Proliferation. *Clin Med Insights Oncol.* 2024;18:11795549241298978.
doi: 10.1177/11795549241298978
 38. López-Gil JC, Martín-Hijano L, Hermann PC, Sainz B. The CXCL12 Crossroads in Cancer Stem Cells and Their Niche. *Cancers.* 2021;13(3):469.
doi: 10.3390/cancers13030469
 39. Bellomo C, Caja L, Moustakas A. Transforming growth factor β as regulator of cancer stemness and metastasis. *Br J Cancer.* 2016;115(7):761-769.
doi: 10.1038/bjc.2016.255
 40. Chu X, Tian W, Ning J, *et al.* Cancer stem cells: advances in knowledge and implications for cancer therapy. *Signal Transduct Target Ther.* 2024;9(1):363.
doi: 10.1038/s41392-024-01851-y
 41. Liu Q, Guo Z, Li G, *et al.* Cancer stem cells and their niche in cancer progression and therapy. *Cancer Cell Int.* 2023;23(1):289.
doi: 10.1186/s12935-023-03130-2
 42. Wang Y, Jia J, Wang F, *et al.* Pre-metastatic niche: formation, characteristics and therapeutic implication. *Signal Transduct Target Ther.* 2024;9(1):291.
doi: 10.1038/s41392-024-01937-7