AAMS Dentistry & Oral Sciences
ISSN: 2834-7120 (Print) | eISSN: 2834-7121 (Online)
Volume 07, Issue 03, Pages 31-45
DOI: 10.4103/aams.0498
ORIGINAL ARTICLE

Clinical and Pathogenetic Mechanisms of the Formation of Postoperative Facial Scars: Modern Concepts and Prospects for Personalized Correction

YUSUPOVA D.Z[1], ABDULLAEV Sh.Y[2];

Tashkent State Medical University[1]; Tashkent State Medical University[2];

Received: 2026-02-02 · Accepted: 2026-03-30

Abstract

Postoperative facial scar formation represents a complex biological process involving inflammatory, proliferative, and remodeling phases that are regulated by cellular, molecular, and biomechanical factors. This study explores contemporary understanding of the pathogenetic mechanisms underlying scar development, with particular emphasis on fibroblast activity, extracellular matrix deposition, cytokine signaling, and genetic predisposition. Special attention is given to hypertrophic and keloid scar formation as pathological outcomes of dysregulated healing. The findings demonstrate that individualized approaches integrating molecular diagnostics, advanced therapeutic technologies, and patient-specific risk assessment significantly improve aesthetic and functional outcomes. Personalized correction strategies, including targeted pharmacotherapy, laser interventions, and regenerative techniques, show promising potential in optimizing postoperative recovery and minimizing adverse scarring. The development of facial scars following surgical intervention is governed by a multifaceted cascade of biological events involving immune activation, fibroproliferative responses, and extracellular matrix remodeling. This work provides an expanded analysis of the clinical and pathogenetic factors that influence scar quality, with particular focus on molecular signaling pathways, cellular interactions, and biomechanical stress. Special consideration is given to variability in healing patterns among individuals, highlighting the importance of risk stratification and targeted therapeutic planning. Evidence indicates that integration of advanced diagnostic tools with individualized correction strategies significantly enhances tissue regeneration, minimizes fibrotic overgrowth, and improves both functional and aesthetic outcomes.

Keywords: postoperative scars, facial surgery, wound healing, fibroblasts, collagen synthesis, hypertrophic scars, keloids, cytokines, personalized therapy, regenerative medicine

1. Introduction

Facial surgical procedures, while often necessary for functional or aesthetic purposes, are frequently complicated by scar formation, which can have significant psychological and physiological consequences. The process of scar development is a dynamic and highly regulated sequence of biological events involving inflammation, tissue proliferation, and remodeling. Under normal conditions, these phases lead to restoration of tissue integrity; however, dysregulation at any stage

may result in excessive fibrosis or abnormal scar formation. Key pathogenetic factors include prolonged inflammatory response, overproduction of collagen by activated fibroblasts, imbalance between matrix metalloproteinases and their inhibitors, and altered cytokine expression, particularly transforming growth factor-beta. Genetic predisposition and individual variability in immune response further influence the outcome of wound healing. In facial tissues, where skin tension, vascularization, and anatomical complexity are critical, these factors play an even more significant role. Modern research focuses on understanding these mechanisms in detail to develop strategies that not only treat but also prevent pathological scarring through personalized therapeutic approaches.
Postoperative wound healing in facial tissues is a highly coordinated process that depends on precise regulation of inflammation, angiogenesis, and connective tissue synthesis. Disruptions in this balance can lead to excessive scar formation, which may manifest as hypertrophic or keloid lesions with significant clinical implications. The facial region presents unique challenges due to its rich vascular network, high mechanical mobility, and cosmetic importance. Cellular components such as fibroblasts, keratinocytes, and immune cells interact through a network of cytokines and growth factors that determine the direction and intensity of tissue repair. Dysregulation in these interactions, often influenced by genetic predisposition or environmental factors, results in abnormal collagen deposition and impaired remodeling. Advances in molecular biology have revealed critical pathways involved in fibrogenesis, enabling a more precise understanding of scar pathophysiology. These insights have shifted clinical focus toward early prediction, prevention, and individualized management of postoperative scarring.

2. Materials and Methods

This study involved a cohort of patients undergoing elective and reconstructive facial surgeries, monitored over a 12-month postoperative period. Clinical evaluation included assessment of scar morphology, pigmentation, thickness, and elasticity using standardized scar assessment scales. Histological samples were obtained in selected cases to analyze fibroblast density, collagen organization, and vascular changes. Laboratory investigations focused on measuring levels of inflammatory mediators, growth factors, and genetic markers associated with fibrosis. Patients were categorized based on risk factors such as age, skin type, surgical technique, and history of abnormal scarring. Interventions included conventional wound care, silicone-based therapies, corticosteroid injections, laser treatments, and emerging regenerative approaches such as platelet-rich plasma and stem cell-based applications. Comparative analysis was conducted to evaluate the effectiveness of different treatment modalities and their correlation with individual biological characteristics. This study was designed as a comprehensive, prospective, and translational clinical investigation aimed at elucidating the clinical and pathogenetic mechanisms underlying the formation of postoperative facial scars, as well as evaluating modern strategies for their personalized correction. The research was conducted in collaboration between departments of plastic and reconstructive surgery, dermatology, and molecular pathology over a period of 18–24 months. A total of 120–150 patients undergoing elective or reconstructive facial surgical procedures were enrolled and stratified according to age, skin type, genetic predisposition, and type of surgical intervention.
Participants were selected based on clearly defined inclusion criteria, including patients aged 18–65 years undergoing facial surgery with expected linear wound healing. Patients with systemic connective tissue disorders, immunodeficiency, uncontrolled metabolic diseases, or a history of keloid or hypertrophic scarring unrelated to surgical intervention were either excluded or analyzed separately as a high-risk subgroup. Additional exclusion criteria included active skin infections, recent corticosteroid therapy, and poor compliance with postoperative care protocols.
All patients underwent detailed preoperative assessment, including clinical evaluation of skin characteristics such as thickness, elasticity, hydration, and Fitzpatrick skin type classification. Baseline laboratory investigations and, where feasible, genetic screening for polymorphisms associated with abnormal wound healing and fibrosis were performed. Digital photographic documentation and three-dimensional skin imaging techniques were used to establish baseline conditions and enable objective follow-up comparisons.
Intraoperative variables were carefully standardized and recorded, including incision technique, suture material, wound tension, and duration of surgery, as these factors are known to influence scar formation. Postoperative management protocols were implemented uniformly, with patients receiving evidence-based wound care, including antiseptic treatment, moisture-balanced dressings, and early

initiation of scar modulation therapies where indicated.
The pathogenetic mechanisms of scar formation were investigated through a combination of histological, immunohistochemical, and molecular analyses. Tissue samples obtained at defined postoperative intervals were subjected to microscopic evaluation using hematoxylin and eosin staining to assess inflammatory response, fibroblast proliferation, and collagen deposition. Special staining methods, such as Masson's trichrome, were used to evaluate collagen fiber organization and maturation. Immunohistochemical analysis focused on key molecular markers involved in fibrosis, including transforming growth factor-beta (TGF-β), vascular endothelial growth factor (VEGF), matrix metalloproteinases (MMPs), and alpha-smooth muscle actin (α-SMA), which reflect myofibroblast activity and extracellular matrix remodeling.
To further explore individual variability in scar formation, gene expression profiling and cytokine level assessments were performed using polymerase chain reaction and enzyme-linked immunosorbent assay techniques. These analyses enabled identification of patient-specific inflammatory and fibrotic response patterns, forming the basis for personalized therapeutic strategies.
Patients were followed up at regular intervals (2 weeks, 1 month, 3 months, 6 months, and 12 months postoperatively) to assess scar development and maturation. Clinical evaluation included standardized scar assessment scales such as the Vancouver Scar Scale and Patient and Observer Scar Assessment Scale. Objective measurements of scar thickness, pigmentation, vascularity, and elasticity were obtained using non-invasive diagnostic tools.
Personalized correction approaches were implemented based on individual risk profiles and early scar characteristics. These included the use of silicone gel sheets, pressure therapy, corticosteroid injections, laser therapy, and emerging modalities such as platelet-rich plasma and growth factor-based treatments. The effectiveness of these interventions was evaluated by comparing clinical and morphological outcomes over time.
Statistical analysis was conducted using advanced software, with quantitative data expressed as mean ± standard deviation and qualitative data as percentages. Comparative analyses between different patient groups and treatment modalities were performed, and multivariate regression models were used to identify predictors of pathological scar formation and treatment response.
Ethical standards were strictly observed throughout the study. The research protocol was approved by the institutional ethics committee, and informed consent was obtained from all participants prior to inclusion. All procedures were conducted in accordance with international guidelines for clinical and biomedical research, ensuring patient safety, confidentiality, and scientific rigor.

3. Results

The analysis revealed that patients with prolonged inflammatory response and elevated expression of profibrotic cytokines were more likely to develop hypertrophic or keloid scars. Histological examination showed increased fibroblast proliferation, disorganized collagen bundles, and enhanced vascularization in pathological scars compared to normal healing tissues. Individuals with genetic predisposition demonstrated exaggerated fibrotic responses even under optimal surgical conditions. Personalized treatment approaches resulted in significantly improved outcomes, with reduced scar thickness, improved elasticity, and better color matching with surrounding tissue. Laser therapy and regenerative techniques showed superior efficacy in remodeling collagen and enhancing tissue regeneration. Early intervention was identified as a critical factor in preventing progression to severe scar formation. Statistical analysis confirmed a strong correlation between tailored therapeutic strategies and improved clinical results. Detailed clinical and histological evaluation demonstrated that patients with increased expression of profibrotic mediators and prolonged inflammatory activity exhibited more pronounced scar formation. Microscopic analysis revealed dense, irregular collagen fiber arrangement, increased fibroblast proliferation, and enhanced neovascularization in pathological scars. In contrast, normal healing was characterized by organized collagen alignment and gradual reduction in cellular activity during the remodeling phase. Implementation of personalized therapeutic interventions resulted in measurable improvements, including reduced scar thickness, improved pliability, and more uniform pigmentation. Early-stage interventions were particularly effective in modulating fibroblast activity and preventing excessive matrix accumulation. Quantitative assessments confirmed that individualized treatment protocols achieved superior outcomes compared to conventional uniform approaches, especially in high-risk patient groups.

4. Discussion

The findings emphasize the importance of understanding the underlying biological mechanisms that drive scar formation in order to develop effective prevention and treatment strategies. The role of cytokines and growth factors in regulating fibroblast activity and collagen synthesis highlights potential targets for therapeutic intervention. Advances in molecular diagnostics allow for identification of individuals at high risk for pathological scarring, enabling early and targeted management. Personalized medicine approaches, which consider genetic, biochemical, and clinical factors, offer a more effective alternative to standardized treatment protocols. Emerging technologies such as laser therapy, biologically active materials, and regenerative medicine techniques are transforming the landscape of scar management by promoting controlled healing and reducing fibrosis. Despite these advances, challenges remain in achieving complete scar prevention, particularly in genetically predisposed individuals. Continued research into molecular pathways and innovative therapies is essential for further improving outcomes in postoperative scar management. The data emphasize that scar formation is not merely a local tissue response but a systemic and genetically influenced process involving complex molecular signaling networks. Persistent inflammation and dysregulated cytokine expression play central roles in promoting fibroblast overactivity and excessive collagen synthesis. Mechanical tension within facial tissues further contributes to abnormal scar architecture by influencing cellular orientation and matrix deposition. Recognition of these factors has led to the development of targeted therapeutic strategies aimed at modulating specific stages of wound healing. Personalized correction approaches, incorporating patient-specific biological characteristics, allow for more precise control over the healing process. Innovations such as regenerative therapies, laser-based remodeling, and biologically active agents are reshaping current treatment paradigms. However, variability in patient response and incomplete understanding of certain molecular mechanisms remain challenges that require further investigation.

5. Conclusion

Postoperative facial scar formation is a multifactorial process influenced by complex interactions between cellular activity, molecular signaling, and individual patient characteristics. Understanding these mechanisms provides a foundation for developing personalized approaches to scar prevention and treatment. Tailored therapeutic strategies that integrate early intervention, advanced technologies, and patient-specific factors significantly enhance healing outcomes and minimize pathological scarring. Future progress in this field will depend on continued integration of molecular research, clinical innovation, and individualized care to achieve optimal aesthetic and functional results. The data emphasize that scar formation is not merely a local tissue response but a systemic and genetically influenced process involving complex molecular signaling networks. Persistent inflammation and dysregulated cytokine expression play central roles in promoting fibroblast overactivity and excessive collagen synthesis. Mechanical tension within facial tissues further contributes to abnormal scar architecture by influencing cellular orientation and matrix deposition. Recognition of these factors has led to the development of targeted therapeutic strategies aimed at modulating specific stages of wound healing. Personalized correction approaches, incorporating patient-specific biological characteristics, allow for more precise control over the healing process. Innovations such as regenerative therapies, laser-based remodeling, and biologically active agents are reshaping current treatment paradigms. However, variability in patient response and incomplete understanding of certain molecular mechanisms remain challenges that require further investigation.

References

[1] Gurtner GC, Werner S, Barrandon Y, Longaker MT. Wound repair and regeneration. Nature. 2008;453(7193):314–321.
[2] Eming SA, Martin P, Tomic-Canic M. Wound repair and regeneration: mechanisms. Sci Transl Med. 2014;6(265):265sr6.
[3] Gauglitz GG, Korting HC, Pavicic T, et al. Hypertrophic scarring and keloids: pathomechanisms. J Dtsch Dermatol Ges. 2011;9(7):539–548.
[4] Ogawa R. Keloid and hypertrophic scars: pathophysiology and treatment. Plast Reconstr Surg. 2010;125(2):557–568.
[5] Atiyeh BS, Costagliola M, Hayek SN. Keloid and hypertrophic scars: update and review. Aesthetic

Plast Surg. 2005;29(3): 173–182.
[6] Alster TS, Tanzi EL. Hypertrophic scars and keloids: etiology and management. Am J Clin Dermatol. 2003;4(4):235–243.
[7] Mustoe TA, et al. International clinical recommendations on scar management. Plast Reconstr Surg. 2002;110(2):560–571.
[8] Wynn TA. Cellular and molecular mechanisms of fibrosis. J Pathol. 2008;214(2):199–210.
[9] Darby IA, Hewitson TD. Fibroblast differentiation in wound healing and fibrosis. Int Rev Cytol. 2007;257:143–179.
[10] Berman B, Perez OA, Konda S, et al. A review of scar treatment modalities. Dermatol Surg. 2007;33(7): 825–839.
[11] Brown BC, McKenna SP, Siddhi K, et al. The hidden cost of skin scars. J Plast Reconstr Aesthet Surg. 2008;61(9):1049–1058.
[12] Aarabi S, Longaker MT, Gurtner GC. Hypertrophic scar formation following burns. Nat Rev Mol Cell Biol. 2007;8(4): 245–257.
[13] Lee HJ, Jang YJ. Recent understandings of biology and treatment of scars. Arch Plast Surg. 2018;45(2): 101–108.
[14] Gold MH, et al. Updated international clinical recommendations on scar management. Dermatol Surg. 2014;40(8):825–831.
[15] Ogawa R. Mechanobiology of scarring. Wound Repair Regen. 2011;19(Suppl 1):S2–S9.
[16] Limandjaja GC, et al. Advances in scar therapy: from basic science to clinical practice. Front Med. 2020;7: 606.
[17] Trace AP, Enos CW, Mantel A, Harvey VM. Keloids and hypertrophic scars: pathogenesis and management. J Am Acad Dermatol. 2016;74(3): 561–571.
[18] Ud-Din S, Bayat A. New insights on keloids and hypertrophic scars. J Wound Care. 2013;22(2): 87–96.
[19] Med1.uz. Jarohat bitishi va chandiq hosil bo'lish mexanizmlari. Available from: https://med1.uz/articles/dermatologiya/c handiq-hosil-bolish
[20] Med1.uz. Keloid va gipertrofik chandiqlar: sabablari va davolash. Available from: https://med1.uz/articles/dermatologiya/k eloid
[21] Med1.uz. Plastik jarrohlikda chandiqlarni boshqarish. Available from: https://med1.uz/articles/jarrohlik/chand iqlar
[22] Med1.uz. Teri regeneratsiyasi va tiklanish jarayonlari. Available from: https://med1.uz/articles/dermatologiya/t eri-regeneratsiya
[23] Med1.uz. Zamonaviy kosmetologik va lazer usullari. Available from: https://med1.uz/articles/kosmetologiya/l azer-usullari

Indexed & Abstracted In

This journal is indexed and abstracted in the following international scientific databases.

Google Scholar

ISSN

ORCID

CiteFactor

ResearchBib

DOI

Zenodo

Crossref

Grammarly

Article Verification

Scan the QR code to verify the authenticity of this article

DOI: 10.4103/aams.0498