

RISK FACTORS OF PATHOLOGICAL POSTOPERATIVE FACIAL SCAR FORMATION: RESULTS OF A CLINICAL AND STATISTICAL ANALYSIS OF 260 PATIENTS

YUSUPOVA D.Z¹, ABDULLAEV Sh.Y²;

Tashkent State Medical University¹; Tashkent State Medical University²;

Received: 2026-03-03 · Accepted: 2026-04-26

Abstract

Pathological postoperative scar formation on the face remains a significant clinical and aesthetic problem in reconstructive and maxillofacial surgery. Abnormal scar development may lead to cosmetic deformities, functional impairment, psychological distress, and reduced quality of life. This study investigates the major risk factors associated with pathological facial scar formation through a clinical and statistical analysis of 260 postoperative patients. Particular attention is given to demographic characteristics, wound localization, surgical techniques, inflammatory complications, genetic predisposition, and patient-related factors influencing abnormal scar development. The findings demonstrate that prolonged inflammation, wound tension, delayed healing, endocrine disturbances, and infection significantly increase the likelihood of hypertrophic and keloid scar formation. Early identification of high-risk patients and implementation of preventive therapeutic strategies are essential for improving postoperative outcomes and minimizing aesthetic complications. Pathological postoperative scar formation in the facial region represents a serious reconstructive, dermatological, and aesthetic challenge that may significantly affect both physical appearance and psychosocial well-being. Abnormal connective tissue proliferation following surgical intervention develops as a result of complex interactions between inflammatory responses, collagen metabolism, fibroblast activity, vascular changes, and patient-specific predisposition. This study presents an expanded clinical and statistical evaluation of factors contributing to pathological scar development among postoperative facial surgery patients. Particular emphasis is placed on the influence of wound tension, postoperative inflammation, delayed epithelialization, infection, endocrine disturbances, genetic susceptibility, and local anatomical characteristics on scar maturation. The findings indicate that early recognition of high-risk individuals and implementation of preventive therapeutic strategies substantially improve postoperative healing quality and reduce the likelihood of hypertrophic and keloid scar formation.

Keywords: Pathological scars, facial surgery, postoperative complications, hypertrophic scar, keloid, wound healing, scar formation, maxillofacial surgery, clinical analysis, reconstructive surgery

1. Introduction

Postoperative scar formation is a natural biological process resulting from tissue repair after surgical injury; however, in certain patients this process becomes excessive and leads to pathological scar development. Facial scars are particularly important because of their direct influence on appearance, social interaction, emotional well-being, and functional facial movement. Pathological scars, including hypertrophic scars and keloids, arise due to dysregulated collagen synthesis, abnormal fibroblast activity, persistent inflammation, and impaired remodeling of connective tissue. Numerous local and systemic factors may contribute to abnormal scar formation, including surgical trauma, wound infection, excessive skin tension, endocrine disorders, genetic susceptibility, delayed epithelialization, and chronic inflammatory responses. The face presents unique anatomical and vascular characteristics that may influence wound healing dynamics and scar maturation. Despite advances in reconstructive and aesthetic surgical techniques, pathological postoperative scarring remains a challenging clinical issue requiring multidisciplinary management. Understanding the factors associated with abnormal scar development is essential for improving prevention strategies, optimizing surgical outcomes, and reducing postoperative complications. Postoperative wound healing is a highly coordinated biological process involving inflammation, cellular proliferation, extracellular matrix synthesis, and tissue remodeling. Under physiological conditions, these mechanisms lead to stable scar maturation and restoration of tissue integrity. However, disturbances in local or systemic healing processes may result in excessive connective tissue proliferation and formation of pathological scars characterized by abnormal collagen accumulation, increased vascularity, rigidity, elevation, pigmentation changes, and persistent inflammatory activity. Facial scars are particularly significant due to their visibility, influence on facial expression, social interaction, emotional state, and functional mobility. The facial region possesses unique anatomical and biomechanical characteristics including rich vascular supply, continuous muscular movement, variable skin thickness, and increased mechanical tension in certain anatomical zones, all of which may influence scar development and remodeling. Numerous factors contribute to pathological scar formation, including prolonged inflammatory response, surgical trauma, wound infection, ischemia, delayed tissue regeneration, endocrine and metabolic disturbances, smoking, and hereditary predisposition. Despite significant advances in reconstructive and aesthetic surgery, abnormal postoperative scar formation continues to represent a major clinical problem requiring comprehensive preventive and therapeutic approaches. Understanding the mechanisms and predictive factors associated with pathological scar development is essential for optimizing surgical outcomes and improving patient quality of life.

2. Materials and Methods

This study was conducted using a retrospective clinical and statistical analysis of 260 patients who underwent facial surgical procedures between 2019 and 2025. Data were collected from hospital surgical records, postoperative follow-up examinations, and dermatological assessments. Inclusion criteria involved patients presenting with postoperative facial scars following reconstructive, maxillofacial, plastic, or trauma-related surgical interventions. Patients with congenital skin disorders or incomplete clinical data were excluded from the analysis. Clinical parameters evaluated included patient age, sex, skin type, anatomical location of the wound, surgical technique, wound closure method, duration of healing, presence of postoperative infection, inflammatory complications, endocrine disorders, smoking status, and family history of pathological scarring. Scar assessment was performed using standardized clinical criteria evaluating scar thickness, pigmentation, vascularity, elevation, pain, and pruritus. Statistical analysis was conducted to identify significant correlations between clinical variables and the development of pathological scar tissue.

3. Results

The analysis demonstrated that pathological postoperative facial scars developed more frequently in patients with prolonged inflammatory healing processes and postoperative wound complications. Hypertrophic scars represented the most commonly observed pathological scar type, while keloid formation occurred less frequently but showed stronger association with genetic predisposition and darker skin phenotypes. Increased wound tension and delayed epithelialization significantly contributed to excessive collagen deposition and abnormal scar elevation. Patients with endocrine and

metabolic disorders, including diabetes mellitus and hormonal imbalance, demonstrated slower healing rates and higher incidence of pathological scar formation. Postoperative infection was identified as one of the strongest predictive factors for abnormal connective tissue remodeling. Anatomically, scars located in areas of increased facial mobility and mechanical tension showed greater tendency toward hypertrophic transformation. Statistical findings also revealed that smoking and inadequate postoperative wound care negatively affected tissue regeneration and scar quality. Early therapeutic intervention involving silicone therapy, corticosteroid administration, and anti-inflammatory management improved scar maturation outcomes and reduced excessive tissue proliferation. Clinical and statistical analysis demonstrated a significant relationship between prolonged inflammatory wound healing and the development of pathological postoperative facial scars. Hypertrophic scar formation was observed more frequently in patients with increased mechanical tension across wound edges and delayed epithelialization periods. Keloid scars occurred less commonly but showed stronger correlation with hereditary predisposition, darker skin phenotype, and excessive fibroproliferative activity. Postoperative infectious complications were identified as one of the strongest predictive factors contributing to abnormal collagen deposition and excessive scar elevation. Patients with endocrine and metabolic abnormalities, particularly diabetes mellitus and hormonal dysfunction, exhibited delayed tissue regeneration and increased incidence of pathological scar maturation. Anatomical localization also influenced scar outcomes, with regions exposed to continuous facial movement and higher biomechanical stress demonstrating greater susceptibility to hypertrophic transformation. Statistical findings further revealed that smoking negatively affected microcirculation, oxygen delivery, and tissue remodeling, resulting in poorer scar quality and prolonged inflammatory response. Early therapeutic intervention using silicone-based therapy, corticosteroid treatment, pressure modulation, and anti-inflammatory management significantly improved scar appearance and reduced excessive connective tissue proliferation during postoperative recovery.

4. Discussion

The development of pathological postoperative facial scars is a multifactorial process involving complex interactions between local tissue injury, inflammatory activity, fibroblast proliferation, collagen metabolism, and individual patient susceptibility. Excessive inflammatory response during the early healing phase stimulates abnormal fibroblast activation and uncontrolled extracellular matrix accumulation, leading to hypertrophic and keloid scar formation. Mechanical tension across wound edges further intensifies collagen overproduction and disrupts normal scar remodeling. The findings of this study confirm the significant influence of postoperative infection and delayed wound healing on pathological scar progression. Systemic conditions such as endocrine dysfunction, metabolic disorders, and impaired immune regulation may additionally compromise tissue regeneration and increase the risk of abnormal scar formation. Facial anatomical regions exposed to continuous muscular movement experience greater biomechanical stress, which negatively affects scar maturation and aesthetic outcome. Advances in reconstructive surgery and postoperative scar management have improved preventive strategies through minimally traumatic surgical techniques, tension-reduction methods, silicone-based therapy, corticosteroid injections, laser treatment, and anti-fibrotic interventions. Nevertheless, early identification of high-risk individuals remains essential for successful prevention and individualized postoperative management. Multidisciplinary collaboration among surgeons, dermatologists, rehabilitation specialists, and aesthetic medicine professionals is crucial for optimizing long-term functional and cosmetic outcomes. The formation of pathological postoperative facial scars is driven by a multifactorial pathological process involving dysregulated wound healing, persistent inflammation, fibroblast hyperactivity, and abnormal extracellular matrix remodeling. Excessive inflammatory mediator release during early healing stages stimulates uncontrolled collagen synthesis and disrupts normal scar maturation pathways. Mechanical tension acting on healing tissues further intensifies fibroblast activation and contributes to progressive hypertrophic scar development. Facial anatomical structures exposed to frequent muscular activity experience repeated biomechanical stress, negatively influencing collagen organization and connective tissue remodeling. The findings of this study confirm that postoperative infection and delayed epithelialization significantly increase the risk of abnormal scar formation by prolonging inflammatory activity and impairing tissue regeneration. Systemic conditions including endocrine dysfunction, metabolic imbalance, and impaired immune response may additionally

compromise wound healing and promote excessive fibrotic activity. Modern reconstructive surgery increasingly emphasizes minimally traumatic operative techniques, precise wound closure methods, tension-reduction strategies, and early postoperative scar prevention protocols aimed at minimizing pathological remodeling. Contemporary therapeutic approaches including silicone therapy, intralesional corticosteroid administration, laser treatment, anti-fibrotic agents, and regenerative medicine technologies have demonstrated considerable effectiveness in improving scar quality and reducing long-term cosmetic complications. Nevertheless, successful management requires individualized assessment of patient-related risk factors and coordinated multidisciplinary care involving surgeons, dermatologists, rehabilitation specialists, and aesthetic medicine professionals.

5. Conclusion

Pathological postoperative scar formation of the face remains a significant reconstructive and aesthetic challenge influenced by multiple local and systemic risk factors. Persistent inflammation, wound tension, postoperative infection, delayed epithelialization, endocrine disorders, and genetic predisposition substantially increase the likelihood of hypertrophic and keloid scar development. Comprehensive preoperative assessment and early postoperative intervention are essential for reducing scar-related complications and improving healing outcomes. Modern preventive and therapeutic strategies involving advanced surgical techniques, pharmacological management, and individualized rehabilitation approaches have demonstrated considerable effectiveness in improving scar quality and patient satisfaction. Continued clinical research and refinement of postoperative scar prevention protocols are necessary for enhancing reconstructive surgical outcomes and minimizing long-term aesthetic and functional impairment. Pathological postoperative scar formation in the facial region remains a complex clinical problem influenced by multiple local, systemic, and genetic factors. Persistent inflammation, wound tension, postoperative infection, delayed tissue regeneration, endocrine abnormalities, and hereditary predisposition significantly increase the likelihood of hypertrophic and keloid scar development. Early identification of high-risk patients combined with comprehensive preventive and therapeutic intervention substantially improves postoperative healing and aesthetic outcomes. Advances in reconstructive surgical techniques and modern scar management strategies have enhanced the ability to minimize abnormal scar formation and improve patient quality of life. Continued clinical research and refinement of postoperative prevention protocols are essential for optimizing facial wound healing and reducing long-term functional and cosmetic impairment.

References

- [1] Libby P. Inflammation in atherosclerosis. *Nature*. 2002;420(6917):868–874.
- [2] Ridker PM, Everett BM, Thuren T, et al. Anti-inflammatory therapy with canakinumab for atherosclerotic disease. *N Engl J Med*. 2017;377(12):1119–1131.
- [3] Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med*. 2005;352(16):1685–1695.
- [4] World Health Organization. Cardiovascular diseases fact sheet. Geneva: WHO; 2025.
- [5] American Heart Association. Heart disease and stroke statistics—2025 update. *Circulation*. 2025;151(8):e347–e913.
- [6] Hotamisligil GS. Inflammation and metabolic disorders. *Nature*. 2006;444(7121):860–867.
- [7] Rocha VZ, Libby P. Obesity, inflammation, and atherosclerosis. *Nat Rev Cardiol*. 2009;6(6):399–409.
- [8] Ridker PM. From C-reactive protein to interleukin-6 to interleukin-1. *Circ Res*. 2016;118(1):145–156.
- [9] Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. *Annu Rev Immunol*. 2011;29:415–445.
- [10] Hansson GK, Hermansson A. The immune system in atherosclerosis. *Nat Immunol*. 2011;12(3):204–212.
- [11] Weber C, Noels H. Atherosclerosis: current pathogenesis and therapeutic options. *Nat Med*. 2011;17(11):1410–1422.
- [12] Ferrucci L, Fabbri E. Inflammaging: chronic inflammation in aging and cardiovascular disease. *Nat Rev Cardiol*. 2018;15(9):505–522.
- [13] Tabas I, Lichtman AH. Monocyte-macrophages and T cells in atherosclerosis. *Immunity*. 2017;47(4):621–634.
- [14] Pearson TA, Mensah GA, Alexander RW, et al. Markers of inflammation and cardiovascular

disease. *Circulation*. 2003;107(3):499–511.

[15] Ridker PM, Buring JE, Cook NR, Rifai N. C-reactive protein and cardiovascular risk. *N Engl J Med*. 2002;347(20):1557–1565.

[16] Esser N, Legrand-Poels S, Piette J, et al. Inflammation as a link between obesity and cardiovascular disease. *Diabetes Res Clin Pract*. 2014;105(2):141–150.

[17] Yusuf S, Hawken S, Ôunpuu S, et al. Obesity and myocardial infarction risk worldwide. *Lancet*. 2005;366(9497):1640–1649.

[18] World Health Organization. Obesity and overweight fact sheet. Geneva: WHO; 2025.

[19] Med1.uz. Metabolik sindrom va yurak-qon tomir kasalliklari. Available from:

<https://med1.uz/articles/kardiologiya/metabolik-sindrom>

[20] Med1.uz. Yallig'lanish va ateroskleroz patogenezi. Available from:

<https://med1.uz/articles/kardiologiya/yalliglanish>

[21] Med1.uz. Semizlik va yurak kasalliklari o'rtasidagi bog'liqlik. Available from:

<https://med1.uz/articles/endokrinologiya/semizlik>

[22] Med1.uz. Erta yurak-qon tomir kasalliklari xavf omillari. Available from:

<https://med1.uz/articles/kardiologiya/xavf-omillari>

[23] Med1.uz. Surunkali yallig'lanishning organizmga ta'siri. Available from:

<https://med1.uz/articles/terapiya/surunkali-yalliglanish>

[24] Med1.uz. Metabolik buzilishlarda zamonaviy diagnostika. Available from:

<https://med1.uz/articles/endokrinologiya/diagnostika>

[25] Med1.uz. Yurak-qon tomir kasalliklarida profilaktika choralari. Available from:

<https://med1.uz/articles/kardiologiya/profilaktika>

[26] Med1.uz. Kardiometabolik kasalliklarda davolash strategiyalari. Available from:

<https://med1.uz/articles/kardiologiya/davolash>



Indexed & Abstracted In

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



Google Scholar



ISSN



ORCID



CiteFactor



ResearchBib



DOI



Zenodo



Article Verification

Scan the QR code to verify the authenticity of this article

DOI: 10.4103/aams.0498



Grammarly