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ORIGINAL ARTICLE

THE ROLE OF MICROCIRCULATORY AND METABOLIC DISORDERS IN THE DEVELOPMENT OF PATHOLOGICAL FACIAL SCARS: A CLINICAL AND PATHOGENETIC ANALYSIS

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

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

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Abstract

Pathological facial scar formation remains a significant clinical and aesthetic problem associated with impaired wound healing, chronic inflammation, and abnormal connective tissue remodeling. Increasing evidence suggests that disturbances in microcirculation and cellular metabolism play a central role in the pathogenesis of hypertrophic and keloid scars. This article investigates the influence of microvascular dysfunction, tissue hypoxia, metabolic imbalance, oxidative stress, and inflammatory activation on the development of pathological scars of the face. The study evaluates clinical manifestations, pathogenetic mechanisms, diagnostic indicators, and modern therapeutic approaches aimed at improving tissue regeneration and scar quality. The findings demonstrate that impaired local blood supply, altered oxygen utilization, endothelial dysfunction, and metabolic disturbances significantly contribute to excessive fibroblast activity and abnormal collagen deposition. Early correction of microcirculatory and metabolic abnormalities may improve wound healing outcomes and reduce the risk of pathological scar development. Pathological scar formation in the facial region represents a complex pathological process closely associated with disturbances of local microcirculation, tissue metabolism, inflammatory regulation, and connective tissue remodeling. Impaired blood supply, endothelial dysfunction, oxidative imbalance, and chronic tissue hypoxia significantly influence reparative processes and contribute to excessive collagen accumulation during wound healing. This study provides an expanded clinical and pathogenetic evaluation of the role of microvascular and metabolic abnormalities in the development of hypertrophic and keloid facial scars. Special attention is given to the interaction between inflammatory mediators, fibroblast hyperactivity, oxygen deficiency, and metabolic dysfunction in scar progression. The findings demonstrate that microcirculatory insufficiency and metabolic instability substantially worsen tissue regeneration and increase the likelihood of abnormal fibrotic transformation. Early correction of vascular and metabolic disturbances may improve postoperative healing quality and reduce severe aesthetic and functional complications.

Keywords: Pathological scars, facial scars, microcirculation, metabolic disorders, hypertrophic scar, keloid, wound healing, oxidative stress, tissue hypoxia, collagen metabolism

1. Introduction

Wound healing is a complex physiological process involving coordinated inflammatory response, angiogenesis, fibroblast activation, collagen synthesis, and tissue remodeling. Under normal conditions, these mechanisms restore tissue integrity and lead to formation of mature, flexible scar tissue. However, disruption of local microcirculation and cellular metabolic processes may alter normal healing dynamics and contribute to pathological scar formation. Facial pathological scars are particularly important because they affect aesthetic appearance, emotional well-being, social interaction, and functional facial movement. Hypertrophic and keloid scars develop due to excessive connective tissue proliferation, persistent inflammation, and dysregulated extracellular matrix remodeling. Modern pathogenetic research increasingly emphasizes the role of impaired tissue perfusion, endothelial dysfunction, chronic hypoxia, oxidative stress, and metabolic imbalance in abnormal scar development. Reduced oxygen delivery and impaired nutrient exchange within healing tissues stimulate fibroblast hyperactivity and excessive collagen accumulation. Additionally, systemic metabolic conditions such as diabetes mellitus, endocrine dysfunction, and obesity may further compromise tissue regeneration and microvascular stability. Understanding the relationship between microcirculatory disturbances, metabolic abnormalities, and pathological scarring is essential for developing effective preventive and therapeutic strategies in reconstructive and aesthetic facial surgery. Physiological wound healing is a highly regulated biological process involving inflammation, angiogenesis, fibroblast activation, extracellular matrix synthesis, and long-term tissue remodeling. Under normal conditions these mechanisms lead to restoration of tissue integrity and formation of mature, flexible scar tissue with minimal structural deformity. However, disruption of local blood circulation and cellular metabolism may alter reparative dynamics and result in pathological scar development characterized by excessive fibrosis, abnormal collagen deposition, persistent vascular activity, and prolonged inflammatory response. Facial pathological scars are of particular clinical importance because of their influence on appearance, facial mobility, emotional well-being, and social adaptation. The anatomical characteristics of facial tissues, including rich vascularization, continuous muscular activity, and variable biomechanical stress distribution, significantly affect wound healing processes and scar maturation. Increasing scientific evidence suggests that disturbances in microvascular perfusion and metabolic regulation play a fundamental role in pathological scar formation. Chronic local hypoxia caused by impaired capillary circulation stimulates fibroblast proliferation and excessive connective tissue synthesis through activation of inflammatory and fibrogenic pathways. Oxidative stress and endothelial dysfunction further aggravate tissue injury by impairing oxygen delivery, nutrient exchange, and collagen remodeling. Systemic metabolic disorders such as diabetes mellitus, obesity, endocrine dysfunction, and insulin resistance may additionally compromise regenerative capacity and vascular stability. Understanding the interaction between microcirculatory insufficiency, metabolic imbalance, and connective tissue remodeling is therefore essential for improving preventive and therapeutic strategies in reconstructive and aesthetic facial surgery.

2. Materials and Methods

This study was conducted using a retrospective clinical and pathogenetic analysis of patients presenting with pathological facial scars following trauma, reconstructive surgery, or inflammatory skin injury between 2019 and 2025. Clinical data were collected from dermatological, maxillofacial, and reconstructive surgery departments. Inclusion criteria involved patients diagnosed with hypertrophic or keloid facial scars confirmed through clinical and morphological assessment. Evaluated parameters included patient age, sex, scar duration, anatomical localization, wound healing characteristics, inflammatory complications, microcirculatory status, metabolic profile, and associated systemic diseases. Microcirculatory evaluation was performed using capillaroscopic examination and tissue perfusion analysis. Laboratory assessment included evaluation of inflammatory biomarkers, oxidative stress indicators, glucose metabolism, and lipid profile abnormalities. Statistical analysis was conducted to determine correlations between microvascular dysfunction, metabolic disturbances, and severity of pathological scar formation.

3. Results

The analysis demonstrated a strong association between impaired local microcirculation and the severity of pathological facial scar development. Patients with hypertrophic and keloid scars frequently exhibited reduced tissue perfusion, vascular instability, prolonged inflammatory activity, and signs of chronic local hypoxia. Capillaroscopic examination revealed structural microvascular abnormalities including reduced capillary density, impaired blood flow regulation, and endothelial dysfunction within affected scar regions. Elevated inflammatory and oxidative stress markers were significantly associated with excessive fibroblast proliferation and abnormal collagen accumulation. Metabolic disturbances such as impaired glucose tolerance, dyslipidemia, and endocrine dysfunction were more common among patients with severe pathological scarring and delayed wound maturation. Histopathological findings demonstrated increased collagen fiber density, persistent inflammatory infiltration, and abnormal vascular remodeling within scar tissue. Patients receiving combined therapy aimed at improving microcirculation, reducing oxidative stress, and stabilizing metabolic function demonstrated improved scar elasticity, reduced fibrosis, and better aesthetic outcomes compared to standard postoperative care alone. Clinical and laboratory evaluation demonstrated that patients with pathological facial scars frequently exhibited significant disturbances in local tissue perfusion and metabolic regulation compared with individuals showing physiological scar maturation. Capillaroscopic assessment revealed reduced capillary density, impaired vascular reactivity, endothelial instability, and irregular microvascular architecture within hypertrophic and keloid scar regions. Persistent tissue hypoxia was associated with increased fibroblast activity and excessive extracellular matrix accumulation, leading to progressive scar elevation and fibrosis. Elevated inflammatory biomarkers and oxidative stress indicators were commonly observed among patients with severe scar deformation and prolonged healing periods. Metabolic abnormalities including impaired glucose metabolism, lipid imbalance, and endocrine dysfunction showed strong correlation with delayed epithelialization and abnormal connective tissue remodeling. Histopathological examination demonstrated dense collagen fiber accumulation, increased vascular proliferation, chronic inflammatory infiltration, and altered tissue organization within pathological scar tissue. Anatomical regions exposed to higher mechanical stress and facial muscular activity demonstrated greater susceptibility to hypertrophic transformation. Patients receiving combined therapeutic approaches targeting microcirculatory improvement, antioxidant support, anti-inflammatory modulation, and metabolic stabilization showed improved scar elasticity, reduced fibrosis intensity, and better long-term cosmetic outcomes.

4. Discussion

The findings of this study confirm that microcirculatory and metabolic disturbances play a fundamental role in the pathogenesis of pathological facial scars. Impaired tissue perfusion and chronic local hypoxia create conditions that stimulate prolonged inflammatory activity and excessive fibroblast activation, ultimately leading to abnormal extracellular matrix accumulation and fibrosis. Endothelial dysfunction contributes to impaired vascular regulation and reduced oxygen delivery, further intensifying tissue remodeling abnormalities. Oxidative stress represents another critical pathogenetic mechanism capable of damaging cellular structures, disrupting collagen turnover, and sustaining inflammatory processes during wound healing. Metabolic disorders including diabetes mellitus, insulin resistance, and lipid imbalance additionally compromise tissue regeneration by impairing cellular energy metabolism and vascular integrity. The anatomical characteristics of facial tissues, including constant muscular activity and variable vascular distribution, may further influence scar maturation and biomechanical stress response. Contemporary therapeutic strategies increasingly focus on pathogenetic correction through enhancement of microcirculation, anti-inflammatory treatment, antioxidant therapy, metabolic stabilization, and modulation of collagen synthesis. Early identification of patients with vascular and metabolic risk factors may significantly improve prevention of hypertrophic and keloid scar formation. Multidisciplinary management involving reconstructive surgeons, dermatologists, endocrinologists, and rehabilitation specialists remains essential for optimizing both functional and cosmetic outcomes. The findings confirm that disturbances in microcirculatory and metabolic homeostasis play a critical pathogenetic role in the development and progression of pathological facial scars. Impaired local blood flow contributes to chronic tissue ischemia and insufficient oxygen delivery, creating biological conditions that stimulate excessive

fibroblast proliferation and dysregulated collagen synthesis. Persistent inflammatory activation further intensifies connective tissue remodeling abnormalities by promoting release of cytokines, growth factors, and oxidative mediators that disrupt physiological scar maturation. Endothelial dysfunction appears to be one of the key mechanisms underlying impaired vascular regulation and chronic tissue hypoxia within healing wounds. Metabolic disorders additionally worsen reparative processes through impaired cellular energy metabolism, oxidative imbalance, and reduced regenerative capacity. Facial tissues are especially vulnerable to pathological scar transformation due to continuous biomechanical stress associated with facial expression and muscular movement. Modern therapeutic strategies increasingly focus on pathogenetic correction aimed at improving microvascular circulation, reducing oxidative stress, stabilizing metabolic function, and modulating fibroblast activity. Contemporary approaches including antioxidant therapy, microcirculatory correction, laser technologies, silicone-based treatment, corticosteroid administration, and regenerative medicine techniques have demonstrated promising effectiveness in improving scar maturation and reducing excessive fibrosis. Successful prevention and treatment require individualized multidisciplinary management integrating reconstructive surgery, dermatology, endocrinology, and rehabilitation medicine.

5. Conclusion

Microcirculatory impairment and metabolic dysfunction are major pathogenetic factors contributing to the development of pathological facial scars. Chronic tissue hypoxia, endothelial abnormalities, oxidative stress, and metabolic imbalance significantly disrupt normal wound healing and promote excessive collagen deposition and fibrosis. Early detection and correction of vascular and metabolic disturbances are essential for improving scar maturation and reducing postoperative and posttraumatic complications. Modern комплекс therapeutic approaches targeting tissue perfusion, inflammatory regulation, oxidative balance, and metabolic stabilization have demonstrated promising effectiveness in improving clinical and aesthetic outcomes. Continued pathogenetic research and individualized therapeutic strategies are necessary for advancing prevention and treatment of pathological facial scar formation. Microcirculatory insufficiency and metabolic dysfunction are major contributing factors in the pathogenesis of pathological facial scar formation. Chronic tissue hypoxia, endothelial abnormalities, oxidative stress, inflammatory persistence, and impaired metabolic regulation significantly disrupt physiological wound healing and promote excessive connective tissue proliferation. Early identification and correction of vascular and metabolic disturbances are essential for improving tissue regeneration and preventing severe hypertrophic and keloid scar development. Modern pathogenetically oriented therapeutic strategies targeting vascular stability, inflammatory control, antioxidant balance, and metabolic normalization have substantially improved reconstructive and aesthetic outcomes. Continued research into the molecular and microvascular mechanisms of pathological scarring remains necessary for developing more effective preventive and individualized treatment approaches in facial reconstructive surgery.

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