OPEN ACCESS Freely available online

Journal of Clinical & Experimental Ophthalmology

Perspective

Fibrovascular Proliferation as a Central Mechanism of Dysregulated Wound Healing

Lucia Moretti*

Department of Translational Medicine, University of Milan School of Medicine, Milan, Italy

DESCRIPTION

Fibrovascular proliferation represents a fundamental pathological process observed in a wide range of diseases where normal tissue repair mechanisms become dysregulated and excessive. Although it is frequently described in histopathological terms, its clinical significance extends far beyond microscopic findings, as it plays a central role in chronic inflammation, wound healing abnormalities, and progressive tissue damage across multiple organ systems. Under normal physiological conditions, tissue injury triggers a tightly regulated cascade involving inflammation, cell migration, extracellular matrix deposition, and vascular repair. Once healing is complete, these processes subside. In fibrovascular proliferation, this regulatory balance is lost, leading to continuous activation of fibroblasts and endothelial cells. In ophthalmology, fibrovascular proliferation is particularly significant because of its impact on transparent and delicate ocular structures. One of the most well-recognized occurs in Pterygium, where fibrovascular tissue originating from the conjunctiva invades the corneal surface. This process reflects not only mechanical extension but also active biological remodeling driven by inflammation, ultraviolet radiation exposure, and molecular signaling pathways. As fibrovascular tissue advances, it alters corneal curvature, induces astigmatism, and may eventually encroach upon the visual axis, leading to visual impairment. The biological mechanisms underlying fibrovascular proliferation are complex and involve multiple interacting pathways.

Fibrovascular proliferation is increased cellular density within the stroma, deposition of collagen fibers, and the presence of newly formed capillary networks. These vessels are often immature and structurally abnormal, reflecting ongoing angiogenic activity rather than stable vascular architecture. Inflammatory cells, including lymphocytes and macrophages, are frequently present, indicating a chronic immune response. The combination of fibrosis, vascularization, and inflammation results in a tissue architecture that is fundamentally different from normal, organized structures, often leading to functional impairment depending on the location and extent of involvement.

Beyond the eye, fibrovascular proliferation is a key feature in several systemic and localized diseases. In proliferative diabetic retinopathy, ischemia-driven angiogenesis leads to the growth of fragile new vessels and associated fibrous tissue within the retina, which can contract and cause tractional retinal detachment. In chronic wounds, excessive fibrovascular tissue can interfere with normal healing and delay epithelial regeneration. Fibrovascular proliferation as a pathological process rather than a disease-specific phenomenon. One of the most important clinical implications of fibrovascular proliferation is its tendency toward recurrence and persistence. Once established, the underlying molecular and cellular pathways often remain active, even after surgical or medical intervention. This has led to increasing interest in adjunctive therapies aimed at modulating wound healing responses and inhibiting pathological angiogenesis.

Chronic exposure to ultraviolet radiation, dust, wind, and dry climatic conditions contributes to repeated epithelial injury and inflammation, particularly in exposed tissues such as the conjunctiva and cornea. These environmental triggers interact with biological susceptibility to create a sustained cycle of injury and repair that ultimately favors fibrovascular overgrowth. The identification of key signaling networks involving growth factors, matrix metalloproteinases, and inflammatory mediators has opened new avenues for therapeutic intervention. Anti-angiogenic agents, immunomodulatory drugs, and inhibitors of fibrotic signaling pathways are being explored as potential to control or reverse abnormal tissue growth. Although many of these approaches are still under investigation, they represent a shift from purely surgical management toward mechanism-based therapy. Fibrovascular proliferation should be viewed as a manifestation of dysregulated repair rather than a standalone pathology. It represents the intersection of inflammation, vascular biology, and extracellular matrix remodeling, all of which are essential components of normal healing but become harmful when persistently activated. Recognizing this continuum is essential for developing more effective for prevention, early detection, and treatment. In conclusion, fibrovascular proliferation is a complex and dynamic pathological process that reflects the failure of normal wound healing regulation. Whether occurring in ocular tissues such as

Correspondence to: Lucia Moretti, Department of Translational Medicine, University of Milan School of Medicine, Milan, Italy, E-mail: lucia.moretti@angiogenesisinstitute.edu

Received: 03-Mar-2025, Manuscript No. JCEO-25-42883; **Editor assigned:** 05-Mar-2025, PreQC No. JCEO-25-42883 (PQ); **Reviewed**: 18-Mar-2025, QC No. JCEO-25-42883; **Revised:** 25-Mar-2025, Manuscript No. JCEO-25-42883 (R); **Published**: 01-Apr-2025, DOI: 10.35248/2155-9570.26.17.1045.

Citation: Moretti L (2025). Fibrovascular Proliferation as a Central Mechanism of Dysregulated Wound Healing. Clin Exp Ophthalmol.17:1045.

Pterygium or in systemic conditions involving ischemia and chronic inflammation, its fundamental features remain consistent excessive fibrous tissue formation combined with abnormal vascular growth. Its clinical importance lies in its ability to disrupt normal tissue architecture, impair function, and contribute to disease progression.