Journal of Medical and Surgical Pathology

OPEN ACCESS Freely available online

Commentary

Morphogenetic Drift in Long-Term Surgical Scars

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DESCRIPTION

The formation of a surgical scar is an inevitable biological response to tissue injury, representing the body's attempt to restore structural integrity after the disruption caused by incision or excision. In its early stages, scar formation appears as a controlled sequence of inflammation, proliferation, and remodeling, all directed toward reestablishing a stable tissue surface. However, over extended periods, even mature scars that appear clinically stable may undergo subtle yet significant changes at the cellular and molecular levels. These long-term alterations, which gradually shift the morphology, texture, and biochemical organization of the scar, can be described as morphogenetic drift. This concept reflects the idea that scars are not static endpoints of healing but dynamic structures subject to slow, continuous remodeling influenced by mechanical forces, local metabolism, and aging.

Morphogenetic drift is rooted in the persistence of cellular and extracellular processes long after the apparent completion of wound healing. When a wound closes, the tissue enters a phase of remodeling where collagen fibers, vascular structures, and fibroblast populations gradually reorganize. In an ideal scenario, this remodeling phase concludes with the formation of a stable and minimally visible scar. Yet, in many cases, this equilibrium remains incomplete. The fibroblasts within the scar, often locked in a semi-activated state, continue to produce collagen and other matrix components in an unbalanced manner. Over time, these processes lead to a slow but measurable alteration in tissue architecture, producing morphological deviations from the initial scar pattern.

Cellular populations within scars also contribute to this long-term evolution. Myofibroblasts, which are the key contractile cells in wound closure, typically undergo apoptosis once healing is complete. In some scars, however, a subset of these cells persists, maintaining low-level contractile activity for years. Their continued presence induces slow remodeling of the extracellular matrix and progressive alteration of tissue tension. Additionally, resident fibroblasts within the scar exhibit altered gene expression profiles compared with those in normal dermis. Epigenetic changes acquired during the healing process may lock them into a state of partial activation, resulting in ongoing synthesis of fibrotic material. This slow and subtle activity fuels morphogenetic drift even decades after surgery.

The vascular component of scars undergoes a similar gradual transformation. The neovascular networks formed during healing often regress, leaving behind hypovascular regions. Reduced blood supply can alter the metabolic state of scar tissue, promoting hypoxia and oxidative stress. These conditions stimulate fibroblasts to produce denser, more cross-linked collagen, which stiffens the tissue furtherMechanical forces exerted on scars by movement, tension, and body growth add another layer to this phenomenon. Every scar exists within a field of mechanical stress defined by the surrounding skin, muscle, and connective tissue. Continuous tension along these vectors influences the orientation and density of collagen fibers. Scars located in high-motion areas such as joints or the abdomen often display more pronounced morphogenetic drift than those in immobile regions. Over time, the repeated mechanical strain stimulates mechanotransduction pathways in scar fibroblasts, leading to renewed matrix deposition and structural adaptation. This mechanical adaptation is rarely symmetrical or controlled, producing irregular thickening, elongation, or distortion of the scar.

Inflammation, even at a subclinical level, contributes to the long-term transformation of scar morphology. While most inflammation subsides after healing, minor immune activity may persist in response to mechanical irritation, microtrauma, or oxidative stress. Low levels of inflammatory cytokines and matrix-degrading enzymes can gradually modify the extracellular architecture. Chronic, mild inflammation promotes continuous turnover of collagen, elastin, and proteoglycans, resulting in slow remodeling and stiffening of the scar matrix. These biochemical processes occur too gradually to be perceived clinically in the short term, but their cumulative effect over years becomes evident as visible textural and color changes in mature scars.

Clinical observation supports the presence of morphogenetic drift in long-term scars. Scars that initially appear flat and well-healed may, after several years, become thickened, depressed, or irregular in pigmentation. The progression is typically slow, often unnoticed by the patient until the changes become prominent. In surgical reconstruction, these late alterations can

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Received: 27-Aug-2025, Manuscript No. JMSP-25-39072; **Editor assigned:** 29-Aug-2025, PreQC No. JMSP-25-39072 (PQ); **Reviewed:** 12-Sep-2025, QC No. JMSP-25-39072; **Revised:** 19-Sep-2025, Manuscript No. JMSP-25-39072 (R); **Published:** 26-Sep-2025, DOI: 10.35248/ 2472-4971.25.10.340

Citation: Baown L (2025). Morphogenetic Drift in Long-Term Surgical Scars. J Med Surg Pathol. 10:340.

compromise both cosmetic appearance and functional mobility. For example, scars near joints may gradually restrict range of motion due to ongoing contracture, while facial scars may distort contours as underlying tissues shift with age. Understanding the underlying biological basis of this phenomenon provides a rationale for long-term monitoring and preventive management of surgical scars.

CONCLUSION

Morphogenetic drift reveals that a surgical scar is not a static relic of injury but a living structure that continues to evolve under the influence of time, tension, metabolism, and aging. The slow reshaping of its architecture represents the long-term dialogue between cellular persistence and mechanical adaptation. Recognizing scars as dynamic tissues invites a new approach to postoperative care, emphasizing not only initial healing but also the lifelong maintenance of structural equilibrium. Understanding and addressing morphogenetic drift offers the potential to transform scar management from a reactive endeavor into a proactive strategy that preserves form, function, and harmony between the repaired and the original tissue.