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Opinion Article

Cellular Resilience Patterns in Surgical Wound Pathology

Oren Slokom*

Departments of Surgical Pathology, University of Bologna, Bologna, Italy

DESCRIPTION

The process of surgical wound healing represents one of the most complex and finely coordinated biological responses in the human body. Every incision or tissue disruption initiates a sequence of cellular and molecular events aimed at restoring anatomical continuity and functional integrity. While much emphasis has been placed on the mechanisms of inflammation, proliferation, and remodeling, an equally important yet often underappreciated concept is cellular resilience. This refers to the capacity of individual and collective cells to adapt, survive, and restore their function under the stresses imposed by surgical trauma. Understanding the patterns of cellular resilience within surgical wound pathology provides a deeper insight into why certain wounds heal efficiently while others develop into chronic or pathological forms.

Surgical injury subjects cells to multiple simultaneous insults including mechanical deformation, ischemia, oxidative stress, and inflammatory cytokine exposure. The initial phase of wounding produces abrupt physical and biochemical disruption that challenges the survival of resident tissue cells. In the immediate aftermath of incision, keratinocytes, fibroblasts, endothelial cells, and immune cells encounter abrupt loss of structural continuity, altered extracellular matrix composition, and fluctuating oxygen tension. The capacity of these cells to endure these stressors depends on intrinsic resilience mechanisms such as rapid metabolic adaptation, activation of stress response proteins, and reprogramming of gene expression to favor survival over specialized function.

One of the first observable patterns of resilience arises from the behavior of keratinocytes at the wound edge. These cells exhibit remarkable plasticity in response to injury. They temporarily suspend normal differentiation and adopt a migratory phenotype, enabling them to crawl over the provisional matrix and cover the exposed tissue. This transition requires extensive cytoskeletal remodeling and altered integrin expression, allowing attachment to new substrates. The success of re-epithelialization depends on the keratinocytes' ability to maintain viability under oxidative and inflammatory stress. Cells that sustain mitochondrial function and avoid apoptosis during this phase display the hallmark of epithelial resilience. Conversely, excessive stress or impaired energy regulation leads to keratinocyte death and delayed closure. Thus, epithelial resilience is a determinant of wound sealing and a key modulator of surgical healing outcomes.

Fibroblasts, which dominate the proliferative phase, exhibit a different but equally crucial pattern of resilience. Following surgical trauma, fibroblasts are recruited from surrounding tissue and bone marrow-derived precursors to the wound site. Their resilience is reflected in their ability to proliferate within a hostile environment rich in reactive oxygen species and proteolytic enzymes. They resist apoptosis through activation of survival pathways mediated by growth factors such as platelet-derived and fibroblast-derived signals. Moreover, fibroblasts adapt metabolically by increasing glycolytic activity to sustain energy production in hypoxic conditions. Those that display high resilience differentiate into myofibroblasts capable of generating contractile force, which contributes to wound contraction. When fibroblast resilience is inadequate or dysregulated, the wound may progress toward atrophy, excessive scarring, or chronic fibrosis.

Endothelial cells within the surgical bed demonstrate another dimension of resilience through their role in angiogenesis. The reestablishment of a microvascular network is essential for oxygen delivery and nutrient exchange in the healing wound. Endothelial cells respond to ischemic stress by undergoing phenotypic transformation, characterized by proliferation, migration, and tube formation. These processes are guided by hypoxia-induced signaling that stimulates the expression of proangiogenic factors. Cellular resilience in this context is defined by the ability of endothelial cells to tolerate transient hypoxia, oxidative injury, and inflammatory mediators without undergoing necrosis. When endothelial resilience is compromised, microvascular integrity fails, resulting in persistent ischemia and impaired healing. Conversely, excessive or uncontrolled endothelial proliferation may lead to abnormal vessel architecture, which can predispose to pathological granulation or hypertrophic scarring.

Molecularly, resilient cells exhibit distinct signatures involving stress-response genes, heat shock proteins, antioxidant enzymes, and autophagy pathways. These systems enable cells to repair

Correspondence to: Oren Slokom, Departments of Surgical Pathology, University of Bologna, Bologna, Italy, E-mail: slokom.oren@k56.it

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damaged proteins, remove dysfunctional organelles, and maintain redox balance. The presence of robust antioxidant capacity, especially through enzymes like superoxide dismutase and catalase, allows cells to buffer against the oxidative bursts that accompany inflammation. Similarly, autophagy serves as a resilience mechanism that prevents accumulation of damaged mitochondria and supports cellular energy equilibrium. These molecular patterns have been observed to correlate with more favorable surgical healing and reduced scar formation.

The metabolic state of the wound further determines how resilience manifests. Surgical wounds often exist in a transiently hypoxic environment, compelling cells to rely on glycolysis rather than oxidative phosphorylation. Those capable of efficient metabolic reprogramming can sustain biosynthesis and repair activities despite oxygen limitation. Metabolic resilience not only ensures cell survival but also prevents the accumulation of lactic acid and acidosis, which would otherwise impair matrix deposition. Studies have shown that enhancing cellular metabolism through controlled oxygen delivery or nutritional supplementation improves wound outcomes by reinforcing these adaptive mechanisms.

CONCLUSION

Ultimately, cellular resilience in surgical wound pathology represents the balance between destruction and renewal, stress and adaptation, survival and function. Each cell type contributes its own strategies to endure the dynamic and often hostile microenvironment of the wound. The orchestration of these strategies determines whether healing proceeds toward restoration or degeneration. Recognizing and reinforcing these resilience patterns not only advances the understanding of wound biology but also opens avenues for improving surgical outcomes through targeted cellular therapy, optimized perioperative care, and molecular interventions designed to enhance the innate strength of healing tissues.