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Perspective

The Role of Cytokines in Inflammation Driven Angiogenesis

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DESCRIPTION

Inflammation-driven angiogenesis is a fundamental pathological process involved in the progression of numerous ocular diseases that threaten vision. Angiogenesis refers to the formation of new blood vessels from pre-existing vascular networks and is a tightly regulated physiological process essential for embryonic development, tissue growth, and wound healing. Under normal conditions, angiogenesis is activated only when required and ceases once tissue repair is complete. In contrast, chronic inflammation disrupts the balance between pro-angiogenic and anti-angiogenic factors, resulting in uncontrolled vascular growth. Within the eye, excessive angiogenesis is particularly harmful because the normal architecture of ocular tissues depends on a precisely organized vascular system that supports retinal metabolism while preserving optical clarity. Persistent inflammation stimulates the continuous production of cytokines that regulate immune responses, endothelial cell function, and vascular remodeling. These cytokines act as key molecular mediators linking chronic inflammation with pathological angiogenesis in diseases such as diabetic retinopathy, age-related macular degeneration, retinal vein occlusion, uveitis, corneal neovascularization, and several ischemic retinal disorders. Understanding the role of cytokines in inflammation-driven angiogenesis has become increasingly important because it has transformed the diagnosis, treatment, and prevention of major causes of blindness worldwide.

The eye is a highly specialized sensory organ with unique anatomical and immunological characteristics. Structures such as the cornea, lens, and vitreous body are naturally avascular to maintain transparency and allow uninterrupted passage of light toward the retina. Conversely, the retina and choroid possess an extensive vascular network that supplies oxygen and nutrients to highly metabolically active neural tissues. This vascular system is maintained through a delicate equilibrium between molecules that stimulate blood vessel formation and those that inhibit excessive vascular growth. Chronic inflammation disturbs this equilibrium by activating immune cells that continuously release cytokines and chemokines. Persistent cytokine signaling stimulates endothelial cell proliferation, migration, survival, and increased vascular permeability, leading to abnormal neovascularization. Newly formed blood vessels are structurally immature, fragile, and highly permeable, allowing fluid, proteins, and blood to leak into surrounding retinal tissues. These pathological changes progressively damage retinal architecture and compromise visual function. Cytokines are low-molecular-weight proteins secreted by immune cells, endothelial cells, retinal pigment epithelial cells, Muller glial cells, fibroblasts, macrophages, and numerous other cell types present within ocular tissues. They function as intercellular signaling molecules that regulate immune activation, inflammatory responses, cellular proliferation, differentiation, apoptosis, and tissue repair. Under physiological conditions, cytokines coordinate protective inflammatory responses that eliminate pathogens and facilitate healing. However, persistent production of pro-inflammatory cytokines during chronic ocular disease creates a sustained inflammatory microenvironment that continuously stimulates angiogenesis. Tumor necrosis factor-alpha (TNF-α) is one of the most extensively studied cytokines involved in ocular inflammation and angiogenesis. It is primarily produced by activated macrophages, monocytes, retinal microglia, T lymphocytes, and retinal pigment epithelial cells in response to injury or infection. TNF-α initiates endothelial cell activation by increasing expression of adhesion molecules that promote leukocyte migration into inflamed ocular tissues. TNF-α stimulates endothelial cell proliferation and migration while increasing production of Vascular Endothelial Growth Factor (VEGF), the principal mediator of pathological angiogenesis. TNF-α also enhances vascular permeability by disrupting endothelial tight junctions, resulting in retinal edema and accumulation of inflammatory proteins within retinal tissues. Although vascular endothelial growth factor is classified as a growth factor rather than a cytokine, it represents the principal downstream mediator through which inflammatory cytokines induce angiogenesis. VEGF is produced by retinal pigment epithelial cells, Muller glial cells, endothelial cells, macrophages, astrocytes, and hypoxic retinal neurons in response to inflammatory stimulation. Persistent VEGF expression is responsible for the pathological neovascularization observed in proliferative diabetic retinopathy, neovascular age-related macular degeneration, retinal vein occlusion, and retinopathy of prematurity. Age-related macular degeneration also demonstrates the central role of cytokines in pathological angiogenesis.

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Aging, oxidative stress, complement activation, and genetic susceptibility contribute to accumulation of drusen beneath the retinal pigment epithelium. Anti-VEGF therapy has become the cornerstone of management for neovascular age-related macular degeneration, diabetic macular edema, proliferative diabetic retinopathy, and retinal vein occlusion by directly inhibiting abnormal blood vessel formation. Intravitreal corticosteroids suppress multiple inflammatory cytokines simultaneously, reducing vascular leakage and retinal edema. In addition to pharmacological treatment, control of systemic inflammation plays an important role in preventing ocular angiogenesis.