

Role of the Vitreous in Diabetic Retinopathy Progression

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Purpose: To demonstrate the role of the vitreous in progression of diabetic retinopathy (DR).

Methods: Review of the literature, ultrastructural and immunohistochemical data, clinical and surgical experience.

Results: The vitreous, and in particular the vitreous cortex plays an important role in DR progression. An attached posterior hyaloid is associated with the development of proliferative DR and diabetic macular edema. Spontaneous resolution of macular edema has been reported following PVD. In ultrastructural terms, the vitreomacular interface is characterized by a layer of vitreous cortex adherent to the retina in almost all eyes. Newly formed vessels grow into the vitreous cortex and follow vitreous structures further. A high number of cells is embedded in the vitreous cortex, including macrophages, fibroblasts, myofibroblasts, fibrous astrocytes, microglial cells, hyalocytes, and others. The cells are forming membranes which contribute to macular edema by exerting traction. In clinical terms, complete PVD rarely occurs spontaneously, and during vitrectomy, the vitreous cortex often comes off in several layers. Splitting of the vitreous cortex, so called vitreoschisis, leaves a layer of collagen back at the retina which can promote repopulation and disease progression. In addition, the vitreous cortex improves the molecular flux of oxygen and other molecules across the vitreoretinal interface.

Conclusion: Treatment strategies should aim at complete vitreous removal, including the vitreous cortex, thereby (1) eliminating the scaffold for fibrovascular proliferation, (2) relieving traction caused by fibrocellular proliferation, and (3) improving retinal oxygen levels.