

Diabetic vitreopathy and role in proliferative diabetic vitreo-retinopathy

Author: J. Sebag, MD, FACS, FRCOphth., Professor of Clinical Ophthalmology, Doheny Eye Institute, University of Southern California Los Angeles, California, USA

Invisible by design^{1,2}, vitreous is a viscoelastic extracellular matrix that normally exists in a gel state as a result of the intricate organization of its macromolecular components³. Hyaluronan (HA) and collagen, primarily type II but also type IX, and a hybrid of types V/XI, are organized into a three-dimensional network that maintains media clarity and provides shock-absorption. The peripheral vitreous cortex consists of densely packed collagen fibrils and has a high concentration of HA. In youth the posterior vitreous cortex is firmly adherent to the internal limiting lamina (ILL) of the retina⁴. While the exact nature of vitreo-retinal adhesion is not known, it most probably results from the biophysical properties of the extracellular matrix molecules found at this interface^{5,6}.

Diabetes causes elevated levels of glucose and advanced glycation end-products in vitreous^{7,8}. These induce structural changes within the corpus vitreus⁹ and at the vitreo-retinal interface that promote the migration and proliferation of vasculogenic cells. Traction upon new vessels growing into the posterior vitreous cortex¹⁰ worsens the prognosis in these patients. Posterior vitreoschisis¹¹ is another manifestation of diabetic vitreopathy that can cause vitreous hemorrhage. Diabetic vitreopathy¹² is also important in macular edema refractory to laser therapy.

Prevention via improved glycemic control and adjunctive therapy to prevent non-enzymatic glycation may mitigate against the effects of diabetic vitreopathy. The induction of posterior vitreous detachment via pharmacologic vitreolysis^{13,14} prior to onset of advanced disease may also greatly improve the prognosis.

1. Sebag J: The Vitreous - Structure, Function, and Pathobiology. Springer-Verlag, New York, 1989.
2. Sebag J: Guest editorial: Classifying Posterior Vitreous Detachment - a new way to look at the invisible. *Brit J Ophthalmol* 81:521-522, 1997.
3. Sebag J: Macromolecular structure of vitreous, In: Polymer Science and the Eye (TV Chirila, ed). *Prog Polym Sci* 23:415-446, 1998.
4. Sebag J: Age-related differences in the human vitreo-retinal interface. *Arch Ophthalmol* 109:966-971, 1991
5. Sebag J, Hageman GS: Interfaces. (Guest Editorial) *Eur J Ophthalmol* 10:1-3, 2000
6. Green WR, Sebag J: Vitreous and the vitreo-retinal interface. In: *Retina* (SJ Ryan, ed) Mosby, St. Louis, 2001; Vol III, pp 1882 – 1960
7. Sebag J, Buckingham B, Charles MA, Reiser K: Biochemical abnormalities in vitreous of humans with proliferative diabetic retinopathy. *Arch Ophthalmol* 110:1472-79, 1992..
8. Sebag J, Nie S, Reiser KA, Charles MA, Yu NT: Raman spectroscopy of human vitreous in proliferative diabetic retinopathy. *Invest Ophthalmol Vis Sci* 35:2976-2980, 1994.
9. Sebag J: Abnormalities of human vitreous structure in diabetes. *Graef Arch Clin Exp Ophthalmol* 231:257-260, 1993.
10. Faulborn J, Bowald S: Microproliferations in proliferative diabetic retinopathy and their relation to the vitreous – corresponding light and electron microscopic studies. *Graef Arch Clin Exp Ophthalmol* 223:130, 1985..
11. Chu T, Lopez PF, Cano MR, et al: Posterior vitreoschisis - an echographic finding in proliferative diabetic retinopathy. *Ophthalmology* 103:315-22, 1996
- 12.. Sebag J: Diabetic Vitreopathy (Guest Editorial). *Ophthalmology* 103:205-206, 1996.
13. Sebag J: Pharmacologic vitreolysis. *Retina* 18:1-3, 1998.
14. Sebag J: Is pharmacologic vitreolysis brewing? *Retina* 22:1-3, 2002.

