Introduction

In developing countries the incidence of diabetes is growing at an alarming rate. In industrialized countries proliferative retinopathy due to diabetes is the leading cause of blindness. (**Zimmet P et al, 2001**).

Proliferative diabetic retinopathy (PDR), the growth of new vessels from the retina or optic nerve, is thought to occur in response to ischemia driven release of vascular endothelial growth factor (VEGF) into the vitreous cavity (**Fong DS et al, 2004**).

The ischemia occurs because diabetes causes microvascular occlusion in the eye resulting in local hypoperfusion. The ischemic retina secretes VEGF, which leads to a number of related events in the eye (**Pe'er J et al, 2005**).

VEGF causes vascular leakage, and increased levels of VEGF are related to macular edema. Increased VEGF levels also can contribute to, although may not be solely responsible for, the formation of new vessels arising from the plane of the retina, which by definition is PDR. VEGF also has a curious property of causing microvascular occlusion, telangiectasis, and microaneurysm formation, all hallmarks of diabetic retinopathy itself (**Watanabe D et al, 2005**).

The microvascular occlusion can lead to increasing ischemia in a self-reinforcing cycle. Accompanying the proliferating vessels is fibroglial tissue, which with the vitreous, may contract and lead to hemorrhage because of traction on the proliferating vessels (**Sone H, et al, 1997**).

Laser photocoagulation currently is the principal therapy for sight-threatening PDR, unless the patient already has extensive vitreous hemorrhage, which would preclude the possibility of laser photocoagulation. Thus one of the principal complications of diabetic retinopathy inhibits its own treatment (**Aiello LP et al, 2004**).

Vitreous hemorrhage complicating PDR currently is managed one of two ways: observation hoping the blood will resorb enough to allow laser photocoagulation or surgical intervention to remove the blood and fibrovascular tissue and at the same time perform laser photocoagulation. However, a third option may be the inhibition of VEGF. A recombinant humanized monoclonal antibody directed against VEGF is available (bevacizumab, Avastin, Genentech) for cancer therapy. Intravitreal bevacizumab in humans has been previously described for the treatment of central retinal vein occlusion and age-related macular degeneration in single case reports (**Ruiz-Moreno et al, 2008**).