SUMMARY

Diabetic macular edema (DME) is the major cause of decrease visual acuity in diabetic individuals. The leakage of plasma in patients with DME visualized by flourescien angiography may be focal or diffuse. Focal macular edema, resulting form focal leakage of mircroaneurysms. However, diffuse macular edema, resulting from generalized breakdown of the inner blood retinal barrier.

Recently, retinal hypoxia has been implicated in the pathogenesis of DME. Hypoxia causes increased expression of vascular endothelial growth factors (VEGF), which is a potent inducer of vascular permeability that has been shown to cause leakage form retinal vessels. Thus, it is reasonable to hypothesize that VEGF contribute to DME.

Previously, described methods of assessing DME include contact and non contact slit lamp biomicroscopy, indirect funduscopy, flourescien angiography, and fundus stereo photography.

Alternative objective methods such as the retinal thickness analyzer and the Heidelberg retinal tomography have been applied. The introduction of optical coherence tomography (OCT) further allow for objective evaluation of DME. OCT also has been shown to be effective in both the qualitative and quantitative description of DME.

Focal laser photocoagulation reduce the risk of visual loss in eye with clinically significant macular edema. Visual acuity had improved in 14.5%, unchanged in 60.9% and decreased in 24% after 3 years of initial grid laser treatment in the eye with diffuse macular edema.

Pars plana vitrectomy (PPV) with or without internal limiting membrane removal has been reported to decrease macular edema and improve visual acuity in patients with DME.

Triamcinolone acetonide, a cortisone suspension has been proposed to decrease macular edema in mean of their anti-angiogenic, anti-inflammatory and blood retinal barrier stabilizing effect.

Intravitreal triamcinolone acetonide has been offered as an alternative theraphy for DME resistant to conventional laser photo coagulation without any toxicity.

Vascular endothelial growth factors (VEGF) are vascular permeability factors, they induce vascular fenestrations as well as an increase in permeability of microvessels leading to deposition of protein in interstitium that facilitate the process of angiogenesis. Increase level of VEGF also lead to Macular edema.

One possible strategy for treating retinal neovascularizton and macular edema is to inhibit VEGF activity by competitively binding VEGF with specific neutralizing anti VEGF antibody. Bevacizumab (avastin), a humanized mono clonal anti VEGF antibody used as an advanced therapy in ophthalmology. Recent clinical data of the intravitreal use of Bevacizumab (avastin), show excellent results in the treatment of macular edema.