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Short Communication

Diabetic Macular Edema

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Diabetic macular edema (DME) is the most common cause of visual impairment in patients with diabetes mellitus. It occurs due to disruption of blood retinal barrier hence resulting in excessive accumulation of fluid within intraretinal layers of macula. DME can occur at any stage of diabetic retinopathy and is caused by excessive vascular permeability resulting in the leakage of fluid and plasma constituents leading to secondary thickening and distortion of the central retina, together with the stretching of neurons and an initial reversible loss of vision. Although spontaneous recovery is not uncommon, 24% of eyes with Chronic significant macular edema (CSME) and 33% of eyes with center involving CSME develop a moderate visual loss (15% or more letters on the ETDRS chart) within 3 years if untreated [1,2].

Grid laser photocoagulation is beneficial for reducing vision loss in patients with DDME. However, it causes visible laser scars that can enlarge postoperatively, leading to decreased vision [3,4]. Even when carefully performed by conventional technique, grid photocoagulation consumes more laser power against ''oedematous'' retina. Laser photocoagulation is associated with side effects like decreased visual field [5,6]. Grid photocoagulation acts by opening of new pathways in the retinal pigment epithelium barrier for fluid transportation and/or a decrease in the photoreceptor population, thereby decreasing oxygen demand leading to reduced blood flow and resolution of macular oedema.

Laser absorption and thermal injury have to act on the retinal pigment epithelium located on the outer most part of the retina, and the laser energy reaches this area via retinal tissue. Therefore, the increased retinal thickness will be associated with more laser power required for retinal photocoagulation. Also, some cytokines, such as interleukin-6 and interleukin-8, related to tissue inflammation are induced by laser photocoagulation, which exacerbate the exisiting macular oedema [7,8].

To diminish intrinsic damage from visible endpoint laser photocoagulation, treatment with posterior subtenon Triamcinolone acetate injection and Bevacizumab before grid treatment against DDME has been proposed. Triamcinolone amplifies the tight junction proteins and local vasoconstriction and decreases the stimulation of VEGF caused by pro inflammatory mediators (pigment derived growth factor and platelet activating factor). It can be administered through subtenon, peribulbar or intravitreal route. Intravitreal anti VEGF inhibitors play an important role by not only decreasing the release of vascular endothelial growth factors but also by causing regression of neovascularization. Numerous studies have assessed the efficacy of intravitreal bevacizumab and Posterior Subtenon Triamcinolone independently and both have been found to be effective [8,9].

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