

Simplified Management of Diabetic Macular Edema for Anterior Segment Surgeons

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Diabetic Macular edema (DME) is one of the most common causes of diminution of vision in diabetic patients besides vitreous hemorrhage, epiretinal proliferations, tractional retinal detachments, macular ischemia and diabetic papillopathy. In a study [1] for patients whose age at diagnosis of diabetes was less than 30 years and who were on insulin treatment, prevalence rates of macular edema varied from 0% in those who had diabetes less than 5 years to 29% in those whose duration of diabetes was 20 or more years, while for those whose age at diagnosis was 30 years or older, prevalence rates of macular edema varied from 3% in those who had diabetes less than 5 years to 28% in those whose duration of diabetes was 20 or more years.

Diagnosis of DME depends on clinical observation of microaneurysms, exudates, hemorrhages, or thickening in the macular area [2]. This should be followed by fundus fluorescein angiography for the sake of staging of the diabetic retinopathy as mild, moderate, or severe non-proliferative diabetic retinopathy, or proliferative diabetic retinopathy. Detecting peripheral ischemia is of utmost importance for laser ablation of ischemic areas which are a source of Vascular Endothelial Growth Factors (VEGF) which are from the main causative factors of macular edema. Fluorescein angiography also detects the pattern of macular leakage as focal, multifocal, or diffuse leakage maculopathy which could be of benefit when adding laser treatment to anti-VEGF injections as will be explained.

Optical Coherence Tomography (OCT) is the main tool for DME diagnosis and follow up [2]. A lot of information can be obtained from OCT scans as presence or absence of traction, average central 1 mm thickness, the presence of central or non-central involving macular edema, the state the photoreceptors and the external limiting membrane, and follow up after treatment.

Treatment of DME involves systemic glyceamic, blood lipids, and blood pressure control [3]. Treating the peripheral ischemia in severe non-proliferative and proliferative diabetic retinopathy by pan-retinal photocoagulation controls the diabetic retinopathy and decreases VEGF vitreous loads. Intra-vitreous injection of anti-VEGF is the mainstay for treatment of visually significant DME. The following algorithms summarize our approach in treatment of DME, generally classified into non-central involving Diabetic Macular Edema (Nci-DME) and central involving diabetic Macular Edema (Ci-DME). It is crucial to point out that we refer diabetic patients with any tractional elements for management by vitreo-retinal specialists.

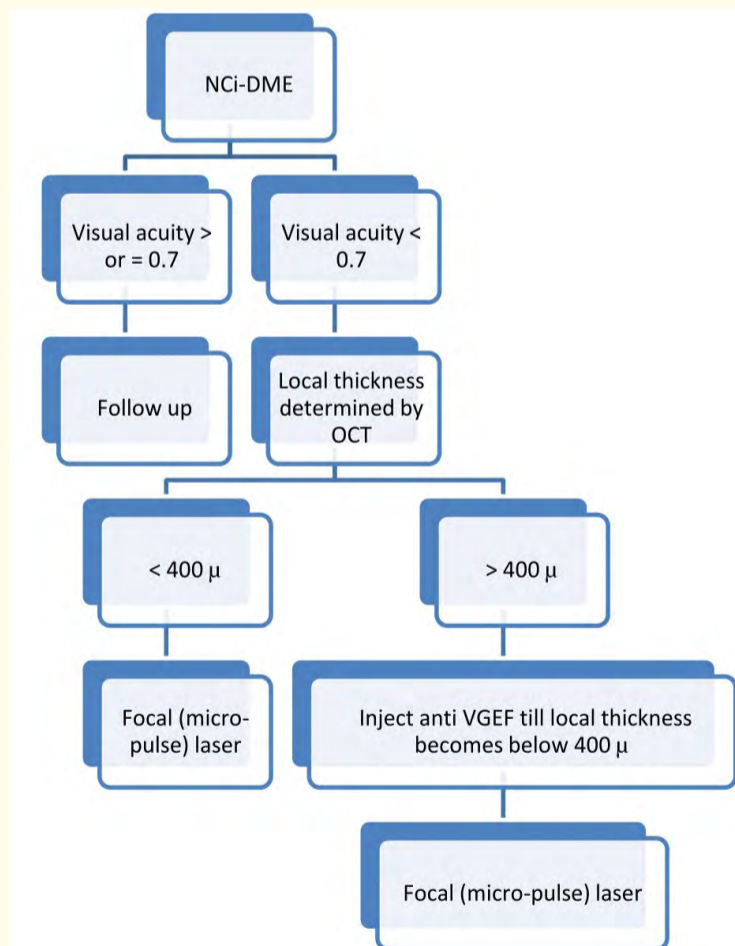


Figure 1: Diagram showing plan of treatment for Non-Ci DME.

N.B: focal laser scars can be destructive and expand in size by time, that is the reason we prefer doing micro-pulse laser nowadays. Although there is not enough evidence, we believe from our experience that micro-pulse laser does not have the destructive effect the conventional laser has, and can decrease the frequency of injections for non-Ci DME.

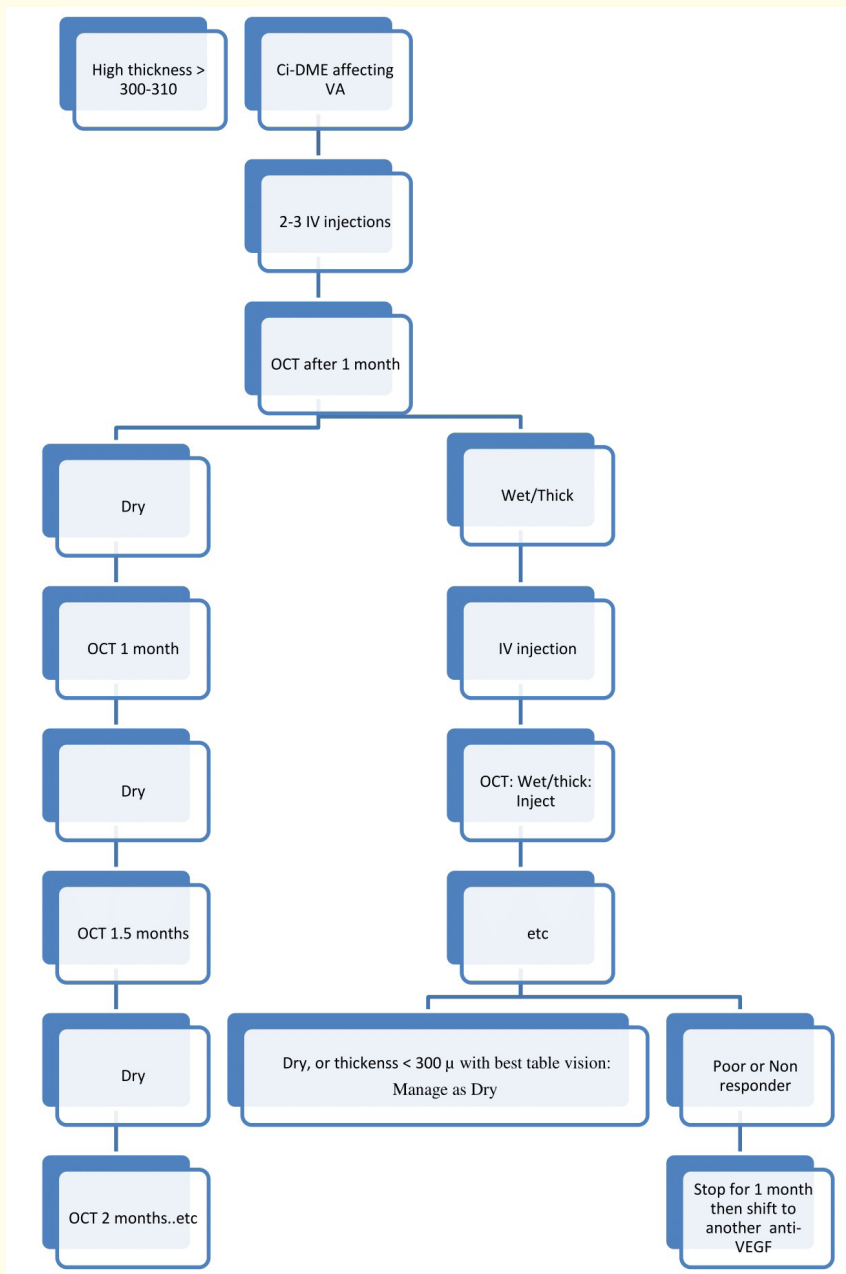


Figure 2: Diagram for management of Central involving edema (Ci-DME).

We consider success after an injection as a decrease in central thickness at least 10% on OCT follow up, while poor responders show no improvement in thickness > 10% on OCT after 3 - 5 injections. In addition, late responders show improvement which begins after 3 to 5 injections. Sometimes a patient improves in thickness (< 300μ) and visual acuity, and stabilizes after 2 - 3 injections with satisfactory vision and no further improvement, in these cases, follow up may show stability for several months even without switching of drugs, which is according to each doctor’s preference as there is still no enough published evidence on switching between anti-VGEFs.

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