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# Diabetic macular edema: An overview

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Dear Friends

Season's Greeting !!

Diabetes mellitus is a leading cause of morbidity in Indian subcontinent.[1] Diabetic retinopathy is a form of micro-vasculopathy involving retinal capillaries in the earliest stage presenting as microaneurysms and leading to micro-vascular leakage, retinal haemorrhages, exudates which may lead to vision loss. End stage diabetic retinopathy involves proliferation of blood vessels over the optic nerve, retina, iris and anterior chamber angle, finally leading to tractional retinal detachments and neovascular glaucoma. It remains one of the major causes of new-onset visual loss in developed countries. If the central part of the retina (i.e., the macula) is involved, it is referred to as diabetic maculopathy. This is the most common cause of vision impairment in individuals with diabetic retinopathy.[2]

Diabetic Maculopathy is defined as hard exudates or retinal thickening involving the macula. Diabetic maculopathy has been divided into four types.

a. Non clinically significant macular edema b. Clinically significant macular edema (CSME). Focal / diffuse c. Ischemic maculopathy d. Mixed type.

It is commonly diagnosed by means of investigations:

1. Fundus photography: Diagnosis of DR is essentially clinical but fundus photography is the best way to record the retinal changes. It helps both in screening as well as to record the progression of retinopathy. A seven field stereo fundus photography is the standard procedure to record the fundus images. Now a day ultra wide field imaging is used primarily as a screening device in diagnoses of DR18. Wide field imaging allows visualisation of up to 200º of peripheral retina captured in a single image.
2. Fundus Fluorescein angiography is not required for screening or diagnosing DR as it can be done clinically but it can be used to classify diabetic macular edema (DME) into focal or diffuse and its treatment.
3. It can also diagnose cystoid macular edema and ischemic maculopathy in defining the extent of capillary non perfusion areas
4. Optical Coherence Tomography (OCT) is a non invasive imaging technique that provides high resolution cross section imaging of the retina, retinal nerve fibre layer and optic nerve head. OCT helps in early diagnosis of macular oedema as it is more sensitive than clinically examination, in deciding the treatment options for DME and also helps to monitor the response of the treatment options available for DME.[3] OCT can help in detecting retinal nerve fibre layer loss as well as early development of oedema by macular thickness measurement in clinically asymptomatic patients.
5. B Scan ultrasonography is a non-invasive tool for imaging intraocular contents in cases of hazy media, leading to difficulty in fundus visualisation. Vitreous haemorrhage, posterior vitreous detachments and tractional retinal detachments can be easily visualised through B scan.

Ophthalmic management of diabetic maculopathy depends on the location and extent of macular thickening. Patients with non centre-involving clinically significant macular oedema (CSMO) may be treated with laser photocoagulation.[4] Patients with centre-involving macular oedema and reduced vision would benefit most from anti-VEGF treatment with or without combination laser treatment. Intravitreal steroid treatment (preservative-free) combined with post-treatment argon laser treatment may be considered particularly in pseudophakic patients, but bearing in mind the risk of raised intraocular pressure.[5,6] For those patients who have been unresponsive to other treatment, the intravitreal fluocinolone implant may be considered but taking into consideration the side-effect profile.[7] Patients unwilling or unsuitable for intravitreal injections may be offered macular laser treatment.

Patients with centre involving macular oedema and good visual acuity (>6/10) may be observed if the leaking microaneurysms are very close to fovea and there are no other treatable lesions suitable or safe to laser, otherwise laser photocoagulation treatment may be considered. Patients with poor visual acuity (below 6/90) may be observed especially if the macular oedema is long standing and there is considerable macular ischaemia. Alternatively intravitreal anti-VEGF or intravitreal steroid preparations may be considered with full consultation and informed consent of the patient.[8,9]

If there is evidence of vitreomacular traction on the OCT scan, vitrectomy may be considered with or without adjunctive anti-VEGF/steroid treatment. Microplasmin injections may be considered as an option when available. 3 (-4) months follow-up is appropriate following macular laser, as long as no other features are present that require more regular follow-up.

For patients undergoing anti-VEGF treatment the evidence shows that patients should be treated with an initial loading phase of 4-6 monthly injections, followed by monthly follow-up with OCT, with continued treatment until the macula is dry or until there is no further improvement. After year 1, the period of time between follow-up appointments may be gradually increased if the eyes are stable off treatment, to a maximum of 12-16 weeks in years 2-3. For patients undergoing intavitreal steroid treatment, regular follow up will be required with OCT scans, IOP monitoring and repeated treatments as required with the aim to keep macula dry.

Patients with early maculopathy (but no CSMO) and background retinopathy (R1) may be followed up in Ophthalmic Imaging assessment Clinics with colour images and spectral domain OCT, at 4- 6 monthly intervals.

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