Management of DME

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Diabetic retinopathy is the most common cause of retinal vascular diseases and diabetic macular edema is the most common cause of vision loss in these patients. Traditionally, diabetic macular edema has been treated by laser photocoagulation. However, after the advent of intravitreal pharmacological agents, the treatment paradigm has shifted from laser to pharmacotherapy. Newer forms of laser are also available that lower the damage caused by conventional laser. With the increasing number of diabetic patients, the prevalence of DME is also increasing. The experts share their knowledge and experience about the changing management patterns and their preferred choices.

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(ASG): In a patient of DME, what all baseline investigations- both systemic and ocular, do you advice before starting treatment?

(AK): Comprehensive systemic investigations include HbA1C levels, Blood sugar both fasting and postprandial, complete blood count, lipid profile, renal function tests including serum creatinine and a check on blood pressure. Ocular investigations that we routinely advice include baseline Fundus picture, OCT (Spectral domain or swept source) and Fundus fluorescein angiography (30 degree, 55 degree, 102 deg or ultra wide field – tailored to each individual). With the availability of OCT-A the vascular architecture including FAZ can be traced easily and becomes essential when we suspect macular ischemia.

(CS): Systemic -Blood Sugar (F and PP), HbA1C, Hemogram, Lipid Profile, KFT and BP
Ocular:- OCT, FFA/OCT A.

(LV): Systemic: Hb, HbA1c Serum lipid profile, BP, Kidney function tests, 24 Hr Urinary Protein; Ocular: BCVA, OCT, FFA.

(RN): Baseline investigations are extremely important in the management of diabetic retinopathy (DR) as such. However, they should not be considered as a barrier to the treatment of diabetic macular edema (DME) in most patients. The first visit to an ophthalmologist is sometimes a wake-up call for the patient as well the physician to have strict control of DM. Good control of sugar, blood pressure, lipids, renal function and anemia have a positive effect on the long-term outcome of DR. Various studies such as DCCT, UKPDS and ACCORD have reported on the need for good control of diabetes. Ocular Investigations: Visual acuity with refraction, look for NVI/ NVA, pupils, fundus examination with good documentation by drawing or fundus photo. OCT is usually sufficient to manage DME. Do not miss examining the periphery for signs of PDR. OCTA/ FA is useful only if there is a mismatch between clinical findings and visual acuity. Systemic Investigations: Our Institute has a well defined protocol for managing patients with Diabetes. The normal profile of investigations includes fasting and Post-prandial blood sugar, blood pressure, renal function test, lipid profile, HbA1c.History is important in all patients, though most ophthalmologists do not spend adequate time eliciting history. This helps in identifying the difficulties the patient has been facing in controlling diabetes, including the fact that they may already be on Insulin and struggling with episodes of hypoglycemia. If a patient has a recent history of insulin treatment, one can expect more cotton wool spots on examination of the fundus. NVE may be subtle in such patients. In cases of multiple previous treatments for DME, I also enquire about sleep apnea, and check their hemoglobin for anemia.

Summary: *Most experts recommend baseline systemic investigations including blood sugar- fasting and post prandial, HbA1c, complete haemogram, kidney function tests, lipid profile, blood pressure monitoring and urinary protien. Ocular investigations recommended at baseline are fundus picture, OCT macula and FFA. OCT-A may help in suspected macular ischema cases.*

(ASG): In today's scenario, what is the role of FFA in the management and follow up of DME?

(AK): FFA is useful to differentiate focal and diffuse leaks in cases of recalcitrant DME. Initial treatment can be started based on OCT findings but FFA might be required in the follow-up if the response to antiVEGF is poor. Baseline FFA is mandatory in patients with associated proliferative diabetic retinopathy to identify points of leakage and to examine the FAZ distortion and to rule out macular ischemia. In severe NPDR cases FFA is performed to identify unnoticed neovascularization. With the advent of Ultrawide field FFA, peripheral non perfusion areas can be picked up easily. Targeted limited peripheral scatter of the peripheral non perfusion areas is usually beneficial in early PDR, severe NPDR and recalcitrant DME cases.

(CS): FFA is important to check macular perfusion and also to detect any co-existing PDR/PPDR when starting treatment and to check for peripheral non perfusion in refractory cases.

(LV): 1. FFA may help to pick up focal leaks in DME
2. Also useful to know presence of associated Neovascularization
3. Presence of CNP, picked on FA, may influence management of DR/DME

(RN): Most patients who present to us have centre involved DME. I rarely advise FFA in DME. In fact, I use it more to confirm or rule out subtle neovascularization, though their primary pathology could be DME. I would often advise OCTA rather than FFA when I suspect a significant component of ischemia. Ischemia should be suspected when the visual acuity does not correlate with the clinical findings.

Summary: *Most experts recommend baseline FFA to look for macular ischema, diffuse or focal leaks at macula, any neovascularisation of retina and peripheral capillary non perfusion areas for targeted laser. However, in centre involving DME and early NPDR, FFA can be delayed. With the advent of OCT-A, the role of FFA for defining macular ischemia has also diminished (Figure 1).*

(ASG): What are the features on OCT/FFA that you look for before deciding what treatment to start - laser or intravitreal injections and the choice of injection?

(AK): My first line of therapy for a centre involving DME will be antiVEGF agent. A base line OCT will guide us in confirming whether the DME is centre or non-centre involving and in quantifying the CMT (CMT > 275 microns with vision <= 6/9 is considered as an indication). OCT helps in analyzing the type of DME (cystic/ spongiform/ neurosensory detachment/ tractional). Cystic and NSD - DME usually responds

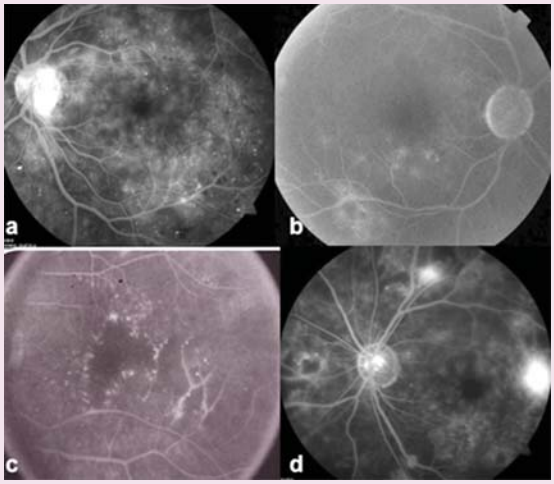

Figure 1: *FFA showing a) Diffuse macular leakage,* ***b)*** *Focal macular leakage,* ***c)*** *Ischemic maculopathy,* ***d)*** *Neovascularisation of retina elsewhere.*

well with anti VEGF whereas the spongiform type shows poor response and is usually associated with Diabetic nephropathy. Tractional DME may require vitrectomy. DRIL (disorganization of retinal inner layers) is picked up on OCT and helps in predicting the visual acuity and suggests poor prognosis. FFA helps in differentiating focal and diffuse DME. Focal non - centre involving DME can be lasered at the baseline. FAZ irregularities/ distortion and enlargement, points towards macular ischemia and can be picked up. Further peripheral ischemic areas can be identified on FFA and lasered in recalcitrant DMEs.

(CS): Non-centre involving macular edema requiring treatment and if causative lesions are amenable to laser treatment go ahead with focal laser.

Centre involving macular edema – Anti-VEGF injections are first line.

Anti-VEGF followed by pan retinal photocoagulation if NVD/NVE seen.

(LV): Treatment in DME depends on Info obtained from: OCT + FFA + BCVA (All combined Info – Not Isolated OCT):

- If OCT shows VMT / ERM : Consider Vitreous Surgery , depending upon BCVA / One-Eyed status etc;
- If OCT shows increased thickness of > 300 and BCVA is < 6/9 – start with Intravitreal Anti VEGF – If inadequate response to 3 monthly Injections – consider Ozurdex;
- If OCT is ++ for edema and BCVA is > 6/9 : Pay attention to Metabolic parameters; Giving Intravitreal Injections in such situations is debatable;
- If OCT thickness is okay and there is no tractional element also : One should see FFA :
- If No leaks / CNP / Neovasc: Follow up this patient;
- If laserable lesions identified: Do appropriate Laser and Follow up.

(RN): Any treatment naïve patient with OCT documentation of centre-involved DME is advised intravitreal injection. Fluid on OCT is the most important factor to decide to go for injections. However, one should look for DRIL and disruption of outer retinal structures to prognosticate the final visual outcome. A naïve patient with edema but having DRIL or disruption of outer retinal structures would still benefit with injections, though is unlikely to get 20/20. I advise micropulse laser in DME not involving the centre. My protocol for chronic DME, previously treated with injections, would differ. I have explained my thoughts in the next few questions.

Summary: *OCT macula helps in defining the type of CME, whether centre involving or non-centre involving, presence of poor prognosticating factors like DRIL and quantifying the CMT. All experts recommend laser (focal/micropulse) to non-centre involving DME. For centre involving DME, the first choice is Anti-VEGF agents. FFA helps in defining focal macular leaks and peripheral CNP areas for laser (Figure 2).*

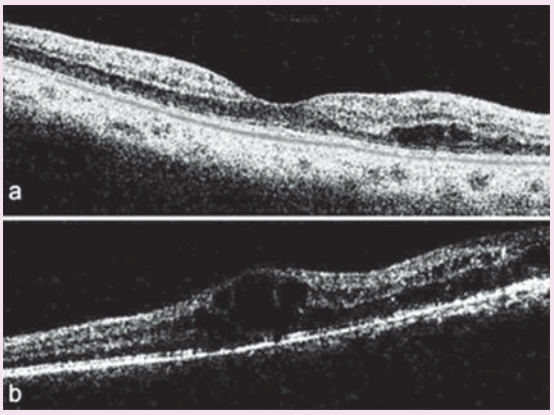

Figure 2: *OCT macula showing a)non-centre involving DME, b) Centre-involving DME.*

(ASG): In a poorly controlled diabetic with DME, do you start treatment for DME immediately or after metabolic control? If you decide to wait, then how long do you wait and till what value of fasting blood sugar/HbA1c?

(AK): A good metabolic control is mandatory for the best response to treatment of DME. Moreover anti-VEGF's being the first line treatment in DME, a good metabolic control and systemic stabilization is required to decrease risk of post-intravitreal endophthalmitis. A fasting sugar of < 125 mg/dl and HbA1c < 7 is desirable however in cases with severe macular edema with increased central macular thickness and NSD, treatment should not be delayed as optimization of systemic status may take time especially in chronic cases and the prognosis may worsen in cases with long standing untreated macular edema. Thus the decision of treatment should be individualized in each case taking into consideration the systemic status and amount of macular edema. So in a patient who

comes to me with creatinine > 4.0mgs% suggestive of CKD ,would be sent for Nephrology expert advice regarding reducing fluid intake etc and I would wait at least 2 weeks prior to ordering a repeat OCT scan and then the anti-VEGF injection. Management of Diabetic macular edema involves comprehensive diabetic care requiring a holistic approach involving the ophthalmologist working in tandem with the endocrinologist, dietician and nephrologist with systemic optimization being the centre stone of treatment.

(CS): Start pharmacological treatment concurrently with trying to improve metabolic control.

(LV): Both Metabolic control and Injection treatment of DME can go parallely. However laser (if indicated) should be avoided in an edematous retina.

(RN): As such, I do not have a cut-off of HbA1c, and I do not delay treatment in DME patients. Outcomes of intravitreal injections for DME are independent of HbA1c levels. Typically, DME studies have excluded patients with HbA1c above 12%, and hence it is difficult to comment on extreme situations. As an Institutional protocol, we inject in patients with RBS less than 250 mg%, though we prefer to get it down to 200 mg%. Simultaneous intensive systemic control is started by our physician.

Summary: *Experts recommend starting pharmacological treatment concurrently with intensive systemic control. They do not recommend delay in treatment if blood sugars are high. DME studies have excluded patients with HbA1c >12% from treatment.*

(ASG): What is your first choice of Agent for Intravitreal Injection in a Phakic Patient with treatment of naive DME?

(AK): The DRCR protocol I / RISE/READ trials support good improvement in visual acuity in the ranibizumab group. My first choice will be injection ranibizumab – 0.5mg. Ranibizumab provides good results with least risk of endophthalmitis. It is a smaller molecule (Mol wt 45Kd) so retinal penetration is thought to be higher. If the patient is non-affording then bevacizumab is a good alternative.

(CS): Anti-VEGF.

(LV): Anti VEGF agent is the First Choice and of the 3 available Anti VEGF`s,
(Ranibizumab, Bevacizumab, Aflibercept), Ranibizumab is preferred for reasons of safety, efficacy and extensive data based on large number of Trials. Avoid Bevacizumab for reason of safety. Keep Aflibercept in reserve for reasons of costing in our country.

(RN): Anti-VEGF is my first choice.

Summary: *Experts recommend Anti-VEGF as first choice in phakic patient with treatment of naive DME.*

(ASG): What is your first choice of Agent for Intravitreal Injection in a Pseudophakic Patient with treatment of naive DME?

(AK): My first choice will still be an antiVEGF injection, often Ranibizumab. If the results are not gratifying after 5-6 monthly injections I would label the eye as having recalcitrant DME and offer my next choice, a sustained-release biodegradable steroid implant (Ozurdex- which is Dexamethasone 700µg) provided there are no issues of pre-existing glaucoma.

(CS): Anti-VEGF.

(LV): Answer still remains the same – as in Ques 5 above.

(RN): Anti-VEGF is my first choice in most cases. Ozurdex could be first choice if patient has good vision at baseline (better than 20/40).

Summary: *Experts recommend Anti-VEGF as first choice in pseudophakic patient with treatment of naive DME.*

(ASG): What is your criteria for response to Intravitreal Injection and when do you label the patient non-responsive to a particular drug?

(AK): Response is considered good if there is ≥ 25% decrease in CMT morphologically on OCT after at least 3 consecutive doses of monthly anti-VEGF injections. Functional response is graded as good when the VA improves by ETDRS 5 letters or Snellens - one line. Clinically recalcitrant DME is suspected when poor response is seen with at least 3 doses of anti-VEGF injections.

(CS): Decrease in macular thickness by 50µ and/or resolution of NSD.
Non-responding if no improvement after 3 injections.

(LV): For Good Clinical response patient should show improvement in BCVA of atleast one line and / or OCT thickness decrease of at least 10-20 %. Maximum improvement occurs in the initial 2-3 Injections. If there is no improvement with initial 3 injections – I label it as Non-Responder/ Refractory.

(RN): I usually look at the response after 1 injection at 1 month.

a. If there is worsening of DME or no improvement after 1 injection-switch to Ozurdex.

b. Marginal or good improvement after 1 injection-Continue with 2 more anti-VEGF injections.

c. Good improvement after every injection (macula gets dry after injection) but continued recurrence after 3 injections-switch to Ozurdex.

d. Persistent fluid after 3 anti-VEGF injections with only marginal improvement (macula never dry) switch to Eylea or Ozurdex.

Summary: *Experts have different criteria for evaluating response on OCT: Decrease in macular thickness by at least 50µ, resolution of NSD, ≥ 20-25% decrease in CMT. Most experts recommend labelling as non-responsive after at least 3 monthly injections (Figure 3).*

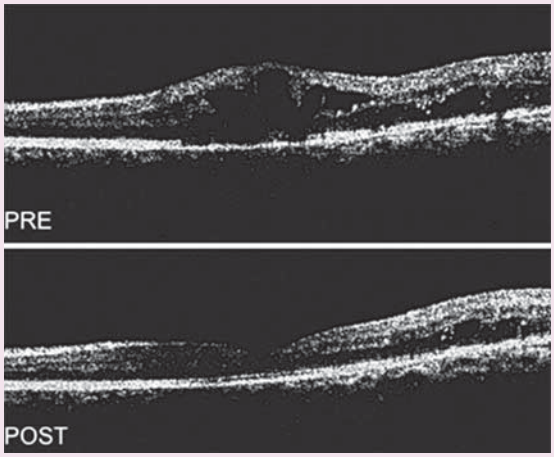

Figure 3: *OCT macula showing resolution of centre involving DME after 2 monthly intravitreal injections of Anti-VEGF.*

(ASG): When do you shift from one agent to the other while treating DME?

(AK): I usually advise, change of antiVEGF in recalcitrant DME. When the patient is showing poor response to anti –VEGF injections, inspite of good glycemic control and no obvious peripheral retinal ischemia a switch of antiVEGF agent might work. Switch over from ranibizumab to aflibercept is a useful option available in eyes with >350µm CMT despite 5-6 antiVEGF injections.

(CS): When not responding as mentioned above or systemic reason to avoid anti-VEGF / ocular reason to avoid steroids.

(LV): Shifting amongst the 3 available Anti-VEGF's (Ranibizumab, Bevacizumab, Aflibercept) does not regularly help. My preference is to start Intravitreal Steroids (Ozudrex).

(RN): As above in question 7.

Summary: *Experts recommend shifting from one agent to the other if there is no response to the current therapy or if there is a systemic or ocular contraindication to the current therapy. The switch can be from one Anti-VEGF agent to the other or to intravitreal steroid and vice versa.*

(ASG): What is the role of focal/ grid laser in the era of intravitreal pharmacotherapy?

(AK): I prefer antiVEGF injections over focal/grid laser. Focal laser can still be performed in obvious cases (non-centre involving focal macular edema – with leaking MCA'a or even a visible circinate retinopathy). In centre involving macular edema – laser is considered as the last resort when other measures fail. As supported by the protocol I, if the response is unsatisfactory after 6 months – then I consider focal/ diffuse laser to non-centre involving leaky lesions. Other options like steroid therapy and switch over anti-VEGF therapy can also be tried according to the individual.

(CS): Focal laser for extra-foveal lesions. Don't do grid laser.

(LV): Classic Grid is history. In Non- Central Involving DME with focal leaks – Focal Laser with 532 laser gives lasting results. Even in central involving DME, if FA shows focal leaks, tend to add laser after reducing macular thickness by Intravitreal Pharmacotherapy.

(RN): Rare. I use more of micropulse laser in non-centre DME.

Summary: *Experts recommend focal/micropulse laser for non-centre involving DME. However, Anti-VEGFs remained the preferred treatment.*

(ASG): In a patient with a recent history of CAD or CVA, do you give intravitreal anti-VEGF? If yes, which Anti-VEGF do you prefer and what is the minimum duration that needs to be kept between the episode of CVA/CAD and the injection?

(AK): Yes recent history of CAD/stroke is a relative contraindication for anti VEGF. A 3 months interval between the episode and the injection is desirable. Systemic level of bevacizumab is comparatively higher than ranibizumab/aflibercept. A three year stroke rate was found to be least (4.3%) with ranibizumab. Hence ranibizumab is preferred in such a scenario.

(CS): Don't give.

(LV): If history of CVA / CAD if less than 3 months – Desirable to Avoid Intravitreal Anti-VEGF treatment. Prefer Intravitreal steroids (Ozurdex) in such cases.

(RN): I do give in patients with CAD or CVA, but after 3 months of the episode if required. While caution is emphasized, it is good to discuss with the physician who is knowledgeable about anti-VEGF drugs and the eye (such physicians are rare). There is no point in trying to shift the responsibility to some other specialty, who do not have enough understanding of the data from Ophthalmology. Personally, I do not believe that anti-VEGF increase the risk of stroke/ CVA. This is my interpretation of multiple trials/ meta-analysis. Having said that, DME is not an emergency, and steroids are always an option.

Summary: *Experts recommend a minimum duration of 3 months between the episode of CVA/CAD and intravitreal Anti-VEGF injection with involvement of a physician. Ranibizumab/Aflibercept appear to be safer than bevacizumab. The patient can be given intravitreal steroids instead.*

(ASG): What is the role of vitrectomy in the management of DME? When do you opt for it?

(AK): I prefer pars plana vitrectomy with or without ILM peeling only for tractional macular edema – (vitreomacular traction or thick ERM often with a thick taut hyaloid on OCT). A trial of antiVEGF or intravitreal steroids can be practiced (according to individual cases) before planning vitrectomy. Only in cases with clear-cut evidence of VMT or ERM disturbing the retinal architecture with associated visual disturbances, I plan for a vitrectomy.

(CS): When there is definite Tractional DME. Not convinced it helps in non-tractional refractory DME.

(LV): Vitrectomy in DME works the best in the presence of Tractional element (VMT / ERM). In Non Tractional DME – Visual results after Vitrectomy are not as great and therefore consider it only after having exhausted with Intravitreal Pharmacotherapy (Anti VEFG and Steroids).

(RN): The role of PPV in DME is at best controversial. Most studies have shown anatomical improvement, but visual improvement is not significant. I have personally had mixed results in my experience.

Summary: *Experts recommend pars plana vitrectomy in only tractional macular edema (VMT/ ERM) (Figure 4).*

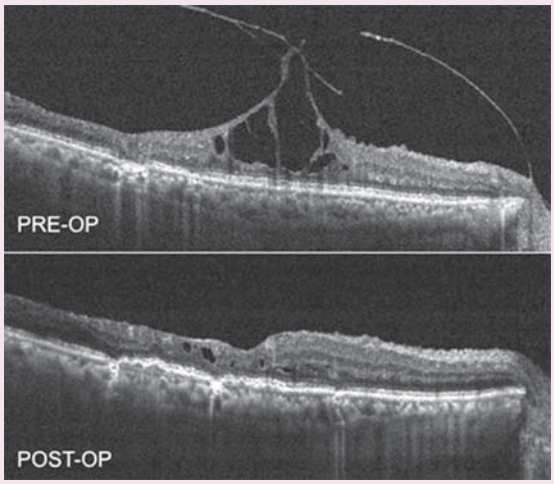

Figure 4: *OCT macula showing resolution of Tractional macular edema after 23 Gauge pars plana vitrectomy.*

(ASG): What is your preferred protocol for intravitreal Anti- VEGF injections for DME, the minimum no. of injections, the duration, follow up and the decision to re-start treatment?

(AK): A 4 weekly/monthly regimen of anti-VEGF agent (ranibizumab mostly) is recommended till the CMT is reduced below 275 microns with no visible cystic changes and a notable improvement in visual acuity- following which I advise antiVEGF as and when required (a pro re nata approach). An initial monthly follow up is advised. When the patient doesn't require intravitreal injections on two consecutive monthly follow-ups the interval can be extended. A minimum of 5 to 6 injections per year may be required initially. In case of repeat recurrence of DME and/or poor response an alternative therapy should be prescribed.

(CS): Usually 3 monthly injections and then PRN. Initial monthly follow up and try to increase interval to 3 months with good patient education regarding need to come earlier.

(LV): Preferred protocol with Intravitreal Anti – VEGF 's for DME is to adminster 2-3 injections of Ranibizumab at 4 weeks interval and follow up with BCVA and OCT. At end of 3 months if response is adequate (See Answer 7) – Repeat FA and if laserable leaks / CNP areas/ Neovascularisation are identified – Add Laser. If no laserable lesions identified on FA – pay attention to Metabolic parameters (including Hb) and can extend the treatment interval. If during ' Treat and Extend ' follow up – a recurrence is noted – then go back to monthly injections. In between – Repeat FFA to identify laserable lesions.

(RN): I give PRN injections with monthly visits in the first 6 months. My minimum number of injections is One. I follow the protocol as in question 7. If the macula is dry on OCT, I recommend follow-up every 2 to 3 months in the first 2 years. If there is mild fluid on OCT but vision is 20/20 on follow-up, I do not inject. But I follow-up them up every month.

Summary: *Experts recommend starting Anti-VEGF therapy (loading dose varying from 1-6 injections monthly) followed by PRN/Treat and extend approach. After the macula is dry, one month follow up is done initially that can be increased slowly. Treatment is restarted if recurrence occurs - with the same agent or alternate agent. Laser may added during the course of treatment if needed.*

(ASG): What is your experience with micro pulse laser? Do you use it for DME and in what situation?

(AK): We have an initial 3-4 months experience using the Subthreshold micropulse diode laser which minimizes collateral damage to the photoreceptors. I still feel this modality is still in primitive stage for DME and with time this might be a valuable alternative for the management of centre involving DME.

(CS): Haven't used.

(LV): Have no personal experience with micropulse laser. Available studies in Literature show micropulse laser to be an upcoming tool to aid in management of Centre – involving DME.

(RN): I have used 577 nm Micropulse laser. It can be an additional option along with anti-VEGF injections in case of centre-involved DME. Micropulse laser can be repeated often, and it helps reduce the number of injections in some cases. In eye with non-centre DME, I use only micropulse laser.

Summary: *Micropulse laser causes minimum collateral damage and can be repeated multiple times. However, its role in centre-involving DME is still not well defined though it appears promising specially in reducing the no. of injections.*

(ASG): What is the role of PRP/targeted peripheral laser for non-responding/recalcitrant DME? Are you doing it?

(AK): Ultra widefield FFA helps in identifying the peripheral non perfused areas. It has been found in studies that the ischemic index (% of non-perfused areas / % of perfused areas) is higher in DR with DME then in DR without DME cases. Hence, I

perform targeted retinal photocoagulation in non-responding/ recalcitrant DMEs. The results are also gratifying.

(CS): There is a role. We do it and have good results in some cases, not all.

(LV): Yes, Yes – As already indicated. Laser does have a role in DME (Laser is NOT DEAD even for Pure DME cases). In pure DME (without PDR) – it seems prudent to repeat FA after adequate Intravitreal Pharmacotherapy and if CNP areas / focal leaks picked up – Do Targeted laser or focal laser. This is done even in cases responsive to Intravitreal pharmacotherapy (to reduce number of Intravitreal Injections) and more so in unresponsive cases.

(RN): I am not doing targeted PRP for DME. I have performed in a few patients, but it had no effect. They still continue to get anti-VEGF or Ozurdex injections.

Summary: *Targeted peripheral laser to peripheral CNP areas (defined on widefield FFA) is being used for cases with recalcitrant DME. However, the results are varying with some patients being benefitted (Figure 5).*

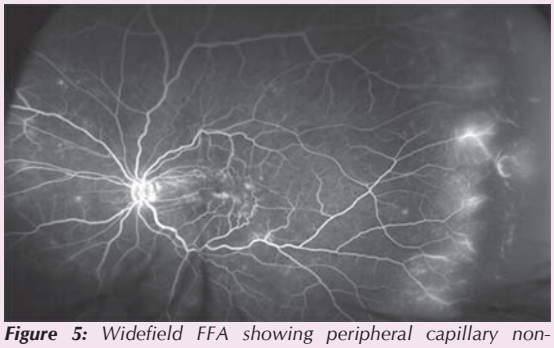

Figure 5: *Widefield FFA showing peripheral capillary non-perfusion areas in a case of DR.*

(ASG): Does Angio-OCT play a part in the management of DME? If yes, how?

(AK): Unlike FFA, the deep capillary plexus can be well visualized with OCT-A. It has been postulated that a distorted/ increased FAZ area at the DCP level is one of the reasons for poor response to antiVEGF. Hence OCT-A can be used as a guide for evaluating such cases. The capillary drop out areas within the arcades can be well appreciated with the help of OCT-A.

(CS): Yes, it does and the role is increasing. Macular perfusion is made out even better than on FA and no contraindication unlike FFA eg. in patients with compromised renal status and allergy to dye.

(LV): OCT–A is a promising upcoming tool. It does help to identify Ischemia, CNP areas as well as neovascularization. Although Flow indices have been described, but their practical utility in day to day management of DME have yet to become a reality.

(RN): OCTA has a limited role in my practice. I get it done in many patients as it is available in my Institution, but my treatment decisions are based on cross-sectional OCT. OCTA is very useful for further understanding of disease pathology, and is a useful research tool. However, in routine practice, OCT is sufficient to take care of our patients.

Summary: *OCT-A is useful for defining macular ischemia and predicting prognosis. There is no contraindication. However, its role in management of DME is still not well defined and its limited availability also restricts its use.*

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