



SDI Review Form 1.6

Journal Name:	Ophthalmology Research: An International Journal
Manuscript Number:	Ms_OR_28445
Title of the Manuscript:	RELATIONSHIP OF HEMOGLOBIN A1C AND OUTCOMES OF TREATMENT OF DIABETIC MACULAR EDEMA
Type of the Article	Original Research Article

General guideline for Peer Review process:

This journal's peer review policy states that **NO** manuscript should be rejected only on the basis of '**lack of Novelty**', provided the manuscript is scientifically robust and technically sound.

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PART 1: Review Comments

	Reviewer's comment	Author's comment (if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)
Compulsory REVISION comments	This study included a large number of patients, but it has some limitation because of retrospective in nature. 1. Please comment about the definition of macular edema. 2. Please comment about retreatment guideline. Such as macular thickness above 300 micrometer? Or worsening of macular edema? 3. If a patient refuse to get treatment, did you exclude the patient's data? 4. Did you measure all the patients' visual acuity with EDTRS chart? It is very time consuming and is not performed in usual clinical condition. I am curious about this because your study is retrospective. 5. Please comment about HbA1C level and mean change. If is confusion that who showed good visual outcome. Patients with good glycemic control? Or unchanged HbA1C level even poor glycemic control? 6. How did you think about treatment regimen? Is there any possibility that there can be different result regarding this (avastin vs lucentis?) 7. Did you have fluorescein angio data? Macular ischemia also affect the visual acuity.	1. The definition of macular edema in our study was determined to be if the foveal architecture was disrupted from intraretinal cystic appearance on SD-OCT or if the CSMT greater than 300 microns. (See lines 68-69) 2. Retreatment for DME was based on persistence of intraretinal cystic spaces on SD-OCT or persistence of CSMT greater than 300 microns. (See lines 69-70) 3. Patients refusing treatment were excluded. Though upon chart review patients did not refuse the recommendation of the treating physician. 4. All measurements of visual acuity were taken on electronic ETDRS charts. See line 64. 5. These are novel results and while confusing at first, we have hypothesized and found support that there is a transient progression of retinopathy that occurs in part due to alteration in blood flow and induced ischemia in patients with improving glycemic control which leads to a possible worsening of macular edema. Though with persistent treatment in the long run these patients will end up



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		doing well in terms of visual acuity. (See lines 180-182.) We did conclude though that regardless of HbA1c level, visual acuity improved and CSMT was reduced overall. 6. Treatment regimen was changed if there had been no improvement of at least 10% of CSMT on SD-OCT at 2 consecutive visits (line 71-72). Based on DRCR protocol T we know that differences in effectiveness in responsiveness of macular edema to intravitreal anti-VEGF injections exists, however a model based on cost-effectiveness and best patient outcomes was applied in this retrospective review. (See lines 187-192 and 328-33) 7. No fluorescein angiography was used in this study as this retrospective review adhered to a OCT-guided treatment regimen. Macular ischemia may play a role in the limitations in visual acuity and we acknowledge this (see lines 199-203)
<u>Minor</u> REVISION comments		
<u>Optional/General</u> comments		