e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2024; 16(5); 416-419

Original Research Article

A Descriptive Analysis of the Different Patterns of Diabetic Macular Edema in OCT and to Observe the Visual Outcome of Intravitreal Anti-VEGF Injection

Deepak Kumar Sinha[1], Gautam Garg[2], Rajiv Kumar Singh[3], Alok Ranjan[4]

[1]Senior Resident, Department of Ophthalmology, SKMCH, Muzaffarpur, Bihar, India

[2]Senior Resident, Department of Ophthalmology, Patna Medical College and Hospital, Patna, Bihar, India

[3]Professor and HOD, Department of Ophthalmology, SKMCH, Muzaffarpur, Bihar, India

[4]Assistant Professor, Department of Ophthalmology, Patna Medical College and Hospital, Patna, Bihar, India

Received: 01-03-2024 / Revised: 10-04-2024 / Accepted: 28-05-2024
Corresponding Author: Dr. Gautam Garg
Conflict of interest: Nil

Abstract

Aim: The aim of the present study was to analyse the different patterns of diabetic macular edema in OCT and to observe the visual outcome of intravitreal anti-VEGF injection.

Methods: A Retrospective study was done in the Department of Ophthalmology, Department of Ophthalmology, SKMCH, Muzaffarpur, Bihar, India. A total of 50 patients were included in the study.

Results: 72% were males and 28% were females. 3% were in the age group of 30-40 years, 24% were in 41- 50 years of age, 38% were 51-60 years of age, 25% 61-70, 10% were >70years of age. 32% had <5 years of diabetes, 64% had 5-10 years, 4% had >10 years of diabetes mellitus. There was a significant reduction in macular edema and improvement in visual acuity between pre and post-injection in Cystoid macular edema and diffuse retinal thickness.

Conclusion: OCT is an essential tool for the quantitative assessment of macular edema. Chronic hyperglycemia is the major cause of diabetic macular edema. Hence the early diagnosis of diabetic macular edema using OCT plays a vital role in preventing permanent vision loss in diabetes patients. CME and DRT have a good visual prognosis for anti-VEGF injections than Posterior hyaloid traction and subretinal detachment. Oct plays a vital role in deciding the treatment modality, explaining prognosis to patients and for predicting the visual outcome.

Keywords: Cystoid macular edema, Diabetic macular edema, OCT, Posterior hyaloid traction

Introduction

Vision loss associated with diabetic retinopathy (DR) is most commonly caused by diabetic macular edema (DME). [1] The Diabetes Control and Complications Trial (DCCT) reported that 27% of patients with type 1 diabetes developed macular edema within 9 years of diabetes onset. [2] Other studies indicate that in type 2 diabetes patients, the prevalence increases from 3% within 5 years of diagnosis to 28% after 20 years. [3] Although several treatment options are available, no consensus on DME treatment based on patient status has been achieved. Vascular endothelial growth factor (VEGF) is an important mediator of abnormal vascular permeability in eyes with DME. [4]

Anti-VEGF injections are generally proposed as first-line therapy for center-involved DME and are effective in improving visual acuity (VA), with 10%–40% of patients achieving significant improvement in VA after 1 year of treatment. [5,6] However, a considerable proportion have unsatisfactory response to anti-VEGF agents; 40% of eyes with DME do not or have suboptimal response to anti-VEGF treatment. [7,8] Nonetheless, there is little information to date about the prognostic factors of poor responders. Optical coherence tomography (OCT) images are readily available to physicians and provide detailed information. Structural changes presumably reflect part of the complex pathophysiologic processes occurring in DME. Furthermore, anatomical measures on spectral-domain (SD) OCT can predict treatment success or failure of various therapies. [9] Distinct structural changes identifiable on SD-OCT could reflect part of the intraocular pathophysiologic

process change after anti-VEGF treatments and help predict the treatment response.

Among patients with DME refractory to anti-VEGF therapy after a loading dose of three consecutive monthly injections, those who were switched to other treatment modalities (e.g., corticosteroids) had better visual and anatomical outcomes at 12 months than did those who continued with anti-VEGF therapy. [10] Post hoc analysis from the DRCR.net Protocol I study also indicates that early central macular thickness (CMT) response to anti-VEGF is a significant prognostic indicator of medium to long-term anatomical outcomes in DME. [11] Accordingly, the early identification of patients who would not benefit from first-line treatment with anti-VEGF therapy is critical. Real-world studies have become increasingly important in providing evidence of treatment effectiveness in clinical practice. They can therefore provide information on the long-term safety, particularly of rare events, and efficacy of drugs in large heterogeneous populations, as well as information on utilization patterns and health and economic outcomes. [12]

The aim of the present study was to analyse the different patterns of diabetic macular edema in OCT and to observe the visual outcome of intravitreal anti-VEGF injection.

Materials and Methods

A Retrospective study was done in the Department of Ophthalmology, Department of Ophthalmology, SKMCH, Muzaffarpur, Bihar, India for 7 months. A total of 50 patients were included in the study.

Inclusion criteria: All patients with diabetic macular edema. Exclusion criteria: Those who have macular edema other than diabetes, Patients with hazy media which makes it difficult to take OCT, OCT scans with poor signal strength less than 5.

All patients were subjected to history taking including age, sex, duration of diabetes, history of other systemic illnesses, previous treatment history. Slit-lamp examination dilated fundus examination using slit-lamp biomicroscopy with +90 D lens, Indirect ophthalmoscope, OCT examination including macular cube, type of macular edema, Central macular thickness value, quadrant wise distribution of edema, cube average thickness and associated findings in OCT were measured. The number of anti–VEGF injection, pre-injection and post-injection macular thickness, pre-injection and post-injection visual acuity, the effect of injection for each type of macular edema were measured.

Results

Table 1: Baseline characteristics

Gender	N	%
Male	36	72
Female	14	28
Age groups in years		
31-40	3	3
41-50	24	24
51-60	38	38
61-70	25	25
>70	10	10
Duration of diabetes		
<5 years	32	32
5-10 years	64	64
>10 years	4	4

72% were males and 28% were females. 3% were in the age group of 30-40 years, 24% were in 41- 50 years of age, 38% were 51-60 years of age, 25% 61-70, 10% were >70years of age. 32% had <5 years of diabetes, 64% had 5-10 years, 4% had >10 years of diabetes mellitus.

Table 2: Distribution of macular edema among various quadrants

Quadrant distribution	Diffuse retinal thickness	Cystoid macular edema	Posterior hyaloid traction	Serous retinal detachment	CME with SRD
ITQ	371.50	437.40	454.00	424.25	432.44
INQ	368.19	432.45	369.00	451.25	441.78
ISQ	376.25	465.25	372.67	434.75	458.89
IIQ	337.88	448.50	392.67	502.75	441.67
OTQ	342.06	363.60	421.00	443.00	335.44
ONQ	316.38	353.35	414.33	386.25	379.44
OSQ	331.44	382.00	373.33	394.00	374.11
OIQ	318.31	355.80	354.67	433.50	369.22

There was a significant reduction in macular edema and improvement in visual acuity between pre and post-injection in Cystoid macular edema and diffuse retinal thickness.

Discussion

Diabetic macular edema is the most common cause of vision loss in diabetes. [13] The prevalence of diabetic macular edema in diabetes is 5% in 5 years and 15% in 15 years. [14] Chronic hyperglycemia is the major risk factor of diabetic macular edema. The incidence of DME over a 10 year period is 20% in patients with younger-onset diabetes versus approximately 40% in older onset diabetes. Chronic hyperglycemia results in deposition of advanced glycated end products which results in disruption of the blood-retinal barrier and an altered vitreoretinal interface. This alteration in the Blood retinal barrier results in interstitial fluid accumulation within the retina and cyst formation. Increased vascular permeability occurs as a result of breakdown of the BRB, due to loss of pericytes, altered glial cells, endothelial cell death, leukostasis in the retinal vasculature, leakage of tight junctions, increased expression of vascular endothelial growth factor (VEGF) and altered vitreoretinal interface with a thickened taut, posterior hyaloid with persistent vitreomacular traction (VMT). [15]

72% were males and 28% were females. 3% were in the age group of 30-40 years, 24% were in 41- 50 years of age, 38% were 51-60 years of age, 25% 61-70, 10% were >70years of age. 32% had <5 years of diabetes, 64% had 5-10 years, 4% had >10 years of diabetes mellitus. There was a significant reduction in macular edema and improvement in visual acuity between pre and post-injection in Cystoid macular edema and diffuse retinal thickness. Basic morphological characteristics of macular edema described by Otani et al [16] are spongy like retinal thickness, cystoid macular edema, and serous retinal detachment. In diffuse retinal edema, there will be an increased retinal thickness with reduced retinal reflectivity. Cystoid macular edema occurs because of two pathological components abnormal collection of extracellular fluid and cystoid space formation. [17]

In a study conducted by Vukicevic et al [18] CME was classified into mild, moderate and severe according to the size of the cysts. The cystoid spaces are mainly located in outer retinal layers in patients with intermediate and severe CME. [19] Posterior hyaloid traction appears as a highly reflective band on the retinal surface. In Subretinal detachment, dark accumulation of SRF under the high reflective and dome-like elevation of a detached retina. The highly reflective band represents the outer surface of the detached retina it differentiates SRF from the intraretinal fluid. In TRD, there will be low signal underlying the highly reflective border of a detached retina. The effect on the improvement of visual acuity is more for central macular thickness than age, fluorescein leakage, the severity of perifoveal capillary occlusion. [20,21]

Conclusion

OCT is an essential tool for the quantitative assessment of macular edema. Chronic hyperglycemia is the major cause of diabetic macular edema. Hence the early diagnosis of diabetic macular edema using OCT plays a vital role in preventing permanent vision loss in diabetes patients. CME and DRT have a good visual prognosis for anti-VEGF injections than Posterior hyaloid traction and subretinal detachment. Oct plays a vital role in deciding the treatment modality, explaining prognosis to patients and for predicting the visual outcome.

References

1. Kempen JH, O'Colmain BJ, Leske MC, Haffner SM, Klein R, Moss SE, Taylor HR, Hamman RF. The prevalence of diabetic retinopathy among adults in the United States. Archives of ophthalmology (Chicago, Ill.: 19 60). 2004 Apr 1;122(4):552-63.
2. White NH, Sun W, Cleary PA, Tamborlane WV, Danis RP, Hainsworth DP, Davis MD, Dcct-Edic Research Group. Effect of prior intensive therapy in type 1 diabetes on 10-year progression of retinopathy in the DCCT/EDIC: comparison of adults and adolescents. Diabetes. 2010 May 1;59(5):1244-53.
3. Klein R, Klein BE, Moss SE, Cruickshanks KJ. The Wisconsin epidemiologic study of diabetic retinopathy XV: the long-term incid ence of macular edema. Ophthalmology. 1995 Jan 1;102(1):7-16.
4. Aiello LP, Avery RL, Arrigg PG, Keyt BA, Jampel HD, Shah ST, Pasquale LR, Thieme H, Iwamoto MA, Park JE, Nguyen HV. Vascular endothelial growth factor in ocular fluid of patients with diabetic retinopathy and other retinal disorders. New England Journal of Medicine. 1994 Dec 1;331(22):1480-7.
5. Massin P, Bandello F, Garweg JG, Hansen LL, Harding SP, Larsen M, Mitchell P, Sharp D, Wolf-Schnurrbusch UE, Gekkieva M, Weichselberger A. Safety and Efficacy of Ranibizumab in Diabetic Macular Edema (RESOLVE Study) A 12-month, randomized, controlled, double-masked, multicenter phase II study. Diabetes care. 2010 Nov 1;33(11): 23 99-405.
6. Mitchell P, Bandello F, Schmidt-Erfurth U, Lang GE, Massin P, Schlingemann RO, Sutter F, Simader C, Burian G, Gerstner O, Weichselberger A. The RESTORE study:

ranibizumab monotherapy or combined with laser versus laser monotherapy for diabetic macular edema. Ophthalmology. 2011 Apr 1;1 18(4):615-25.
7. Bressler SB, Ayala AR, Bressler NM, Melia M, Qin H, Ferris FL, Flaxel CJ, Friedman SM, Glassman AR, Jampol LM, Rauser ME. Persistent macular thickening after ranibizumab treatment for diabetic macular edema with vision impairment. JAMA ophthalmology. 2016 Mar 1;134(3):278-85.
8. Gonzalez VH, Campbell J, Holekamp NM, Kiss S, Loewenstein A, Augustin AJ, Ma J, Ho AC, Patel V, Whitcup SM, Dugel PU. Early and long-term responses to anti–vascular endothelial growth factor therapy in diabetic macular edema: analysis of protocol I data. American journal of ophthalmology. 2016 Dec 1;172:72-9.
9. Zur D, Iglicki M, Busch C, Invernizzi A, Mariussi M, Loewenstein A, Cebeci Z, Chhablani JK, Chaikitmongkol V, Couturier A, Fraser-Bell S. OCT biomarkers as functional outcome predictors in diabetic macular edema treated with dexamethasone implant. Ophthalmology. 2018 Feb 1;125(2): 2 67-75.
10. Busch C, Zur D, Fraser-Bell S, Laíns I, Santos AR, Lupidi M, Cagini C, Gabrielle PH, Couturier A, Mané-Tauty V, Giancipoli E. Shall we stay, or shall we switch? Continued anti-VEGF therapy versus early switch to dexamethasone implant in refractory diabetic macular edema. Acta diabetologica. 2018 Aug; 55:789-96.
11. Dugel PU, Campbell JH, Kiss S, Loewenstein A, Shih V, Xu X, Holekamp NM, Augustin AJ, Ho AC, Gonzalez VH, Whitcup SM. Association between early anatomic response to anti–vascular endothelial growth factor therapy and long-term outcome in diabetic macular edema: an independent analysis of protocol i study data. Retina. 2019 Jan 1;39 (1):88-97.
12. Blonde L, Khunti K, Harris SB, Meizinger C, Skolnik NS. Interpretation and impact of real-world clinical data for the practicing clinician. Advances in therapy. 2018 Nov;35:1763-74.
13. Maurya RP. Diabetic retinopathy: My brief synopsis. Ind J Clin Exp Ophthalmol. 2015;1 (4):189–90.
14. Aiello LP, Gardner TW, King GL, Blankenship G, Cavallerano JD, Ferris FL, et al. Diabetic Retinopathy. Diabetes Care. 1998; 21(1):143–56.
15. Maurya RP. Dabetic macular edema: An overview. Indian J Clin Exp Ophthalmol. 2019 ;5(1):1-2.
16. Otani T, Kishi S, Maruyama Y. Patterns of diabetic macular edema with optical coherence tomography. Am J Ophthalmol. 1999; 127(6): 688–93. doi:10.1016/s0002-9394(99)00033-1.
17. Tso MO. Pathology of cystoid macular edema. Ophthalmol. 19822;89:902–15.
18. Vukicevic M, Gin T, Al-Qureshi S. Prevalence of optical coherence tomography-diagnosed postoperative cystoid macular oedema in patients following uncomplicated phaco-emulsification cataract surgery. Clinical & experimental ophthalmology. 2012 Apr;40(3): 282-7.
19. Koleva-Georgieva DN, Sivkova NP. Types of diabetic macular edema assessed by optical coherence tomography. Folia medica. 2008 Jul 1;50(3):30-8.
20. Diabetic Retinopathy Clinical Research Network. Relationship between optical coherence tomography–measured central retinal thickness and visual acuity in diabetic macular edema. Ophthalmology. 2007 Mar 1;1 14(3):525-36.
21. Baskin DE. Optical coherence tomography in diabetic macular edema. Current opinion in ophthalmology. 2010 May 1;21(3):172-7.