RESEARCH ARTICLE

OCT DETERMINED MACULAR THICKNESS IN DIABETIC RETINOPATHY AND RELATION TO COLOUR VISION DEFICIENCY PATTERNS

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DOI: 10.5455/ijmsph.2013.111220131 **Received Date: 02.12.2013** **Accepted Date: 11.02.2014**

ABSTRACT

Background: Diabetic retinopathy is the most common cause of blindness in industrialised nations and the incidence of diabetes is expected to rise over the next 10 years. Early treatment of proliferative diabetic retinopathy and diabetic maculopathy improves visual outcome and with effective screening, blindness could be reduced. Patients suffering from diabetes mellitus show alterations in their colour perception. Colour vision testing provides a sensitive, non-invasive method to assess macular damage in diabetic retinopathy and any deterioration in colour vision often precedes changes in other clinical measures such as visual acuity and morphological changes. Optical coherence tomography (OCT) provides cross-sectional images of the retina, information concerning internal retinal structure as well as reproducible measurements of retinal thickness.

Aims & Objective: This study was intended to examine the relationship between macular thickness on OCT (Optical coherence tomography) and colour vision deficiency patterns in diabetics.

Material and Methods: A total of 100 patients with established diabetic retinopathy visiting hospital were examined and a detailed history of diabetes, duration and inability to differentiate colours was taken. Retinal examination for evidence of diabetic retinopathy and colour vision patterns on Ishihara colour vision chart were noted. OCT was performed on these patients.

Results: 21% (21 out of 100) of patients under study showed a gross deficiency of red green pattern of colour blindness. The colour vision deficiency increased with increasing macular thickness. Macula thickness on OCT in subjects varied from 188 to 462 microns. The average macular thickness of patients with total colour vision deficiency was 290 microns that with red green was 274 micron and those without any colour vision deficiency was 228. Prevalence of colour vision deficiency increased with duration of diabetes and severity of retinopathy.

Conclusion: Higher prevalence of red green colour blindness was observed in patients of diabetes with macular oedema. Impaired colour vision was more in patients with macular thickness above 228 micron.

Key-Words: Diabetes; Macular Thickness; Optical Coherence Tomography (OCT); Colour Vision

Introduction

Diabetes, a complex metabolic disorder is frequently encountered in ophthalmic practice. Well documented reports on colour vision defects in diabetes mellitus are very few. An analysis of such defects has recently been done by Kinnear et al[1], who found colour discrimination among diabetics was poorer than non-diabetic population. Colour vision testing provides a sensitive, non-invasive method to assess macula damage. Deterioration in colour vision often precedes changes in other clinical measures such as visual acuity and morphological changes.[2] Several studies have shown a correlation between tritan colour vision deficiency and stage of diabetic retinopathy.[3]

Studies have demonstrated that duration diabetes correlates strongly with increases in lens optical density, even among patients with relatively short diabetes duration.[4,5] It has been shown that patients with diabetes suffer an increased rate of lens yellowing similar to that in non-diabetics older than 60 years.[6] Consequently, any tritan deficit seen in diabetics may be wholly or partly due to the preretinal absorption of short wavelength light resulting from lens yellowing.[7]

Patients suffering from diabetes mellitus can show alterations in their colour perception. Metabolic alterations caused by chronically raised blood glucose levels cause capillary degeneration, hypoxia, and cell death in the retina of diabetics. The role of hypoxia in the colour vision changes has recently been stressed by the findings of Dean et al[8], who show that the colour deficits in diabetics with retinopathy can be partially reversed by inhalation of oxygen. The aetiology of diabetic retinopathy remains unclear, and the initial retinal sites affected by the hypoxia are as yet unknown. Most studies have been performed on patients with diabetic retinopathy and report a deficiency at the short end of the spectrum. Alterations have been found in photoreceptor function as well at the post receptoral stage of retinal processing, with some patients showing deficits at both retinal loci.[9-12] It is known that microvascular complication becomes more frequent with increasing diabetes duration[13] and high glycosylated haemoglobin values.[14]. However, previous studies[15], did

not show any association between either titan defect and duration of diabetes.

The aim of this study was to ascertain the prevailing colour vision defects in patients with diabetes mellitus, the correlation between duration of diabetes, macular thickness on OCT and colour vision.

Materials and Methods

This observational study was done on 100 patients with diabetes mellitus attending outpatient department of ESIC Medical College and PGIMSR, Bangalore over one year and five months between April 2012 and September 2013. Diabetic retinopathy was classified based on the international clinical diabetic retinopathy disease severity scale as in the American academy of ophthalmology October 2002 guidelines.

Inclusion Criteria: (i) All patients with diabetes mellitus with visual acuity of 6/24 or better; (ii) Presence of at least mild diabetic retinopathy

Exclusion Criteria: (i) Diabetic patients with visual acuity 6/36 or lesser as the Ishihara interpretation may not be accurate; (ii) Known cases of colour vision deficiency, Central serous retinopathy, optic neuritis; (iii) Patients on Anti Tubercular therapy; (iv) Chronic uveitis, glaucoma; (v) Family history of colour vision disorders; (vi) History of any vitreoretinal surgeries in the past; (vii) Presence of cataract.

After obtaining informed consent, a detailed history on demographic data, ocular, and medical history was taken. All the participants underwent a complete ophthalmic examination, including visual acuity (VA) measurement, refraction, and slit lamp examination, dilated fundus examination by indirect ophthalmoscopy, +90D and direct ophthalmoscopy. IOP measurement by Goldmann applanation, gonioscopy using three mirror lens and macular thickness on 3D OCT. blood investigations including fasting and post prandial blood glucose levels were tested. Colour vision testing was done using standard Ishihara's colour vision plates. All findings were tabulated. Optical coherence tomography was performed using commercially available equipment through a dilated pupil by an experienced examiner who was aware of the clinical findings.

The OCT examination comprised six radial 6-mm-long scans of each eye, centered on the patient's fixation point, at intervals of 30 degrees. Retinal thickness was computed automatically, using OCT retinal mapping software. This mapping averaged the six scans to give the central macular thickness in an area 500 micron in diameter. The average macular thickness was noted for the study.

Results

A total of 100 patients with diabetes mellitus were included in the study. The age group varied from 26 years to 75 years among males and 34 years to 75 among females. The average age of presentation of diabetes among males was 52.8 years and 56.3 years among females. Diabetic retinopathy had a higher prevalence among men was about 61% (61/100) and 39 % in females (39/100). Out of the 100 diabetic patients examined, 46 were found to have colour vision deficiency. i.e., 46% was the prevalence rate.

The prevalence of colour vision defects was found to be more in men 31 of the 61 patients (51%) than women 38% (15 of 39) as shown in table 1. Colour vision deficiency observed was maximum for red green patterns. About 21 of subjects had deficient red green pattern, 13 had protanopia, 4 had deuteran defect and 8 had total colour blindness. The macular thickness determined on OCT of patients varied from 188 micron to 388 micron among males with an average of 266 micron and ranged from 188 to 462 micron in females with an average of 245 micron. Colour vision defects increased with duration of diabetes and had significant association with severity of diabetic retinopathy too. Increasing macular thickness on OCT was associated with higher prevalence of colour vision defects.
The average macular thickness of patients who did not have any colour vision defect was 227 micron in males and 230 in females. Patients with an average macular thickness of 270 (males) and 274 (females) showed protan and deuteran colour defect. The average central macular thickness in males with red green defect was 274 micron in males and 260 in females but above 290 micron showed total colour vision defects. Patients with proliferative diabetic retinopathy showed a higher incidence of colour vision defects 17 out of 23 (74%) showing colour defect followed by 13 of 23 i.e. 57 in severe NPDR. The duration of diabetes too had a positive correlation to colour vision defects along with the severity of diabetes. The average duration of diabetes in patients with colour vision defects was 7.3 yrs and those without was 6.2 years.

Table-1: Sex wise distribution of colour defects

	MALE	FEMALE
Total under study	61	39
Colour defects	31	15
%	51	38

Table-2: Defects with severity of retinopathy

	Mild NPDR	Moderate NPDR	Severe NPDR	PDR
Colour Defects	6	10	13	17
Total	30	24	23	23
%	20	41.6	56.5	74

Discussion

The colour vision defects in diabetics have recently been well documented by Kinnear et al[1] who reported a very high incidence of yellow-blue losses among diabetics even without retinopathy. Macular oedema occurs in systemic disorders such as diabetes mellitus or uveitis. Histopathologic reports have shown that the extent and location of the intraretinal oedema varies according to the etiology of oedema[16], and that other features such as serous retinal detachment (SRD) may be associated with ME. Biomicroscopy is not sensitive enough to evaluate structural abnormalities or retinal thickness. Hence, the need for additional techniques such as optical coherence tomography (OCT), which is based on low coherence interferometry and provides cross-sectional images of the retina. OCT also provides information concerning internal retinal structure[17] as well as reproducible measurements of retinal thickness[18,19].

The mechanism and causation of defective colour perception in diabetics is a matter of conjuncture. In fact the complexities of diabetic retinopathy as well as the associated defects of colour perception are little understood and theoretically a discolouration of lens, Kinnear[1]; disturbances of cone pigments, Krill and Fishman[20]; faulty perception or transmission owing to the pathological changes in neurones Bloodworth[21]; may individually or collectively contribute to loss of brightness or colour perception. However, they found the mean red green losses within normal limits in diabetics even with retinopathic changes. An appreciable number of Tritan defect was noted in diabetics with retinopathic changes. Protan and Deutan defects were not revealed by any diabetic patient.

Diabetic retinopathy is the most common cause of blindness in industrialised nations among the 20–60 year old age group and the incidence of diabetes is expected to rise over the next 10 years.[22,23] Early treatment of proliferative diabetic retinopathy and diabetic maculopathy improves visual outcome and with effective screening blindness in patients under the age of 70 could be reduced by 10%.[24] Higher resolution scans such as optical coherence tomography (OCT) and the retinal thickness analyser (RTA) have demonstrated considerable thickening of the diabetic retina before the onset of diabetic retinopathy.[25,26]

OCT employs the principles of low-coherence interferometry and is analogous to ultrasound B-mode imaging but uses light instead of sound to acquire high resolution images of ocular structures. In brief, a low coherence near-infrared (840 nm) light beam is directed onto a partially reflective mirror (beam splitter) that creates two light beams, a reference and a measurement beam. The measurement beam is directed onto the subject's eye and is reflected from intraocular microstructures and tissues according to their distance, thickness, and different reflectivity. The reference beam is reflected from the reference mirror at a known, variable position. Both beams travel back to the partially reflective mirror, recombine, and are transmitted to a photosensitive detector. The pattern of interference is used to provide information regarding distance and thickness of retinal structures. Bi dimensional images are created by successive longitudinal scanning in transverse direction.[27]

Colour vision testing provides a sensitive, non-invasive method to assess macula damage. Deterioration in colour vision often precedes changes in other clinical measures such as visual acuity and morphological changes.[28] Several studies have shown a correlation between tritan colour vision deficiency and stage of diabetic retinopathy.[29] Our observations, thus, were in partial agreement with that of Kinnear et al.[1] Tritan defects are more common in diabetics with retinal involvement and colour vision testing should be utilised as an adjunct in evaluating patients with diabetes.

It is well established that colour vision is affected by diabetic retinopathy. Colour vision is a function of the cones, and patients with exudative maculopathy would be expected to demonstrate the most marked defects of colour vision. Our results confirm this; 46 of 100 (46% of subjects) with maculopathy had an abnormal colour vision. In some cases of proliferative retinopathy areas of capillary closure may be situated peripherally in the retina where they might be expected not to influence colour vision, while in other patients intraretinal microvascular abnormalities or capillary closure may affect the macular circulation and lead to a deterioration of colour vision.[30] In our proliferative retinopathy group 17 of 23 (74% of subjects) had defective colour vision.

In addition to these more or less expected observations we also found significant defects of colour vision in many diabetics with moderate retinopathy or severe NPDR. In these two categories 24 and 23 of patients (41.6% and 56.5 % of subjects) respectively showed higher prevalence of colour vision defects. The mechanism of this deterioration

of colour discrimination remains obscure, but it may result from early damage to the cones or their neuronal connections, which cannot be detected ophthalmoscopically, as previously suggested by Kinnear.

Conclusion

The Ishihara colour vision test demonstrates deteriorating colour discrimination with increasing severity of diabetic retinopathy. Increasing macular thickness had higher prevalence of colour vision defects seen on OCT. The test though, is not on its own sufficiently specific to be of value as a screening test for the identification of serious diabetic retinopathy requiring laser treatment still provides an effective tool to assess early macular damage.

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Cite this article as: Sadiqulla M, Khan IA, Rahmath AG. OCT determined macular thickness in diabetic retinopathy and relation to colour vision deficiency patterns. Int J Med Sci Public Health 2014;3:265-268.
Source of Support: Nil
Conflict of interest: None declared