EVALUATION OF OPTICAL COHERENCE TOMOGRAPHY PATTERNS IN DIABETIC MACULAR OEDEMA

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ABSTRACT

BACKGROUND

The aim of this study is to identify, categorise and analyse the Optical Coherence Tomography patterns of Diabetic Macular oedema.

MATERIALS AND METHODS

In this observational study, 43 eyes of 25 patients with Diabetic Macular oedema (DME) were evaluated. DME was defined as the retinal thickening due to fluid leakage and pooling in the macular area in patients with Diabetes Mellitus. Macular oedema due to other ocular illness was excluded. All patients underwent best corrected visual acuity assessment by Snellen's visual acuity chart, dilated slit-lamp Biomicroscopic examination, Fundus Fluorescein Angiography (FFA) and Optical Coherence Tomography (OCT) by the same examiner. OCT patterns were analysed. Central foveal thickness was also measured by OCT and macular oedema classified into mild (201 μm-300 μm), moderate (301 μm-400 μm) and severe (≥400 μm).

RESULTS

Of the total 25 patients in the age group 35-75 years (Mean age 54.08), males predominated in this study (M: F ratio of 2.6:1). OCT examination revealed that 30% eyes had Cystoid macular oedema and 26% had Sponge-like retinal thickness. Mixed cystoid and spongeform pattern was observed in 28%, Epiretinal membrane (ERM) in 9%, Plaque of hard exudates in 7%, Serous macular detachment in 9%, and Vitreomacular traction in 5%. 32% eyes had mild macular oedema, 21% had moderate and 35% had severe forms.

CONCLUSION

Various patterns can be easily identified by OCT and treatment may be modified accordingly. Cystoid macular oedema was the predominant form of DME according to this study. Both eyes of a same patient can present with different DME patterns.

KEYWORDS

Optical Coherence Tomography, Diabetic Macular oedema, Cystoid Macular Oedema, Epiretinal Membrane, Central Foveal Thickness, Vitreomacular Traction.


BACKGROUND

Diabetic Macular Oedema (DME), a microvascular complication which is caused by the breakdown of the blood-retinal barrier, promotes neurigial dysfunction and concomitant visual disturbance. It is the commonest cause of visual loss in patients with non-proliferative diabetic retinopathy and a common cause of visual loss in proliferative diabetic retinopathy.

Diabetic macular oedema is diagnosed stereoscopically as retinal thickening in the macula using slit-lamp biomicroscopy. The ETDRS defined DME as retinal thickening or presence of hard exudates within 1 DD of the centre of the macula. To characterise the severity of macular oedema, and for treatment guidelines the term Clinically Significant Macular Oedema (CSME) is used. Macular oedema is clinically significant, if one of the following conditions is present: 1. Retinal thickening at or within 500μ of the centre of the macula. 2. Hard exudates at or within 500μ of the centre of the macula if associated with thickening of retina. 3. A zone or zones of retinal thickening one disc area or larger, any part of which is within one disc diameter of the centre of the macula.

Diabetic macular oedema tends to be a chronic disease. Although spontaneous recovery is not uncommon, 24% of eyes with CSME and 33% of eyes with centre involving CSME will have a moderate visual loss (15 or more letters on the ETDRS chart) within 3 years if untreated.

The incidence of DME over a 10-year period was 20.1% among patients diagnosed before age 30 years (younger onset) and 39.3% among patients diagnosed after 30 years. As the severity of overall retinopathy increases, the proportion of eyes with macular oedema also increases. 3% in eyes with mild non-proliferative diabetic retinopathy (NPDR), 38% with moderate-severe NPDR and 71% with proliferative diabetic retinopathy (PDR) develop DME.

Optical coherence tomography (OCT) is a fast and noninvasive tool for examining the retina in cross sectional images that correlate reasonably with the retinal histology. It is not only helpful in detecting DME early, but has the added advantage of being able to reveal not only the presence of cystoid macular oedema, but subfoveal serous retinal...
detachment, vitreomacular traction or an epiretinal membrane which cannot be detected in FFA.

Moreover, the macular thickness map gives us a very accurate idea of central retinal thickness and can quantify the degree of improvement or worsening following therapy.

**Aim of Study**
The aim of the study was to identify, categorise, and analyse the OCT patterns of Diabetic Macular Oedema.

**MATERIALS AND METHODS**
This was an observational study done between October 2010 and March 2011 in patients who attended the retina clinic of Govt. Medical College, Thrissur. 43 eyes of 25 patients with Diabetic Macular Oedema were evaluated. The study group included both insulin dependent and non-insulin dependent proliferative diabetic retinopathy and non-proliferative diabetic retinopathy between the ages of 35-75 years.

None of the patients in our study had undergone previous focal laser or pan-retinal photocoagulation, or ocular surgery in the past six months. Other exclusion criteria were dense cataract, macular oedema owing to other ocular illness and advanced diabetic retinopathy.

Diabetic macular oedema is diagnosed stereoscopically as retinal thickening in the macula. The patients were diagnosed as having Diabetic Macular Oedema by slit-lamp biomicroscopic examination with 90D lens. A detailed history regarding onset of visual loss and duration of diabetes, treatment taken, etc was taken and recorded. All these patients underwent best corrected visual acuity assessment by Snellen's visual acuity chart, and dilated slit-lamp biomicroscopic examination. Fundus photographs were taken and macular oedema was confirmed by Fundus Fluorescein Angiography. Spectral domain OCT (OPKO) was taken on the same day, by the same examiner. OCT was done in all eyes, a line scan program was chosen and the image processed and analysed for pattern characterisation. Central macular thickness was measured with the retinal thickness map. Macular oedema was categorised into mild (with a thickness of 201-300µ), moderate (301-400µ) and severe (≥400µ).

**RESULTS**
Of the 25 patients we analysed, there were 3 (12%) patients in the age group 30-39 years, 2 (8%) in 40-49 years age group, 11 (44%) in 50-59 years age group, 9 (32%) in 60-69 age groups and 1 (4%) in 70-79 age group. Males predominated with M: F ratio of 2.6:1. 67.3% had NPDR and 32.7% PDR. Mean diabetic age was 14.08 years.

Biomicroscopic examination of all the patients showed Diabetic macular oedema. 11% patients showed DME associated with cystoid macular oedema (CME), and 2% had DME with vitreomacular traction (VMT). No patients had Epiretinal membrane (ERM) or Serous Macular Detachment with Subretinal Fluid (SRF) clinically.

Ocular Coherence Tomography analysis showed seven patterns of macular oedema in our patients.

**Spongiform Pattern**
Eyes with spongy oedema showed diffuse thickening of macula. It mostly involved the outer retinal layers, while the internal layers maintained their normal reflectivity. Cross sectional scans show swelling of the retina giving it a spongy appearance with increased retinal thickness.
Cystoid Pattern
Eyes with CME showed large cystic spaces in the foveolar and parafoveal region. It involves various depth of retina and has intervening septa in between.

Mixed Pattern
Some eyes showed both spongiform thickening of outer retinal layers and cystoid spaces in the inner retina.

Serous Macular Detachment was seen as a hyporeflective area between neurosensory retina and RPE.

Vitreomacular Traction or VMT was seen as hyper-reflective band in the vitreous, which was adherent to the fovea, either centrally or paracentrally causing traction and pulling up the macula.

Epiretinal membrane or ERM was identified as a hyper-reflective thickening at the level of ILM, causing distortion and flattening of the foveal surface.

Hard Exudates Plaque was seen as hyper-reflective intraretinal plaque which cast a shadow due to the blockade of light transmission.

<table>
<thead>
<tr>
<th>Pattern of DME in OCT</th>
<th>% of Eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spongy oedema</td>
<td>26</td>
</tr>
<tr>
<td>Cystoid oedema</td>
<td>30</td>
</tr>
<tr>
<td>Mixed spongy and cystoid oedema</td>
<td>28</td>
</tr>
<tr>
<td>ERM</td>
<td>9</td>
</tr>
<tr>
<td>Submacular detachment</td>
<td>9</td>
</tr>
<tr>
<td>VMT</td>
<td>5</td>
</tr>
<tr>
<td>Hard exudate plaques</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 1. Distribution of Eyes with Various OCT Patterns in Patients with DME

15% of patients presented with differing OCT patterns in both eyes.

<table>
<thead>
<tr>
<th>Type of DME</th>
<th>Biomicroscopy</th>
<th>OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td>CME</td>
<td>11%</td>
<td>30%</td>
</tr>
<tr>
<td>SRF</td>
<td>Nil</td>
<td>9%</td>
</tr>
<tr>
<td>VMT</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>ERM</td>
<td>Nil</td>
<td>9%</td>
</tr>
</tbody>
</table>

Table 2. Comparison of DME Characteristics Identified by Biomicroscopy and OCT

Measurement of macular thickness revealed 33% of eyes with mild macular oedema, 21% moderate oedema and 35% with severe diabetic macular oedema.
DISCUSSION
Optical Coherence Tomography is a fast and noninvasive tool for examining the retina in cross sectional images that correlates reasonably with the retinal histology. Till recently slit-lamp biomicroscopy and FFA were the tools for the diagnosis and management of DME. It is true that these are highly sensitive for the qualitative detection of DME. OCT enables us to detect and understand the accurate subclinical retinal changes associated with DME that may not be detectable even in FFA. Yang et al have suggested that OCT up imaging. Visual impairment in -ctions and the 86 invas ophthalmol, 7, Moss SE. Quantitative, Lee FL, et al J same patient can present with different DME patterns as per 
Thus biomicroscopy and 63% were not detected even by FFA. That 40% of CME detected on OCT were not detected by to 11% detected by biomicroscopy. Ozdek et al also found represents a chronologically later stage of DME.
Diabetic macular our retina clinic is with longer diabetic age and thus their the fact that the ret spongy and cystoids oedema study revealed that 26% had macular thickening with spongy macular thickening. In his series, OCT identified spongy retinal thickness seen in 58% of eyes.6 Otani et al found spongy retinal thickness in 88%, CME in 47%, SRF in 15% of eyes with CSME. Kim et al found spongy retinal swelling in 97%, CME in 55%, SRF in 7%, VMT in 13% of eyes with DME.7 Ozdek et al8 had reported spongy swelling in 66%, CME in 16%, SRF in 10% of eyes with DME. In our series, cystoid macular oedema was the common form of presentation. Our study revealed that 26% had macular thickening with spongy oedema, 30% with cystoid changes, 29% with mixture of spongy and cystoids oedema, 9% ERM, 9% with serous retinal detachment, 5% with vitreomacular traction and 12% with plaques of hard exudates. The higher incidence of cystoid form of macular oedema in our series could be due the fact that the section of diabetic population presenting to our retina clinic is with longer diabetic age and thus their diabetic macular oedema a longstanding one. CME pattern represents a chronologically later stage of DME.
In our study, 30% of the eyes had CME on OCT, compared to 11% detected by biomicroscopy. Ozdek et al also found that 40% of CME detected on OCT were not detected by biomicroscopy and 63% were not detected even by FFA. Thus, OCT tends to be a better diagnostic tool in detecting CME than biomicroscopy or FFA.
In our study, 9% of eyes had SRF with subfoveal retinal detachment, which could not be detected by biomicroscopy. Most series have found SRF in 8-12% of eyes with DME.
According to our study, 5% had VMT as per OCT and 2% as per biomicroscopy. VMT has been reported by various authors between 10-60% of eyes with DME.
Another important finding of our study was both eyes of a same patient can present with different DME patterns.

CONCLUSION
Diabetic macular oedema is a major cause of visual disability in diabetic patients. DME may be more easily and accurately diagnosed in an early stage with OCT as compared to slit-lamp biomicroscopic examination. Various patterns can be easily identified and treatment may be modified accordingly. Being noninvasive, its acceptance as a followup imaging modality to monitor the course of DME and response to therapy is high. It helps to selectively identify cases like VMT and ERM which needs surgical intervention.

REFERENCES