Assessment of Macular Involvement in Blunt Trauma Using SD OCT and Multifocal ERG
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**Aim:** To evaluate the macular changes that happen during blunt trauma using Spectral Domain Optical Coherence Tomography (SD OCT) and Multifocal ERG (MFERG). **Methodology:** 18 eyes with macular involvement were evaluated to assess the damage to the retinal structure using SD OCT and functional changes using MFERG. **Results:** Of the 12 eyes with foveal involvement, 5 had changes including transient foveal lucency, macular holes and atrophy. 2 eyes had choroidal rupture through the fovea. 5 eyes had commotio retina. 9 of 12 eyes had loss of visual acuity corresponding to IS-OS loss and disruption. 6 eyes had choroidal rupture at extrafoveal locations that appeared as anterior protrusion of the RPE and RPE dehiscence (2 eyes). Multifocal ERG showed mild reduction in the inner 3 rings during the acute phase and persistent reduction in foveal atrophy and scarring. **Conclusion:** SDOCT and MFERG are important tools to explain the extent, cause and prognosis for vision in blunt trauma to the macula.

In blunt trauma to the eye, damage is caused by direct contusive force or counter-coup type injury. Traumatic Maculopathy due to closed globe contusion can manifest as commotio retina, traumatic macular holes, indirect choroidal ruptures, retinal hemorrhages and sub ILM hemorrhages. Commotio retinæ result in retinal opacification following blunt trauma. A mild commotio retina usually settles spontaneously with minimal sequelae but more severe cases are associated with visual loss. Sipperley et al noted fragmentation of photoreceptor outer segments and damaged cell bodies in their study of the histological changes in commotio retinæ in primate retinas. Persistent visual impairment may occur in severe cases of commotio retinæ due to damage to the photoreceptors or retinal pigment epithelia. Choroidal ruptures are known to cause severe visual loss especially if they pass through the fovea. They may cause visual loss even if extrafoveal as they may cause pigmentary changes and photoreceptor atrophy. Choroidal Neovascularisation can occur after choroidal rupture which can affect vision by macular involvement. Traumatic Macular hole formation occurs due to contusive force and not by the mechanisms that are responsible for idiopathic senile macular holes and hence may not be amenable to surgery.
With the advent of advanced diagnostic tools like spectral domain OCT and multifocal ERG\textsuperscript{5} the patho-physiology behind the development of these features are more clearly demonstrated. Long term complications include development of choroidal neovascularisation, foveal atrophy and optic atrophy.\textsuperscript{6}

**MATERIALS AND METHODS**

18 eyes of 18 patients who had received contusive force trauma were included in the study. The nature of injury and the size of impacting force were noted. The visual acuity and anterior segment features were evaluated. Posterior segment evaluation included fundus evaluation with slit lamp biomicroscopy, indirect ophthalmoscopy, Spectral Domain OCT by Carl Zeiss Cirrus OCT and multifocal ERG by Roland electrodiagnostics. The presence of the following features were noted in the OCT.


The multifocal ERG was done for the 61 hexagons. The central hexagon P1 values and the summated P1 values for the central 19 hexagons were noted to assess correlation between areas of commotio and retinal sensitivity depression.

Exclusion criteria included any patient with significant media opacity to exclude a proper and repeatable MFERG or OCT.

**RESULTS**

There were 13 males and 5 female patients.

Road Traffic Accidents were the cause of trauma in 7 patients; Trauma with an object not much larger than the orbital rim was seen in 9 patients. 2 patients had trauma with large objects much larger than the orbital rim e.g. football and a large fruit. 3 patients had coexisting orbital fractures. Visual acuity ranged from 6/12 to CFCF.

Macular involvement was either foveal or extrafoveal. 12 patients had changes involving the fovea and 6 patients had entirely foveal sparing changes.

Of the 12 eyes with foveal involvement: 5 had commotio like changes, 3 patients had significant RPE alterations involving the fovea, 2 patients had choroidal rupture passing through the fovea, 1 developed a macular hole and 1 eye developed a large foveoschisis and a BRAO like picture. 6 eyes had extrafoveal involvement in the form of choroidal ruptures.

Hemorrhage in the posterior pole were seen in 11 patients, 8 of these were associated with choroidal ruptures while the other two eyes had superficial retinal hemorrhages.
**Fig. 1:** OCT features of commotio and macular hole

A. Changes in reflectivity of layers with outer lamellar translucency.
B. After a week of area of translucency collapses and produces foveal atrophy.
C. Changes in reflectivity of layers seen in commotion.
D. Traumatic macular hole.

**Fig. 2:** Clinical Presentations of choroidal ruptures

A. Extrafoveal rupture with hemorrhage involving the fovea.
B. FFA of the same patient.
C. Extrafoveal choroidal rupture.
D. Choroidal rupture passing through the fovea.

**Fig. 3:** OCT features in a choroidal rupture

A. Extrafoveal rupture with involvement of the hemorrhage. There is disruption of the IS OS junction.
B. Foveal choroidal rupture.
C. Extrafoveal choroidal rupture.
D. RPE map demonstrating the choroidal rupture. Arrows represent site of rupture.

**Fig. 4:** ONH pit in communication with schisis and retinal ischemia

A. Optic Nerve Head Pit.
B. Area of Retinal Ischemia in contact with circular area of Schisis.
C. Schisis and SRF Cavity in communication with Pit.
D. Macular Schisis.

**Mid peripheral and Peripheral Retinal changes**

Mid peripheral commotio was seen in 7 eyes and a peripheral retinal tear was seen in 1 eye. One eye had preexisting lattices with hole and SRF. Both these eyes underwent laser barrage of the lesions.

**OCT characteristics**

The prominent feature in eyes with commotio retina was variable reflectivity of the inner retinal layers. These reflectivity changes resolved with time and
became imperceptible later and did not have any relation with poor visual outcome later. Some patients who clinically had commotio retina and RPE alterations in the form of a yellowish hypo pigmented area at the fovea revealed on OCT areas suggestive of outer lamellar translucency. Whether this corresponds to an area of tissue loss and separation or whether is just an area of translucency in not known. It is likely to correspond to an area of photoreceptor disruption. After 7 days this area of outer retinal lucency quickly subsided resulting in an area of foveal atrophy and loss of IS OS junction.

**Macular hole formation:** One patient developed a macular hole with a diameter of 460 microns.

**OCT features of choroidal rupture**

Choroidal rupture presented as areas of elevation, which is often associated with subretinal fluid or subretinal hemorrhage. The OCT representation suggest a thinning of the double layer and herniation/protrusion of the underlying layers through this dehiscence. Later OCT evaluation revealed reduction in the protrusion with loss of integrity of IS OS junction.

One patient had a picture that simulated a BRAO with pallid retinal edema suggestive of retinal ischemia. On closer evaluation this patient was found to have an optic nerve pit.

**MFERG characteristics**

The multifocal ERG showed a mean depression of 67% as compared to the normal eye in the central hexagon in patients with acute commotio retinae. After a month this value became nearly same in both eyes, which had vision better than 6/12. The MFERG was depressed in eyes that had worse vision, however a statistically significant difference was not obtained because of the small sample size. The changes in the inner 3 rings were noted when there was diffuse commotio beyond the fovea. The MFERG showed depression of the hexagons in a pattern corresponding to the area of commotio only when there was severe damage and did not show any change in mild to moderate commotio.

**DISCUSSION**

Our series had 8 choroidal ruptures of which two (25%) passed though the fovea. In a study by Wood et al 5 of 30 (17%) eyes they studied had ruptures through the fovea. In a further ten eyes they noted that visual acuity was affected by pigmentary changes involving the fovea or photoreceptor loss. Only 17/30 eyes with choroidal rupture attained a vision of 6/12. They noted delayed complications in their series of CNVM (5), glaucoma (1) and retinal detachment (2). Wood concluded that temporal rupture was more associated
with defective vision, multiple ruptures were associated with macular and optic nerve damage and those without anterior uveal involvement had the best prognosis.

**Macular Hole**

Previous studies have utilized OCT imaging in documenting the natural progression and spontaneous closure of traumatic macular holes anatomically.\(^7\)\(^8\) The exact pathogenesis of macular holes remains uncertain. Ho et al\(^9\) outlined the three basic historical theories regarding etiology—the traumatic theory, the cystic degeneration and vascular theory, and the vitreous theory. Of these, the latter has gathered the most support in the context of idiopathic macular holes. Ismail et al\(^4\) described the occurrence of a macular hole in a case of mild commotio with possible photoreceptor damage. The OCT imaging revealed that the edges of the macular hole were elliptical and irregular with no associated PVD, cortical vitreous condensation, or overlying prefoveal opacity. The characteristics suggest a different mechanism of hole formation from that proposed in idiopathic senile macular holes. Ismail et al hypothesize that mechanical distortion of the retina, relative to the vitreous and underlying sclera, created disruption of the photoreceptor outer segments and creation of a FTMH. It is at the fovea and photoreceptor outer segment level that the retina has the least support from Müller cells and is therefore likely to undergo greatest deformation.

**Retinal Ischemia**

In the patient with the pallid edema there was extensive retinoschisis, which seemed to be connected to an area of prior foveoschisis of an ONH pit. We hypothesize that there was a sudden extension of the schisis due to contusive effects and this was responsible for vascular damage secondarily and this was not a case of BRAO secondary to trauma or Purtscher’s retinopathy.

**Commotio retina**

The multifocal ERG showed a significant reduction in the central hexagons when there was significant visual loss after resolution of commotio, indicating photoreceptor damage. There was no obvious geographic difference in cases with mild commotio but there was involvement of the hexagons in the affected area when there was severe commotio. In a study by Lai et al\(^5\) areas of severe commotio were confirmed objectively by the well-demarcated reduction in retinal response density in the MFERG. The MFERG findings supported the suggestion that permanent visual loss following commotio retinae may occur due to permanent loss of photoreceptors.

**Conclusion**

OCT and MFERG provide very specific indicators of retinal damage that occurs in blunt trauma to macula. They may be prognostic indicators to predict which
patients will go on to have poor visual acuities and larger studies are required to quantify their predictive values.

REFERENCES


