Unusual optical coherence tomography and fundus autofluorescence findings of eclipse retinopathy

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A 63-year-old female patient complained of dimness in the central field of vision in the left eye after viewing an annular partial eclipse without adequate eye protection on 22 July 2009. Funduscopy showed a wrinkled macular surface. Fundus autofluorescence study revealed well-demarcated hyperautofluorescence at the fovea. Optical coherence tomography demonstrated tiny intraretinal cysts. Fluorescein angiography and indocyanine green angiography were unremarkable. Epimacular membrane developed in the following month with deteriorated vision. Vitrectomy, epiretinal membrane and internal limiting membrane peeling were performed. Vision was restored to 20/20 after the operation. Direct sun-gazing may damage the retinal structures resulting in macular inflammation and increased focal metabolism, which explains the hyperautofluorescence. It may also induce epimacular membrane. Fundus autofluorescence might represent a useful technique to detect subtle solar-induced injuries of the retina. The visual prognosis is favorable but prevention remains the mainstay of treatment. Public health education is mandatory in reducing visual morbidity.

Key words: Eclipse retinopathy, epimacular membrane, fundus autofluorescence, optical coherence tomography

Eclipse retinopathy is a specific foveolar lesion caused by direct sun gazing. Typically, it initially produces a yellow-white spot lesion with surrounding gray zone at fovea. The spot fades and can be replaced by a reddish spot with a pigment halo several days later. RPE depigmentation and permanent photoreceptor damage have been reported in severe cases. Fluorescein angiography (FA) may initially appear normal but window defects will develop over time. Visual acuity could range from 20/20 to 20/400 in the acute phase but generally returns to between 20/20 and 20/40 three to six months later.

On 7/22/2009, the excitement of eclipse viewing spread in Africa and Asia. However, improper eye protection may cause eclipse retinopathy. Our patient was one of the victims. Much to our surprise, her clinical manifestations were quite different.

Case Report

This 63-year-old woman watched an eclipse on 7/22/2009. She gazed at the sun directly without adequate eye protection for about 10 minutes. Blurred vision in the left eye developed thereafter. She visited our outpatient clinic 2 days later. On presentation, her best-corrected visual acuity (BCVA) was 20/30 in the left eye. There was no afferent pupillary defect. Anterior chamber and vitreous cavity showed no inflammation. Funduscopy revealed wrinkled surface and grayish discoloration in a left macula [Fig. 1]. FA and indocyanine green angiography were the unremarkable. Fundus autofluorescence (FAF, excitation 488 nm, emission 500 nm) showed hyperfluorescence in the left foveola [Fig. 2]. Optical coherence tomography (OCT) showed mild foveal elevation [Fig. 3]. Epimacular membrane developed after one month [Fig. 4]. Her BCVA dropped to 20/40.

Sutureless vitrectomy with membrane peeling was performed uneventfully after 4 months of observation. Her BCVA improved dramatically to 20/20 three weeks after the operation. Fundus appeared normal. FAF showed resolution of previous autofluorescent lesion [Fig. 5]. OCT also revealed no more epimacular membrane [Fig. 6].

Discussion

Eclipse retinopathy usually produces a small foveal or parafoveal yellow-white spot with a surrounding gray zone. The spot may become reddish several days later. There can be depigmentation of RPE in severe cases. Visual recovery is usually poor if OCT detects loss of the hyper-reflective layer in the photoreceptors’ outer segments.

In our case, however, the formation of epimacular membrane progressed rapidly with quick deterioration of vision. Fortunately, vision improved well after surgical removal of the membrane. To our knowledge, no similar findings have been published in the current literature. There may be some argument regarding the relation between the development of epiretinal membrane and sunlight exposure. In our opinion, since the patient had no identifiable risk factor for epiretinal membrane formation, such as diabetes, retinal vascular disease, or previous laser treatment, it was too fast for an idiopathic retinal membrane to develop just in 1 month. There must have been some predisposing factor. We believe that it was the photic injury and related inflammation of retina that induced the rapid formation of epiretinal membrane.

FAF is a relatively novel, non-invasive imaging technique based on the autofluorescent properties of natural occurring retinal fluorophores. It is generally accepted that the lipofuscin-laden cells of the RPE are the main source of FAF. Subtle RPE abnormalities, overlooked on cursory ophthalmoscopy, can be efficiently imaged using FAF. Reduction of the content of lipofuscin, the main fluorophore
Figure 1: Fundoscopy revealed wrinkled macular surface with grayish discoloration

Figure 2: Fundus autofluorescence showed hyper-autofluorescence in foveola

Figure 3: Optical coherence tomography showed mild foveal elevation

Figure 4: Epimacular membrane developed after one month

Figure 5: Fundus autofluorescence showed resolution of previous autofluorescent lesion

Figure 6: Optical coherence tomography revealed no more epimacular membrane

of the RPE, has been documented following light-induced loss of photoreceptors.[4]

In dell’Omo’s presentation, FAF of chronic solar retinopathy showed small areas of decreased autofluorescence at fovea,
surrounded by a ring of faint increased autofluorescence. The hypo-autofluorescent areas were thought to be secondary to degeneration of RPE cells and photoreceptor cells, with consequent reduced accumulation of lipofuscin. The OCT also showed a defect in photoreceptor and RPE layer, proving his thesis.

In our case, however, there was hyper-autofluorescence at the fovea. And OCT did not show the defect in photoreceptor or RPE layer. We think the difference may be due to relatively early phase and smaller extent of photo injury in our case. Due to these two factors, photoreceptors and RPE cells were injured but not totally destructed at the fovea. Hyper-autofluorescence might be a sign of increased metabolism. There should be self-repair in process and caused increased metabolism in this area.

Issa also presented a patient of solar retinopathy, in whom fundus autofluorescence showed a mottled, increased signal at the fovea. The patient, however, had solar exposure many years before presented to Issa. We think the persistent macular lesion on AF and impaired vision were due to more intense sunlight exposure and more severe macular injury compared to our patient.

Direct sun-gazing may damage the retinal structures resulting in macular inflammation and increase focal metabolism, which explains the hyper-autofluorescence. It may also induce epimacular membrane, but larger case series are required to confirm this finding. OCT and FAF are useful and non-invasive techniques to detect subtle solar-induced injuries of the retina. The visual prognosis is favorable but prevention remains the mainstay of treatment. Public health education is mandatory in reducing visual morbidity.

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