Saidova L.X., Makinskaya D.B.

GÜNƏŞ MAKULOPATİYASI (KLİNİK HAL)

XÜLASƏ

Göz dibinin nadir patologiyalarından biri "Günəş makulopatiyası" klinik halı təqdim edilir. Müayinə zamanı bu xəstəliyin diaqnozu üçün xüsusi müayinə metodlarının - pattern-elektroretinogram (pattern-ERG), mikroperimetriya və optik koherens tomoqrafiya (OKT) - ən informativ olduğu təsdiq edilmişdir.

Təyin olunan müalicədən sonra xəstənin görmə itiliyi 0,4-dən 1,0-ə yüksəlmişdir.

Günəş makulopatiyasının inkişafının qarşısını almaq üçün pasiyentlərə günəş işığının potensial mənfi təsirləri barədə məlumat verilməli, əhali üçün maarifləndirmə tədbirləri təşkil edilməli və göz qoruyucu vasitələrdən istifadə etmələri tövsiyə olunmalıdır.

Açar sözlər: günəş makulopatiyası, OKT, pattern-ERG, mikroperimetriya

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SOLAR MACULOPATHY (CLINICAL CASE)

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SUMMARY

The article presents a clinical case of the patient with rare retinal pathology called "Solar Maculopathy." During the examination, it was found that the most informative diagnostic methods for this disease are special techniques such as pattern-electroretinogram (pattern-ERG), microperimetry, and optical coherence tomography (OCT).

After the prescribed treatment, the visual acuity increased from 0.4 to 1.0.

To prevent the development of solar maculopathy, patients should be informed about the potential negative effects of sunlight, educational activities for the population should be conducted, and the use of eye protection should be recommended.

Key words: solar maculopathy, OCT, pattern-ERG, microperimetry

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СОЛНЕЧНАЯ МАКУЛОПАТИЯ (КЛИНИЧЕСКИЙ СЛУЧАЙ)

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Авторы заявляют об отсутствии конфликта интересов (финансовых, личных, профессиональных и других). Поступила 07.02.2025 Принята к печати 04.04 2025 Представлен клинический случай редкой патологией глазного дна - «Солнечная макулопатия». В ходе обследования для диагностики данного заболевания была подтверждена наибольшая информативность специальных методов обследования - паттернэлектроретинограмма (паттерн-ЭРГ), микропериметрия и оптическая когерентная томография (ОКТ).

После назначенного лечения острота зрения увеличилась от 0,4 до 1,0.

Во избежание развития солнечной макулопатии следует информировать пациентов о возможных негативных эффектах солнечного света, проводить образовательные мероприятия населения, а также рекомендовать пользоваться защитными средствами для глаз.

Ключевые слова: солнечная макулопатия, ОКТ, паттерн-ЭРГ, микропериметрия

Sunlight, which is the main source of life on Earth, can cause dangerous and irreversible eye damage under certain circumstances. Solar maculopathy or light-induced retinopathy is damage to the macula caused by exposure to high-intensity light radiation in the longwavelength visible, ultraviolet (UV), and infrared (IR) spectra. This disease can develop after prolonged observation of a solar eclipse without protection, as well as after extended direct exposure to the sun or sunlight reflected from surfaces [1, 2]. Eye damage from nonionizing electromagnetic radiation can also be caused by artificial light sources such as arc projectors, mercury-quartz lamps, welding high-pressure machines, xenon lamps. medical laser scalpels, ophthalmocoagulators, and other devices [2, 3]. Retinal photodamage may occur in children after attending laser shows or playing with laser pointers [3].

Excessive exposure of solar energy on the eyes often leads to a decrease in visual acuity and/or the appearance of central or paracentral scotomas [1, 4]. Studies on the harmful effects of solar radiation on the eyes and cases of solar retinopathy were recorded as early as the 18th century.

The risk of developing this pathology primarily depends on the intensity, duration, and spectrum of exposure. Risk factors for macular damage include dilated pupils, young age, the presence of transparent optical media (such as a transparent lens), and albinism. Protective factors include high refractive error, cataracts, and dark pigmentation of the retina [5].

Solar radiation reaching Earth's surface spans from 250 to 1800 nm, with 2% ultraviolet (UV), 40% visible, and 58% infrared (IR) radiation. UV radiation below 250 nm and IR radiation above 1400 nm, produced by artificial light sources, can damage the eyes [6]. Light passing through the eye's optical media reaches the retina, including the pigment epithelium and photoreceptor layer. The cornea and lens protect against harmful UV rays by blocking most radiation below 400 nm, while allowing visible and infrared light (400-1400 nm) to reach the retina [5, 7].

There are two mechanisms of UV radiation's damaging effects on the structures of the retina: thermal and photochemical. The thermal mechanism occurs due to protein coagulation. Short-wavelength rays, refracted in the transparent media, reach and focus on the retina. The energy of these rays is sufficient to coagulate proteins during the time of a single blink reflex [2, 8].

According to the research of Wu and his colleagues [8, 9, 10], photochemical damage to the retina occurs through Type 1 (direct reactions with proton or electron transfer, leading to the formation of free radicals) and Type 2 (reactions involving active forms of oxygen, due to the development of oxygen-dependent toxicity) mechanisms [9, 10].

Purpose - to analyze a clinical case of solar maculopathy using the results of specialized diagnostic methods: OCT, ERG, multifocal ERG, pattern-ERG, and microperimetry.

Clinical Case

PatientA.Sh., born in 2008, presented to the National Ophthalmology Centre named after Academician Zarifa Aliyeva on November 26, 2024, with complaints of sudden vision loss in both eyes. From the anamnesis: On November 23, 2024, during the day, the patient spent an hour looking at the sun without protective measures, after which he noted blurry vision. Routine examination methods were conducted: refraction, tonometry, ophthalmobiomicroscopy, as well as special methods such as OCT, ERG, multifocal ERG, pattern ERG, and microperimetry. Due to the onset of an allergic reaction in the patient during the test, fluorescein angiography (FA) was not performed.

During the examination, visual acuity in both eyes was 0.4 (did not respond to correction).

IOP OD = 17.1 mmHg; IOP OS = 14.5 mmHg

Pathological changes in the anterior segment of the eyes were not detected during the examination.

Upon ophthalmobiomicroscopic examination, the following was noted: OU – the optic nerve discs were pink, and the borders were clear. In the central zone, the foveolar reflex was slightly dull, and the parafoveal area had a yellowish hue. The course and caliber of the arteries and veins were normal. No pathological changes were detected in the peripheral retina either.

OCT was performed using the ZEISS CIRRUS 5000 machine (country of origin – Germany). During the examination of the outer segment of the photoreceptors, the ellipsoid zone, and the outer limiting membrane, a destructive focus was identified in the shape of a rectangle (height – 340 μ m; width – 22 μ m). A stripe of damage was visualized in the foveolar area, extending outward to the retinal pigment epithelium (Figure 1).

The results of the conducted ff-ERG (Roland Consult Super Color Ganzfeld Q450 SC-Germany) showed no pathological changes (Figure 2).



Figure 1. *OCT images of the right and left eye of the patient during the initial examination. A line of damage is determined, extending from the inner layer to the retinal pigment epithelium (RPE).*











Figure 2. Scotopic and photopic ERG examination of a patient with solar maculopathy: the functions of the rods and cones of the retina are within the normal range.

The results of the conducted ff-ERG (Roland Consult Super Color Ganzfeld Q450 SC-Germany) showed no pathological changes (Figure 2).

In the multifocal ERG, performed on the ROLAND CONSULT (Germany), retinal activity remained within the normal range **(Figure 3).**

According to the pattern-ERG (Roland Consult Super Color Ganzfeld Q450 SC-Germany), the latency of the N35-P50 component was within normal limits, unlike its amplitude, which was moderately reduced. However, the latency and amplitude of the P50-N95 component were within the normal range (no changes in the ganglion cells were detected) (Figure 4).

Microperimetry was performed using the MAIA Macular Integrity Assessment (Italy). A decrease in retinal light sensitivity was observed in the central part. Additionally, a small change in functional activity in the foveal area was found. An anomaly was recorded, which appeared as "noise" in the central visual field (Figure 5).



Figure 3. Multifocal ERG: a) right eye; b) left eye.



Figure 4. *Pattern-ERG: changes in the latency and amplitude of the N35-P50 and P50-N95 components are shown.*



Figure 5. *Microperimetry of both eyes. OU: a decrease in the sensitivity of the macular area of the retina is determined.*

Based on the medical history and diagnostic examination results, the patient was diagnosed with "Solar Maculopathy." Local treatment was prescribed (non-steroidal antiinflammatory drugs (NSAIDs), citicoline, and retinal protectors).

Follow-up examinations were conducted 1 week, 2 weeks, 1 month, and 2 months after the treatment started. One week after the visit to the clinic and the initiation of treatment, the destructive focus in the area of the outer limiting membrane, ellipsoid zone, and outer segment of the photoreceptor layer was absent (Figure 6, a).

After 2 weeks, the patient subjectively noted an improvement in vision. Objectively, visual acuity was 0.8, and OCT results showed no changes in the middle and inner layers of the neuroretina. A defect was present in the outer limiting membrane, ellipsoid zone, and outer segment of the photoreceptor layer (Figure 6, b).

After one month, visual acuity in both eyes was 0.9. OCT showed restoration of the integrity of the outer limiting membrane and a significant reduction in the defect in the ellipsoid zone.

Upon repeating the pattern-ERG, positive dynamics were observed: latency of the N35-P50 component was within the normal range, amplitude in the right eye was slightly reduced, and in the left eye, it showed moderate reduction. Latency and amplitude of the P50-N95 component were within normal limits. After treatment, repeat microperimetry showed restored retinal sensitivity in the



Figure 6. *Example of the structural condition of the macula in OCT examination: a) after 1 week; b) after 2 weeks.*

central area. Improvement in light sensitivity was observed in areas previously reduced due to photoreceptor damage. The conducted antioxidant therapy resulted in improved microperimetry results, such as a reduction in "noise" in the visual field and enhanced fixation accuracy (Figure 7).

After 2 months, the patient's visual acuity was 1.0, and all electrophysiological indicators and OCT data were within normal limits (Figure 8).

Discussion

It is known that the retina consists of 10 layers, but in solar maculopathy, the outer layers (pigment epithelium, photoreceptor layer, outer limiting membrane, and ellipsoid zone) are damaged [11]. Changes in these layers are most prominently observed on OCT. This pathology was first described using OCT by Behman and his colleagues [12, 13].

In daily life, retinal damage from sunlight typically does not occur because the eye is protected by an effective antioxidant system. Pigments, such as kynurenines in the lens, and melanin in the choroid and retina, absorb radiation and scatter its energy, preventing damage. Light radiation can only cause damage to the tissue in which it is absorbed. Histological studies have shown that the pigment epithelium and the outer segments of photoreceptors are particularly susceptible to solar damage [14].

The primary damage likely occurs in the RPE [15]. Compared to other retinal layers, the RPE layer has the highest absorption coefficient (over 60%) for the visible spectrum. RPE cells, which play a critical role in maintaining retinal health, are the first to absorb light energy.

These cells contain melanin, which is responsible for absorbing and scattering light. When exposed to excess light, particularly UV radiation, RPE cells absorb more energy than they can handle, leading to oxidative stress and cellular damage.

Photoreceptor cells (rods and cones) are the next layer affected by phototoxicity. These cells convert light into electrical signals, but when exposed to excessive sunlight, their photopigments (like rhodopsin) can undergo photobleaching. Photobleaching is a process in which light destroys the photopigment molecules, impairing the cell's ability to process light and leading to cell dysfunction or death. Furthermore, the lack of protection







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Figure 7. *Results of examinations one month after the start of treatment: a) OCT of the right and left eye; b) microperimetry of the right eye of the patient; c) microperimetry of the left eye; d) pattern-ERG of both eyes.*



Figure 8. After 2 months OCT of the right and left eye. Restoration of the defect in the foveal area is noted.

of the fovea by the ganglion cell layer makes it more vulnerable to solar radiation exposure [9, 16]. The second most informative method is pattern-ERG. Unlike ff-ERG and m-ERG, which provide information about the entire retina's response and give a topographic image of the central retina's function, respectively, pattern-ERG allows the assessment of the electrical activity of both macular cells and ganglion cells of the retina.

FA is also the third most important diagnostic tool. In solar maculopathy, FA results can vary depending on the stage and severity of the condition. In the early stage, hypofluorescence is observed, which is associated with damage to the RPE. In the later stages, hyperfluorescence is observed due to leakage from damaged vessels. Additionally, window defects are noted due to damage to the RPE.

There is a number of evidence regarding the spontaneous recovery of solar maculopathy without specific treatment. In most cases, solar maculopathy presents as temporary changes, and depending on the extent of retinal damage, partial or full recovery of its functionality is possible. Spontaneous recovery may occur in less severe forms of solar maculopathy, where damage to photoreceptors and other macular cells is minimal.

This is because cellular structures may partially regenerate, and neuroplasticity can compensate for the loss of function. However, prolonged or intense exposure to sunlight can lead to irreversible damage to retinal cells, and in such cases, spontaneous recovery is limited, requiring treatment or specialized therapy.

The rationale behind using medication for solar maculopathy is to help manage symptoms, reduce inflammation, and potentially speed up recovery or minimize long-term damage, especially if the condition is severe. Anti-inflammatory medications, like NSAIDs, may be used to reduce this inflammation and prevent further damage to the retina.

In some cases, medications or treatments can potentially speed up recovery, especially if the damage to the retina is significant. Some research suggests that antioxidants or other specific treatments may help promote retinal healing [17].

It took two months for our patient to recover his vision. According to some authors, improvement in visual acuity begins one week after the exposure. Full recovery typically occurs within 3-6 months, after which vision remains stable [18].

Currently, the effect of steroid treatment on macular edema, which develops as a complication of this condition, is being studied. From the clinical cases described, treatment with systemic corticosteroids led to the development of central serous chorioretinopathy [19].

Conclusion

Solar maculopathy is a retinal condition caused by direct sunlight exposure, leading to a sharp decline in vision lasting from weeks to months. To prevent it, patients should be informed about the risks of not following protective measures, such as avoiding direct sun exposure and using protective eyewear. This condition primarily affects the retina's outer layers and pigment epithelium. Key diagnostic methods include OCT, microperimetry, and pattern-ERG. The prognosis is favorable, with vision recovery within 3 to 6 months. Young age is a risk factor.

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