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CASE REPORT

Accidental focal laser injury—a correlation of electrophysiology, perimetry and clinical findings

Barbara Link · Georg Michelson · Folkert K. Horn · Anselm Jünemann

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Abstract *Background* Laser injuries to the eyes occasionally occur in occupational accidents or during free-time activities. The aim of this study was to compare clinical and functional findings. *Methods* We describe a 34-year-old male complaining about reduced visual acuity after an accidental laser injury. We correlate changes in the multifocal ERG with visual field defects due to focal laser application 1.5 years after the trauma. *Results* A small central retinal scarring resulting in a perimetric defect and a focal reduction of amplitude in the multifocal ERG have been observed in an area closely corresponding to the area where the scarring and the field losses occurred. *Conclusion* Structural and perimetric damage as a consequence of laser injury can be associated with focal change in multifocal ERG.

Keywords Laser burn · Multifocal ERG · Perimetry

Introduction

Accidental laser injuries to the eye are a well-known threat to central vision (e.g. [1, 2]). Damage to the eye by accidental laser application can occur by photomechanical, photothermal or photochemical mechanisms (see [3] for a short summary of each of these mechanisms). Trauma due to laser injuries can have a tremendous impact on visual function, either by direct damage (retinal holes, scarring or haemorrhages) to the globe or by inducing long-term changes leading to visual dysfunction (i.e. cataract, choroidal neovascularisation) [4]. Here we report about a patient suffering from an accidental laser injury and show a correlation between electrophysiology, perimetry, angiography and fundus autofluorescence.

Patient and methods

A 34-year-old male Caucasian complained about loss of vision in his right eye after an accidental occupational laser injury with an Alexandrite laser (755 nm, pulse energy: 25 J/cm^2). The patient worked as an engineer in a laboratory and was familiar with this type of laser. The injury presumably occurred during visual inspection of the laser crystal due to an unintentional discharge of the high-voltage condenser when the device was in stand-by mode.

The first examination in our department took place 4 weeks after the trauma. Fluorescence angiography was performed several times during the follow-up; no leakage was present in either of the angiograms. Multifocal ERG (mfERG) (RetiScan, 61 segments, five cycles of 40 s each, distortion factor 4:1, fibre

B. Link (✉) · G. Michelson · F. K. Horn · A. Jünemann
Augenklinik mit Poliklinik, Universität Erlangen-Nürnberg, Schwabachanlage 6, 91054 Erlangen, Germany
e-mail: barbara.link@uk-erlangen.de

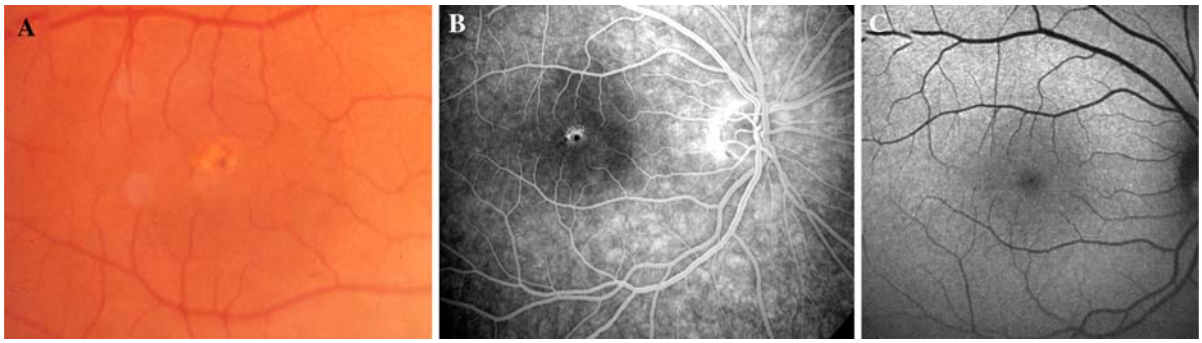

Fig. 1 (**a**) Focal scarring in central retina (1.5 years after trauma), (**b**) fluorescence angiography (4 weeks after trauma), and (**c**) fundus autofluorescence (1.5 years after trauma)

electrode (DTL), 190 cd/m^2 bright fields, 9 cd/m^2 dark fields, 90 cd/m^2 background, pupils dilated), perimetry (Octopus 101 (C08) and Goldmann) and fundus autofluorescence (Heidelberg Retina Angiograph II (HRA II), Heidelberg Engineering) were performed on the last examination 1.5 years after the trauma.

All tests were performed in the context of a routine medical examination or a medical estimate. Informed consent was obtained prior to each examination, after having been explained thoroughly to the patient.

Results

The left eye did not show any sign of pathology of any kind, either on clinical examination or on further examination.

At the patient's first presentation 1 month after the accident, visual acuity was 20/100 (OD, eccentric fixation) and 20/20 (OS), intraocular pressure was 22 mmHg (OD) and 9 mmHg (OS). Visual acuity did not change during the follow-up, intraocular pressure in the last follow-up was 18 mmHg (OD) and 11 mmHg (OS).

Clinical examination in the last follow-up demonstrated the following findings in the right eye: A segmental iridal stromal atrophy was visible in the upper nasal quadrant and melanin granula were found on the surface of the whole iris. An increased transillumination, a reduced and delayed direct light reflex and deformation during pupil dilatation in this segment could be observed. The lens was centrally clear with a slight opacification beneath the iridal defect. The anterior chamber was deep and showed neither anterior synechia nor intraocular irritation, the chamber angle showed abundant deposition of melanin granula. The vitreous was attached, the optic nerve head did not show signs of atrophy and peripheral retina was attached without signs of any disease. Central retina showed a scar with RPE hyper- and hypopigmentation (Fig. 1a).

Fluorescence angiography showed a focal hyper- and hypofluorescence due to central atrophy and proliferation of the RPE. There were no signs of choroidal neovascularisation (Fig. 1b).

Fundus autofluorescence of the right eye did not demonstrate any abnormality (Fig. 1c). Perimetry of the central 10° showed reduced sensitivity about 16 dB in the central six points (Fig. 2a, b).

MfERG examination was performed as described above. The central and most peripheral amplitudes were slightly larger in the right eye. Mainly segment 22 corresponds to the area of retinal damage by the laser injury: in this field, an amplitude reduction could be observed (N1–P1) in comparison with the intraocular adjacent areas as well as in relation to the corresponding field of the contralateral eye. The amplitude of this segment was smaller; peak time was equal to the corresponding 23 in the other eye (Fig. 2c–f): right eye, field 22: amplitude (N1–P1) 59 nV/deg^2, peak time (P1) 37.7 ms; left eye, field 23: amplitude (N1–P1) 72 nV/deg^2, peak time (P1) 38.7 ms.

Discussion

In this report we demonstrate a small retinal scar resulting in focal changes in the mfERG.

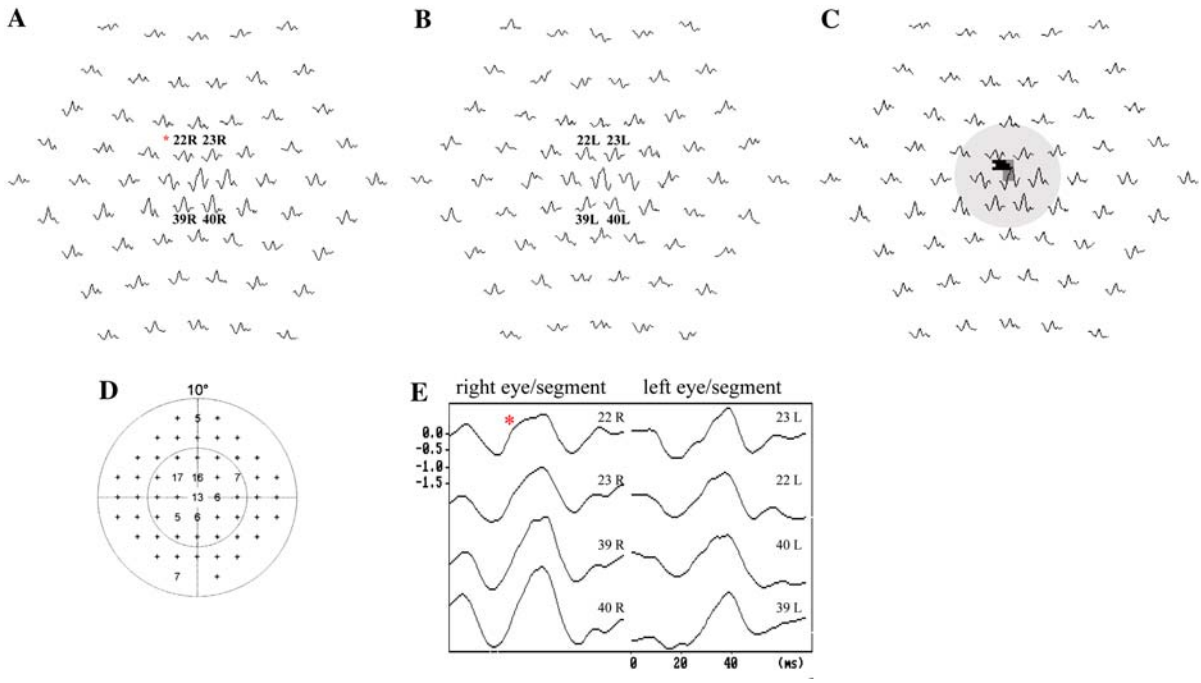

Fig. 2 (**a**, **b**) MfERG of the right (**a**) and left eye (**b**); the site of the laser injury (right eye) is marked by an asterisk. Scale is identical in both figures. (**c**) Amplified corresponding curves of right and left eye: The ratio of amplitudes between right and left eye points up the defect in the right eye: nasal upper field (22R/23L) (scar in OD!): 0.82, temporal upper field (23L/23R): 1.23, nasal lower field (39R/40L): 1.78, temporal lower field (40L/39R): 1.46. This ratio indicates amplitude reduction in the field 22R by an asterisk. This correlates well with the area damaged by the laser. (**d**) Perimetry of the central 10° demonstrating the central defect in the right eye. Values show the depth of the defect in dB relative to a normative database. (**e**) Right eye: Superimposed perimetry on mfERG shows a correlation of reduced ERG amplitude with localised perimetric defect

Although in this patient we found a difference in intraocular pressure, we could not find any signs of glaucomatous atrophy. The presence of melanin granula on the surface of the iris and the chamber angle of the traumatised eye explained the asymmetrical intraocular pressure.

Fluorescein angiography helps to confirm the diagnosis and is an important tool to exclude choroidal neovascularisation that can occur in the course of retinal laser injury [4].

Autofluorescence is mainly from the lipofuscin within the retinal pigment epithelium [5]. Lipofuscin accumulates as a byproduct of phagocytosis of the photoreceptor outer segments in the pigment epithelial cells. In an earlier report fundus autofluorescence decreased immediately after laser treatment, increased after 1 month and decreased again after 1 or 2 years to normal autofluorescence [6]. This is in line with the findings in our patient 1.5 years after a laser injury.

This report correlates changes in the mfERG with visual field defects after focal laser application in humans. In this patient we saw slightly reduced amplitude in the affected area. Peak times did not change in the affected segment. Pathology of the mfERG correlates well with perimetry. Lens opacification can alter electrophysiological recordings like flash ERG and multifocal ERG. In the patient described here, the central part of the lens was clear with only a slight opacification in the periphery. Thus it can be assumed that there is no influence of the cataract on the central 10° in mfERG in this particular case. A full-field ERG was not performed at any of the visits, as mfERG, angiography, measurement of fundus autofluorescence and perimetry were considered more decisive and patients' compliance and patience tend to be limited the more examinations are performed.

The present finding is in line with earlier observations that the mfERG is able to detect focal damage

to the retina [7, 8]. Changes in mfERG in cases of focal retinal impairment due to solar retinopathy [9, 10] and other eye diseases such as retinal vascular branch occlusion [11] have been reported previously. However, the pathological findings in those patients are larger and electrophysiological changes in the mfERG more extensive. Our report shows the ability of the mfERG to detect even very small retinal disturbances.

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