Reversible retinoschisis following high voltage electrical injury evaluated with optical coherence tomography and electrophysiology

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ARTICLE INFO

Keywords:
Electrical injury
Retinoschisis
Electrophysiology

ABSTRACT

Purpose: To report a carefully studied case of high voltage electrical injury of the retina and optic nerve with anatomically reversible retinoschisis.

Methods: Observational case report.

Results: A 22 year old power company worker was electrocuted with 12,000 V, with his left forehead being the exit point of the current. After regaining consciousness he reported decreased vision with both eyes. He was extensively tested with optical coherence tomography (OCT) and angiography (OCT-A), fundus photography, fluorescein angiography (FA), multifocal electroretinography (mfERG), full field electroretinography (fERG), visual evoked potentials (VEP), and Goldmann-type Octopus automated perimetry in addition to careful clinical examinations. Our investigations revealed severe visual field constriction in both eyes, severe coagulative damage leading to inner and outer retinal atrophy, subretinal fluid collection, retinoschisis cavities, and papillitis. Initially he was treated with 100 mg prednisone per day for one week and 250 mg acetazolamide per day which was continued for 3 months. Over time the OCT signs of retinoschisis resolved but visual acuity and visual field improvement did not occur.

Conclusion: Resolution of retinoschisis cavities following electrical damage does not necessarily lead to improvement in visual function due to the many accompanying structural injuries.

1. Introduction

Ocular damage from electrical injury is well reported, the most common being corneal injuries and cataracts. Retinal and choroidal changes are infrequently seen but do include macular edema, macular holes, central retinal vessel attenuation or occlusion, and retinal detachment. However, few of these cases have been studied systematically with OCT or electrophysiology.

We report a case of high voltage electrical injury where the only permanent injury was ocular in nature and consisted mainly of multilayer retinoschisis OU combined with optic neuropathy. The patient was tested extensively with OCT, fundus photography, FA, multifocal mfERG, full field ERG, VEP, and Goldmann-type Octopus automated perimetry, in addition to careful clinical examinations.

2. Case presentation

A 22 year old power company employee with no past medical or ocular history was electrocuted by 12,000 V with the point of contact at his left forehead. He reported bilateral decreased vision upon awakening in the hospital. Subjectively his vision continued to decline with both eyes for one week until stabilization. He had severe electrical burns diffusely over his face and scalp, and suffered one seizure after the electrocution. While in the hospital he was started on topical ketorolac twice a day in both eyes. He presented to our clinic 28 days following the injury. His visual acuity was 20/70 OD and CF3 OS. Intra-ocular pressure (IOP) was within normal limits. He had a 4+ afferent pupillary defect (APD) OS. His confrontational fields were severely constricted OD > OD. Posterior exam was significant for mild disc pallor OU and mild vascular attenuation. He underwent optical coherence tomography (OCT) (Cirrus HD-OCT 5000, Zeiss, Germany) which showed abnormal retinal nerve fiber layer (RNFL) thickness bilaterally, and ganglion cell complex (GCC) thinning bilaterally as seen in Fig. 1.

Fig. 1 images C-F show the OCT findings prior to treatment. The right eye had multilevel retinoschisis cavities with IS/OS disruption in
the macula, and multilevel retinoschisis cavities including incomplete detachment of the ILM likely with vitreoretinal traction secondary to thermally induced vitreous contraction. The left eye showed more extensive damage, with pronounced inner and outer retinal atrophy, subretinal fluid, retinoschisis cavities around the macula and optic nerve. The relatively higher degree of damage OS is likely secondary to proximity to the point of contact at his left forehead.

Fluorescein angiography (FA) performed by the referring ophthalmologist showed no leakage and thus the OCT changes were consistent with retinoschisis. The patient was started on acetazolamide 250mg bid for 3 months and prednisone 100mg orally for one week.

He returned for follow up 3 months after electrocution with no subjective changes in his vision. However, repeat OCT showed improvement in the retinoschisis when compared to the previous OCT.

Fig. 1 G-J show the patient’s OCT findings following treatment. The retinoschisis cavities have significantly improved in both eyes, however there is also more pronounced inner and outer retinal atrophy, and optic nerve atrophy OS > OD. He underwent additional evaluation including microperimetry (MP) microperimeter, Nidek Technologies, Italy), multifocal electoretinography (mfERG) (VERIS™ 5.2, Electrodiagnostic Imaging, LTD, Redwood City, CA) (Fig. 2), full-field elec
toretinogram (ERG) (Espion V5, Diagnosys LLC, Lowell, MA), visual evoked potential (VEP) (Espion V5, Diagnosys LLC, Lowell, MA), and automated Goldmann-type visual field (GVF) evaluation (Octopus 900, Haag-Streit International, Switzerland).

Microperimetry of the right eye (Fig. 2A) showed loss of the sensitivity in the entire nasal half of the macula, with spared parafoveal sensitivity and foveal loss. Left eye showed complete loss of macular sensitivity, with pronounced eccentric fixation (Fig. 2B). Multifocal ERG appeared consistent with the microperimetry findings, showing significant depression of the electrical activity of the retinal cells in the nasal macula in the right eye and severe depression of the mfERG responses in the left macula, with unrecordable activity within the central 15° (Fig. 2C–D). Full-field ERG showed low borderline peak amplitudes.

Fig. 1. (A–B): Fundus photos prior to treatment of the right eye (A) and the left (B) eye, showing pigmentary changes. (C) OCT of right eye macula prior to treatment showing discontinuity in the IS/OS, schisis in the ganglion cell layer (GCL), inner plexiform layer (IPL), inner nuclear layer (INL), outer plexiform layer (OPL), and outer nuclear layer (ONL); primarily in the OPL. (D) OCT of the right eye optic disc prior to treatment showing IS/OS disruption, schisis in the GCL, IPL, INL, OPL, and ONL, and vitreoretinal traction. (E): OCT of left eye macula prior to treatment showing loss of the IS/OS and ELM, photoreceptor shedding, schisis in the GCL, IPL, INL, OPL, ONL. (F): Left eye showing similar findings to the macula. (G): OCT of the right eye macula 3 weeks after treatment showing improvement in schisis cavities, inner and outer retinal atrophy, and disruption of the IS/OS junction. (H): OCT of the right eye optic nerve 3 weeks after treatment showing improvement in schisis cavities, improved vitreoretinal traction, and inner and outer retinal atrophy. (I): OCT of the left eye macula 3 weeks after treatment showing improvement in schisis cavities and severe inner and outer retinal atrophy. (J): OCT of the left eye optic nerve 3 weeks after treatment showing improvement in schisis cavities, severe inner and outer retinal atrophy, and papillary atrophy.
in the right eye and depressed peak amplitudes in the left eye, with delayed peak times for both the rods and the cones in both eyes (Fig. 2, E-F). VEP showed delayed P100 peak times in both eyes. Automated perimetry was possible only on the right eye, as the left eye was unable to see the fixation target. Right eye visual field showed mild constric-
tion of the peripheral isopters, especially in the superior and nasal
quarters, and a large cecocentral scotoma.

He was again examined 6 months later and OCT Angiography (Zeiss
AngioFlex OCTA Angiography) was obtained showing decreased perfu-
sion around the nerve and macula more severe in the left eye than the
right (Fig. 3).
3. Discussion

As noted by Spies and Trohman, tissue injury from electricity primarily results from thermal energy, the severity of which is determined by the amount of current, voltage, resistance, type of current, pathway of current and duration of contact with the electrical source. 7 Tissues like dry skin, bone and fat that have relative high resistance tend to increase in temperature leading to protein denaturation and coagulation. Another mechanism for cellular damage is electroporation, which is the creation of pores in cell membranes by electrical current. 8 Electroporation can cause cell death without significant thermal effects.

Macular tissue is particularly sensitive to electrical damage due to the high resistance from the high density of cells, and concentration of intracellular melanocytic granules. 9 Thermal shrinkage of the vitreous can also lead to vitreomacular traction and cause tears or holes in the retina. 10 Optic nerve tissue has a relatively low resistance to electrical current relative to the surrounding retina and is less likely to exhibit damage. When it does occur, optic nerve damage may be from electroporation of the cells under high electrical load and from thermal coagulation of the surrounding retinal tissue immediately adjacent to the nerve. 3

In a case series by Boozalis et al. of 149 patients with high voltage burns, the most common ocular sequelae were cataracts, recurrent iritis, macular holes, and one patient with central retinal artery occlusion. 1 Corneal injury is a common ocular injury from electrical current, secondary to damage or loss to any layer of corneal cellular structure including endothelium, stroma, or epithelium. Depending on extent of damage, this can be transient or permanent. Anterior chamber signs including recurrent iritis and hyphemas have been reported, with occasional development of secondary glaucoma from chronic uveitis. 7 The most common complication of high voltage electrical injury is the development of lenticular opacities.

Macular holes seem to be more common after lightning strike injuries. In a case of bilateral macular holes after high voltage electrical shock, Ravikumar and colleagues reported that surgical repair allowed for anatomical improvement, however there was no improvement in visual outcome. 3

Previous evaluations of ocular electrical injury with OCT have not found evidence of retinoschisis or documented structural changes following treatment, and to our knowledge there have not been reports including the electrophysiology we have presented.

Given the extensive damage that our patient sustained, it is likely the retinoschisis cavities and subretinal fluid had a multifactorial origin. The retina suffered severe coagulative damage leading to inner and outer retinal atrophy, vitreoretinal traction from thermal vitreous contraction, and possibly electroporation of the vasculature allowing for chronic leakage and development of the retinoschisis cavities.

OCTA one year after the injury showed significant vascular attenuation which corresponded to the OCT atrophy in both eyes. We attribute the lack of functional improvement despite the resolution of the retinoschisis cavities to many OCT findings including severe inner and outer retinal atrophy, disruption of the IS/OS junction, photoreceptor shedding, neurosensory detachment of the macula, and a thermal papillitis.

Financial interests

None of the authors have any financial interests to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://
doi.org/10.1016/j.ajoc.2020.100760.

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