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Development of Optic Neuropathy and Foveal Pseudocyst in a Case of High-Voltage Electrical Injury: A Three-Year Follow-Up

Yüksek Voltajlı Elektrik Yaralanması Olan Olguda Optik Nöropati ve Foveal Psödokist Gelişimi: Üç Yıllık Takip

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Summary
A 35-year-old male patient had a decreased vision in his right eye, with a visual acuity of 20/400, after a high-voltage electrical injury. Corneal edema, Descemet's membrane folds, cataract, macular hole, and optic neuropathy were detected in his right eye. Intravenous pulse steroids were administered. On the third day of treatment, the visual acuity in the right eye improved to 20/200 and the corneal edema resolved. Two months later, the patient's visual acuity was still 20/200, while optic disc pallor, epiretinal gliosis, a non-full-thickness macular hole, and pseudo-cyst formation were detected in his right eye. Three years later, the macular pseudo-cyst formation transformed into an atrophic scar, while the patient's visual acuity was the same. In conclusion, as a result of high-voltage electrical injury, serious ocular complications may develop. It is sufficient to refer those patients to an ophthalmologist for appropriate treatment and follow-up. (Turk J Ophthalmol 2014; 44: 410-2)
Key Words: Electrical injury, optic neuropathy, macular hole, foveal pseudo-cyst

Özet
Otuz beş yaşında erkek hastanın; yüksek voltajlı elektrik yaralanması sonrası sağ gözde görmede azalma şikayeti gelişti. Görme keskinliği sağda 20/400 olan hastanın sağ gözünde korneal ödem, Descemet membran kırışıklıkları, katarakt, maküler delik ve optik nöropati saptandı. İntravenöz pulse steroid tedavisine başlandı. Tedavinin üçüncü gününde sağ gözde görme keskinliği 20/200 oldu, korneal ödem geriledi. İki ay sonra, hastanın görme keskinliği aynı iken, sağ gözde optik disk solukluğu, epiretinal gliozis, tam kat olmayan maküler delik, psödokist oluşumu saptandi. Üç yıllık takip sonunda, görme keskinliği sağda 20/200 iken maküler psödokist oluşumunun atrofik skara dönüştüğü izlendi. Sonuç olarak, olgumuzda olduğu gibi, yüksek voltajlı elektrik yaralanması sonrası ciddi oküler komplikasyonların gelişimi mümkündür. Bu sebeple hastaların uygun tedavi ve takip için en kısa sürede bir oftalmolog tarafından değerlendirilmesi gereklidir. (Turk J Ophthalmol 2014; 44: 410-2)
Anahtar Kelimeler: Elektrik yaralanması, optik nöropati, maküler delik, foveal psödokist

Introduction

During an electrical shock, electric current flows through the body between two contact points.[1] The clinical outcomes of an electrical injury are influenced by numerous factors including voltage, current intensity, tissue resistance and sensitivity, type of the current, duration of the contact, contact area of the current, and the route of current that travelled within the body.[1,2]

More than half of all lightning victims suffer from some form of ophthalmic injury, most commonly involving the cornea. The symptoms and findings of anterior segment injuries include blepharospasm, edema and necrosis of eyelids, thermal keratopathy, uveitis, iridocyclitis, superficial or deep corneal opacities, corneal scars, chemosis, midriasis, hyphema, anterior and posterior subcapsular cataract, and lens dislocation.[3] Posterior segment injuries include vitreous hemorrhage, retinal edema, retinal hemorrhage, retinal detachment, cystoid macular edema, chorioretinal rupture, lightning maculopathy, macular hole, central retinal vein occlusion, and central retinal artery occlusion.[3,4] Neurological injuries include thermal papillitis, optic neuropathy, loss of pupillary reflex, anisocoria, Horner's syndrome, multiple cranial nerve palsies, and nystagmus.[3,4]

The purpose of this paper is to report a case of high-voltage electrical injury with central corneal edema, posterior subcapsular cataract, macular pseudohole/cyst/atrophy, and optic neuropathy in the right eye.

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Case Report

A 35-year-old male patient, who was exposed to high-voltage electricity during work, had an injury as a result of an electrical shock that started from his right ear and passed to his right arm and emitted from his right lower extremities. He had severe burn wounds, therefore detailed eye examination with biomicroscopy could not be performed at that time. He was treated in the burn unit of another center until he was referred to our clinic for decreased vision in his right eye, nearly two weeks after the injury. In the first examination, which was performed two weeks after the injury, the patient's best corrected visual acuity (BCVA) was 20/400 in the right eye and 20/20 in the left eye according to Snellen card. Intraocular pressure was 17 mmHg in the right eye and 16 mmHg in the left eye. The patient had right peripheral facial paralysis. In the biomicroscopic examination, corneal punctate epithelial defects, central corneal edema, Descemet's membrane folds, and subconjunctival hemorrhage were detected in the right eye. In the fundus examination of the right eye, mild vitreous blurring due to corneal edema and probable mild vitreous reaction and a hypopigmented suspicious area at the temporal part of the optic disc were noted (Figure 1a). Anterior segment and fundus examination revealed no pathological findings in the left eye. The light reflex was poorly detected in the right eye due to severe corneal edema. At that time, only topical medication was administered to the patient for the corneal edema. Two days after the intense topical steroid (prednisolone sodium phosphate, 1%, eight times a day) and antibiotic (ofloxacin, five times a day) treatment, the corneal edema resolved, but visual acuity was still 20/400 in the right eye. Mild posterior subcapsular cataract, indistinct and blurry borders of the right optic disc, and the appearance of macular hole were marked (Figure 1b). A hypopigmented elevated area located temporally to the right optic disc and small dot-shaped hemorrhages compatible with possible electrical injury were also detected.

The patient, who had no color vision and had right relative afferent pupillary defect (RAPD), was treated with intravenous pulse steroid therapy (intravenous metil-prednisolone, 1000 mg/day, for three days). On the third day of the steroid treatment, the visual acuity in the right eye was 20/200 and oral steroid therapy (oral metil-prednisolone, 1 mg/kg/day) was administered. The dosage of the oral steroid was tapered by 5 mg in every three days and steroid therapy was continued for two months. Non-full-thickness macular hole, pseudocyst formation was detected on optical coherence tomography (OCT) (Figure 2a). A foveal cyst was evident as hyporeflective lucency, visible just anterior to the retinal pigment epithelium (RPE)/choriocapillaris complex in the patient's right eye. The remaining foveal internal layers of the retina and posterior hyaloid layer bridged over the cyst. BCVA was found to be 20/200 in the right eye, the right optic disc was pale, and epiretinal gliosis with cystic changes was present (Figure 2b). In the mid-phase of fundus fluorescein angiography (FFA), hyperfluorescent areas were detected at the fovea and at the temporal part of the optic disc (Figure 2c). At the last visit after three years of follow-up, an atrophic macular scar was detected on OCT (Figure 2d). Optic atrophy and

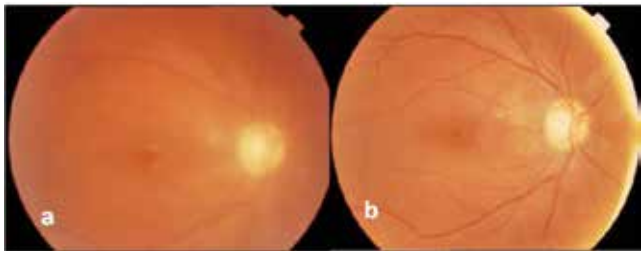

Figure 1. a) The colored fundus photograph of the right eye shows mild vitreous blurring and a hypopigmented spot on the fovea; **b)** Two days after treatment, colored fundus photograph of the right eye showing no vitreous blurring and a hypopigmented spot that simulated macular hole on the fovea and a hypopigmented area at the temporal part of the optic disc

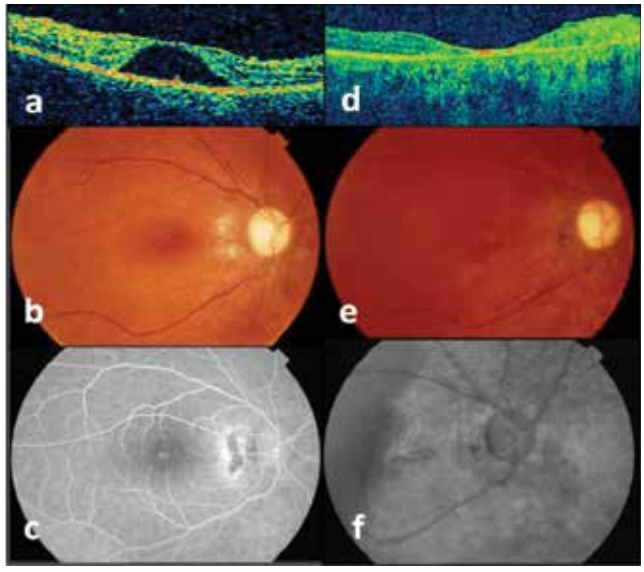

Figure 2. a-f) OCT (a), fundus photograph (b) and FFA (c) of the right eye, two months after the injury, showing retinal cyst formation with retinal thinning on OCT, epiretinal membrane formation and wrinkling, hypopigmented spots on the fovea, and hypopigmented area on the temporal part of the optic disc on colored fundus photograph as well as peripapillary window defects involving the fovea on FFA. OCT (d), fundus photograph (e), and FFA (f) of the right eye, three years after the injury, showing retinal atrophy on OCT, wide atrophic changes at posterior macula and peripapillary area on colored fundus photograph, and wide peripapillary window defects involving the fovea and RPE stippling at the peripheral area on FFA

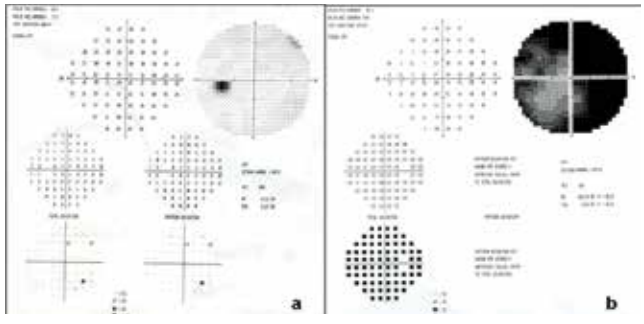

Figure 3. Visual field testing of the (a) left eye is normal and of the (b) right eye demonstrates generalized peripheral constriction

window defects in wide peripapillary area due to the atrophic changes and RPE stippling at peripheral areas were detected on colored fundus photo and FFA images of the right eye (Figure 2 e-f). In the visual field test, generalized peripheral constriction was detected, in the right eye while the infero-nasal area was not affected and the left eye was normal (Figure 3a, 3b). In the late period, no improvement was detected in the visual field test. BCVA in the right eye was 20/200 and right RAPD persisted.

Discussion

The macula is very sensitive to thermal damage because of the high content of melanin granules of RPE, which constitute the main obstacle to the current flow.[4,5] Melanin acts as a resistance to electric current, which may produce thermal denaturation of the outer retina and RPE. Electrical current could damage RPE by electrolysis. Lightning can also emit a shockwave, which can mechanically injure the RPE. Localized inflammation in response to lightning injury could contribute to RPE dysfunction. Intra-retinal edema could result from decreased transport of fluid out of retina or development of retinal vascular incompetence.[5]

Macular edema that is seen early after lightning strike may be replaced by lesions described as a cyst, macular hole, or solar maculopathy.[4,5] Lee at al.[4] reported that initial macular holes due to lightning injury may undergo spontaneous closure subsequently. It is important to differentiate between macular cystic changes and full-thickness macular holes due to lightning or electrical injury, because maculopathy with cystic changes may spontaneously resolve, but for a full-thickness macular hole, surgical intervention is sometimes recommended.[6-9] The prognosis of early macular pseudocyst or hole formation can be different; it can progress to atrophic scar formation or regress spontaneously.[4,6] OCT is an important diagnostic tool for the differentiation and detection of macular damage.

Our case was initially considered as a full-thickness macular hole until the OCT images revealed that there was a non-full-thickness macular hole with macular pseudo cyst formation, which progressed to atrophic macular scar during the three-year follow-up.

In one study,[10] the histopathological alterations in one eye enucleated ten days after electrical injury were detected and it was found that the choroid was edematous, hemorrhagic and infiltrated by acute inflammatory cells. Retinal pigment epithelium showed focal areas of edema and atrophy, while the retinal vessels were dilated, congested and incompetent with inflammatory exudates in subretinal space. In the literature, there are a few cases about optic neuropathy due to electrical injury.[9] In our case, when the patient was referred to us almost two weeks after the electrical injury, the mild pallor appearance of the right optic disk and decreased direct light reflex were marked. The patient was diagnosed as traumatic optic neuropathy and intravenous pulse steroid therapy was administered for three days. The neuronal damage in such injuries is caused by prolonged depolarization, direct neuronal damage, secondary tissue damage from edema, ischemia, and reperfusion injury.[3] The high-dose steroid therapy has several effects on the key steps of the neuronal injury cascade such as reducing lipid peroxidation, decreasing prostaglandin synthesis, and increasing lipocortin production.[3] The actual effectiveness of steroid treatment cannot be proven as it is not ethical to obtain control groups of patients who do not receive any treatment.[11] There is not enough evidence on the issue of the corticosteroid treatment modalities for traumatic optic neuropathy cases. However, the dose schedule of the National Acute Spinal Cord Injury Study Protocol (NASCIS-2), which consists of initial intravenous bolus, methyl prednisolone 30 mg/kg administered over 15 minutes, followed by a 45-minute pause before instituting the maintenance infusion of methyl prednisolone 5.4 mg/kg per hour for 23 hours, can be followed.[12] It is not clear when the visual damage becomes irreversible after such injuries, however, studies support the idea that response to the medical treatment is better in early stages.[11] In our case, the visual acuity improved to 20/200 from 20/400. However, full recovery of visual acuity could not be achieved probably because of the damage that occurred in the macula and the optic nerve and also the delay in steroid therapy for two weeks after the injury. We think that if the patient had received proper intravenous steroid therapy earlier, visual recovery might have been more satisfactory.

In conclusion, ocular damage due to electrical injury appears in a wide spectrum and requires urgent and appropriate treatment approach. It is very important to evaluate the ophthalmic damages as soon as possible and to implement early treatment modalities on time for better visual outcomes. Steroid therapy might be helpful, and longtime follow-up of these cases is mandatory to understand the effect of electrical injury to the ocular tissues better.

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