Ocular Injuries Caused by Lightning Strikes: Review of the Literature and Presentation of Two Clinical Cases

Abstract: Secondary injuries caused by lightning strikes are not frequent; however, survivors have important sequel in organs and tissues. We describe two cases. The first one with lightning-induced maculopathy and the other case involving lens damage. This paper discusses factors that determine the extent of injuries and review the management for each one. The study and data collection complied with local legislation and with the principles of the Declaration of Helsinki.

Key words: lightning strike; eye injury; maculopathy; cataract; electric shock.

INTRODUCTION

Electrocution by lightning or electrofulguration is the most frequent cause of injuries by natural phenomena and it is associated with high mortality (20-30%) and morbidity (75%) with permanent sequel among survivors.1 Asystolic cardiac arrest4 or ventricular fibrillations are the most frequent mechanisms of death5 and these are caused by different degrees of cardiac depolarization.

The injuries caused by electricity are classified as low voltage (less than 1000 volts), high voltage (more than 1000 volts) or lightning (3 to 200 million volts).7 Several factors determine the magnitude of the injuries, these include: voltage, current (amperage), type of current (alternate or direct), current pathway across the body, duration of contact with the current source, and individual susceptibility, among others.8,9

Although ocular involvement is infrequent for victims of low-voltage electrical burns, in victims of electrocution by lightning, it can reach up to 50%.10 The most affected ocular tissues are generally the cornea (where epithelial erosions predominate11 and the lens where the percentage of formation of anterior and posterior subcapsular cataracts can reach 6-7%.12 A minor percentage of victims can present with uveitis, iridocyclitis, intraocular hemorrhage or thrombosis, macular cysts, thermal papillitis, and chorioretinitis. Glaucoma is more frequent in electrocution survivors compared to the general population.

The army as a profession is ranked in first place for being at high risk of electrical accidents caused by lightning.13 These young people have high morbidity and mortality and suffer from important sequel.

We report on two cases of ocular injuries caused by lightning among Colombian soldiers. Anatomical involvement is seen at different levels: the first one initially presented with unilateral uveitis, which was then associated with a cataract and subfoveal cyst; the second case later developed a subcapsular bilateral cataract. We describe the clinical presentation, tomography, management, and include a review about the pathophysiologic mechanisms that generated the ocular injury in each case.

Figure 1 Subcapsular Cataract
CASE REPORT 1

A 20-year-old soldier came to the ophthalmology service after being struck by lightning in the left frontal region of the head. He reported intense ocular pain and moderate visual loss in the left eye. On examination his best-corrected visual acuity was 20/20 RX + 0.50-0.25x 160 in the right eye and 20/400, RX +0.75-0.50x 40 in the left. Ocular bilateral movements were preserved. The eyelids, adnexa, anterior segment, and tonometry of the right eye were all normal. The left eye had a clear cornea with cellularity ++ in the anterior chamber.

He was treated with topical corticosteroids and cycloplegic agents for several days and he experienced clinical improvement. A week later, he presented again with iritis associated with important lens opacification (Figure 1) and macular pigmentary changes in the left eye upon ophthalmoscopic examination. He was treated for two more weeks with topical corticosteroids and cycloplegic agents that controlled the iritis, but a new control demonstrated a full-thickness macular hole with retinal traction. Optical coherence tomography confirmed the detachment of the pigmentary epithelium, a subfoveal cyst (Figure 2), and thickness of the internal retinal membrane (Figure 3) and the hyaloid. The patient required a surgical approach with phacoemulsification with intraocular lens implantation, vitrectomy, laser, and gas achieving the complete closing of the hole and retinal attachment. One week postoperative his best-corrected visual acuity was counting fingers at a distance of 50 cm from the left eye. After five months, his best-corrected visual acuity was 20/50 and the retina was attached.

CASE REPORT 2

An asymptomatic 26-year-old soldier is evaluated after being struck by lightning. The ophthalmological examination is completely normal with a visual acuity of 20/20 AO. Thirteen months later, he consults for progressive bilateral visual loss. His best-corrected visual acuity is 20/60 and there is lens opacification (Figure 4) in both eyes. Ocular intrinsic and extrinsic movements are preserved and there are no changes in pupil reflexes, intraocular pressure, or retinal mapping. Interferometry reached 20/30. Phacoemulsification with intraocular lens implantation was consecutively done in both eyes with improvement in his visual acuity to 20/30 one week postoperative. Two months later, his visual acuity was 20/20 in both eyes.

DISCUSSION

The pathological effects of electrical power are seen in several active mechanisms. The current destroys cells, damages the integrity of the cellular membranes and alters their potential; the consequences are edema and irreversible cellular damage. This process is called electroporation.

The electrical current can initiate its pathway across the orbit; in this case, it produces alterations in the annexes like the loss of eyebrows and eyelashes. Painless deep burns form unaesthetic scars with or without lagophthalmos and even ankyloblepharon in the eyelids. The metallization of the skin is typical at the point of contact with the current source.

The pathway of electrical current across the body produces changes and reactions in tissues and organs that exponentially increase in cases of electrofulguration. These reactions affect the organism’s structural composition on several levels (anatomical, histological, and biochemical) and make for a series of electric traumas.

The changes that are observed in the conjunctiva can be hyperemia, with some degree of ciliary injection, and/or chemosis. Eventually hyposphagma and hematic impregnation of the cornea have been described, especially when the electric shock is intense.

Different injuries can occur in the cornea: from dotted epitheliopathy, to punctate erosions, localized or diffused subepithelial opacities. Sometimes widespread
transitory edema appears, but a localized edema that topographically corresponds with lens opacity is the most typical sign.

According to Waardenburg et al,15 injuries begin appearing 24 hours after the trauma, with disturbances occurring in the deepest layers of the stroma. After a week, the whole cornea is cloudy, slowly becoming transparent nearly six weeks later. If an electrical burn formed, the epithelium necrotized, and was associated with the loss of the corneous sensation and suffered permanent scarring. In the worst cases, a purulent infiltration of the whole cornea appeared, with extensive necrosis of the tissues, which culminated in perforation and phthisis.16

In the iris and ciliary body, inflammatory changes occurs frequently. The iritis is moderate and transient, generally beginning between 1 and 8 weeks after the trauma.17 Although it is infrequent in severe traumas, an intense iridocyclitis28 can cause the formation of hypopyon and synchiae.29 It is interesting that in all cases, including ours, uveitis has been associated with cataracts—suggesting that lens capsule disruption leads to the release of angiogenic substances.30

The appearance of hyphaema is exceptional. Iris atrophy, either focal or generalized, can be associated with the pigment of the anterior lens capsule, superficial stroma of the iris, endothelium, and trabecular meshwork, being able to cause a secondary glaucoma.25

The early changes that can be recognized in the lens consist of multiple vacuoles directly behind the anterior capsule.26 These vacuoles are located in half of the periphery and for this reason, they can go unnoticed if the patient is not dilated. These changes do not produce an immediate reduction of visual acuity. A few days or weeks later, these vacuoles disappear and are replaced by thin, linear irregular opacities in the anterior subcapsular region. In some cases, especially in young patients, all of these lens opacities disappear, returning to transparency.21

Cataracts are not generally diagnosed immediately (usually, within two to six months after the trauma); however, cataracts have been identified immediately up until eleven years after the trauma. If they are bilateral, the nearest eye to the point of contact is the most affected. In general, industrial accidents affect the anterior cortical of the lens, whereas the injuries caused by light imply the involvement of the anterior and posterior subcapsular areas.27

The mechanisms of these lens changes are not clear. Ferreiro et al28 postulated that the voltage does not influence the severity of the cataract as much as the point of contact with the electrical burn. An existing hypothesis states that the current coagulates the proteins of the lens’ cells. Galezowski and Knies29 suggest that the contraction force of the ciliary muscle mechanically displaces the fibers of the lens, in the same way that occurs in contusions. Other proposed mechanisms include changes in the permeability of the lens capsule, vascular involvement of the previous eye segment, and direct injury by heat or absorption of radiant energy.

Lightning retinal injuries are rare; the most frequent are macular edema and dotted pigmented degeneration, which generally persist as pigmented atrophy. The macula is particularly sensitive to the thermal damage because of its high concentration of melanocytic granules that lead to major dissipation of the heat when it is injured by the electrical current. The elevated temperature found in the retina’s pigmented epithelium and choroidal ischemia caused by focal vascular changes can explain macular injuries. Edema can be a temporary reaction or it may be followed by the formation of cysts or holes.30 There are few reports in the literature of these macular cysts and holes like those found in our patient.28,29

Other retinal changes described are arterial constriction, intra and sub retinal hemorrhages secondary to venous thromboses or arterial occlusions for edema, and clots in the intima layer.31 In the periphery, we can find patches of chorioretinal atrophy that resemble traumatic choriorretinitis,32 a serious injury such as retinal detachment.33

Nervous injuries can be thermal papillitis, unilateral or bilateral neuropathy, optic atrophy, and paralysis of extraocular muscles, ptosis, nystagmus, facial paralysis and Horner’s syndrome, among others.34 These injuries lead to functional alterations such as photophobia and blepharospasm. Visual loss ranges from transitory blindness to a permanent decrease of all or part of the field of vision. Ring scotomas and concentric field reductions have also been described.

The injury of the nervous tissues takes place in several mechanisms. The nervous tissues can present a decrease in the conductivity or necrosis for coagulation similar to the one observed in the muscle. In addition, it can suffer a consequential damage in the patient’s vascular supply or myelinic injury. The signs of neuronal injury can appear immediately or delay several hours or even days to appear.35

**References**

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