Lightning Induced Ocular Complications: A Case Report

Chandana Chakraborti

Lightning injuries range from various forms of transient focal damage to instant death. Eye injuries from lightning are not uncommon. We report a case of lightning injury in a 30 - year old female who presented to us one month after the injury. On examination her best corrected visual acuity (BCVA) was 6/24 in right eye (RE) and 6/60 in left eye (LE). Slit lamp biomicroscopy revealed anisocoria, bilateral uveitis and sphincter tear in left eye. There was lightning induced cataract in both eye and macular hole in left eye. Optical Coherence Tomography (OCT) revealed macular cyst in right eye (RE) and a full thickness macular hole (FTMH) in left eye. Patient was treated with topical corticosteroid and cycloplegics in both the eyes. At subsequent follow up cataract surgery was done in both eyes (BE). Post-operative best corrected visual acuity at 1 month was 6/9 and 6/12 in right and left eye. There was a spontaneous resolution of the macular lesion in both eyes.

Lightning injury can cause multi system damage and often results in high mortality. The consequences of being struck by lightning depend on several factors at the time of the incident. Important features are the type of stroke, the current and its distribution, and the victim’s position and clothing1. Ocular injuries like thermal keratopathy, uveitis, hyphaema, anterior and posterior subcapsular cataract, lens dislocation have been reported2. Posterior segment complications like vitreous hemorrhage, retinal edema and haemorrhage, retinal detachment, vascular occlusion, choroidal rupture, macular involvement in the form of cystoid macular edema and macular hole have been documented. Lightning induced nystagmus, anisocoria, optic neuropathy, Horner's syndrome and multiple cranial nerve palsies has been reported2-4.

We report a rare case of ocular injury due to lightning strike involving both anterior and posterior segments with good final visual outcome.

CASE REPORT

A 30 - years old female presented with painless diminution of vision in BE following a lightning strike one month back. She was struck by lightning when she along with her two family members were sitting on the veranda of their house while it was raining outside. The neighbour who accompanied her gave the rest of the history. She was unaware of the incident. All three became unconscious following a lightning strike on a coconut tree situated near the house. The coconut tree was burnt out. All three were admitted in hospital in unconscious state and treated conservatively. Among the three one sustained skin burns around her necklace, her husband sustained a fracture of right humerus, may be due to fall on ground. But these two did not develop any ocular complaints. After seven days of the incident she complained of blurring of vision and redness in both eyes. She was examined by an ophthalmologist and diagnosed as bilateral uveitis and was treated with topical corticosteroid and cycloplegics. Systemic examination like pulse, BP and renal function were normal. There was no skin or eyelid burns. She developed loss of hearing in the left ear due to rupture of tympanic membrane.

On examination, her BCVA was 6/24 and 6/60 and intraocular pressure was 16 and 18 mm Hg respectively in RE and LE. Slit lamp examination
revealed bilateral resolving iridocyclitis, anisocoria (LE > RE) and sphincter tear at 5-o’clock position and anterior and posterior subcapsular cataract in both eyes (LE > RE) (Fig. 1). Slit lamp biomicroscopy with a 90 D revealed pigmentation of macula in RE (Fig. 2) and macular hole in LE (Fig. 3). The Watzke - Allen sign was positive in LE. OCT revealed macular cyst in RE (Fig. 4) and a full thickness macular hole with a thin bridging tissue over it in LE (Fig. 5). Non steroidal anti-inflammatory drops were prescribed in both eyes for the uveitis.

The patient reviewed with us 3 months later, when she had developed a total cataract in her LE and advanced immature cataract in RE. Small incision cataract surgery was done in BE. Post-operative BCVA was 6/9 in RE and 6/12 in LE. Pigmentary changes
were found in macular area of BE. The Watzke – Allen sign was negative in LE. We assume that there was a spontaneous closure of the macular hole in LE but the OCT could not be done because of patient’s unwillingness.

**DISCUSSION**

Lightning is an uncommon cause of ocular injury. The power of lightning is 10,000 to 2,00,000 amperes and a voltage of 20 million to 1 billion volts. Lightning exposure time is only 1 to 100 milliseconds, taking less time to cause damage of tissue. Tissue destruction is caused by both thermal effect and electrolysis. Non-nervous tissue offering high resistance is responsible for the thermal effects of lightning resulting in rapid coagulation of the cellular proteins. Lightning induced cataract is mostly bilateral. Possibility of cataract formation increases with the proximity of the contact area to the eye.

Lightning may reach its victims by any of the four routes and causes injuries:

1. Direct strike: when the major current flows directly through the victim and is facilitated by metal objects.
2. Splash: where lightning strikes an object first and then arcs through the path of least resistance.
3. Contact: the bolt strikes an object the victim is in contact with i.e. electrocution while telephonic conversation.
4. Ground current: here the lightning travels along the surface towards the victim after striking the ground.

Our patient probably sustained the injury by the second mechanism mentioned; lightning passed through the nearby coconut tree and through the ground. Current passed initially through the left side of the body as indicated by left sided hearing loss, poorer vision in LE (6/60) than RE (6/24) and denser posterior sub-capsular cataract in LE. Severity of uveitis and macular damage was also more in the LE.

In case of bilateral cataract, it starts in the eye on the more affected side first and there may be a gap of 1 – 10 months between the two eyes. Lightning induced cataract may be attributed to, decreased permeability of lens capsule, protein coagulation by electrical current, nutritional impairment of lens due to iritis and mechanical damage to the lens fibers. Lightning induced cataract is morphologically characteristic, affecting both anterior and posterior parts of the lens. Regression of the opacity as a whole or partially has been reported. In our case there was both anterior and posterior-sub-capsular cataract in BE (LE > RE).

The macula is very sensitive to thermal damage because of the high melanin content of the retinal pigment epithelium (RPE). Electrical current damages the RPE by electrolysis. Melanin resists the electric current leading to thermal denaturation of the outer retina and RPE. Lightning strike may cause localized inflammation of the RPE. Retinal edema may result from decreased transport of fluid out of retina or development of retinal vascular incompetence. Early development of macular edema seen after lightning strike may lead to formation of macular cyst, macular hole or solar maculopathy. Lightning maculopathy is supported by the lack of posterior vitreous detachment and operculum.

We advised our patient macular hole surgery when she presented to us after the injury but the patient denied. Subsequently after cataract surgery patient had good visual outcome in both eyes. We presume that the hole had resolved on its own which was clinically proven by negative Watzke – Allen sign (LE). Lightning induced macular holes may undergo spontaneous closure with a good visual outcome which was a feature in our case though there is no OCT documentation.

It is important to differentiate between lightning induced macular cyst and full-thickness macular hole, as cystic changes may resolve spontaneously but for full – thickness macular whole surgery may be required.

To the best of our knowledge, this is the first case reporting bilateral lightning induced anterior and posterior segment ocular damage with a good final visual outcome in both the eyes.

**Author’s Affiliation**

Dr. Chandana Chakraborti  
Assistant Professor  
Dept. of Ophthalmology  
Calcutta National Medical College and Hospital  
Kolkata 700006  
West Bengal  
India

**REFERENCES**