Review Article

Taiwan J Ophthalmol 2022;12: 130-137

Access this article online

Quick Response Code:

Website:
www.e-tjo.org

DOI:
10.4103/tjo.tjo_27_20

Lightning injuries of the posterior segment of the eye

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Abstract:

Lightning causes serious injuries and deaths worldwide every year. Ophthalmic injuries due to lightning are due to direct or indirect transmission of electric current, resistance-induced heat, and heat-induced shock wave. PubMed search of articles related to posterior segment injuries caused by lightning using keywords (lightning injury, ophthalmic manifestations, ocular injuries, and posterior segment) was conducted, and 19 case reports in 17 articles including 29 eyes with lightning injury to the posterior segment of the eye from 1984 to 2019 were reviewed. The majority of case reports (*n* = 10, 53%) were from North America. Eleven patients (58%) were in the age group of 10–30 years. Most patients (*n* = 10, 53%) had bilateral injury. The macula was the most common site of involvement with retinal pigment epithelial changes (*n* = 14, 48%) being the most common manifestation. A variety of other retinal, vitreous, and electrophysiological abnormalities have also been reported. We conclude that although lightning injuries are usually mild injuries, with vision remaining, either stable or showing some improvement in the majority of cases, severe visual loss due to optic atrophy and maculopathy may occur in long term.

Keywords:

Cystoid macular edema, lightning injuries of eye, lightning maculopathy, macular hole

Introduction

Worldwide, there are approximately 50,000 thunderstorms every day, which produce about 8 million lightning strikes daily.[1,2] The magnitude of discharge in lightning has been estimated at many millions to 1 billion Volts, the current at 12,000–200,000 Amps, and the current's duration at 1/1000th to 1/100th of a second. The contact temperature may rise up to approximately 8000°C–300,000°C.[1,3] More than half of all lightning victims suffer from some form of lightning injury.[4] Ophthalmic injuries due to lightning occur mainly from direct or indirect transmission of electric charge, resistance-induced heat, or heat-induced shock wave.[5] Intraocular injury secondary to lightning strike has been appreciated since 1772 when Saint Yves reported the first lightning-induced cataract.[6,7] It has been earlier reported that the most common affected ocular structures after lightning injury are generally the cornea, where epithelial erosions being the most common injury,[8] and the lens, where the percentage of formation of anterior and posterior subcapsular cataracts can reach 6%–7%;[9] however, in the past decade, with the advent of optical coherence tomography (OCT), more number of posterior segment pathologies due to lightning injury were picked up. In the present article, PubMed search of articles related to posterior segment injuries caused by lightning using keywords (lightning injury, ophthalmic manifestations, ocular injuries, posterior segment, and retinopathy) was conducted and 19 case reports in 17 articles including 29 eyes with lightning injury to the posterior segment of the eye from 1984 to 2019 were reviewed.

Discussion

When a person is struck by lightning, he/she becomes highly charged. If he/she

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Submission: 28-01-2020
Accepted: 19-05-2020
Published: 25-05-2021

How to cite this article: Rishi E, Indu VP, Sharma U. Lightning injuries of the posterior segment of the eye. Taiwan J Ophthalmol 2022;12:130-7.

is grounded, the current passes through his body to the ground. The current may produce violent muscular contractions that can propel the victim to a surprising distance. Thus, there may be a mechanical injury in addition to the electrical injury of a lightning strike.

Mechanism of lightning injury

There are five types of lightning strike.[1,2,10-13] (a) Direct strike that occurs when the victim is struck directly by the lightning discharge. Most fatalities and severe injuries occur after this type of lightning strike. (b) Side flash that occurs when a nearby object such as a tree is struck and the current then traverses through the air to strike the victim. (c) Contact strike that occurs when lightning strikes an object the victim is holding and the current is transferred from the object to the person to the ground. Common contributors to lightning contact are a golf club, an umbrella, or a set of keys in the person's hand. (d) Ground current that occurs when lightning hits the ground and the current is transferred through the ground to a nearby victim. (e) A weak upward streamer that does not become connected to the completed lightning channel.[14] Significant blunt injury can be sustained from the shock waves associated with the expansive and implosive forces generated by surrounding air as the lightning bolt passes. Victims are often thrown against objects, resulting in deceleration trauma.

There are several proposed mechanisms for ophthalmic injuries due to lightning.[10] First, the electrical current passes through ocular tissues, causing disruption of cell membranes. Second, the heat that is converted from the electrical current can cause damage. Finally, tissue ischemia can develop secondary to vasoconstriction, and damage can also occur secondary to localized inflammation or reperfusion injury.[15]

The pigments of the iris, retinal pigment epithelium, and choroid act as resistors to electrical conduction.[15,16] Intracellular melanin gives retinal pigment epithelium the highest electrical resistance in the eye, and it will generate the most heat when conducting electrical current, reaching a temperature that is 10°C–20°C above ambient temperature in experimental models. Since macular retinal pigment epithelium contains more melanin than nonmacular retinal pigment epithelium and the retina overlying the foveola is relatively thin, thermal damage at the foveal zone may be enhanced. Combined, these observations could account for both generalized retinal pigment epithelial damage and the predilection for more severe central macular damage in lightning-induced ophthalmic injuries.[7]

Most reports of lightning-induced maculopathy quote direct or indirect electric transmission as the cause of injury. However, a high-voltage electric current can also induce photic retinopathy without contribution from the electric charge.[5]

Clinical manifestations of lightning injury

Lightning causes damage to a wide range of body systems including cardiopulmonary, neurological, vascular, cutaneous, ophthalmic, and otological injuries.[2,13,17-21] Cardiac injuries such as cardiac arrest or arrhythmia and neurologic damage are responsible for most of the morbidities and mortalities of lightning.[1,3,11,12,17,22,23]

Lightning-induced ocular injury comes in many forms.[1,9] Anterior segment injuries[1,24] include adnexal burns, thermal keratopathy, uveitis,[17,25] hyphema, anterior and posterior subcapsular cataract,[1,11,16,26,27] dislocated lens, raised intraocular pressure,[25] and dilated unresponsive pupils.[17,28] Neuro-ophthalmologic injuries include thermal papillitis, optic neuropathy, multiple cranial nerve palsies, nystagmus, dilated and unresponsive pupil, ptosis and loss of accommodation, and optic atrophy.[1,29-31]

Posterior segment manifestations of lightning injuries

Of the 19 case reports in 17 articles from 1984 to 2019 [Table 1], the majority (53%, *n* = 10) of the articles were from North America, followed by 26% (*n* = 5) from Europe and 21% (*n* = 4) from Asia.[4,7,10,15,16,31-42]

Most of the patients (*n* = 11, 58%) were in the age group of 10–30 years. Males outnumbered females, with a male-to-female ratio of 10:7. Of the 19 cases, 9 (47%) had unilateral and 10 (53%) had bilateral lesions. In unilateral cases, the left eye was twice (*n* = 6, 67%) more commonly involved than the right eye (*n* = 3, 33%). The time of presentation ranged from 1 day to 10 months (mean: 39 days, standard deviation [SD]: 68 days), although Harris *et al*.[41] have reported a case of severe choroidal atrophy four decades after lightning injury. Presenting visual acuity varied widely from counting finger to 0.18 logMAR (20/30). The mean presenting visual acuity was +0.7 logMAR (SD +0.8, range: 0–3). Harris *et al*.[42] have reported a case of optic atrophy presenting with nil perception of light four decades after lightning injury.

The macula was the most common site affected by lightning [Table 2]. Retinal pigment epithelial (RPE) changes at the posterior pole were the most commonly reported lesion (*n* = 14, 48%) followed by macular hole (MH) (*n* = 11, 38%) and cystic changes at the macula (*n* = 6, 21%). Peripheral retinal pigmentary alterations were reported in 3 (10%) eyes.

The early changes noted at the macula following lightning injury included cystoid macular edema (CME), absent foveal reflex, and serous macular detachment.

Table 1: Summary of articles

Author/years	Eye affected	Age	Gender	Time of presentation	Visual acuity at presentation	Findings at presentation	Follow-up and treatment
Campo and Lewis, 1984[32]	OD	15	Female	2 weeks	0.46 logMAR (20/50)	Cyst at macula	MH with surrounding cystic changes at macula. Foveal RPE WD on FFA
Handa and Jaffe 1994[33]	OD OS	25	Male	5 weeks	1.0 logMAR (20/200) 0 logMAR (20/20)	OD - cysts OS - solar-like maculopathy	OS - MH at 5 months OU - 14 months- Macular RPE changes, resolution of hole and cysts OU - cataract surgery
Lagrèze *et al*., 1995[7]	OD OS	13	Male	4 days	0.18 logMAR (20/30) 0.48 logMAR (20/60)	OU- missing FR, RPE defects at macula, retinal folds	OU - central yellow-white lesion with orange discoloration at RPE WD on FFA, EOG - reduced amplitude , ERG - normal OU
Augustin *et al*., 1995[34]	OD OS	43	Male	6 weeks	0.3 logMAR (20/40) 0.7 logMAR (20/100)	Target-like alterations at fovea. FFA-WD OD-decreased scotopic ERG, EOG-1.6	8 months; OD-scotopic ERG -reduced amplitude; photopic ERG-WNL; OU-decreased dark adaptation
Augustin *et al*., 1995[34]	OS	60	Male	2 months	0.3 logMAR (20/40)	Target-like alterations at fovea FFA - parafoveal WD	8 months, decreased night vision
Espaillat *et al*., 1999[16]	OD OS	30	Male	1 week	0.7 logMAR (20/100) 1.0 logMAR (20/200)	OD- PVD, MH OS- PVD, MH, rhegmatogenous RD	OS- underwent vitrectomy + FGE + sclera buckling Recurrence of RD at 1 month
Yi *et al*., 2001[31]	OD OS	32	Male	10 days	HM 1.0 logMAR (20/200)	OD-peripapillary retinal edema ERG OD-extinguished scotopic blue flicker, reduced scotopic white 30-Hz flicker OS-reduced photopic and scotopic ERG PVER-reduced amplitude in OS	50 mg prednisolone/ day for 4 days and multivitamins After 13 days OD-20/200, OS-20/100
Lee *et al*., 2002[10]	OS	14	Male	2-3 weeks	0.46 logMAR (20/50)	Serous macular detachment	MH at 2 weeks Spontaneous closure of hole at 3 months, ERM
Lee *et al*., 2002[10]	OD OS	13	Male	5 weeks	CF 0.48 logMAR (20/60)	OD - PVD, cherry red spot due to CRAO OS - mild macular edema OU - reduced flow in CRA on orbital Doppler	OD - MH at 12 weeks, underwent MH repair + IOL implantation OS- cataract surgery + IOL
Lin *et al*., 2002[35]	OS	39	Male	2 days	0.46 logMAR (20/50)	OS - missing FR, WD at fovea on FFA	3 months - macular RPE mottling, decrease in ERG amplitude, decrease in EOG amplitude and Arden ratio Cataract surgery

Contd...

Table 1: Contd...

Author/years	Eye affected	Age	Gender	Time of presentation	Visual acuity at presentation	Findings at presentation	Follow-up and treatment
Moon *et al*., 2005[36]	OD OS	20	Female	1 month	0.6 logMAR (20/80) 0.54 logMAR (20/70)	OU - cysts at macula WD on FFA	1 year - OU resolution of cysts, RPE pigmentary changes OU - cataract surgery
Rivas-Aguiño *et al*., 2006[37]	OD OS	14	Female	2 months	0.48 logMAR (20/60) 1.3 logMAR (20/400)	OU - cystic changes at fovea on OCT	Observation. Follow up OCT- NA
Rao *et al*., 2009[4]	OD OS	16	Female		0.6 logMAR (20/80) 0.18 logMAR (20/30)	OU - MH, RPE hyperpigmentation in periphery FFA - OU macular WD	NA
Armstrong *et al*., 2010[15]	OS	29	Female	3 days	0.48 logMAR (20/60)	Dull FR, OCT - double U shaped inner foveal contour with elevation of IS-OS junction	2 weeks-peripheral pigmentary changes, 3 weeks- loss of central PR, cysts, disruption of IS - OS junction, loss of RPE. 4 months - resolution of CME, loss of RPE, thinning of NSR
Stefaniotou *et al*., 2012[38]	OD	60	Male	1 day	0.48 logMAR (20/60)	OD - white fluffy lesion at posterior pole, OCT - PR thinning, RPE unevenness	5 months - OCT - small parafoveal cysts, recovery of PR layer and RPE changes
Dhillon and Gupta, 2015[39]	OD	77	Female	2 weeks	0.46 logMAR (20/50)	Macular edema	Observation and NSAIDS. MH at 2 months-advised surgical intervention.
Liu *et al*., 2016[40]	OD	Early 20s	Female	4 days	0.46 logMAR (20/50)	OCT-subtle irregularity of the outer retinal layers, most notably nasal to fovea	2 weeks-vision 20/100, OCT-intraretinal cystoid spaces, subfoveal EZ disruption; 7 weeks-decreased cystoid spaces, small subfoveal hyporeflective space and further loss of outer retinal layers in nasal macula; 4 months-subfoveal hyporeflective space in inner retina, restoration of outer retina
Rishi *et al*., 2016[41]	OS	40	Male	10 months	0.18 logMAR (20/30)	LMH on OCT, peripheral RPE alterations	Status quo after 1 year
Harris *et al*., 2019[42]	OD OS	58	Male	4 decades	0.09 logMAR(20/25) NPL	OU-disc pallor, choroidal atrophy	

CF=Counting finger, CRA=Central retinal arteries, CRAO=Central retinal artery occlusion, CME=cystoid macular edema, EOG=Electro-oculogram, EZ=Ellipsoid zone, ERG=electroretinogram, FFA=Fundus fluorescein angiography, FR=Foveal reflex, FGE=Fluid-gas exchange, IOL=Intraocular lens, LMH=Lamellar macular hole, MH=Macular hole, NA=Not available, NSR=Neurosensory retina, NPL=No, NSAID=Non-steroidal anti-inflammatory drugs, OS=Oculus sinister, OD=Oculus dextrus, OU=Oculus unitas, PL=Perception of light, PR=Photoreceptors, PVD=Posterior vitreous detachment, PVER=Pattern visual evoked response, RD=Retinal detachment, RPE=Retinal pigment epithelium, WD= Window defects, WNL=within normal limits

Table 2: Various manifestation of posterior segment injury with lightning strike in published literature

	n (%)
Posterior segment manifestation* (*n*=29)	
RPE changes	14 (48)
Macular hole	11 (38)
Cyst	7 (24)
MH + RRD	1 (3)
Outer retinal defect	2 (7)
NSD	1 (3)
CRAO	1 (3)
LMH	1 (3)
ERM	1 (3)
Choroidal scar + optic atrophy	2 (7)
Peripapillary retinal edema	1 (3)
Management given (*n*=21)	
Conservative	13 (62)
VR surgery	1 (5)
Cataract surgery	6 (29)
VR + cataract surgery	1 (5)
Outcome of macular hole (*n*=5)	
Spontaneous closure	3 (60)
Surgical closure	1 (20)
Nonclosure	1 (20)
Visual outcome (*n*=25)‡‡	
Initial visual acuity	+0.7 logMAR (SD +0.8, range 0-3)
Final visual acuity	+0.6 logMAR (SD +0.79, range 0-3)
Improvement in visual acuity	14 (56)
Stable visual acuity	4 (28)
Worsening of visual acuity	7 (16)

*Some eyes had combination of more than one finding, ‡‡One eye presented with no perception of light, four decades after lightning injury. This eye was excluded from the analysis. CRAO=Central retinal artery occlusion, ERM=Epiretinal membrane, LMH=Lamellar macular hole, MH= macular hole, NSD=Neurosensory detachment, RPE= Retinal pigment epithelial, RRD=Rhegmatogenous retinal detachment, SD=Standard deviation, VR=Vitreo-retinal

CME[15] was found to be very common following lightning injuries. Intraretinal edema could result from decreased transport of fluid out of the retina or due to development of retinal vascular incompetence.[4] CME was found to resolve spontaneously in a few cases over a variable period. Spontaneous resolution of macular edema mostly resulted in foveal thinning or residual retinal pigment epithelial changes at the macula. There were also reports of CME evolving into full-thickness MHs.[32,39]

Although the macula is most commonly affected site, lightning can also cause vitreous and peripheral retinal lesions. Posterior vitreous detachment (PVD) was the most common vitreous change that can be induced by lightning.[5,10,16] The heating of retinal surface, concussive forces on the eye, and a sudden lateral contraction of attached vitreous can result in PVD and retinal breaks. Espaillat *et al*.,[16] described a patient who developed PVD with unilateral inferior retinal break and retinal detachment needing surgical repair. Peripheral retinal pigment epithelial mottling and pigmentation have also been described with lightning injuries.[4,41]

We also have had experience with lightning injuries at our institution. A 40-year-old male who was struck by lightning presented to us 10 months later with complaints of metamorphopsia in the left eye. The best corrected visual acuity was 0.00 LogMAR in the right eye and 0.18 logMAR in the left eye along with a lamellar hole at the macula and a group of pigment clumps arranged in a wedge-shaped pattern in the temporal periphery of the retina.[41]

Vascular occlusions after lightning injuries, though uncommon, have been documented. After nerves, blood vessels are the next tissue most susceptible to electrical injury.[1,10,39] Central retinal artery occlusion (CRAO) can occur in lightning injuries from either thrombosis or severe vasoconstriction, damaging the central retinal arteries (CRAs). Lee *et al*.[10] described a patient who developed bilateral incomplete CRAO following lightning strike. Orbital Doppler showed reduced flow in both CRA even after 2 years of follow-up, though his visual acuity had improved.

Norman *et al*.[1] have also described vitreous hemorrhage, chorioretinal rupture, and central retinal vein occlusion in lightning strike injuries. Harris *et al*.[42] have reported a case of severe choroidal atrophy and optic atrophy four decades after lightning injury. Yi *et al*.[31] reported a case of peripapillary retinal edema after lightning injury.

Photic injury at the macula following direct viewing of lightning strike can occur without any direct hit by lightning as described by Shukla *et al*.[5] There were hyperreflectivity and interruption of outer retinal layers, as seen on OCT, similar to the acute and chronic changes observed in welding arc and solar retinopathy. Shukla *et al*. emphasized the potential for phototoxic effects from viewing lightning at close range and added another dimension to the spectrum of lightning maculopathy.[5]

Imaging characteristics

Earlier reports relied on fundus fluorescein angiography for the diagnosis of macular lesions in lightning injuries. The most common lesion noted on fluorescein angiogram were retinal pigment epithelial window defects at the fovea and sometimes in the peripheral retina due to retinal pigment epithelial loss.[4,7,34-36,40]

OCT has revolutionized the diagnosis of macular lesions in recent times. OCT analysis of the macula was done in

five case reports of lightning injuries.[15,37,38,40,41] OCT helps in the accurate diagnosis and follow-up of CME, MHs, serous macular detachment, RPE atrophy, and foveal thinning and thereby helps in determining the visual prognosis. Armstrong *et al*.[15] described the evolution of lighting maculopathy using OCT. Earliest change noted was a double U-shaped elevation of inner foveal contour with focal elevation of inner segment-outer segment (IS-OS) junction. This evolved to hyporeflective cystic changes at the macula with a loss of foveal photoreceptors, focal loss of RPE cells and widespread disruption of IS-OS junction over 3 weeks. By 4 months, there was a complete resolution of cystic changes but persistence of increased choroidal reflectivity due to focal loss of RPE and associated thinning of neurosensory retina. The final visual acuity had thereby decreased to 20/200 due to foveal atrophy.[15] Liu *et al*.[40] reported a case of lightning injury in young woman, who presented 4 days after being struck by lightning directly to the forehead while hiking. They described the evolution of cystoid abnormalities at the fovea in this patient. Four days after injury, the OCT revealed subtle irregularity of the outer retinal layers, most notably nasal to fovea; 2 weeks later, there was transient development of intraretinal cystic spaces; 7 weeks later, there were decreased cystic spaces with subfoveal outer retinal hyporeflective space; at 4 months, there was persistence of subfoveal inner retinal hyporeflective space with restoration of outer retinal structures.[40] These case reports highlight the importance of OCT in diagnosis and prognostication of macular lesions associated with lightning injuries. Foveal atrophy and retinal pigment epithelial changes following resolution of macular lesions result in poor final visual acuity.

Electrophysiological changes

Electrophysiological tests are usually normal in lightning injuries. However, there were four case reports of abnormal electrophysiological tests after lightning injuries. Lagrèze *et al*.[7] reported a patient with a history of lightning strike, with normal scotopic and photopic electroretinography (ERG) in both eyes, but the electrooculogram (EOG) revealed pathologically low amplitudes bilaterally. They proposed that widespread RPE damage could have contributed to this. Augustin *et al*.[34] reported a patient with reduced scotopic ERG and normal photopic ERG. This same patient also had decreased night vision. Lin *et al*.[35] also described a patient struck by lightning, whose EOG showed reduced amplitudes in one eye. The amplitudes of scotopic and photopic ERG B-waves were also reduced in the same eye. Maximal amplitude varies with the number of functional B-wave generators. Bipolar and Müller cell death could account for the decline in amplitude. Yi *et al*.[31] reported a case of peripapillary retinal edema with extinguished scotopic blue flicker and reduced scotopic white 30-Hz flicker in the right eye and reduced photopic and scotopic ERG in the left eye. In the same patient, they reported reduced amplitude in pattern visual evoked response.

Surgical outcome of lightning-induced macular hole

There were many reports of development of full-thickness MHs following lightning injury. The earliest presentation was by 1 week of injury. MHs may occur early soon after the injury or late following long-standing CME.[10,32,39] In some of these patients, MHs were found to close spontaneously with residual RPE changes or epi-retinal membrane formation at the macula. However, surgical repair was needed for persisting holes with poor visual acuity.[10,18] Espaillat *et al*.[16] reported a case of MH with rhegmatogenous retinal detachment following lightning injury, treated surgically. The case presented with recurrent retinal detachment at 1 month of follow-up.

The majority of cases (n = 13, 62%) were managed conservatively. Vitreo-retinal surgery was done in one eye (5%) having MH with rhegmatogenours retinal detachment,[16] it was advised in one eye (5%) for MH,[39] and combined cataract surgery + vitreo-retinal surgery was done in one eye (5%) with MH.[10]

Cataract surgery was done in 6 (29%) eyes. The mean age of patients in which cataract surgery was done was 24 years (SD: 11 years, range: 13–39 years).

Visual prognosis

The mean visual acuity at the final follow-up was +0.6 logMAR (SD +0.79, range: 0–3). Fourteen eyes (56% eyes) had improved visual acuity, seven eyes (28% eyes) had worsened visual acuity, while in four eyes (16% eyes), the visual acuity was maintained. The final visual acuity of three eyes was not available. One eye turned blind due to lightning injury.

Visual prognosis in patients with lightning-induced ocular injury depends on the extent of involvement of ocular structures, and in the absence of anterior segment manifestations, irreversible retinal damage as well as optic nerve damage is the major determinant factor.[5] Late onset of pigmentary changes at the fovea and papillomacular bundle may further prevent visual improvement in these patients. Therefore, long-term follow-up of these patients is recommended.[27]

Prevention of lightning injuries

Most of the injuries and deaths by lightning happens because of misinformation and inappropriate behavior during thunderstorms. There are safer locations and locations which should be avoided during

thunderstorms. Large structure with plumbing and electrical wirings (e.g., houses, schools, and office buildings) and fully enclosed metal vehicles (e.g., cars, trucks, buses, and enclosed farm vehicles) are safer areas. It is important to roll up windows and avoid contact with metal or conducting surfaces inside or outside the metal vehicle. Areas to avoid include those near tall objects, such as towers or trees, and those near water or open areas. Familiarity with and implementation of lightning safety guidelines can decrease injuries.[18,43-47]

Conclusion

Lightning injury can cause a wide range of injuries to posterior segment of eye of varying severities. Although lightning injuries are usually mild injuries, with vision remaining, either stable or improved in the majority of cases, severe visual loss due to optic atrophy and maculopathy may occur in long term. Hence, long-term follow-up of these eyes is recommended.

Financial support and sponsorship
Nil.

Conflicts of interest
The authors declare that there are no conflicts of interests of this paper.

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