Outer lamellar hole with severe visual loss following High Tension Electric shock

ABSTRACTS
The authors report a case of macular cyst following electric shock by high tension current with serial SD-Optical Coherence Tomography and its natural history to outer lamellar hole leading to irreversible loss of vision.

INTRODUCTION
Tissue damage from electric shock may occur through one or more mechanisms: transmission of electric current directly through tissues, conversion of electrical energy to thermal energy, which is subsequently absorbed by the tissues, or end organ ischemia caused either by generalized vascular constriction or cardiac arrhythmia. The extent of damage to the tissues depends on the intensity of the current, duration of the tissue exposure, and the tissues resistance to the current. Resistance is variable in different body tissues and is known to be the greatest in bones with decreasing resistance in fat, tendon, skin, muscles, blood vessels and nerves. For ocular tissues, retina and optic nerve have a low resistance and thought to be primarily affected by ischemia resulting from coagulation and necrosis of vascular tissues that feed them.

CASE REPORT
A 39 years old otherwise healthy male present to outpatient department with defective vision in both eyes following electric shock from high tension wire since 11 days. Visual loss typically started after day 3 following electric shock. He had grade I burns over both the hands. He had giddiness, headache, vomiting following the episode for which he was medically managed. Computerized tomography of brain was normal. There are was no history of sun gazing, exposure to welding arc or solar eclipse previous to this episode.

His best corrected visual acuity in right eye and left eye was 20/60 and 20/40 respectively. Anterior segment examination showed anterior capsular changes in both eyes (fig.1A and 1B). Fundus examination (2A & 2 B) showed cystic changes at the fovea which was confirmed on OCT (2C &2D), which also showed IS-OS junction defect. There was no evidence of vitreomacular traction or posterior hyaloid separation. Fundus camera based autofluorescence (Topcon TRC 50 DX) imaging showed increased central hypoautofluorescence surrounded by decreased parafoveolar hypoautofluorescence (2E &2F).

On his subsequent visit after 3 months, his BCVA was dropped to 6/24 in RE and 6/36 in LE, which is partly attributed to anterior and posterior diffuse subcapsular cataract (fig. 3A &3B).

OCT of both eyes showed outer lamellar hole(fig. 3C & 3D) on his last visit.

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FIGURE 2 (For description see next page)

FIGURE 3
Figure (A) and (B) shows color fundus images of posterior pole demonstrating pale yellow spot at the center of the fovea in both the eyes. Spectral Domain Optical Coherence Tomography (horizontal 6 mm scan) shows outer lamellar defect with few cystoid changes just below internal limiting membrane and hyperreflectivity at the level of Retinal pigment epithelium in right eye (C) and cystoid edema with with cystic changes in inner nuclear layer along with interruption of external limiting membrane and inner segment and outer segment junction in the left eye (D). Central macular thickness was 206µ in OD and 310µ in OS. Fundus camera-based autofluorescence images showed increased central hyperautofluorescence corresponding outer lamellar defects and decreased hypoautofluorescence in perifoveal region (E) and (F).

Discussion
Electric current can injure a tissue by several mechanisms. First, it can directly destroy cells and body structure. It can also damage retinal pigment epithelium by electrolysis. Second, melanin retinal pigment epithelium offers resistance to electric current which produces heat causing thermal injury.1,4 Localized inflammation in response to injury could contribute to pigment epithelium dysfunction. Third, damage to the posterior pole can occur by shock wave generated by lightning strike.4 The homeostasis of the retina could be compromised by mechanical, thermal, or inflammatory injury. Intraretinal edema could result from injury to Muller cells, which are involved in the active transport of fluid out of the retina.7 Cataract formation is the most common sequel of electric burn and can present in 5% cases of electric shock above neck region.6 Handa JT et al reported a case of lightning maculopathy with cystic changes at the fovea along with diffuse posterior subcapsular cataract which progressed subsequently in both eyes. However, 14 month after injury and cataract extraction, visual acuity of 20/20 was noted in both eyes.6 Campo and Lewis reported a case of full thickness macular hole following lightning strike with visual acuity of 20/40, however, Watzke Allen test was not reported in the study and OCT was not available then.10 None of these reports of lightning induced macular cyst or macular hole have OCT documentation for confirmation.

Shukla et al have reported a case of lightning maculopathy following visualization of distant lightning strike. SD-OCT images showed central hyperreflective echoes with disruption of inner segment and outer segment junction in each eye. Fundus autofluorescence images showed bilateral increased hypoautofluorescence and decreased parafoveolar hypoautofluorescence.11 However, by 12 months visual acuity returned to 20/20 with IS-OS disruption on SD OCT findings.

We believe the damage to outer retina at the fovea was caused by thermal reaction to resistance posed by retinal pigment epithelium to passage of electric current. We don’t know that why it was concentrated only on the fovea.

References