Peripapillary retinal thermal coagulation following electrical injury

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In this study, we have presented the case report of a 20 year old boy who suffered an electric injury shock, following which he showed peripapillary retinal opacification and increased retinal thickening that subsequently progressed to retinal atrophy. The fluorescein angiogram revealed normal retinal circulation, thus indicating thermal damage to retina without any compromise to retinal circulation.

Key words: Cystoid edema, electrical injury, retinal opacification, retinal atrophy

Electrical injury from high-tension wires can result in a significant ocular morbidity. Tissue damage can occur through the transmission of electrical current directly through the tissues, and conversion of electrical energy to thermal energy, which gets subsequently absorbed by tissues, thus causing end-organ ischemia either by generalized vascular constriction or by cardiac arrhythmia. The extent of damage to the tissues depends on the intensity of the current, duration of tissue exposure, and the tissue’s resistance to the current. The optic nerve and retina have a low resistance and are thought to be primarily affected by ischemia resulting from coagulation and necrosis of the vascular tissues that feed them. The sequence of events following such an injury to retinal tissues is not known. We report a case which illustrates that the initial acute response following electric shock injury is the retinal opacification with increased retinal thickness that subsequently progresses to retinal atrophy.

Case Report

A 20 year old boy presented to our department with decreased vision, redness and watering of both eyes following electrocution with overhead transmission wires six days ago. The electricity transmission wires were carrying a high voltage of approximately 20,000V. He had multiple eschars on the back, scalp and arms along with facial erythema [Fig. 1]. His best-corrected visual acuity was light perception in the right eye and counting fingers in the left eye with normal intraocular pressures. There was a lid scaling and charring of the eyelashes. Anterior segment examination showed bilateral circumcorneal congestion, sluggish pupillary reactions, superficial punctate keratitis and cataract in the left eye. Fundoscopy revealed an area of peripapillary retinal opacification and a dull foveal reflex in both eyes [Figs. 2 and 3]. Fundus fluorescein angiogram showed a normal retinal circulation with late hyperfluorescence corresponding to the area of opacification. Optical coherence tomography (OCT) showed retinal thickening with few cystoid spaces in the right eye [Fig. 4]. Similar changes were seen in the left eye (not shown). Patient was started on oral corticosteroids (prednisolone 30 mg/day) and acetazolamide (750 mg/day in three divided doses). Steroids were given to control inflammation arising from the injury, if any, and oral carbonic

Figure 1: Photograph of the face of the patient in the study following electrical burn showing eschars, erythema and facial erythema

Figure 2: Fundus photograph of the right eye of the patient in the study at presentation showing areas of peripapillary retinal opacification with a dull foveal reflex
anhydrase inhibitor were added to reduce cystic fluid collection by possible stimulation of the retinal pigment epithelium (RPE) pump. Acetazolamide was withdrawn after 1 month and oral steroids were discontinued after another month by gradual tapering. One month later, his best corrected visual acuity was counting fingers 1 meter in the both eyes with posterior subcapsular cataract and peripapillary retinal atrophy in both the eyes [Fig. 5]. OCT of both the eyes showed retinal atrophy with disorganization of all retinal layers and the RPE [Fig. 6]. Patient underwent cataract surgery after 6 months from the time of injury in both the eyes with no improvement in the visual acuity.

Discussion
Optic nerve is very good conductor of electricity, with the nerve tissue conducting electricity as any metal rod. The retina usually suffers the thermal effects of injuries in the form of immediate coagulation of proteins and cells, and thus the initial whitening/opacification of the retina can be explained. The absorption of energy by the RPE results in retinal damage which was evident in the peripapillary and macular area in our patient. A normal fluorescein angiogram indicates that the retinal opacification was not as a result of ischemia, but was due to the thermal coagulation of the retinal tissue. According to a recently published report, peripapillary meridional changes in the eye have been noted in a patient suffering from electric injury. However, OCT changes underlying the pathology have not been studied in that study. Our case illustrates that initial acute response following electric shock injury is the retinal opacification with increased retinal thickness, that subsequently progresses to retinal atrophy.
References


