Electrical Cataracts: A Case Report and Review of the Literature

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ABSTRACT
A case report of electrically-induced cataracts and a review of the literature is presented. Awareness of the possibility of this complication and screening of high-risk patients is stressed. The majority of cases respond well to surgery, but final visual acuity may be decreased secondary to retinal or optic nerve injury caused by the electrical current.

Although rare, cataracts secondary to electrical injury have been described.1-4,9,10,12,15 We present a case report and review the literature.

CASE REPORT
A powerline carrying 7200 volts fell onto the left shoulder of a 32-year-old power company employee. He grasped the line with both hands and was electrocuted. Examination at a burn center revealed severe facial edema and burns (Figure 1). His right pupil was reactive but his left eye could not be visualized due to marked lid edema.

The patient first complained of blurred vision LE approximately 6 weeks after his accident. Visual acuity was 20/20 RE, 20/40 LE. Pupils were 3.5 mm RE/3.0 mm LE, briskly reactive bilaterally to direct and consensual light. The lens on the right showed a fine feathery anterior subcapsular cataract in a ring shape (Figure 2). The lens on the left showed a diffuse anterior subcapsular cataract with inferior vacuoles (Figure 3).

Nine days later visual acuity was 20/20 RE, 20/200 LE. The cataract in the right eye was unchanged, but the lens in the left eye had developed a diffuse posterior subcapsular opacity in addition to worsening of the anterior subcapsular opacity. He also developed iritis LE.

Figure 1: External photo of face, neck, and shoulders after injury.

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Approximately 3 weeks later (11 weeks after the accident) visual acuity in the RE was 20/25, despite the progression of the cataract to a complete anterior subcapsular opacity (Figure 4). Visual acuity LE being hand motion, the patient underwent extracapsular cataract extraction with posterior chamber lens implantation. One week later his visual acuity RE dropped to 20/300, leading to another cataract extraction with lens implantation. No evidence of increased capsular fragility or zonular weakness was noted. Final visual acuity was 20/20 RE and 20/30 LE. Postoperative examination was unremarkable RE, but a macular hemorrhage and wrinkling of the internal limiting membrane LE was revealed. ERG testing was normal in both eyes.

**DISCUSSION**

As shown in the Table, electrical accidents can damage virtually every part of the eye.

The first description of electrically-induced (lightning) cataracts was by Saint-Yves in 1722. A cataract produced by artificially generated electrical current was reported by Desbriers and Bargy in 1905. The average incidence of cataract formation following electrical injury appears to be approximately 6%, relative to the voltage experienced.

Because such injuries are usually life threatening, the eyes are often not examined until days to weeks after the accident. Cataracts usually form two to six months later, but they can appear immediately or as late as 11 years later. The time involved seems to have no relation to the experienced voltage. There is a general tendency toward progression, but occasionally the cataracts remain stable or regress. Fraunfelder and Hanna reported an 82% incidence and Safflé reported a 77% incidence of electrical cataracts progressing to the point where surgery was required.

The closer the point of contact is to the eye, the greater is the likelihood of cataracts. If they are bilateral, the cataract on the side of contact is worse. For unknown reasons, electroconvulsive therapy does not cause cataracts.
The typical lesions following electrical burn can be seen in the lens within a few days. The earliest changes are multiple fine vacuoles beneath the anterior capsule, usually midperipheral in location and sometimes requiring pupillary dilatation for visualization. Thomas and Hanna produced electric cataracts in rabbit eyes. Lens vacuoles appeared initially, increased in size, and gradually disappeared. These vacuoles were replaced by anterior subcapsular cataracts. Ultrastructurally the vacuoles were located between the epithelial cells. The adjacent epithelial cells were greatly dehydrated and grossly deformed. As the vacuoles disappeared, the surrounding epithelium began to divide and differentiate into small lens fibers. These new fibers produced new lens capsule material which correlated with the gross appearance of the white fibrillar anterior opacities (Figure 2). The individual fibers produced opaque plaques that coalesced to form a central elevated anterior subcapsular cataract (Figures 3 and 4). The lens vacuole formation seen shortly after electrical injury has been shown to be predictive of future lens changes.8

In general, industrial electrical accidents affect the anterior superficial lens cortex, while lightning injuries involve both anterior and posterior subcapsular areas. Saffie, however, reported a 40% incidence of posterior subcapsular cataracts in his review of industrial electrical accidents.3

The severity of electrical injury is determined by Ohm's Law, which states that amperage is equal to voltage divided by resistance. Usually only the voltage can be determined in an electrical injury. Although the usual skin resistance is between 3000 to 5000 ohms, it may be reduced to around 300 ohms by wetting. Current less than 500 volts rarely overcomes skin resistance. Voltage causing cataracts in reported cases ranged from 220 volts to 60,000 volts.14

The pathogenesis of these lens changes is not completely known. One theory holds that the current has direct coagulative effect on the proteins of lens cells. If this were the case, however, one would expect more immediate cataract formation. Galezowski and Knies suggested that the powerful contraction of the ciliary muscle mechanically displaced the lens fibers, causing a concussion type injury. This theory would be supported by the occasional ring shape of the cataract and its location peripheral to the pupillary border. Heat has also been suggested as a factor in cataract formation, but Long9 has shown that the temperature at the point of contact is not increased above body temperature. Changes in capsular permeability were first noted by Hess and the experimental work of Kuwabara, Croci, and Bellows and Chinn lends support to this view.

REFERENCES