

Late Retinal Manifestations of Electric Shock Retinopathy: A Case Report

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Abstract

Purpose: We report a 40 year old male who presented with extensive areas of chorioretinal atrophy, pigmentary changes, optic disc pallor and mild vascular attenuation years after an electric shock injury from an exposed wire.

Observation: 40-year-old man presented with bilateral decreased vision following a high-voltage injury years ago. No significant family or ocular history was found. Anterior segment examination was normal and bilateral fundus examination showed optic disc pallor, presence of extensive areas of chorioretinal atrophy and pigmentary changes surrounding the optic nerve extending up to mid-peripheral fundus, partially involving the macula with spared areas on superior retina and mild retinal vascular attenuation.

Conclusion and Importance: Late retinal manifestation with chorioretinal atrophy and pigmentary changes following electric injury can resemble other diseases with similar clinical pattern and highlight the importance of the medical history and clinical evaluation.

Keywords: Electrical Injury; Choroidal Atrophy; Retinal Atrophy; Pigmentary Retinopathy

Introduction

High voltage electric currents produces ocular damage at several levels and its mechanism includes disruption of cell membranes and vasoconstriction that finally leads to ischemia and tissue necrosis. Retinal damage and cataracts are the most common injuries produced by this mechanism [1,2].

The electric current travels through the retinal pigment epithelium (RPE) then becomes heat and destroys the overlying retina; since the RPE of the macula has the highest concentration of melanin, it is the most affected site, but the entire retina can be affected. The extent of damage will depends on the intensity of the current, duration of tissue exposure, and the tissue's resistance to the current [3]. Damage to the optic nerve and retina is mainly caused due to its low resistance to electric current and by ischemia [1,3].

Retinal lesions due to an electrical injury can be unspecific and highly variable, including vitreous hemorrhage, retinal edema, macular hole, pigmentary alterations, central vein occlusion, chorioretinal rupture and retinal detachment [1,2]. We report a case showing late retinal manifestations with extensive chorioretinal atrophy and pigmentary changes following an electrical burn accident years ago.

Case Report

Clinical history

A 40-year-old man presented with bilateral acute loss of vision 10 years ago following a high-voltage injury. He sustained the injury when his head accidentally touched an exposed electric wire during work. He had multiple burns on the head, scalp, back and arms

requiring multiple facial surgeries. Months after the incident, he developed bilateral cataracts, and surgery was performed at that time with no vision improvement. No significant family or ocular history was elicited. He was clinically diagnosed to have pigmentary retinopathy in both eyes.

At the time of presentation (ten years later) the best-corrected visual acuity (BCVA) was counting fingers in the right eye and 6/120 in the left eye. Anterior segment examination revealed in the bag intra ocular lens in both eyes and normal intraocular pressure. Bilateral fundus examination (Figure 1 and 2) revealed optic disc pallor and the presence of chorioretinal atrophy surrounding the optic nerve extending up to mid-peripheral fundus, partially involving the macula associated with diffuse pigment hyperplasia and some spared areas on the superior retina. Mild retinal vascular attenuation was also noted.

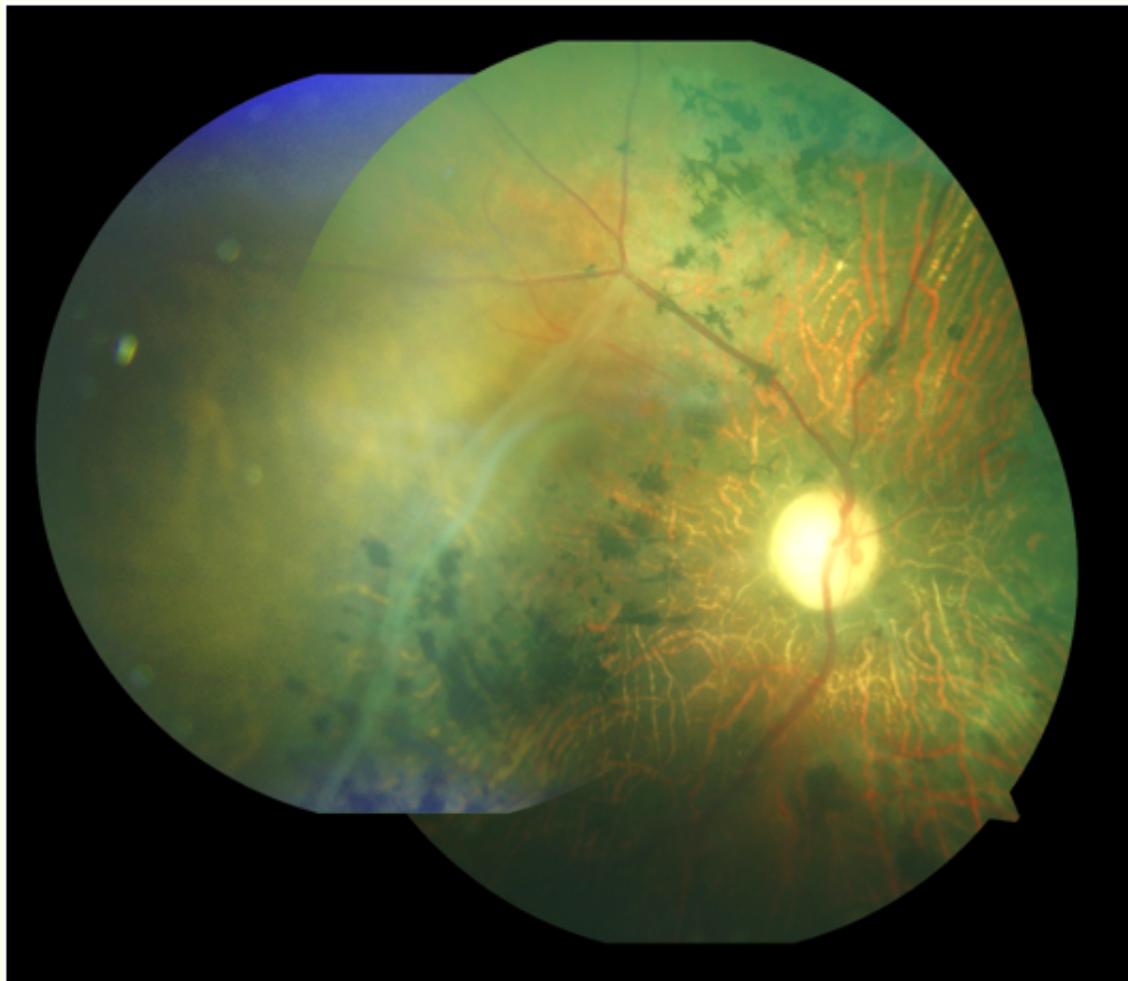


Figure 1: Color fundus photographs of the right eye showing extensive peripapillary chorioretinal atrophy with pigmentary changes, extending up to the mid-peripheral fundus, pale optic disc, normal spared superior retina and generalized vascular attenuation in both eyes.

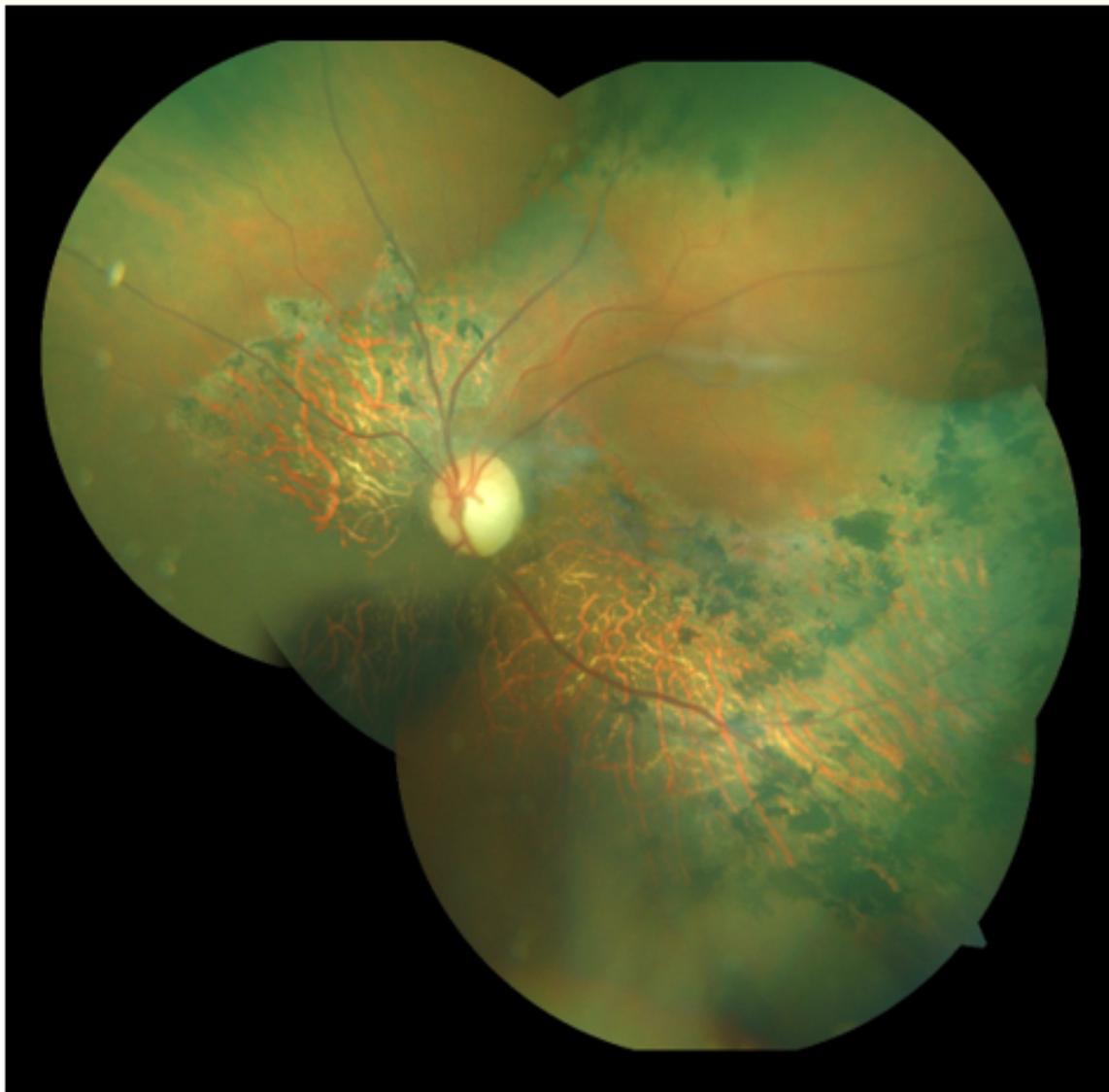


Figure 2: Color fundus photographs of the left eye showing extensive peripapillary chorioretinal atrophy with pigmentary changes, extending up to the mid-peripheral fundus, pale optic disc, normal spared superior retina and generalized vascular attenuation in both eyes.

Fundus fluorescein angiography (FFA) (Figure 3) showed extensive areas of hypofluorescence to due a blocking effect and hyperfluorescence, indicating transmission window defects. Optical coherence tomography (OCT) (Figure 4) showed severe choroidal and retinal atrophy in affected areas and retinal thinning of the outer retinal layers of the superior less affected retina. The patient also had burn scars on his head, hand, left leg and scalp.

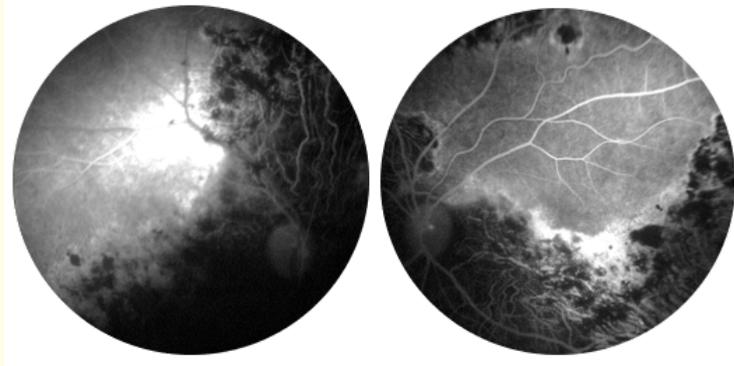


Figure 3: Fundus fluorescein angiography (FFA) showing extensive areas of hypofluorescence due to a blocking effect and hyperfluorescence, indicating transmission window defects.

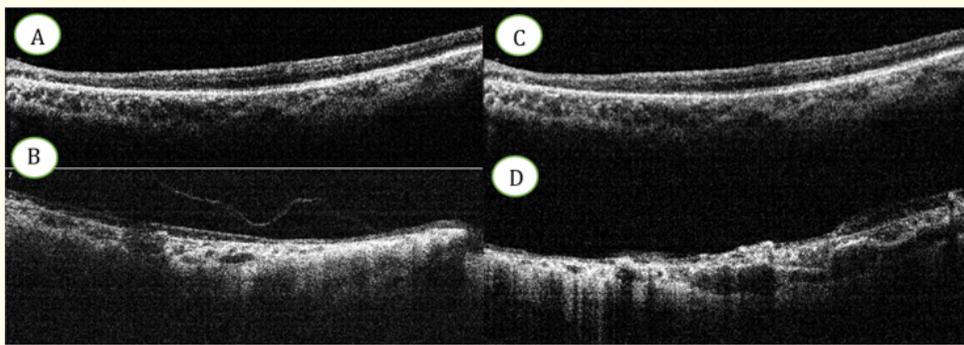


Figure 4: Optical coherence tomography (OCT) showing severe choroidal and retinal atrophy in affected areas (B and D, right and left eye respectively) and retinal thinning of the outer retinal layers of the superior less affected retina (A and C, right and left eye respectively).

Discussion

The injuries resulting from electrical burns can be very severe since the resulting damage is secondary to the direct passage of electrical current through the tissues. Electrical injuries produces tissue damage by direct cell injury or by the conversion of electricity into heat. The acute cell membrane depolarization leads to cellular dysfunction and death, and the dissipated heat causes severe burns at the entry and exit points, and all along the path of the current. Electrical energy is converted to thermal energy, which is absorbed by tissues, causing ischemia that finally leads to tissue necrosis [1,2]. Optic nerve is a good conductor of electricity [4]. The retina usually suffers the thermal effects due to the absorption of energy by the RPE in the form of immediate coagulation of proteins and cells [2].

Retinal manifestation are mainly seen surrounding the optic nerve and retinal periphery. Fundus findings may be non-specific (Table 1) [2] ranging from initial retinal whitening, and edema [5], hemorrhages, retinal detachment and choroidal rupture in acute injury to severe chorio retinal atrophy as late manifestations [1,5-10]. Late optic atrophy and retinal vascular damage may occur. Unilateral or bilateral optic neuropathy has also been reported [11,12]. Izzy, *et al.* [12] and Grover, *et al.* [4] reported unilateral ischemic anterior optic neuropathy following an electrical injury. Bilateral retinal detachment can also occur in the acute phase and has been reported by

Faustino., *et al.* and [7], Koytak., *et al* [8]. Another frequent retinal finding reported by Sony., *et al.* [6], Faustino., *et al.* [7], Miller., *et al.* [2] and Lakosha., *et al.* [10] are the formation of macular cysts that in some cases progressed to full thickness macular holes and even retinal detachment.

Bilateral retinal detachment	Faustino., <i>et al.</i> [7], Koytak <i>et al</i> [8]
Chorioretinal atrophy	Verma., <i>et al.</i> [8]
Pigmentary retinopathy	Verma., <i>et al.</i> [8]
Isquemic optic neuropathy	Izzy., <i>et al.</i> [12], Grover., <i>et al.</i> [4]
Macular cysts	Sony., <i>et al.</i> [6], Faustino., <i>et al.</i> [7], Miller., <i>et al.</i> [2], Lakosha., <i>et al.</i> [10].
Retinal edema	Tandon., <i>et al.</i> [10]

Table 1: Retinal manifestations from electric injury.

Our case illustrates late retinal findings after an electric injury. Other causes with a similar clinical pattern such as hereditary and inflammatory diseases need to be differentiated and a very detailed history regarding symptoms of night and/or color blindness in the patient and family besides the presence of consanguinity are particularly very important. Extensive areas of chorioretinal atrophy involving up to the mid-peripheral fundus may be present in both conditions so differentiating this condition from other entities based only with electroretinography (ERG) is difficult. Inflammatory diseases usually present with inflammatory reaction in the anterior chamber or vitreous, a greater extent of retinal involvement, vascular changes such as sheathing and recurrent episodes with poorer vision. OCT may be normal or show chronic cystoid changes or central thinning depending on the stage of the inflammatory disease. FFA and OCT showing unspecific defects (diffuse and severe chorioretinal atrophy with indistinct edges) are seen in the present case.

Retinal manifestations following an electric injury are non specific and late manifestations as the ones reported in the present case are very difficult to distinguish from another entities without a meticulous medical, family and clinical history.

Conclusion

Late retinal manifestation with chorioretinal atrophy and pigmentary changes following electric injury can resemble other diseases with similar clinical patterns and highlight the importance of the medical history and clinical evaluation.

Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient have given his consent for his images and other clinical information to be reported in the journal. The patient understand that his names and initials will not be published and due efforts will be made to conceal his identity but anonymity cannot be guaranteed.

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Conflicts of Interest

The authors have no financial disclosures nor conflict of interest.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

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