

## Case Report

## Late onset reversible cortical blindness following electrocution



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## ARTICLE INFO

## Article history:

Received 4 April 2015

Received in revised form 14 July 2015

Accepted 11 October 2015

Available online 11 November 2015

## Keywords:

Cortical blindness

Electrocution

Reversible

Steroids

Delayed onset

## ABSTRACT

An elderly gentleman presented with acute onset of bilateral visual blurring and generalized headache after 1 week post electrocution injury. Clinically, the symptoms were attributed to cortical lesion. Magnetic resonance imaging (MRI) of brain revealed bilaterally symmetrical diffusion restriction in parietal and occipital areas. Treatment with intravenous steroids resulted in remarkable improvement in symptoms. Neurological injury secondary to electrocution is a well described entity having a variety of clinical presentation. We put forward our experience with this unique case presenting as post electrocution delayed onset of visual symptoms. Discussion and review of literature related to this clinical entity will also be presented.

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## 1. Introduction

Electrical injuries are one of the most common occupational and household hazards. Local injuries as an immediate effect of electrical current are a well-known phenomenon. The spectrum of medical disorders secondary to electrical current injury is extensive and complex. Neurological manifestations secondary to such injuries are well known but literature description of case reports is limited. Multiple mechanisms have been considered responsible for the genesis of these symptoms, but none of them have received a unified approval. The neurological symptomatology following electrical injury involves multiple anatomical substrates and also has a temporal diversity in relation to the occurrence of electrocution. We describe a case having delayed onset of visual symptoms with symmetrical cortical lesions after electrocution, which in our experience has not been reported earlier in literature.

## 2. Case report

A fifty year old gentleman was working at a factory on a rainy day, surrounded by electrical appliances. He suffered from an electrocution, probably from one of the loosened wires lying on a wet floor. He apparently became unconscious immediately and suffered from the burns over his right leg and left hand without any significant head trauma. On regaining his senses after 30 min, he had generalized body ache and severe pain over his right leg. He

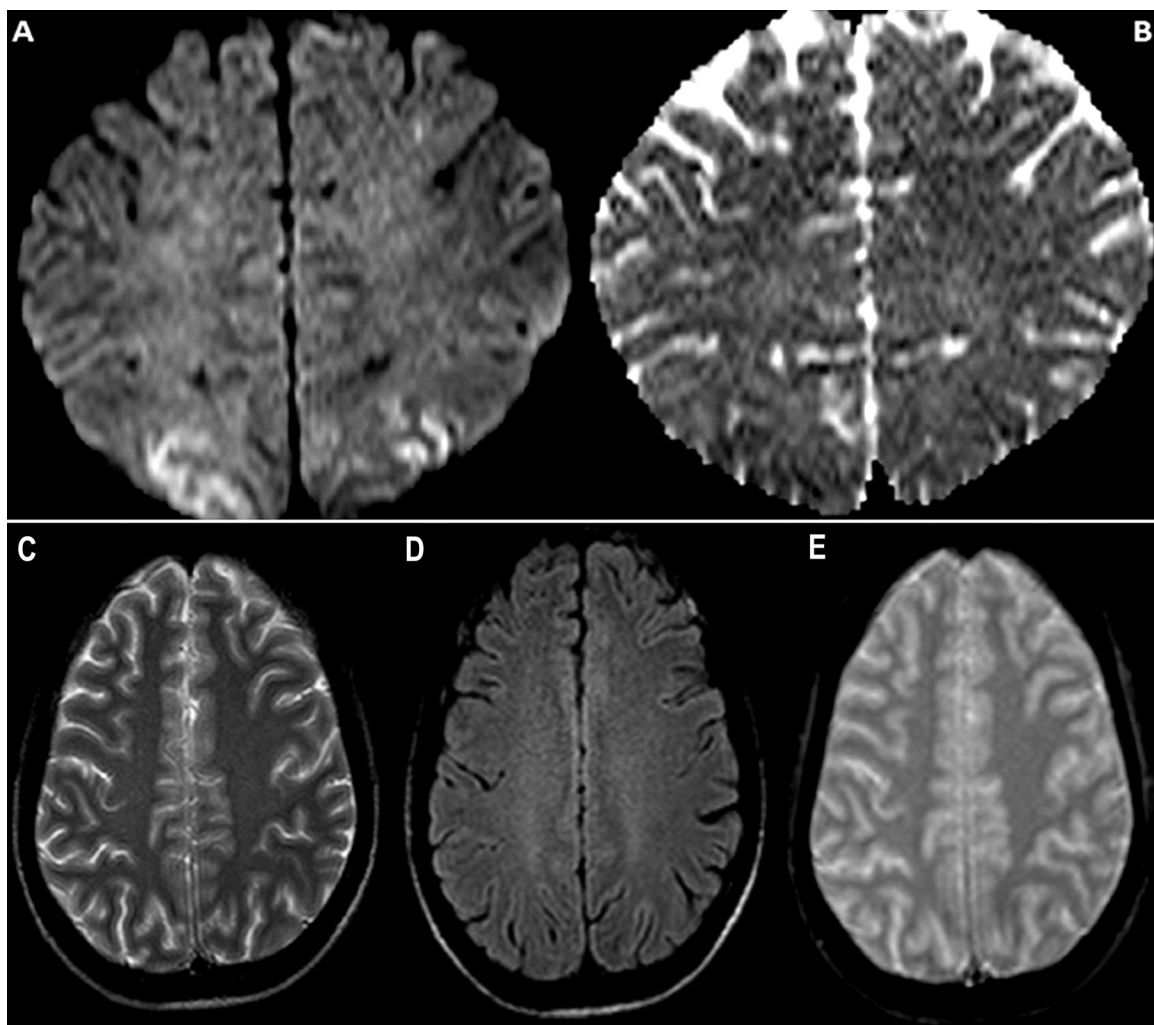
was rushed to the hospital, where he was given primary management and was admitted for observation. He was discharged in a few days following an uneventful stay at hospital with no residual symptoms. One week later, he woke up in the morning with sudden onset of generalized headache and bilateral visual blurring. He did not have any history of similar headache in the past. The headache was severe and associated with vomiting. The visual blurring was similar in both eyes and was more prominent in lower half of his visual field. Headache and visual blurring progressed over 2–2 h after the onset, until they became static and continuous. He denied diplopia, local eye symptoms or drooping of eye lids. It was not associated with any other neurological symptoms. He did not have any vascular risk factors and his previous medical history was not significant.

When evaluated by an ophthalmologist, local ocular examination was normal. He barely had perception of light in both eyes. Color vision and perimetry were not possible due to poor visual acuity. Pupillary reflexes were intact bilaterally without relative afferent pupillary defect. Neurological examination did not reveal any other abnormal signs, systemic examination was unremarkable. He had healing burn wounds over right leg and left hand. Blood pressure was within normal range, carotid bruit was absent and all peripheral pulsations were palpable. Routine blood investigations and electrocardiogram did not show any abnormality.

Magnetic resonance imaging (MRI) of brain showed presence of symmetrical diffusion restriction with corresponding low apparent diffusion coefficient values in parieto-occipital regions. The lesions were inhomogeneous and irregular, beyond vascular territories and strictly cortical in location. T2 and Fluid Attenuation Inversion Recovery (FLAIR) sequences showed subtle hyperintensity in

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**Fig. 1.** MRI brain shows symmetrical diffusion restriction in bilateral occipital and parietal areas (A) and corresponding low intensity in ADC image (B). The T2 (C) and FLAIR (D) sequences show subtle high intensity signals in similar areas. GRE image (E) is negative for any abnormalities.

corresponding areas. Intracranial plus extracranial angiogram and venogram were normal (Fig. 1). Radiological possibilities of infarction, edema or demyelination were considered.

Investigations to diagnose underlying vascular risk factors were negative. Blood pressure monitoring did not show any fluctuations. After 24 h of onset, the headache became more severe with worsening of vision as well. He was initiated on intravenous steroids at the dose of 1 g methylprednisolone per day, which was continued for a total of 3 days. His symptoms of headache and visual blurring improved significantly after steroid therapy was started. He was able to count fingers at 4 m distance. He was discharged after 6 days of hospital stay with improved vision and no residual headache. He followed up as an outpatient after 4 weeks with an improved visual acuity of 6/9 (Snellen's chart) bilaterally. He was functionally independent and was able to carry out his social and occupational responsibilities.

### 3. Discussion

Electrical current is governed by three parameters: voltage, resistance and amperage. Severe form of electrical injury has been described as electrocution. Electrocution accidents are a common occupational hazard and are also seen in household environment. The nature of current, alternating current (AC) and direct current (DC), also influences the severity of injuries sustained in these

accidents. Most of the accidents are secondary to exposure to AC which is the more destructive as compared to DC. The extent of injury is also decided by other factors such as voltage, duration of exposure, resistance offered by body parts, area of contact, presence of protective devices, etc. Effect of electricity on the body can be broadly classified as direct and indirect effects. The direct or local effects are well described and more common in occurrence. However, it is the indirect effect which is more complex in terms of anatomical involvement and temporal profile after an electrocution.

Neurologically speaking, electric current can affect almost any part of the neural axis. The symptoms can be as nonspecific as headache, lethargy, fatigue, exhaustion, spasms, cramps, joint weakness or physical sensitivity [1]. Specifically diagnosable syndromes affecting peripheral nerves, muscles, autonomic symptoms, spinal cord, cerebral cortex and psychiatric manifestations have been recognized. It requires a high index of suspicion and knowledge regarding the temporal association so as to diagnose these entities.

Cherington [2] proposed four categories of neurological sequelae that could result from electrical trauma. The categories are:

- 1) Immediate and transient: symptoms occur at the time of incident and spontaneous remission occurs over hours to days.

**Table 1**

Previously reported cases with stroke or stroke like presentation after the electrocution and delay of onset after the incident.

Author	Delay from incident	Deficits
Critchley	9 months	Right hemiparesis and aphasia
Langworthy	1 day	Right hemiplegia
Haase and Luhan	3 weeks	Right hemiplegia
Gans and Glaser	4 days	Right homonymous hemianopia
Present case	7 days	Cortical blindness

Examples under this category are sudden loss of consciousness and retrograde amnesia.

- 2) Immediate and prolonged or permanent: symptoms occur at the time of incident and persist for days to years or indefinitely. Brain hematoma, infarction, etc. are common examples.
- 3) Delayed and progressive: symptoms are absent at the time of incident but present after a delay and are progressive in nature. The delay is variable, ranging from few days to years after the incident. This category is exemplified by disorders like demyelinating diseases, movement disorders, hydrocephalus, thrombosis, etc.
- 4) Linked or coupled: the indirect effects of the incident occur. For example, head injuries following a fall after the electrocution.

The spectrum of neurological manifestations following electrical injury is vivid. Literature describes cases with peripheral neuropathy, seizures, central nervous system complications (myelopathy, encephalopathy, venous sinus thrombosis), movement disorders, headache and memory disturbances. Neuropsychological manifestations include insomnia, emotional disturbances, anxiety, phobia to electricity, inability to concentrate, post-traumatic stress disorder, etc.

As in our case, acute stroke or stroke like presentation has been described in previous case reports. Gans and Glaser described a case with acute visual symptoms 4 days after the electrocution secondary to left occipital cortical infarct (Table 1) [3].

The mechanisms for genesis of delayed neurological symptoms are speculative and variable. One of the earliest description of brain biopsy specimen after death from electrocution showed incomplete perivascular necrosis, demyelination, swelling of oligodendroglia with compression of cortical gray ribbon cells. Later, it was suggested alteration of proteins in the endothelial cells which may lead to thrombosis and vascular compromise in the affected areas. Another hypothesis is that over excitation of neuronal cells through the electric current releases glutamate in excess which is capable of cell death. Excess glutamate also results in generation of free radicals through oxidative stress mechanisms and result in cytotoxicity. It is possible that the effects of oxidative stress are delayed and cumulative. Theory of extraporation [4] explains that electric field induces 'pores' in the cell membrane, resulting in temporary dysfunction or cell death. This theory explains those cases which have transient symptoms following the electric injury and are self-limiting. The end result of above mentioned mechanisms

possibly leads to edema, dysmyelination, ischemia or infarction, which in turn manifest as acute or delayed onset symptoms. However, other aspects of pathogenesis such as variability in delay of onset, involvement of isolated areas of neural axis in an individual, severity of symptoms and underlying pathological variations still remains unexplained.

Our speculation of causal relation is based on sudden onset of symptoms with radiological correlate occurring after latency of 7 days of the incident. Almost all the possibilities leading to our clinical scenario were ruled out through history, examination and investigations. The clinical profile along with the radiological picture resembles that of posterior reversible leukoencephalopathy syndrome, and a possible role of electric injury leading to this syndrome cannot be refuted with certainty in this case. Also, since the initial event of loss of consciousness was unwitnessed, possibility of delayed clinical presentation of initial anoxia cannot be denied. However, the temporal aspect of this case, reversibility with steroids and previous literature description of delayed neurological insults after electrocution cannot be ignored. Our decision to treat with steroids was based on possibility of edema or demyelination. We reviewed a similar case report of visual symptoms post electrocution, which improved with steroid therapy [5]. Still, role of steroids in such cases remains elusive and needs further correlation with significant number of similar cases.

#### 4. Conclusion

In summary, neurological sequelae following electrocution have wide diversity in terms of symptoms, neuroanatomical involvement, temporal profile and pathogenesis. Even the involvement of site remote from contact can be affected. It is important to remember the entity of delayed onset neurological injury post electrical injury. Radiological lesions may help in understanding the pathology in some cases. To the best of our knowledge, this is the first description of a reversible blindness of delayed onset after electrocution with radiological evidence. The occurrence of delayed onset of cortical blindness following electrocution and its reversibility with steroids marks the salient features and learning points in this case.

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