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Case report

Different sequelae of electrical brain injury — MRI patterns

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ARTICLE INFO

Article history:
Accepted 14 March 2017

Keywords:
Electrical injury
Brain
MRI
Diagnosis

ABSTRACT

Purpose: Electrical injury to the central nervous system may lead to neurologic compromise via pleiotropic mechanisms. It may cause current-related, thermal or nonthermal damage followed by secondary mechanisms.
Methods: We herein report a case of a 20-year old man, who experienced a low-voltage electric injury due to an occupational accident.
Results: Magnetic resonance imaging (MRI) one week after the insult allowed differentiation of pathophysiologic features including thermal, nonthermal and hypoxic cerebral lesions.
Conclusion: The capability of MRI assessing a variety of lesions for diagnostic and potentially prognostic reasons is presented.

1. Introduction

Electrical injury may affect multiple organ systems. Neurologic compromise may present in different ways including cerebral or spinal cord injury as well as peripheral nerve injuries. The clinical manifestation might be acute or delayed [1]. Depending on the entry site, the nervous system might be affected via a pleiotropic route of action resulting in direct thermal-induced or current related insults as well as secondary injuries such as disruption of transmembrane ion gradients due to electroporation of proteins [2]. Therefore cells with a relatively large surface – like neurons – are most likely more susceptible to electric injury [3]. Nowadays MRI may allow the objective differentiation between several pathophysiological mechanisms after severe electric injury to the central nervous system.

2. Case report

A previously healthy 20-year-old man experienced a low voltage electric injury (400V, 70A) in an occupational accident with loss of consciousness and cardiac arrest followed by

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http://dx.doi.org/10.1016/j.burns.2017.03.012

successful cardiopulmonary resuscitation. The suspected entry point of the current was the left ear with development of extensive necrosis thereafter. Exit wounds were localized predominantly at the right body site. The intubated and ventilated patient was transported to the local hospital.

On arrival initial brain CT (multisclice CT with reconstructions) demonstrated a slightly decreased density of the white matter of the left temporal lobe. Follow-up brain CT on day 3 revealed a sharply demarcated crescent-shaped (16 mm × 55 mm × 39 mm) hypodense lesion of the cortex and the subcortical white matter of the left temporal lobe and decreased density of the left temporo-parieto-occipital white matter. Anticonvulsants were started on day 3 due to seizures. Follow-up brain CT on day 6 revealed progressing brain edema of the left hemisphere and a 5 mm midline-shift to the right. Due to extensive burning in the area of the left ear and increased intracranial pressure, the patient was further transferred to our trauma center for multidisciplinary management in our intensive care unit.

On day 7 after the injury multiplanar multisequence MRI of the brain with administration of a contrast agent was performed including axial SE T1-weighted (T1w), TSE T2w, FFE T2*w, EPI-DWI (with ADC-correlate) and sagittal 3-D FLAIR (with multiplanar reconstructions) sequences. Imaging data were acquired on a 1.5 T system (Philips Achieva, Eindhoven, The Netherlands). A 8-channel SENSE Head coil was used.

Firstly axial T1w contrast-enhanced and axial DWI MRI showed diffuse enhancement and cytotoxic edema in the central region (Fig. 1A–C).

Secondly axial T1w and axial T1w contrast-enhanced MRI demonstrated hyperintense lesions of the cortex in the left temporal gyri (Fig. 2A,B). In addition, cortex and subcortical white matter of the left temporal gyri were also hyperintense in T2w MRI and FLAIR, indicating ongoing edema in the cortex (Fig. 2C,D).

Thirdly axial DWI and ADC-correlate showed cytotoxic edema in the white matter of both hemispheres with the exception of the right frontotemporal region (Fig. 2E,F).

The clinical condition deteriorated throughout the next 8 days. The serum level of the neuron specific enolase (NSE) was markedly increased. The patient passed away 15 days after the accident without regaining consciousness. Any prior neurosurgical intervention was denied by the parents according to the patient's most probable wish.

3. Discussion

Electrical injuries are increasing and occur almost exclusively in young men as a result of occupational hazards and in children [4]. There are several pathways by which electricity may cause injury: thermal and nonthermal mechanisms as well as event-associated injuries, for example trauma and hypoxic injuries due to ventricular fibrillation [4].

The International Electromechanical Commission defines high voltage as above 1000 V for alternating current [5]. In general, high-voltage electric injury is thought to be more dangerous as low-voltage electric injury, because the former is associated with more severe tissue destruction and higher mortality. However, in low voltage electric trauma, immediate death may occur either from current induced-ventricular fibrillation or from respiratory arrest, and victims who survive may remain in a persistent vegetative state as a result of global hypoxia [6].

Our patient suffered cardiac arrest followed by successful cardiopulmonary resuscitation. MRI revealed typical findings for global hypoxia [7]: Axial T1w contrast-enhanced and axial DWI MRI showed diffuse enhancement and cytotoxic edema in the cortex of the central region (Fig. 1A–C).

Although the severity of thermal injuries is determined primarily by the voltage, low-voltage injuries can be just as dangerous as high voltage ones under certain conditions for instance in coexisting high electric field strength (70 A in our case). According to Joule's law, the power (heat) is defined as amperage squared times resistance [8]. However, in the majority of previously published reports information about the amperage is missing.

In our patient thermal injury was assumed due to the extensive burning in the area of the left ear and the sharply demarcated hypodense lesion of the cortex and the subcortical white matter of the left temporal lobe in the follow-up CT. In thermal injury, electricity generates heat, the amount of which

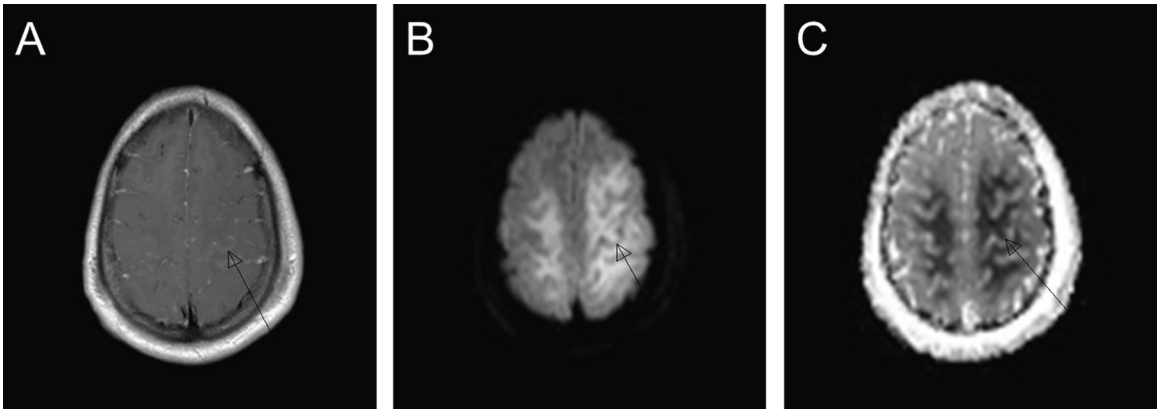

Fig. 1 – Cranial MRI of hypoxic injury.
Axial SE T1-weighted (TE 15 ms, TR 704 ms) contrast-enhanced (7 ml Gadovist i.v.) cMRI (A) shows diffuse enhancement in the central region. Axial diffusion-weighted (B-value 1000) imaging (B) and ADC-correlate (C) show cytotoxic edema in the central region.