Resuscitation from cardiac arrest secondary to electrical injury: description of a case

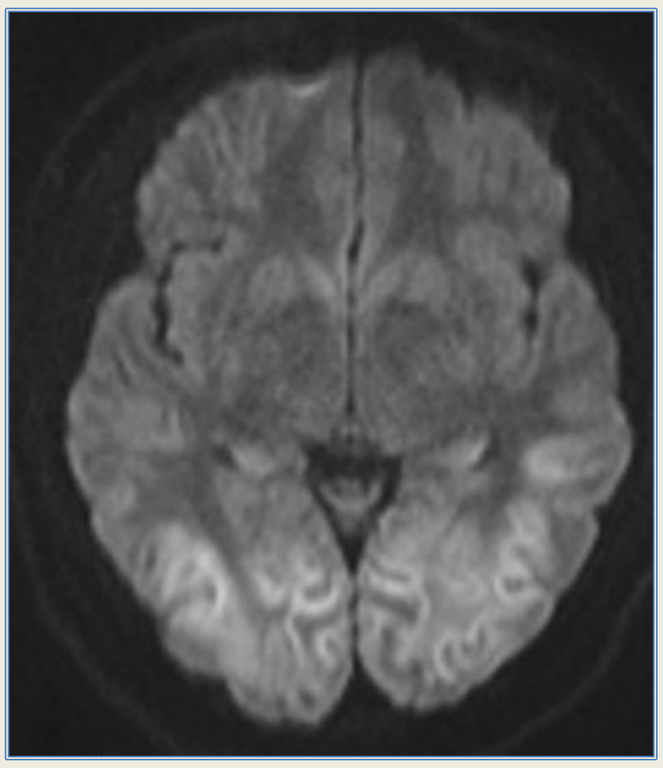
M. Rossi[1,2], C. Fassio[1], G. Bellaviti[1], A. Carolei[2], C. Pistarini[1]
[1] IRCCS Fondazione Salvatore Maugeri, Department of Neurorehabilitation (Pavia)
[2]Department of Neurology, University of L'Aquila (L'Aquila)

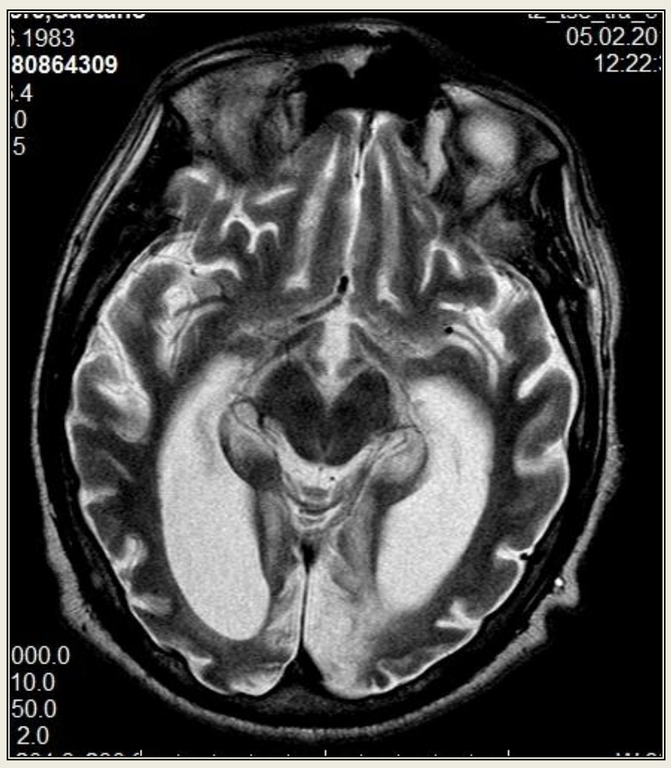
BACKGROUND

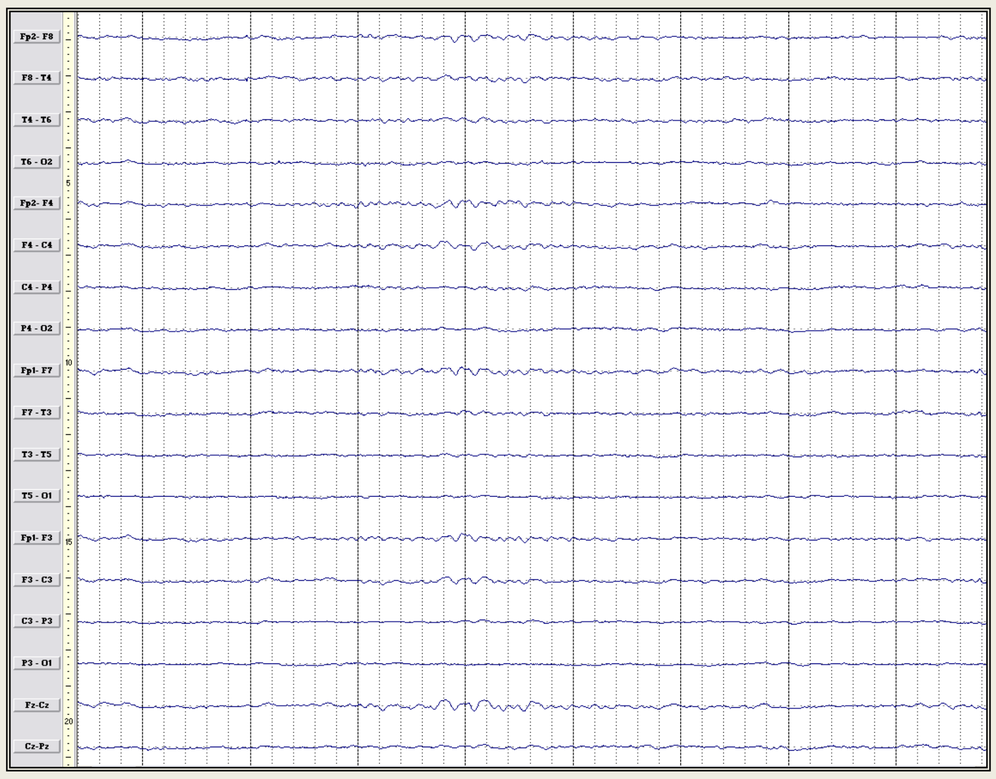
We describe unusual and severe sequelae after cardiac arrest secondary to electrocution, resistant to usual treatment.

CASE REPORT

A 30-year-old male grasped one end of a live electrical cable with his fishing rod Carbon. He received a severe electrical shock and he lost consciousness. Rescuers arrived immediately on the place and found the patient in cardiac arrest. They proceeded to defibrillation and ROSC (return of spontaneous circulation) in 20 minutes. On physical examination they found two small electrical burns on his right hand and foot. Brain MRI showed bilateral diffuse cortical damage, mostly in the parietal, occipital, and perirolandic areas. EEG showed diffuse slow brain waves without epileptiform activity. Clinically, the patient soon presented neurovegetative symptoms and hypertonia. After one month he was admitted to our Rehabilitation Unit with a GCS=6 (O2, M3, V1).







During hospitalization the patient progressively developed several neurological symptoms like dystonia, flexor spasms, exaggerated startle reaction, severe diffuse hypertonia, continuous shouts and crying. Dystonia affected initially the upper limb and the trunk, and then gradually became generalized. Moreover the symptoms were stimulus sensitive causing consistent problems in physiotherapy and hygiene. Repeated brain MRI showed symmetrical, bilateral cortical damage that affects calcarine region of both hemispheres; T2 sequences showed hyperintensities involving the white matter of the semioval centers and the lenticular nuclei. During the hospitalization various pharmacological treatments were prescribed, such as carbamazepine, clonazepam, levetiracetam at high doses, without improvement of any reported symptom. Because of progressive worsening of the symptoms, the infusion of midazolam in continuous was undertaken, and this was the only therapy to give benefit. After 5 months, with the implantation of the intrathecal baclofen infuser and the introduction of treatment with high dose of risperidone, valproate and pregabalin, it was possible to better control spasms, dystonia, and shoutsinterrupting the infusion of midazolam.

DISCUSSION AND CONCLUSION

Clinical manifestations of hypoxic brain injury may be widely different in relation to several factors including age, duration of hypoxia and possibly also the different causes of cardiac arrest. Not all cases of cerebral post-anoxic injury develop so complex neurological symptoms as in our case and we wonder if the electric event itself rather than only the hypoxic injury may have contributed to this unusual clinical presentation. Notably, while clinical manifestations were changing, MRI showed a progressive worsening of the damage in focal areas as the lenticular nuclei. Treatment was also difficult and not completely satisfactory.

References

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