Accidental High Voltage Electrocution: a Rare Neurosurgical Problem

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Summary

The authors describe a case of accidental electrocution from a high voltage current in a young worker, who was struck by the electric shock in the mid-occipital region.

The case is especially interesting due to the improbability of anyone surviving after receiving a shock of more than 1000 V., and to the development of bilateral parieto-occipital haemorrhagic infarction with spastic paraparesis, directly caused by the high voltage current and not indirectly by heat generation or secondary head trauma.

Keywords: Electrocution; intracerebral haemorrhage; electricity vasospasm.

Case Report

A 28 years old male was admitted to our hospital after being involved in an accident at work 8 days previously. While he was inside an electrical cabin he accidently touched a live high voltage cable, receiving a 3500 V. electric shock in the right parieto-occipital region. The current left the body through the right arm, which was resting on an iron girder. The patient lost consciousness immediately and collapsed to the ground, but without suffering further injuries, e.g. head trauma.

His fellow workers, who had witnessed the accident, immediately took him to a local hospital. Here he was admitted to the intensive care unit and remained comatose under assisted ventilation, for two days, presenting a marked quadraparesis.

The CT scan taken on admission, (Fig. 1) showed a right parieto-occipital hypodense area surrounding a small hyperdense cortical lesion. Standard x-rays of the skull, particularly of the occipital bone, proved negative.

Starting from the third day after admission, the patient began to gain consciousness, so that the breathing apparatus was removed. In the days following mobility of the arms improved bilaterally, so that the patient was able to help his right hand with his left.

On the fifth day he was able to speak again. However, the spastic paraparesis remained unchanged in spite of medical treatment with Mannitol 18 per cent 500 ml and Dexametasone 8 mg per day.

On admission to our hospital, 8 days after the accident, he was still unable to lift both legs off the bed, while flexion-extension of the feet was preserved. On his right arm there was a large skin defect with partial necrosis of the muscular structures (biceps and triceps) which were covered by free cutaneous grafts taken from the thighs. There was also a large cutaneous defect in the parieto-occipital region extending to the right retroauricular region. The skull was exposed over an area 6 x 8 cm, presenting a central area of osteolysis.

A second operation was carried out 15 days after the accident removing the necrosed bone. The dura mater appeared to be intact and no cranioplasty was made so as to avoid foreign body reactions. A large pedicle skin graft was taken from the dorsal region to cover the defect. The skin grafts healed satisfactorily.

One month after the accident, the patient was able to walk with support on both sides. The muscular strength of his left arm is normal, while on the right he is only able to move his hand, partly caused by the large muscle loss. A recent CT scan control (Fig. 2) shows the persistence of a small hypodense occipital area, while the haemorrhagic infarction has disappeared.

Discussion

The singularity of this case is due to its rarity, i.e. very few people survive an electric shock of more than 1000 V. Also, direct involvement of the cerebral structures is rare, depending upon the direct passage of the current from the head to the extremities. Generally speaking, in accidents of this type hand-to-hand or hand-to-feet discharges are much more common than the direct involvement of the brain.

In addition, the nervous system seems to be generally resistant to electrical injuries, as is confirmed by necropsy of such patients, where the autopsy examination of the brain is negative. In some cases of head-extremities electrocution, the finding of direct brain lesions at necropsy, such as coagulation and hardening of the brain, is induced by the high temperature caused by the electrical current.
I. Iob et al.: Accidental High Voltage Electrocution: a Rare Neurosurgical Problem

Fig. 1. CT scan on admission. Bilateral hypodense lesions in the parieto-occipital lobes (more extensive to the right), surrounding a hyperdense area. The latter probably represents a haemorrhagic infarction. No sign of osteolysis is detectable.

Fig. 2. CT scan control about 1 month after the accident. Persistence of occipital craniectomy. The right side is here at the left of the photograph a small hypodensity in the right parieto-occipital lobe. Signs of right.

This may happen if the time of exposure to the discharge is quite long, i.e. several seconds, whereas the mechanism of direct brain injury due to high temperature cannot take place when the patient collapses to the ground, interrupting the flow of the current, as happened in our case.

Another interesting point is that, when subdural or intracerebral haemorrhages are discovered at necropsy, they are usually thought to be the result of a direct head injury caused by a fall, especially if the victim has been working high above the ground, as is often the case with electrical linesmen.

In this case, however, there was no head injury, and it is more likely that the haemorrhagic lesions shown up by the CT scan were due to the effect of the electrical shock, probably by the rupture of small cortical veins.

On the other hand, about forty years ago, Stevenson in a work about the effects of electricity on brain tissue, observed diffuse haemorrhagic lesions distributed irregularly, focal demelination, ganglios degeneration and fragmentation of axons, as well as some areas of perivascular necrosis, which he interpreted as a consequence of the hypoxic damage due to vasospasm provoked by electrocution.

In the same period Echlin observed, from animal experiments that a passage of alternating 110 V. current through the brain of a cat for a period or 1–3 seconds induced a transient but marked vasospasm of the meningeal arteries.

From these autopsy and experimental findings the results of the CT scan in our case may be interpreted as areas of ischaemic brain necrosis surrounding some points of haemorrhagic infarction and/or breackage of the small vessels close to the entry zone of the current.