Transient quadripareisis after electric shock

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Abstract
A case of acute transient flaccid quadripareisis after a low voltage electric shock is reported. The patient recovered completely with in three days.

Case report
A 45 year old man was brought to the accident and emergency department after developing weakness of all four limbs. About six hours previously he had suffered a 220 volt alternating current electric shock when an iron pole which he was holding in both hands touched an electric wire. The shock caused him to fall to the ground. He soon recovered and started working again but about one hour later he noticed weakness in both lower limbs and difficulty in walking. The weakness gradually progressed to involve his upper limbs in the next few hours. There was no history of recent febrile illness or bleeding disorder. He did not complain of pain in the neck or muscle cramps.

On admission he was fully conscious and orientated and all vital signs were stable. There were no superficial electric burns. He had a flaccid quadripareisis with muscle power of grade I to II in all four limbs and loss of all sensations below C6 spinal level. Deep tendon reflexes were lost, plantar reflexes were not elicitable, and he had to be catheterised for retention of urine. All cranial nerves were intact. There was no local tenderness over the cervical spine and the neck movements were painless. Routine blood investigations (including serum electrolytes) and cerebrospinal fluid studies gave normal results. Radiography of the cervical spine did not show any bony or soft tissue abnormality.

Magnetic resonance imaging of the cervical spine was planned and an intravenous hydrocortisone sodium succinate infusion was started. Twenty four hours after admission he showed a remarkable improvement in muscle power and sensations in all four limbs. After 72 hours he had no residual neuropathy and went home walking. At one year he was well and had no symptoms attributable to the accident.

Discussion
Electric shock can affect any tissue of the body. It can cause transient or permanent damage to the central and peripheral nervous systems. The effect may be of acute onset or delayed for months or years.1-3 The cerebral involvement may present as hemiplegia or coma whereas spinal cord injury can manifest as transverse myelitis, quadriplegia,4,5 or motor neurone syndrome.6 Factors that determine the extent of electrical injury include the voltage of the current, the exposure time, the path of the current, and moisture at the contact areas.7 The sites of the entry and exit wounds may help in determining the path of the current. Electrical injury to the spinal cord occurs when the path of the current is either from arm to arm or from arm to leg.1 As skin is relatively resistant to electrical injury the underlying tissue damage may be masked.2 Electric shock acts either by generating local heat or by causing breakdown of the cell membrane and cell lysis.3 It can produce features of tissue anoxia such as oedema, perivascular infiltration, intravascular thrombosis, and arteritis.8

Our patient had no skin burns but the electric shock was able to cause alterations in the nerve cell physiology. We propose that the electric current produced changes in membrane potentials of nerve cells in the upper part of the spinal cord leading to cellular oedema that manifested as quadripareisis. Sudden insult to the cord led to a spinal shock syndrome. Since the shock was of low voltage with a short exposure time it failed to produce irreversible changes at the cellular level and the patient fully recovered. The steroid may have helped in his recovery by its membrane stabilising property and reducing oedema, but its role in such cases needs further investigation. The emergency physician treating such patients should be aware of the spectrum of clinical presentations and the consequences of electrical injuries.

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References