DELAYED NEUROLOGICAL MANIFESTATIONS SECONDARY TO ELECTRICAL INJURY—A CASE REPORT

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SYNOPSIS

A case of progressive spinal atrophy developing two years after an electrical injury is reported. Possible neurological syndromes arising from electrical injuries and likely mechanisms of delayed sequelae are reviewed.

INTRODUCTION

With the advent of electronics and the use of electrical appliances, injuries resulting from electrical mishaps have increased and aroused a greater awareness in medical and lay thinking. Such injuries alone have accounted for more than a thousand deaths yearly in the United States and another hundred in England. The first report of fatality was made in 1897. The immediate effects of electrical injuries are well known, but the delayed sequelae may be missed.

The occurrence of neurological manifestations after lightning strokes have been known for over a century. The first case of spinal amyotrophy after a lightning stroke was reported by Le Roy de Mericourt in 1860, in which he described the gradual onset of atrophic motor paralysis with slight sensory involvement in the right arm and thigh of a patient who experienced a lightning discharge conducted into his right hand. Since then reports of similar cases have appeared in the literature sporadically.

This report describes a case of progressive spinal atrophy developing twenty-four months after an electrical injury. Spinal atrophy secondary to electrical injury was first described by Koeppen and Panse in 1955.

CASE REPORT

In 1965, W. s/o T., a 26 year old Indian male laboratory assistant received an electrical shock when his right forearm came into contact with a live wire of estimated 220 volts. He was at the time standing bare-footed on the ground. He did not lose consciousness and felt physically well except for a sensation of numbness over the posterior and medial aspect of his right forearm and upper arm. This numbness lasted for about two days. Thereafter the patient had no further symptoms.

In 1967, i.e. two years after the electrical injury, while the patient was exercising with some weights he experienced a sudden sharp pain over the medial aspect of the ventral surface of the right forearm at the junction of the distal one-third and the proximal two-thirds associated with swelling over the same region. He also felt numb over the ulnar aspect of the right forearm and the palmar aspect of all the fingers. After two to three days the pain and numbness subsided. However, a few months later, the patient noticed that he could not adduct his right little finger. He also found that he was not as dextrous with his right hand as before in performing tasks like holding a pen, opening bottle caps or clipping finger nails. This disability increased progressively and two years later he could not sustain a good grip of a racket while playing badminton.

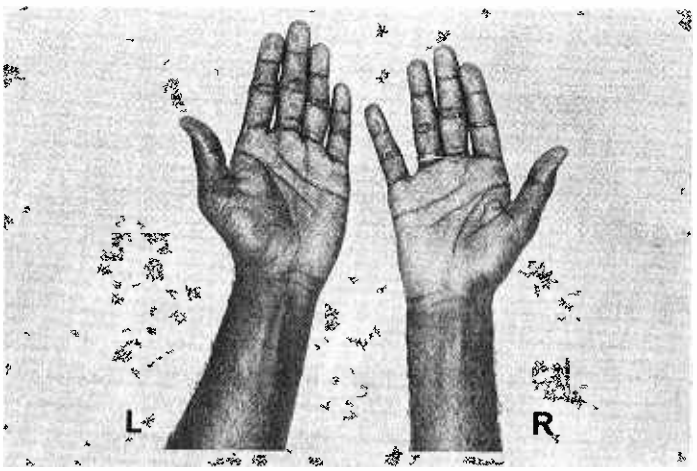

Fig. 1

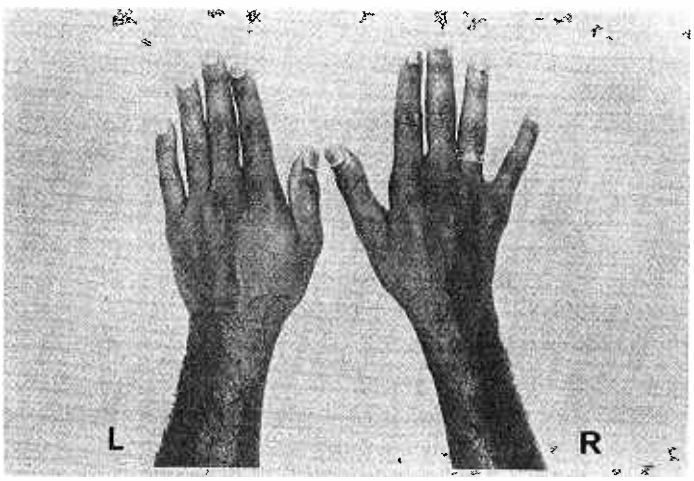

Fig. 2

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In 1971, six years after the electrical injury the patient sought medical attention at the University Hospital, Kuala Lumpur.

Physical examination then revealed a well built individual. His blood pressure measured 160/95 mm. Hg. in the right arm supine and the pulse was 76 per minute. There were no burn marks on the skin. Abnormal physical signs were found in the nervous system, all other systems were normal. The patient's mental status was normal, his cranial nerves were intact, and there was no cerebellar dysfunction.

In the right upper limb there was wasting of all the small muscles of the right hand and muscles on the ulnar aspect of the forearm. Motor power was decreased in the long flexors of the wrist and fingers, and in all the intrinsic muscles of the hand. There was decreased sensation to pin prick over the area supplied by the seventh and eighth cervical and the first thoracic nerves and a decreased sensation to temperature and light touch over the medial half of the palm. Vibration and position sense were preserved. No fasciculations were seen. No trophic changes were seen in the right arm. There were no abnormalities found in the other limbs. The tendon reflexes were present and equal on both sides except for the finger jerk which was absent on the right side.

Blood and urine examinations showed no abnormalities. The fasting blood sugar was 92 mg.%. A lumbar puncture yielded clear spinal fluid with an opening pressure of 120 mm. Hg. There was a quick rise and fall with Queckenstedt's test. The spinal fluid contained 4 rbc/ul, 2 wbc/ul, sugar of 66 mg.% and a protein of 25 mg.%, no organisms were present. The VDRL test of both cerebrospinal fluid and blood were negative. X-ray films of chest and cervical spine revealed no abnormality. A myelogram done showed free flow of myodil through the entire spinal canal.

Electromyographic studies done on the right abductor digiti minimi, the first dorsal interosseous and flexor carpi ulnaris showed no spontaneous activity at rest. On mild contraction many broad polyphasic motor units were seen. In addition many of them were giant motor unit potentials of more than 3,000 uv, some as high as 7,000 uv. Isolated interference patterns were seen in all 3 muscles on maximal contraction. Motor nerve conduction studies of the right ulnar nerve were within normal limits. The electromyogram findings are compatible with that seen with a chronic neuropathic lesion of muscle. With a normal conduction velocity and giant motor unit potentials the changes may indicate involvement of the anterior horn cells.

DISCUSSION

Lesions as a result of electrical injury could occur days to months later, (Panse, F., 1970). Various forms of spinal syndromes occur when electricity passes from trunk to limb or from limb to limb (Critchley, 1972). The type of spinal cord lesion depends on the path taken by the electric current through the body. In the patient reported above it was only possible to establish the site of entry of the current i.e. the right forearm. Charter (cited by Panse, 1970) described a case in which the current travelled from the right forearm to earth via the feet. Four months later spinal atrophic paralysis of the right arm developed involving the muscles supplied by the fifth to seventh cervical nerves and a sensory deficit corresponding to the same nerve supply but including the area supplied by the first thoracic nerve as well.

Farrel (1968) reported a case of a 67 year old man who was struck by a current which travelled from his right hand to the left foot. Twenty-four months after the injury, paraesthesiae, atrophy, weakness, fasciculations of the left thigh became apparent. Studies were consistent with neurogenic atrophy of this region.

Besides spinal syndromes other sequelae of electrical injury include cerebral syndromes (Critchley, 1934), peripheral nerve syndromes (Hartford, 1971), autonomic nervous system disorders (Panse, 1970), and psychiatric disorders (Critchley, 1934; Silversides, 1964).

The pathological changes in the nervous system as seen in autopsies of electrocuted humans and experimental animals include bleeding into subarachnoid spaces and the cord, patchy chromatolysis and demyelination, peculiar ballooning of myelin sheaths (Critchley, 1934), cavitations in the pia arachnoid, and swelling and softening of nervous tissues.

Attempts have been made by workers to explain the pathological changes. The electrolytic, mechanical and heating effects of electricity appear insufficient to account for the production of lesions in the central nervous system. Pritchard in 1934, postulated that in victims not grounded, induced electrostatic charges accumulate in the body tissues which then repel each other, causing sudden expansion with resultant decompression waves under the epidermis. He believed that similar forces operate in the nervous system, these being most marked in the fluid spaces, where rigidity or cohesion is minimal. However, it would be difficult to imagine that the body can accumulate such large quantities of induced charges to produce the lesions that have been mentioned earlier. Farrel and Starr in 1968 attempted to explain why neurological sequelae are delayed by comparing them to those observed after irradiation. They postulate that electricity like irradiation can induce protein changes in the ultra-structure of cells, especially the rapidly dividing endothelial cells of blood vessel walls, leading to cell death or altered cell behaviour.

The patient in this report developed progressive weakness, wasting and sensory deficit of the right hand and forearm, two years after sustaining an electrical injury. This is probably due to progressive spinal atrophy, a delayed sequelae of the injury. The

possibility of a traction injury to the right brachial plexus was considered unlikely owing to the lack of a history of trauma. Diabetic amyotrophy or radiculopathy were excluded on a normal fasting blood sugar and cerebro-spinal fluid examination. Radiological investigation did not reveal any extra-medullary lesions in the cervical region that could have accounted for the neurological changes seen in this patient.

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