



12-1-1999

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Recommended Citation

Lo, Cheng-Jen; Chuang, Yao-Chia; and Yang, Baii-Jia (1999) "Delayed Myelopathy After Electrical Burn : Report of Two Cases," *Rehabilitation Practice and Science*: Vol. 27: Iss. 4, Article 8.

DOI: <https://doi.org/10.6315/3005-3846.2086>

Available at: <https://rps.researchcommons.org/journal/vol27/iss4/8>

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Delayed Myelopathy After Electrical Burn :

Report of Two Cases

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Electrical burns dramatically affect the nervous system, muscles, bones, and other tissues within the human body. Myelopathy after an electrical burn has seldom been mentioned.

This article describes two cases of myelopathy admitted to our burn center after a high-voltage electrical burn incurred in the workplace, with symptoms onset around 16th and 4th day, respectively. The patients both experienced a course of deterioration, followed by a slow and partial recovery. The possible mechanisms and pathophysiology associated with electric burns are discussed. (J Rehab Med Assoc ROC 1999; 27(4): 235 – 240)

Key words: myelopathy, electrical burn

INTRODUCTION

Electrical burns have a high mortality rate, accounting for approximately 3% of all burn center admissions. In particular, damage to the nervous system is one of the most serious sequelae of electrical burns. Spinal cord damage, however, is a rare episode. In our burn center from 1993 to 1997, 103 patients were admitted due to electrical burn. Among which only one patient was diagnosed with myelopathy after an electrical burn during hospitalization. This article describes two patients who had spinal cord lesions, manifested with motor function loss, and paresthesia after an electrical burn injury.

CASE REPORT

Case I

A 31-year-old male patient suffered from a severe

electrical injury after coming into contact with a 22,000 V power cable while repairing high-tension equipment. Upon arrival to our emergency room, he was conscious, with the burnt area covering the neck, head, bilateral shoulders, as well as anterior side of bilateral arms and left lower limb. The entrance wound was on his right forearm, and a large blow-out exit wound appeared on his left eyebrow. A second or third degree electrical burn that covered 20% of the patient's total body surface area (TBSA) was diagnosed. No abnormal neurological signs and symptoms were observed.

On the 16th day after the electrical burn, he developed a symmetrical weakness in the bilateral lower limbs (hip extensor: 3-/5, knee extensor: 4/5, ankle dorsiflexor: 1/5, ankle plantar flexor: 1/5, trunk stability: poor, knee jerk: 3+, Babinski's sign: (+), Hoffman's sign: (+), sensory: hypoesthesia below C5 level, sphincter: intact). Although x-ray results of the lumbar spine were normal, MRI revealed a linearly high signal intensity

Submitted date: July, 6, 1999

Revised date: Sep. 10, 1999

Accepted date: Oct. 29, 1999

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(T2W1) over the anterior aspect of spinal cord from the bony level of C3 to C6 in the left paramedial portion (Fig.1). In addition, electromyography and nerve conduction velocity study were performed, indicating no evidence of peripheral neuropathy. Somatosensory evoked potentials (SSEP) study of median and tibial nerves revealed a prolonged N20, P37 as well as central conduction time (CCT) on both sides, a lesion above cervical spinal cord was suggested.

His muscle strength gradually improved in 2 months after injury, and the rehabilitation program with Mat exercise, balance training continued. Approximately 6 months after injury, he could walk with a quadricane. Three years after injury, the functional status was still the same as that of 6 months after injury.

Case II

A 44-year-old patient received a 10,000 V electrical burn while working and became unconscious. He regained consciousness upon arrival to our emergency room. The area of burnt skin involved the head, neck, left forearm, and left thigh. The entrance wound was his left forearm, and the exit wound appeared on his left anterior thigh. A second to third degree electrical burn covering 16% of TBSA was diagnosed. No abnormal neurological signs and symptoms were detected.

On the 4th day after the injury, he developed a symmetrical weakness and numbness in the bilateral lower limbs (hip flexor: 3/5, knee extensor: 3/5, ankle dorsiflexor: 3/5, ankle plantar flexor: 3/5, sensory: hypoesthesia below T10, knee jerk: 3+, ankle clonus(+), Hoffman's sign(+)). On the 6th day after injury, the patient had difficulty in defecating, and recovered about one month later. The motor function deteriorated and, by the 15th day, he developed bilateral complete paraplegia, with hypoesthesia below L4. Brain CT and spine CT (C7-L5) were normal. The MRI of spine revealed a focal area of high signal intensity (T2W1) in the spinal cord from the bony level of T7 to T10 on transverse image (Fig.2 and Fig.3); ischemic change was suspected. EMG and NCV studies revealed that consistent with an upper motor neuron lesion. SSEP study from bilateral posterior tibial nerves revealed prolongation of P37 latencies with borderline latencies of N20 and CCT from bilateral median nerves, indicating a lesion in the spinal cord.

Approximately one and a half months after the injury, mild motor function recovery was observed (hip flexor: 2/5, knee extensor: 2/5, ankle dorsiflexor: 1/5). A rehabilitation program with Mat exercise, strengthening of bilateral lower limbs, standing and ambulation training continued. He could walk fairly well using a walker during his last visit.

DISCUSSION

Three types of electrical burns ^[1] are A. arc burns or flash burns, as caused by the arc when it jumps the gap between the source and the conductor; B. electrothermal burns, which destroy the tissues along the pathway of the current through the tissues according to Ohm's law; C. flame burns, which are a combination of the above two types. The two cases reported herein are flame burns.

Severity of an electrical injury is related to the voltage and the amperage ^[2]. According to Ohm's law, with a constant voltage, the lower the resistance of a tissue implies a larger amperage it has. The resistance of human tissues, in decreasing order, is bone, fat, tendons, skin, muscles, blood vessels and nerves ^[3]. Other factors, e.g. moisture, may modify the impedance of the skin. Above a certain voltage, however, the resistance of the intact skin is negligible ^[2]. Low voltage electrical currents follow the pathway of the least resistance; high voltage currents above 1KV flow along the shortest paths as well ^[4]. Blood vessels and nervous tissue are the most conductive tissues. Coagulation necrosis and central nervous system (CNS) damage are thus the most common complications in electrical injuries ^[5,6]. A related investigation indicated that large electrical currents not only produce Joule heat, but also generate electrical fields, resulting in a breakdown of the cell membrane and ultimately causing lysis of the adjacent cells ^[7].

Neurological involvement can be categorized by either the time of onset, immediate or delayed; or by the anatomic site, peripheral or central. Immediate injury to the CNS results in various levels of change in consciousness, respiratory distress, and motor paralysis ^[8]. Delayed injury to the CNS is particularly devastating and has permanent sequelae ^[1,6,9-14]. Symptoms may appear in the first days after injury or may take as long as 3 years to evolve ^[1,15,16]. Spinal cord injury is the most common



Fig. 1. MRI of C spine (T2W1 on sagittal section): linear high signal intensity in the anterior aspect of spinal cord from the bony level of C3 to C6 in the left paramedial portion (as black arrows).

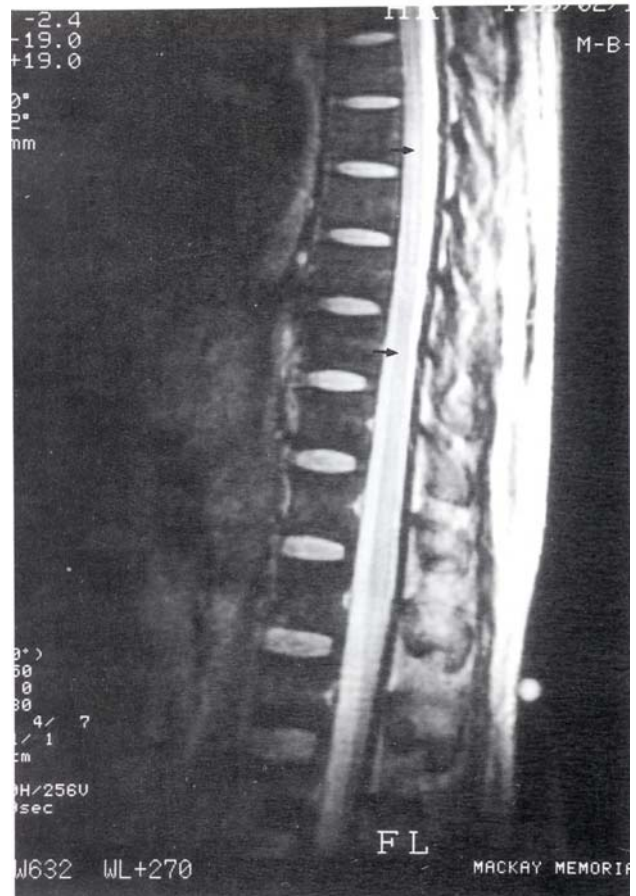


Fig. 2. MRI of T spine (T2W1 on sagittal section): linear high signal intensity in the spinal cord from the bony level of T7 to T10 (as black arrows).

permanent sequela, and the level of lesion is related to the pathway of the current. For instance, hand-to-hand current passage can affect the cervical cord between C4 and C8^[15]. Most clinicians confer that immediate neurological syndromes have a better prognosis for recovery, as compared with that of delayed neurological syndromes.

Levine et al. found only two cases with partial recovery out of forty patients with late myelopathy after electrical injuries^[16]. Christensen et al. described one case of late ascending paralysis with full recovery^[15]. While reviewing pertinent literature, Varghese et al. reported on an incidence of spinal cord injury involving

between 2 and 27% in various series, although 6% appeared to be an average figure in the larger series^[19]. In our two cases, delayed symptoms of the first case appeared on the 16th day, and gradual recovery was noted on the following 2 months. He could walk well with a quadricane about 6 months after injury. At that time, the functional improvement reached a plateau. Neurological symptoms were noted in the second case on 4th day, and functional improvement was noted about one and a half months after injury. More functional recovery may be obtained in the second case if the patient has longer period follow-up. Continued OPD visit was recommended.



Fig. 3. MRI of T spine (T2W1 on transverse section): focal area of high signal intensity in the spinal cord of bony level of T9 (as black arrow).

Possible mechanisms^[17] for spinal cord injury in electrical burn included: vascular damage, which caused hemorrhage due to rupture of the small vessels of the cord; thermal damage; radiation-like effect, which induced protein changes of tissues; electrostatic force which lead to violent tissue disruption.

Results of SSEP study in the two cases of this study reveal a lesion of the spinal cord. The potentials are transmitted predominantly via the proprioceptive pathways in the dorsal columns in the spinal cord, the medial lemniscal pathways of the brain stem and, to some extent, by the spinocerebellar pathways. The scalp potentials are generated by the primary somatosensory cortex. SSEP facilitate the detection or confirmation of lesions involving the proprioceptive and somatosensory pathways within the spinal cord, brainstem, and cortex. In our two cases, the first case showed prolonged N20, P37 as well as CCT on bilateral median and tibial nerves, indicating a lesion above the cervical spinal cord. The normal brain CT, MRI findings, and a series of physical examinations help confirm the precise lesion level on cervical cord. SSEP study of the second case revealed prolonged P37 latencies of bilateral tibial nerves, with

borderline latencies of N20 and CCT from bilateral median nerves, indicating a lesion in the spinal cord. The other imaging and physical findings confirm a precise lesion on the thoracic spinal cord.

Review of the early MRI findings on admission, high signal intensity in the spinal cord (T2W1) is noted in the two cases, implying change after electrical injury. Follow-up of MRI (3 years after injury in case I, and 3 months after injury in case II) indicated that no significant abnormal finding is noted in the two cases. This finding suggests resolution of ischemic change on imaging level, and it is compatible with the functional improvement in clinical observations. Comparing the MRI findings reveals that the most likely mechanism of myelopathy after electrical injury is vascular damage in our two cases.

Survivors of electrical burns may face multiple complications and must confront unique rehabilitation challenges. As with all burn-related injuries, rehabilitation should begin early in the acute stage. The patient should participate in his or her own program planning and goal setting. Early range of motion of involved area is advocated and the use of passive motion

machines has been proven effective in certain electrically burned patients ^[18]. Strength training in the four limbs, trunk are necessary to satisfy the new requirements of transfer and mobility. The amount of muscle tissue lost due to necrosis and debridement is occasionally so large that the normal function and rhythm of a joint may be impaired. The therapist must become aware of the extent of muscle damage when teaching early mobility because some movements may be awkward and stressful to joints. Overuse of the muscle and joint should be avoided in the early stage.

CONCLUSION

The two cases presented in this study revealed delayed injury to spinal cord, with symptom onset around the 16th and the 4th day, respectively. They both experienced a course of deterioration followed by partial recovery, 3-year follow-up in case I and 3 months in case II. This finding is compatible with other investigations. Imaging findings suggest that vascular damage is the most likely mechanism of delayed myelopathy in the two cases.

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電傷後的延遲型脊髓病變：兩病例報告

駱政仁 莊曜嘉 楊百嘉

馬偕紀念醫院復健科

電傷會影響人體的許多系統及組織，包括神經系統、肌肉、骨骼...，而電傷後的脊髓病變在文獻上的記錄，是相當少見的。

我們報告兩例高壓電傷造成的脊髓病變，這兩個例子都是在工作時不慎被高壓電電傷，之後分別於第十六天及第四天產生症狀。而且兩個例子都同樣經歷了一個惡化的過程，之後才又慢慢恢復，但恢復並不完全。在文章中，我們也將討論可能的致病轉機及生理病理變化。（中華復健醫誌 1999; 27(4): 235 - 240）

關鍵詞：脊髓病變 (myelopathy)，電傷 (electrical burn)