IMAGES

Acute Cerebral Infarction Caused by Cerebral Vasospasm Due to High-Voltage Electrical Injury

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A 44-year-old male patient was exposed to a 22,900 V wire while doing electrical work. The entry of electricity was thought to be at his right hand and the exit to be the left inguinal area. The affected area was approximately 45% of the area of the body surface (Fig. 1). He did not have any specific medical history. After the accident, he had a loss of consciousness for about five minutes. Neurologic examinations were normal. The brain computed tomography (CT) was normal. The laboratory test results showed SGOT 406 U/L, SGPT 127 U/L, CPK 23,253 IU/L, and CK-MB 3 15 ng/mL. The other laboratory findings were normal.

Fluid therapy was initiated for the 45% electrical injury. While measuring the urine output, the amount of fluids was adjusted to maintain 1 mL/kg/hr. The second day after the injury, the patient had two generalized tonic clonic type seizures for about one minute. In the laboratory test performed after the seizure, the potassium was elevated to 5.2 mmol/L, and the other electrolytes were within the normal range.

In the diffusion-weighted magnetic resonance imaging (MRI), multifocal scattered high-signal intensity was seen in the right frontoparietal subcortical regions (Fig. 2). The muscular strength of the left upper limb was motor grade 2 while the muscular strength of the left lower limb was motor grade 3. The muscular strength of the right upper and lower limbs was motor grade 4+. In consultation with the Department of Neurology, as a result of not finding an electrolyte imbalance that was causing the seizure except for an increase in potassium to 5.2 mmol/L, we concluded that the seizure was caused by cerebral edema or cerebral infarction. We diagnosed the patient with cerebral infarction by MRI. The echocardiography conducted on the third day indicated no abnormal conditions. In the transcranial doppler study (TCD), the right middle cerebral artery (MCA) blood flow velocity was 107 cm/sec and the left MCA blood

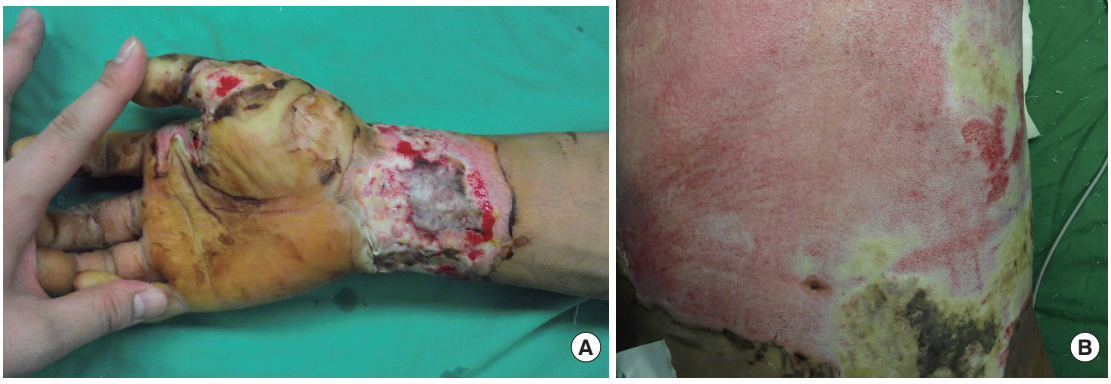

Fig. 1.
Burn wound of patient. Fourteen days after the burn (A) Eschars were found on his right wrist. (B) Eschar and granulation tissue were found on the abdomen and left inguinal area.

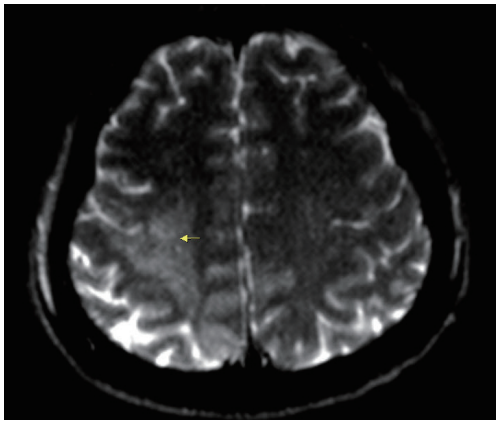

Fig. 2.
Brain magnetic resonance imaging (MRI) on the second day following the burn injury. In the diffusion-weighted MRI, multifocal scattered high-signal intensity was seen in both frontoparietal subcortical regions (yellow arrow). It was more distinct on the right side.

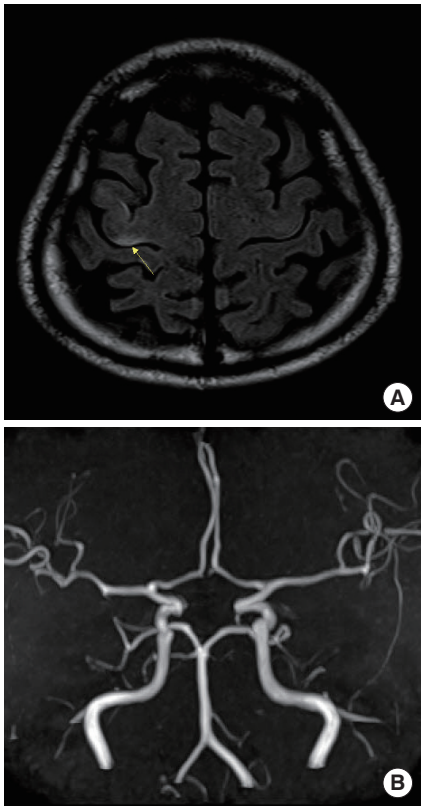

Fig. 3.
Brain magnetic resonance imaging (MRI) and magnetic resonance angiogram (MRA) three months following the burn injury. (A) Three months after the injury, in the follow-up brain MRI T2-weighted image, evidence that indicated ischemic lesion (high-signal intensity) was detected in the right frontoparietal subcortical region (yellow arrow). (B) MRA did not show any traces of angiostenosis.

flow velocity was 98 cm/sec, which were higher than the normal MCA blood flow velocity of approximately 60 cm/sec. Phenytoin (400 mg/day), valproic acid (2,400 mg/day), and 20% mannitol (100 mL) were administered to the patient. In addition, aspirin (100 mg/day) and clopidogrel (75 mg/day) continued to be administered. From three days before the surgery, when the patient was at higher risk of bleeding, low molecular heparin (5,700 U) was injected instead of aspirin and clopidogrel by subcutaneous injection until the day before the surgery. After the surgery, the aspirin and clopidogrel were administered again. The tendon was exposed in his right wrist, and flaps and skin grafts were employed for wound coverage. The other wounds were covered by skin grafts. Three months after the injury, the motor function of the left-side upper and lower limbs had recovered to above grade 4. In the follow-up brain MRI T2-weighted image, evidence that indicated an ischemic lesion (high-signal intensity) due to acute cerebral infarction was detected in the right frontoparietal subcortical region. The magnetic resonance angiogram did not show any traces of angiostenosis (Fig. 3).

Electrical injuries are categorized generally into thermal burns caused by direct contact or fire, arc burns, and direct electrical injury [1]. Numerous complications can develop in high-voltage electrical injury, ranging from wound infections to cardiac infarction. Fleury et al. [2] reported a case where a cardio-embolic cerebellar stroke occurred due to mitral valve rupture following high-voltage electrical injury, while Lim et al. [3] reported a case of chronically exposed dura following an electrical scalp burn.

Extensive research has already been conducted focusing on the mechanism of tissue damage caused by electrical injury. In case of a severe burn, decreased antithrombin, which inhibits the p38 mitogen-activated protein kinase pathway, can cause injury to myocardial cells [4]. Furthermore, deficiency of antithrombin could increase thrombogenicity because of its role as a natural anticoagulant. According to research on the lesion progression mechanism following electrical injury, a large volume of cytokines, such as prostaglandin F2a and thromboxane A2, is produced from the tunica vasculosa and blood platelets. Due to their hemagglutination reaction and vasospasm, the blood vessels are blocked and congested in some areas and tissues have ischemia [5]. However, the difference in this study is that cerebral infarction was not caused by cardiac-origin embolus but was developed by cerebral vasospasm caused by systematic action.

In the present case, we diagnosed cerebral infarction based on the decline in motor function and the brain MRI results. Based on the results of the TCD study, we could confirm that the cause was associated with cerebral vasospasm. It showed that the right MCA contraction period blood flow velocity was 107 cm/sec

and the left MCA contraction period blood flow velocity was 98 cm/sec, which was higher than the normal MCA contraction period blood flow velocity of approximately 60 cm/sec. To diagnose cerebral vasospasm, the blood flow velocity from the TCD should be above 120 cm/sec. As the test was performed three days after the injury, it could be assumed that cerebral vasospasm had been improving. Cardiac-origin embolus can occur in patients with atrial fibrillation or cardiac valve disease. However, in this case, the electrocardiography and echocardiography were normal. Therefore, the cause of cerebral infarction was not a cardiac-origin embolus. In addition, based on the entry and exit of the current pathway, the cause of brain damage was not direct electrical injury. However, we cannot completely exclude cerebral infarction caused by necrotic emboli, which is similar to MRI findings, since there are no applicable tests to determine whether the cause of cerebral infarction is necrotic emboli.

Nevertheless, based on examinations and comprehensive tests, we believe that the cause of cerebral infarction was vasospasm rather than necrotic emboli. In the present case, we experienced cerebral infarction caused by high-voltage electrical injury. Accordingly, active evaluation was needed for brain injury in the high-voltage electrical injury. In addition, thorough examinations and comprehensive tests were critical in the treatment of various complications, as they can be easy to miss because of severe external injuries by high-voltage electrical injury.

With a review of the relevant literature, this paper has discussed a case of cerebral infarction caused by cerebral vasospasm in a 44-year-old male patient who suffered from high-voltage electrical injury from contact with 22,900 V.

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Images

Internal Jugular Phlebectasia in a Patient with Facial Trauma

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Internal jugular phlebectasia (IJP) is defined as a fusiform dilatation of the internal jugular vein without tortuosity [1]. IJP is diagnosed on physical

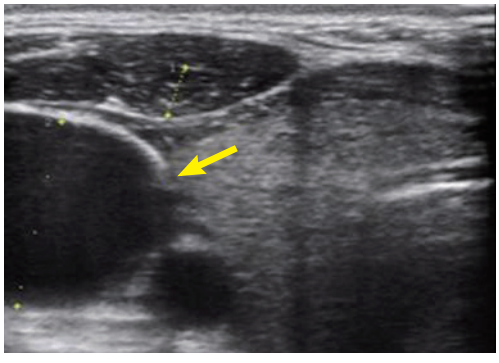

Fig. 1.
Ultrasonography of the right neck. The yellow arrow shows a marked dilatation of the right internal jugular vein without abnormalities of the adjacent structures.