
Acute Stroke in the Burn Patient

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A 3-year-old boy was brought to our hospital with 62% TBSA burn injury by boiled water. He received modern burn therapy, including early débridement and skin grafts. The patient discharged 70 days after being burned. Unfortunately, he sustained an acute right-sided hemiparesis 7 days after discharge. Magnetic resonance imaging revealed left middle cerebral artery territory infarction, and a magnetic resonance angiogram showed nonvisualization of left distal intra-cranial carotid artery and proximal middle cerebral artery. Thickened walls of these arteries indicated acute dissection. The patient received a follow-up magnetic resonance angiogram study 10 years later to check cerebral vascular lesions that may not have been apparent on the original image studies. It showed only persistently thickened left distal intra-cranial carotid artery and middle cerebral artery walls, indicative of old dissection. Therefore, carotid artery dissection must be considered as one cause of acute stroke in pediatric burn patient, even in the recovery phase of burn injury. (*J Burn Care Res* 2007; 28:351–354)

The altered physiological and homeostatic responses after burn injury make the victim susceptible to a variety of complications. Central nervous system complications include seizure, focal neurological deficits, and changes in consciousness and result mostly from dehydration, hypoxia, and metabolic disturbance.¹ Acute ischemic infarction in the acute phase of burn injury also has been reported.² However, stroke induced by acute carotid artery dissection in the recovery phase of burn injury has not been reported in the literature. We describe a case study of this injury and discuss the possible etiologies.

CASE REPORT

A 3-year-old boy presented to our emergency department on January 7, 1994, with a burn injury caused by boiling water on his trunk, both upper arms, legs, and the dorsums of both feet. Second-degree burns

constituted 45% TBSA, and third-degree burns constituted 17% TBSA. He received modern burn care, including optimal fluid resuscitation, early wound débridement, and several rounds of split-thickness skin grafting (STSG). Central venous catheters were introduced, via bilateral internal jugular veins and femoral veins, for fluid resuscitation and medication administration during the first 5 weeks. The central lines were removed before he was transferred to the general ward from the burn center. He was discharged 70 days after the accident.

The patient returned to the emergency department 7 days later with right lower-extremity weakness. His body temperature was increased to 38.2°C. On the next day, he presented additional symptoms, including mouth angle deviation to the left side, loss of right corneal reflex, and decreased power in his right trapezius muscle. A hypodense lesion was identified in his left frontal lobe and basal ganglia under computed tomography (CT scan). A Magnetic resonance imaging (MRI) study showed high signal in T1-weighted views and low signal in T2-weighted views of the left basal ganglia and frontal areas (Figure 1). These pictures confirmed left middle cerebral artery (MCA) territory infarction. The use of magnetic resonance angiography (MRA) demonstrated nonvisualization of the left internal carotid artery and branches of the MCA. Other laboratory examinations were normal. No cardiac anomalies were found under echocardiography, and carotid duplex confirmed the absence of

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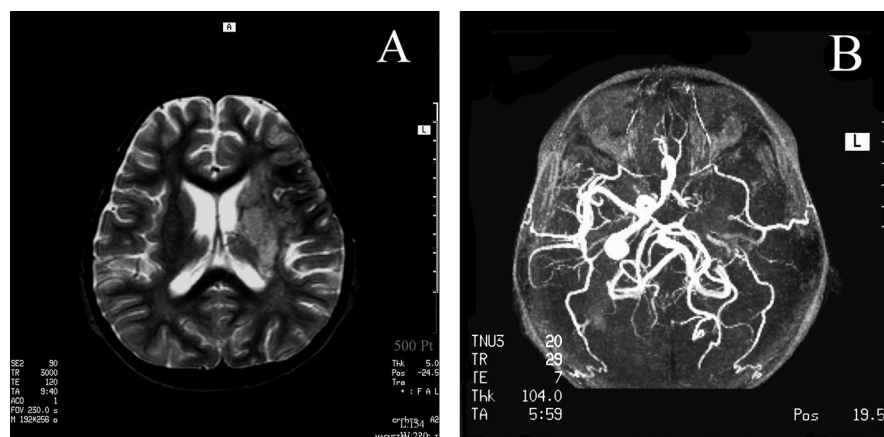


Figure 1. Magnetic resonance imaging shows increased signal on T2W image (A), compatible with acute ischemic infarction. The left intra-cranial carotid artery and middle cerebral artery are absent from the magnetic resonance angiography image (B).

thrombi or vegetations in bilateral extracranial carotid arteries. The patient had normal platelet number, a normal prothrombin time, and a normal partial thromboplastin time. He had neither bleeding problems during the acute burn period nor a family history of autoimmune diseases. The antinuclear antibody (ANA) titer was normal (1:40), and the protein C level also was in the normal range. The patient's family refused to pay for protein S and anti-thrombinIII examinations by themselves. The patient was discharged 18 days after admission when his condition was stabilized. He has been under regular follow-up at our out patient clinic.

Ten years later, the patient leads a normal life except sustaining grade IV/V muscle power (Medical Research Council grading system) on his right forearm and hand. He underwent another brain MRI

examination to rule out brain lesions that may not be apparent in its early stage. The MRI showed left MCA territory old infarction. The thickened wall of the distal intra-cranial carotid artery (ICA) and proximal MCA indicates old ICA dissection (Figure 2). There is no evidence of other vascular tumors, including Moyamoya-like lesions, in his brain.

DISCUSSION

Antoon, Andreasen, and Sevitt in the 1970s observed that some patients presented with neurological dysfunction after burn injury. They termed this phenomenon "burn encephalopathy" because the causes leading to the condition were still obscure at that time.^{1,3,4} Later hypoxia, hypovolemia, metabolic disturbances, infection, and vascular diseases

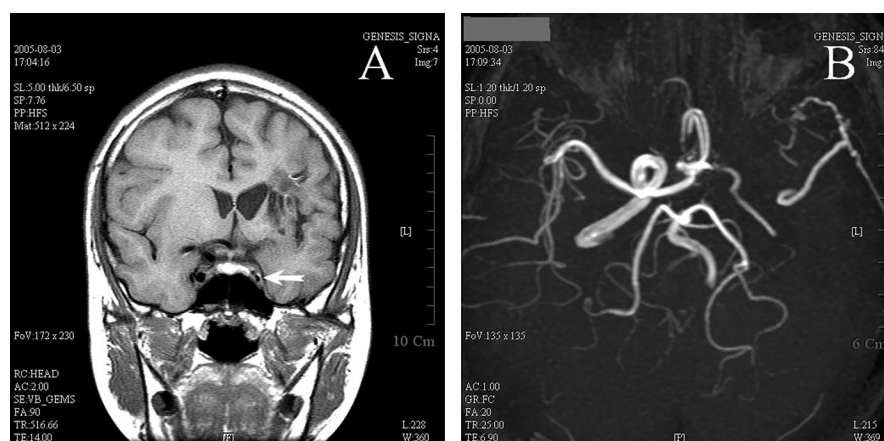


Figure 2. Magnetic resonance imaging 10 years after the tragedy shows the thickened wall of the left intra-cranial carotid artery (A, arrow), evidence of organization of previous thrombus formation. Magnetic resonance angiography reveals a small vessel indicating recanalization of left intra-cranial carotid artery and middle cerebral artery (MCA), but no branches from MCA can be seen. A collateral artery could be seen from the dural artery in the left MCA territory (B).

were reported to be the inducing factors of burn encephalopathy.³ Shahar et al² reported a 13 month-old girl suffering from acute ischemic infarction in the acute phase of burn injury. However, a report of brain infarction during the recovery phase of burn injury in children has not appeared in the literature before. Moreover, the relation between these two devastating events has never been discussed, either.

The reported incidence of stroke in children is approximately 7 per 100,000 children per year.⁵ Pediatric stroke often is multifactorial.⁶ The MRA examinations performed in search for the risk factors for stroke in this 3-year-old boy revealed only distal ICA and MCA occlusion. The MRI also demonstrated thickening of the wall of the distal ICA. These findings raised the initial suspicions of ICA dissection, vasculitis, or Moyamoya disease. Ten years later, the repeat MRI revealed no Moyamoya-like vascular collaterals or other vascular lesions. Persistently thickened walls of the distal ICA and MCA were noted, compatible with the previous image suggestive of vessel wall dissection. Given that the patient suffered only one episode of stroke, it would appear that the disease process is not progressive. We checked the protein C level for possible hereditary hypercoagulable state and serum ANA to rule out autoimmune disease. To further clarify his coagulable status, protein S and antithrombin III levels should be examined. For his normal platelet number, normal prothrombin time, partial thromboplastin time, protein C, and absent past history of bleeding tendency as well as the normal ANA level, coagulopathy or autoimmune diseases do not seem to be the cause in this instance.

Stroke in children frequently is associated with the complication of Varicella zoster. Askalan et al⁷ found that 31% of the children with acute ischemic infarction have had primary varicella zoster infection in the past year. The mechanism of varicella induced stroke is unknown, but virus-induced vasculitis was one accepted cause.⁸ The mean interval between infection and stroke onset was 5.2 months.⁷ Angiography often reveals focal stenosis or beading of the vessels.^{7,9} Although the locations of the infarction in this patient are in the basal ganglia and the frontal lobe, but beading does not feature in his MRA. Moreover, this 3-year-old boy had not suffered a varicella infection before the burn injury.

It is possible that central venous catheter insertion may also induce stroke. It is reported that accidental puncture of the carotid artery could cause thromboembolism of the intracranial arteries or carotid artery dissection, thus inducing stroke.^{10,11} However, according to the previous reports, the time between

puncture of the carotid artery and the onset of neurological symptoms would be several hours to 2 days.^{10,11} In our case report, this patient presented with right hemiparesis in the recovery phase of his burn injury long after the central venous catheter had been withdrawn. Furthermore, the dissection in this patient was intracranial. No lesion was observed in the extracranial carotid artery when we did a neck duplex study. Thus, central venous catheter induced stroke is not favored. There were neither signs of wound infection nor evidence of urinary tract infection or airway infection. The white blood cell count was not increased. The blood cultures were negative. The cardiac and carotid ultrasonography revealed no vegetations. Thus, septic emboli causing stroke is also not likely in this instance.

Carotid dissection in children generally is classified into spontaneous or traumatic dissection. They account for 10% to 25% of ischemic stroke in young and middle aged patients.¹² The patient has no identifiable history of neck trauma. In spontaneous carotid artery dissection, the intracranial arteries are involved more often than the extracranial arteries in children.¹³ However, the exact cause of spontaneous carotid artery dissection in children is unknown.¹³ Studies in adults have suggested that certain arteriopathy and a minor precipitating event, like neck hyperextension or rotation, may cause carotid artery dissection.^{12,13} The patient might have had a history of rapid neck extension or rotation that was overlooked just before the onset of neurological symptoms. Most cervicocerebral artery dissections are single events. The recurrence rate of infarction is 2% to 10%.^{12,13} The clinical condition of this patient and his image studies are all compatible with these aforementioned characteristics.

Stroke in children is always multifactorial.⁵ A triggering event that accompanies the hypercoagulable state may make the patient succumb to thromboembolic complications like pulmonary embolism, deep venous thrombosis, and even cerebral infarction. Anticoagulation or antiplatelet agents may be beneficial to the patient. Anticoagulant therapy has been reported to decrease recurrence rates in patients with cervicocerebral artery dissection.¹²⁻¹⁴ However, the beneficial effect in burn patients with stroke is difficult to demonstrate, given the rarity of this condition.

CONCLUSION

Acute ischemic stroke is a rare complication of burn injury, especially in the recovery phase. Numerous etiologies have been suggested for acute ischemic stroke. In a pediatric burn patient, carotid artery dis-

section is one possibility and must be considered as the cause of acute stroke.

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