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Temporary Neurological Deterioration Caused by Hyperperfusion after Extracranial-intracranial Bypass

—Case Report and Study of Cerebral Hemodynamics—

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Abstract

A 64-year-old female with occlusion of the left internal carotid artery (ICA) developed temporary neurological deterioration after superficial temporal artery to middle cerebral artery anastomosis. Preoperative single photon emission computed tomography (SPECT) showed marked reduction of the cerebral perfusion reserve in the left ICA territory. She suddenly developed aphasia 18 hours after operation. Follow-up SPECT revealed temporary hyperperfusion in the left temporal lobe, strongly suggesting that this unusual complication resulted from bypass flow into the brain tissue with chronic severe ischemia and impaired autoregulation.

Key words: acetazolamide, cerebral blood flow, cerebral vasoreactivity, extracranial-intracranial bypass, hyperperfusion syndrome

Introduction

Transient neurological deterioration may occur after extracranial-intracranial (EC/IC) bypass.[5,6] Neuroimaging and cerebral angiography may demonstrate specific associated abnormalities in regional cerebral blood flow (rCBF) and volume, but the pathogenesis remains unclear.

We describe a 64-year-old female who developed transient neurological deterioration after EC/IC bypass surgery for left internal carotid artery (ICA) occlusion, and discuss the pathogenesis based on preoperative cerebral hemodynamics.

Case Report

A 64-year-old female with a past history of hypertension suddenly experienced right hemiparesis and motor aphasia on June 26, 1991. She was admitted to another hospital, and cerebral infarction was diagnosed. Her symptoms gradually improved over a few days. She was admitted to our hospital on August 22, 1991, for further examination. Neurological examination demonstrated dysarthria, mild motor aphasia, mild right hemiparesis including facial nerve paresis, and hyper-reflexia of the right upper extremity.

Magnetic resonance (MR) images revealed cerebral infarction in the left middle cerebral artery-anterior cerebral artery (MCA-ACA) watershed zone (Fig. 1). Left carotid angiograms showed complete occlusion of the left ICA (Fig. 2). There was no significant occlusive lesion on the right carotid and vertebral angiograms. The rCBF was measured by the xenon-133 inhalation single photon emission computed tomography (^{133}Xe SPECT) on September 5, 1991. To evaluate the cerebral perfusion reserve, vasoreactivity was also measured 15 minutes after intravenous injection of 10 mg/kg acetazolamide, as described previously.[8,9] These studies revealed reduced rCBF and a "paradoxical" decrease after acetazolamide injection in the left ICA territory, strongly suggesting marked reduction of the cerebral perfusion reserve (Fig. 3).[8,9,13]

On September 18, 1991, she underwent superficial temporal artery (STA)-MCA double anastomosis to prevent further ischemic stroke. The frontal branch

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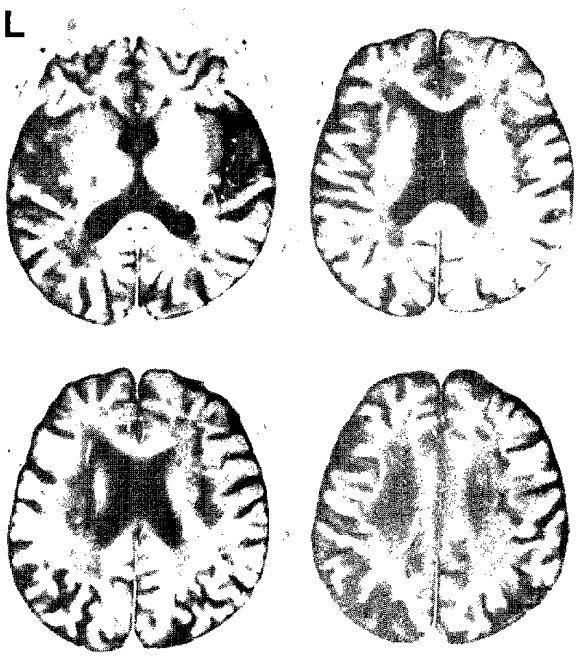

Fig. 1 Preoperative MR images (SE; TR 3000 msec, TE 80 msec), revealing cerebral infarction in the left MCA-ACA watershed zone.

Fig. 2 Preoperative left carotid angiogram, showing complete occlusion of the left ICA.

of the STA was anastomosed to the prefrontal artery, and the parietal branch of the STA to the middle temporal artery without difficulty. No marked neurological change was detected when she awoke from anesthesia, but total aphasia suddenly developed 18 hours after the operation. Her blood pressure was 160/90 mmHg and the anastomosed STA was well palpable. Computed tomographic (CT) scans showed mild swelling of the left temporal lobe. On the 2nd postoperative day, an abnormal rCBF increase in the left temporal lobe was observed by SPECT 15 minutes after intravenous injection of N-isopropyl-p-(I-123) iodoamphetamine (^{123}I-IMP) (Fig. 4 *upper*). Normal blood pressure was maintained with antihypertensive agent, and dehydration was induced with glycerol (800 ml/day) to resolve the cerebral edema. Her aphasia gradually improved, returning to the preoperative state within 1 week of the operation. The brain swelling in the left temporal lobe also disappeared in a few days. Hyperperfusion in the left temporal lobe was also resolved on ^{133}Xe SPECT scans 12 days after the operation (Fig. 4 *lower*). Left external carotid angiograms 30 days after the operation demonstrated good patency of the bypass arteries (Fig. 5).

She has experienced no further ischemic attack. Follow-up ^{133}Xe SPECT scans 3 months after operation also revealed normalization of the rCBF and reactivity to acetazolamide in the left ICA territory, suggesting improvement of the cerebral hemodynamics.

Study of Clinical Features

The clinical findings of our 34 patients with occlusion of the ICA were studied retrospectively. All patients had experienced transient ischemic attack or minor completed stroke in the ipsilateral hemispheres. rCBF was measured by ^{133}Xe SPECT (HEADTOME SET-031; Shimadzu Co., Kyoto), and calculated using the "sequential picture" method.[7] Each patient underwent two rCBF measurements before and 15 minutes after intravenous injection of 10 mg/kg acetazolamide. A circular region of interest of about 12.8 cm^2 was designated in the territory of the ipsilateral MCA. The regional cerebral vasoreactivity (rCVR) to acetazolamide was calculated as follows:

$$\text{rCVR (\%)} = (\text{acetazolamide rCBF} - \text{resting rCBF}) / \text{resting rCBF} \times 100$$

Normal control values of rCBF (mean ± SD 43.1 ± 3.0 ml/100 g/min) and rCVR (20.3 ± 5.3%) were obtained from eight healthy males, aged from 34 to 74 years (mean 50.4 yrs).[8]

Twelve patients had reduced rCBF and disturbed reactivity to acetazolamide in the ipsilateral ICA territory like the present case. Nine of these 12 patients underwent STA-MCA double anastomosis, but none

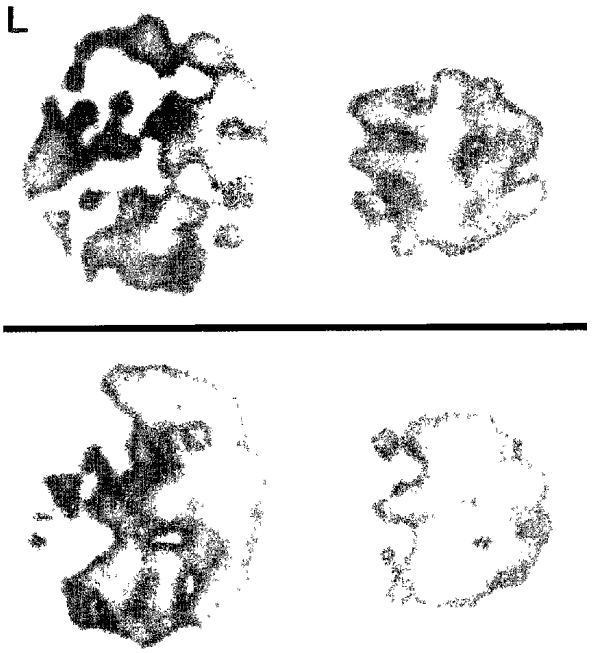

Fig. 3 Preoperative ^{133}Xe SPECT scans, demonstrating reduced rCBF and disturbed reactivity to acetazolamide in the territory of the left ICA. *upper*: Resting state, *lower*: after acetazolamide injection.

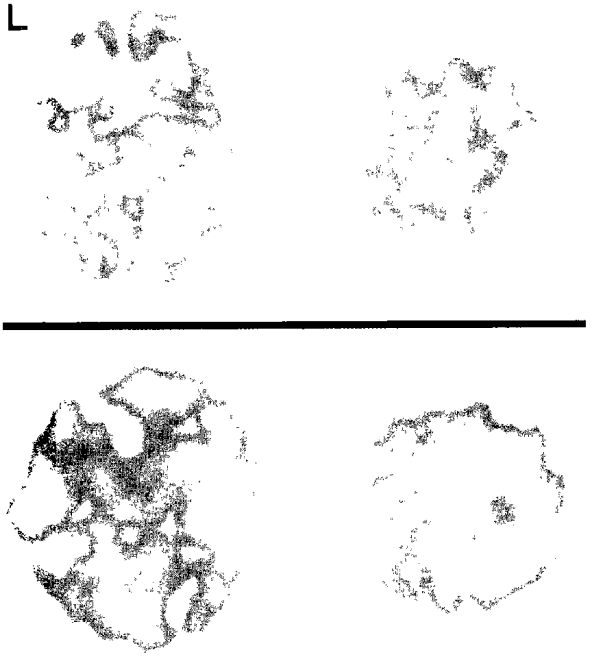

Fig. 4 *upper*: ^{123}I-IMP SPECT scans 2 days after operation, showing abnormal hyperemia in the left temporal lobe. *lower*: ^{133}Xe SPECT scans 12 days after operation, revealing resolution of abnormal hyperemia in the left temporal lobe.

experienced transient neurological deterioration after operations.

Comparison of the clinical findings of the present case with these nine previous patients showed no significant difference in age, clinical symptoms, rCBF, or reactivity to acetazolamide in the ipsilateral MCA territory (Table 1). The period between the cerebral ischemic attack and SPECT scan/operation was significantly longer in the present patient.

Discussion

The unusual complication of EC/IC bypass in our case was considered to result from postoperative hyperperfusion in the operated hemisphere, because 1) marked reduction of the cerebral perfusion reserve was shown by preoperative rCBF studies (decreased rCBF and disturbed reactivity to acetazolamide in the ipsilateral ICA territory),[8,9,13] 2) transient edema and hyperperfusion occurred in the ipsilateral temporal lobe, and 3) the temporary neurological deterioration (global aphasia) gradually resolved over 1 week. This phenomenon could not be attributed to temporary occlusion of the cortical

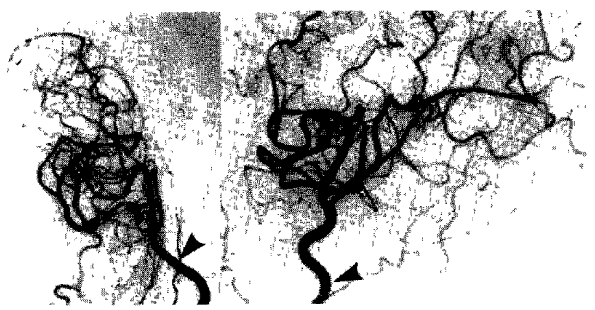

Fig. 5 Left external carotid angiograms 30 days after operation, showing good filling of the MCA territory through the patent STA graft (*arrowhead*).

branches and general anesthesia, because her neurological signs remained unchanged when she awoke from anesthesia, only deteriorating 18 hours later.

Similar complications have been reported previously. Heros *et al.*[5] reported five patients who developed temporary neurological deterioration after STA-MCA anastomosis, possibly resulting from post-

Table 1 Clinical findings in the present patient and nine similar patients but without postoperative hyperperfusion

	Present patient	Other nine patients
Sex	female	males
Age (yrs)	64	62.9 ± 6.1
Clinical diagnosis	minor completed stroke	TIA, minor completed stroke
Period (days)		
onset-SPECT scan	70*	33.7 ± 13.1
onset-operation	83*	50.4 ± 15.4
rCBF (ml/100 g/min)	35	31.4 ± 2.4
rCVR (%)	−7.7	−6.8 ± 6.6

*Longer than mean + 2 SD for other nine patients.
TIA: transient ischemic attack.

operative hyperperfusion because no definite abnormality was seen on CT scans and cerebral angiograms. Higashi *et al.*[6] also reported a patient who experienced postoperative neurological deterioration lasting for 1 week. A transient abnormal increase in rCBF and regional cerebral blood volume was observed in the operated hemisphere, suggesting that this phenomenon was caused by postoperative hyperperfusion into the ischemic brain tissue. In our patient, transient hyperperfusion could have resulted from bypass flow into the ischemic brain tissue with disturbed autoregulation. However, why this neurological deterioration occurred 18 hours after operation is obscure.

A higher incidence of postoperative hyperperfusion occurs after carotid endarterectomy, especially in patients with high-grade ICA stenosis.[1,2,10-12] The most frequent complications are brain swelling and intracerebral hemorrhage. Many investigators have suggested that postoperative vasoparalysis could induce this hyperperfusion syndrome in regions with impaired autoregulation due to critical reduction of perfusion pressure. This condition has been described as "unilateral normal perfusion breakthrough."[1,10]

Experimental studies show medial hypertrophy, loss of contractile strength, and decreased resistance in the arterioles of chronically underperfused vascular bed.[3,4] Hypercellularity and proliferation of endothelium and smooth muscle occur in the small artery walls of patients who develop cerebral edema and hemorrhage after carotid endarterectomy.[2] These findings suggest that such histological changes in the cerebral resistance vessels could cause hyperperfusion syndrome after cerebral vascular reconstruction.

In contrast to carotid endarterectomy, STA-MCA anastomosis rarely causes transient hyperperfusion syndrome, probably because the bypass flow through the STA is rather less than the ICA flow.[5,6] However, our case suggests that chronic severe ischemia may be important in the pathogenesis of postoperative hyperperfusion syndrome.

Hyperperfusion syndrome after STA-MCA anastomosis is rare, but careful perioperative management is required to prevent this complication in patients with chronic severe hemodynamic compromise, as well as after carotid endarterectomy. In particular, the systemic arterial pressure must be strictly controlled in the normal or lower range.[5,6] Hyperperfusion syndrome as well as postoperative ischemia must be considered in patients manifesting neurological deterioration after STA-MCA anastomosis, because the required treatments are precisely the opposite.

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