

Augmentation of the Patency of an Extracranial-Intracranial Bypass Accompanied by the Occlusion of an Intracranial Stenotic Lesion

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We describe a case that showed augmention of the superficial temporal artery (STA) pedicle's patency 15 months after extracranial to intracranial (EC-IC) bypass surgery for a carotid artery occlusion with contralateral intracranial internal carotid artery stenosis. It is rare that meager patency of the STA pedicle in the early postoperative angiogram can be become well augmented with time where most branches of the middle cerebral artery (MCA) are robustly filled with blood from the STA. A 28-year-old woman with a history of a previous left hemispheric stroke presented with slurred speech after several bouts of seizure. Magnetic resonance imaging showed a new infarct on the right hemisphere in addition to an old infarct on the left hemisphere. Carotid angiography revealed stenosis of the right carotid siphon and occlusion of the left carotid artery. The patient underwent EC-IC bypass on the right side. Even though the early postoperative angiogram showed meager filling of MCA with no significant stenotic lesion change, a subsequent angiogram taken 15 months later, demonstrated a widely patent STA pedicle with occlusion of the previous intracranial stenotic lesion. Selected cases with an inaccessible intracranial stenotic lesion can benefit from EC-IC bypass surgery; however, its clear indication should first be established.

KEY WORDS: Extracranial-intracranial bypass · Stenosis · Cerebrovascular disease.

Introduction

cranial (EC-IC) arterial anastomosis was first introduced by Yasargil and Donaghy in 1967. After the publication of an international study on EC-IC bypass surgery in 1985, this approach was thought to be abandoned as a treatment of symptomatic carotid artery occlusion¹⁴. However, this was greatly criticized by many authors^{1,3,5-7,13)}. It was argured that the study failed to analyze separately a subgroup of patients with reduced cerebral perfusion pressure in whom surgical revascularization might have been more beneficial. Moreover, even though the number of bypass procedures has declined, the procedure is useful in Moyamoya disease and for planned ligation of the internal carotid artery (ICA) for an unclippable aneurysm or

skull base tumor. It has been reported that EC-IC bypass surgery

is also beneficial in selected patients with ICA occlusion with a

known poor cerebrovascular reserve^{11,12)}. We present a case of

rebral revascularization through an extracranial to intra-

augmented patency of superficial temporal artery (STA) pedicle 15 months after EC-IC bypass surgery for a carotid artery occlusion with contralateral intracranial internal carotid artery stenosis, which progressed to occlusion in follow-up angiograms.

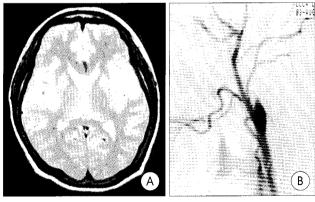


Fig. 1. A: T2—weighted magnetic resonance image showing focal infarction in the left frontal cortex. B: Left carotid angiogram showing marked stenosis of the cervical internal carotid artery.

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Case Report

Asyear-old woman presented with a sudden onset of slurred speech. She experienced two bouts of tonic and clonic convulsion, preceded by numbness of right fingers 20 days prior to presentation. She had experienced a left hemispheric stroke 5 years ago and resulted in mild right hemiparesis and aphasia. MRI at that time revealed a hemorrhagic infarct in the left frontal cortex (Fig. 1), brain SPECT revealed diminished perfusion in the same region. Carotid angiography showed marked stenosis of the left cervical internal carotid artery and that the left cerebral hemisphere was perfused via the collateral circulation of the posterior communicating artery. She had experienced significant neurological improvement and had been treated with oral hypoglycemic agents and antiplatelets.

On admission, she was alert and well oriented. Her neurological examination was nonspecific, except for mild dysarthria. Blood chemistry showed hyperlipidemia (total cholesterol: 357mg/dL, triglyceride: 963mg/dL) and a controlled sugar level. Brain MRI demonstrated a bifrontal infarct and

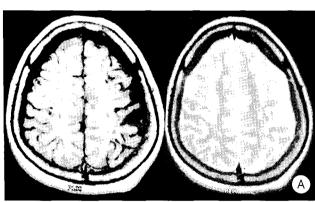




Fig. 2. A: Brain magnetic resonance image demonstrating a bifrontal infarct and an old infarct in the left fronto—parietal region. B: A cerebral angiogram demonstrating complete occlusion of the left internal carotid artery and stenosis of the right carotid siphon.

an old infarct in the left fronto-parietal region. A cerebral angiogram demonstrated complete occlusion of the left internal carotid artery and stenosis of the right carotid siphon (Fig. 2). Brain SPECT showed perfusion defect in bilateral hemispheres. Right ECIC bypass surgery was performed to pr



Fig. 3. Initial postoperative external carotid angiogram revealing faint middle cerebral artery filling through the superficial temporal artery (14 days after operation).

event additional infarction and her postoperative course was uneventful. Postoperative external carotid angiogram showed faint filling of the middle cerebral artery (MCA) through the bypassed superficial temporal artery after 14 days (Fig. 3). Good pulsation over the bypassed artery was noted during regular visits. A subsequent angiogram, 15 months after operation, showed that the previous stenotic lesion had become occluded and that the patency of STA pedicle had been augmented to extent that the main branches of the MCA were well perfused (Fig. 4). She has remained symptom-free for 5 years.

Discussion

The role of EC-IC bypass for symptomatic inaccessible internal carotid stenotic lesion remains uncertain. The pathophysiology of cerebrovascular ischemia has been exte-

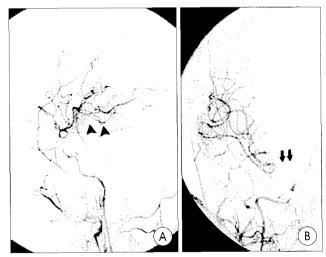


Fig. 4. A, B: Subsequent carotid angiogram (15months after operation) showing that the previous stenotic lesion had became occluded (black arrow) and that the patency of the superficial temporal artery became greatly augmented that main branches of middle cerebral artery were well perfused (arrowhead).

nsively studied and sophisticated physiologic evaluation of cerebrovascular insufficiency has been used in clinical practice. It has become clear that many patients benefit from cerebrovascular revascularization as it reduces the risk of stroke based on careful selection. Spetzler¹¹⁾ described current indications for bypass surgery as follows: 1) Patient who has failed maximal medical therapy, 2) lesion that cannot be managed by endovascular or direct surgical means, 3) patient who has a poor cerebrovascular reserve as demonstrated by xenon-enhanced computed tomography or positron emission tomography, and where patient's symptoms correlate with radiological findings. In the present case, the patient developed a new bifrontal infarct on brain MRI despite antiplatelets and oral hypoglycemic agents. Her cerebral angiogram showed occlusion of the left internal carotid artery and stenosis of the contralateral carotid artery that had a normal diameter 5 years previously, and brain SPECT revealed a bifrontal perfusion defect. Angioplasty may have been a suitable treatment modality in this case, but its role and the possibility of restenosis remain controversial. Thus, we decided to perform bypass surgery on the right side because the left hemisphere was supplied by collateral circulation through the posterior communication artery and because perfusion defect was more prominent on the right side.

The natural structural evolution of carotid siphon stenosis is not well-known. Bauer et al.⁴⁾ reported that 11 of 13 patients with intracranial ICA stenosis remained stable over a 2-year period, whereas stenosis worsened or progressed into occlusion in the other two patients.

The results of EC-IC bypass for a stenotic lesion have not been always convincing. One series of EC-IC bypass for carotid stenosis showed a better outcome than that seen in natural evoluation and surgical benefit was demonstrable. Recently Nussbaum et al.¹¹⁾ reported good results from EC-IC bypass surgery in 20 patients including 2 supraclinoid internal carotid stenosis cases, but they didn't describe the follow-up angiographic findings. On the other hand, no difference in outcome was observed between the medical and surgical treatment of patients with severe inaccessible carotid stenosis in an international randomized trial¹⁵⁾. Some intracranial stenotic lesions that were converted to occlusion associated with ischemic symptoms after EC-IC bypass surgery have been reported^{2,8)}. Awad et al.20 observed significant change of a stenotic lesion to occlusion or improvement in 9 of 18 cases after EC-IC bypass. One pattern of change involved stenotic lesion occlusion that occurred within several weeks of operation and was associated with ischemic complications, though all grafts remained patent. The other pattern was of improvement in the degree of stenosis or complete resolution of the stenotic lesion. Gumerlock et al.89 documented that stenotic vessels became occluded with new symptoms after bypass surgery in 4 of 10 patients

with ICA stenosis. The majority of these changes occurred within 6 months of EC-IC bypass surgery. Even though these changes may have been due to natural evolution, bypass surgery itself might play a role in stenotic lesion changes in view of the frequency of these changes and their rapidity. It was assumed that EC-IC bypass could modify hemodynamic parameters across a stenotic lesion and thus predispose them to improvement or worsening^{2,8,10)}.

In the present case, early post-operative angiography showed poor cortical filling through the STA and no significant change in stenotic lesion. However, follow-up angiography after 15 years revealed occlusion at the previous stenotic site and robust filling of MCA branches from the STA. It is not uncommon to observe a STA pedicle becoming wider with time than that shown in early postoperative angiograms after bypass surgery. But it was unexpected to see the patency of a pedicle to become greatly augmented that most branches of the MCA are filled from the pedicle 15 months after surgery, especially when its patency was in doubt based on the early postoperative angiogram. Awad's series involved symptomatic occlusion of a stenotic lesion in 4 of 18 cases that underwent EC-IC bypass for intracranial arterial stenosis, but unfortunately they only performed early postoperative angiography in these 3 cases²⁾. Therefore, long-term postoperative sequential changes of stenotic lesions and bypasses could not been evaluated in these cases. Nonetheless, it was observed that most symptomatic occlusions of stenotic lesions occurred during the early postoperative period.

Poor cortical filling on early postoperative angiograms, as in the present case, might be due to slight blood pressure differences between donor and recipient vessel, spasm of the vessels and acute thrombosis at the anastomosis site. The lack of a significant stenotic lesion change on the early postoperative angiogram suggested that progression of the stenotic lesion was not caused by an early effect of surgical intervention. However, early poor STA patency might have slightly affected with hemodynamic parameters and thus caused stenosis progression. The progression of a carotid stenotic lesion might encourage bypass patency and a widely patent bypass could protect against ischemia. Two possible explanations have been speculated in our case. First, total cerebral flow may have not been sufficiently compromised to promote the flow of STA pedicle. Second, bypass may have been small and it may have taken longer time to grow.

Conclusion

We conclude that selected cases with an inaccessible intracranial stenotic lesion could benefit from EC-IC bypass surgery, but its clear indications must first be established.

References

- 1. Ausman JI, Diaz FG: Critique of the extracranial-intracranial bypass study. Surg Neurol 26: 218-221, 1986
- Awad IA, Furlan AJ, Little JR: Changes in intracranial stenotic lesions after extracranial-intracranial bypass surgery. J Neurosurg 60: 771-776, 1984
- Awad IA, Spetzler RF: Extracranial-intracranial bypass surgery: a critical analysis in light of the international cooperative study. Neurosurgery 19: 655-664, 1986
- Bauer RB, Boulos RS, Meyer JS: Natural history and surgical treatment of occlusive cerebrovascular disease evaluated by serial arteriography. AJR 104: 1-17, 1968
- Day AL, Rhoton AL Jr, Little JR: The Extracranial-intracranial bypass study. Surg Neurol 26: 222-226, 1986
- Goldring S, Zervas N, Langfitt T: The extracranial-intracranial bypass study. A report of the committee appointed by the American Association of Neurological Surgeons to examine the study. N Engl J Med 316: 817-820, 1987
- Grubb RL Jr, Powers WJ: Risks of stroke and current indications for cerebral revascularization in patients with carotid occlusion. Neurosurg Clin N Am 12: 473-487, 2001
- 8. Gumerlock MK, Ono H, Neuwelt EA: Can a patient extractanial-intracranial bypass provoke the conversion of an intracranial arterial stenosis

- to a symptomatic occlusion? Neurosurgery 12:391-400, 1983
- Link MJ, Herzog GA: Extracranial-Intracranial Bypass -Technical achievement, controversies and indications in the treatment of ischemic stroke. Jacksonville medicine 49: 505-509, 1998
- Moon KY, Kang SD, Kim YS: Transcranial Doppler examination following EIAB in patients with hemodynamic cerebral ischemia. J Korean Neurosurg Soc 35: 273-277, 2004
- Nussbaum ES, Erickson DL: Extracranial-Intracranial bypass for ischemic cerebrovascular disease refractory to maximal medical therapy. Neurosurgery 46: 37-43, 2000
- Schmiedek P, Piepgras A, Leinsinger G, Kirsch CM, Einhaupl K: Improvement of cardiovascular reserve capacity by EC-IC arterial bypass surgery in patients with ICA occlusion and hemodynamic ischemia. J Neurosurg 81: 236-244, 1994
- Sundt TM Jr: Was the international randomized trial of extracranialintracranial arterial bypass representative of the population at risk? N Engl J Med 316: 814-816, 1987
- The EC/IC Bypass Study Group: Failure of extracranial- intracranial bypass to reduce the risk of ischemic stroke. Results of an international randomized trial. N Engl J Med 313: 1191-2000, 1985
- Weinstein PR, Baena RR, Chater NL: Results of extracranial-intracranial arterial bypass for intracranial internal carotid artery stenosis: review of 105 cases. Neurosurgery 15: 787-794, 1984