Acta Neurochir (Wien) (2006) 148: 1001–1004
DOI 10.1007/s00701-006-0851-0

Acta Neurochirurgica
Printed in Austria

Case Report

Delayed re-opening of an STA-MCA bypass graft

I. F. Dunn, D. B. Ellegala, and **A. L. Day**

Cerebrovascular Center, Department of Neurosurgery, Brigham and Women's Hospital, Harvard Medical School, Boston, USA

Received May 17, 2005; accepted May 31, 2006; Published online July 17, 2006

Summary

We describe the case of a 47-year-old female with symptomatic right MCA stenosis who had undergone cerebral revascularization through a superficial temporal artery-to-middle cerebral artery (STA-MCA) bypass. Despite clear patency in the operating room, post-operative angiography showed no flow in the bypass. Her ipsilateral internal carotid artery (ICA) was widely patent. She remained asymptomatic and follow-up angiography four years later showed a widely patent bypass graft in the setting of critical stenosis of the ipsilateral ICA. That the graft was found opened up and supplying the hemisphere was presumably stimulated by an increased "demand" and flow gradient promoting its patency.

Keywords: Cerebral revascularization; hemispheric ischemia; STA-MCA bypass.

Introduction

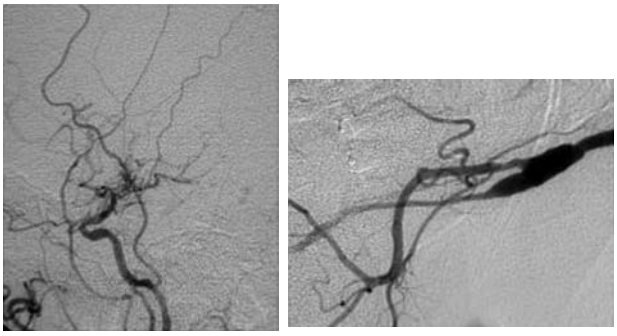
Superficial temporal artery-to-middle cerebral artery (STA-MCA) bypass is an important and effective technique considered by cerebrovascular surgeons whenever a need for cerebral revascularization is anticipated [13]. Indications for this particular technique generally include symptomatic ischemia associated with inadequate cerebral perfusion and exhausted cerebrovascular reserve (secondary to cervical or intracranial vessel occlusion or stenosis), and to prevent ischemia during procedures such as cranial base tumor resection or complex cerebral aneurysm surgery, during which a large vessel sacrifice is anticipated [9, 10, 12].

Once performed successfully, a patent bypass is expected to produce alterations in blood flow in the recipient vessel territory. The graft itself often accommodates for its new role by dilation and hypertrophy over time, providing a "growing" source of collateral circulation into the treated region [2, 11]. Nearby vessels that formerly supplied that region can also be affected. Dilation of neighboring leptomeningeal collaterals may recede as the need for such channels diminishes. Progression of proximal vessel stenosis to occlusion after STA-MCA bypass has also been described [1, 3, 6, 7] and is likely precipitated by a significant drop in the relative cerebral perfusion pressure (rCPP) across the stenosis after the distal bypass has been installed. In this case report, we describe an interesting case where an STA-MCA bypass was done as treatment of an MCA stenosis, immediately following which the graft was found to be occluded. Several years later, in the setting of progressive ipsilateral internal carotid artery stenosis, the graft was found opened up and supplying the hemisphere. Several possible explanations for this re-opening are discussed.

Case report

A 47-year-old right-handed woman presented to our institution with mild left-sided weakness and increasingly frequent right hemispheric transient ischemic attacks (TIAs). Past history was significant for marked intracranial and peripheral vascular disease, diabetes, hypertension, and hyperlipidemia. She had three small strokes in her thirties, and underwent a right carotid endarterectomy in one year prior to presentation. She had also had a previous coronary artery bypass grafting (CABG) procedure.

Catheter-based angiography (Fig. 1a) revealed a high-grade stenosis of the right M1 segment of the middle cerebral artery (MCA) with substantial alteration of flow throughout its territory. The anterior and middle opercular branches filled in a delayed fashion, while the angular artery and posterior branches did not fill in an anterograde manner. The cervical internal carotid artery was mildly stenosed (Fig. 1b), and the size of the anterior trunk of the STA was quite favorable to serve as a



a b

Fig. 1. (a) Lateral view of a catheter-based angiogram showing high-grade RMCA stenosis with no anterograde filling of the angular artery and its posterior branches. The ipsilateral STA is visible. (b) RCCA injection showing a small RICA with no obvious stenosis

bypass donor vessel. After a Diamox SPECT tolerance test demonstrated poor vascular reserve, a revascularization procedure was performed, during which the anterior branch of the right STA was anastomosed to an MCA cortical branch. During the procedure, good flow was documented both visually and by intraoperative Doppler. Her post-operative clinical course was unremarkable. No untoward events such as hypotension, focal compression from eyeglasses, etc were noted, nor was the status of a palpable pulse recorded during the ensuing week. A post-operative common carotid angiogram with selective external carotid injection, done one week later as part of a standard protocol to check patency prior to discharge, however, showed filling of the stump of the STA, but no flow through the bypass either early or in a delayed fashion later in the study (Fig. 2). The remainder of the angiogram was unchanged from her pre-operative study. She was placed on anti-platelet therapy, and was free of symptoms thereafter.

Four years later, she experienced acute loss of vision in the inferior aspect of her right eye visual field. Ophthalmologic evaluation revealed an infarction in superior retina of the right eye, suggesting an embolic occlusion of a branch of the central retinal artery. Computed tomographic angiography (CTA) revealed a ''string sign'' in the right cervical ICA. Catheter-based angiography (Fig. 3) revealed a tapering occlusion of the proximal right ICA, with partial reconstitution of the intracranial ICA via dural connections with the right occipital artery. Interestingly, the STA-MCA bypass – formerly bereft of flow – was now widely patent. Also noted was a proximal right M1 occlusion, extensive filling of the distal right MCA via the bypass, and partial reconstitution of the right ACA territory by splenial branches off the right PCA. Since her right ICA was completely occluded and collateral appeared good to the

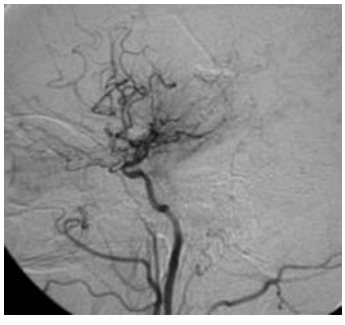
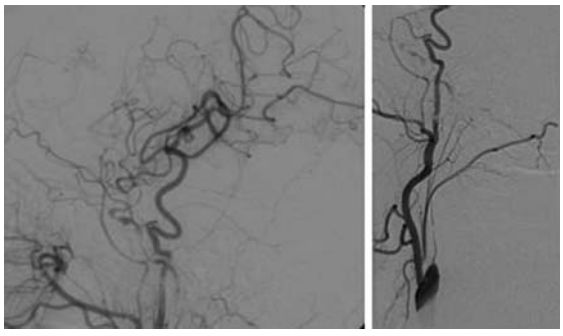


Fig. 2. Post-operative lateral view showing no flow through the STA-MCA bypass graft and hence no filling of the MCA distal to the stenosis



a b

Fig. 3. (a) Follow-up RCCA injection showing robust bypass graft with filling of the ipsilateral MCA territory. (b) RCCA injection showing critical stenosis of the ipsilateral ICA

ipsilateral hemisphere, another anti-platelet agent was added, and she was discharged home without further intervention.

Discussion

We report a rare case of previously occluded STA-MCA bypass graft that apparently opened up and progressively enlarged concomitant with progressive ipsilateral ICA stenosis. Bypass graft patency rates, as documented by post-operative angiogram, were reported as high as 96% one month after surgery in the EC-IC bypass trial [5]. Other large series have reported graft patency rates of between 96 and 99% [8, 11] at varying times after surgery.

If patency is maintained, the donor vessel is not a static conduit but usually dilates with time and accommodates increased cerebral blood flow [2]. In one study, up to 70% of EC-IC bypass grafts hypertrophied over subsequent angiograms [8]. The altered cerebral hemodynamics after bypass grafting may also influence the nature and severity of the bypassed vessel and ipsilateral vessels. Early observers [3, 6, 7] noted that when EC-IC bypass were performed for symptomatic ICA or MCA stenosis, changes in the degree of the stenosis occurred post-operatively in two patterns. In the first, the stenotic vessel became occluded within days of bypass, accompanied in most cases by a stroke despite a widely patent graft. In these cases, the increased distal cortical flow through the bypass presumably produced a critical drop in the perfusion pressure gradient across the stenotic segment, resulting in stasis and thrombotic occlusion. Perforators arising from thrombosed segment were also occluded, and a patent bypass graft was often not sufficient to prevent cortical infarction, suggesting propagation of the thrombus and reduced communication between adjacent vessel territories.

In another pattern, an improvement in the degree of stenosis (or even resolution) of the bypassed vessel was noted on follow-up arteriography. In these cases, graft occlusion was commonplace; in cases where the graft remained patent, cortical filling was poor, suggesting that stenosis resolution increased the distal arterial pressure, thus encouraging occlusion of the donor vessel. Conversely, Andrews *et al.* described a patient treated with a successful STA-MCA bypass for severe MCA stenosis who developed severe ipsilateral headaches and a more prominently palpable STA ten years later. Repeat arteriography revealed a high-grade ipsilateral ICA stenosis; after treatment with endarterectomy, the symptoms completely resolved [1].

It is clear that many high cervical or intracranial arterial stenoses are not static, and that part of the ''stenosis'' is coming from clot either within the vessel wall or adjacent lumen, rather than fixed atheromatous disease. Time may allow recanalization of some of these obstructions, while others worsen. Because of these divergent outcomes, we consider an endarterectomy under such circumstances when appropriate, but do not perform a bypass for inaccessible stenosis until the lesion has been documented to be stable or worsening on serial examinations despite best medical treatment [4].

Our case is particularly interesting because of the relationship between the patient's STA-MCA bypass graft and obstruction of the ipsilateral ICA. When the ICA was only mildly stenosed (despite a tandem MCA stenosis), the bypass was not patent. Sometime later, the graft opened; when that occurred, and the factors involved, are unclear. When the ICA became occluded, the graft opened up widely and presumably prevented hemispheric ischemia. Did the closure of the ICA prompt or contribute to the reopening of the bypass?

While other reports have documented progressive ICA stenosis after bypass with a widely patent graft, none describes the reverse situation – eventual opening of an occluded STA-MCA bypass graft and concomitant progressive ipsilateral ICA stenosis. One explanation for this observed phenomenon is that the STA-MCA bypass graft was in spasm post-operatively secondary to temporary clip application, leading to an absence of flow on the post-operative angiogram. The spasm could have resolved over the following weeks, permitting flow through the graft and expediting ICA stenosis. Good intraoperative flow had been observed after clip removal, however, making severe early vasospasm less likely. Another explanation could be an intraluminal thrombus that dissolved over time. The freedom of symptoms despite an occluded graft suggests an ''early'' delayed graft opening, and may favor the latter two possibilities. Her strong anti-platelet regimen, however, may also have contributed to this clinical endpoint.

Alternatively, it is possible that the graft was thrombosed, and that the progressively worsening right carotid disease served as a stimulation to graft recanalization. Another explanation is that the graft was patent all along, but that competitive anterograde flow from the ICA prevented its filling (similar to the way a posterior communicating artery is patent, but may not be visible on either a carotid or vertebral artery injection). While we cannot be certain about the exact factors contributing to this phenomenon, this case underscores the dynamic interrelationship between bypass grafts and neighboring arterial disease.

References

1. Andrews BT, Chater NL, Weinstein PR (1985) Extracranial-intracranial arterial bypass for middle cerebral artery stenosis and occlusion: operative results in 65 cases. J Neurosurg 62: 831–838
2. Latchaw RE, Ausman JI, Lee MC (1979) Superficial temporal-middle cerebral artery bypass. A detailed analysis of multiple pre- and postoperative angiograms in 40 consecutive patients. J Neurosurg 51(4): 455–465
3. Awad I, Furlan A, Little JR (1984) Changes in intracranial stenotic lesions after extracranial-intracranial bypass surgery. J Neurosurg 60: 771–776
4. Day AL, Rhoton AL, Quisling R (1980) Resolving siphon stenosis following endarterectomy. Stroke 11: 278–281
5. The EC/IC Bypass Study Group (1985) Failure of extracranial-intracranial arterial bypass to reduce the risk of ischemic stroke. Results of an international randomized trial. N Engl J Med 313(19): 1191–1200
6. Furlan AJ, Little JR, Dohn DF (1980) Arterial occlusion following anastomosis of the superficial temporal artery to middle cerebral artery. Stroke 11: 91–95
7. Gumerlock MK, Ono H, Neuwelt EA (1983) Can a patent extracranial-intracranial bypass provoke the conversion of an intracranial arterial stenosis to a symptomatic occlusion? Neurosurgery 12: 391–400
8. Jack CR, Sundt TM Jr, Fode NC, Gehring DG (1988) Superficial temporal-middle cerebral artery bypass: clinical pre- and postoperative angiographic correlation. J Neurosurg 69: 46–45
9. Lawton MT, Hamilton MG, Morcos JJ, Spetzler RF (1996) Revascularization and aneurysm. Neurosurgery 38: 83–94
10. Spetzler RF, Schuster H, Roski RA (1980) Elective extracranial-intracranial arterial bypass in the treatment of inoperable giant aneurysms of the internal carotid artery. J Neurosurg 53: 22–27
11. Sundt TM Jr, Whisnant JP, Fode NC, Piepgras DG, Houser OW (1985) Results, complications, and follow-up of 415 bypass operations for occlusive disease of the carotid system. Mayo Clin Proc 60(4): 230–240
12. Wanebo JE, Zabramski JM, Spetzler RF (2004) Superficial temporal artery-to-middle cerebral artery bypass grafting for cerebral revascularization. Neurosurgery 55(2): 395–398
13. Yasargil MG (ed) (1969) Microsurgery applied to neurosurgery. Georg Thieme Stuttgart, Stuttgart, pp 105–115

Comment

The authors present the case of a 47 year old female with a symptomatic MCA stenosis in the setting of a previously treated mild ipsilateral carotid stenosis. The patient was treated with a STA-MCA bypass graft. The initial control angiogram demonstrated non-patency of the bypass graft. Anti-platelet therapy was initiated and the patient's symptoms resolved. The patient presented four years later with a retinal infarction. An angiogram revealed a widely patent bypass graft and a progressive ICA stenosis. Concervative management was pursued with a second anti-platelet.

This paper represents an interesting observation, demonstrating a nuance of this rarely performed procedure. Most of us would have presumed that an occluded STA-MCA bypass graft would have been a permanent finding. This case clearly illustrates that secondary flow phenomena can occur long after the procedure which may be unanticipated. What we do not know, however, is the frequency with which this unusual finding may be found in other patients whose grafts have similarly been found to be non-functional. The authors are to be congratulated for adding this unusual but important observation to the literature.

James T. Rutka
The University of Toronto

Correspondence: Arthur L. Day, MD, Harvard Medical School, Department of Neurosurgery, Brigham and Women's Hospital, Boston MA 02115, USA. e-mail: aday1@partners.org