The Paramount Role of the Anterior Communicating Artery in the Collateral Cerebral Circulation

Miloslav Spacek, MD¹ David Tesar, MD, PhD¹ Josef Veselka, MD, PhD, FESC, FSCAI, FICA¹

¹Department of Cardiology, University Hospital Motol, Charles University, Prague, Czech Republic

Int J Angiol 2015;24:236–240.

Address for correspondence Josef Veselka, MD, PhD, FESC, FSCAI, FICA, Department of Cardiology, University Hospital Motol, 2nd Medical School, Charles University, V Úvalu 84, 150 06, Prague 5, Czech Republic (e-mail: veselka.josef@seznam.cz).

Abstract

Keywords

- carotid occlusion
- transcranial Doppler
- cerebrovascular reserve
- collateral circulation

Clinical manifestation of carotid occlusive disease is largely dependent on the severity of stenosis and the capability of collateral circulation. However, due to the complexity and difficulty in evaluation, cerebral collateral circulation has, so far, remained underappreciated. We report a patient with advanced extracranial arterial disease (including the right subclavian steal, occlusion of the right external carotid artery, and severe stenosis of the left vertebral artery), who underwent transient right internal carotid artery occlusion during carotid intervention. Throughout the occlusion, the flow into the right hemisphere (monitored by transcranial Doppler ultrasound in the right middle cerebral artery) was sufficient despite almost totally dependent on the anterior communicating artery, which highlights its role as the most potent collateral pathway.

Carotid artery stenting (CAS) is the method of choice for selected patients with carotid artery disease. The indication criteria are currently based on the severity of stenosis considering patient's symptoms.¹ Increasingly, the procedure is performed with protection devices that involve endovascular clamping of the common carotid artery to arrest or reverse flow in the internal carotid artery (ICA). These systems resemble surgical clamp and make perfusion of the ipsilateral hemisphere totally dependent on the collateral blood supply. In addition, it has been demonstrated that patients with impaired cerebrovascular reserve are more prone to neurologic complication.^{2,3} Therefore, it is of utmost importance to expand our understanding of collateral cerebral circulation to better select and treat patients. This case emphasizes the complexity of cerebral circulation and highlights the role of the anterior communicating artery (ACom) as the major collateral pathway.

Case

A 65-year-old man with known carotid artery disease was referred to our institution because of ultrasound signs of severe right ICA in-stent restenosis. Complete evaluation of the patient's history revealed that he had previously under-

gone bilateral CAS. The patient had also been diagnosed with subclavian steal resulting from an occlusion of his right subclavian artery. There was no history of stroke or transient ischemic attacks. The patient was accustomed to hard work in his garden and denied having any syncope events or claudication in his right arm despite routine periods of strenuous activity. His physical examination was unremarkable except for a barely discernible right radial and brachial pulses. The ultrasound examination showed severely calcified right ICA in-stent restenosis. The peak systolic velocity was 350 cm/s with end-diastolic velocity of 115 cm/s, suggesting 70 to 79% restenosis based on modified criteria for in-stent velocities.

Before carotid angiography, a transcranial Doppler (TCD) ultrasound examination was performed to test the patient's cerebrovascular reserve and to reveal collateral channels: this is a routine part of the preprocedural evaluation in our institution. The patient was found to have mildly decreased mean flow velocity (MFV) in his right middle cerebral artery (MCA) (35–40 cm/s) compared with the left MCA (51–53 cm/s). The patient was also found to have reversed flow in the right anterior cerebral artery and nonreversed flow in the right ophthalmic artery. His cerebrovascular reserve (measured during the breath holding test) was normal with a breath

Fig. 1 Selective angiography of the right common carotid artery. Note the absence of anterior cerebral artery filling (area of question mark), tight in-stent restenosis of the right internal carotid artery (thick arrow), occlusion of the right external carotid artery (thin arrow), and retrograde filling of the basilar and right vertebral artery (arrowheads). Also note that the right vertebral artery flow is only slightly contributed (diluted) by the left vertebral artery. Transcranial Doppler head frame is marked by an asterisk.

holding index (BHI) of 1.40 and 1.28 for the left and right MCAs, respectively. The BHI was measured repeatedly, requiring the apnea period of 25 to 30 seconds. The angiography study showed that the left ICA as well as the left external carotid artery was without severe stenosis or restenosis. The right subclavian artery was occluded before the origin of the verte-

bral artery, which showed reverse flow. The right external carotid artery was occluded and the right ICA had an 85% eccentric in-stent restenosis and a kink just distal to the stent. The patient's left vertebral artery had antegrade flow and was severely stenosed just proximal to the junction with the right vertebral artery. The basilar artery had reverse flow and was fed from both ICAs through the circle of Willis. In addition, the anterior cerebral arteries could not be visualized through the right carotid angiography, which confirmed reversed flow and its role in cross-filling as shown on the TCD. **►Fig. 1** shows the right carotid angiography and illustrates the hemodynamic situation.

The patient's condition meant that he was a candidate for dilation of the restenosis and possibly opening of the occluded right subclavian artery. Due to the occlusion of the right external carotid artery, the presence of tight in-stent restenosis and the kink, it was not possible to (safely) deploy any protection system and the procedure was performed unprotected despite the higher risk of distal embolization. Throughout the procedure, MFV of the right MCA was monitored using TCD to anticipate any clinical intolerance. Dilation of the restenosis was performed with a 5 × 40 mm drug-eluting balloon (In.Pact Pacific, Invatec, Brescia, Italy); balloon inflation lasted for approximately 40 seconds. However, the residual stenosis was still 40 to 50% due to significant recoil caused by the severely calcified lesion (**►Fig. 2**). TCD monitoring during prolonged dilation showed MFV drop of approximately 31% from baseline 32 to 22 cm/s (**►Fig. 3**) with an immediate return to baseline values after balloon deflation. Even though the procedure was well tolerated by the patient, residual flow during transient occlusion was significantly blunted. Therefore, considering the risk of repeated restenosis in a severely calcified lesion, the operator decided to open the right subclavian artery to provide an additional source of collateral circulation through the posterior communicating arteries. Recanalization was subsequently accomplished with gradual balloon dilations and the placement of a 6 × 18 mm stent (Racer RX, Medtronic, Minneapolis,

Fig. 2 Right carotid angiography showing reduction in the severity of in-stent restenosis (arrow) following balloon dilation. Left oblique view (A) before and (B) after treatment.

Fig. 3 Transcranial Doppler monitoring of the right middle cerebral artery flow—pulsed wave spectral waveform display: (A) comparison of pulsatile flow before carotid occlusion and (B) blunted flow during carotid occlusion.

MN) into the proximal segment (►Fig. 4). No neurological complications were observed during the procedure or during hospitalization; the patient was discharged the next day following an ultrasound examination to determine baseline flow values.

Discussion

Clinical manifestation of carotid occlusive disease is largely dependent on the severity of the stenosis and the extent of recruitable collaterals.^{3,4} This case illustrates the complexity of collateral cerebral circulation and highlights the importance of ACom as the most potent collateral pathway.

The circle of Willis constitutes the main cerebral collateral network which is immediately available when large arterial occlusions occur. Other collateral pathways such as the leptomeningeal anastomoses or reversible ophthalmic artery

flow are considered secondary networks, which need time for proper functioning and are presumed to be recruited once the primary collaterals have failed.⁵

Several authors have prioritized the role of ACom in the setting of acute carotid occlusion.^{6,7} Visser et al analyzed preoperative TCD variables in patients undergoing carotid endarterectomy and concluded that patients with adequate cross-filling (detected as reversed flow in the ipsilateral anterior cerebral artery) never needed a shunt.⁶ Doblar et al studied the role of the anterior and posterior communicating arteries by measuring the MCA flow velocity throughout the clamp period. They concluded that the posterior communicating artery was a less potent collateral pathway and contributed little if there was a functional ACom.⁷ One must, however, keep in mind that an absent or hypoplastic posterior communicating artery is a risk factor for acute ischemic strokes.⁸

It is known from pathoanatomical studies that more than 50% of patients do not have a complete circle of Willis, and ACom can be absent or nonfunctional in as many as 25% of subjects.^{9,10} Thus, with only 3 to 7% of patients reported not to tolerate carotid clamp,¹¹ it seems that well-adapted non-ACom collaterals might be sufficient to permit isolated carotid occlusion.

Furthermore, the role of the ACom is even more evident in patients with a carotid-subclavian steal phenomenon. Although it was initially believed that the presence of an isolated subclavian steal was associated with cerebral ischemia, it is now accepted that most patients are rarely symptomatic unless one or more other extracranial arteries supplying the brain are severely diseased.¹²

On the contrary, patients with severe innominate artery (IA) disease have more significant alterations in extracranial hemodynamics, which may lead to flow compromise.^{13–15} In patients with IA occlusion, blood “stolen” by the right vertebral artery perfuses the right carotid as well as right brachial artery—a phenomenon called “carotid recovery.” During strenuous upper limb exercise or in case of inadequate collateral circulation through the vertebral artery, flow through the carotid artery may even reverse leading to a phenomenon known as “subclavian-carotid double steal.”

Our patient presented with advanced extracranial arterial disease suggesting reduced flow to the right hemisphere. Despite that, he was symptom free and his cerebrovascular reserve was well preserved, presumably due to residual flow through the ICA and active anterior cross-filling.

Fig. 4 Selective angiography of the innominate artery following endovascular treatment. Note the restored antegrade flow in the subclavian artery (arrowheads) and the vertebral artery (thick arrow) as well as diminished anterior cross-filling (restored flow to the anterior cerebral artery [thin arrow]).

Fig. 5 Schematic illustration of major hemodynamic changes in cases of (A) carotid recovery phenomenon, (B) right internal (balloon) and external carotid occlusion together with right subclavian steal and severe stenosis of the left vertebral artery—case patient, and (C) subclavian-carotid double steal phenomenon.

Interestingly, focusing on the anterior collateral circulation, prolonged transient occlusion of the right ICA simulated a state of IA occlusion—specifically a borderline condition between the less severe phenomenon of “carotid recovery” and the sinister phenomenon of “subclavian-carotid double steal” as depicted in ▶Fig. 5. This led to the flow reduction in the MCA, which under basal conditions during procedure, was clinically well tolerated despite leaving the patient almost totally dependent on the ACom. This highlights the potential of anterior cross-filling. Similarly, Muñoz et al described a completely asymptomatic patient with severe IA stenosis with well-developed cross-filling through the circle of Willis.¹⁶ Nevertheless, the blunted flow pattern of MCA flow during prolonged occlusion suggested a depleted cerebrovascular reserve and helped the operator decide on performing a complex carotid-subclavian intervention to alleviate the risk of hemodynamic compromise in case of a repeated restenosis.

It is also of note that residual flow through a severely stenosed ICA could preserve vasomotor reaction despite contributing to the subclavian steal circulation. This finding supports the concept that residual flow through the ICA plays an important role in the preservation of cerebrovascular reserve and is in accordance with the results of Visser et al who found that, in unselected patients scheduled for carotid endarterectomy, cerebrovascular reserve was a less reliable predictor of carotid occlusion intolerance compared with the carotid compression test.⁶ In other words, the less severe the ICA stenosis, the more unpredictable will be the flow changes during carotid clamping and the less reliable will be predictions regarding occlusion intolerance based on cerebrovascular reserve testing.

Conclusion

Collateral cerebral circulation constitutes an important aspect of carotid occlusive disease, which has, so far, remained

largely unappreciated. With this case, we illustrate the complexity of collateral circulation and highlight the importance of ACom as a major collateral pathway.

Acknowledgments

This study was supported by the projects of Ministry of Health, Czech Republic, for conceptual development of research organization 00064203 (University Hospital Motol, Prague, Czech Republic) and NT13319.

References

- Meier B, Frank B, Wahl A, Diener HC. Secondary stroke prevention: patent foramen ovale, aortic plaque, and carotid stenosis. *Eur Heart J* 2012;33(6):705–713, 713a, 713b
- Sfyroeras G, Karkos CD, Liasidis C, et al. The impact of carotid stenting on the hemodynamic parameters and cerebrovascular reactivity of the ipsilateral middle cerebral artery. *J Vasc Surg* 2006;44(5):1016–1022, discussion 1022
- Silvestrini M, Vernieri F, Pasqualetti P, et al. Impaired cerebral vasoreactivity and risk of stroke in patients with asymptomatic carotid artery stenosis. *JAMA* 2000;283(16):2122–2127
- Hedera P, Bujdaková J, Traubner P. Effect of collateral flow patterns on outcome of carotid occlusion. *Eur Neurol* 1995;35(4):212–216
- Romero JR, Pikula A, Nguyen TN, Nien YL, Norbash A, Babikian VL. Cerebral collateral circulation in carotid artery disease. *Curr Cardiol Rev* 2009;5(4):279–288
- Visser GH, Wienenke GH, van Huffelen AC, Eikelboom BC. The use of preoperative transcranial Doppler variables to predict which patients do not need a shunt during carotid endarterectomy. *Eur J Vasc Endovasc Surg* 2000;19(3):226–232
- Doblar DD, Plyushcheva NV, Jordan W, McDowell H. Predicting the effect of carotid artery occlusion during carotid endarterectomy: comparing transcranial Doppler measurements and cerebral angiography. *Stroke* 1998;29(10):2038–2042
- Chuang YM, Liu CY, Pan PJ, Lin CP. Posterior communicating artery hypoplasia as a risk factor for acute ischemic stroke in the absence of carotid artery occlusion. *J Clin Neurosci* 2008;15(12):1376–1381

- 9 Hoksbergen AW, Fülesdi B, Legemate DA, Csiba L. Collateral configuration of the circle of Willis: transcranial color-coded duplex ultrasonography and comparison with postmortem anatomy. *Stroke* 2000;31(6):1346–1351
- 10 Liebeskind DS. Collateral circulation. *Stroke* 2003;34(9):2279–2284
- 11 Cremonesi A, Rigattieri S, Liso A, et al. Proximal protection in carotid artery stenting: rationale and recent findings. *EuroIntervention* 2007;3(2):269–274
- 12 Moran KT, Zide RS, Persson AV, Jewell ER. Natural history of subclavian steal syndrome. *Am Surg* 1988;54(11):643–644
- 13 Mozersky DJ, Barnes RW, Sumner DS, Strandness DE Jr. Hemodynamics of innominate artery occlusion. *Ann Surg* 1973;178(2):123–127
- 14 Tyagi S, Girish MP, Gupta MD. Endovascular revascularization of bald aortic arch for severe cerebral ischemia. *Catheter Cardiovasc Interv* 2013;81(7):1213–1216
- 15 Filis K, Toufektzian L, Sigala F, et al. Right subclavian double steal syndrome: a case report. *J Med Case Reports* 2008;2:392
- 16 Muñoz R, Pulido L, Gállego J. Asymptomatic subclavian-carotid double steal phenomenon due to innominate artery stenosis. *Neurology* 2012;78(20):e125