

Ipsilateral hyperperfusion after neck clipping of a giant internal carotid artery aneurysm

Case report

HIDEKI MURAKAMI, M.D., D.M.Sc., MAKOTO INABA, M.D.,
AKIYOSHI NAKAMURA, M.D., D.M.Sc., AND TAKAKAZU USHIODA, M.D.

Departments of Neurosurgery and Neuroradiology, Ashikaga Red Cross Hospital, Tochigi, Japan

✓ A 48-year-old woman exhibited hyperperfusion soon after undergoing a successful clip operation involving multiple clip placement for a giant internal carotid artery (ICA) aneurysm. Intraarterial digital subtraction angiography demonstrated a left paraclinoid giant aneurysm. Multiple clips were placed to obliterate the aneurysm during a 7-minute temporary ICA occlusion. Intraoperative Doppler ultrasound flowmetry showed that the blood flow through the ICA distal to the aneurysm increased from 71.6 ml/minute before clipping to 123.3 ml/minute after. The patient exhibited right hemiparesis and motor aphasia after the operation. Postoperative imaging studies revealed an increase in perfusion and diffuse edema in the left cerebral cortex. The symptoms and diffuse brain edema gradually resolved. In this case, increase in blood flow through the ICA distal to the aneurysm may have played an important role in the circulatory disturbance.

KEY WORDS • giant aneurysm • hypoperfusion • hyperperfusion • internal carotid artery

ANEURYSMS have been reported to increase in volume during the cardiac cycle.⁵ It is likely that part of the blood flow through the parent artery is slowed by the increase in the volume of the aneurysm. Assuming this to be true, blood flow increases through the parent artery distal to the aneurysm after the neck has been clipped. If the volume of increased blood flow is large enough to exceed the cerebral autoregulatory abilities, hyperperfusion will occur.

Hyperperfusion is known to occur after carotid endarterectomy, extracranial–intracranial bypass surgery, and percutaneous transluminal angioplasty;^{2,8–10} however, it has never been reported after clipping the neck of an aneurysm.

Case Report

History. This 48-year-old right-handed woman presented with progressive loss of vision and a nasal visual field defect in her left eye. The patient had no history of stroke or other vascular risk factors.

Abbreviations used in this paper: ACA = anterior cerebral artery; CT = computerized tomography; DS = digital subtraction; FLAIR = fluid-attenuation inversion recovery; HMPAO = hexamethylpropyleneamine oxime; ICA = internal carotid artery; MCA = middle cerebral artery; MR = magnetic resonance; POD = postoperative day; SPECT = single-photon emission CT; TOF = time of flight; 3D = three-dimensional.

Examination. Magnetic resonance imaging revealed a nonthrombosed giant aneurysm. A 3D TOF MR angiography study also revealed the lesion as well as an attenuation in ipsilateral anterior circulation (Fig. 1A). Intra-

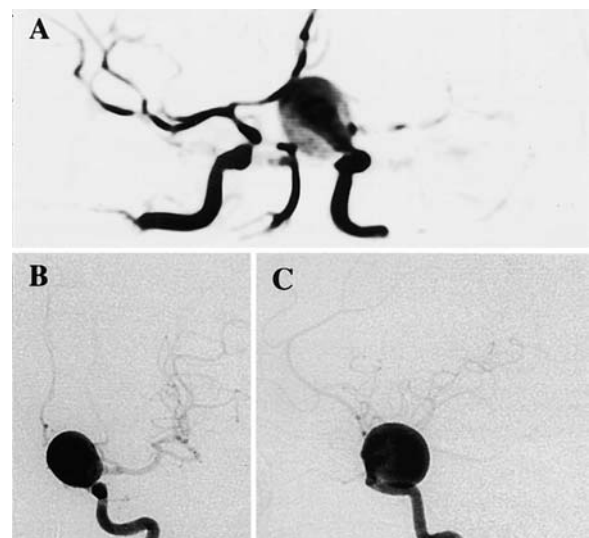


FIG. 1. Preoperative imaging studies. A: An MR angiogram showing the aneurysm and attenuation in ipsilateral anterior circulation. B and C: Frontal and lateral DS angiography views of the left ICA revealing a paraclinoid giant aneurysm with poor filling of the normal hemisphere branches.

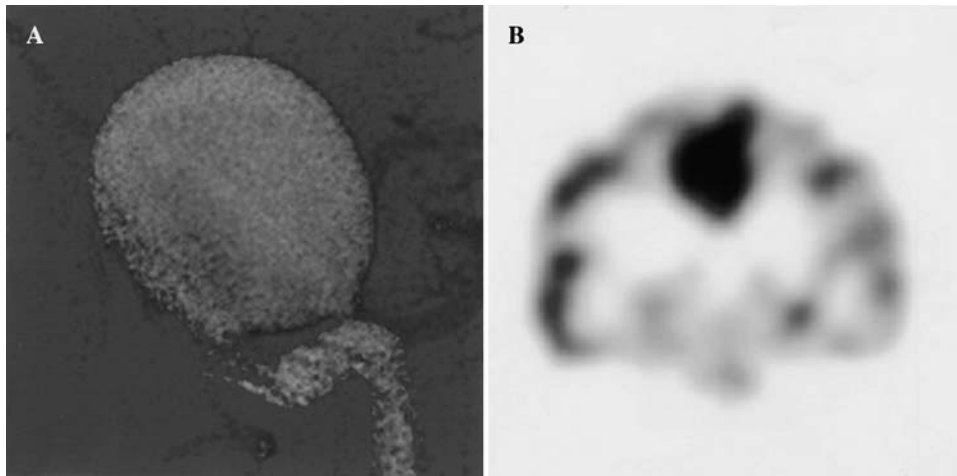


FIG. 2. A: A DS angiogram revealing the changes in the size of the aneurysm. B: A SPECT scan, coronal section, demonstrating a slight decrease in left hemisphere perfusion.

arterial DS angiography demonstrated a left paraclinoid giant aneurysm with poor filling of the left hemisphere branches (Fig. 1B and C). Digital subtraction angiography revealed that the minimum diameter of the aneurysm increased from 21.3 to 21.8 mm and the maximum diameter increased from 25.2 to 25.9 mm (Fig. 2A). The increase in the volume of the aneurysm was calculated by the method of Meyer, et al.,⁵ to be approximately 0.5 ml/one cardiac cycle. Stenotic lesions were not observed in the extracranial or intracranial arteries. Using ^{99m}Tc-HMPAO SPECT a slight decrease in perfusion in the left hemisphere was demonstrated (Fig. 2B). A balloon occlusion test of the left ICA was performed. The left ACA and the MCA were filling through the ipsilateral posterior communicating artery but not through the anterior communicating artery. No neurological deficits were observed during the 15-minute occlusion, but ^{99m}Tc-HMPAO SPECT did reveal a decrease in perfusion in the left hemisphere.

Operation. A left frontotemporal craniotomy was performed. After removing the anterior clinoid process, the broad neck of the aneurysm was clearly identified. The

neck of the aneurysm arose from the ventromedial aspect of the ICA and was located between the ophthalmic artery and the posterior communicating artery. After achieving temporary clip occlusion of the clinoid segment of the ICA, the aneurysm was successfully obliterated by using four serial Sugita clips and the ICA was preserved without stenosis. The temporary clip was in place for a total of 7 minutes. Intraoperative Doppler ultrasound flowmetry (DVM-4300; Nihon Kohden Co., Ltd., Tokyo, Japan) showed that the blood flow through the ICA distal to the aneurysm had increased from 71.6 ml/minute before the neck was clipped to 123.3 ml/minute thereafter. Both the systolic and diastolic flow velocity in the ICA distal to the aneurysm increased after neck clipping (Fig. 3).

Postoperative Course. Soon after the operation, the patient exhibited right hemiparesis and aphasia. An emergency DS angiography study demonstrated the complete obliteration of the aneurysm and a patent ICA without stenosis (Fig. 4A and B), but a CT examination showed diffuse swelling in the left hemisphere cortex. A CT scan obtained on POD 1 revealed no significant edema in the frontal and temporal lobes. A ^{99m}Tc-HMPAO SPECT scan also obtained on POD 1 demonstrated that perfusion was still increased in the left hemisphere (Fig. 4C). The patient's blood pressure was monitored closely and remained normotensive. Although the patient's visual acuity improved, she required 4 weeks to recover completely from the hemiparesis and aphasia. Follow-up CT scanning performed on POD 5 revealed prominent brain swelling in the left cortex, but the swelling had improved by POD 28 (Fig. 5A and B). A series of FLAIR MR images obtained on POD 4 demonstrated a high signal intensity in the left frontoparietal cortex; the area of high signal intensity had been reduced by POD 28 (Fig. 5C and D). Cerebral infarctions were not observed in the white matter or basal ganglia. These imaging findings confirm that the lesion was a cortical brain edema resulting from hyperperfusion. The patient had no neurological deficits other than a nasal visual field defect when discharged from the hospital.

Discussion

Giant aneurysms have a dismal natural history and

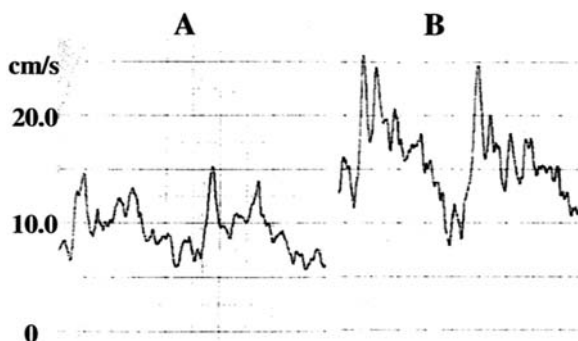


FIG. 3. Intraoperative Doppler ultrasonograms of the ICA distal to the aneurysm, revealing an increase in velocity after aneurysm neck clipping. A: Before the aneurysm neck is clipped, the systolic velocity is 14.1 cm/second and the diastolic velocity is 6.3 cm/second. B: After neck clipping, the systolic velocity increased to 24.8 cm/second and the diastolic velocity increased to 10.4 cm/second.

Hyperperfusion after neck clipping of giant aneurysm

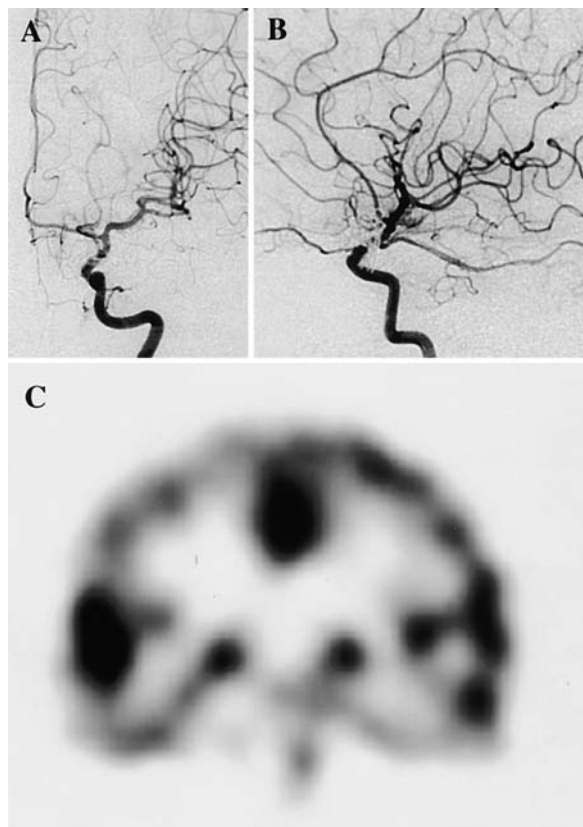


FIG. 4. A and B: Postoperative DS angiograms of the left ICA revealing the complete obliteration of the aneurysm, with a patent ICA and normal well-filled hemisphere branches. C: A SPECT scan, coronal section, obtained on POD 1, demonstrating increased perfusion in the left hemisphere.

often result in hemorrhage, cerebral compression, and thromboembolism. The mortality rate in patients with untreated giant aneurysms has been reported to be 68% after 2 years and 85% after 5 years.⁶ Although surgical treatment is controversial because of the anatomical complexity and surgical difficulty, direct aneurysm neck clipping with preservation of the parent artery and branch arteries is considered to be the preferred treatment method.³ Ischemia resulting from temporary arterial occlusion, stenosis, and occlusion of the parent artery or branch arteries have been reported as complications of direct aneurysm neck clipping;^{3,6} however, hyperperfusion has never been reported to cause focal neurological signs after neck clipping for aneurysm obliteration.

Two postischemic circulatory disturbances associated with temporary arterial occlusion have been known to occur in gerbils and cats.^{1,4} The first disturbance is hyperperfusion, which promptly follows the release of arterial occlusion and lasts for up to 5 minutes. The second is hypoperfusion, which develops later and lasts for up to 6 hours. Hyperperfusion is associated with the opening of the blood-brain barrier to serum proteins, resulting in edema and hypoperfusion.^{1,4} Thus the hyperperfusion associated with the temporary arterial occlusion is transient and lasts for a short period. In this case, hyperperfusion was confirmed by the SPECT scanning performed on POD 1. Moreover, neurological deficits were not observed

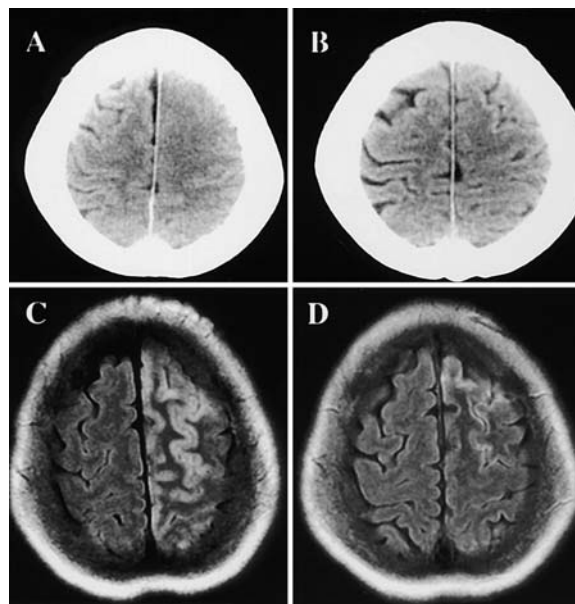


FIG. 5. A: A CT scan obtained on POD 5, revealing brain swelling in the left hemisphere. B: A CT scan on POD 28, revealing an improvement in brain swelling. C: A FLAIR MR image obtained on POD 4, demonstrating high signal intensity in the left frontoparietal cortex. D: A FLAIR MR image obtained on POD 28, demonstrating a reduction in the high signal intensity area.

after the 15-minute balloon occlusion test, suggesting that the hyperperfusion occurring after release of the 7-minute temporary clip had not caused any neurological deficits. Therefore, this postoperative hyperperfusion was probably not caused by the temporary occlusion.

Hyperperfusion is thought to occur when perfusion is rapidly increased in a chronically hypoperfused area.⁸⁻¹⁰ In our patient, ischemic symptoms were not observed before admission, and preoperative SPECT scanning revealed slight hypoperfusion in the left cerebral cortex. Although the cerebral perfusion reserve was not evaluated, this finding may indicate that the arterial network in the left cortex became dilated to maintain the cerebral blood flow, resulting in an impairment of normal autoregulation, compared with the right side. The stenotic lesions of the ICA proximal and distal to the aneurysm were not detected during the DS angiography study. Moreover, no compression-related stenosis of the ICA caused by the aneurysm was seen on 3D CT angiography nor was it observed during the operation. Therefore, this slight hypoperfusion is considered to have been caused by the giant aneurysm.

In 3D TOF MR angiography, the signal intensity is influenced by the flow velocity, slice thickness, repetition time, and flip angle.⁷ In a series of 3D TOF MR angiography studies performed in a single patient, the slice thickness, repetition time, and flip angle are constant. Therefore, the signal intensity is dependent on the flow velocity. In this case, MR angiography revealed decreased signal intensity in the left ACA and MCA and DS angiography demonstrated that the diameters of the left ACA and MCA were almost equal to those of the right. These findings indicate that the flow velocity was decreased; therefore, blood flow through these arteries was reduced on the left side. The mechanism by which the giant aneurysm de-

creased the blood flow through these arteries remains uncertain.

Meyer, et al.,⁵ reported that unruptured aneurysms exhibit a $17.6 \pm 8.9\%$ increase in volume between cardiac systole and diastole, as measured using cine phase-contrast MR angiography. If this is true, part of the blood flow volume is probably lessened when the volume of the aneurysm increases during systole. As a result, systolic blood flow through the distal parent artery of the aneurysm would be decreased. During diastole, the blood retained in the aneurysm during systole regurgitates from the aneurysm into the parent artery. In the present case, a patent ICA without stenosis was confirmed during the operation and by an emergency DS angiography. The increase in the systolic velocity of the ICA distal to the aneurysm after the neck was clipped may support the aforementioned assumption. The regurgitated blood might have then disturbed the blood flow through the parent artery, because the diastolic velocity was also increased after the aneurysm neck was clipped. According to the DS angiography findings, the volume of the aneurysm in the present case increased by 0.5 ml/cardiac cycle. The wasted blood volume can be calculated to be 40 ml/minute because the heart rate was approximately 80 beats/minute during aneurysm neck clipping. The blood flow through the ICA distal to the aneurysm increased by 51.7 ml/minute after obliteration (71.6–123.3 ml/minute). Therefore, it seems reasonable to suppose that the giant aneurysm was responsible for the reduction in blood flow through the ICA distal to the aneurysm and might have caused the slight hypoperfusion in the left cerebral cortex. Assuming this to be true, blood flow through the ICA distal to the aneurysm would have suddenly increased after the aneurysm was obliterated by clipping; the dilated vessels might not have been able to regulate this new blood flow, leading to hyperperfusion and capillary breakthrough, which resulted in edema.

Conclusions

The giant ICA aneurysm in this case may have caused hypoperfusion in the left cerebral cortex of the patient. After aneurysm neck clipping, the increase in blood flow

through the ICA distal to the aneurysm may have caused hyperperfusion. Preoperative SPECT scanning and MR angiography may be useful in identifying potential cases of hyperperfusion.

References

1. Gourley JK, Heistad DD: Characteristics of reactive hyperemia in the cerebral circulation. *Am J Physiol* **246**:H52–H58, 1984
2. Heros RC, Scott RM, Kistler JP, et al: Temporary neurological deterioration after extracranial-intracranial bypass. *Neurosurgery* **15**:178–185, 1984
3. Lawton MT, Spetzler RF: Surgical strategies for giant intracranial aneurysm. *Acta Neurochir Suppl* **72**:141–156, 1999
4. Masaoka H, Klatzo I, Tomida S, et al: Role of circulatory disturbances in the development of post-ischemic brain edema. *Neurochem Pathol* **9**:21–29, 1988
5. Meyer FB, Huston J III, Riederer SS: Pulsatile increases in aneurysm size determined by cine phase-contrast MR angiography. *J Neurosurg* **78**:879–883, 1993
6. Peerless SJ, Wallace MC, Drake CG: Giant intracranial aneurysms, in Youmans JR (ed): *Neurological Surgery*, ed 3. Philadelphia: WB Saunders, 1990, Vol 3, pp 1742–1763
7. Perl J II, Turski PA, Masaryk TJ: MR angiography: techniques and clinical applications, in Atlas SW (ed): *Magnetic Resonance Imaging of the Brain and Spine*, ed 2. Philadelphia: Lippincott-Raven, 1996, pp 1547–1618
8. Schoser BGH, Heesen C, Eckert B, et al: Cerebral hyperperfusion injury after percutaneous transluminal angioplasty of extracranial arteries. *J Neurol* **244**:101–104, 1997
9. Sundt TM Jr, Sharbrough FW, Piepgras DG, et al: Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy: with results of surgery and hemodynamics of cerebral ischemia. *Mayo Clin Proc* **56**:533–543, 1981
10. Yoshimoto T, Houkin K, Kuroda S, et al: Low cerebral blood flow and perfusion reserve induce hyperperfusion after surgical revascularization: case reports and analysis of cerebral hemodynamics. *Surg Neurol* **48**:132–139, 1997

Manuscript received February 26, 2002.

Accepted in final form July 16, 2002.

Address reprint requests to: Hideki Murakami, M.D., Department of Neurosurgery, Ashikaga Red Cross Hospital, 3-2100 Honjo, Ashikaga-city, Tochigi 326-0808, Japan. email: hidekim@mail2.takauji.or.jp.