31 Posterior Communicating Artery Aneurysm: Surgical Trapping of a Giant Posterior Communicating Artery Aneurysm – Balloon Test Occlusion and Clip Ligation for Trapping

Amer Haj, Alexander Brawanski, Christina M. Wendl, and Karl-Michael Schebesch

Abstract

A 51-year-old man presented with a persistent headache and blurred vision. Magnetic resonance imaging (MRI) and digital subtraction angiography (DSA) showed a giant aneurysm of the right internal carotid artery (ICA) measuring approximately 30 × 25 mm. After a balloon test occlusion (BTO) of the right ICA, preoperative DSA showed good crossflow from the left-hand ICA via the anterior communicating artery (AcomA). Interdisciplinary discussion resulted in the decision to go for microsurgical treatment. The giant aneurysm was approached via a right-hand pterional craniotomy and completely excluded from circulation using the trapping technique with two clips on the ICA. Postoperative DSA confirmed the complete occlusion of the aneurysm. The patient did not develop any new neurological symptoms during the follow-up. The management of large and giant aneurysms of the PcomA by means of the trapping technique is the main topic of this chapter.

Keywords

Internal carotid artery · Posterior communicating artery aneurysm · Giant aneurysm · Microsurgical trapping · Balloon test occlusion · BTO · Crossflow

Patient

A 51-year-old man with no significant past medical history presented with a persistent headache and blurred vision.

Diagnostic Imaging

Magnetic resonance imaging (MRI) showed a giant aneurysm of the right internal carotid artery (ICA) with minor perianeurysmal gliosis; however, neither there was intra-aneurysmal thrombus nor calcification at the neck of the aneurysm. A subsequent digitally subtracted angiogram (DSA) revealed a giant aneurysm of the right posterior communicating artery (PcomA) with a fundus diameter of 30 × 25 mm. No additional aneurysms nor any other vascular abnormalities were detected. The preoperative balloon test occlusion (BTO) showed good crossflow from the left ICA via the anterior communicating

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artery (AcomA) and was tolerated for 20 min (Fig. 1).

Treatment Strategy

The treatment goal was to isolate the aneurysm from normal circulation in order to avoid any further growth and a potential rupture of said aneurysm. The case was extensively discussed among vascular neurosurgeons and interventional neuroradiologists. Clipping the aneurysm was considered difficult but possible, and should it be successful, it would be the preferred treatment modality since the mass effect of the aneurysm would immediately resolve. Endovascular coil occlusion was rejected since it would not have reduced the mass effect of the aneurysm; indeed, it may even have increased it. Flow diversion was considered risky due to the known phenomenon of delayed aneurysmal rupture after a flow diverter stent had been implanted in large and giant aneurysms. Parent vessel occlusion by trapping was the preferred backup plan should selective aneurysm clipping have turned out to be impossible. Given how well the patient had tolerated balloon test occlusion (BTO), sacrificing the parent artery without bypass surgery was deemed possible because of the apparently sufficient crossflow from the left ICA. After careful anesthetic assessment of cardiac and pulmonary risks and to prepare for the possibility that pressure and blood flow in the aneurysm and the parent artery would need to be reduced, everything was set up to enable cardiothoracic surgeons to induce deep hypothermic circulatory arrest, should it be required during the operation.

Treatment

Procedure, 26. 10. 2015: microsurgical clipping of a giant aneurysm of the right ICA with deep hypothermic circulatory arrest available on standby

Anesthesia: general anesthesia, TIVA

Technical equipment: Zeiss Pentero 900 operating microscope, indocyanine green angiography (ICG), Micro-Doppler, and neuromonitoring

Course of treatment: a general anesthetic was given and the patient intubated, placed on a ventilator, and laid out in a supine position. His head was fixed in a Mayfield holder and rotated 30° to the left with moderate posterior extension. The inguinal region on the right side was draped and prepared for the femoral artery to be cannulated, enabling it to be connected to the heart-lung machine should hypothermia need to be induced. After a pterional craniotomy, the optic chiasm was dissected, the carotid cistern opened, and cerebrospinal fluid (CSF) was released. The Sylvian fissure was opened and split. The visible parts of the aneurysm were dissected as well as the middle cerebral artery (MCA) and the anterior cerebral artery (ACA). Perforating vessels were detected and preserved. The three vessels connected to the aneurysm (ICA, MCA, and ACA) were temporarily clipped, which allowed the aneurysm to soften. However, clip reconstruction was not possible because the aneurysm could not be compressed any further. Afterward, the parent carotid artery was temporarily clipped. Doppler sonography on the MCA and ACA showed that flow conditions in both vessels were unaffected; therefore, the decision was made to trap the aneurysm. A plain permanent Sugita clip (Mizuho) was placed onto the ICA proximal to the aneurysm neck and a second permanent Yaşargil 90° curved clip (Aesculap) was attached distal to the neck of the aneurysm. Blood flow in the MCA and ACA was uncompromised, as confirmed by Micro-Doppler sonography. Indocyanine green (ICG) video angiography showed good perfusion of the MCA and ACA but no flow inside the aneurysm. Extracorporeal circulation was not set up. The surgical cavity was filled with sterile saline, the dura sutured, and the bone reimplanted. After surgery, the patient was admitted to the neurosurgical intensive care unit (ICU) (Fig. 2).

(a) Transsylvian view of the giant aneurysm and the adjacent vessels
(b) Temporary clipping of the parent ICA
(c) Monitoring the flow in the MCA and ACA by Doppler sonography
(d) Permanent clipping with a plain Sugita clip on the ICA proximal to the basis of the aneurysm and a second Yaşargil 90° curved clip attached distal to the neck of the aneurysm

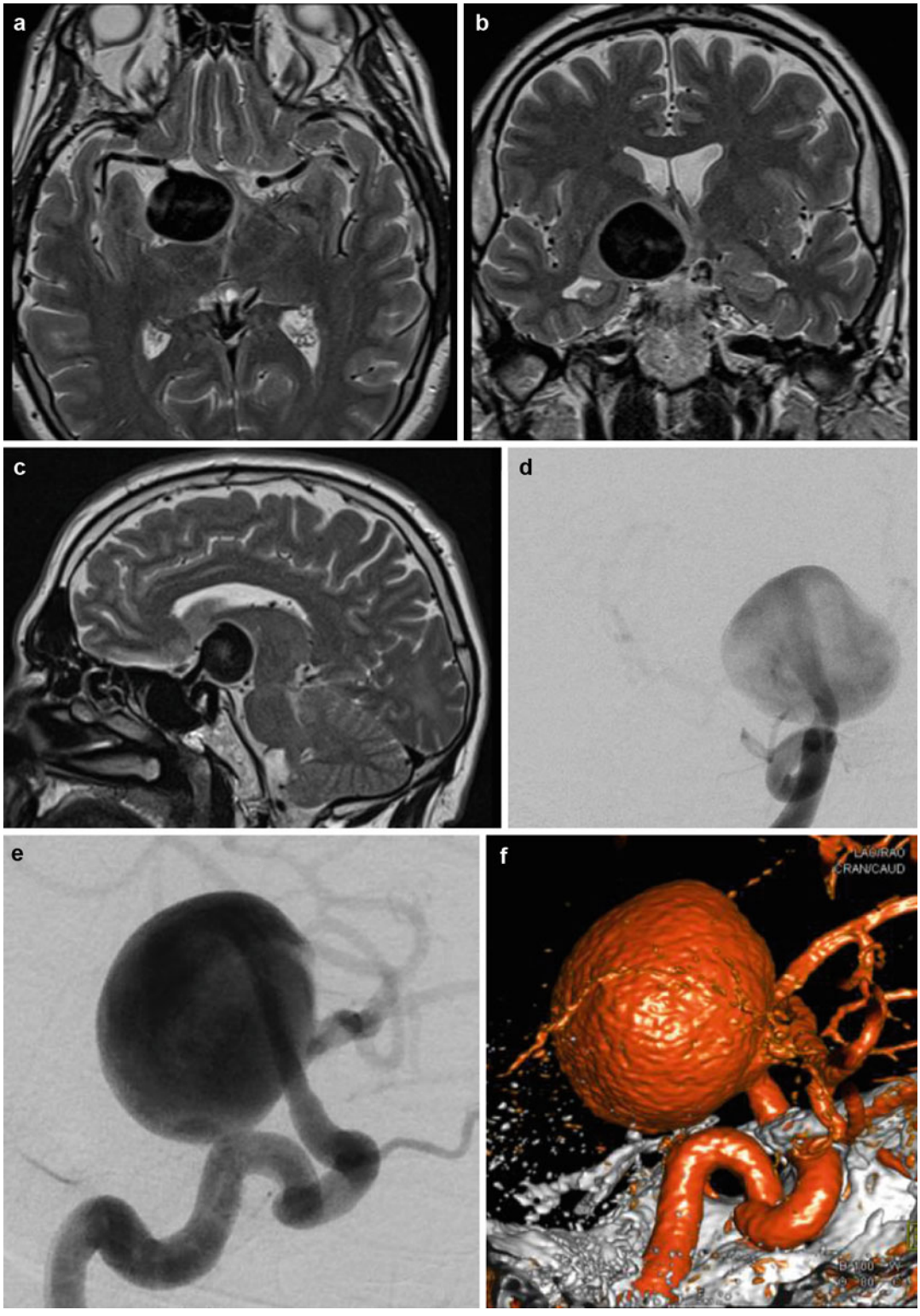

Fig. 1 (continued)

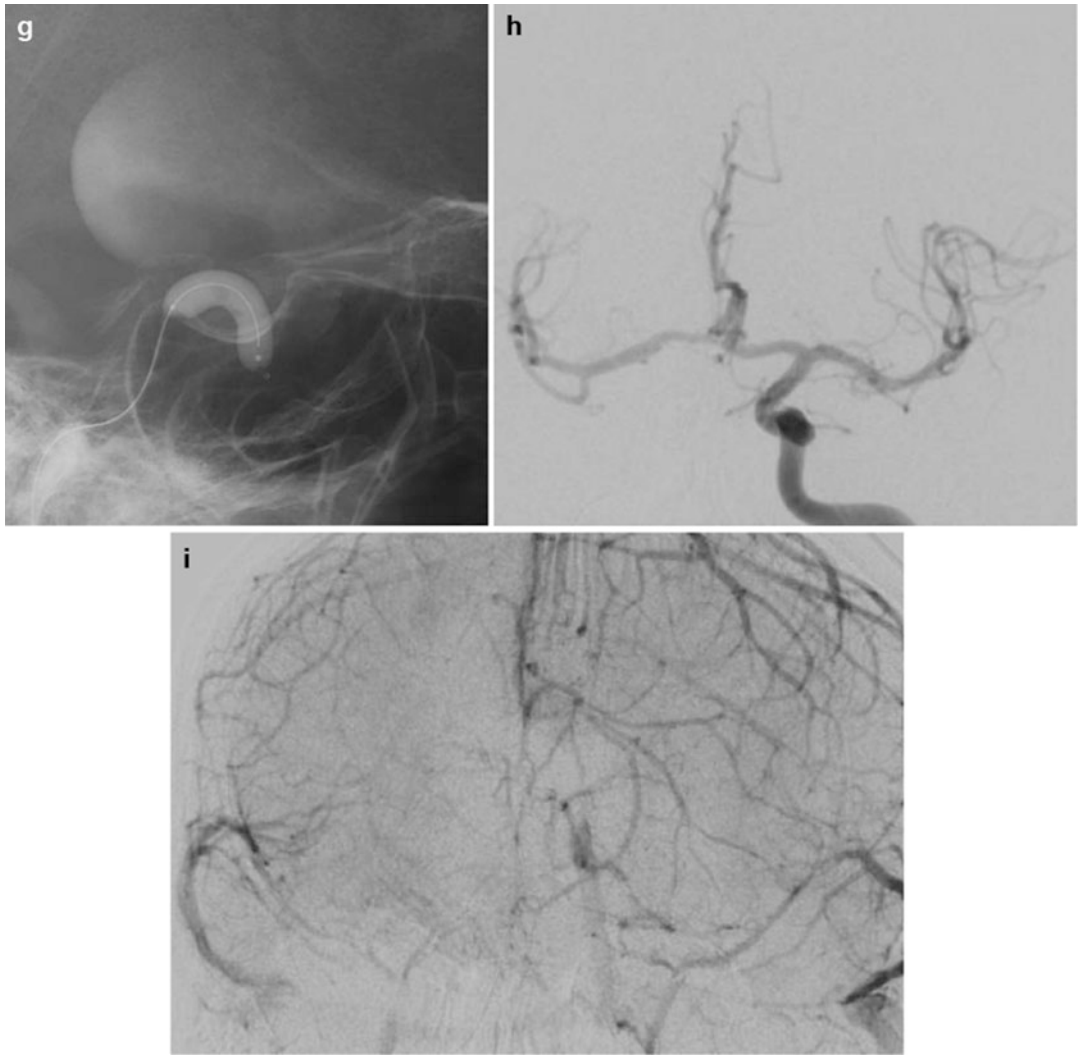

Fig. 1 Diagnostic imaging in a symptomatic giant right-hand ICA/PcomA aneurysm. Axial (**a**), coronal (**b**), and sagittal (**c**) T2WI MRI showed a giant aneurysm of the right ICA surrounded by minor gliosis and with no calcification of the aneurysm wall. Baseline DSA (posterior-anterior projection (**d**), lateral projection (**e**), 3D reconstruction from rotational DSA images (**f**)) showed the supraclinoid origin of the narrow aneurysm neck. The right PcomA was not opacified. Balloon test occlusion of the right ICA (**g**) showed crossflow from the left ICA to the right anterior circulation via the AcomA (**h**) with almost synchronous venograms of both hemispheres (**i**). The BTO was tolerated for 20 min without a neurological deficit

(e) Final Micro-Doppler test confirming sufficient flow after permanent clipping and trapping has been carried out
(f) Final photograph after the aneurysm has been trapped

Duration: 3 h 27 min
Complications: none

Follow-Up Examinations

Postoperative CT ruled out any hemorrhagic or ischemic complications. DSA conducted 1 week after surgery showed complete occlusion of the aneurysm together with the parent artery and neither vasospasm nor any compromised intracranial arterial vessels (Fig. 3).

Clinical Outcome

The clinical status of the patient was rated as excellent, mRS 0. Moreover, the previous headache and blurred vision had abated within 4 weeks.

Discussion

Giant aneurysms are rare but potentially lethal and account for approximately 5% of all intracranial aneurysms (Barrow and Alleyne 1995). Aneurysms located on the PcomA usually

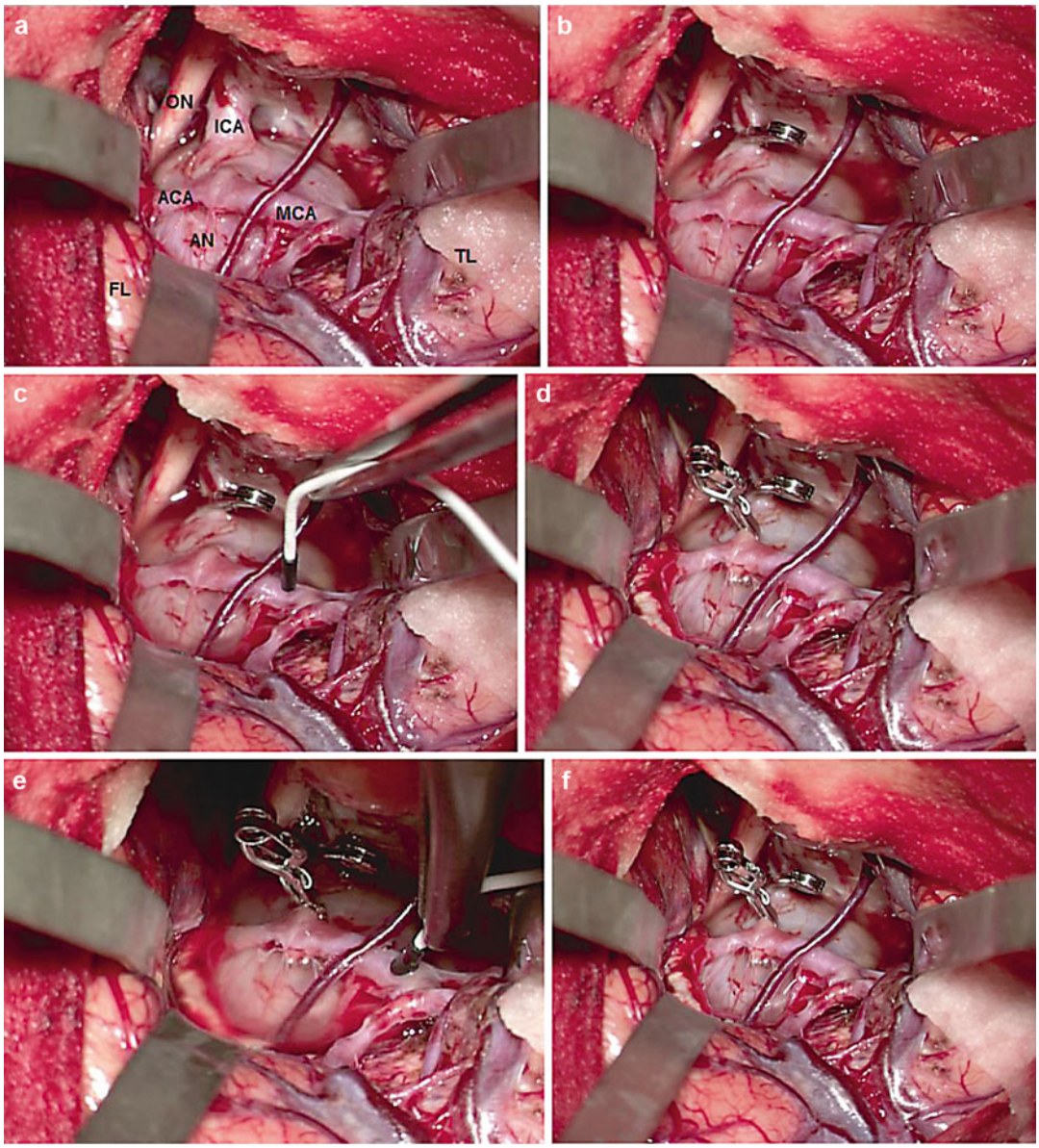

Fig. 2 Intraoperative microscopic transsylvian view of the giant aneurysm, ICA and PcomA. Exposure of the PcomA via the pterional approach (AN, aneurysm; FL, frontal lobe; TL, temporal lobe; ON, optic nerve)

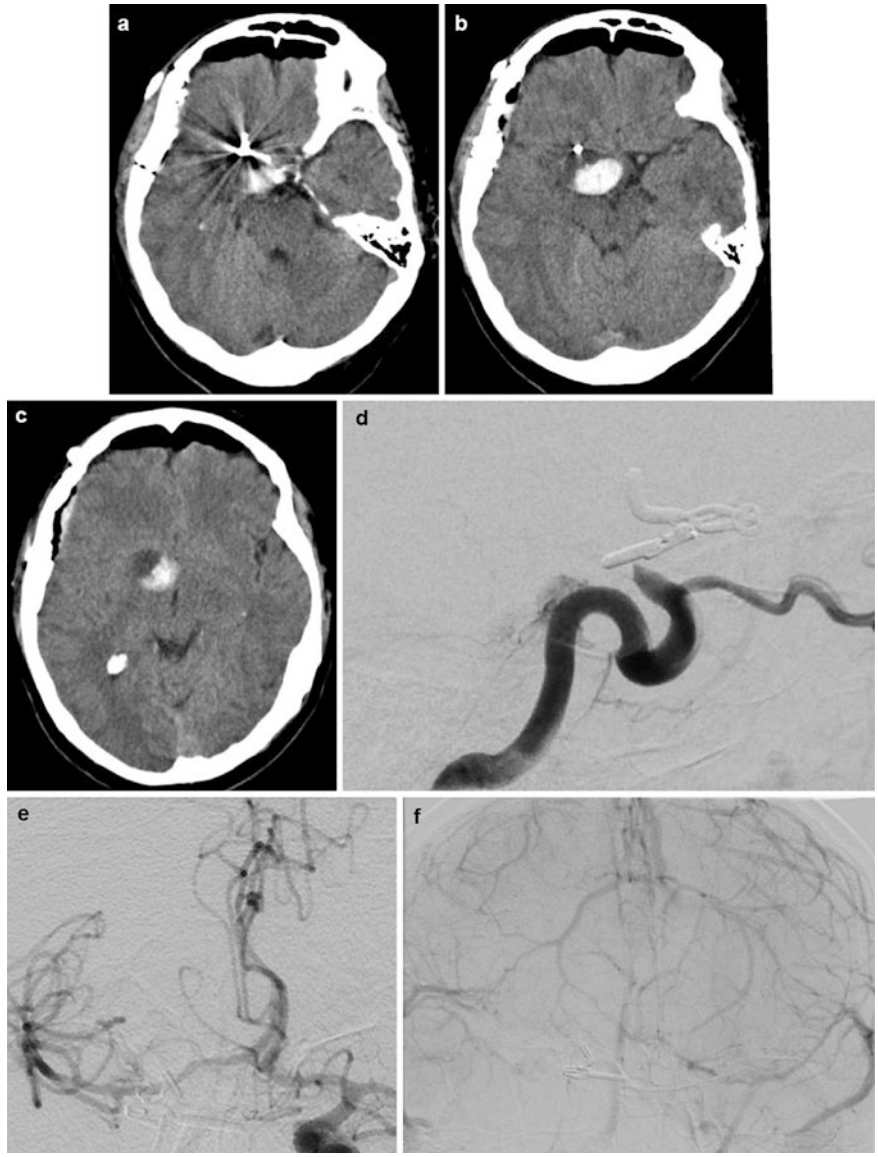

Fig. 3 Postoperative imaging after the surgical trapping of a giant right ICA/PcomA aneurysm. NCCT after microsurgical clipping of the right ICA proximal and distal to the origin of a giant PcomA aneurysm (**a**–**c**) showed hyperdense thrombus (T) inside the aneurysm sac but no hemorrhage or ischemic lesions. Follow-up DSA with contrast medium injected into the right-hand ICA confirmed the clip ligation of the said vessel in the supraclinoid segment (lateral view (**d**)). Injecting contrast medium into the left-hand ICA showed the crossflow from the left to the right anterior circulation via the AcomA (posterior-anterior view (**e**)) and the synchronous venograms of the both anterior circulations (posterior-anterior view (**f**))

present with clinical symptoms or ruptures before growing giant in size. Therefore, giant PcomA aneurysms are extremely rare and constitute only 4% of all giant intracranial aneurysms (Drake 1979).

These lesions are adjacent to critical neuronal structures. The perforating branches of the PcomA have been found to vascularize the medial and lateral hypothalamus, the cerebral peduncle, and the optic tract. Giant PcomA aneurysms may

also cause severe headaches, seizures, visual acuity loss, double vision, ptosis, and mydriasis due to oculomotor nerve palsy and other cranial nerve deficits (Barrow and Alleyne 1995; Brisman et al. 2006; Gnanalingham et al. 2003). Our patient presented with a persistent headache and blurred vision.

Given the fact that PcomA aneurysms in general have a distinct hemodynamic environment compared to other cerebral aneurysms, giant PcomA aneurysms are known to have a relatively high rupture risk. The 5-year cumulative rupture rate is as high as 50% as reported in the International Study of Unruptured Intracranial Aneurysms (ISUIA) trial (Wiebers et al. 2003). When left untreated, giant aneurysms have a reported mortality rate of 68% within 2 years after diagnosis and of 85% after 5 years from diagnosis (Misra et al. 2014). Therefore, effective and fast management is vital for patients with such a poor prognosis.

Microsurgical clipping and endovascular therapy are treatment options for giant intracranial aneurysms. However, the complication rates of endovascular therapy and the need for supplementary treatment are significant. Jahromi et al. (2008) reported on the endovascular treatment of 38 patients with giant aneurysms and found a 26% morbidity and a 29% mortality in their series.

Microsurgical clipping of giant ICA aneurysms is considered a high-risk procedure with mortality rates of 6% to 22%. However, procedure-related morbidity and mortality rates have significantly reduced in recent years, mainly due to modern preoperative neuroimaging with MRI, DSA, and contrast-enhanced CT as well as intraoperative diagnostic tools such as Doppler ultrasound and intraoperative video angiography and not at least due to interdisciplinary assessment by neuroradiologists and neurosurgeons (Gewirtz and Awad 1996; Lawton and Spetzler 1995; Locksley et al. 1966; Wiebers et al. 2003).

Direct surgery on PcomA aneurysms is even more challenging because of the deep and partly hidden anatomical location of the parent vessel, the presence of important neuronal structures such as the optic chiasm and the oculomotor nerve adjacent to the aneurysm, and the difficulty of identifying and preserving perforating arteries (Matsukawa et al. 2014).

An excellent or good clinical outcome has been achieved in 61% to 87% of patients undergoing surgical treatment for giant intracranial aneurysm (Lawton and Spetzler 1995; Yaşargil 1984). Velat et al. (2012) in a series of 11 patients with giant PcomA aneurysms reported a 36% morbidity and a 18% mortality.

Before surgery, the patient's tolerance of ICA occlusion was tested to reduce the risk of ischemia. An angiographic balloon test occlusion (BTO) is crucial for evaluating ischemic risks before complete occlusion (Sorteberg et al. 2008). This preoperative test is used to evaluate whether the carotid artery concerned could be sacrificed and to ascertain the capacity of collateral blood flow. Poor collaterals can be compensated for by an extra-intracranial bypass before the parent vessel occlusion (Lesley and Rangaswamy 2009). The routine usage of clinical BTO of the ICA has been associated with significantly reduced post-occlusion morbidity (Linskey et al. 1994). The complications rate of BTO is approximately 3.2% (Mathis et al. 1995).

Since our patient had passed the BTO, thus showing good crossflow and collateral circulation from the left ICA via the AcomA, a surgical occlusion of the parent artery was deemed possible with no bypass required. The BTO was supplemented by the intraoperative use of Doppler sonography and indocyanine green angiography (ICG), for real-time monitoring of cerebral hemodynamics. Both methods are proven to be reliable and sensitive intraoperative tools (Balamurugan et al. 2011; Stendel et al. 2000).

During surgery, the carotid artery was temporarily clipped and Doppler sonography showed good blood flow in the MCA and ACA. After the BTO, good crossflow was confirmed by Doppler sonography and ICG; therefore, the aneurysm was occluded with the trapping technique, and the parent artery was sacrificed.

To provide a safety net to prevent emergency bypass surgery should there be an intraoperative aneurysm rupture, and in line with our policy of trying to preserve the parent arteries whenever

possible, preparations were made to enable the immediate induction of deep hypothermic cardiac arrest. However, crossflow from the contralateral side via the AcomA was high enough to mean that no further intervention was required. The patient did not show any clinical or radiographic perfusion deficits.

One week after surgery, the patient was still not presenting with any neurological deficits, vision had returned to normal, and the headaches had disappeared. Postoperative imaging including NCCT and DSA showed complete occlusion of the aneurysm, preserved perforators, and no cranial nerve deficit (especially N. III, IV, V, and VI). A recent follow-up phone interview with the patient confirmed that he is still healthy with no neurological deficit.

Therapeutic Alternatives

Endovascular Coil Occlusion
Endovascular Parent Vessel Occlusion
Microsurgical Clipping

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