



Is surgical parent artery occlusion effective for intracranial aneurysms measuring over 10 mm in size? Result from long-term follow-up of size changes and outcomes

Shintaro Arai¹ · Tohru Mizutani¹ · Tatsuya Sugiyama¹ · Kenji Sumi¹ · Takato Nakajo¹ · Masaki Matsumoto¹ · Katsutoshi Shimizu¹

Received: 4 October 2018 / Accepted: 26 November 2018 / Published online: 4 December 2018
© Springer-Verlag GmbH Austria, ein Teil von Springer Nature 2018

Abstract

Background There have been no long-term follow-up reports pertaining to chronological size changes in large or giant unruptured intracranial aneurysms treated with surgical parent artery occlusion (PAO). The object of this study is to investigate the utility and safety of surgical PAO by conducting a long-term follow-up of chronological aneurysm size changes and outcomes.

Methods A retrospective study of 21 unruptured intracranial aneurysms measuring over 10 mm (20 patients) treated with surgical PAO in the period 2012–2017 was conducted. For aneurysms presenting with anterior circulation, high/low flow bypass was chosen and carried out concomitantly on the basis of preoperative balloon occlusion test results. Aneurysm size before and after surgery was evaluated chronologically using maximum diameter measurements taken from the same slice of MRI T2-weighted images. Moreover, post-surgery outcomes were evaluated according to a modified Rankin scale (mRS) at discharge.

Results PAO aiming for blind-alley formation was performed in 20 of 21 aneurysms (95.2%). Aneurysm size reduction was confirmed in 20 aneurysms (95.2%) after proper PAO, with an average reduction rate of 63.1% (range, 28–95%), during an average follow-up period of 27 months (range, 4–54 months). Eighteen (90.4%) of the 20 patients with 21 aneurysms returned to previous life with mRS score 0–2. With regard to preoperative symptoms, diplopia and visual impairment had improved in three patients (50%) and one patient (100%), respectively. Ischemic complications had occurred in five patients, two (9.6%) of whom were symptomatic and three (14.3%) were asymptomatic. The mortality rate in this study was 0%.

Conclusions Surgical PAO for unruptured intracranial aneurysms measuring over 10 mm has been shown to be an effective method of treatment, eliciting a reduction in aneurysm size.

Keywords Blind-alley formation · Flow alteration · Giant aneurysms · Large aneurysms · Perforators · Size changes · Surgical parent artery occlusion

This article is part of the Topical Collection on *Vascular Neurosurgery—Aneurysm*

Previous presentation: The contents of this paper were presented in the 47th Annual Meeting of the Japanese Congress for Stroke 2018 on March 16, 2018.

✉ Shintaro Arai
sarai@med.showa-u.ac.jp

¹ Department of Neurosurgery, Showa University School of Medicine, 5-8 Hatanodai 1-chome, Shinagawa-ku, Tokyo 142-8666, Japan

Introduction

For large or giant unruptured cerebral aneurysms, curative surgical treatments, such as clipping and trapping, are generally difficult, and parent artery occlusion (PAO), to induce thrombosis by reducing the blood flow pressure into the aneurysm, is often required [6]. In surgical PAO, having observed the perforators surrounding the aneurysm, it is possible to carry out blind-alley formation with ligation or with placement of clip properly, and an added advantage is that it can be performed concomitantly with bypass surgery. In order to establish whether or not these types of flow-alteration treatments

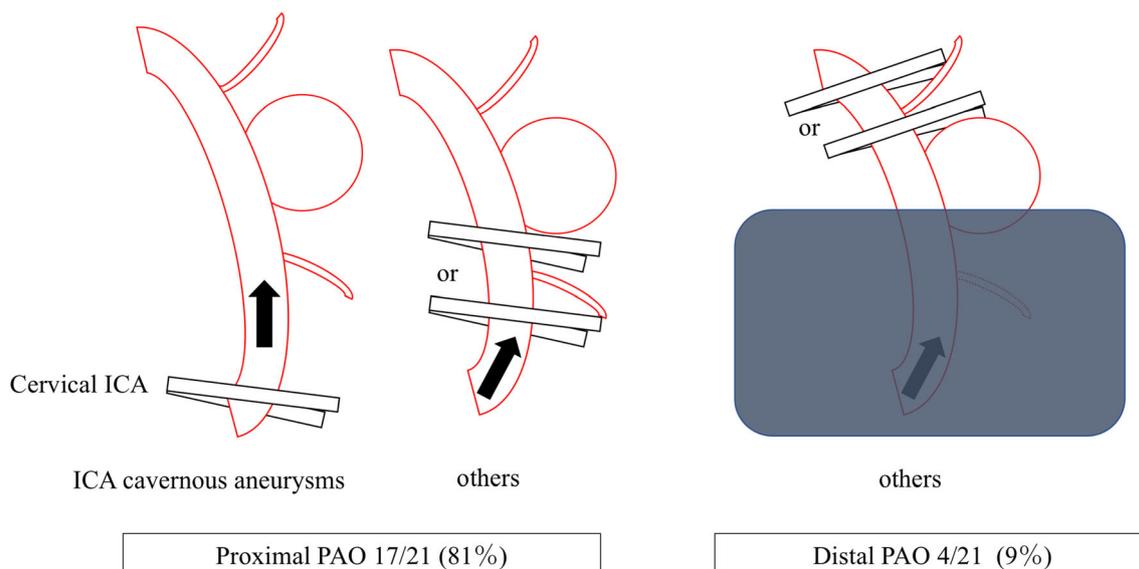


Fig. 1 Therapeutic strategies and methods

can actually cure, long-term aneurysm size reduction needs to be assessed. Although there have been some reports of the effect of surgical PAO for large or giant unruptured intracranial aneurysm [8, 17], no long-term detailed follow-up pertaining to chronological size changes have been reported. In terms of long-term follow-up post-endovascular PAO, Clarençon et al. have reported confirmed size reduction in all 18 cases of internal carotid artery aneurysm observed for an average period of 6.1 years [3]. In contrast, there have also been reports of recanalization of the parent artery post-treatment in 0–8.3% of cases [2, 3, 11, 13, 22]. In the current paper, we have investigated the utility and safety of surgical PAO treatment for unruptured intracranial aneurysms measuring over 10 mm by conducting a long-term follow-up of chronological aneurysm size changes and outcomes post-surgery.

Methods

All patients agreed to provide all information including images for the present study, and approval from the ethical committee was obtained for this study.

A retrospective study of 21 aneurysms (20 patients) of unruptured intracranial aneurysms (measuring over 10 mm) treated with surgical PAO (excluding trapping) in the period 2012–2017 was conducted. Occlusion of the proximal artery is the first choice in PAO procedures. For internal carotid artery (ICA) aneurysms located in cavernous portion, ICA was ligated at cervical portion. For the other aneurysms, meanwhile, a clip was placed at the portion as near as the aneurysm without including major artery aiming for the blind-alley formation of the aneurysm. However, in aneurysms wherein it is difficult

to safely expose the proximal parent artery or when perforating or cortical arteries have been confirmed from the aneurysm itself and it has been judged that there is an increased risk of symptoms from performing a proximal PAO, occlusion of the distal artery is chosen. The schema of therapeutic strategies and methods are shown in Fig. 1. In PAO for ICA aneurysms, a preoperative balloon occlusion test (BOT) was carried out in all patients, and in the aneurysms of vertebral artery (VA), basilar artery (BA), or posterior cerebral artery (PCA), superselective BOT was occasionally conducted. For aneurysms presenting with anterior circulation including middle cerebral artery (MCA), high/low flow bypass was chosen and carried out concomitantly on the basis of preoperative BOT results. Aneurysm size before and after surgery was evaluated chronologically by a neurosurgeon and neuroradiologist, using averaged maximum diameter measurements taken from the same slice of MRI T2-weighted images. Moreover, post-surgery outcomes were evaluated by a neurosurgeon according to mRS at discharge.

Results

Patient characteristics

All patient data are summarized in Table 1. Of the 20 patients, 7 were male and 13 were female, with an average age of 59.2 years (range, 29–80 years). Of the 21 aneurysms, 10 aneurysms (47.6%) located in ICA, 2 aneurysms (9.5%) located in MCA, 6 aneurysms (28.6%) located in VA, 1 aneurysm (4.8%) located in BA, and 2 aneurysms (9.5%) located in PCA, with an average size

Table 1 Patient characteristics and results

Patient	Age (years)/sex	Aneurysm		Thrombosed	Presenting symptoms	BOT tolerance	PAO	Follow-up		Reduction rate (%)	
		Site	Size (mm)					Time (months)	Outcome (mRS)		Complication
1	69/F	Rt. ICA cavernous	20	No	–	Yes	Proximal/high flow bypass	47	0	–	62
2	80/F	Rt. ICA PcoA	29.2	No	–	Yes	Proximal/low flow bypass	33	4	Symptomatic infarction	140
3	71/F	Rt. ICA AChoA	21.7	No	–	Yes	Proximal/low flow bypass	17	0	–	66
4	39/M	Rt. ICA ophthalmic	30.5	Yes	Visual loss	Yes	Distal/low flow bypass	54	0	–	52
5	65/F	Rt. ICA cavernous	26.1	No	Diplopia	Yes	Proximal/low flow bypass	39	1	Asymptomatic infarction	76
6	64/F	Lt. ICA cavernous	40.3	No	Diplopia	Yes	Proximal/high flow bypass	35	2	Asymptomatic infarction	55
6	65/F	Rt. ICA cavernous	26.7	No	Diplopia	Yes	Proximal/high flow bypass	25	2	–	45
7	65/F	Lt. ICA cavernous	26.7	No	Diplopia	No	Proximal/high flow bypass	31	1	–	52
8	39/F	Lt. ICA cavernous	30	No	Diplopia	No	Proximal/low flow bypass	32	1	–	72
9	60/F	Lt. MCA M2	38.3	Yes	–	N/A	Proximal/low flow bypass	19	0	–	81
10	76/F	Lt. MCA M1	27	No	–	N/A	Distal/high flow bypass	40	4	Symptomatic infarction	35
11	59/F	Rt. ICA cavernous	27	No	Diplopia	No	Proximal/high flow bypass	5	0	–	60
12	72/M	Rt. VA	14	Yes	–	N/A	Proximal	9	0	–	94
13	59/F	Rt. PCA P2	14	No	–	Yes	Distal	20	0	Asymptomatic infarction	43
14	64/F	Rt. VA	16	Yes	–	Yes	Proximal	22	0	–	91
15	46/M	Rt. PCA P1/2	14.4	No	Symptomatic infarction	No	Distal	40	0	–	72
16	52/M	Rt. VA	20.5	Yes	–	Yes	Proximal	25	0	–	37
17	29/F	BA	22	No	–	Yes	Proximal	14	0	–	28
18	69/M	Rt. VA	11	No	–	N/A	Proximal	4	0	–	79
19	47/M	Lt. VA	10	No	–	N/A	Proximal	6	0	–	95
20	59/M	Rt. VA	11	No	–	N/A	Proximal	46	0	–	68

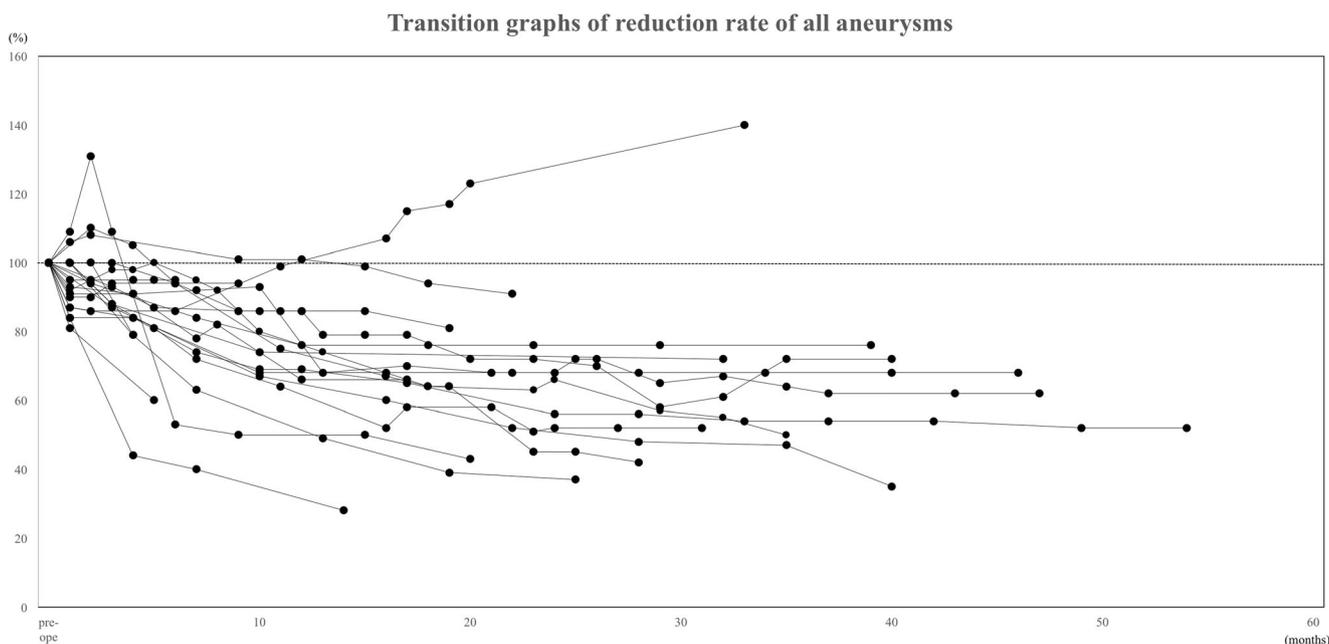


Fig. 2 Transition graphs of reduction rate of all aneurysms

of 22.7 mm (range, 10–40.3 mm), and 5 aneurysms (23.8%) of confirmed partial thrombosis prior to surgery. There were eight symptomatic aneurysms (38.2%), including six patients with double vision (28.6%), one patient with visual loss (4.8%), and one patient with cerebral infarction onset (4.8%). The PAO breakdown in terms of proximal versus distal artery occlusion was 17 aneurysms (81%) to 4 aneurysms (19%), respectively.

Aneurysm size changes

Figure 1 details the size changes for all patients. PAO aiming for blind-alley formation was performed in 20 of 21 aneurysms (95.2%). Aneurysm size reduction via MRI T2-weighted images was confirmed in 20 aneurysms (95.2%) after proper PAO, with an average reduction rate of 63.1% (range, 28–95%), during an average follow-up period of 27 months (range, 4–54 months). On the other hand, the only one aneurysm (patient 2) was treated with insufficient blind-alley formation; aneurysm size increased up to 140%.

In patient 1 (Fig. 2), a right ICA cavernous giant aneurysm showed typical shrinkage with reduction rate of 62% at 47 months post-surgery after concomitant proximal PAO and high flow bypass.

In patient 2 (Fig. 3), a right ICA-posterior communicating artery (PcoA) giant aneurysm was treated with proximal PAO and low flow bypass. Digital subtraction angiography (DSA) on the next day and 14th day after surgery showed the

retrograde flow into the aneurysm through PcoA branched from the midst of the aneurysm dome and right ophthalmic artery (OA) via right middle meningeal artery, respectively. Aneurysm had begun to increase in size from 11 months after surgery, reaching to 140% at 33 months due to insufficient blind-alley formation.

In patient 16 (Fig. 4), a right VA large thrombosed aneurysm was treated with proximal PAO. No infarction occurred in the area of right posterior inferior cerebellar artery (PICA) and anterior spinal artery (ASA) branched from the VA just distal to the aneurysm. Aneurysm size had reduced to 37% at 25 months after surgery.

Five aneurysms of pre-surgery partial thrombosis were included (Fig. 5). Of the five aneurysms, one aneurysm showed a temporary increase after surgery (patient 14); however, two aneurysms showed early-stage shrinkage (patients 4 and 16).

Three aneurysms temporarily enlarged after surgery (Fig. 6; patients 6, 13, 14).

Neither recanalization of the parent artery post-surgery nor de novo aneurysm due to high blood flow were recognized.

Outcome

Eighteen (90.4%) of the 20 patients with 21 aneurysms returned to previous life with mRS score 0–2.

With regard to preoperative symptoms, diplopia and visual impairment had improved in three out of six patients (50%) and one patient (100%), respectively.

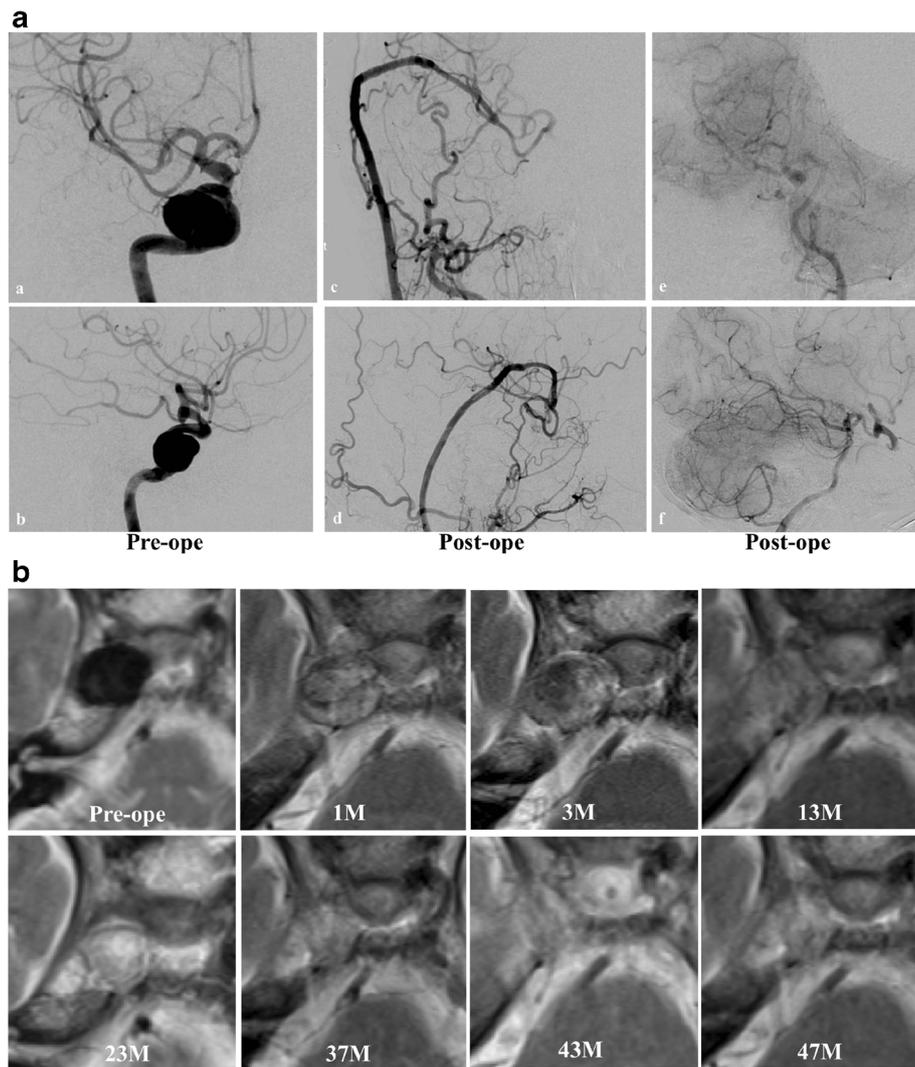


Fig. 3 (A) **a** AP view of right ICA, **b** lateral view of right ICA, **c** AP view of right common carotid artery, **d** lateral view of right common carotid artery, **e** AP view of left VA, **f** Lateral view of left VA. (B) MRI T2-weighted images. (C) The black line is the transition graph of patient 1

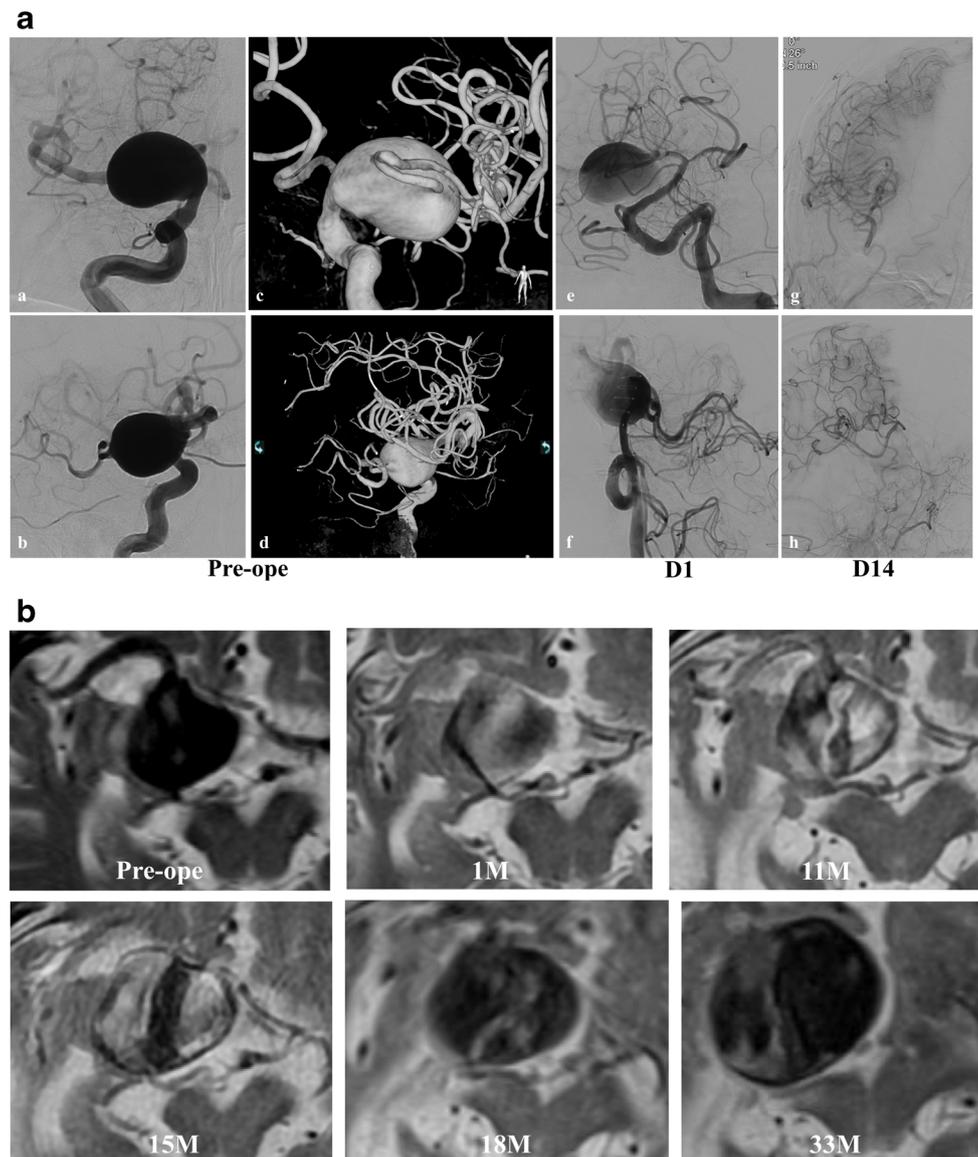


Fig. 4 (A) **a** AP view of right ICA; **b** lateral view of right ICA; **c**, **d** 3D view of right ICA—PcoA branched from dome of the aneurysm; **e**, **f** DSA on the next day after surgery showed the retrograde flow into the aneurysm through PcoA branched from the midst of the aneurysm dome; **g**, **h** the retrograde flow through right ophthalmic artery (OA) via right middle

meningeal artery was also confirmed by DSA on the 14th day post-surgery. (B) MRI T2-weighted images. (C) Three days and 17 months after surgery, the new infarctions in the area of the PcoA perforator and AChoA were found, respectively. (D) The black line is the transition graph of patient 2

Ischemic complications had occurred in five patients, two (9.6%; patients 2, 10) of whom were symptomatic and three (14.3%; patients 5, 6, 13) were asymptomatic. Patient 2 with right ICA-PcoA giant aneurysm had prolonged left hemiparesis and sensory impairment due to a new infarction in the distribution of the PcoA perforator occurred on the third post-surgical day, then she was transferred to a rehabilitation hospital with mRS score 4 at 2 months after surgery. At 17 months post-surgery, furthermore, along with progressive thrombosis of the aneurysm, another infarction occurred in the distribution of the anterior choroidal artery (AChoA)

branched from the aneurysm. Patient 10 with left M1 giant aneurysm presented right hemiparesis 10 h after surgical distal M1 occlusion and high flow bypass due to delayed infarction in the area of lenticulostriate artery (LSA) branched from the aneurysm. Right hemiparesis was prolonged; however, she returned home with mRS score 4 after rehabilitation. In patients 5 and 6, a punctate infarction of the frontal cortex, as suggested by the artery-to-artery embolism, was confirmed. In patient 13, a right PCA (P2) aneurysm was treated with distal PAO. An infarction of the medial temporal lobe occurred due to the lack of peripheral perfusion in parent artery,

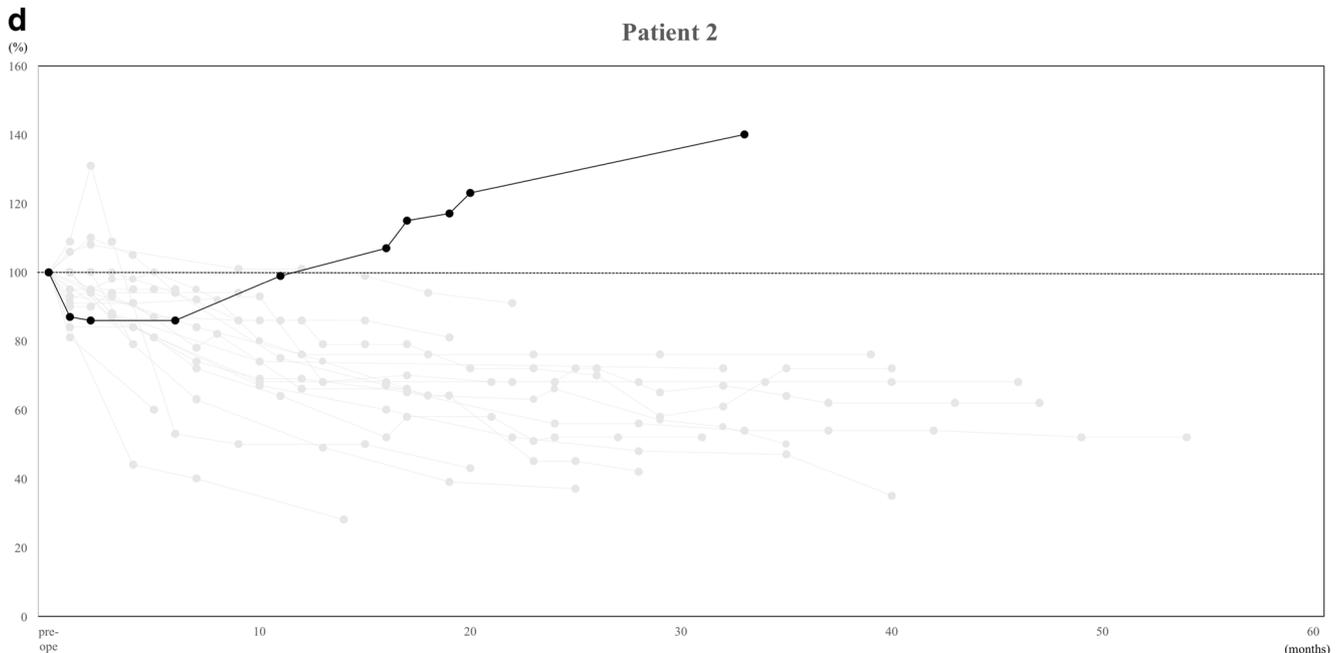
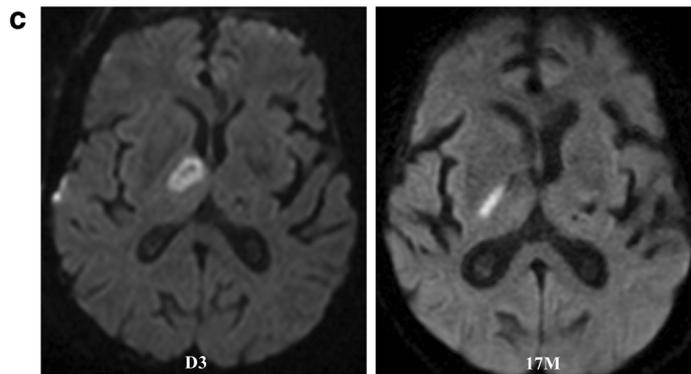


Fig. 4 continued.

while no infarction occurred in the P1 or PcoA perforator area.

There were no other complications and the mortality rate was 0%.

Discussion

Long-term size changes and morbidity/mortality

To confirm the effectiveness of PAO for large and giant intracranial aneurysms, long-term size reduction of aneurysm should be demonstrated. The present study showed 95.2% of the intracranial aneurysms revealed long-term size reduction after the proper PAO aiming for blind-alley formation as much as possible without including major arteries other than perforators. Nevertheless, in the aneurysm treated with insufficient blind-alley formation, such as that of patient 2, aneurysm size

increased again, and it becomes necessary to pursue alternatives, such as flow diverter treatment.

No obvious characteristics of aneurysm size changes were found in a comparison of anterior versus posterior circulation, proximal PAO versus distal PAO, and presence versus absence of thrombosis.

In terms of safety, results from the current series showed that morbidity (9.6%) and mortality (0%) are comparable with past reports of morbidity (6%) and mortality (9–13%) involving surgical clipping of large or giant internal carotid artery aneurysms [4, 5, 12, 16, 20, 21]. Furthermore, the current results can be interpreted as being equivalent when compared with reports [2, 9, 11, 13, 22] from endovascular treatments of symptomatic infarction (0–12.3%) and morbidity (0–8%). Moreover, although recanalization rates of 0–8.3% have been reported for endovascular treatments [2, 3, 11, 13, 22], recanalization in the current series was 0%, indicating that surgical PAO has an extremely low risk. Although flow diverters have been approved for the treatment of large and giant aneurysms

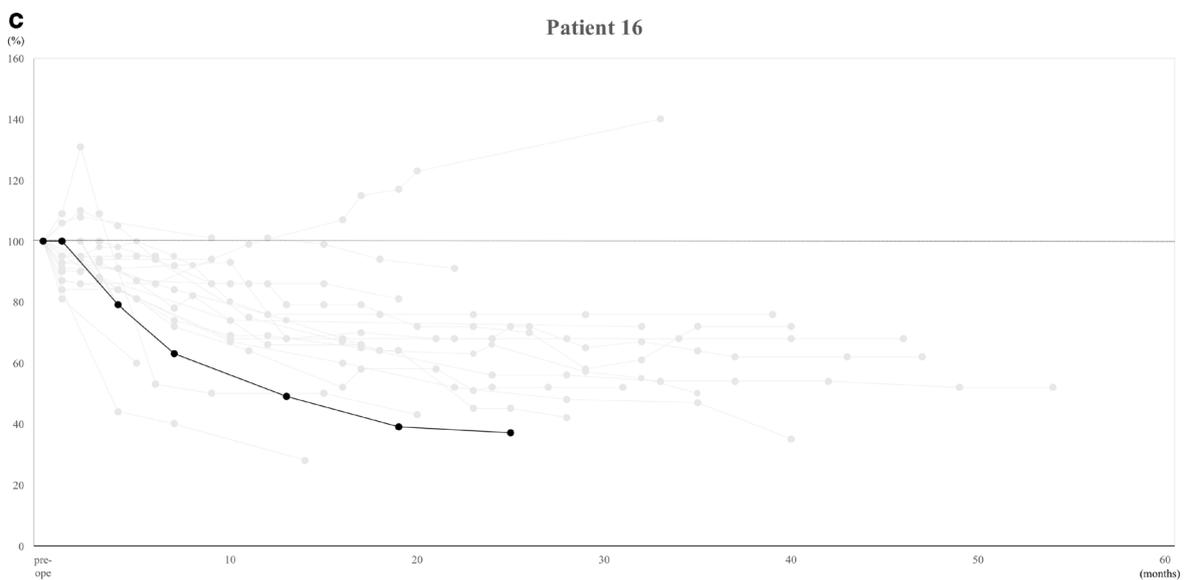
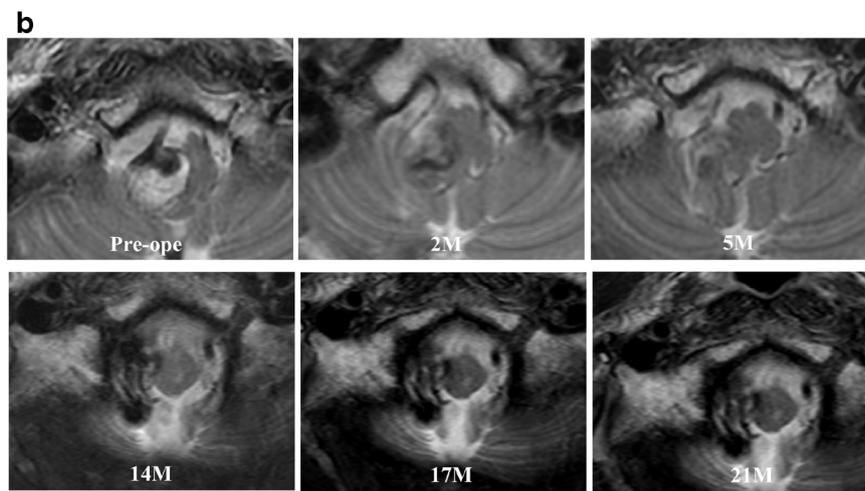
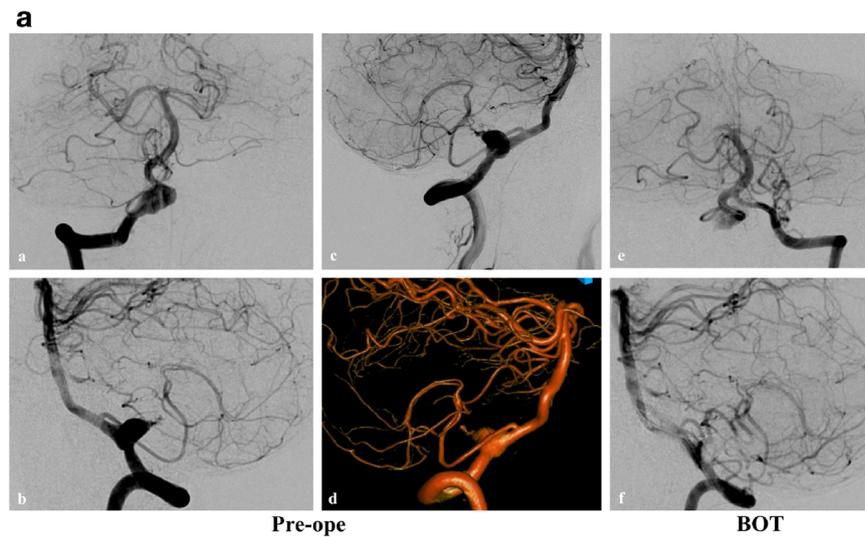


Fig. 5 (A) **a** AP view of right VA; **b** lateral view of right VA; **c** right PICA and ASA on the distal side of the aneurysm were confirmed; **d** 3D view of right VA; **e, f** The flow from left VA via the union to right PICA and ASA was confirmed by BOT on the proximal side of the aneurysm. (B) MRI

T2-weighted images. (C) The black line is the transition graph of patient 16. The black lines are the transition graphs of pre-surgery thrombosed aneurysms. The black lines are the transition graphs of aneurysms of temporary size increase

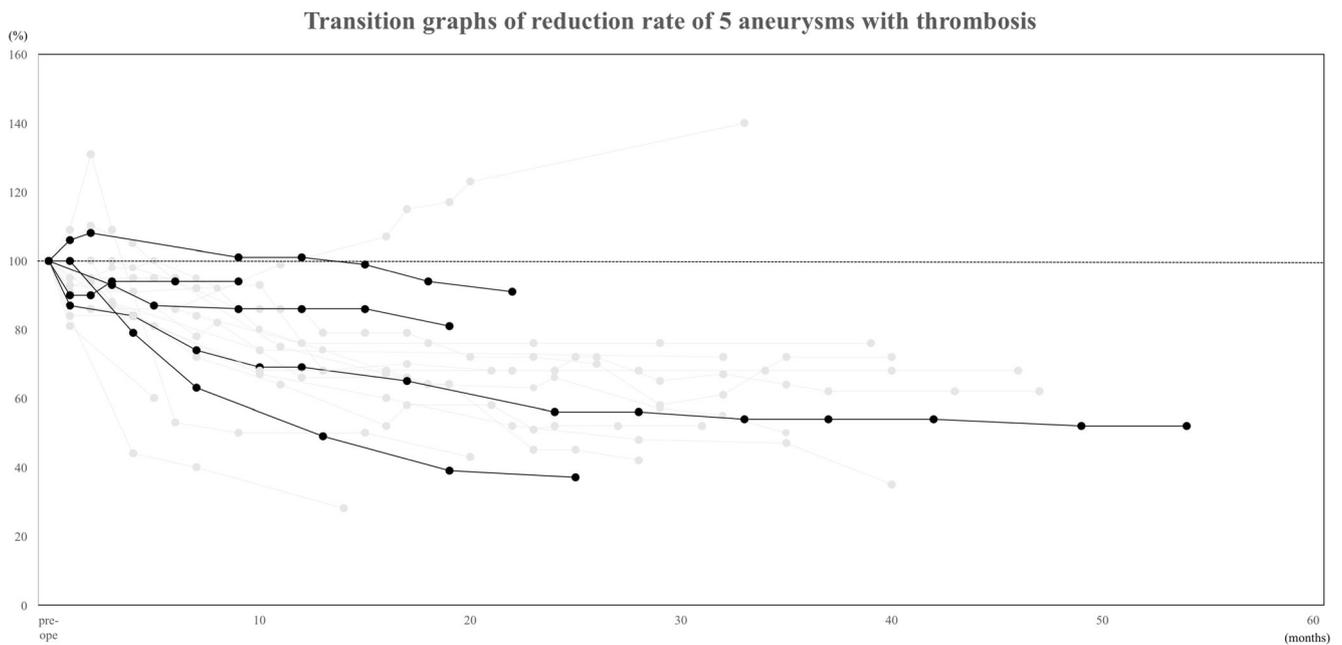


Fig. 6 Transition graphs of reduction rate of five aneurysms with thrombosis

especially for ICA cavernous, the results of FIAT trial have showed that flow diversion is not as safe and effective as hypothesized with 5.3% rate of morbidity and 10.7% rate of mortality [18].

Surgical design and infarction patterns

Table 2 shows infarction patterns, the hemodynamic conditions of perforating and cortical arteries of the aneurysmal areas, as well as the presence or absence of infarction.

The current series had five patients with postoperative cerebral infarctions; however, cerebral angiography suggests that artery-to-artery embolism was the cause for two of five patients. Of the remaining three patients, one (patient 13) presented with a right PCA (P2) aneurysm, with an adult-type PcoA branched from the dome. Tolerance was confirmed with a superselective BOT at the right P1, and distal PAO was performed since exposing the proximal parent artery proved difficult during the surgery. As such, infarction was not found in the P1 perforator

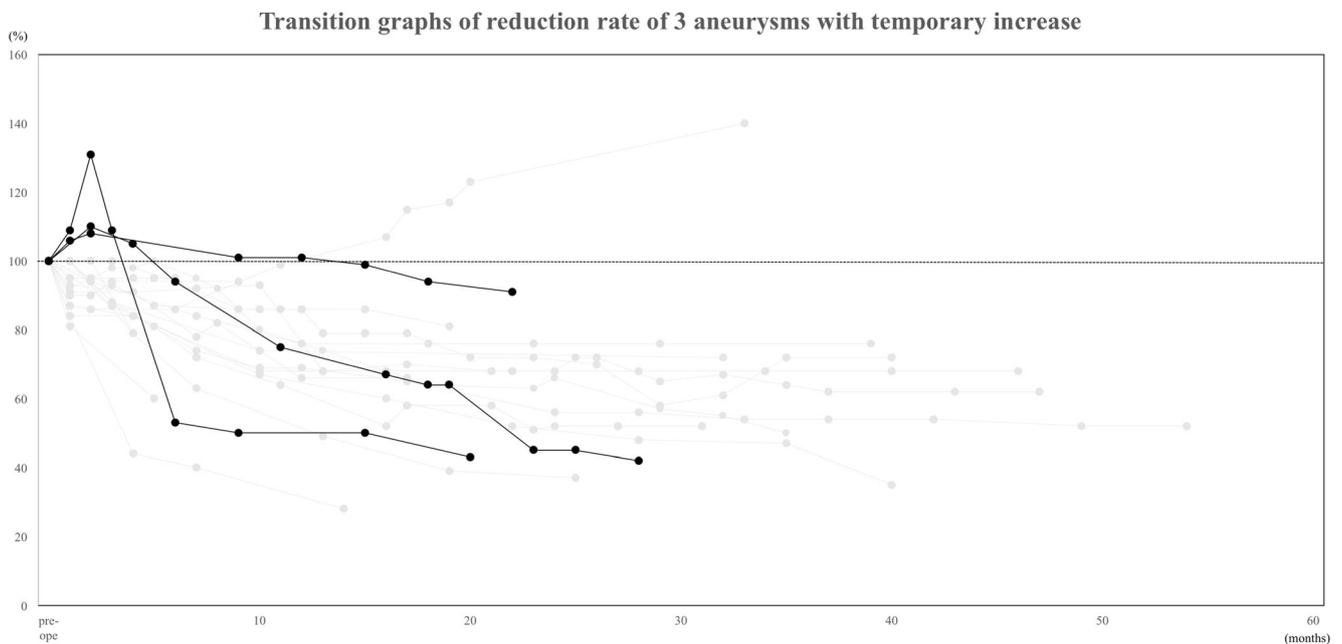


Fig. 7 Transition graphs of reduction rate of three aneurysms with temporary increase

Table 2 Infarction patterns

Aneurysms	Total	Patient	Vessels (perforator/cortical branch) related to aneurysms		Timing, area of infarction
			Antegrade flow	Retrograde flow	
ICA PcoA	1	2		OA, AChoA, PcoA	POD3, PcoA perforator POD17mon, AChoA
ICA AChoA	1	3	OA	AChoA, PcoA	–
ICA ophthalmic	1	4	OA	AChoA, PcoA	–
ICA cavernous	7	1		OA, AChoA, PcoA	–
		5		OA, AChoA, PcoA	POD1, artery-to-artery embolism
		6		OA, AChoA, PcoA	POD1, artery-to-artery embolism
		6		OA, AChoA, PcoA	–
		7		OA, AChoA, PcoA	–
		8		OA, AChoA, PcoA	–
		11		OA, AChoA, PcoA	–
MCA M1	1	10		LSA	POD0, LSA
MCA M2	1	9	LSA		–
VA	6	12		PICA, ASA	–
		14		PICA, ASA	–
		16		PICA, ASA	–
		18	P1 perforator, PICA	ASA	–
		19	PICA	ASA	–
		20	AICA-PICA	ASA	–
BA	1	17	BA perforator, P1 perforator		–
PCA P1–P2	2	13	P1 perforator		POD1, distal perfusion area of the parent artery
		15	P1 perforator		–

area; however, an asymptomatic cerebral infarction was confirmed on the distal perfusion area of the parent artery upon which PAO was performed. It has been reported that, for fetal-type PCA, PAO is suggested to carry a risk of infarction onset due to insufficient collateral blood circulation, indicating that preoperative BOT evaluation is crucial [23]. However, there was a patient of adult-type PCA and tolerance confirmed by BOT in the current series, indicating that hereafter re-evaluation of BOT false positives and false negatives, as well as investigation of concomitant bypass prior to PAO, should be considered. On the other hand, there were no perioperative complications related to high/low flow bypass for anterior circulation aneurysms such as graft occlusion, scalp necrosis, postoperative intracranial hemorrhage, and ischemic troubles. Cerebral infarctions in perforator areas were confirmed in the remaining two patients (patients 2 and 10). However, in the patients of ICA cavernous and VA aneurysms in particular, there have been no infarctions of vessels areas around the aneurysms.

Considering the temporary size increase mechanism

As indicated by Fig. 7, in the present series, there were three aneurysms of temporary enlargement following surgery (patients 6, 13, 14). Apart from insufficient aneurysmal blind-alley formation, a number of possible factors exist to explain the mechanism of post-surgery enlargement. One of these is the vasa vasorum, which is thought to be involved in the repeated hemorrhage and thrombosis mechanism of the small blood vessels of the aneurysmal wall [7, 10, 19]; however, vasa vasorum is assumed to be present only in the proximity of the internal carotid and vertebral arteries [1]. In terms of other possible factors, Nagahiro et al. [15] have reported on the development of the intrathrombotic capillary channels, and Marunishi et al. [14] have reported on rapid thrombus formation causing intramural hemorrhage and aneurysmal growth. In the current series, the involvement of the vasa vasorum in patients 6 and 14 cannot be refuted, yet in both patients, post-surgical thrombosis was markedly progressive, and hemorrhage was confirmed within the aneurysm, so this

was considered to be the cause of the temporary volume increase.

Limitations

Limitations of the present series are the small number of participants, difficulty of accurate comparison between patients due to the various aneurysmal locations, and lack of unified timing for MRI follow-up.

Conclusion

Surgical PAO treatment of unruptured intracranial aneurysms measuring over 10 mm has been shown to be an effective method of treatment, eliciting a reduction in aneurysm size. However, for aneurysms in which blind-alley formation is unsuccessful, there are those which present with enlargement, as such an alternative treatment needs to be pursued.

Funding No funding was received for this research.

Compliance with ethical standards

Conflict of interest The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent For this type of study, formal consent is not required.

Disclosure of funding None.

References

1. Aydin F (1998) Do human intracranial arteries lack vasa vasorum? A comparative immunohistochemical study of intracranial and systemic arteries. *Acta Neuropathol* 96:22–28
2. Berenstein A, Ransohoff J, Kupersmith M, Flamm E, Graeb D (1984) Transvascular treatment of giant aneurysms of the cavernous carotid and vertebral arteries. Functional investigation and embolization. *Surg Neurol* 21:3–12
3. Clarençon F, Bonneville F, Boch AL, Lejeune L, Biondi A (2011) Parent artery occlusion is not obsolete in giant aneurysms of the ICA. Experience with very-long-term follow-up. *Neuroradiology* 53:973–982
4. Diaz FG, Ohaegbulam S, Dujovny M, Ausman JI (1988) Surgical management of aneurysms in the cavernous sinus. *Acta Neurochir* 91:25–28
5. Dolenc V (1983) Direct microsurgical repair of intracavernous vascular lesions. *J Neurosurg* 58:824–831
6. Drake CG, Peerless SJ, Ferguson GG (1994) Hunterian proximal arterial occlusion for giant aneurysms of the carotid circulation. *J Neurosurg* 81:656–665
7. Iihara K, Murao K, Sakai N (2003) Continued growth of and increased symptoms from a thrombosed giant aneurysm of the vertebral artery after complete endovascular occlusion and trapping: the role of vasa vasorum. Case report. *J Neurosurg* 98:407–413
8. Ishii R, Tanaka R, Koike T, Takeda N, Takeuchi S, Sasaki O, Okada K (1983) Computed tomographic demonstration of the effect of proximal parent artery ligation for giant intracranial aneurysms. *Surg Neurol* 19:532–540
9. Kashiwazaki D, Ushikoshi S, Asano T, Kuroda S, Houkin K (2013) Long-term clinical and radiological results of endovascular internal trapping in vertebral artery dissection. *Neuroradiology* 55:201–206
10. Krings T, Alvarez H, Reinacher P (2007) Growth and rupture mechanism of partially thrombosed aneurysms. *Interv Neuroradiol* 13:117–126
11. Larson JJ, Tew JM Jr, Tomsick TA, van Loveren HR (1995) Treatment of aneurysms of the internal carotid artery by intravascular balloon occlusion: long-term follow-up of 58 patients. *Neurosurgery* 36:26–30
12. Lawton MT, Quiñones-Hinojosa A (2005) Thrombotic intracranial aneurysms: classification scheme and management strategies in 68 patients. *Neurosurgery* 56:441–454
13. Lubicz B, Gauvrit JY, Leclerc X, Lejeune JP, Pruvo JP (2003) Giant aneurysms of the internal carotid artery: endovascular treatment and long-term follow-up. *Neuroradiology* 45:650–655
14. Maruishi M, Shima K, Chigasaki H (1994) Giant intracranial aneurysm with rapid thrombus formation and intramural hemorrhage. Case report. *Neurol Med Chir (Tokyo)* 34:829–831
15. Nagahiro S, Takada A, Goto S (1995) Thrombosed growing giant aneurysms of the vertebral artery: growth mechanism and management. *J Neurosurg* 82:796–801
16. Nanda A, Sonig A, Banerjee AD et al (2014) Microsurgical management of giant intracranial aneurysms: a single surgeon experience from Louisiana State University, Shreveport. *World Neurosurg* 81:752–764
17. Pozzati E, Fagioli L, Servadei F, Gaist G (1981) Effect of common carotid ligation on giant aneurysms of the internal carotid artery: computerized tomography study. *J Neurosurg* 55:527–531
18. Raymond J, Gentric JC, Darsaut TE, Iancu D, Chagnon M, Weill A, Roy D (2017) Flow diversion in the treatment of aneurysms: a randomized care trial and registry. *J Neurosurg* 127:454–446
19. Schubiger O, Valvanis A, Wichmann W (1981) Growth mechanism of giant intracranial aneurysms demonstrated by CT and MR imaging. *Neuroradiology* 29:266–271
20. Sharma BS, Gupta A, Ahmad FU et al (2008) Surgical management of giant intracranial aneurysms. *Clin Neurol Neurosurg* 110:674–681
21. Shi X, Qian H, Fang T et al (2015) Management of complex intracranial aneurysms with bypass surgery: a technique application and experience in 93 patients. *Neurosurg Rev* 38:109–120
22. Vazquez Anon V, Aymard A, Gobin YP, Casasco A, Rüffenacht D, Khayata MH, Abizanda E, Redondo A, Merland JJ (1992) Balloon occlusion of the internal carotid artery in 40 cases of giant intracavernous aneurysm: technical aspects, cerebral monitoring, and results. *Neuroradiology* 34:245–251
23. Xu J, Xu L, Wu Z, Chen X, Yu J, Zhang J (2015) Fetal-type posterior cerebral artery: the pitfall of parent artery occlusion for ruptured P2 segment and distal aneurysms. *J Neurosurg* 123(4):906–914