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COMMENTS

Shojima et al present a well-written report using computational fluid dynamic (CFD) simulations to propose a mechanistic role for wall shear stress in the progression of cerebral aneurysms. Wall shear stress is the tangential force exerted on the arterial walls by flowing blood. The authors suggest that low shear stress may serve as a trigger for aneurysm progression represented by blister formation, and distinguish this from the hemodynamic factors related to aneurysm formation. Of 82 unruptured aneurysms studied with 3 dimensional (3D) rotational angiography and followed with serial magnetic resonance angiography, 2 enlarged and 2 ruptured with blister formation during the mean observation period of 10.1 months. In these aneurysms with documented blister formation, 1 that enlarged (MCA) and 2 that ruptured (AComA and basilar apex) comprise the subject of this report. Three-D rotational angiogram images obtained before and after blister formation were overlaid to characterize the regions of blister formation. The aneurysm geometry before blister formation was then analyzed with phase contrast magnetic resonance velocimetry to simulate 3D pulsatile blood flow and to model shear magnitude at various sites within segmented parent arteries, whole aneurysms, and areas of blister formation on the aneurysm wall. Since shear magnitude values less than 1 Pa were defined as subphysiologic, the spatially averaged shear magnitude at the area of blister formation on these 3 aneurysms was concluded to be low, as measurements ranged from 0.40 to 0.61 Pa. The authors conclude that low shear magnitude at the blister-forming area is associated with progression and rupture of cerebral aneurysms.

This report adds to the growing amount of image-based data implicating intra-aneurysmal hemodynamic characteristics in aneurysm growth and rupture. These 3 cases extend the previous work by these authors describing low wall shear stress in MCA aneurysms, and is unique in focusing upon the area of the aneurysm walls that subsequently form blisters. The effects of patient-specific flow characteristics upon CFD simulations are becoming increasingly clarified. Despite limitations of these modeling techniques, as evidenced by the exclusion of the anterior communicating artery aneurysm that enlarged in this series owing to the presence of inflow from both anterior cerebral arteries, this field of hemodynamic research is immensely exciting in its potential for improving our understanding of the natural history of this disease. Substantial efforts have already been targeted at elucidating the mechanisms of mechanotransduction, namely the interaction between hemodynamic biomechanical forces and endothelial cell function, and further studies focusing upon these mechanisms in cerebral aneurysms will likely lead to enhanced insights into the risk of aneurysm growth and rupture.

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The role of hemodynamic forces in blister formation on cerebral aneurysms is currently poorly understood. To complicate matters, recent data have suggested that the forces that promote aneurysm formation (high shear magnitude) may, in fact, be opposite to those that promote blister formation. Shojima et al have retrospectively evaluated 3 cerebral aneurysms that have undergone blister formation among their series of 82 unruptured aneurysms. A middle cerebral artery, anterior communicating artery, and basilar artery aneurysm with blister formation were isolated. Using elegant CFD simulations, the authors have demonstrated markedly subphysiologic shear magnitudes in the regions bordering blister sites in all 3 cases. These findings suggest that regions of low shear stress, resulting in an adjacent high shear gradient, may result in endothelial changes that weaken the vessel wall and, over time, promote degeneration and ultimately blister formation.

The present study has several important limitations including small sample size, lack of some critical preblister formation data, and typical CFD limitations regarding the assumptions made for model generation and analysis. Nonetheless, Shojima et al have provided interesting data not previously evaluated. The continued refinement of CFD techniques and their application to neurosurgical disease provides a very real opportunity for substantial knowledge gains in cerebrovascular pathophysiology. Investigators, such as Shojima et al, who contribute to this burgeoning field, should be commended; however, we would also emphasize the importance of developing reliable ways to correlate these virtual findings with actual biologic cascades and events.

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Unruptured Intracranial Aneurysms: Current Perspectives on the Origin and Natural Course, and Quest for Standards in the Management Strategy

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Abstract

Unruptured intracranial aneurysms are relatively common, and can cause subarachnoid hemorrhage. Management of unruptured intracranial aneurysms requires knowledge of the natural course and management risks of individual aneurysms. Current knowledge on the natural course and management risks is summarized and our current management strategy presented. Extensive literature review was conducted to identify risk factors influencing the natural course and management outcome of unruptured intracranial aneurysms. Our consecutive surgical series from October 2006 through June 2009 were reviewed retrospectively. The risk factors for rupture were size and location, as well as history of subarachnoid hemorrhage in small aneurysms. Management morbidity was significantly influenced by the size, location, and patient's age. Since 2006, we have monitored motor evoked potentials in all surgeries of cerebral aneurysms and utilized endoscope control, and skull base and bypass techniques in selected cases. In 133 consecutive surgeries, two patients (1.5%) suffered severe neurological morbidity. Unruptured intracranial aneurysms have various clinical characteristics and we need to stratify management strategy according to the aneurysm features such as size, location, shape, and patient's clinical status. In Japan, with national efforts to elevate management standards, morbidity associated with the treatment of the unruptured intracranial aneurysms is relatively low. To improve future care further, we need to continue seeking better and less invasive management modalities and technique.

Key words: unruptured intracranial aneurysm, natural course, rupture, growth, management

Introduction

Unruptured intracranial aneurysms are relatively common, occurring in 2–6% of the adult population,[23] and can have grave consequences after rupture resulting in subarachnoid hemorrhage. Surgical intervention significantly reduces the risk of rupture but the risk of such prophylactic treatment cannot be ignored. To determine management strategy, we need to assess the natural history and management risks of individual aneurysms. Since the majority of the patients with this pathology are asymptomatic, management must minimize the interventional risks as far as possible.

This review summarizes the current knowledge of the origin and the natural course, and standard management strategy of unruptured intracranial aneurysms.

Characteristics of Unruptured Intracranial Aneurysms

I. Origin and rupture

The origin and cause of rupture of cerebral aneurysms remain unclear. The impact of the blood stream may damage the arterial wall, especially at the arterial bifurcation, and cause the aneurysm. However, a recent hemodynamic study has proven that the impact force at the originating site is not significantly different from other sites.[35] On the other hand, the shear stress was relatively high adjacent to the origin of the aneurysm, which is thought to work as the force tearing the vessel wall. The aneurysm then grows with physiological remodeling of the arterial wall caused by the relatively low shear stress at the aneurysm dome.[34] Histological studies of intracranial aneurysms have demonstrated that both symptomatic unruptured and ruptured aneurysms incorporate degenerative processes of the arterial wall similar to atherosclerosis, and such features are not commonly seen in asymptomatic unruptured intracranial aneurysms.[5] Recently, atherosclerosis has been considered to originate in inflammation of the arterial wall. Such inflammatory processes are considered to be important in the growth and rupture of the aneurysms. A recent experimental study showed statins inhibit the growth of experimental cerebral aneurysms.[1] Development of medical treatments for unruptured intracranial aneurysms are awaited.

II. Natural course of unruptured intracranial aneurysms

Many studies have investigated the natural course (analysis of risk of rupture) of unruptured intracranial aneurysms.[8,10,20,30,37,38,41,42,45,46] Most studies were retrospective and some were prospective, but no randomized clinical trials have been published. In general, higher rupture risks have been reported in retrospective studies than in prospective studies. Also, the independent risk factors are rather difficult to clarify in retrospective series. Table 1 summarizes the natural course of unruptured intracranial aneurysms from retrospective series. The annual rupture risk of all intracranial aneurysms was reported as between 1% to 3% in retrospective studies. However, there is inevitable selection bias in the included patients. Elderly, sicker patients or patients with aneurysms carrying high management risks tend to be observed conservatively.

Table 2 shows the three prospective studies published. The international study of unruptured intracranial aneurysms (ISUIA) including 1,692 prospective cases demonstrated the rupture rate is about 0.78% annually, and is strongly related with aneurysm size and anterior or posterior location (Table 3).[42] Small anterior circulation aneurysms rarely ruptured (0% for <7 mm aneurysms located in the anterior circulation without history of subarachnoid hemorrhage). On the other hand, large aneurysms frequently ruptured (8% or more annually). The SUAVe study in Japan included all small aneurysms less than or equal to 5 mm, and demonstrated that even small aneurysms can grow and rupture.[46] The annual rupture rate in the follow-up period of 375 aneurysm-years was 0.8%. An additional 18 aneurysms (4.7%) grew more than 2 mm, and 5 of them underwent repair of the aneurysm before its rupture. Considering the growing aneurysm

Table 1 Natural history of unruptured intracranial aneurysms from retrospective series

Author (Year)	No. of cases	Mean age (yrs)	Follow up	Annual rupture rate (%)	Factors affecting rupture
Yasui et al. (1997)[45]	234 pt, 303 an	59.6	75.1 mos	2.3	multiplicity
Rinkel et al. (1998)[30]	3907 pt·yr	—		1.9 (1.5–2.4); 0.7: <1 cm, 4.0: >1 cm	symptomatic, sex, size, location
ISUIA (1998)[38]	727 pt, 977 an	56	8.3 yrs, 12,023 pt·yr	0.05: <1 cm, 0.5: >1 cm	size
	722 pt, 960 an	49.4		0.5	
Juvela et al. (2000)[10]	142 pt, 181 an	41.9	19.7 yrs, 2575 pt·yr	1.3	smoking, older age, size
Tsutsumi et al. (2000)[37]	62 pt	70.8	4.8 yrs	2.3	size
Morita et al. (2005)[20]	911 pt		3801 pt·yr	2.7	size, posterior, symptom
Wermer et al. (2007)[41]	4705 pt, 6556 an		26122 pt·yr	1.2: <5 yrs, 0.6: 5–10 yrs, 1.3: >10 yrs	age >60 yrs, female, Japanese or Finnish, size >5 mm, posterior, symptom

an: aneurysms, pt: patients, pt·yr: patient·year.

Table 2 Natural history of unruptured intracranial aneurysms from prospective series

Author (Year)	No. of cases	Mean age (yrs)	Follow up	Annual rupture rate (%)	Factors affecting rupture
ISUIA (2003)[38]	1692 pt, 2686 an	55.2	4.1 yrs, 6544 pt·yr	0.78	size, posterior location, history of SAH
Yonekura (2004)[46]	329 pt, 380 an, all ≤5 mm	62	375 an*yr	0.8 (0.2–3)	multiple, female, ACom, basilar location, age >70 yrs
Ishibashi et al. (2009)[8]	419 pt, 529 an		2.5 yrs, 1039 pt·yr	1.4	history of SAH, size, posterior location

ACom: anterior communicating artery, an: aneurysms, an*yr: aneurysm-year, pt: patients, pt·yr: patient·year, SAH: subarachnoid hemorrhage.

Table 3 **Risk specific natural history (annual rupture rate) according to the prospective cohort of ISUIA II**

ISUIA II	Size				
	<7 mm		7–12 mm	13–24 mm	≥25 mm
	Group I	Group II			
Cavernous IC (n=210)	0	0	0	3.0%	6.4%
AC/MC/IC (n=1037)	0	0.3%	0.5%	2.9%	8%
Post-PCom (n=445)	0.5%	0.7%	2.9%	3.7%	10%

AC: anterior cerebral, Group I: not associated with subarachnoid hemorrhage, Group II: aneurysm found in patients with previous subarachnoid hemorrhage, IC: internal carotid, ISUIA II: International Study of Unruptured Intracranial Aneurysms Investigators published in 2003, MC: middle cerebral, PCom: internal carotid-posterior communicating, Post: posterior circulation.

Table 4 **Summary of reports on the growth of unruptured intracranial aneurysm**

Author (Year)	No. of cases	Follow up	Enlargement rate	Factors affecting rupture
Matsubara et al. (2004)[18]	140 pt, 166 an	17.7 mos	10 (6.4%); 2.4%: <5 mm, 8.8%: 5–10 mm, 50%: >10 mm; 2.5%: <1 yr, 8%: 2 yrs, 17.6%: 3 yrs; 40%: BA, 0%: MCA	size, location (BA), follow-up period
Yonekura (2004)[46]	329 pt, 380 an, 375 an*yr	12 mos	18 (4.7%)	age >70 yrs, ACom, BA location, multiplicity, female
Burns et al. (2009)[3]	156 pt, 191 an	47 mos	10%; 6.9%: <8 mm, 25%: 8–12 mm, 83%: >13 mm	size, location, multiplicity

ACom: anterior communicating artery, an: aneurysms, an*yr: aneurysm-year, BA: basilar artery, MCA: middle cerebral artery, pt: patients, pt·yr: patient·year.

could have ruptured unless treated, the annual rupture rate is not negligible in such patients. A follow-up study on untreated 419 patients at a single institution[8] found the rupture rate was 1.4% per year and was significantly influenced by history of subarachnoid hemorrhage, posterior location, and size of the aneurysm. Currently, in Japan, two prospective studies (UCAS Japan and UCAS II) have being conducted, and the preliminary results indicate that the natural course is significantly related to size as well as specific locations of the aneurysms. Aneurysms located at the anterior communicating and internal carotid-posterior communicating arteries tended to rupture more frequently than other locations, even if small (unpublished data).

Several studies compared three-dimensional (3D) images between groups of ruptured and unruptured intracranial aneurysms. Aneurysms with large height and high ASPECT (dome height /neck width) ratio, and located on the posterior communicating artery and anterior communicating artery, as well as the posterior circulation are more commonly ruptured.[25] Irregular shape and coexistence of bleb are also found more in ruptured aneurysms.[29] These findings suggest that the aneurysms with such features can rupture easily.

Table 4 summarizes the reports on aneurysm growth. Larger aneurysms often grew more than smaller aneurysms.[3,17,46] Posterior location and multiple aneurysms also tended to grow frequently. Rate of growth is exponentially increased along with the follow-up year. Such findings coincide with the risk factors found for aneurysm rupture.

III. Management outcome and risks

Numerous studies on the management outcome for unruptured intracranial aneurysms are summarized in Table 5.[9,14,22,28,42,43] Reported and published clinical outcomes are significantly different between various types of studies.[47] The ISUIA[42] found that mortality was 1.6% and significant morbidity (modified Rankin scale 3 or below or mini-mental state examination (MMSE) score below 25) was 10.9% at the one month follow-up. Decline of cogni-

Table 5 Outcome of management of unruptured intracranial aneurysms

Author (Year)	No. of cases	Type of study	Risk	Risk factors
Wirth et al. (1983)[43]	107 pt, 119 an open surgery	multicenter retrospective cohort, 12 centers	mortality: 0%; morbidity: 6.5%	size, location
King et al. (1994)[14]	733 pt open surgery	meta-analysis, 28 studies	mortality:1.0% (0.4–2.0%); morbidity: 4.1% (2.8–5.8%)	none
Raaymakers et al. (1998)[26]	2,460 pt open surgery	meta-analysis, 61 studies	mortality: 2.6% (2.0–3.3%); morbidity: 10.9% (9.6–12.2%)	old publication, giant an, posterior circulation
Murayama et al. (1999)[22]	115 pt, 120 an endovascular	case series retrospective	morbidity: 4.3%; complete occlusion: 91%, unsuccessful coil: 5%, delayed rupture: 1%	early series
Johnston et al. (2001)[9]	2,069 pt 1699: open surgery, 370: endovascular	multicenter retrospective cohort	mortality: 3.5% (open surgery), 0.5% (endovascular); morbidity: 25% (open surgery), 11% (endovascular)	open surgery
ISUIA (2003)[38]	1917 pt open surgery	multicenter prospective cohort, 61 centers	mortality: 1.5%; morbidity: 11.7% (1 mo)	size, age, location, ischemic disease, symptom
	451 pt endovascular		mortality: 1.7%; morbidity: 7.3% (1 mo); complete occlusion: 51%, unsuccessful: 5%	

an: aneurysms, pt: patients.

tive function accounted for the major part of the morbidity, and the importance of measuring high cognitive function for the clinical assessment of surgical outcome of the patients was stressed. Risks were higher in elderly patients and patients with posterior circulation aneurysms. Endovascular treatment had better outcomes in the group of elderly patients. Survey of the outcomes of aneurysm surgery in Japan found the surgical mortality of 4,396 UIAs was 0.2%.[7] The case volume of the institution did not affect the surgical outcome in this survey. Quality of life (QOL) score was also analyzed in several outcome studies.[27,39,44] Some reported significant decline of QOL long time after aneurysm repair. However, preoperative anxiety was relieved by the repair of the aneurysm and QOL also improved.[44] Meticulous surgical repair resulted in decline of cognitive function in only a minimal number of cases.[24] Our recent study (UCAS II) also followed the QOL of patients (unpublished data). QOL scores measured by SF-8 and EQ5D immediately after treatment did not show any decline compared to prior to surgery. Depression score measured by SF36 mental health and vitality domain was low compared to the Japanese normal population and did not show change immediately after the treatment. We are following these scores in the longer term.

Surgical Strategy and Case Series in Our Institution

I. Surgical strategy

To accomplish the safest intervention, we need to carefully assess the patient and involve updated surgical technique and assist devices. In our institution, we follow the recommendations of the Japanese Society of Screening of Asymptomatic Cerebral Disorders in deciding on repair of unruptured intracranial aneurysms. Basically, aneurysm repair is indicated in patients with management risks considered lower than the natural risks for rupture or physical factors for the next 10 years. When we decide to treat the aneurysm of specific patients, either open craniotomy and clipping or endovascular coiling is selected individually according to the following strategy.

A: Clipping is the preferred method when the surgical risk in the specific patient is similar or lower to that of coiling. Aneurysm repair in the location of the basilar tip or in patients with very high medical risks is often managed better by endovascular methods. However, wide-based basilar aneurysms are repaired by open clipping after thorough discussion with endovascular specialists.

B: If the patient strongly prefers endovascular management after thorough discussion, endovascular treatment is indicated.

In our institution, surgical managements are assisted by the following advanced techniques to diminish surgical morbidity: Pre-operative thorough evaluation of aneurysm images including 3D angiography, 3D computed tomography (CT) angiography and magnetic resonance (MR) angiography, MR imaging with Fast Imaging Employing STeadystate Acquisition (FIESTA), and Preoperative 3D simulation.

The gold standard for the diagnosis of cerebral aneurysms is digital subtraction angiography. This technique is still essential for assessing cerebral circulation and obtained details of the cerebral vascular anatomy including the venous system. However, recent advances in CT and MR angiography have reduced the needs for catheter angiography. With advanced imaging techniques, we now can visualize and anticipate perforators around the aneurysm, and imagine the space between the aneurysm and the adjacent parent arteries. CT and MR angiography provide the internal shape of the aneurysm and FIESTA images provide the external shape of the aneurysm, and detect adjacent perforator and cranial nerves.[32] More accurate imaging incorporating heart beat influence will provide information on the distortion of the aneurysm by the heart beat and the wall thickness of the aneurysm. Combination of such 3D images can provide very useful virtual reality simulations for surgical procedures.[13] Currently, we can create an actual-sized elastic hollow model of individual aneurysms from the preoperative 3D images. These models were very useful in deciding how to apply clips to occlude the closure line of the aneurysm and combination of aneurysm clips for complex-shaped aneurysms.[11]

Electrophysiological monitoring: Electrophysiological monitoring is essential to reduce operative complications. Motor evoked potential monitoring is very useful for reducing managing morbidity during aneurysm surgery, especially around the perforating branches such as the anterior choroidal, posterior communicating, M_1, and vertebro-basilar arteries.[31] Visual evoked response is also very useful in reducing risks to visual acuity during intervention for paraclinoid aneurysms.[6]

Meticulous microsurgical, skull base surgical and bypass technique: For open aneurysm surgery, the basic surgical technique involves meticulous microsurgical procedures preserving all possible venous and arteriolar structures. To accomplish appropriate clipping, we need to fully dissect and expose the aneurysm dome and neck with sharp dissection. Also, with the widespread availability of endovascular technique, open surgeons are now facing more complex cerebral aneurysms, which cannot be simply obliterated using the usual microsurgical techniques. We should be able to utilize skull base and bypass techniques as routine additional microsurgical techniques. The anterior clinoid process including optic canal unroofing and/or posterior clinoid process should be drilled in treating paraclinoid aneurysms or basilar aneurysms to widely expose the adjacent anatomical structures and create extra room for safe access. Such procedures can be safely performed using the SONOPET ultrasonic bone curettage instrument (UST-2001; Stryker Japan, Tokyo) without the fear of causing rolling-up injury to the important structures by the drills.[4] Other cranial base approaches including anterior petrosectomy and combined petrosal approach, and transcondylar approaches are utilized in managing vertebrobasilar complex aneurysms. Bypass technique including superficial temporal artery (STA) to middle cerebral artery, A_3-A_3 anastomosis or high flow technique, is often required in the management of some wide neck aneurysms to salvage parent arteries, especially aneurysms incorporating wide or thrombosed wall.[12,33,36]

Combination of open and endovascular technique in selected cases: If cases present possible difficulty for both open and endovascular techniques, we should combine both techniques. Especially in cases with wide neck aneurysm combined with vessel wall calcification, or adjacent perforator difficult to dissect from the neck, we can intentionally partially occlude the aneurysm neck with clips and obliterate the aneurysm completely with endovascular coils. Endovascular procedure can be achieved safely after the aneurysm neck is narrowed and the clipping can be done without sacrificing the perforators. In Japan, advanced intracranial stent technique is currently under trial and only performed in selected institutions. When such endovascular techniques including the soft flexible stenting method are available widely, we should actively incorporate such techniques.[16,40] However, we need to carefully assess the long term outcome of such endovascular procedures, since the ISAT showed frequent rebleeding after endovascular procedures.[18] Current endovascular technique is not completely reliable protection against bleeding from aneurysms.

Application of endoscopy and Indocyanine Green (ICG) angiography: Endoscopy and ICG are very useful techniques in assessing the patency of adjacent perforating and parent arteries. In selected cases, clipping can be accomplished under the control of the endoscope to prevent occlusion of the perforating vessels.[15,21] Recently developed ICG technique is useful in assessing the patency of adjacent arteries.[26]

Cosmetic skin incision and craniotomy repair: To reduce patient's burden in social life, we should always consider cosmetic concerns in assessing surgical outcome. We should avoid facial nerve palsy after craniotomy and minimize surgical depression of the temporal area. Muscle atrophy should be avoided by meticulous dissection technique, avoiding injury to the temporal muscle nerves and vasculature, and reduce the bone defect caused by the craniotomy.

II. Our series of open aneurysm surgeries

From October 2006 through June 2009, 133 open aneurysm surgeries for unruptured intracranial aneurysms were performed by the authors utilizing the above mentioned techniques. Bypass surgery was included in 13 procedures and cranial base technique was involved in 18 cases (anterior clinoidectomy in 13, posterior clinoidectomy in 3, anterior petrosal approach in 1, and combined posterior petrosal approach in 1). Endovascular procedures (coiling) were added in 2 cases for planned incomplete occlusion of the aneurysm. The endoscope was used in all cases except for middle cerebral aneurysms. Electrophysiological monitoring was applied in all cases. At discharge, two patients (1.5%) suffered severe morbidity (modified Rankin scale less than or equal to 2, or Mini-mental State Examination Score less than or equal to 25). Both patients had giant thrombosed aneurysms (Table 6).

Postoperative MR imaging, obtained in all open surgical cases, showed unexpected T_2 hyperintensity in 6 cases. In addition to the severe morbidity, four patients experienced four subdural hematomas, which required surgical evacuation one to 3 months after surgery. Seizure occurred in 5 patients, which was controlled with antiepileptic medication. Wound complication was found in 2 patients, including one patient who required surgical repair for CSF leak. No other clinical complication was noted.

Table 6 Morbidity of 133 consecutive open surgical procedures on unruptured intracranial aneurysms in Kanto Medical Center, NTT Ec during October 2006 through June 2009 (mRS ≥2, or MMSE ≤25 at discharge)

Location	Size	
	<10 mm	≥10 mm
Anterior circulation	0/92	1/26 (3.8%)
Posterior circulation	0/3	1/2 (50%)

MMSE: mini-mental state examination, mRS: modified Rankin scale, NTT Ec: Nippon Telegraph and Telephone East Corporation.

III. Illustrative cases

Case 1 (Fig. 1): This 63 year-old female presented with a left asymptomatic small posterior communicating artery aneurysm. Left pterional craniotomy was performed and clipping was performed under endoscopic control. After initial clipping, her right upper-extremity motor evoked potential diminished and endoscopic imaging showed the clip had occluded a perforator from the posterior communicating artery. The clip was repositioned to not constrict the perforator circulation. Motor evoked response returned and the patient recovered uneventfully.

Case 2 (Fig. 2): This 72-year old female presented with a large left basilar superior cerebellar artery aneurysm. Preoperative FIESTA MR imaging demonstrated several perforators on the right back side of the aneurysm. Endovascular intervention was not indicated because the left superior cerebellar artery was at risk of occlusion. Surgery was performed through the left modified pterional-anterior temporal approach involving anterior and posterior clinoid resection using SONOPET. During surgery,

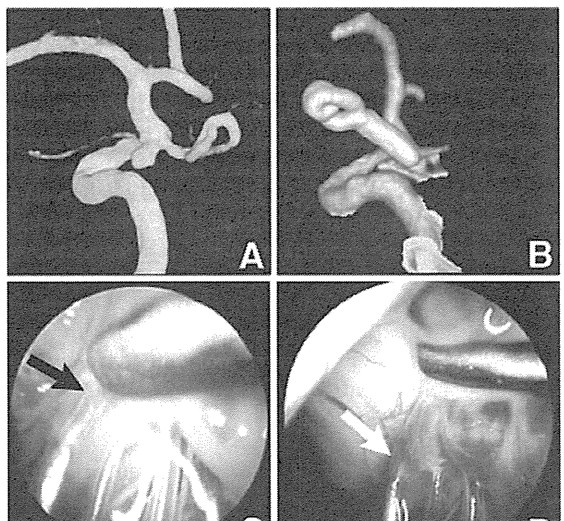

Fig. 1 Illustrative Case 1. A, B: Pre- and post-operative three-dimensional angiograms showing a small left internal carotid-posterior communicating aneurysm successfully occluded by a clip graft. C: Endoscopic image after initial clipping showing occlusion of the perforating vessel (*arrow*). D: After repositioning the clip, the perforators were spared (*arrow*) and motor evoked potential recovered.

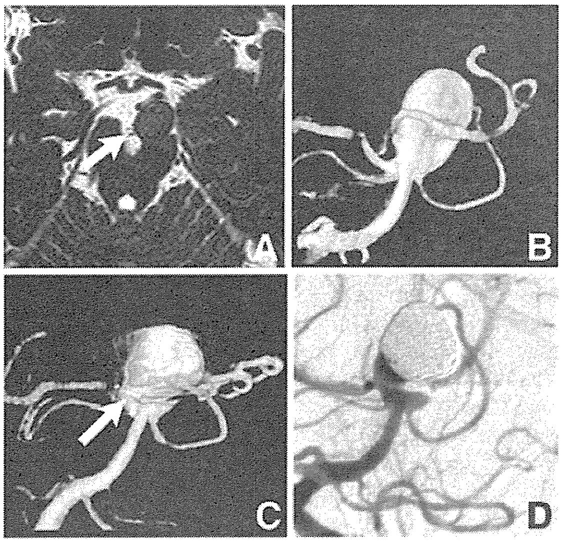

Fig. 2 Illustrative Case 2. A: Preoperative magnetic resonance image using the Fast Imaging Employing STeadystate Acquisition sequence showing several perforators in the right back side of the aneurysm (*arrow*) and the left P_1 segment attached to the aneurysm dome. B: Three-dimensional angiogram showing the large basilar-superior cerebellar artery aneurysm with a relatively wide neck through the narrowed neck (*arrow*). C: After partial clipping across the neck, the aneurysm dome was still patent. D: Endovascular coiling was performed to occlude the majority of the dome.

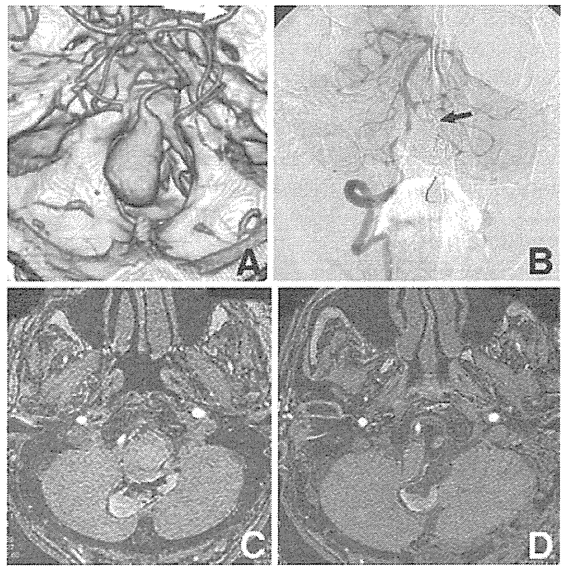

Fig. 3 Illustrative Case 3. A: Three-dimensional angiogram showing a large left vertebral artery aneurysm. B: After coil obliteration of the aneurysm, there is back flow from the distal left vertebral artery to the top of the aneurysm (*arrow*). C: Magnetic resonance angiogram showing a large mass compressing medulla. D: After clipping of the distal and proximal left vertebral artery, aneurysm mass and coil substance was partially removed and the brain stem was decompressed.

the clip could not be pushed across the neck due to the toughness of the wall and adhesion of the left P_1 segment to the aneurysm wall. So the clip was intentionally placed partially over the neck to produce a narrow aneurysm neck. Since this aneurysm did not occlude spontaneously, the remaining dome was filled with coils through the smaller aneurysm neck. She suffered temporary left oculomotor palsy, which subsided in 2 months. No other complication was noted and follow-up MR imaging findings have been stable.

Case 3 (Fig. 3): This 50-year-old male presented with a left giant vertebral artery aneurysm with progressive brainstem signs after coil embolization in another hospital. He initially presented with subarachnoid hemorrhage 10 years ago. He was found to have left vertebral artery dissecting aneurysm and was conservatively managed. In 2005, he developed progressive right heaviness and giant vertebral aneurysm was diagnosed. Aneurysm obliteration was performed using detachable platinum coils in another hospital. However, even after the occlusion, the aneurysm continued to grow by blood back flow from the distal vertebral artery and his neurological function deteriorated including dysphagia, dizziness, and right hemiparesis. Endovascular trial to occlude the left distal vertebral artery was unsuccessful. We planned to occlude the distal vertebral artery by clipping and decompression of the aneurysm at the second stage surgery. Left anterior petrosectomy was chosen and the clip was placed on the left distal vertebral artery just above the aneurysm. There were several perforators from the vertebral artery, which were spared. Three weeks later, the aneurysm mass with coils compressing the medulla was removed after proximal left vertebral artery clipping. Postoperative course was complicated with hydrocephalus, pneumonia, and persistent orthostatic hypotension. He was transferred to a rehabilitation facility and resumed his job 7 months after surgery. At the last follow-up examination, he was ambulatory and dysphagia was improved. At the last clinical visit, his modified Rankin scale was 1. (This case was managed in collaboration with the Endovascular Unit at Jikei Medical School.)

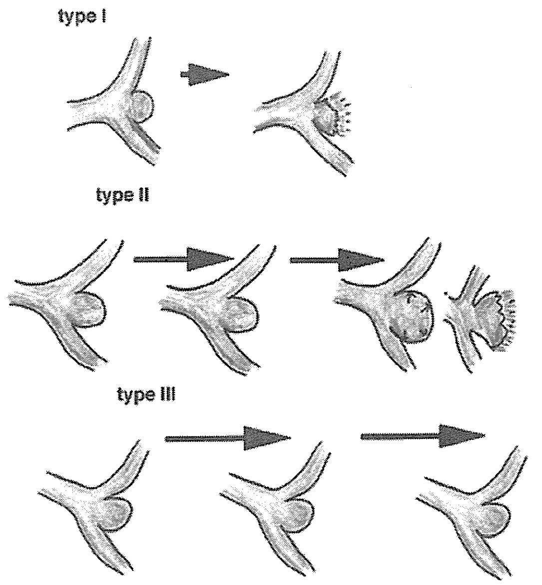

Fig. 4 Conceptual types of the natural course of intracranial aneurysms. Three types of natural course occur after the formation of intracranial aneurysms.[46] In type I, the aneurysm continues to enlarge till the phase of rupture. Most subarachnoid hemorrhage (SAH) could be caused by such aneurysms, since many small aneurysms cause SAH, whereas small unruptured aneurysm rarely ruptures. In type II, the aneurysm stops growing, but with some physiological or some other external stimuli, regrowth starts and eventually ruptures. There might be several steps repeating these grow and halt phases. In type III, the aneurysm stops growing and remains stable.

Discussions

Current knowledge on the origin and growth of the cerebral aneurysms suggests three types of origin and natural course of unruptured intracranial aneurysms (Fig. 4[46]). Type 1 is an aneurysm which ruptures immediately after formation. Type 2 is an aneurysm which stops enlarging sometime after formation, but grows due to some inflammatory process and ruptures. Type 3 is an aneurysm which stops growing after formation, but still may start regrowth due to some unknown biological cues. Type 1 should present as ruptured aneurysm, so unruptured intracranial aneurysms are likely to be type 2 or 3. To determine whether an aneurysm should be treated, we need to know the type of the individual aneurysm and the process that induces the growth of the aneurysm.

The risk factors influencing the growth and rupture of intracranial aneurysms can be summarized as follows: Group 1 risk factors (evidenced by high level prospective studies), size ($\geq$7 mm), location (posterior circulation > anterior circulation), specific locations such as anterior communicating, posterior communicating and basilar artery aneurysms, and history of subarachnoid hemorrhage; Group 2 risk factors (evidenced by retrospective case series and studies), history of smoking, multiplicity of aneurysms, higher age, symptomatic aneurysm, Japanese or Finnish population, and shape of aneurysm (irregular or high dome/neck ratio). Such evidence leads us to believe that unruptured intracranial aneurysms should not be considered as a single disease entity. We should stratify unruptured aneurysms according to the specific features such as location, size or shape, and patient factors.

Risk factors that influence management outcomes can be summarized as follows: Group 1 management risk factors (evidenced by high level prospective studies), size, location (vertebro-basilar > anterior circulation), history of subarachnoid hemorrhage, and age; Group 2 management risk factors (evidenced by retrospective case series, studies), history of ischemia, and type of management. The management of unruptured intracranial aneurysms is not risk free and the published outcomes vary according to the study design and the measurement of outcome. We should not apply published outcomes to our own decision making. Before discussing risk communication, we should clarify our own clinical outcome. Thereafter, we need to carefully decide the indications for management by assessing the natural course and institutional outcome in managing individual aneurysms.

According to the ISUIA study, recent management recommendations for unruptured intracranial aneurysm did not advocate surgical management for incidental small intracranial aneurysms unless the patient is young or has other specific hemodynamic features.[2] However, a recent study of the long-term outcome of ISUIA cases with mean follow-up of 9.2 years reported that the overall outcome was better in treated than untreated patients. Relatively risk-matched patient groups were extracted from the prospective patient cohort of ISUIA using propensity analysis score and compared untreated and treated patients (ISUIA report at the AANS 2010, unpublished data). Although the case risks in both groups matched relatively well, there might be hidden bias as sicker patients may not be indicated for treatment. Nevertheless, we need to realize the importance of long term assessment to evaluate the real outcome and benefit of treatment of unruptured intracranial aneurysms.

In Japan, even carefully designed prospective studies showed relatively low overall morbidity and mortality in managing unruptured intracranial aneurysms.[19] To achieve low-risk intervention, meticulous care in preoperative assessments and involvement of advanced surgical and interventional techniques are mandatory, including high resolution MR imaging, preoperative 3D simulation, meticulous surgical technique, skull base technique, bypass surgery, advanced neuro-physiological and neuro-imaging monitoring, and collaboration with neuro-interventional techniques. In illustrative Case 1, the authors demonstrated the need for electrophysiological monitoring and endoscopic control in managing simple small aneurysms. In Cases 2 and 3, we illustrated the need for cranial base approaches as well as combination of surgery and endovascular techniques in managing difficult cases. Brain stem compression caused by the mass effect of the aneurysm and endovascular material must be decompressed to alleviate clinical signs, and can be effective even after months of progressive symptoms. Well organized and dedicated rehabilitation efforts and physical care is mandatory to obtain good recovery.

In 2008, the revised guidelines of the Society of Screening of Asymptomatic Cerebral Disorders in Japan were published. Recommendations were made according to the published series of natural course and management outcome. The Japanese experiences were stressed rather than reports from western countries since the natural course of UIA might be slightly different between Japan and other western countries.[41] The management outcomes in our prospective series are also relatively better than those reported by ISUIA.

The recommendations for managing unruptured intracranial aneurysms are as follows (http://www.snh.or.jp/jsbd/pdf/guideline2008.pdf).

i) Interventions for unruptured cerebral aneurysms should be determined by the age and health status of the patient, size, location and nature (shape and other characteristics) of the aneurysm, the expected natural course of the aneurysm, and the institutional or surgeon's management outcome. Full detailed informed consents should be obtained and good risk communication between caregivers and patients should be created before deciding on intervention or careful follow up.

ii) Surgical or endovascular management is recommended for patients with the following aneurysms with life expectancy of more than 10–15 years.

ii-i) Aneurysms more than or equal to 5 to 7 mm

ii-ii) Smaller than above but

ii-ii-i) Symptomatic

ii-ii-ii) Located at anterior communicating, internal carotid-posterior communicating arteries or posterior circulation.

ii-ii-iii) Aneurysm with daughter sac, irregular shape, or large dome-neck aspect ratio.

iii) Risk benefit analysis based on large previously published cohort studies can be a useful indicator in deciding the management of unruptured cerebral aneurysms in general. However, the natural course, quality of life, mental status of each patient and aneurysm, and institutional surgical outcome should be carefully assessed.

iv) If the unruptured cerebral aneurysm is to be followed up without surgical intervention, smoking and excessive alcohol intake should be avoided and hypertension needs to be treated. Aneurysm should be carefully followed every half or one year by high quality imaging devices.

v) If any change of shape or increasing size is detected by the follow-up imaging, management of aneurysms should be re-assessed.

vi) Long-term follow up is mandatory after endovascular and surgical clipping of aneurysms.

Conclusions

Unruptured intracranial aneurysms show various clinical characteristics and risk analysis should be based on their features. Indication and decision of management should be decided carefully based on the aneurysm features and updated institutional management risks. All possible measures to achieve safe surgery for this disease, which is mostly asymptomatic, should be deployed. To advance future care of the unruptured intracranial aneurysms, we need continue to obtain more accurate data on the natural course by well designed prospective studies and develop less invasive management measures with minimal morbidity including medical treatment.

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Preoperative Evaluation of Unruptured Cerebral Aneurysms by Fast Imaging Employing Steady-State Acquisition Image

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BACKGROUND: In aneurysm surgery, understanding the microanatomy around the aneurysm such as perforating arteries and cranial nerves is mandatory.

OBJECTIVE: To assess the usefulness in determining the microanatomy around the cerebral aneurysms by the use of fast imaging employing steady-state acquisition (FIESTA) images of magnetic resonance imaging preoperatively, in addition to computed tomography and digital subtraction angiography.

METHODS: Between October 2006 and June 2009, 123 patients with 140 unruptured cerebral aneurysms were treated in our institution. Eighty-two patients were assessed with FIESTA by the operators on the workstation of the magnetic resonance image before surgical clipping of the aneurysms. The small vessels and cranial nerves were confirmed intraoperatively before or after obliteration of the aneurysms.

RESULTS: Sensitivities and specificities of FIESTA imaging were 100% in detecting hypothalamic artery around anterior communicating artery aneurysms, oculomotor nerve attachment to the posterior communicating artery aneurysm domes, and anterior choroidal artery adhesion to the posterior communicating artery aneurysms. This technique was also useful for predicting adhesion between the aneurysm and adjacent main trunks or perforators. Although the specificity was 100%, sensitivity was 56% in detecting vessel adhesion around the middle cerebral aneurysms. This technique can provide limited information in large aneurysms or aneurysms located in minimal cerebrospinal fluid space. The overall outcomes of the patients included 120 excellent recoveries, 1 moderate deficit, 1 severe deficit, and 1 persistent vegetative state according to the Glasgow Outcome Scale.

CONCLUSION: By giving information on the minute anatomical structure around the aneurysm, FIESTA can contribute to thorough preoperative evaluations of cerebral aneurysms.

KEY WORDS: Anatomy, Clipping, Complication, FIESTA, Perforator, Unruptured cerebral aneurysm

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To accomplish safe aneurysm surgery, it is important to understand the microanatomy around the aneurysm such as perforating arteries and cranial nerves preoperatively.

ABBREVIATIONS: Acom, anterior communicating artery; **CISS,** constructive interference in steady-state imaging; **CTA,** computed tomography angiography; **DICOM,** digital imaging and communications in medicine; **FIESTA,** fast imaging employing steady-state acquisition; **MCA,** middle cerebral artery; **UCA,** unruptured cerebral aneurysm

To assess the microanatomy before surgery, several kinds of modalities are used. Two-dimensional digital subtraction angiography with or without 3-dimensional (3D) rotational angiography is the gold standard in aneurysm surgery. On the other hand, there have been reports recently that 3D computed tomography angiography (CTA) is sufficient for the assessment for the surgery.[1-6] Magnetic resonance imaging (MRI) can provide minute anatomical information on structures such as cranial nerves and cerebellar tentorium.[7-12] Each has its advantages and is used complementarily. But because the CTA and MR angiography usually demonstrate only intraluminal anatomy of cranial vessels and aneurysm, we use fast imaging employing steady-state acquisition (FIESTA) images of MRI, in addition to 3D CTA and digital subtraction angiography when necessary, to see the outer environment of the aneurysm to anticipate whether perforating arteries or cranial nerves adhere to the aneurysm.

A steady-state acquisition sequence, FIESTA intensifies the contrast between water and solid structure with high signal-to-noise ratio.[13-15] In brain MRI, minute anatomic structures such as blood vessels and cranial nerves are contrasted to the cerebrospinal fluid (CSF), so we can understand the microanatomy of cranial nerves and vessels around the aneurysm.

The purpose of this study was to evaluate whether FIESTA can provide enough information about the perforating arteries and cranial nerves preoperatively.

MATERIALS AND METHODS

Subjects

From October 2006 to June 2009, 123 patients with 140 unruptured cerebral aneurysms (UCAs) were treated in our institution. Eight cases were symptomatic. Surgical clipping was performed for 114 patients with 133 aneurysms, and FIESTA images were taken in 82 patients. MRI and MR angiography were routinely obtained for preoperative evaluation of the unruptured intracranial aneurysms, and a FIESTA sequence was added as our department protocol. Adding this sequence was not assessed by institutional review board. The locations and sizes of the aneurysms are shown in Table 1.

Three-dimensional CTA is used mainly to decide on surgical indication and to determine the treatment modality. Digital subtraction angiography with 3D rotational angiography is performed routinely.

FIESTA Images

Imaging was performed by a 1.5-T MRI system (Signa CV/i, GE Medical Systems) using a standard quadrature head coil. The FIESTA images were obtained with other scanning modalities with the following parameters: echo time, 1.9 milliseconds; minimum repetition time, 6.3 milliseconds; flip angle, 45°; and scan time, 3 minutes 16 milliseconds, according to the installed program. The field of view was 160 × 160 mm; the matrix was 256 × 192; and the slice thickness was 1.0 mm.

Evaluation

The FIESTA images were assessed preoperatively on the screen of the workstation by the primary surgeons (T.K., A.M., S.S.) to determine whether the hypothalamic artery arises closely to anterior communicating artery (Acom) aneurysms and whether any perforating arteries, small vessels, or cranial nerves are adhered to the aneurysm in middle cerebral artery (MCA) or internal carotid artery aneurysms. When visualizing the outside of the aneurysm and adjacent arteries, if there is no CSF between them, we speculated that there might be tight adhesion between these structures.

During operation, the aneurysm was dissected circumferentially before and sometimes after obliteration of the aneurysm. The perforators or other nervous structures indicated by FIESTA images were explored. The presence of perforating arteries was confirmed with the endoscope if needed.

In internal carotid–posterior communicating artery cases, the oculomotor nerve was observed before or after the application of the clip and confirmed whether there was attachment or adhesion between the nerve and the aneurysm. In aneurysms at other locations, the existence of perforators and the adherence of nearby arteries or nervous structures also were assessed.

RESULTS

Location-specific statistical analysis of usefulness of FIESTA is summarized in Table 2.

TABLE 1. Characteristics of All Aneurysms and Outcome[a]

Patients						
Sex, n (%)						
Male	40 (32.5)					
Female	83 (67.5)					
Mean age (range), y	61.7 (39-89)					
Location	Small (< 7 mm)	Medium (7-14 mm)	Large (15-24 mm)	Giant (≥ 25)	Total, n	%
Acom	17	10	0	0	27	20.0
IC-PC	10	13	3	0	26	19.5
MCA	24	20	3	1	48	36.1
VA-BA	1	1	2	2	6	4.5
Others	13	11	2	0	26	19.5
Total					133	
Size, mm	8.1 (3-53)					
Glasgow Outcome Scale						
VS	1					
SD	2					
MD	1					

[a]Acom, anterior communicating artery; IC-PC, internal carotid-posterior communicating artery; MCA, middle cerebral artery; MD, moderate disability; SD, severe disability; VB-BA, vertebrobasilar system; VS, persistent vegetative state.

TABLE 2. Sensitivities and Specificities Detecting Surrounding Structures Around the Aneurysms by Fast Imaging Employing Steady-State Acquisition Images[a]

	Identification of Presence or Adhesion During Actual Surgery	
	Yes	No
Detection of hypothalamic artery around Acom complex aneurysm (n = 20)		
Hypothalamic artery detected by FIESTA +	18	0
Hypothalamic artery detected by FIESTA −	0	2
Sensitivity, 100%; specificity, 100%		
Detection of surrounding vessels around MCA aneurysms (n = 36)		
MCA branches around aneurysm by FIESTA +	5	0
MCA branches around aneurysm by FIESTA −	4	27
Sensitivity, 56%; specificity, 100%		
Detecting adhesion between IC-PCom aneurysm and oculomotor nerve (n = 18)		
IC-PCom An–CN III attachment by FIESTA +	10	0
IC-PCom An–CN III attachment by FIESTA −	0	8
Sensitivity, 100%; specificity, 100%		
Detecting adhesion of the IC-PCom aneurysm to the anterior choroidal artery (n = 18)		
IC-PCom An–Achor artery adhesion detected by FIESTA +	2	0
IC-PCom An–Achor artery adhesion detected by FIESTA −	0	16
Sensitivity, 100%; specificity, 100%		

[a]ACom, anterior communicating artery; AChor, anterior choroidal; An, aneurysm; CN, cranial nerve; FIESTA, fast imaging employing steady-state acquisition; IC, internal carotid; MCA, middle cerebral artery; PCom, posterior communicating artery.

Anterior Communication Artery Aneurysms

Among 20 cases with anterior communicating artery aneurysms assessed with FIESTA image, the interhemispheric approach was used in 9 cases and the pterional approach was used for the others. The Acom complex was dissected circumferentially in all cases. In 18 aneurysms (90%), the FIESTA image indicated the presence of a hypothalamic artery close to the aneurysm and no other perforating braches. In all of them, the origins of the hypothalamic arteries were confirmed during surgery at the site that preoperative FIESTA image had shown, with no other perforating arteries around the Acom complex (Figure 1). In the other 2 aneurysms (10%), no definite perforator was seen on preoperative FIESTA image. During actual surgery, only several small arteries were observed around the Acom complex. The sensitivity and specificity of the FIESTA technique were 100% for the detection of hypothalamic branches around the Acom aneurysms (Table 2).

In 1 thrombosed large Acom aneurysm, the right A2 was adhered tightly to the aneurysmal dome, which was well anticipated because the preoperative FIESTA image showed a lack of CSF between the aneurysm and the parent artery (Figure 1).

MCA Aneurysms

In 36 aneurysms assessed with FIESTA image, 5 aneurysms (14%) seemed attached to the anterior temporal artery or other small arteries and required dissection from the arteries. Another 31 aneurysms (86%) showed no adhering artery on the preoperative image. However, during surgery, in 4 of them (13%), the perforating arteries were found to arise from the parent arteries adhered to the aneurysms, which required dissection.

Overall, the sensitivity and specificity of FIESTA for detecting a MCA branch were 56% and 100%, respectively (Table 2).

In a case of symptomatic giant thrombosed aneurysm, it was difficult to assess the adhesion of vessels owing to the absence of CSF between the aneurysm and brain, but the lenticulostriate artery was identified preoperatively (Figure 2).

Internal Carotid–Posterior Communicating Aneurysms

The relationship between the aneurysm and the oculomotor nerve and anterior choroidal artery was assessed in 18 internal carotid–posterior communicating artery aneurysms, including symptomatic cases (Figure 3). In 10 cases (56%), the oculomotor nerve appeared to fuse with the aneurysm on the preoperative FIESTA image. In all 10 cases, it was confirmed that there was at least attachment between them during surgery (Figure 4). In the other 8 aneurysms (44%), there seemed to be CSF space between the 2 structures by the preoperative FIESTA images. During surgery, the oculomotor nerve was separate from the aneurysm in all of them.

The detection rate of oculomotor nerve attachment to the internal carotid–posterior communicating artery aneurysm was 100% sensitivity and 100% specificity (Table 2).

In this series, only 2 cases (12.5%) showed adhesion of the anterior choroidal artery to the aneurysm on preoperative FIESTA images. This finding was confirmed in surgery, and no other aneurysm had adhesion to the anterior choroidal artery (Table 2).

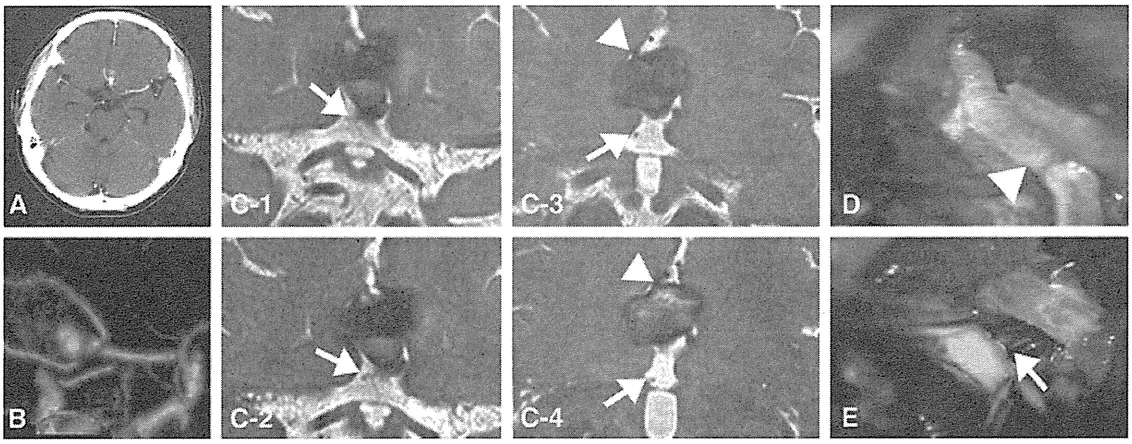

FIGURE 1. *A thrombosed anterior communicating artery aneurysm.* A, *computed tomography after contrast enhancement.* B, *virtual reality image of the aneurysm.* C-1 *through* C-4, *consequent fast imaging employing steady-state acquisition images (several slices are omitted). Arrows point to the hypothalamic artery; arrowhead points to the A2 segment of the anterior cerebral artery.* D, *intraoperative view of the right A2 segment. Note that the artery adheres tightly to the aneurysm (arrowhead).* E, *after careful and meticulous dissection of the A2 segment from the aneurysm. Arrow points to the hypothalamic artery behind the A2 segment.*

Other Aneurysms

Small arteries or perforating arteries around the aneurysm were also assessed preoperatively in aneurysms of other locations. Critical arteries such as thalamoperforating arteries around basilar bifurcation aneurysms were precisely depicted in preoperative FIESTA images (Figure 5). Arteries larger than approximately 0.5 mm in diameter were detected as long as there was some CSF space around the aneurysm or the aneurysm was not giant.

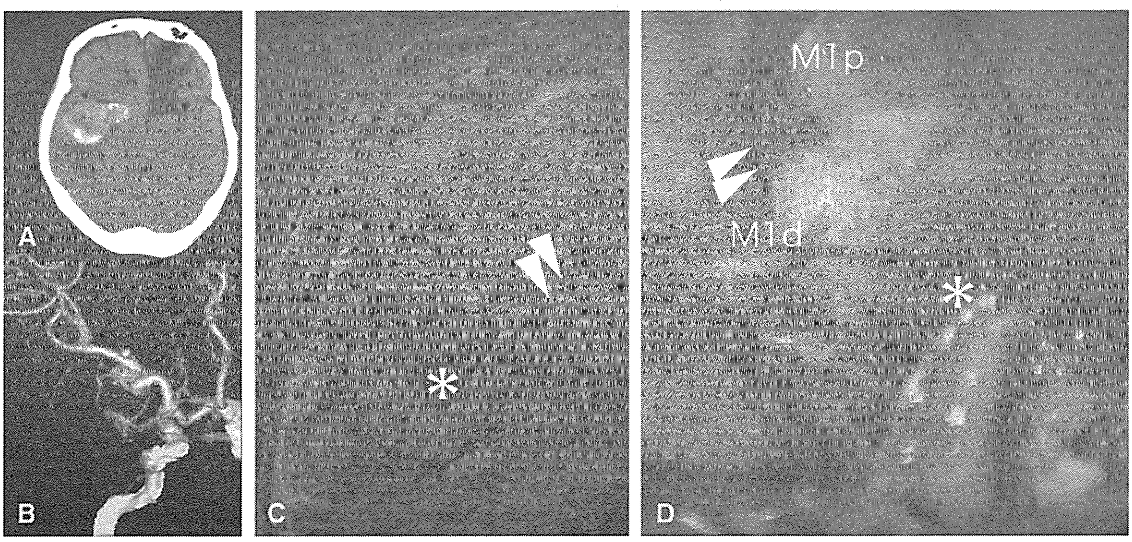

FIGURE 2. *A case of giant thrombosed middle cerebral artery aneurysm.* A, *plain computed tomography (CT) shows perianeurysmal edema and slight midline shift. Left frontal low-density area is due to a previous subarachnoid hemorrhage.* B, *virtual reality image of CT angiography.* C, *no cerebrospinal fluid signal can be identified. The lenticulostriate artery can be identified (double arrowheads). The aneurysm is shown by an asterisk.* D, *intraoperative view after resection of large part of the aneurysm. M1p and M1d, proximal and distal portion of the horizontal segment of the middle cerebral artery.*

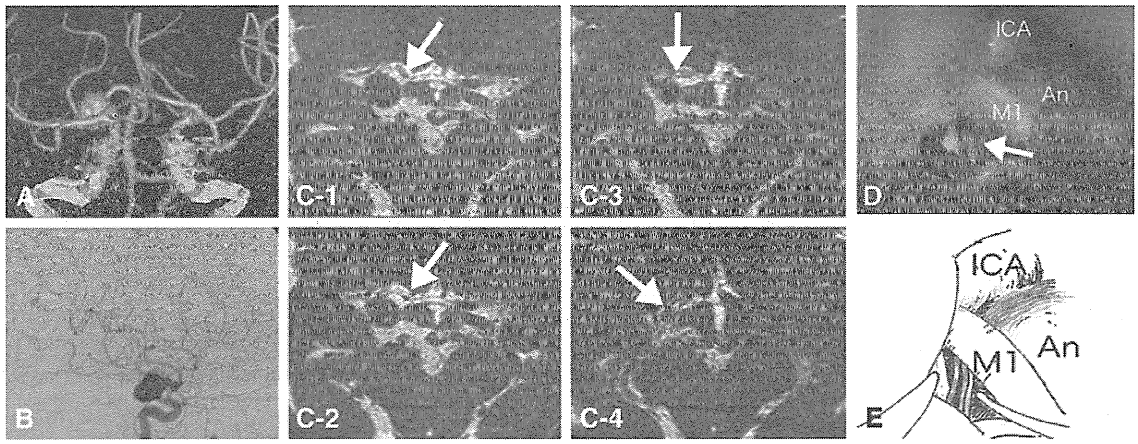

FIGURE 3. *A large internal carotid-posterior communicating artery aneurysm.* A, *preoperative virtual reality image of computed tomography angiography.* B, *lateral image of digital subtraction angiography. It is difficult to distinguish the origin of the anterior choroidal artery.* C1 *through* 4, *consecutive fast imaging employing steady-state acquisition (FIESTA) images. Arrow points to the anterior choroidal artery originating from the internal carotid artery (ICA;* C-1*) and running near the aneurysm* (C-4). D, *during the operation, the anterior choroidal arteries were identified behind the aneurysm (An), as was expected from the FIESTA image.* E, *schematic drawing of* D.

Clinical Outcome

There were no morbidities associated with taking MRIs, and no cerebral infarctions resulting from circumferential dissection were detectable on postoperative CT. Overall clinical outcome is shown in Table 1. The surgical outcomes of those patients with FIESTA imaging at 3 months after the operation included 79 excellent recoveries, 1 moderate deficit, 1 severe deficit, 1 persistent vegetative state, and no deaths according to the Glasgow Outcome Scale. Two morbidity cases were those with symptomatic thrombosed giant aneurysms.

DISCUSSION

Management strategies for UCAs are determined by balancing between natural rupture risk and surgical morbidity. The rupture

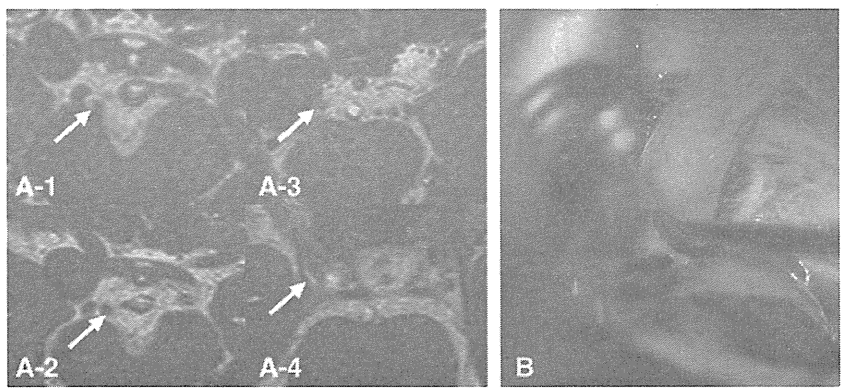

FIGURE 4. *The same case as in Figure 3.* A1 *through* A4, *fast imaging employing steady-state acquisition images. The oculomotor nerve (arrowhead) is followed from the midbrain* (A-1) *to the porus oculomotorius* (A-4). B, *intraoperative observation of the oculomotor nerve by compressing the aneurysm posteriorly under temporary clipping of the cervical internal carotid artery and ipsilateral A1 segment of the anterior cerebral artery.*

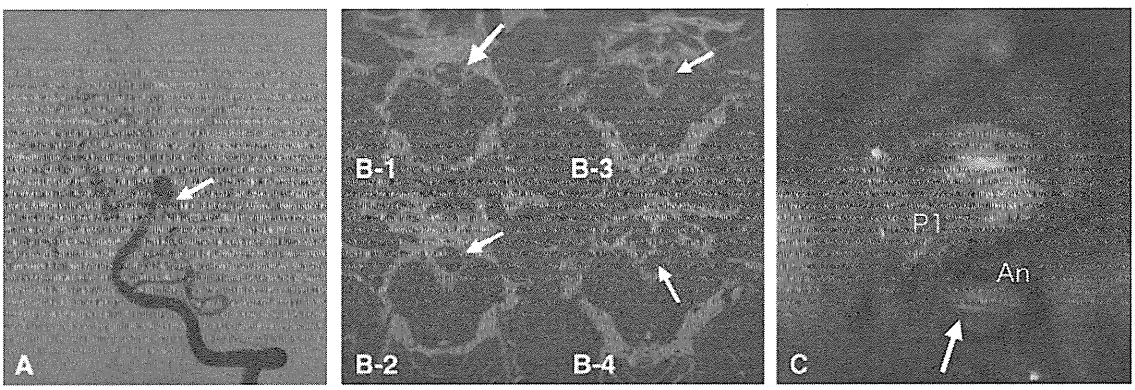

FIGURE 5. *A case of basilar bifurcation aneurysm.* A, *anterior-posterior view of the digital angiography.* **B1** *through* **B4**, *fast imaging employing steady-state acquisition images. Origin of the right thalamoperforating artery (TPA) is very close to the neck of aneurysm* (**B-1**, *arrow). The left TPA is slightly apart from the neck of the aneurysm and goes rightward over the dome of the aneurysm* (**B-4**). C, *intraoperative view via the left orbitozygomatic anterior temporal approach. Arrow indicates the TPA. An, aneurysm; P1, P1 segment of the posterior cerebral artery.*

rate of UCAs varies from 0.05%/y to 2.7%/y.[16-18] Nonetheless, if rupture occurs, clinical outcome is grave; hence, surgical interventions are recommended in a selected group of patients. Because the rupture rate is not very high, surgical morbidity must be very low to intervene in the natural history.

Several factors are associated with surgical morbidity such as age, size, posterior circulation, and patient comorbidities. In actual surgery, preserving perforating arteries and cranial nerves around the aneurysm is important to avoid clinical morbidity. By understanding the relationship and location of the perforating arteries or cranial nerves and the aneurysm preoperatively, we can assess and minimize the risk of the intervention. This knowledge helps surgeons communicate risk to patients. In some complicated cases, such knowledge can direct interventional method (open surgery vs endovascular) and the surgical approach to take.

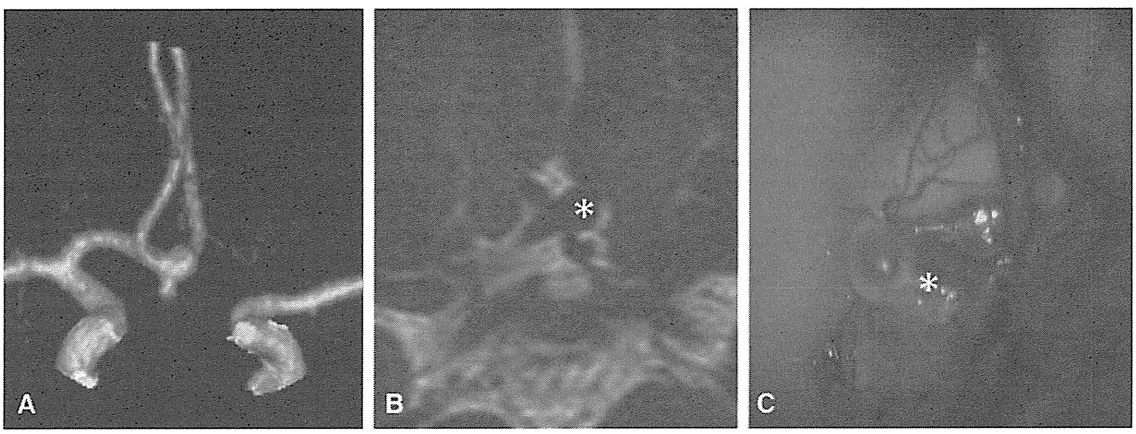

FIGURE 6. *Anterior communicating artery (Acom) aneurysm with large lamina terminalis cistern.* A, *virtual reality image of the computed tomography angiogram.* B, *fast imaging employing steady-state acquisition image shows a relatively large cerebrospinal fluid space around the aneurysm. The aneurysm is shown by an asterisk.* C, *intraoperative view through the left pterional approach. The Acom complex is exposed widely by gentle retraction without resection of the rectal gyrus after minimal dissection of arachnoid membrane and trabecula.*

For this purpose, 3D CTA and MRI have been recruited in addition to, or instead of, conventional or 3D rotational angiography. Three-dimensional CT and its original image data can provide information about the relationship between the aneurysm and bony structure, which is especially important for assessing paraclinoid and posterior fossa aneurysms. MRI can show the CSF and its contrast with soft tissue such as nerves and brain. Several reports state that either is sufficient for the preoperative evaluation for the surgery of aneurysms.[1,3-6,11,19,20] However, these modalities are complementary and should be used together.[21]

Magnetic resonance imaging has many characteristic sequences, and MR angiography might be sufficient to understand the aneurysm and nearby large arteries. In posterior fossa surgery such as microvascular decompression or removal of acoustic neurinoma, constructive interference in steady-state imaging (CISS) is often used to assess the cisternal structures.[22,23] In CISS, the contrast between the CSF and the surrounding structures is intensified; as a result, the nerves or small vessels are depicted.

Like CISS, FIESTA is a field echo sequence in which free induction decay, spin echo, and stimulated echo signals produced by radiofrequency pulse are combined simultaneously, so high signal-to-noise ratio can be accomplished. Owing to complete rephasing, this sequence has flow compensation, resulting in images with fewer flow artifacts compared with CISS imaging.[14,15]

In the preoperative assessment of UCAs, the contrast between water and solid structure is intensified, so we can understand the relationship of the nerves and vessels around the aneurysm, both of which are located in the CSF. In this study, we proved that the sensitivities and specificities of FIESTA imaging were 100% in detecting hypothalamic artery around Acom aneurysms, oculomotor nerve attachment to the posterior communicating artery aneurysm domes, and anterior choroidal artery adhesion to the posterior communicating artery aneurysms. Although the specificity was 100%, sensitivity was 56% in detecting vessel adhesion around the MCA aneurysms. In cases with MCA aneurysms, there may be unexpected perforating branches during surgery that could not detected by FIESTA images. However, if the FIESTA shows some branches, there will definitely be some important branches around the aneurysm.

In addition, we can predict the extent of the cistern around the aneurysm, which correlates with the difficulty in dissecting the aneurysm. For example, we prefer an interhemispheric approach for Acom aneurysms because of the ease of recognizing the anatomic structures of the Acom complex, including the origin of hypothalamic arteries. In those cases with large lamina terminalis cistern on preoperative images, however, we use the pterional approach because it is easy to expose the Acom complex without damaging the rectal gyrus (Figure 6).

Because the absence of CSF between an aneurysm and known structures such as adjacent main arterial trunks indicates adhesion, FIESTA images are useful for assessing the risk for aneurysmal dissection from adjacent arteries and clipping and for considering other treatment modalities.

It is often difficult to distinguish which line or dot stands for nerves or vessels on a single slice. However, by examining consequent slices to detect connecting structures, we can judge what they represent. Using a workstation or a personal computer with an imaging tool for digital imaging and communications in medicine (DICOM) data sets of the FIESTA image, it becomes much easier to grasp the course of the perforators and cranial nerves by showing the images consequently.

Cranial nerves were distinguished from small vessels in all cases by following their course from the brainstem or dural edge using the knowledge of microneuroanatomy. Thus, for an internal carotid–posterior communicating artery aneurysm, we could assess whether the oculomotor nerve was attached to the aneurysm by following it from the emergence at the midbrain. Although it was still difficult to assess how tight the adhesion was, there was a tendency for tighter adhesion between the aneurysm and oculomotor nerve in the actual surgery in cases with the more numerous slices of attachment on the FIESTA images.

Although FIESTA can provide much information on the microscopic anatomy around the aneurysm, this technique has several limitations. Sensitivity in detecting adjacent arteries around the MCA aneurysms was relatively low (56%), probably owing to a relatively small amount of CSF around these aneurysms. Small arteries that adhered to and had become flat on the surface of rather large aneurysms could not be distinguished on the preoperative FIESTA image. It was also difficult to judge whether there were any vessels when the aneurysm was large and buried in the cerebral cortex because there is no CSF between the aneurysm and the brain tissue. In MCA aneurysms or large aneurysms, a FIESTA image should not be relied on to depict all the important arteries around the aneurysm.

In this series, only open surgical cases were assessed by FIESTA images. In our institution, the majority of referred UCAs are treated surgically because of our specialty. We have performed 13 endovascular coilings during the same period, which were indicated because of patient preferences and surgeon judgment, including anticipated difficulty in separating perforating arteries by FIESTA images as stated above.

Imaging with FIESTA is introduced mainly to provide thorough information about the aneurysms before surgery. During actual surgery, after such preoperative prediction, we use modern surgical adjunctives such as neuroendoscopy and indocyanine green videoangiography. Both are very useful for detecting perforating arteries and nervous tissue and confirming intactness after clip application. Using all available techniques should lead to minimizing surgical morbidity. Although the introduction of FIESTA does not seem to contribute directly to surgical outcome, careful preoperative anatomic prediction is an essential step for successful surgeries, and this method can provide very useful anatomic information to minimize surgical morbidity and to decide on management modalities.