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Clinical Article

Intracranial Fusiform Aneurysms : It's Pathogenesis, Clinical Characteristics and Managements

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Objective : The objective of this study is to investigate clinical characteristics, management methods and possible causes of intracranial fusiform aneurysm.
Methods : Out of a series of 2,458 intracranial aneurysms treated surgically or endovascularly, 22 patients were identified who had discrete fusiform aneurysms. Clinical presentations, locations, treatment methods and possible causes of these aneurysms were analyzed.
Results : Ten patients of fusiform aneurysm were presented with hemorrhage, 5 patients with dizziness with/without headache, 4 with ischemic neurologic deficit, and 1 with 6th nerve palsy from mass effect of aneurysm. Two aneurysms were discovered incidentally. Seventeen aneurysms were located in the anterior circulation, other five in the posterior circulation. The most frequent site of fusiform aneurysm was a middle cerebral artery. The aneurysms were treated with clip, and/or wrapping in 7, resection with/without extracranial-intracranial (EC-IC) bypass in 6, proximal occlusion with coils with/without EC-IC bypass in 5, EC-IC bypass only in 1 and conservative treatment in 3 patient. We obtained good outcome in 20 out of 22 patients. The possible causes of fusiform aneurysms were regard as dissection in 16, atherosclerosis in 4 and collagen disease or uncertain in 2 cases.
Conclusion : There is a subset of cerebral aneurysms with discrete fusiform morphology. Although the dissection or injury of internal elastic lamina of the cerebral vessel is proposed as the underlying cause for most of fusiform aneurysm, more study about pathogenesis of these lesions is required.

KEY WORDS : Fusiform aneurysm · Cerebral aneurysms · Dissection · Atherosclerosis · Lamina elastica · Collagen disease.

INTRODUCTION

Intracranial aneurysms are classified according to their shape into saccular and nonsaccular types. Fusiform aneurysms are nonsaccular dilatations involving the entire vessel wall for a short distance[2,4,9,25]. They are termed cylindrical if it involves a somewhat longer length[34]. This type of aneurysm may be caused by dissection or atherosclerosis[6], by disorders of collagen and elastin metabolism, by infections and-very rarely-by neoplastic invasion of the arterial wall[34]. Fusiform aneurysms have different underlying pathologies, hemodynamics, anatomical distributions, natural histories and treatments than do the saccular variety[6]. The patient often has symptoms and signs of occlusion, arterial rupture, or a mass effect.

Intracranial fusiform aneurysms are rare, although the number of cases has increased in recent years. They represent about 3%-13% of all intracranial aneurysms[2] and are usually located in the vertebrobasilar system[7-9]. Fusiform aneurysms in the anterior circulation remain rare and occur mostly in the middle cerebral artery and internal carotid artery[6,13]. There are some sporadic case reports about the treatment of fusiform aneurysms[2,4,13,34] and a few reports on the clinical characteristics, pathogenesis and treatment methods of these aneurysms in large series[6-8,11].

We have reviewed 22 cases of fusiform aneurysms that have been treated at our institute, and we describe the clinical characteristics, treatment methods and outcomes after management of patients with these lesions. We also discussed the possible pathogenesis of these aneurysms and present various examples of fusiform aneurysms caused by dissection, atherosclerosis, collagen disease, or unknown factors.

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MATERIALS AND METHODS

We have reviewed the medical records of 22 patients with fusiform aneurysms among 2,458 patients treated for

aneurysms between September 1982 and June 2007. Diagnoses were made from the findings of computed tomographic angiography (CTA), magnetic resonance angiography (MRA) and cerebral angiography. Typical dissecting aneurysms of the vertebral artery were excluded, because of their well known different mechanisms of development. The medical records of these patients were reviewed to determine their clinical characteristics, where the aneurysms originated, treatment methods and outcomes after management. The clinical presentation here is based on the symptoms of the patient at the time of diagnosis; an incidental case is defined as that discovered during surgery for another symptomatic aneurysm or workup for other disease. The sites within the middle cerebral artery (MCA) and anterior cerebral artery (ACA) are defined as follows: M1, the horizontal portion of the MCA proximal to the genu; M2, the insular segment of the MCA lying within the insular compartment of the Sylvian fissure; M3, the region distal to M2; A1, the horizontal segment of the ACA proximal to the anterior communicating artery (Acom); A2, the region distal to Acom. Treatment is defined as follows. Wrapping followed by clipping means that the fusiform aneurysm was wrapped initially with pieces of Bemsheets (Kawamoto Corporation, Osaka, Japan) or periosteum, because the aneurysmal neck could not be clipped, followed by securing the reinforced wrapping materials with aneurysmal clips. Clipping followed by wrapping means that the aneurysm was occluded incompletely, so the remaining portion of the aneurysmal neck was reinforced by wrapping with pieces of Bemsheets or periosteum. Extracranial-intracranial (EC-IC) bypass was performed by anastomosis of the superficial temporal artery (STA) to the MCA branch in all cases. Conservative treatment means medication with antiplatelet drugs in patients who presented with ischemic symptoms. Outcome is defined as follows : excellent-the patient was neurologically normal; good-neurological abnormalities were detected on examination but the patient returned to full activities of normal living; fair-neurological abnormalities prevented the patient from returning to normal life.

We also describe the possible developmental causes and mechanisms of these aneurysms, based on the neuroimaging, pathological, intraoperative findings and literature review.

RESULTS

Clinical characteristics

The ages of the patients ranged from 15 months to 68 years (mean 45.1 years). More than half of the patients were younger than 50 years (60%). Thirteen were men and nine were women.

The clinical presentations are summarized in Table 1. The presenting features were categorized as subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), neurological deficit from ischemia, neurological deficit from the mass effect of the aneurysm, nonspecific symptoms such as dizziness with or without headache, and incidental findings. Eight patients presented with SAH and one of these developed it following an ischemic attack. Two patients presented with ICH; four with ischemic neurological deficits; one with abducens nerve palsy caused by compression from the aneurysm and two with incidental aneurysms.

The locations of aneurysms are summarized in Table 2. Seventeen aneurysms were located in the anterior circulation and five in the posterior circulation. Among the cases involving the anterior circulation, 12 were located on the MCA, three on the internal carotid artery (ICA) and two on the ACA. Among the five cases of posterior circulation aneurysms, two were located on the posterior inferior cerebellar artery (PICA), two on the vertebral artery (VA), and one on the posterior cerebral artery (PCA). The most frequent site of fusiform aneurysm in this study was the MCA.

Table 1. Clinical presentation in patients with fusiform aneurysm

Presentation	No. of cases
Subarachnoid hemorrhage	8*
Intracerebral hemorrhage	2
Neurological deficit (ischemia)	4
Dizziness with/without headache	5
Cranial nerve deficit from mass effect	1
Incidental findings	2

*One case presented with a SAH followed an ischemic attack

Table 2. Location of fusiform aneurysm

Location	No. of cases
Anterior circulation	17
ICA	3
MCA	12
M1	2
MCA bifurcation	3
M2	4
M3	3
ACA	2
A1	1
A2	1
Posterior circulation	5
PCA (P2)	1
VA	2
PICA	2

ICA: internal carotid artery, MCA: middle cerebral artery, ACA: anterior cerebral artery, PCA: posterior cerebral artery, VA: vertebral artery, PICA: posterior inferior cerebral artery

Treatment methods and outcome after management

The treatment methods are summarized in Table 3. Several treatment options were used in the treatment of intracranial fusiform aneurysms. Three patients were treated with wrapping of the aneurysm followed by clipping of the wrapping materials; two with clipping of the aneurysm followed by wrapping of the remaining portion; two with clipping alone; six with resection; five by proximal occlusion with a coil after EC-IC bypass or not, and one with EC-IC bypass only. The other three aneurysms were treated conservatively.

Table 3. Treatment methods of fusiform aneurysm

Treatment	No. of cases
Wrapping followed by clipping	3
Clipping followed by wrapping	2
Clipping	2
Resection	6
Proximal occlusion with coil with (1)*/ without (4)* EC−IC bypass	5
EC−IC bypass only	1
Conservative treatment	3

*Number of case(s). EC−IC : extracranial−intracranial

Table 4. Outcomes of patients with fusiform aneurysm

Outcomes	No. of cases
Excellent	17
Good	3
Fair	1*
Dead	1†

*due to vasospasm, †due to rebleeding after clipping

Table 5. Possible causes of fusiform aneurysm

Causes	No. of cases
Dissection	16
Atherosclerosis	4
Collagen disease or unknown	2

The results of treatment and the final clinical outcomes for patients are summarized in Table 4. Seventeen patients recovered completely after treatment. Some neurological deficits developed in three patients, but they returned to full normal activities ultimately. Severe neurological deficit occurred in one patient caused by vasospasm during treatment of a ruptured aneurysm and another patient died because of rebleeding from a rupture of the clipped aneurysm. In particulr, five patients among the 22 patients, who were treated with wrapping or clip with wrapping, needed longer follow up for the results. Of these patients, one died, one didn't have follow up, one had follow up for 3 years, one had 2 year follow up and one developed ICH on the other side thalamus in 5 years, that indicates patients had longer than two years of follow up.

Possible causes and mechanisms

The possible causes of fusiform aneurysms in this study are summarized in Table 5. The causes of 16 cases were vessel dissection, four were caused by atherosclerosis (Fig. 5A) and two by a collagen disease or unknown factor (Fig. 5B).

Day et al.[6] proposed a sequence of the evolution of spontaneous fusiform aneurysms caused by dissection. Based on this, six stages of fusiform aneurysm were noted

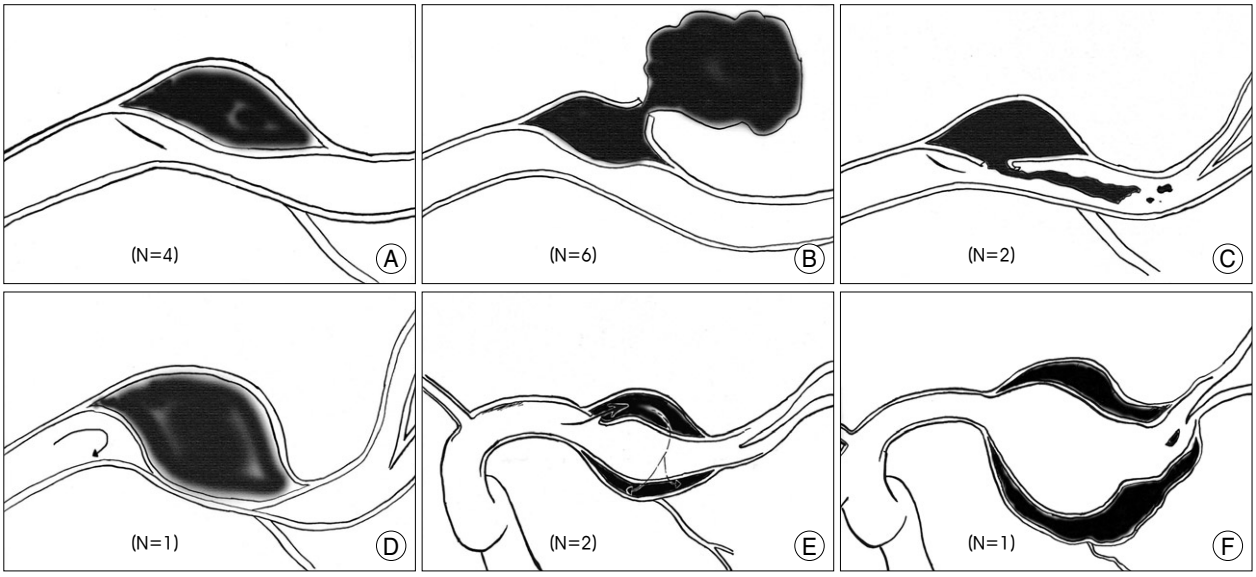

Fig. 1. Drawings show pathogenesis of fusiform aneurysm by dissection[6]. Arrows indicate direction of blood flow. A : Arterial dissection with intramural hemorrhage between the intima and media producing focal narrowing of vessel. B : Arterial dissection with rupture producing extension of blood into subarachnoid space or brain. C : Rupture of a dissection into the arterial lumen producing a distal embolization. D : Expansion of intramural clot leading to vessel occlusion. E : Progress enlargement of dissection both laterally and longitudinally. F : Serpentine channel within dissected thrombotic aneurysm. ( ) : number of case(s).

in the present study (Fig. 1). Arterial dissection with intramural hemorrhage between the intima and media producing focal narrowing of vessel was noted in 4 cases (Fig. 1A, 2A , B). Arterial dissection with rupture producing bleeding into the brain or subarachnoid space was noted in 6 cases (Fig. 1B, 2C, D). Rupture of a dissection into the arterial lumen producing a distal embolization was noted in 2 cases (Fig. 1C, 3A, B) and the further expansion of an intramural clot leading to vessel occlusion was noted in 1 case (Fig. 1D, 3C, D). Progress enlargement of dissection both laterally and longitudinally was noted in 2 cases (Fig. 1E, 4A-C) and serpentine channel within dissected thrombotic aneurysm was noted in 1 case (Fig. 1F, 4D-G).

Histopathology of a specimen of resection showed focal loss of the internal elastic lamina along part of the vessel wall with or without marked thickening of intima (Fig. 2D, 3D, 4C).

DISCUSSION

Fusiform aneurysm is a morphological term with no reference to the origin or clinical features of the lesion[13]. It is defined as a circumferential arterial dilatation resulting from pathological involvement of the entire artery[2,4,9,28,34]. All aneurysms exhibit a spindle shape when viewed externally[6]. Conceptually, there is still confusion as to the etiological, clinical and radiological features of fusiform aneurysms. Several investigators have applied the term "atherosclerotic" as the cause of fusiform aneurysms[7-9,23,25]. In fact, advanced atherosclerotic arteries have a slightly fusiform appearance. However, the classic dissecting aneurysm also has a fusiform appearance[23] and several authors have recently reported the presence of fusiform aneurysms caused by dissection[2-6,9-11,13,16,17,21,23,24,26,34]. Therefore, all cases of fusiform aneurysms based on external view without

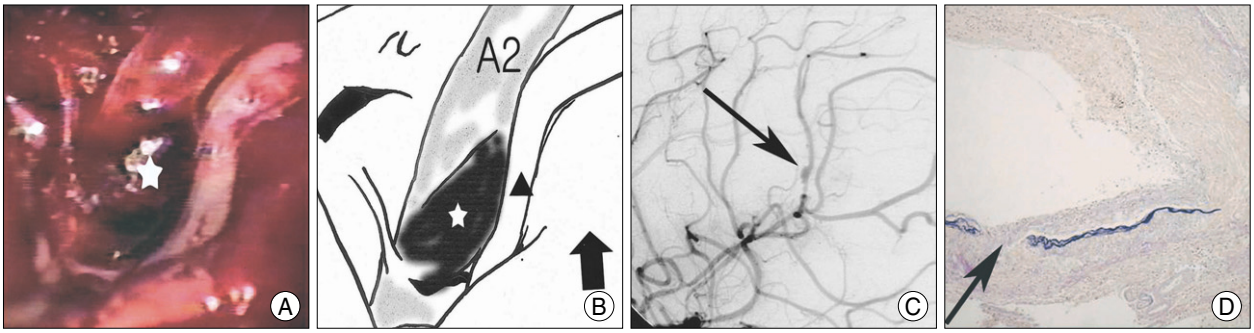

Fig. 2. Example of arterial dissection with intramural hemorrhage producing focal narrowing of vessel (A & B) and rupture producing bleeding into brain (C & D). A : Intraoperative surgical microscopic picture of A2 in a 55 year-old male who received surgery for ruptured Acom aneurysm shows arterial dissection with intramural hemorrhage (marked in asterisk) with narrowing of vessel. B : Schematic drawing for the photograph(A) showing A2 with arterial dissection near partial resected gyrus rectus (arrowhead) and frontal lobe (arrow). C : Cerebral angiogram, lateral view, showing fusiform aneurysm (arrow) at central branch of right middle cerebral artery in 27 year-old male who presented with intracerebral hematoma. D : Histopathological findings of that aneurysm of resection showing focal loss of the internal elastic lamina (arrow) along part of the vessel wall (Elastic stain, original magnification ×100).

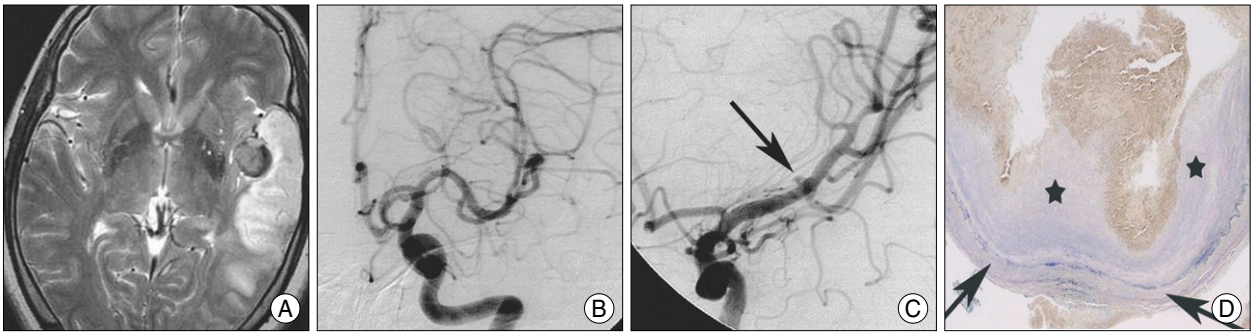

Fig. 3. Example of rupture into the true lumen with distal embolization(A & B) and expansion of intramural clot leading to vessel occlusion (C & D). A : Brain magnetic resonance image, T2-weighted axial view, showing a round thrombosed aneurysm surrounding with perilesional edema in 53 year-old female who presented with speech disturbance. B : Cerebral angiogram, A-P view of arterial phase, showing filling of small part of thrombosed aneurysm. This aneurysm was occluded with Guglielmi detachable coils by endovascular method. C : Cerebral angiogram, oblique view of arterial phase, showing abrupt occlusion of middle cerebral artery branch (arrow) in 42 year-old female who presented with a subarachnoid hemorrhage. D : Histopathologic findings of aneurysm of resection showing an organized thrombus, irregularly thickened intima (asterisk), and a fragmented or degenerated internal elastic lamina (arrows) along the vessel wall. There was no evidence of atheromatous plaque (Elastic stain, original magnification ×40).

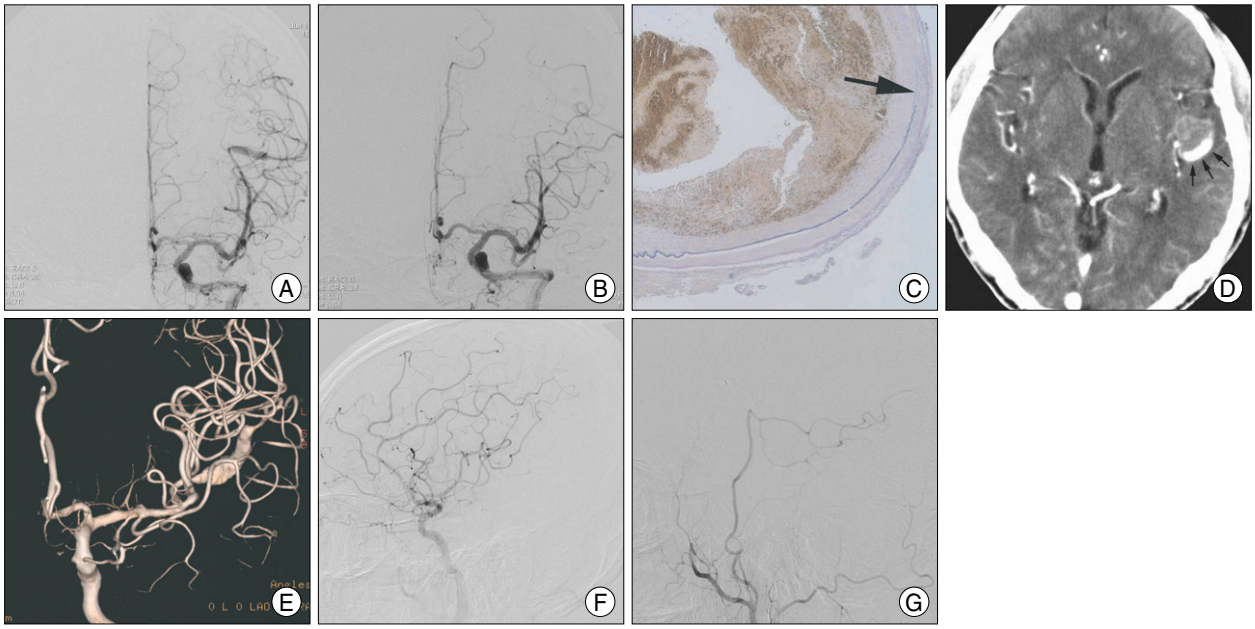

Fig. 4. Example of progress enlargement of dissection both laterally and longitudinally (A, B and C) and serpentine channel within dissected thrombotic aneurysm (D, E, F, and G). A : Cerebral angiogram, anterior-posterior (A-P) view of arterial phase, showing fusiform dilated vessels at left M3 in a 38 year-old male who presented with SAH. B : Postoperative angiogram, A-P view of arterial phase, showing disappearance of previous fusiform aneurysm. C : Histopathologic findings of aneurysm showing disconnected internal elastic lamina (arrows) along the vessel wall. (Elastic stain, original magnification ×40). D : Axial view of enhanced brain computed tomography showing serpentine channel within thrombosed aneurysm(arrow) at left sylvian fissure in 68 year-old male who presented with dizziness. E : Three dimensional cerebral angiogram showing fusiform aneurysm at left M2. The aneurysm was resected followed by a superficial temporal artery-middle cerebral artery anastomosis. F & G : Postoperative internal (F) and external carotid (G) angiogram, lateral views of arterial phase, showing disappearance of previous aneurysm and filling of blood to distal M3 through superficial temporal artery.

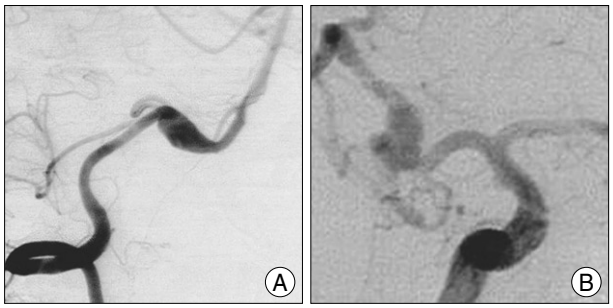

Fig. 5. Example of pathogenesis of fusiform aneurysm by atherosclerosis (A) and by collagen disease or unknown cause (B). A : Oblique view of right vertebral artery (VA) angiogram showing a fusiform aneurysm of VA at proximal to posterior inferior cerebellar artery in 54 year-old male who presented with right abducens nerve palsy. This aneurysm was treated with occlusion of right VA and the aneurysm with Guglielmi detachable coils by endovascular method. B : Oblique view of cerebral angiogram showing a fusiform aneurysm at bifurcation of right middle cerebral artery in 37 year-old female who complained of headache. This aneurysm was treated with clipping of aneurysm followed by wrapping with periosteum.

consideration of etiology were included in this study except well known typical cases of dissecting aneurysm of the vertebral artery.

The age and sex distribution of patients with fusiform aneurysms differ from those with saccular aneurysms. The mean age of patients in this study was 45.1 years and the male/female ratio was 1.4:1. This contrasts with that of patients with saccular aneurysms[36]. Our study confirms previous reports that spontaneous fusiform aneurysms are more often found in younger patients[2,4,6,9,13,19,26,29,31,34,35] and are more frequent in men[6,13,28,35].

The clinical features of fusiform aneurysms are categorized morphologically. They can progress from a small focal dilatation or vessel narrowing, to a relatively thick-walled, tortuous dilatation and elongation of the artery. They can be incidental or asymptomatic, discovered during workup for unrelated symptoms. They can present as a nonspecific headache without hemorrhage or other neurological signs or symptoms, as ischemia, transient ischemic attack, or complete stroke, as mass effect with or without seizure, or as hemorrhage, subarachnoid or intraparenchymal lesions. Day et al.[6] reported that patients with small, large and giant aneurysms with focal dilatations of their lumen had subarachnoid hemorrhage rates of 80%, 62% and 23%, respectively, whereas ischemic symptoms such as transient ischemic attack or complete stroke were the presenting feature in 31% of their patients. They also mentioned that hemorrhage was the most common presentation in patients with small lesions with focal dilatation, whereas ischemic symptoms were the most common presentation of patients

with stenosis or occluded vessels. Sato et al.[33] reported a case of fusiform aneurysm of the vertebral artery presenting hemifacial spasm by mass effect, which was treated by intravascular embolization. In the present study, fusiform aneurysms presented with hemorrhage in ten (45%) of the 22 patients, neurological deficit from ischemia in four, dizziness with or without headache in five, cranial nerve deficit from mass effect in one and incidental findings in two.

Fusiform aneurysms in this study were found in the anterior circulation in 17 (77%) of the patients and in the posterior circulation in 23%. Dissection has been proposed as the main underlying cause of fusiform aneurysms and most commonly involves the posterior circulation, especially vertebral and basilar arteries[1,3,5-10,15,16,19,20,24-26,32,35] However, typical cases of dissecting aneurysm in the posterior circulation were excluded in this study. Dissecting aneurysms can originate in any regions of the anterior circulation, such as the ICA[1,29], MCA[2,4,6,10,13-16,22,26,30], the ACA[3,10,16-18,24,27,30,32,35] and rarely in the anterior choroidal artery[21]. The MCA is the most common[6]. Day et al.[6] reviewed 102 cases of spontaneous fusiform middle cerebral artery aneurysms. They found that 69% of the aneurysms originated proximal to the MCA genu (M1 segment), 21% were insular (M2 segment) and 10% were distal (M3 or M4 branches). In the present study, the MCA was also the most frequent site of origin, followed by the ICA and ACA.

Various etiological factors for fusiform aneurysms have been proposed, including atherosclerosis, vessel dissection and association with other diseases such as von Recklinghausen's disease, fibromuscular dysplasia, systemic lupus erythematosus and various collagen-associated vascular diseases[4,6,30,34]. Based on the radiological, operative and histopathology findings in this study, vessel dissection was the leading cause, followed by atherosclerosis and collagen disease or unknown factors. The initial event in the formation of atherosclerotic fusiform aneurysms is thought to be lipid deposition in and beneath the intima. This disrupts the internal elastic membrane and infiltrates the muscular wall[35]. Intramural hemorrhage and rupture of the atheroma leads to transmural extension of the thrombus and thickens the intima to create the fusiform shape of the aneurysm[23,35]. Rupture of the vasa vasorum by shear forces or by stress on the luminal wall then causes intimal tear and fracture of the internal elastic membrane. This permits bleeding into the arterial wall to form a hematoma[1,6]. If the dissection occurs between the internal elastic lamina and the media, the vessel lumen becomes narrow or occluded with an intramural hematoma and the patients present with ischemic symptoms[1,3,6,13,23,35]. If dissection occurs between the media and adventitia, the aneurysm can rupture and the patient will present with SAH or ICH[1,3,6,13,23,35]. An intramural thrombus that ruptures into the lumen will cause a distal embolization and further expansion of the intramural clot will lead to vessel occlusion[6]. After occluding vessel by intramural clot, it can be recanalized and enlarged the dissection both laterally and longitudinally. Serpentine channel forms as disease extends longitudinally, combined with varying degrees of intraluminal thrombosis. In this study, disconnection or injuries of the internal elastic membrane were recognized in histopathology of the surgical specimens (Fig. 2D, 3D, 4C).

Treatment of fusiform aneurysms should be based on the presence and type of symptoms, the lesion size and location and the risk of any accompanying intervention. Day et al.[6] have suggested guidelines for the treatment of patients with dissecting aneurysms of the MCA. They recommend that most small and some large focal dilatations, especially those that are asymptomatic, should be treated conservatively unless serial neuroimaging assessment indicates significant enlargement over time. However, the appearance of symptoms requires aggressive intervention. Lanzino et al.[17] and Nikawa et al.[26] also recommended conservative treatment in patients with dissecting aneurysms without neurological deterioration or recurrent SAH because of the possibility of spontaneous evolution of a dissecting aneurysm. In general, we agree with the recommendation of conservative treatment for non symptomatic dissecting aneurysms. However, we also recommend clipping using encircling clips for focally dilated dissecting aneurysms if they are found during surgery for another symptomatic aneurysm, and aggressive surgical treatment for fusiform aneurysms which are not caused by dissection because of the possibility they will progress.

Day et al.[6] and several other authors[3,8,17,32,35] recommend that patients with stenotic or occlusive lesions presenting with acute ischemic symptoms should be treated conservatively. However, Kurino et al.[16] reported a patient with a dissecting aneurysm in the MCA who presented with ischemic symptoms and who showed a poor outcome after conservative treatment. They recommend surgical revascularization distal to the compromised artery. We also agree with the need for conservative treatment for patients with stenotic or occlusive lesions and acute ischemic symptoms. However, we experienced a case with a fusiform dilatation dissecting aneurysm in the PICA with cerebellar ischemic symptoms, which bled later after conservative treatment. The aneurysm was treated successfully with endovascular method. Therefore, we recommend consideration of aggressive treatment with endovascular or surgical methods for focal dilating fusiform aneurysms.

Several authors[6,26] have mentioned that direct "aneurysm only" clip application was associated with a high rate of regrowth of the lesion or rebleeding caused by preserving flow in the normal half of the affected vessel, despite excellent intraoperative aneurysm obliteration. Therefore, they recommended a proximal occlusion or trapping with or without resection combined with end-to-end anastomosis or EC-IC bypass[3,4,6,9-13,17,28,31,34,35]. We also experienced one patient who died from rebleeding after clipping of a fusiform aneurysm at A1. However, we do not agree that all fusiform aneurysms should be treated with proximal occlusion or trapping combined with EC-IC bypass. Some can be treated using encircled aneurysm clips, such as the Sundt clip, by partial clipping followed by wrapping if the aneurysm is not ruptured case, and by occlusion of the aneurysm and parent vessel with packing of the coils by an endovascular method. Other authors[5,12,19,20,33] have reported that fusiform or dissecting aneurysms can be treated using endovascular methods.

CONCLUSION

We have reviewed twenty two cases of patients with fusiform aneurysms who have been treated at our institute. We investigated the clinical characteristics, treatment methods and outcomes after treatment of these lesions. We have discussed the possible causes of these aneurysms, such as dissection, atherosclerosis, collagen disease, or unknown factors. Based on the spectrum of clinical, neuroimaging and pathological findings, there is clearly a distinct subset of cerebral aneurysms with fusiform morphology. Although dissection or injury of internal elastic lamina of the cerebral vessel is the probable underlying cause for most fusiform aneurysms, more studies on their pathogenesis are required.

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