Giant fusiform aneurysms in the middle cerebral artery presenting with hemorrhages of different origins

Report of three cases and review of the literature

NOBUTAKA HORE, M.D., NOBUAKI TAKAHASHI, M.D., SHOJI FURUCHI, M.D., KATSUHARU MORI, M.D., MASANARI ONIZUKA, M.D., KEISUKE TSUTSUMI, M.D., AND SHOBU SHIBATA, M.D.

Department of Neurosurgery, Nagasaki University School of Medicine, Nagasaki; and Department of Neurosurgery, Fukuoka Kinen Hospital, Fukuoka, Japan

Three cases of giant fusiform aneurysms in the middle cerebral artery (MCA) presenting with hemorrhages of different origins are reported, and appropriate literature is reviewed to investigate the characteristics of these lesions. Two patients had suffered a subarachnoid hemorrhage and the other had an intramural hemorrhage (dissection). Pathologically, these aneurysms presented with hemorrhages of different origins; classic rupture type (Case 1), dissection type (Case 2), and atherosclerosis-related thrombosis type (Case 3).

Based on surgical and pathological investigations in these three cases and a review of the reported literature, the authors propose that giant fusiform aneurysms in the MCA are characterized by weaknesses in the internal elastic lamina with intimal thickening. Therefore, these lesions have the potential to present with hemorrhage in each of the three types. This finding indicates that there is a strong relationship between the pathological features of giant fusiform aneurysms and their clinical course, and that it is necessary to determine appropriate therapy for giant fusiform aneurysms in the MCA by evaluating cerebral blood flow, even if the lesions are found incidentally.

KEY WORDS • giant fusiform aneurysm • middle cerebral artery • hemorrhage • ruptured aneurysm • dissecting aneurysm • atherosclerosis

Case Reports

Case 1

History. This 35-year-old woman with no significant medical history was admitted to our hospital in a semicomatose state 1 hour after the onset of severe headache and vomiting.

Examination. At presentation, the patient had right hemiplegia. Admission CT scans demonstrated SAH in the suprasellar cistern and hematoma in the left sylvian fissure (Fig. 1A and B). Left ICA angiography revealed a giant fusiform aneurysm in the temporal branch (M2) of the left MCA (Fig. 1C); the wall of the aneurysm was smooth.

Course of Illness. The patient underwent an emergency left frontotemporal craniotomy. On opening of the left sylvian fissure, a giant cerebral aneurysm was exposed. A rupture point was identified at the distal site of the aneurysm. The lesion was resected and end-to-end anastomosis was performed using the STA (Fig. 1D and E). The patient had a satisfactory postoperative course, and CT scans obtained postoperatively (Fig. 1F) revealed no infarction except for a small amount of brain damage caused by the initial intra-
cerebral hematoma. She was discharged with no neurological deficit 2 months postsurgery.

Pathological Examination. The aneurysm had a fragmented or completely absent IEL and equally thickened intima. The luminal surface was smooth, and no atheromatous deposits were detected (Fig. 1G).

Case 2

History. This 37-year-old male pilot with no significant medical history was hospitalized for acute onset of left oculodynia and vomiting during a flight.

Examination. The results of a neurological examination were unremarkable. An analysis of his cerebrospinal fluid showed normal findings. Admission MR imaging demonstrated an aneurysm in the left sylvian fissure; the lesion had an intramural hematoma that appeared hyperintense on T1-weighted and hypointense on T2-weighted images (Fig. 2A and B). A subintimal flap was demonstrated on T2-weighted images. Left ICA angiography revealed a giant fusiform aneurysm in the temporal branch (M2) of the left MCA. The aneurysm had an irregular, stenotic portion near the proximal end (Fig. 2C).

Course of Illness. The patient underwent an emergency left frontotemporal craniotomy. On opening of the left sylvian fissure a giant fusiform aneurysm was exposed; it was dark red and hard on the frontal side, and a normal color and soft on the temporal side. The temporal branch (M2) was larger in the distal than in the proximal portion, and the anterior temporal artery was well developed. The aneurysm was trapped with a straight clip (proximal 7 mm, distal 9 mm) after STA–MCA double anastomoses for the middle temporal and posterior temporal arteries were performed, because end-to-end anastomosis to the distal end was technically difficult (Fig. 2D and E). We assumed that dissection had occurred in the giant fusiform aneurysm, judging from presentation, MR imaging findings, angiographic findings, and intraoperative findings. The patient experienced transient right hemiparesis and aphasia after surgery, but postoperative CT scanning (Fig. 2F) revealed protection of the surrounding brain. He was discharged with no neurological deficit 1 month after surgery.

Case 3

History. This 75-year-old man with previously diagnosed hypertension was admitted to our hospital in a comatose state with right hemiplegia 1 hour after onset of coma.

Examination. Admission CT scans demonstrated SAH in the suprasellar cistern and a mass in the left sylvian fissure (Fig. 3A). Left ICA angiography revealed a giant fusiform aneurysm with an irregular contour in the temporal branch (M2) of the left MCA (Fig. 3B).

Course of Illness. The patient underwent an emergency left frontotemporal craniotomy. On opening of the left sylvian fissure a giant cerebral aneurysm was exposed; it was dark red and very hard. A rupture point was identified at the distal site of the aneurysm. The lesion was resected after...
end-to-side and end-to-end double anastomoses in which the STA was used (Fig. 3C and D). Unfortunately, the patient’s general condition grew steadily worse and he died 2 days later.

**Pathological Examination.** The aneurysm had a degenerated IEL, unequally thickened intima, and organized thrombus (Fig. 3E). The luminal surface was irregular and narrowed. The wall of the aneurysm had atheromatous deposits and hemorrhage in the atheroma (Fig. 3F).

**Discussion**

“Fusiform aneurysm” is a morphological term with no reference to the origin or clinical features of the lesion. Giant fusiform aneurysms (≥ 2.5 cm in length) are defined as those having circumferential arterial dilation resulting from pathological involvement of the entire artery. This type of lesion is rare, accounting for 5 to 17.6% of giant aneurysms, which themselves represent 3 to 13% of all intracranial aneurysms. Giant fusiform aneurysms are reportedly more common in the vertebrobasilar circulation. Nevertheless, autopsy data indicate that these aneurysms occur just as frequently in the anterior circulation. A consideration of the possible pathogenetic mechanisms offers no clues to support the observation that fusiform aneurysms occur predominantly in the posterior circulation. Giant fusiform aneurysms, when present, are frequently multiple, but our patients had no other lesions.

In our patients, the giant fusiform aneurysms were located on a trunk of the left MCA. Some authors have discussed the treatment of these lesions in the MCA, but few have discussed the relationship between pathological features and clinical courses. Pathogenesis of Giant Fusiform Aneurysms

In the intracranial arteries, the IEL containing the elastic tissue is the most important layer for determining the strength of the vessel wall. Therefore, vessels are more prone to damage if the elastic tissue is defective. Hemodynamic stress is presumed to be the primary factor causing remodeling, degeneration, and loss of the IEL. In contrast, the intima thickens as a physiological response to hemodynamic stress. We recognize that fusiform aneurysms are the final product of a dynamic pathological process in the arterial wall.

There have been few pathological studies of fusiform intracranial aneurysms in the vertebrobasilar artery, and those performed have focused intently on the atherosclerotic changes in the parent vessels.
reported that loss of the arterial elastic membrane, arteriosclerosis, and hypertension seemed to be the three main factors underlying the pathogenesis of arterial ectasia. Shokunbi and colleagues reported that microscopic examination of the walls of fusiform aneurysms revealed atheromatous degeneration, focal wall attenuation, mural hemorrhage, rupture, and acute and chronic inflammatory cell infiltration. These findings indicated that rupture is not rare and that atherosclerosis is but one mechanism in the pathogenesis of such lesions. In contrast, several authors have recently reported the presence of fusiform aneurysms that were unrelated to atherosclerosis.

Pathologically, our cases presented with hemorrhages of different origins; classical rupture, dissection, and atherosclerosis-related thrombosis types (Fig. 4). In all cases, the aneurysm wall was mostly thinned and fibrotic with either an absence of or several fragmented portions of IEL and a markedly reduced muscle layer. There was no evidence of atheromatous lesions such as plaque, calcification, or ulceration in the wall of the aneurysms in Cases 1 and 2. Because these patients were relatively young, the findings contradicted the idea that atherosclerosis was the pathogenic process; the presence of other factors such as congenital abnormalities is indicated.

Yasui and colleagues reported that the presence of a fusiform aneurysm in the vertebral artery is a predisposing factor for a dissecting aneurysm in that vessel. Anson, et al., reported that fusiform and dissecting aneurysms have the same pathological features in the vertebral artery, and that fusiform lesions have the potential to become dissect-
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ing aneurysms or to grow. We suggest that fusiform aneurysms in the MCA have the same mechanism, and also have the potential to grow and dissect (Case 2).

In contrast to the first two cases, the wall of the aneurysm in Case 3 showed marked atherosclerotic degeneration. Once the artery is dilated, the blood flow becomes sluggish, predisposing an artery to thrombosis. It is not clear, however, whether giant fusiform aneurysms form thrombi in younger patients without atherosclerosis. Nagahiro and colleagues\(^{19}\) stressed that a small hemorrhage in the thrombus has a critical role in aneurysm rupture; they called this small vessel a capillary channel.\(^{19}\)

In our patients, origins of presenting hemorrhages were all different clinicopathologically, but fragmentation or loss of the IEL played a critical role in growth or rupture in each case. Therefore, strict control of blood pressure is necessary in patients with giant fusiform aneurysms in the MCA. Because there has been little pathological confirmation of the sources of these aneurysms, pathological findings in other patients will be needed to reinforce these speculations.

**Method of Treatment**

The surgical management of intracranial giant fusiform aneurysms is controversial, but these lesions have a poor natural history that differs from that of typical, saccular aneurysms.\(^{19}\)

One surgical option is reconstruction of the vessel wall by using multiple clips, but it is difficult to perform. This was not thought to be feasible in our cases, because of technical difficulty (Case 1), intramural hemorrhage (Case 2), and organized thrombus (Case 3). Resection of the aneurysm and end-to-end anastomosis or EC–IC bypass is another possible treatment.\(^{1}\) We performed resection of the aneurysm with EC–IC bypass in Cases 1 and 3 to improve the mass effect at the acute stage. Proximal clipping or aneurysm trapping with EC–IC bypass is probably the most common treatment; we used this procedure in Case 2. We used the STA (a relatively low-flow type of donor artery) for EC–IC bypass because of the risk of hyperperfusion if using a higher-flow bypass with a vein or the radial artery. We would use a higher-flow bypass for ICA occlusion. Furthermore, we think that a single anastomosis to the distal end of the MCA is enough for an EC–IC bypass, but we performed double anastomoses to cortical branches of the MCA because of the technical difficulty involved with end-to-end anastomosis to the distal end (Case 2) or its vascular structure (Case 3).

Wrapping the aneurysm or conservative treatment has been described, but it is ineffective because of the high risk of rupture.\(^{1}\) Recently, Hoh, et al.,\(^{13}\) reported on the combined surgical and endovascular techniques of flow alteration to treat giant fusiform aneurysms. Their idea is reasonable because hemodynamic stress plays a critical role in the growth of fusiform lesions. We emphasize that no treatment option is perfect in this situation. Determination of appropriate therapy by evaluating the cerebral blood flow is necessary for giant fusiform aneurysms, even if they are found incidentally. Nakatomi, et al.,\(^{20}\) reported that the formation of intramural hemorrhage within the thrombus seemed to be a critical event necessary for lesions to become symptomatic and to progress. Monitoring of intramural hemorrhage with MR imaging can help us determine the timing of surgical intervention.

**Conclusions**

Giant fusiform aneurysms in the MCA have the potential to present with hemorrhages of different origins: classic rupture, dissection, and atherosclerosis-related thrombosis types. This indicates that there is a strong relationship between the pathological features of aneurysms and their clinical course. Critical control of blood pressure and determination of appropriate therapy by evaluating cerebral blood flow are necessary for giant fusiform aneurysms in the MCA, even if they are found incidentally.

**References**


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Address reprint requests to: Nobutaka Horie, M.D., Department of Neurosurgery, Nagasaki University School of Medicine, 1-7-1, Sakamoto-machi, Nagasaki-shi, Nagasaki, 852-8501, Japan. email: n-horie@net.nagasaki-u.ac.jp.