

# Non-Atherosclerotic Fusiform Cerebral Aneurysms

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**ABSTRACT: Background:** Fusiform cerebral aneurysms are dilatations of the entire circumference of a segment of cerebral artery, usually considered due to atherosclerosis in adults. They are relatively thick-walled and elongated, causing neural compression or ischemia when discovered. We have noted a subset of fusiform cerebral aneurysms that vary from this common description. **Patients:** Out of a series of 472 intracranial aneurysms treated over 11 years, 11 patients between the ages 16 and 67 years (mean age 37) were identified who had discrete fusiform aneurysms unassociated with generalized cerebral atherosclerosis, connective tissue disorder or inflammation. Three presented with hemorrhage, six with neural compression by the aneurysm and two were discovered incidentally. **Results:** Nine aneurysms were located in the posterior circulation, the other two in the intracranial carotid artery. Their mean length and width were 16.3 and 11 mm, respectively. Three aneurysms contained thrombus. The eight aneurysms that were exposed surgically were partly or substantially thin-walled with normal appearing parent arteries. Eight were treated with proximal occlusion and three were circumferentially “wrapped”. Parent artery occlusion caused one death and one mild disability and the remaining patients made good recoveries (follow-up 0.5 - 10 years). **Conclusions:** There is a subset of cerebral aneurysms with discrete fusiform morphology, apparently unrelated to cerebral atherosclerosis or systemic connective tissue disease, thin-walled in part or whole, more common in the vertebrobasilar system, and possessing a risk of rupture. Treatments currently available include proximal occlusion or aneurysm “wrapping”, different approaches than neck-clipping or endovascular coiling of side-wall saccular cerebral aneurysms that leave the parent artery intact.

**RÉSUMÉ: Anévrismes cérébraux fusiformes non athéroscléreux. Introduction:** Les anévrismes cérébraux fusiformes sont des dilatations de toute la circonférence d'un segment d'artère cérébrale, habituellement attribuées à l'athérosclérose chez les adultes, avec une paroi relativement épaisse et allongée, causant des compressions nerveuses ou de l'ischémie lors du diagnostic. Nous avons observé un sous-groupe d'anévrismes cérébraux fusiformes qui diffère de cette description générale. **Patients:** Dans une série de 472 anévrismes intracrâniens traités sur une période de 11 ans, 11 patients entre l'âge de 16 et 67 ans (âge moyen 37 ans) ayant des anévrismes fusiformes discrets non associés à une athérosclérose cérébrale généralisée, à des maladies du tissu conjonctif ou à une maladie inflammatoire, ont été identifiés. Trois patients ont présenté une hémorragie, six une compression neurologique due à l'anévrisme et deux ont été découverts fortuitement. **Résultats:** Neuf anévrismes étaient localisés au niveau de la circulation postérieure, les deux autres dans la carotide intracrânienne. Leur longueur et leur largeur moyennes étaient de 16.3 et 11 mm respectivement. Trois anévrismes contenaient un thrombus. Les huit anévrismes qui ont été explorés chirurgicalement étaient en tout ou en partie des anévrismes à paroi mince et l'artère qui y avait donné naissance était d'apparence normale. Huit ont été traités au moyen d'une occlusion proximale et trois ont été “enveloppés”. L'occlusion de l'artère mère a provoqué un décès et un cas d'invalidité légère; les autres patients ont bien récupéré (suivi de 0.5 à 10 ans). **Conclusions:** Certains anévrismes cérébraux ont une morphologie fusiforme discrète, ne sont apparemment pas en relation avec une athérosclérose cérébrale ou une maladie systémique du tissu conjonctif, ont une paroi mince en tout ou en partie, sont plus fréquents au niveau du système vertébrobasilaire et sont à risque de rupture. Les traitements disponibles présentement sont l'occlusion proximale ou l'enveloppement de l'anévrisme, des approches différentes de la pose d'un clip ou d'une prothèse endovasculaire au niveau des anévrismes cérébraux sacculaires dont l'artère mère est intacte.

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Fusiform intracranial aneurysms have been so often associated with atherosclerosis that the term “atherosclerotic aneurysm” has been used interchangeably.<sup>1-5</sup> “Dolichoectasia” has also been used to refer to fusiform aneurysms, alluding to their elongated (dolichos) and distended (ectasia) shape. Most commonly affecting the distal vertebral and basilar arteries in older patients, fusiform aneurysms are usually associated with neural compression or ischemia and, rarely, rupture.<sup>4-6</sup> Cerebral aneurysms with fusiform morphology have more rarely been associated with a number of systemic connective tissue diseases and arteritides.<sup>7</sup> Presumably any congenital or acquired frailty of

the cerebral artery wall can sometimes lead to fusiform dilatation of the vessel.

We have treated a number of patients with fusiform cerebral aneurysms who do not suffer from detectable atherosclerosis or

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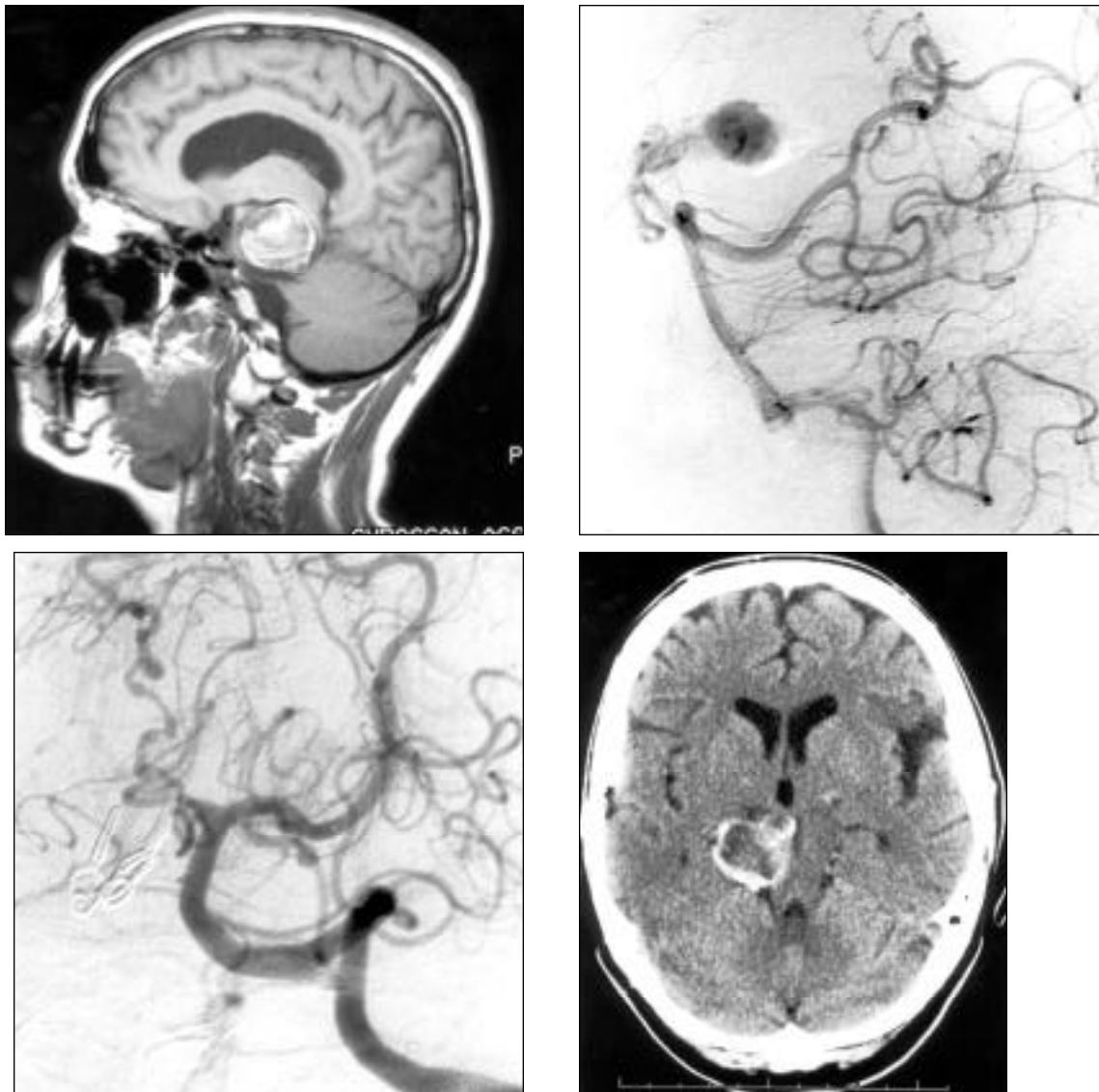
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any other systemic and/or heritable connective tissue or vascular disease. Like atherosclerotic dolichoectasias, these aneurysms are commonest in the vertebrobasilar circulation and frequently cause compressive symptoms but, unlike them, these aneurysms occur in younger patients, are relatively discrete and arise from otherwise normal arteries. The series of patients presented here suggests that not all aneurysms with fusiform morphology have a clear atherosclerotic etiology or are related to a detectable connective tissue disease.

#### PATIENTS

Out of a series of 472 intracranial aneurysms treated by a single neurovascular surgeon over 11 years, and not including six

additional patients with intracranial arterial dissections, 11 patients (2.3% of the total) between the ages of 16 and 67 (mean age 37) were identified who had discrete fusiform intracranial aneurysms which were unassociated with detectable cerebral atherosclerosis or a diagnosed hereditary or acquired connective tissue or inflammatory disorder, such as Marfan's syndrome, pseudoxanthoma elasticum, Ehlers-Danlos syndrome, neurofibromatosis, or any type of intracranial infectious or inflammatory condition. Specifically, cerebral atherosclerosis was considered very unlikely when the aneurysm appeared discrete and there was no evidence of either diffuse cerebrovascular disease or atherosclerosis elsewhere as determined by cerebral arterial imaging and/or by direct



**Figure 1:** A 46-year-old woman presented with a one-year history of spastic left-sided weakness and tremor (patient no. 3), due to a partially thrombosed right-sided proximal posterior cerebral artery aneurysm (upper left panel, sagittal T1 weighted MRI, and upper right, left vertebral angiogram showing partial and sluggish opacification of the aneurysm). Treated with proximal clip occlusion of both the right posterior cerebral artery and posterior communicating artery after a fronto-temporal and trans-sylvian exposure (lower left, left vertebral angiogram), the patient's clinical condition improved substantially over one year and the aneurysm was seen to involute on CT over the same interval (lower right, noncontrast CT scan one year following surgery).

**Table:** Summary of Patient Series

Patient name, age	Clinical Presentation	Aneurysm location, length & width (mm)	Intraluminal thrombus	Treatment	Outcome*
1. Mrs. L.K., 67 y.o.	SAH	left PCA, P1 and P2 segments 8 x 3.5 mm	no	saccular bleb clipped, remainder wrapped	Good, 10 years f/u
2. Mr. A.B., 16 y.o.	Painful right 3 & 5 cranial neuropathies	right cavernous ICA, 20 x 11 mm	no	EC-IC bypass, proximal ICA occlusion	Good, 6 years f/u
3. Mrs. S.T., 47 y.o.	Spastic left hemiparesis, tremor	right PCA, P1 and P2 segments, 30 x 22 mm	yes	proximal PCA clip occlusion	Good, 5 years f/u
4. Mr. V.M., 38 y.o.	left 9, 10 & 11 cranial neuropathies, facial numbness, vertigo	left intradural VA, 12 x 4 mm	no	wrapped	Good, 5 years f/u
5. Mrs. D.G., 18 y.o.	left 3 & 5 cranial neuropathies	left SCA, 10 x 6 mm	yes	endovascular coil occlusion (x 2)	Good, 3 years f/u
6. Rev. E.E., 49 y.o.	incidental	right intradural VA, 10 x 5 mm	no	proximal clip occlusion	Good, 2 years f/u
7. Mrs. L.G., 47 y.o.	recurrent SAH	left PCA, P2 segment, 3 x 5 mm	no	proximal and distal clip occlusion	Good, 2 years f/u
8. Mrs. A.S., 46 y.o.	right-sided weakness	left PCA, P2 segments, 30 x 20 mm	yes	proximal clip occlusion	Good, 20 months f/u
9. Mr. G.B., 24 y.o.	recurrent SAH	BA trunk, 30 x 25 mm	no	proximal clip occlusion	Died
10. Mr. D.V., 53 y.o.	right hemiparesis	VA-BA junction, 19 x 15 mm (both terminal VAs)	no	right VA balloon occlusion	Good, 1 year f/u
11. Mrs. M.J., 43 y.o.	incidental	left terminal ICA, 7 x 4 mm	no	wrapped	Good, 6 months f/u

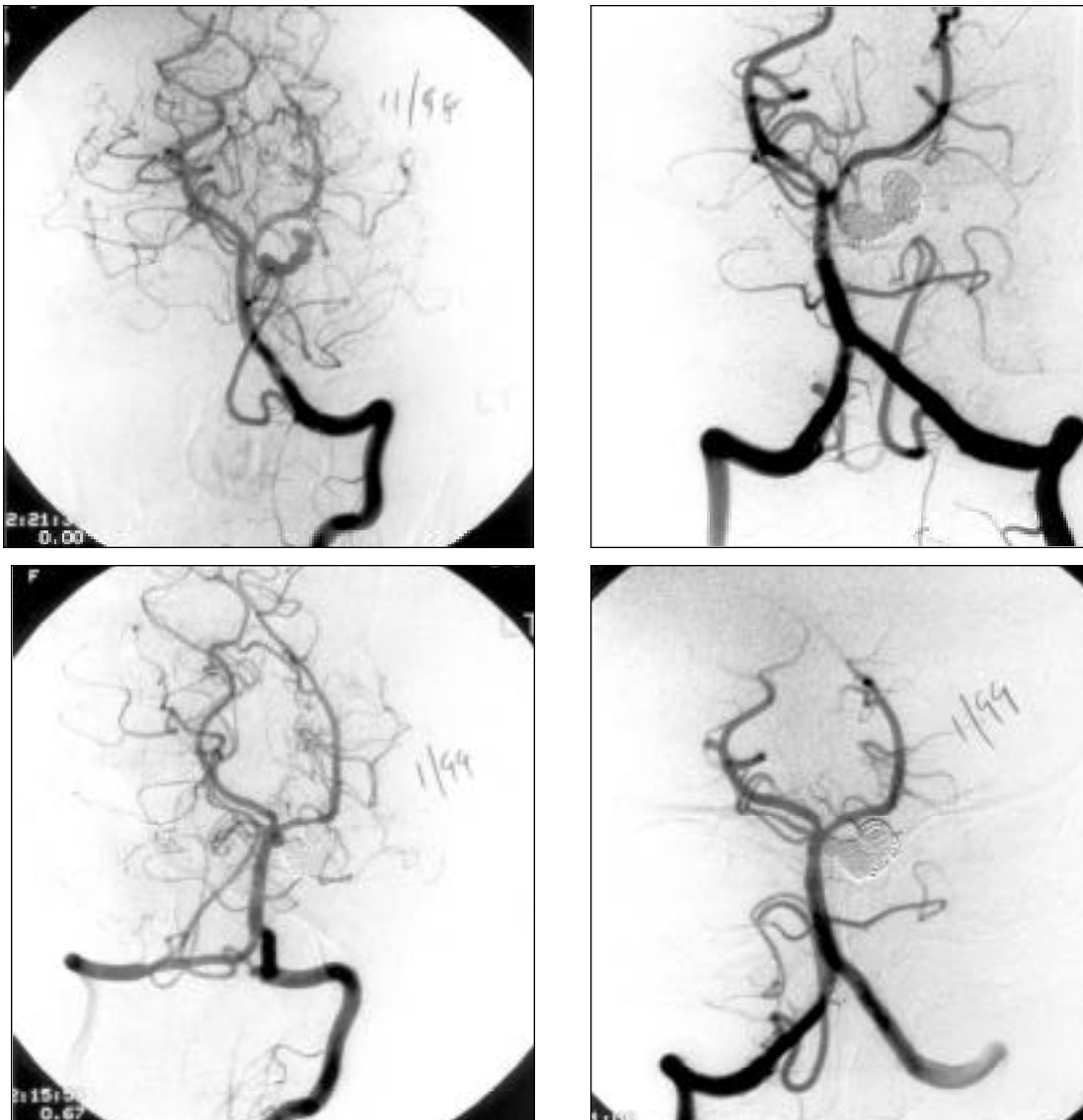
SAH = subarachnoid hemorrhage, ICA= internal carotid artery, VA= vertebral artery, BA=basilar artery, PCA=posterior cerebral artery, SCA=superior cerebellar artery, P1=precommunicating segment of the posterior cerebral artery, P2=post-communicating segment of the posterior cerebral artery to the quadrigeminal cistern,

\*Glasgow Outcome Scale<sup>39</sup>

inspection of the parent and adjacent arteries at the time of surgery. At the time of clinical presentation and in follow-up, connective tissue disorders were ruled out on the basis of standard clinical criteria.<sup>8</sup> None of the patients had detectable intracranial sepsis.

Three patients presented with subarachnoid hemorrhage, six with symptoms and signs of either cranial nerve or brain stem compression by the aneurysm and two aneurysms were incidental discoveries (Table). None had calcification of the aneurysm wall and three had intra-aneurysmal thrombus (laminated clot) prior to treatment (Figure 1). Seven of the 11

patients underwent preoperative magnetic resonance imaging (MRI) (patients nos 2, 3, 4, 6, 8, 10 and 11) and none had evidence of cerebral infarction related to the aneurysm. Angiographic assessment of every patient and direct surgical inspection of the eight patients who underwent surgery indicated normal parent arteries and normal arteries adjacent to the fusiform aneurysm. The mean length and width of the aneurysms was 16.3 mm and 11 mm, respectively. In recent years we have found three-dimensional computerized tomographic angiography (CTA) very useful in the anatomical characterization of these aneurysms.



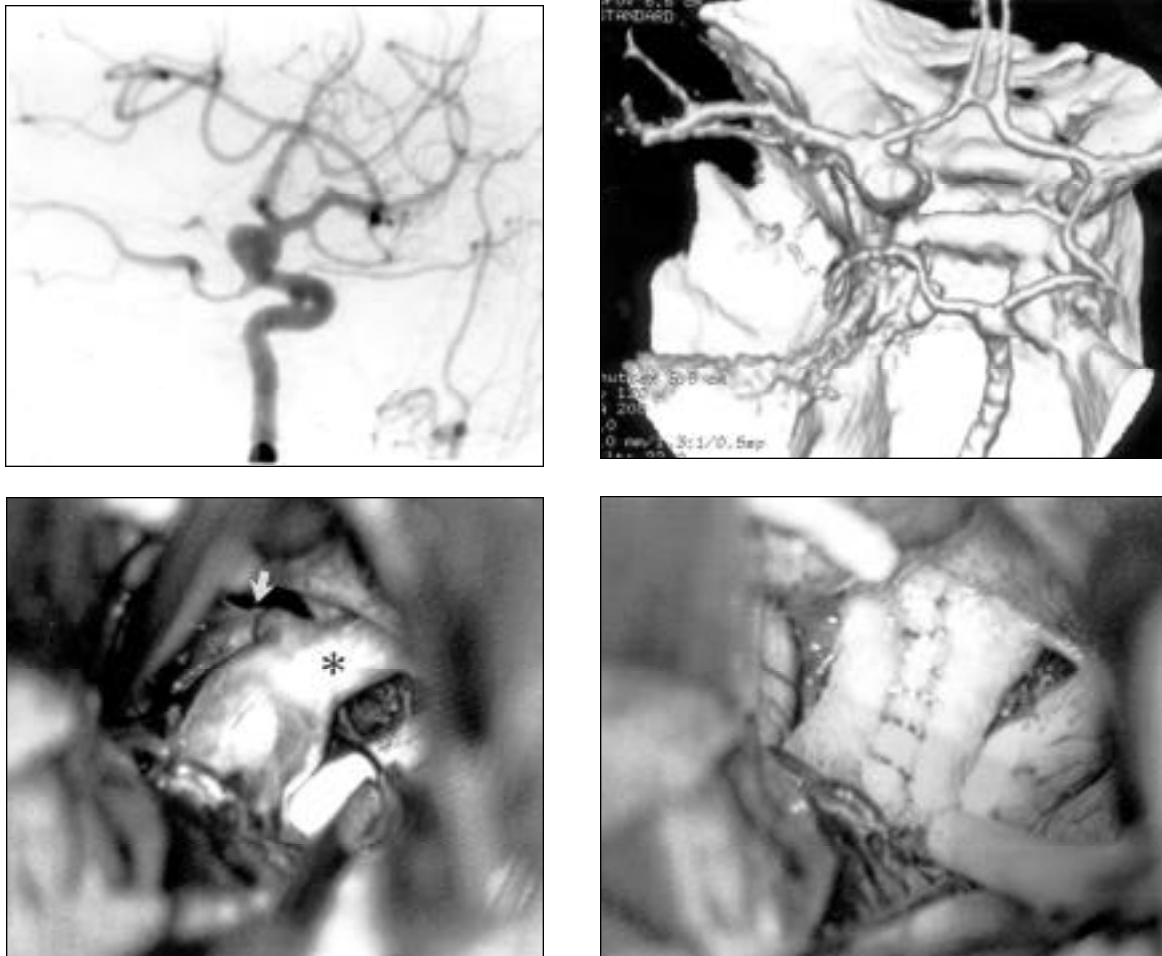
**Figure 2:** An 18-year-old and 36 week pregnant young woman (patient no. 5) presented with painful and partial left third and fifth nerve palsies, due to compression from a fusiform aneurysm of the left superior cerebellar artery (upper left panel, left vertebral artery angiogram). Outflow from the aneurysm into a distal vessel could not be readily appreciated and thrombus within the aneurysm was seen on CT (not shown). The aneurysm was occluded with a total of seven detachable coils (upper right), which did not result in cerebellar infarction. Two months later, following safe delivery of her child, repeat angiography showed aneurysmal expansion of the proximal parent artery (lower left), which was occluded with an additional two coils (lower right). Repeat angiography two years later has showed persistent complete aneurysm occlusion.

**TREATMENT AND OUTCOME**

All of the patients in this series were treated in some manner. Eight patients underwent proximal parent artery occlusion, five with microsurgical clipping (one of whom also underwent trapping of a small fusiform aneurysm [patient no. 7]), two with proximal balloon occlusion and one with intra-aneurysmal endovascular Guglielmi detachable (Target Therapeutics, Fremont, CA) coil embolization (Figure 2). Three aneurysms were circumferentially wrapped with cotton sheets that were held clipped or stapled around the fusiform aneurysm (Figure 3).

One of these patients (patient no.1) also had a saccular bleb arising from her posterior cerebral artery fusiform aneurysm (thought to have caused her subarachnoid hemorrhage) that was clipped directly prior to wrapping the entire fusiform segment of artery. One patient, no. 2, underwent a superficial temporal artery-middle cerebral artery bypass prior to balloon occlusion of a cavernous segment internal carotid artery aneurysm.

Outcome has been generally good. One patient with a giant basilar trunk aneurysm who underwent proximal basilar artery clip occlusion suffered brain stem infarction and died several days following surgery (patient no. 9) (Figure 4). Another with a large



**Figure 3:** A 43-year-old healthy woman (patient no. 11) was found incidentally in a chronic headache investigation to have a fusiform aneurysmal dilatation of her left internal carotid artery (ICA) between the origin of the posterior communicating artery and the terminus (upper left, left carotid angiogram, upper right CT angiogram of the circle of Willis from above). The fusiform expansion of the ICA (lower left, image taken from the operative video, \* = proximal ICA, arrow = posterior communicating artery) was circumferentially wrapped with a cotton sheet made by splitting a surgical cottonoid, and the ends were joined by microstaples (lower right, image taken from the operative video, stapler manufactured by AutoSuture Company, a division of the United States Surgical Corporation, Norwalk, CT).

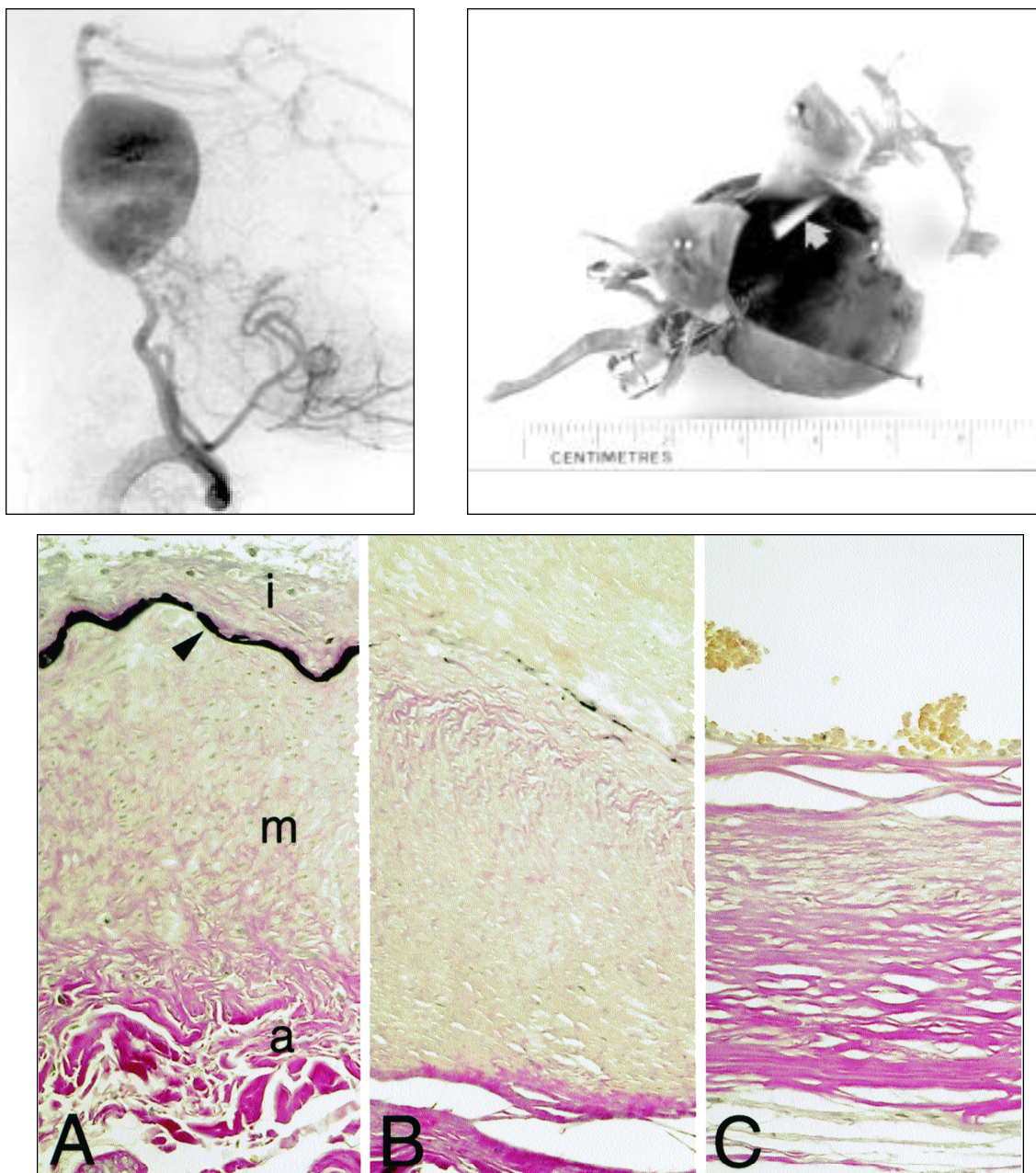
“pantaloon” aneurysm of the vertebrobasilar junction suffered a small medullary infarction following occlusion of the right vertebral artery, from which he made a good recovery (patient no. 10) (Figure 5). Follow-up between six months and ten years has not detected clinical aneurysm recurrence in any patient.

## DISCUSSION

Intracranial arterial aneurysms are classified and described both etiologically and morphologically and, for many aneurysms, there is a correlation between their cause and shape. The most common type of intracranial aneurysm, making up over 90% of the total, are “developmental” in origin, acquired rather than congenital lesions arising at arterial bifurcations, branch points or curves. These locations are subjected to particular hemodynamic stresses that over time lead to focal vessel wall degeneration and aneurysm formation.<sup>9,10</sup> Developmental aneurysms have a saccular morphology (hence the older term

“berry” aneurysms), can be of any size, and have an orifice and neck arising from an underlying normal parent vessel.

Fusiform intracranial aneurysms have been characterized as thickened, widened and elongated arteries due to atherosclerosis in an older population (geriatric men most commonly), occurring most often in the vertebrobasilar arteries where they can become quite large and compress cranial nerves or brain stem causing symptoms such as trigeminal neuralgia, hemifacial spasm, deafness, or sensorimotor dysfunction of the pharynx, larynx or limbs.<sup>4,11-14</sup> Intraluminal thrombosis and embolism can cause ischemia and infarction.<sup>4,5</sup> Histopathological examination of the walls of these aneurysms shows deficiencies in the elastic lamina, fibrotic replacement of the muscularis, calcium and lipid deposition, mural hemorrhage and infiltration with both acute and chronic inflammatory cells.<sup>6,15-17</sup> Although quite uncommon, cerebral arterial ectasia and fusiform intracranial aneurysms have also been reported to occur in younger persons with certain systemic connective tissue disorder, including Ehlers-Danlos

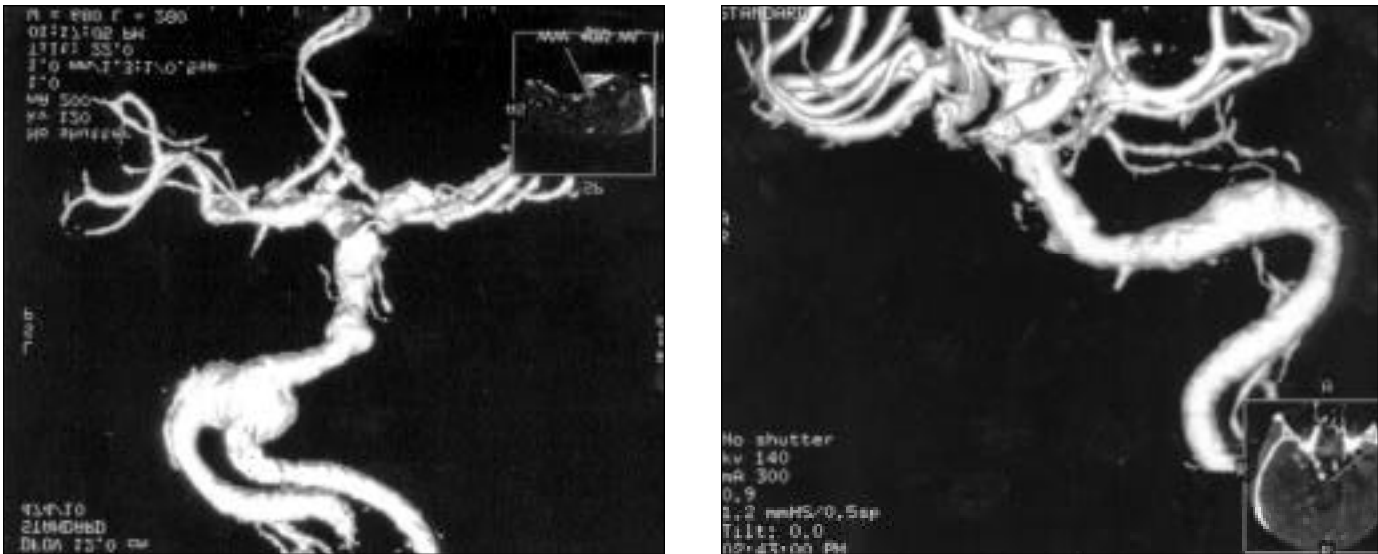


**Figure 4:** A 24-year-old healthy young man (patient no. 9) collapsed while motor-bike racing and, within 24 hours, suffered a recurrent subarachnoid hemorrhage from a large mid-basilar artery aneurysm (upper left panel, left vertebral angiogram). At surgery it was found that the aneurysm was fusiform so the proximal basilar artery was clip-occluded, since it was known that the patient had posterior communicating arteries to provide collateral flow to the upper brain stem. The aneurysm thrombosed following, causing brain stem infarction and the patient died several days later. Autopsy confirmed the fusiform nature of the aneurysm (upper right, showing the opened aneurysm, clips occluding the proximal basilar artery and a wooden stick [arrow] inserted into the distal basilar artery lumen). Pathological examination (lower panel, Verhoeff Van Gieson staining) showed the proximal parent basilar artery (A) with normal tunica intima (i), elastic lamina (arrow head), tunica media of smooth muscle (m) and tunica adventitia (a). The aneurysm neck begins to lose elastic lamina and tunica media (B), and the aneurysm wall shows a complete loss of these two elements (C), replaced by degenerative fibrous tissue. No changes consistent with atherosclerosis were found.

syndrome, Marfan's syndrome, pseudoxanthoma elasticum, tuberous sclerosis and neurofibromatosis.<sup>18,19</sup>

Intracranial arterial dissections can result in stenosis, occlusion, double-lumens and also fusiform arterial dilatations causing subarachnoid hemorrhage when the adventitial layer is

breached. The possibility that some of the aneurysms described in this report were intracranial dissecting aneurysms must be considered, especially since intracranial dissections more commonly occur in the vertebral and basilar arteries.<sup>20-22</sup> The majority of patients with intracranial arterial dissections (and all



**Figure 5:** A 53-year-old healthy man (patient no. 10) presented with a one-day history of right hemiparesis. Imaging revealed a fusiform dilatation of the vertebral basilar junction (a “pantaloons” aneurysm) (left, CTangiogram) that appeared to be compressing the left pyramid on magnetic resonance imaging (there was no evidence of brain stem ischemia). Treated with right vertebral artery balloon occlusion, the patient experienced a new left hemiparesis (due to a small medullary infarct) that improved over several months. Subsequent CT angiography showed partial aneurysm thrombosis resulting in a tortuous but more normal appearing left vertebral artery (right). The patient has been able to return to work with minimal disability.

of the 65 patients with typical dissections in Mizutani et al’s report<sup>21</sup>) present with subarachnoid hemorrhage and imaging demonstrates irregular luminal stenosis at either end of a fusiform enlargement, often with a tell-tale intimal flap. Among the three patients in our series who presented with subarachnoid hemorrhage, one died and pathological examination of the basilar aneurysm ruled out a dissection, and the radiological appearances of the other two posterior cerebral artery aneurysms that ruptured were not typical of dissection (as described above). Nevertheless, in the absence of neuropathological proof, it remains possible that these and perhaps other fusiform aneurysms in our series were atypical intracranial arterial dissections.

There are a number of other more rare and special intracranial aneurysms, including traumatic,<sup>23</sup> neoplastic,<sup>24,25</sup> inflammatory,<sup>26,27</sup> infectious<sup>28-31</sup> and radiation induced types,<sup>32</sup> where focal arterial wall injury, invasion or destruction leads to aneurysmal dilatation that is usually irregular and sometimes fusiform in morphology. However, these types of aneurysms are quite unlike the fusiform aneurysms described in this report, arising usually in peripheral arteries, normally only several millimeters in size and, because of their distal location, causing parenchymal hemorrhage when they rupture.

The patients presented in this report are unique in that their aneurysms had distinct fusiform morphologies but there was no evidence of adjacent or widespread cerebral atherosclerosis, nor was there any clinical evidence of connective tissue disorder, localized infection or vessel wall inflammation. Also inconsistent with an atherosclerotic etiology was the young mean age of our patients (37 years) as well as the absence of clinical or radiological evidence of cerebral ischemia associated with the aneurysms. It needs to be acknowledged, however, that without

histopathological proof obtained at autopsy (available for only one patient in our series) we cannot absolutely rule out either atherosclerosis or occult systemic connective tissue disease. Long-term follow up of several patients suggests that once treated, the underlying process does not spread to adjacent vessels. It is interesting that the aneurysms in this series showed a proclivity for the posterior circulation, and the posterior cerebral artery in particular, which is similar to the more common atherosclerotic fusiform aneurysms. Similar findings to these were recently reported by Drake and Peerless.<sup>33</sup> Although the 120 fusiform intracranial aneurysms they treated surgically were all giant (> 2 cm) in size, they found only three due to an identifiable atheriopathy and only six in patients with atherosclerosis. In the remaining 111 patients the etiology of their giant fusiform aneurysms could not be determined. In these much younger patients, with an almost even sex ratio, aneurysms predominated in the basilar trunk and posterior cerebral artery (90% affected the posterior circulation), and 20% bled, findings similar to our own series of smaller aneurysms. Idiopathic fusiform aneurysms affecting the middle cerebral artery were also described by those authors and have been reported by others,<sup>34-36</sup> although we have not yet encountered one.

The morphology of fusiform aneurysms requires that treatment, when indicated by either the presence of symptoms (nine of the 11 patients in this series), or the predicted long-term risk of growth and possible rupture (two of the 11 patients in this series) consist of either proximal occlusion, proximal and distal occlusion (“trapping”), or circumferential wrapping with or without clip reinforcement to stabilize and strengthen the aneurysm wall.<sup>37,38</sup> When Hunterian proximal occlusion or trapping are being considered, success is dependent upon adequate collateral circulation to both the distal vessels as well

as any branches or perforator vessels arising from the fusiform aneurysm itself.<sup>33</sup> That collateral circulation may arise from the circle of Willis, bypass vessels created surgically, or natural leptomeningeal or deep arteriolar collateral flow. Treatment must be individualized to the patient, but basilar trunk aneurysms present the greatest difficulties while posterior cerebral artery fusiform aneurysms are often simply and effectively managed by proximal posterior cerebral artery occlusion, since this artery usually has good collateral blood supply.

The underlying cause of the type of fusiform aneurysms described in this report is unknown at this time but appears to bear no clear relationship to cerebral atherosclerosis. The principle aim of this report is to emphasize that fusiform and atherosclerotic aneurysms are terms that should not be used interchangeably. Fusiform atherosclerotic aneurysms should be distinguished from those that are fusiform in shape but idiopathic in etiology.

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#### REFERENCES

- Boeri R, Passerini A. The megadolichobasilar anomaly. *J Neurol Sci* 1964;1:476-484.
- Sacks J, Linderberg R. Dolichoectatic intracranial aneurysms. *Johns Hopkins Med J* 1969;125:95-106.
- Nishizaki T, Tamaki N, Takeda N, et al. Dolichoectatic basilar artery: a review of 23 cases. *Stroke* 1986;17:1277-1281.
- Echiverri HC, Rubino FA, Gupta SR, Gujrati M. Fusiform aneurysm of the vertebrobasilar arterial system. *Stroke* 1989;20:1741-1747.
- Pessin MS, Chimowitz MI, Levine SR, et al. Stroke in patients with fusiform vertebrobasilar aneurysms. *Neurology* 1989;39:16-21.
- Shokunbi MT, Vinters HV, Kaufmann JCE. Fusiform intracranial aneurysms. *Surg Neurol* 1988;29:263-270.
- Weir BKA. Special Aneurysms (nonsaccular and saccular). In: Brown CL, Eckhart C, eds. *Aneurysms Affecting the Nervous System*: Williams & Wilkins, 1987:134-208.
- Schievink WI, Michels VV, Piepgras DG. Neurovascular manifestations of heritable connective tissue disorders. A review. *Stroke* 1994;25:889-903.
- Stehbens WE. Etiology of intracranial berry aneurysms. *J Neurosurg* 1989;70:823-831.
- Ferguson GF. Physical factors in the initiation, growth, and rupture of human intracranial saccular aneurysms. *J Neurosurg* 1972;37:666-677.
- Hayes W, Bernhardt H, Young J. Fusiform arteriosclerotic aneurysm of the basilar artery. *Vasc Surg* 1967;1:171-178.
- Deeb Z, Jannetta P, Rosebaum A, Kerber C, Drayer B. Tortuous vertebrobasilar arteries causing cranial nerve syndromes: screening by computed tomography. *J Comp Ass Tomog* 1979;3:774-778.
- Waga S, Morikawa A, Kojima T. Trigeminal neuralgia: compression of the trigeminal nerve by an elongated and dilated basilar artery. *Surg Neurol* 1979;11:13-15.
- Gibson W, Wallace D. Basilar artery ectasia. An unusual cause of a cerebello-pontine lesion and hemifacial spasm. *J Laryng Otol* 1975;89:721-731.
- Nakatomi H, Segawa H, Kurata A, et al. Clinicopathological study of intracranial fusiform and dolichoectatic aneurysms. *Stroke* 2000;31:896-900.
- Vinters HV. Pathology of cervical and intracranial atherosclerosis and fibromuscular dysplasia. In: Hunt Batjer H, ed. *Cerebrovascular Disease*. Philadelphia: Lippincott-Raven, 1997:41-51.
- Vale F, Hadley MN. Pathology of intracranial aneurysms and vascular malformations. In: H. Hunt Batjer, ed. *Cerebrovascular Disease*. Philadelphia: Lippincott-Raven, 1997:65-77.
- Muhonen MG, Godersky JC, VanGilder JC. Cerebral aneurysms associated with neurofibromatosis. *Surg Neurol* 1991;36:470-475.
- Guha A, Montanera W, Hoffman HJ. Congenital aneurysmal dilatation of the petrous-cavernous carotid artery and vertebral basilar junction in a child. *Neurosurgery* 1990;26:322-327.
- Sasaki O, Ogawa H, Koike T, Koizumi T, Tanaka R. A clinicopathological study of dissecting aneurysms of the intracranial vertebral artery. *J Neurosurg* 1991;75:874-882.
- Mizutani T, Miki Y, Kojima H, Suzuki H. Proposed classification of non-atherosclerotic cerebral fusiform and dissecting aneurysms. *Neurosurgery* 1999;45:253-259.
- Mizutani T, Kojima H. Clinicopathological features of non-atherosclerotic cerebral arterial trunk aneurysms. *Neuropathology* 2000;20:91-97.
- Haddad FS, Haddad GF, Taha J. Traumatic intracranial aneurysms caused by missiles: their presentation and management. *Neurosurgery* 1991;28:107.
- Murata J, Sawamura Y, Takahashi A, Abe H, Saitoh H. Intracerebral hemorrhage caused by a neoplastic aneurysm from small-cell lung carcinoma: case report. *Neurosurgery* 1993;1:124-126.
- Furuya K, Sasaki T, Yoshimoto Y, Kirinio T. Histologically verified cerebral aneurysm formation secondary to embolism from cardiac myxoma. *J Neurosurg* 1995;83:170-173.
- Hashimoto N, Handa H, Taki W. Ruptured cerebral aneurysms in patients with systemic lupus erythematosus. *Surg Neurol* 1986; 26:512-516.
- Sasaki T, Morimoto T, Utsumi S. Cerebral transmural angiitis and ruptured cerebral aneurysms in patients with systemic lupus erythematosus. *Neurochir* 1990;33:132-135.
- Venger BH, Aldama AE. Mycotic vasculitis with repeated intracranial aneurysmal hemorrhage. *J Neurosurg* 1988;69:775-779.
- Brust JCM, Dickinson PCT, Hughes JEO, Holtzman RNN. The diagnosis and treatment of cerebral mycotic aneurysms. *Ann Neurol* 1990;27:238-246.
- Kurino M, Kuratsu J, Yamaguchi T, Ushio Y. Mycotic aneurysm accompanied by aspergillotic granuloma: a case report. *Surg Neurol* 1994;42:160-164.
- Barami K, Ko K. Ruptured mycotic aneurysm presenting as an intraparenchymal hemorrhage and nonadjacent acute subdural hematoma: case report and review of the literature. *Surg Neurol* 1994;41:290-293.
- Scodary DJ, Tew JM Jr, Thomas GM, Tomsick TH, Liwnicz BH. Radiation-induced cerebral aneurysms. *Acta Neurochir* 1990;102:141-144.
- Drake CG, Peerless SJ. Giant fusiform intracranial aneurysms: review of 120 patients treated surgically from 1965 to 1992. *J Neurosurg* 1997;87:141-162.
- Kumabe T, Kaneko U, Ishibashi T, Kaneko K, Uchigasaki S. Two cases of giant serpentine aneurysm. *Neurosurgery* 1990;26: 1027-1033.
- Isla A, Alvarez F, Roda JM, et al. Serpentine aneurysm: regrowth after a superficial temporal artery-middle cerebral artery bypass and internal carotid artery ligation. *Neurosurgery* 1994;34:1072-1074.
- Horowitz MB, Yonas H, Jungreis C, Hung TK. Management of a giant middle cerebral artery fusiform serpentine aneurysm with distal clip application and retrograde thrombolysis: case report and review of the literature. *Surg Neurol* 1994;41:221-225.
- Bederson JB, Zabramski JM, Spetzler RF. Treatment of fusiform intracranial aneurysms by circumferential wrapping with clip reinforcement. Technical note. *J Neurosurg* 1992;77:478-480.
- Anson JA, Lawton MT, Spetzler RF. Characteristics and surgical treatment of dolichoectatic and fusiform aneurysms. *J Neurosurg* 1996;84:185-193.
- Jennett B, Bond M. Assessment of outcome after severe brain damage. A practical scale. *Lancet* 1975;1:480-484.