Giant Fusiform Aneurysm of the Cerebral Arteries

JOHN R. LITTLE, M.D., PHILLIP ST. LOUIS, M.D., MEREDITH WEINSTEIN, M.D., AND DONALD F. DOHN, M.D.

SUMMARY Giant fusiform aneurysm of the cerebral arteries was found in 11 patients during a 20-year period. The 7 males and 4 females ranged in age from 9 to 68 years (mean: 49 years). The supraclinoid segment of the internal carotid artery (ICA) and the M-1 segment of the middle cerebral artery (MCA) were the most frequently involved arteries. Multiple aneurysms were identified in 3 patients. Compression of adjacent intracranial structures was the usual cause of symptoms, and only 2 patients experienced subarachnoid hemorrhage. One patient presented with transient ischemic attacks. Computed tomography, with and without Hynaque infusion, clearly demonstrated the aneurysms in the 6 of 7 patients studied. Thrombus was invariably seen in the lumen of the aneurysm. Cerebral angiography in 11 patients displayed marked dilatation and elongation of the involved artery. The dilatation frequently extended into connecting arteries. Surgical treatment was carried out in 6 patients, including 2 with aneurysm entrapment and decompression, 2 with proximal ICA ligation, 1 with wrapping and 1 with wrapping and superficial temporal artery (STA) to MCA anastomosis. Death occurred in 2 patients not treated surgically.

Stroke, Vol 12, No 2, 1981

GIANT FUSIFORM ANEURYSM of the basilar artery is a well-recognized clinical and pathological entity. Information regarding giant fusiform aneurysm of the cerebral arteries, however, is limited and derived from a relatively small number of reports. The object of this investigation was to study the clinical presentation, diagnosis, and treatment of patients with this unusual arterial abnormality.

Analysis of Patients

Giant fusiform aneurysm of the cerebral arteries was defined as a marked concentric dilatation, 2.5 cm or more in length, involving the intracranial internal carotid artery (ICA) and/or the anterior (ACA), middle (MCA), or posterior (PCA) cerebral arteries. Eleven patients with this arterial abnormality were seen during a 20-year period from 1959 to 1979. The diagnosis was made by cerebral angiography in all patients. Multiple giant aneurysms were found in 3.

Clinical Presentation. The 7 male and 4 female patients ranged in age from 9 to 68 years (mean: 49 years). Presenting symptoms were relatively diverse and without any clear-cut pattern (table 1). Only 2 patients (Nos. 1 and 8) had subarachnoid hemorrhage. The duration of symptoms ranged from one day to 15 years (mean: 2.5 years). Hypertension was a pre-existing condition in 5 patients (Nos. 5, 6, 9, 10 and 11). One patient (No. 4) had unilateral optic atrophy and bilateral peripapillary chorioretinitis. The chorioretinitis was thought to be the result of toxoplasmosis. Another patient (No. 11) had multiple aneurysms of the superficial temporal arteries and right radial artery which were palpated on general examination. Four patients previously he had been found to have an autoimmune hemolytic anemia.

Investigations

Skull x-rays. Skull x-rays were normal in 9 patients. Suprasellar calcification and erosion of the dorsum sella in the 9-year-old patient with an ICA aneurysm was initially thought to represent a craniopharyngioma. Another patient had erosion of the anterior clinoid process on the same side as the ICA aneurysm.

Computed Tomography. Seven plain CT scans and 7 with Hynaque infusion were performed during the initial investigation of 7 patients. The findings of these studies are listed in table 2 (figure). A follow up CT scan in a medically-treated patient (No. 4) 3 years after diagnosis was unchanged from the initial study.

Cerebral Angiography. The findings of the initial studies are listed in table 3. There were no patients with angiographic findings of fibromuscular dysplasia or coarctation of the aorta.

Follow up angiography in one patient (No. 3) a year following ICA ligation for a giant fusiform aneurysm of the ICA showed substantial reduction in size of the aneurysm.

Other Studies. Five patients had a lumbar puncture. The cerebrospinal fluid was bloody in two (Nos. 1 and 8) with subarachnoid hemorrhage. No abnormalities were found in the other 3.

A high titer of toxoplasma antibodies was demonstrated in one patient (No. 4) who had presented with chorioretinitis and optic nerve atrophy. Serologic testing for syphilis in 7 patients was negative. The erythrocyte sedimentation rate was markedly elevated in 1 patient (No. 11).

Treatment and Outcome. Six patients had surgical treatment. Two (Nos. 1 and 3) had ICA ligation only. One of them (No. 3) is alive and well 13 years following surgery. The other (1) has recurrent headache.

One patient (No. 5) underwent craniotomy and trapping of a giant fusiform aneurysm of the ICA. The aneurysm was opened and thrombotic material removed. The fifth and sixth nerve deficits cleared.

From the Section of Cerebrovascular Diseases and the Departments of Neurosurgery and Neuroradiology (Dr. Weinstein), Cleveland Clinic Foundation, Cleveland, OH.
TABLE 1. Clinical Presentation in 11 Patients with Giant Fusiform Aneurysm of the Cerebral Arteries.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Symptoms</th>
<th>Neurological examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>20</td>
<td>M</td>
<td>Sudden, severe headache</td>
<td>Stiff neck</td>
</tr>
<tr>
<td>2.</td>
<td>32</td>
<td>M</td>
<td>Severe recurrent headache; R. face pain; loss of vision</td>
<td>L. homonymous hemianopsia</td>
</tr>
<tr>
<td>3.</td>
<td>50</td>
<td>F</td>
<td>Loss of vision, headache</td>
<td>R. homonymous hemianopsia; R. hyperreflexia</td>
</tr>
<tr>
<td>4.</td>
<td>9</td>
<td>F</td>
<td>Loss of vision; headache</td>
<td>R. optic atrophy; R. peripapillary chorioretinitis</td>
</tr>
<tr>
<td>5.</td>
<td>65</td>
<td>M</td>
<td>Double vision; headache</td>
<td>L. 6th nerve palsy; hypalgesia L. trigeminal nerve (V1) distribution</td>
</tr>
<tr>
<td>6.</td>
<td>63</td>
<td>M</td>
<td>Severe, recurrent headache; unsteady gait</td>
<td>Slight spastic weakness of L. lower extremity; impaired upward gaze</td>
</tr>
<tr>
<td>7.</td>
<td>58</td>
<td>M</td>
<td>R. cerebral transient ischemic attacks; headache</td>
<td>Normal</td>
</tr>
<tr>
<td>8.</td>
<td>49</td>
<td>M</td>
<td>Sudden, severe headache</td>
<td>Stiff neck; drowsy</td>
</tr>
<tr>
<td>9.</td>
<td>65</td>
<td>F</td>
<td>Loss of memory; unsteady gait; headache</td>
<td>Demented; spastic gait</td>
</tr>
<tr>
<td>10.</td>
<td>68</td>
<td>F</td>
<td>Loss of memory; unsteady gait; headache</td>
<td>Demented; spastic gait</td>
</tr>
<tr>
<td>11.</td>
<td>65</td>
<td>M</td>
<td>Double vision; headache; pulsatile swellings in forehead and R. wrist</td>
<td>L. 6th nerve palsy</td>
</tr>
</tbody>
</table>

L = left; R = right.

postoperatively. He remains asymptomatic 20 years later.

One patient, (No. 2) who presented with severe headache and a left homonymous hemianopsia, had a craniotomy and wrapping of a giant fusiform aneurysm of the right PCA. Following surgery, there was dramatic relief of severe right-sided headache and face pain and the CT scan showed thrombosis of the aneurysm. Six months later he returned with slowly progressive left hemiparesis. Recanalization and enlargement of the aneurysm were identified on CT scanning. He had a second craniotomy with aneurysm excision. After one year, he was functional but had a mild residual left spastic hemiparesis and a left homonymous hemianopsia. A second patient, (No. 8), did well without further symptoms following craniotomy and wrapping of the aneurysm.

A right superficial temporal artery (STA) to MCA anastomosis and wrapping of a giant fusiform aneurysm of the right MCA were performed in patient No. 7 presenting with right cerebral transient ischemic attacks. The anastomosis was carried out because of stenosis of the right MCA trunk distal to the aneurysm. Following surgery, the patient developed a severe left hemiparesis. CT scan with Hypaque infusion revealed a deep right cerebral infarct and thrombosis of the aneurysm.

Five patients were not treated surgically. A 65-year-

TABLE 2. CT Scan Findings in 7 Patients with Giant Fusiform Aneurysm of the Cerebral Arteries.

<table>
<thead>
<tr>
<th>Patient</th>
<th>No. of Studies</th>
<th>Plain CT scan</th>
<th>CT scan with Hypaque infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>2</td>
<td>Isodense L. anterior temporal mass; recent intracerebral hematoma; edema of adjacent brain</td>
<td>5 cm fusiform aneurysm of L. MCA; partly thrombosed lumen</td>
</tr>
<tr>
<td>2.</td>
<td>2</td>
<td>Isodense R. mid-temporal mass; compression R. cerebral peduncle</td>
<td>4 cm fusiform aneurysm of R. PCA with partly thrombosed lumen</td>
</tr>
<tr>
<td>4.</td>
<td>2</td>
<td>1 cm calcified lesion in L. parasellar region</td>
<td>4 cm fusiform aneurysm of L. ICA extending into L. MCA</td>
</tr>
<tr>
<td>6.</td>
<td>2</td>
<td>Round, hyperdense L. parasellar mass</td>
<td>5 cm fusiform aneurysm of L. ICA with partly thrombosed lumen; 3 cm fusiform R. ICA</td>
</tr>
<tr>
<td>7.</td>
<td>2</td>
<td>Isodense L. anterior temporal mass</td>
<td>4 cm fusiform aneurysm of L. MCA; partly thrombosed lumen</td>
</tr>
<tr>
<td>10.</td>
<td>2</td>
<td>Isodense R. parasellar and preptoneal masses; moderate hydrocephalus</td>
<td>3 cm fusiform aneurysm of R. ICA; 5 cm fusiform aneurysm of BA, extending into L. cerebellopontine angle</td>
</tr>
<tr>
<td>11.</td>
<td>2</td>
<td>Normal</td>
<td>1.5 cm saccular aneurysm of R. MCA</td>
</tr>
</tbody>
</table>

ICA = internal carotid artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; BA = basilar artery; L = left; R = right.
Radiologic studies in Patient No. 6. A) Plain CT scan shows a circular area of increased density in the left parasellar region. As well, there is slight increased density along the course of the right MCA. B) CT scan with Hypaque infusion provides a cross sectional view of the giant fusiform aneurysm of the left ICA. The lumen is partly filled with clot. The right ICA-MCA aneurysm also can be seen. C) CT scan with Hypaque infusion at a higher level shows the upper limit of the left ICA aneurysm. The anterior horn of the left lateral ventricle appears compressed. D) Anteroposterior view of the left carotid angiogram showing the giant fusiform aneurysm of the left ICA. The proximal segments of the left ACA and MCA are dilated.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Angiographic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5 cm fusiform aneurysm of M-1 segment of L. MCA extending into major MCA branches, proximal ACA, and distal ICA; stretching and narrowing of supraclinoid L. ICA</td>
</tr>
<tr>
<td>2</td>
<td>4 cm fusiform aneurysm of R. PCA extending into major PCA branches; slow filling of major PCA branches</td>
</tr>
<tr>
<td>3</td>
<td>4 cm fusiform aneurysm of supraclinoid L. ICA</td>
</tr>
<tr>
<td>4</td>
<td>4 cm tortuous fusiform aneurysm of supraclinoid L. ICA extending into proximal ACA and MCA; diffuse dilatation of intrapetrous and intracavernous L. ICA; abnormal small vessel collection in R. basal ganglia</td>
</tr>
<tr>
<td>5</td>
<td>6 cm fusiform aneurysm of infraclinoid and supraclinoid L. ICA</td>
</tr>
<tr>
<td>6</td>
<td>5 cm fusiform aneurysm of supraclinoid L. ICA extending into proximal ACA and PCA; R. side not studied</td>
</tr>
<tr>
<td>7</td>
<td>4 cm fusiform aneurysm of M-1 segment of L. MCA; distal L. MCA stenosis</td>
</tr>
<tr>
<td>8</td>
<td>3 cm fusiform aneurysm of anterior communicating artery extending into A-1 segment of both ACA’s</td>
</tr>
<tr>
<td>9</td>
<td>4 cm fusiform aneurysm of supraclinoid R. ICA</td>
</tr>
<tr>
<td>10</td>
<td>3 cm fusiform aneurysm of supraclinoid R. ICA extending into proximal ACA and MCA; 5 cm tortuous fusiform aneurysm of BA</td>
</tr>
<tr>
<td>11</td>
<td>3 cm fusiform aneurysm of intracavernous segment of both ICA’s; 1.5 cm saccular aneurysm of R. MCA; multiple fusiform aneurysms of both STA’s and R. internal maxillary artery</td>
</tr>
</tbody>
</table>

ICA = internal carotid artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; ACA = anterior cerebral artery; STA = superficial temporal artery; BA = basilar artery; L = left; R = right.
Giant fusiform aneurysm in some patients may be associated with a saccular aneurysm. In the presented patients, this association was found in 4 patients. In each case, a thick-walled aneurysm was exposed at surgery in 4 patients. A 2.5 cm intracerebral hematoma was also found in one patient. 

**Pathological Findings.** The giant fusiform aneurysm was exposed at surgery in 4 patients. In each case, a thick-walled aneurysm was found to compress the adjacent neural and vascular structures. A 2.5 cm intracerebral hematoma was also found in one patient. 

**Discussion**

Giant aneurysms constitute approximately 5 percent of all intracranial aneurysms. Most of these are the saccular variety. In recent series of giant aneurysm reported by Sundt and associates, Onuma and Suzuki, and Hosobuchi, only one case of giant fusiform aneurysm of the cerebral arteries was described. In our patients, 2 giant fusiform aneurysms were found. In another patient, 2 giant fusiform aneurysms were present. Giant fusiform aneurysm in some patients may represent a unique form of arteriopathy. Pathological examination of the aneurysm wall in younger patients consistently has demonstrated extensive deficiencies in the muscularis and internal elastic lamina compared with a more focal defect seen in saccular aneurysm.

Findings such as irregularity in the thickness of the media, abrupt gaps in the internal elastic lamina, occasional foci of muscularis, thickened fibrous tissue, hypertrophic and swollen connective tissue, and absence of intimas, have been described. Severe atherosclerosis, with or without associated hypertension, seen in those aneurysms occurring in the older age group may represent the primary mechanism of dilatation. However, atherosclerosis in some of them may represent a secondary change in an already abnormal arterial segment.

Giant fusiform aneurysm also has been reported in patients with Marfan's syndrome, pseudoxanthoma elasticum, syphilis, and coarctation of the aorta. Giant cell arteritis, probably on an autoimmune basis, appeared to be an etiologic factor in one of our patients. No instances have been reported in conjunction with toxoplasmosis. The relationship, if any, of this infection and giant fusiform aneurysm in our patient is unclear.

Compression of adjacent intracranial structures is the usual cause of symptoms and neurologic deficit in patients with a giant fusiform aneurysm of the cerebral arteries. Subarachnoid or intracerebral hemorrhage, cerebral ischemia, and impairment of CSF circulation also occur but are less common. In contrast, subarachnoid hemorrhage appears to be more common with giant saccular aneurysm.

Headache is the most common complaint of patients with a giant fusiform aneurysm of the cerebral arteries. The pain is usually located on the same side of the head as the aneurysm and occasionally is so intense as to result in complete incapacitation. In our series, recurrent headache was present in 9 of 11 patients. The other 2 patients experienced sudden, severe headache from subarachnoid hemorrhage.

Headache is thought to be the result of pressure of the aneurysm on adjacent pain-sensitive structures. In one of our patients (No. 2), dramatic relief of severe, recurrent headache and right face pain occurred after cottonoid gauze was wrapped around a giant fusiform aneurysm of the right PCA, thereby separating it from the floor of the middle fossa. Headache also was improved in another patient after trapping and decompression of an ICA aneurysm. ICA ligation only relieved the headache in one patient with an ICA aneurysm but did not prevent the development of headache in another patient with an MCA aneurysm.

Optic nerve compression with visual loss and optic atrophy has been reported frequently in patients with giant fusiform aneurysm of the ICA. In some, an altitudinal field defect has been identified. In one of our patients, 2 giant fusiform aneurysms were found. In another patient, 2 giant fusiform aneurysms and a saccular aneurysm were present. Giant fusiform aneurysm in some patients may represent a unique form of arteriopathy. Pathological examination of the aneurysm wall in younger patients consistently has demonstrated extensive deficiencies in the muscularis and internal elastic lamina compared with a more focal defect seen in saccular aneurysm.
with giant fusiform aneurysm of the cerebral arteries. Hilton and Hoyt\(^9\) described a patient with bitemporal hemianopia suggestive of a tumor arising in the sella turcica. An atherosclerotic fusiform aneurysm of the ACA was subsequently identified. In our series, 2 patients were found to have homonymous hemianopia, including one with a PCA aneurysm, and another with a tortuous ICA aneurysm. Two patients with an ICA aneurysm had horizontal diplopia which was the result of sixth nerve compression in the cavernous sinus by the infraclinoid component of the aneurysm.

Cerebral ischemia is occasionally seen in patients with a giant aneurysm, either saccular or fusiform.\(^8\)-\(^10\) Embolization from the thrombus contained within the aneurysm appears to be an important mechanism, although thrombosis with impaired flow through the malformation also may play a role. In our series, transient cerebral ischemic attacks seen in one patient appeared to be the result of severe MCA stenosis distal to an atherosclerotic MCA aneurysm. Embolization from the thrombus-containing aneurysm or transient occlusion of perforating arteries, however, could not be ruled out.

Blood vessels of the brain can be visualized with CT scanning if their walls contain calcium or if their lumens contain contrast material. The plain CT appearance of giant fusiform aneurysm may be difficult to differentiate from an intracranial neoplasm. In the investigation of a patient suspected of having a giant fusiform aneurysm, a CT scan should be performed as soon as possible after the injection of contrast material to obtain maximum visualization. The enhanced CT appearance of dolichoectasia or giant fusiform aneurysm is virtually pathognomonic.\(^1\)-\(^4\), \(^18\)

A definitive statement on treatment of this anomaly cannot be made on the basis of the 11 patients presented. The decision regarding medical versus surgical treatment is related to the unique features in the individual patients. When recommended, surgical treatment should be directed toward exclusion of the giant fusiform aneurysm from the cerebral circulation. This can be achieved by trapping or by proximal artery ligation. Reduction of the mass effect by excision of the aneurysm wall and/or removal of thrombus should be performed whenever possible in order to help relieve symptoms. Trapping and decompression of the aneurysm was successfully accomplished in 2 of our patients, including one with an ICA aneurysm, and another with a PCA aneurysm. A patient with an ICA aneurysm did well after proximal ligation only.

The use of regional cerebral blood flow measurement techniques and cerebral revascularization may play an important role in preventing ischemic complications in selected patients undergoing entrapment of the aneurysm or proximal artery ligation.\(^7\) Gelber and Sundt\(^6\) have shown that the risk of infarction can be reduced in certain patients by performing an anastomosis between the STA and a branch of the MCA before proximal ligation for giant aneurysm of the ICA. Regional cerebral blood flow studies were used to select the patients requiring the pre-occlusion revascularization procedure. In their study, only one of 6 patients undergoing ICA ligation after anastomosis was unable to tolerate the occlusion.

Trapping of a giant fusiform aneurysm involving the M-1 segment of MCA or the anterior communicating artery is potentially more dangerous because of the presence of important perforating branches. In our series, one patient suffered a large deep cerebral infarct following thrombosis of a giant fusiform aneurysm of the M-1 segment. Thrombosis appeared to be the result of relative stasis in the aneurysm following a STA to MCA anastomosis.\(^9\)

Ammerman and Smith\(^19\) described a case of giant fusiform aneurysm of the left M-1 segment treated by intracranial ICA and distal M-1 ligation (i.e., not complete entrapment) together with STA to MCA anastomosis. Following surgery, the patient was dysphasic and had right hemiparesis. These deficits improved during the subsequent 2 months.

Excision of a giant fusiform aneurysm involving a major MCA branch, however, appears to carry less risk of cerebral infarction. In 2 patients, Segal and McLaurin\(^10\) described successful excision of a giant fusiform aneurysm involving a major MCA branch only.

References

15. Pinto RS, Krichoff II, Butler AR, Murulli R: Correlation of


17. Ley A: Compression of the optic nerve by a fusiform aneurysm of the carotid artery. J Neurol Neurosurg Psychiatry 13: 75-86, 1950


Concordance of Inhalation rCBFs with Clinical Evidence of Cerebral Ischemia

JAMES R. EWING, M.S., E. GREGORY KEATING, PH.D., PAUL R. SHEEHE, SC.D.,
CHARLES J. HODGE, M.D., MARTIN CHIPMAN, M.D.,
AND CORLEEN T. BROOKS, B.S.

SUMMARY Using the 133-Xenon inhalation technique, cerebral blood flow (CBF) and hemispheric blood flow (HBF) were determined serially in 45 patients with acute stroke undergoing pharmacologic trials and in 8 transient ischemic attacks (TIA) scheduled for superficial temporal-middle cerebral artery anastomoses. Both patient populations had lower blood flow than a control group of similar ages. Patients in both populations with lateralized clinical signs demonstrated an asymmetry in HBF which corresponded to their clinical signs. In the stroke population, the trend we expected over time toward development of asymmetrical HBF as the non-infarcted hemisphere recovered from diaschisis did not appear.

Stroke, Vol 12, No 2, 1981

THE NON-INVASIVE 133-Xenon inhalation technique for measuring regional cerebral blood flow (rCBF) needs to be evaluated for its sensitivity to ischemia. Toward this end, we have compared our measurements of rCBF in patients suffering a lateralized hemodynamic deficit to prior results using invasive techniques on similar patients.

A number of studies using invasive techniques report bilateral measurements in hemispheric blood flow (HBF) following stroke. Inhalation technique studies in patients with stroke have also been performed. Rao et al., using the fast compartment clearance rate, $k_1$, assessed the inhalation technique in a population of patients with acute stroke, but did not report differences in HBF. Blauenstein et al., in a similar population, reported interhemispheric differences in Tissue Volume Perfused, a parameter related to uptake of 133-Xenon indicator. Recently, in a short note, Halsey et al. have reported interhemispheric differences in the Initial Slope Index (ISI) of Risberg et al. in a population of patients with stroke, in which it was not stated whether large asymmetries in ISI were reliable indicators of laterality of stroke.

A stable parameter is necessary for the summary of inhalation technique results in a population of patients with stroke. In patients with ischemic infarct studied with other techniques, most useful flow estimates have come from analyses which do not assume a certain number of distinct tissue compartments. The Kety-Schmidt technique measures of CBF or HBF and the hydrogen clearance technique measurements of HBF are model independent, as is
Giant fusiform aneurysm of the cerebral arteries.
J R Little, P St Louis, M Weinstein and D F Dohn

doi: 10.1161/01.STR.12.2.183

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/12/2/183