Intracranial dissecting aneurysm

Case report

OSAMU SATO, M.D., JAMES F. BASCOM, M.D., AND JOHN LOGOTHETIS, M.D.
Departments of Neurology and Surgery, Hennepin County General Hospital, and
Department of Neurology, University of Minnesota Medical School, Minneapolis,
Minnesota

The case of a 6-year-old boy who died 4 days after the acute onset of a
left middle cerebral artery aneurysmal dissection is described. A review of
the 30 reported cases with similar lesions reveals their relative rarity. The
age distribution, sex incidence, intracranial vessels affected, and postulated
causes of the dissection are discussed.

KEY WORDS · dissecting aneurysm · intracranial · middle cerebral
artery

Dissecting aneurysms of the intracranial arteries have been included
among the less common etiological factors in obstructive cerebrovascular
disease. A dissecting aneurysm is caused by leakage of the circulating blood into the ves-
sel wall so as to separate its layers. Cerebral dissecting aneurysms are characterized by
the subintimal site of dissection, as con-
trasted to dissections of the aorta that usu-
ally split the media. It is the purpose of this
communication to describe a case of left
middle cerebral artery occlusion from a dis-
secting aneurysm observed in a 6-year-old
boy, and also to review the clinical and
pathological features of the cases previously
reported. It is felt that, in spite of its rare oc-
currence, a dissecting aneurysm of an intra-
cranial vessel should always be kept in mind
as a cause of an acute stroke, especially in a
younger individual where arteriosclerotic
occlusion is uncommon.

Case Report

A 6-year-old white boy was admitted to the Hennepin County General Hospital,
Minneapolis, on March 29, 1960, for stupor
of 2 hours' duration. About 4:30 p.m.
following a normal school day, he had stag-
gered into his home unable to talk. He had a
swollen left eye and looked strange. He went
to the bathroom hurriedly and then fell on
the floor unresponsive. There had been no
known anoxia, jaundice, or cyanosis at his
normal birth. His growth and development
had been normal. Two to 3 months prior to
admission, and again 3 days before admis-
sion, he had an unexplained left black eye.

Examination. The patient was in a semi-
comatose condition. Blood pressure was 140
/104, pulse 88 irregular, respiration 28/
min, body temperature 94.4°F. No seizure
activity or meningeal signs were observed.
Shortly after admission he could respond to
simple commands but was unable to talk.
Optic fundi were normal. The pupils were
round, regular, equal, and reactive. He ap-
peared to have a right homonymous hemian-
opsis and a right hemiparesis. There was a
Babinski sign and unsustained ankle clonus
on the right. Lumbar puncture released
clear, colorless spinal fluid at a pressure of
Osamu Sato, James F. Bascom and John Logothetis

Fig. 1. Left middle cerebral artery under low-power magnification. A blood clot occupies nearly the entire lumen of the vessel. × 25.

140 mm H₂O; it contained 3 white blood cells (WBC) and 107 red blood cells (RBC), all crenate, and a protein content of 21 mg%. Blood hemoglobin was 12.9 gm, and the peripheral white blood cell count was 14,500/cu mm. Skull films were normal.

A left carotid angiogram on March 30 showed good filling of the common carotid and the anterior cerebral arteries but no direct filling of the middle cerebral artery, which filled only partially through collaterals. The common carotid was in spasm.

On the evening of March 31, the blood pressure steadily rose to 220 mm Hg systolic and 120 diastolic. This was attributed to increasing cerebral edema. In spite of the use of Urevert and dexamethasone, the patient's condition rapidly deteriorated. Late on April 1 he developed irregular breathing and shortly after became apneic. An endotracheal tube was inserted and connected to Bird apparatus. An emergency bilateral subtemporal decompression was done, but the patient grew steadily worse during the next 2 days and died on April 4.

Postmortem Examination. Gross examination of the brain revealed mild swelling and almost complete softening of the entire left temporal lobe. There was an extensive tentorial herniation on the left measuring about 1 cm in width, with displacement of the midbrain by the swollen temporal lobe. The large cerebral arteries of the circle of Willis were carefully examined; the middle cerebral artery on the left, just before the trifurcation, was distended for a distance of about 1 cm. Sections through the left middle cerebral artery showed the vessel to be filled with clot. One branch of the trifurcation contained a very firm clot and was lighter in color than the rest of the clot. The other vessels at the base of the brain were normal. Microscopic examination revealed necrosis of the cerebral parenchyma and a good deal of ischemic necrosis of the cortical neurons. The left middle cerebral artery showed the most dramatic changes (Figs. 1 and 2). There was a large hemorrhage into the wall of the vessel, splitting off the intimal layer and displacing the lumen of the vessel to one side with only a very small vessel lumen remaining patent. The picture was typical of a dissecting aneurysm.

Discussion

Dissecting aneurysms are most commonly found in the aorta and its branches and only occasionally have been described in other vessels. Even more rare are dissecting aneurysms involving intracranial vessels. Table 1 and Fig. 3, which summarize previous cases, show that this lesion has rarely occurred before the age of 10 years.

Norman and Urich⁹ reported a patient who at 6 months became hemiplegic and subsequently developed convulsions and mental deficiency. He later died of pulmonary tuberculosis at age 15, and autopsy revealed a dissecting aneurysm of the right middle cerebral artery. This report is also noteworthy, as the authors postulated dis-
### TABLE 1

**Summary of cases of dissecting aneurysm of the cerebral arteries**

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Age, Sex</th>
<th>Remarks</th>
<th>Vessels Involved</th>
<th>Postulated Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turnbull (1915)</td>
<td>— —</td>
<td></td>
<td>middle cerebral artery</td>
<td>luetic arteritis</td>
</tr>
<tr>
<td>Scholefield (1924)</td>
<td>47 M</td>
<td>died 15 days after the onset</td>
<td>right vertebral and basilar arteries</td>
<td>—</td>
</tr>
<tr>
<td>Stern (1933)</td>
<td>24 M</td>
<td>died in 6 days</td>
<td>basilar artery, posterior cerebral artery, post. comm. ant. choroid arteries</td>
<td>—</td>
</tr>
<tr>
<td>Hyland (1933)</td>
<td>42 M</td>
<td>died in 20 days</td>
<td>basilar artery</td>
<td>mucoid degeneration of the media</td>
</tr>
<tr>
<td>Hassin (1937)</td>
<td>37 M</td>
<td>accidental electrocution</td>
<td>&quot;large blood vessels&quot;</td>
<td>trauma</td>
</tr>
<tr>
<td>Szabo (1939)</td>
<td>35 F</td>
<td></td>
<td>right vertebral artery</td>
<td>luetic arteritis</td>
</tr>
<tr>
<td>de Veer &amp; Browder (1942)</td>
<td>42 M</td>
<td>died 57 hours after a fall</td>
<td>right middle cerebral artery</td>
<td>trauma</td>
</tr>
<tr>
<td>Dratz &amp; Woodhall (1947)</td>
<td>21 F</td>
<td>died 2 days after car accident</td>
<td>left internal carotid from post. comm. into anterior and middle cerebral arteries</td>
<td>trauma</td>
</tr>
<tr>
<td>Ramsey &amp; Mosquera (1948)</td>
<td>47 M</td>
<td>occipital headache for 2 weeks, left hemiparesis, unconsciousness, died 6 days after admission</td>
<td>right middle cerebral artery</td>
<td>—</td>
</tr>
<tr>
<td>Poppen (1951)</td>
<td>— —</td>
<td>patient recovered after surgery</td>
<td>angular branch of middle cerebral artery</td>
<td>—</td>
</tr>
<tr>
<td>Poppen (1951)</td>
<td>— —</td>
<td>well 8 years after ligation of internal carotid artery</td>
<td>medial trunk of middle cerebral artery</td>
<td>—</td>
</tr>
<tr>
<td>Sinclair (1953)</td>
<td>27 F</td>
<td>severe migraine for several years, sudden pain, and death 4 days after admission</td>
<td>right middle cerebral artery</td>
<td>migraine</td>
</tr>
<tr>
<td>Bigelow (1955)</td>
<td>46 F</td>
<td>died 1 day after excision of berry aneurysm</td>
<td>right middle cerebral artery</td>
<td>postop trauma or congenital defect</td>
</tr>
<tr>
<td>Watson (1956)</td>
<td>32 M</td>
<td>died in 4 days</td>
<td>basilar artery</td>
<td>cystic degeneration of the media</td>
</tr>
<tr>
<td>Norman &amp; Urich (1957)</td>
<td>15 M</td>
<td>hemiplegia, convulsion and mental arrest at age 6 months, died of tbc. at age 14</td>
<td>right middle cerebral artery</td>
<td>—</td>
</tr>
<tr>
<td>Wolman (1959)</td>
<td>16 M</td>
<td>sudden onset, died in 4 days</td>
<td>right middle cerebral artery</td>
<td>congenital defect</td>
</tr>
<tr>
<td>Wolman (1959)</td>
<td>19 F</td>
<td>right-sided spasticity and hemiparesis at 3 months of age; behavior disorder, epilepsy; left hemispherectomy at age 19, specimen examined</td>
<td>left middle cerebral artery</td>
<td>congenital defect</td>
</tr>
<tr>
<td>Wolman (1959)</td>
<td>33 F</td>
<td>severe headache, sudden collapse, died in 7 days</td>
<td>basilar artery</td>
<td>congenital defect</td>
</tr>
<tr>
<td>Scott, et al. (1960)</td>
<td>29 F</td>
<td>died 6 days after tonsillectomy</td>
<td>right internal carotid artery at the bifurcation of middle and anterior cerebral arteries</td>
<td>—</td>
</tr>
<tr>
<td>Wisoff &amp; Rothbailer (1961)</td>
<td>11 F</td>
<td>died 5 days after left hemiparesis, 2-wk history of mild intermittent headache</td>
<td>right internal carotid at the bifurcation</td>
<td>cystic media degeneration</td>
</tr>
<tr>
<td>Ritchie (1961)</td>
<td>17 M</td>
<td>died 5 days after drinking and fighting</td>
<td>left internal carotid and left middle cerebral arteries</td>
<td>trauma</td>
</tr>
<tr>
<td>Spudis, et al. (1962)</td>
<td>30 F</td>
<td>left hemiparesis, died 11 days later</td>
<td>right internal carotid and right middle cerebral arteries</td>
<td>migraine</td>
</tr>
<tr>
<td>Nedwich, et al. (1963)</td>
<td>30 F</td>
<td>died 3 days after collapse</td>
<td>right middle cerebral artery</td>
<td>congenital defect</td>
</tr>
</tbody>
</table>

*Table 1 continued on next page*
TABLE 1 (Continued)

<table>
<thead>
<tr>
<th>Author, Year</th>
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<th>Remarks</th>
<th>Vessels Involved</th>
<th>Postulated Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duman (1963)</td>
<td>18 M</td>
<td>left the hospital with left hemiparesis 27 days after admission</td>
<td>right middle cerebral artery</td>
<td>trauma</td>
</tr>
<tr>
<td>Duman (1963)</td>
<td>60 M</td>
<td>left the hospital with left hemiparesis 27 days after hospitalization</td>
<td>right middle cerebral artery</td>
<td>trauma</td>
</tr>
<tr>
<td>Duman (1963)</td>
<td>21 M</td>
<td>hemiplegic 4 days after accident, died 16 days after his initial fall</td>
<td>left middle cerebral artery</td>
<td>trauma water-skiing</td>
</tr>
<tr>
<td>Robert, et al. (1964)</td>
<td>20 F</td>
<td>died 3 days after developing headache, left hemiparesis, and sensory impairment</td>
<td>right middle cerebral artery</td>
<td>—</td>
</tr>
<tr>
<td>Perier, et al. (1964)</td>
<td>22 F</td>
<td>severe headache 15 days after giving birth, comatose, left oculomotor palsy and left hemiparesis, died on 54th day</td>
<td>basilar artery</td>
<td>—</td>
</tr>
<tr>
<td>Hayman &amp; Anderson (1966)</td>
<td>15 M</td>
<td>mental retardation, found semicomatose and right hemiplegic, died 8 weeks after admission</td>
<td>basilar artery</td>
<td>—</td>
</tr>
<tr>
<td>Walb, et al. (1967)</td>
<td>30 F</td>
<td>headache for 4 weeks prior to severe headache and decerebration, died in 7 days</td>
<td>basilar artery</td>
<td>congenital defect</td>
</tr>
<tr>
<td>Sato, et al. (1971)</td>
<td>6 M</td>
<td>left black eye, sudden collapse, rt hemiparesis, died 4 days later</td>
<td>left middle cerebral artery</td>
<td>—</td>
</tr>
</tbody>
</table>

The middle cerebral artery appears to be the most frequently involved intracranial vessel, constituting more than 50% of all reported cases (Table 2). The right side is more often involved, with a ratio of 3:1. Dissecting aneurysms of the internal carotid arteries were reported in five occasions, with no predilection as to side. The basilar artery has been involved in seven cases and two, while congenital defects in the cerebral arterial wall have been cited as possible causes by others. Luetic arteritis as an underlying cause is mentioned by several. Wisoff and Rothbailer attributed the dissection in their patients to degenerative change in the media of the affected vessels. Two authors have suggested a causal relation between migraine and the dissecting aneurysm.

In our case, in spite of no documentation...
Intracranial dissecting aneurysms

TABLE 3
Postulated causes in cases of dissecting aneurysm of the cerebral arteries

<table>
<thead>
<tr>
<th>Suggested Cause</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>head trauma</td>
<td>8</td>
</tr>
<tr>
<td>leutic arteritis</td>
<td>2</td>
</tr>
<tr>
<td>degenerative change of the media associated with migraine headache</td>
<td>4</td>
</tr>
<tr>
<td>congenital defect of the vessel wall undetermined</td>
<td>10</td>
</tr>
</tbody>
</table>

of injury, the left black eye noted 2 to 3 months before admission and also 3 days before the acute episode speaks in favor of head trauma as the cause of the dissection of the arterial wall.

References

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Address reprint requests to: Osamu Sato, M.D., Department of Neurosurgery, Nagoya University, 65, Tsurumai-cho, Showa, Nagoya, Japan.