**Short Communication**

**Fusiform Basilar Aneurysm as a Cause of Embolic Stroke**

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**SUMMARY** Giant fusiform basilar aneurysms (dolicho-ectatic basilar anomalies) are rare and have not previously been reported to cause embolic infarction in territory distal to the aneurysm. They most commonly present as posterior fossa mass lesions with brainstem compression and cranial neuropathies. Originally considered atherosclerotic in etiology, recent authors feel that they may represent a unique arteriopathy characterized by loss of elastin in the vessel wall. We report a case which presented solely as an occipital lobe infarction. To our knowledge, this is the first case in which a fusiform basilar aneurysm presented with an embolic infarction as its only manifestation.

**Case Report**

A 59-year-old man with mild hypertension and Type IV hyperlipidemia experienced abrupt onset of right periorbital headache accompanied by loss of vision in the left visual field. He had no previous history of transient ischemic attacks (TIAs) or stroke. Several weeks prior to the event, he noted frequent right periorbital headaches lasting 45–60 minutes without neurologic symptoms. Nine years previously he underwent surgery for repair of an abdominal aortic aneurysm. His risk factors included a 20 year history of smoking, hypertriglyceridemia, and moderate obesity.

His general physical examination was normal except for early retinal vessel sclerosis. Blood pressure was 130/84 mm Hg. There were no carotid bruits. Neurologic examination demonstrated a left superior quadrantanopsia, but was otherwise unremarkable.

The following studies were normal: serum electrolytes, complete blood count, syphilis serology, urinalysis, coagulation profile, echocardiogram, and lumbar puncture. Serum triglycerides were 261 mg/dl (normal, 30–150 mg/dl). The electrocardiogram demonstrated borderline nonspecific ST-T wave changes and rare multifocal premature ventricular contractions.

A computed tomographic (CT) scan of the head demonstrated a nonhemorrhagic right occipital lobe infarction (fig. 1), and a large eccentrically positioned basilar artery, with calcification in its walls, extending well above the posterior clinoids (fig. 2a). The contrast enhanced (fig. 2b) and reformatted coronal (fig. 3) images revealed the presence of incomplete thrombosis in the aneurysm which measured up to 2 cm in diameter. Pancerebral angiography (fig. 4) confirmed the diagnosis of a partially thrombosed giant fusiform basilar aneurysm and showed the lesion to be continuous with an ectatic and elongated dominant right vertebral artery. In addition, a clinically silent, high-grade left internal carotid artery stenosis with ulcerative changes was found. The calcarine branches of the posterior cerebral arteries were normal and neither posterior communicating artery was patent.

He was treated with platelet inhibitors. The left superior quadrantanopsia resolved within two months. Three months after the event he underwent an uncomplicated left carotid endarterectomy; follow-up neurologic examinations have been normal.

**Discussion**

The fusiform basilar aneurysm (B. I. in the classification of Pia) is an uncommon vascular abnormality characterized by tortuosity, elongation, and ectasia of the basilar artery. In his review, Pia found that only 6.2% (33 of 538) of intracranial aneurysms were located in the vertebrobasilar system. These included 4 fusiform basilar aneurysms, 0.7% of the total. In two
TABLE 1  Fusiform Basilar Aneurysms: Clinical Syndromes

<table>
<thead>
<tr>
<th>Neurologic syndrome</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemifacial spasm</td>
<td>14</td>
</tr>
<tr>
<td>Trigeminal neuralgia</td>
<td>8,12,21</td>
</tr>
<tr>
<td>Oculomotor paralysis</td>
<td>1,20</td>
</tr>
<tr>
<td>Cerebellopontine tumor</td>
<td>12,13</td>
</tr>
<tr>
<td>Supranuclear ophthalmoplegia</td>
<td>1,19</td>
</tr>
<tr>
<td>Multiple cranial neuropathies</td>
<td>4,9,12</td>
</tr>
<tr>
<td>Obstructive hydrocephalus</td>
<td>4,16</td>
</tr>
<tr>
<td>Brainstem compression</td>
<td>1,9,16</td>
</tr>
<tr>
<td>TIA</td>
<td>6,18</td>
</tr>
<tr>
<td>Visual field loss</td>
<td>1,3,4,5</td>
</tr>
</tbody>
</table>

autopsy series comprising 20,500 cases, only 7 fusiform basilar aneurysms were found. Dandy called it the “S” aneurysm. Other authors deny that the lesion is a true aneurysm and suggest that it be called an anomaly. Mitts used the noncommittal term “fusiform enlargement.” The disorder usually presents as a posterior fossa mass with diverse symptomatology, including brainstem compression, multiple cranial neuropathies, hemifacial spasm, trigeminal neuralgia, supranuclear ophthalmoplegia, or obstructive hydrocephalus (table 1). Isolated embolic occipital lobe infarction, arising from a partially thrombosed basilar aneurysm, has not previously been reported.

Cohen and Antunes have established criteria by which a diagnosis of arterial embolism from an intracranial aneurysm may be established: 1) clinical TIA or stroke, 2) arteriographic or pathologic confirmation of the aneurysm, 3) no other lesion which could produce TIA or stroke, and 4) no evidence of subarachnoid hemorrhage or vasospasm. Our case satisfies these criteria. In addition, the abrupt onset, rapid resolution, and arteriographically demonstrated patency of the calcarine vessels further suggest an embolus. Thrombus within the lumen of the basilar artery was the likely source of the embolus. Even if the right posterior communicating artery had been patent, it is extremely unlikely that an embolus from the left carotid artery lesion would pass to the diagonally opposite posterior cerebral artery.

Emboli arising from intracranial aneurysms are rare. Only one of 73 cases of giant saccular or fusiform aneurysms had symptoms of cerebral emboli in the form of TIAs. Antunes reported 2 cases of cerebral emboli arising from aneurysms of the internal carotid-posterior communicating artery juncture with arteriographically or pathologically demonstrated thrombus in the aneurysm. In the only case examined at autopsy, emboli were not found in the distal vessels. Hirsch and Gonzalez reported a patient who had left internal carotid territory TIAs which failed to cease after carotid endarterectomy. Only after the patient suffered a brainstem infarction was his fusiform basilar aneurysm discovered.

Cohen concisely summarized why emboli seldom arise from intracranial aneurysms. First, giant aneurysms, those most likely to contain thrombus, account for only 3 to 5% of intracranial aneurysms. Second, thrombus formation and embolus release depend on critical, unstable, relationships among aneurysmal size, orifice size, stagnation, and turbulence. The size of the aneurysm and its orifices determine the degree of turbulence or stagnation of blood within it. Excessive turbulence is not conducive to thrombus formation; excessive stagnation will not promote embolization. The precise balance between turbulence and stagnation, necessary to promote thrombus formation.

TABLE 2  Visual Syndromes Caused by Fusiform Basilar Aneurysms

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Mechanism</th>
<th>Patients</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bitemporal hemianopsia</td>
<td>Chiasmal compression</td>
<td>2</td>
<td>3&amp;4</td>
</tr>
<tr>
<td>Homonymous hemianopsia</td>
<td>Compression of right optic tract</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Homonymous hemianopsia</td>
<td>Suspected occipital compression</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Homonymous hemianopsia</td>
<td>Not reported</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>
and release, may only exist transiently in the natural
der than of an intracranial aneurysm. Finally, intracran-
ial aneurysms, in contrast to extracranial aneurysms,
are not subject to distortion or pressure from adjacent
structures which may dislodge an embolis.

Dandy stated that the "S" aneurysms "are unques-
tionably of arteriosclerotic origin."9 The five cases
reported by Hayes et al.9 all had extensive atheroscle-
rosis. Nijensohn et al.10 reviewed the clinical and gross
pathological features of 27 saccular and 23 fusiform
basilar aneurysms. Fusiform aneurysms correlated
with male sex, age over 60, atherosclerotic heart dis-
 ease, and aneurysmal dilation of other vessels, espe-
cially the abdominal aorta. Saccular aneurysms pre-
dominated in a younger, largely female, population
without associated atherosclerotic heart disease. The
incidence of hypertension was the same in both
groups.

However, Greitz and Lofstedt5 in 1954 noted the
absence of atherosclerotic changes in 2 of 3 cases ex-
amined pathologically. In 1959 they reported that 3 of
4 patients with dilation and elongation of the basilar
artery had abnormal focal defects in the elastic fibers
of the intima, not only of the atherosclerotic vessels,
but also of ectatic vessels without atherosclerosis.29
Boeri and Passerini1 suggested that atrophy of the elas-
tica and muscularis lead to dilation and tortuosity of
the vessels. Sacks and Lindenburg11 studied 34 cases
of cerebral artery ectasia histopathologically and found
irregularity in the thickness of the media, multiple gaps
in the intimal elastic membrane, and atrophy of the
muscularis with replacement by hypertrophic, swollen
connective tissue. They concluded that the cause of the
dolicho-ectatic anomaly was a primary dysplasia of the
muscularis with secondary changes in the elastic tissue.
FUSIFORM BASILAR ANEURYSM AS A CAUSE OF EMBOLIC STROKE/Steel et al.

Figure 4. Selective right vertebral angiogram demonstrates marked ectasia of this vessel, and the patent channel through basilar artery. A) Anteroposterior projection illustrates left lateral location of lumen and lack of sharp interface of contrast material medially due to thrombus. B) Lateral view reveals marked posterior location of basilar artery lumen (arrow) with respect to clivus and sella turcica (dashed lines).

Due to arterial distention, or, a congenital deficiency of the elastic tissue which predisposes vessels to dilate when subjected to hypertension. Relevant to this discussion, Busuttil et al.30 detected collagenase activity in the vessel walls of patients with resected abdominal aortic aneurysms but not in the walls of atherosclerotic aortas. Abnormal persistence of this proteolytic enzyme may weaken vessel walls, predisposing to dilation and elongation and secondary atherosclerotic changes.

At the present time the etiology of fusiform aneurysms remains obscure. Comparison of collagenase activity in the walls of resected fusiform aneurysms versus that in the walls of stenotic lesions may illuminate this question.

References
1974
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