The Incidence and Clinical Significance of Fenestrations in the Horizontal Segment of the Anterior Cerebral Artery Detected by Conventional Angiography and Magnetic Resonance Angiography

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Objective: We examined the incidence of fenestration in the proximal segment of the anterior cerebral artery (ACA) and reviewed its clinical significance.

Methods: Cerebral angiography was performed in 843 patients and magnetic resonance angiography (MRA) in 1,787; all patients had, or were suspected to have, cerebrovascular disease. We subsequently reviewed the angiography and MRA data.

Results: Fourteen patients (six men and eight women, 35–81 years of age, median age = 67 years) had proximal ACA fenestrations. Three of the 843 patients (0.36%) undergoing cerebral angiography had fenestrations of proximal ACA as did eleven of the 1,787 MRA patients (0.62%). Seven fenestrations were located on the right side and seven fenestrations were located on the left side. The fenestrated ACA in one patient was associated with a saccular aneurysm at its proximal end. None of the 120 patients who underwent both conventional cerebral angiography and cranial MRA showed fenestration of proximal ACA.

Conclusion: Although fenestration of ACA has little clinical significance, knowledge and recognition of this condition is important in the interpretation of cerebral images, especially during neurosurgical procedures.

KEY WORDS: Fenestration • Anterior cerebral artery • Incidence.

Introduction

Fenestration of the cerebral arteries is well-known incidental angiographic or autopsy observation. Although usually not related to any clinical symptoms, there is evidence suggesting a relationship between fenestration and the incidence of intracerebral aneurysms, transient ischemic attack, and other clinical conditions. It is important to understand the complex anatomy of fenestrations and aneurysms when Guglielmi detachable coil embolization is used as an alternative treatment method for cerebral aneurysms. There have been three Korean reports describing fenestration in the proximal segment of ACA (A1), associated with aneurysm. The purpose of the present study is to determine the incidence and clinical implications of fenestration of A1.

Materials and Methods

A retrospective review of 843 conventional cerebral angiograms and 1,787 cranial MRAs performed over an eight-year period identified patients with intracranial arterial fenestrations. Conventional cerebral angiography (Philips V-5000, Philips Medical System, Eindhoven, Netherlands) was performed on 494 patients between July 1997 and June 2004, and cranial MRA (1.5T, Sigma MRi, General Electric, Milwaukee, WI, USA) on 880 patients between May 2001 and June 2004 in one hospital. In addition, conventional cerebral angiography (Philips V-3000, Philips Medical System, Eindhoven, Netherlands) was performed on 349 patients between June 2002 and August 2005, and cranial MRA (1.5T, Intera-achieva, Philips Medical System, Eindhoven, Netherlands) was performed on...

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907 patients between August 2003 and August 2005 in a second hospital. Both cerebral angiography and MRA were performed on 120 patients. The initial MRA and angiographic studies were undertaken for a variety of clinical reasons, including symptoms of cerebral ischemia, cerebral infarction, hemorrhagic contusion, intracerebral hemorrhage, headache, and dizziness.

One hospital acquired MRAs using a three-dimensional time-of-flight (TOF) technique with a neurovascular phased array coil (MRI devices, Milwaukee, WI, USA) plus a multiple overlapping thin slab acquisition technique. In the second hospital, a three-dimensional TOF technique with a neurovascular phased array coil (SENSE-Head-8, Philips Medical System, Eindhoven, Netherlands) was used. The images were obtained either from a routine diagnostic study or from the initial diagnosis during an intervention procedure. We subsequently evaluated all of the angiograms and MRAs for cerebral arterial anatomic variations. In patients exhibiting fenestration, special attention was given to defining the origin and size of the fenestrated vessels. We recorded the presence of associated vascular lesions, including cerebral aneurysms, vascular stenoses or occlusions and other anomalies.

**Results**

Fourteen patients (six men and eight women, 35 to 81 years of age, median age 67 years) had fenestrations of proximal ACA. Seven fenestrations were located on the right side and seven fenestrations were located on the left side. None of the 120 patients who underwent both conventional cerebral angiography and cranial MRA showed fenestration of A1. Except one case, fenestration of A1 was discovered by a MRA (Fig. 1) and an angiographic finding (Fig. 2) incidentally. One patient (Case 3) had a fenestrated anterior cerebral artery associated with a saccular aneurysm at its proximal end (Fig. 3).

Three (0.36%) of the 843 patients who underwent cerebral angiography showed fenestration in A1, while eleven (0.62%) of the 1,787 MRA patients had a fenestration of A1 (Table 1).

Vascular anomalies associated with fenestration included an ipsilateral middle cerebral artery aneurysm, an ipsilateral posterior communicating artery aneurysm, and an aneurysm at the proximal end of the fenestrated A1.

**Discussion**

Incidence of proximal ACA fenestration

Fenestration of the A1 segment was first described in 1905 by Fawcett and Blaschford. They encountered this anomaly in 0.14% of the 700 brains investigated. Fenestration of ACA...
Table 1. Summary of 14 patients exhibiting fenestrations of the proximal anterior cerebral artery detected by transluminal cerebral angiography and magnetic resonance angiography

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/ Sex</th>
<th>Location</th>
<th>Associated vessel anomaly</th>
<th>Symptom</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F/68</td>
<td>left, distal of A1</td>
<td>left MCA aneurysm</td>
<td>SAH</td>
<td>A</td>
</tr>
<tr>
<td>2</td>
<td>F/69</td>
<td>right, distal of A1</td>
<td>right P–com aneurysm</td>
<td>SAH</td>
<td>A</td>
</tr>
<tr>
<td>3</td>
<td>M/43</td>
<td>left, distal of A1</td>
<td>left ACA aneurysm</td>
<td>SAH</td>
<td>A</td>
</tr>
<tr>
<td>4</td>
<td>F/68</td>
<td>right, distal of A1</td>
<td>no</td>
<td>headache</td>
<td>M</td>
</tr>
<tr>
<td>5</td>
<td>F/67</td>
<td>left, mid–distal of A1</td>
<td>no</td>
<td>headache</td>
<td>M</td>
</tr>
<tr>
<td>6</td>
<td>F/45</td>
<td>right, mid–distal of A1</td>
<td>no</td>
<td>headache</td>
<td>M</td>
</tr>
<tr>
<td>7</td>
<td>F/57</td>
<td>right, distal of A1</td>
<td>no</td>
<td>vertigo</td>
<td>M</td>
</tr>
<tr>
<td>8</td>
<td>F/35</td>
<td>right, distal of A1</td>
<td>no</td>
<td>infarction</td>
<td>M</td>
</tr>
<tr>
<td>9</td>
<td>M/69</td>
<td>left, distal of A1</td>
<td>no</td>
<td>infarction</td>
<td>M</td>
</tr>
<tr>
<td>10</td>
<td>M/69</td>
<td>left, distal of A1</td>
<td>no</td>
<td>TIA</td>
<td>M</td>
</tr>
<tr>
<td>11</td>
<td>F/65</td>
<td>right, distal of A1</td>
<td>no</td>
<td>facial palsy</td>
<td>M</td>
</tr>
<tr>
<td>12</td>
<td>M/81</td>
<td>right, distal of A1</td>
<td>no</td>
<td>dizziness</td>
<td>M</td>
</tr>
<tr>
<td>13</td>
<td>M/67</td>
<td>left, distal of A1</td>
<td>no</td>
<td>infarction</td>
<td>M</td>
</tr>
<tr>
<td>14</td>
<td>M/50</td>
<td>left, distal of A1</td>
<td>no</td>
<td>IH</td>
<td>M</td>
</tr>
</tbody>
</table>

Abbreviations: MCA = middle cerebral artery; SAH = subarachnoid hemorrhage; P–com = posterior communicating artery; A1 = terminal cerebral angio–
graphy; A1 = proximal segment of anterior cerebral artery; M = magnetic resonance angiography; ACA = anterior cerebral artery; TIA = transient ischemic attack; IH = intracerebral hemorrhage.

has been described anatomically\textsuperscript{12,20}, but its precise incidence is still unknown. Sanders et al.\textsuperscript{13} in their review of 5,190 cerebral angiograms, reported 37 patients with 38 fenestrated arteries comprising 16 basilar (0.5%), 10 vertebral (0.19%), 9 middle cerebral (0.17%), and three anterior cerebral (0.06%) arteries. Three fenestrations of A1 associated with aneurysms at their proximal ends have been reported in Korea\textsuperscript{14,16}. In the present study, three (0.36%) of the 843 patients who underwent cerebral angiography showed fenestration in A1 as did eleven (0.62%) of the 1,787 patients studied by MRA.

Friedlander, et al.\textsuperscript{11} reported that 16 of 58 cases (29 identified in postmortem examinations, 18 during cerebral arteriography, and 8 during surgery) had a fenestrated A1 associated with a saccular aneurysm at the proximal end of the fenestration (27.6%). In our study, a fenestrated ACA associated with a saccular aneurysm at its proximal end was observed only once (Case 3) in 14 patients (7.1%).

Pathogenesis of proximal ACA fenestration

Fenestration of the basilar artery can be explained by its embryological formation from the fusion of two longitudinal arteries\textsuperscript{19}. However, fenestration of the A1 segment is still not clearly understood. As we observed, these fenestrations occur only in the distal end of the A1 segment. Various theories describing the generation of the A1 fenestration have been proposed; fenestration might occur as a result of partial duplication, an incomplete fusion, or the anomalous course of a nonvascular structure through the vascular precursor\textsuperscript{20}. However, a fenestration of the A1 segment that only occurs in the distal end of the A1 segment is not as easily explained. Minakawa, et al.\textsuperscript{12} have suggested that such an A1 fenestration may be a remnant of the plexiform anastomosis between the primitive ophthalmic artery and the ACA. In Padgett\textsuperscript{4,5} illustration of a 14mm embryo, one can see the plexiform anastomosis between the ACA and the primitive ophthalmic artery. In addition, in 18mm and 24mm embryos, there is a fenestration in the distal end of the ACA. However, by the 43mm stage, no such fenestration is indicated. These facts suggest that in 18–43mm embryos, fenestrations of the distal A1 segment may result from the failure of fusion in the plexiform anastomosis present in the distal primitive ACA\textsuperscript{5}.

Clinical consideration of proximal ACA fenestration

Most aneurysms of the ACA arise either at the anterior communicating complex or at the junction of the pericallosal and callosomarginal arteries. A1 aneurysms are relatively rare, accounting for only about 1% of all aneurysms\textsuperscript{3,21}. A1 aneurysms have been reported to be associated most commonly with other vascular abnormalities such as A1 fenestration, the presence of an accessory middle cerebral artery (MCA), abnormally elongated A1, azygos ACA, and so on\textsuperscript{4,11,20,21}. There are various explanations for the origin of cerebral aneurysms. Some emphasize the importance of vascular anomalies in the circle of Willis in their genesis\textsuperscript{19}. Stehbens\textsuperscript{19} has suggested that the circulatory dynamics of vascular anomalies play an important role in the development of aneurysms: fenestration exerts unusual stress on its proximal end, which receives the greater force of the pulse wave\textsuperscript{10}. In addition, defects in the medial layer may be present at the proximal end of an arterial fenestration as Crompton\textsuperscript{9} reported from his autopsy specimen. These findings may explain the genesis of the aneurysm in our study. Crompton\textsuperscript{9} described the histological appearance of a fenestration and noted a defect in the media at the proximal end. Such a fenestration of the MCA could give rise to an aneurysm independent of any branching. Black and Aasbacher\textsuperscript{21} studied the histological appearance of an aneurysm at the proximal end of a basilar artery fenestration. They also found a defect in the media at the proximal and distal ends of the fenestration, and an aneurysm associated with the proximal bifurcation. Yamada, et al.\textsuperscript{20} describe a medial defect at the neck of an aneurysm formed at an A1 fenestration. Arterial wall weakness, in part, provides a mechanical explanation for the high incidence of aneurysms associated with arterial fenestrations. In addition, hemodynamic factors involved in the formation of aneurysms at the site of arterial bifurcations may play a similar role at the proximal end of arterial fenestrations\textsuperscript{3}. Clinicians need to be familiar with this association between intracranial arterial fenestrations and aneurysms.
In our Case 3, we did not discover the presence of the aneurysm or fenestration from the initial brain computed tomographic angiogram or cerebral angiogram. When the angiography was repeated five days later, we observed the A2 aneurysm plus fenestration. Various angiographical projections and delayed studies are therefore necessary to prevent misdiagnosis, especially in cases where an aneurysm arises from the fenestration. Recognition of this unusual anomaly is important from a surgical point of view. Indeed, the aneurysmal sac may be difficult to differentiate from the fenestrations due to its small size, as in our case; on the other hand, a short fenestration might be obscured by an aneurysm.

When considering the surgical removal of an A2 aneurysm, it should be noted that these aneurysms most often occur at the takeoff point of small perforating arteries. This suggests that more careful attention is required during the clipping procedure. These small arteries are easily occluded during clipping and tend to close even if the clip appears to be properly positioned during the operation. Furthermore, spontaneous rotation or shifting of the clip body can cause kinking of the arteries as the brain recovers in situ, even if these arteries are not very close to the aneurysm. In view of the potential for clip movement, it seems very important to use a small clip as possible in the treatment of these delicate aneurysms.

The probability of a thrombus or embolus formation is much higher in a fenestrated artery than a normal one. Hemodynamic disturbances and turbulent blood flow at the site of fenestration may be the cause of the thrombosis that occurred in the artery. Such a thrombus or embolus may also be related to a transient ischemic attack or infarction. Fenestrations of the internal carotid artery or MCA associated with an ischemic attack have been reported. Vertebral artery fenestration after repeated ischemic stroke attacks and treatment (proximal balloon occlusion) has been described in a Korean patient. A large intraluminal thrombus was located just proximal to the fenestration in this case. However, we could find no reported case with ischemic attack due to ACA fenestration. We think that the absence of a causal relationship between ACA fenestration and transient ischemic attack may be related to the less blood flow and the smaller ACA vessel diameter, compared with the MCA.

**Conclusion**

The incidences of A2 fenestration found in this study were 0.36% (3/843) in conventional cerebral angiograms and 0.62% (11/1787) in MRA measurements. In addition to medial defects, flow patterns at the proximal ends of fenestrations, where hemodynamic stress and increased turbulence are present, may contribute to aneurysm formation. Although A2 fenestrations have little clinical significance, knowledge and recognition of these may be important in the interpretation of cerebral images and during neurosurgical procedures.

**Acknowledgement**

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Commentary

In this interesting article, the authors summarized 14 cases with fenestration in the horizontal segment of the anterior cerebral artery. In their study, fenestration was observed in the A1 segment of anterior cerebral artery in 0.36% and 0.62% of patients who underwent cerebral angiography and MRA, respectively. Vascular anomalies such as an aneurysm on the ipsilateral side were also found to be associated with fenestration in three cases. Most cases of fenestration were incidental findings except for one case with a ruptured aneurysm at the proximal end of anterior cerebral artery.

This study is of great value in reporting the incidence of the fenestration of A1 in a large series of patients. In addition, the authors discussed several embryological hypotheses for the pathogenesis and clinical consideration of proximal ACA fenestration such as the small size of an associated aneurysm, which might create difficulties in making a diagnosis, its anatomical relationship with the small perforators and the hemodynamic instability in a fenestrated artery. This highlights the importance of anatomical awareness of this variation during a radiological interpretation and surgical procedures.

However, this article did not mention a specific methodology for making a diagnosis nor did it provide a precise description of the fenestration i.e. its diameter, length, location, morphologic classification and the anatomic relationship between the lesions and perforators. The study also has a limitation in that further information could not be obtained from this article regarding the radiological differential diagnosis and the anatomic/hemodynamic correlation of the fenestrations in the proximal anterior cerebral artery coexisting with other variations such as a fenestrated anterior communicating artery, duplication, multiplication or hypoplastic A1, internal carotid-anterior communicating artery anastomosis or an accessory middle cerebral artery.

I totally agree with the authors that in order to prevent a misdiagnosis and establish desirable therapeutic plan, an aneurysm originating from the proximal end of an A1 fenestration should be detected with special attention in multiple projections. A meticulous inspection of the radiological data using a combined evaluation with three dimensional CT angiography would be helpful in detecting vascular abnormalities associated with a fenestration of the anterior cerebral artery.

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