Clinical study of enlarged infundibular dilation of the origin of the posterior communicating artery

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Although some aneurysmal dilations of the origin of the posterior communicating artery (PCoA) that are revealed on carotid angiogram are true aneurysms or preaneurysmal lesions, the authors warn that diagnosis should not be based only on the size and shape of the dilation, especially when the PCoA does not fill.

In the present retrospective study, the authors analyzed intraoperative and angiographic findings in 32 patients with 34 lesions of the PCoA that were preoperatively diagnosed as aneurysms. Only 10 of the lesions were true aneurysms (six cases) or enlarged infundibular dilations with reddish bulges (that is, preaneurysmal bulge; four cases) at the origin of the PCoA. All of the other 24 lesions, including one lesion with PCoA occlusion, were merely enlarged infundibular dilations without any wall abnormality.

A well-developed PCoA was observed only in cases of true aneurysm and dilation with reddish bulge. No other clinical or angiographic characteristics proved useful in identifying the type of lesions. These findings may be helpful to the physician in evaluating the clinical features of such lesions and in determining the diagnosis of enlarged infundibular dilation of the PCoA.

Key Words • posterior communicating artery • infundibulum • infundibular dilation • aneurysm • preaneurysmal bulge • diagnosis

FUNNEL-shaped dilations most frequently affect the origin of the posterior communicating artery (PCoA) at its junction with the internal carotid artery (ICA). This type of PCoA dilation is not a true aneurysm and has been referred to as infundibular widening,13 infundibulum,4 or infundibular dilation,8,11 when it is round or conical in shape and less than 3 mm in diameter, has a wide neck, and when the PCoA arises from its apex. However, when the dilation is greater than 3 mm in diameter or when filling of the PCoA is absent, it is difficult to distinguish angiographically whether the dilation is nonaneurysmal or a true aneurysm. In addition, it remains controversial whether this dilation may be a preaneurysmal condition.

With the advent of high-resolution magnetic resonance (MR) imaging and MR angiography, unruptured aneurysms have been identified with increasing frequency. Our policy is to recommend surgery for otherwise healthy patients with asymptomatic aneurysms greater than 3 mm in diameter, although management decisions in these instances continue to be controversial.12,14,19 Diagnostic evaluations and clinicopathological investigations of enlarged infundibular dilations of the PCoA should be more intensively pursued.

Intraoperatively, we observed 34 lesions that were angiographically visualized as aneurysmal dilations of the PCoA origin, larger than 3 mm in diameter, and not classified as infundibular widening. In this report, we present our analysis of the intraoperative and angiographic findings of these 34 lesions and discuss various diagnostic and therapeutic issues pertaining to these ambiguous PCoA dilations.

Clinical Material and Methods

From April 1983 through December 1993, 32 patients, who were admitted to the Toyama Medical and Pharmaceutical University and affiliated hospitals, underwent surgery for a total of 34 funnel-shaped dilations that were diagnosed as unruptured aneurysms of the ICA–PCoA junction. This group of patients consisted of 10 men and 22 women with a mean age of 62.5 years (range 54–73 years).

On carotid angiography, all of the lesions appeared to be greater than 3 mm in diameter and the PCoA was absent or did not extend from the apex of the dilation. The dilations appeared round, conical, or triangular. The patients’ underlying diseases were subarachnoid hemor-
rhage (SAH) caused by rupture of another aneurysm in nine cases, brain tumor in three, cerebral infarction in 15, and other disease in five cases. In the 20 cases of patients with cerebral infarction or other underlying disease, surgery was performed as a prophylactic measure.

We retrospectively analyzed the intraoperative features, angiographic findings, and therapeutic results of these cases to assess the precise nature of these lesions.

Results
Intraoperative Features of the Lesions
During surgery, six of the lesions were found to be true aneurysms of the ICA–PCoA junction. Each of these aneurysms had a wide neck and protruded in a posterolateral–inferior direction. A large PCoA was identified in all six lesions (Fig. 1). Four other lesions were found to be enlarged funnel-shaped dilations of the PCoA origin with a reddish bulge in the lateral wall that protruded slightly in a posterolateral direction. We suspected that these bulges were a preaneurysmal condition (Fig. 2). Twenty-three of the lesions were simply enlarged funnel-shaped dilations of the PCoA origin, which were associated with a small PCoA that extended from the apex. No abnormal bulge or irregularity in the wall of the PCoA origin was observed in these lesions (Fig. 3). In the remaining lesion, a marked atherosclerotic change was observed, and the portion of the PCoA adjacent to the dilated origin was occluded (Fig. 4).

On the basis of the intraoperative findings, the 34 lesions could be classified into the following types: 1) true aneurysm of the ICA–PCoA junction; 2) enlarged infundibular dilation with bulge; 3) enlarged infundibular dilation without bulge; and 4) anomalously enlarged dilation associated with PCoA occlusion (Table 1). The clinical features of the patients undergoing surgery, including age, sex, and underlying disease, showed no relationship to the type of lesion.

Angiographic Findings
On angiography the lesion with PCoA occlusion displayed an anomalously shaped dilation measuring 10 mm in diameter (Fig. 4). All of the other aneurysmal dilations were round, conical, or triangular, and ranged in size from 3.5 to 6 mm in diameter (mean diameter 4.9 mm). The angiographic characteristics of size and shape of these lesions were not evaluated preoperatively to assess the true pathological features.

Alteration in the PCoA filling pattern was the only noteworthy finding in lesions of these types. The PCoA was visualized on angiography as large (equal to or larger than the posterior cerebral artery (PCA); that is, belonging to the fetal/juvenile type), small (smaller than the PCA; belonging to the adult type), or nonfilling. In the six cases of true aneurysm and the four cases of enlarged dilation with bulge, filling of the PCoA was observed and six of these cases were associated with a large PCoA. In the 23 cases of enlarged dilation without wall abnormality, nine lesions were associated with a small PCoA and 14 with a nonfilling PCoA (Table 1).
Therapeutic Results of Surgery

The six true aneurysms (six patients) were treated uneventfully with aneurysmal neck clipping. Of the 28 nonaneurysmal lesions (26 patients), five had fairly thin walls and in those cases the PCoA was not apparent on the carotid angiograms. These five lesions were treated by clipping the PCoA origin. The remaining 23 lesions, including the four with reddish bulge, were treated with total or partial coating of the dilated PCoA. The postoperative courses of 31 of the patients were uneventful; however, one patient treated with clipping of the PCoA origin developed ipsilateral thalamic infarction. In three of the four patients with an enlarged dilation with bulge, no changes in the size or shape of the dilations were confirmed on follow-up angiograms, which were obtained approximately 1 year after surgery. Postoperative angiograms were not obtained in the other patients; however, no patients suffered from SAH during the follow-up period (range 14 months to 10 years).

Discussion

Clinical Interpretation of PCoA Infundibular Dilation

The reported incidence of PCoA infundibular widening detected on angiography or at autopsy ranges from 6% to 17%, and this incidence increases with age. Angiographic findings for this lesion are difficult to interpret, and it is sometimes misdiagnosed as an ICA–PCoA junction aneurysm. Particularly when the dilation is larger than 3 mm in diameter and no PCoA filling is observed, it can be difficult to distinguish an enlarged infundibular dilation from an aneurysm. The 34 lesions in this series did not fulfill the angiographic criteria for infundibular widening and

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<tr>
<th>Intraoperative Features</th>
<th>No. of Lesions</th>
<th>PCoA on Carotid Angiograms†</th>
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<tr>
<td></td>
<td></td>
<td>Large</td>
</tr>
<tr>
<td>true aneurysm</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>EID with reddish bulge</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>EID with PCoA occlusion</td>
<td>1</td>
<td>0</td>
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<tr>
<td>total</td>
<td>34</td>
<td>6</td>
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* EID = enlarged infundibular dilation.
† Large = fetal-type PCoA, with PCoA larger than posterior cerebral artery; small = adult-type PCoA, with PCoA smaller than posterior cerebral artery.
were surgically treated under the preoperative diagnosis of unruptured aneurysm. Operative results, however, revealed that only six of the lesions were true aneurysms. Instead, the other 28 lesions that had displayed an angiographic appearance of aneurysmal dilation were merely enlarged dilations of the PCoA origin.

A few instances of an aneurysm developing from an infundibular widening have also been reported. Epstein and coworkers3 obtained normal histological findings in cases of SAH heretofore ascribed to unknown origin. Debate continues in the literature as to whether a PCoA infundibulum causes a ruptured infundibular widening. Epstein and coworkers1 obtained normal histological findings for the vasculature of the infundibulum and concluded that this lesion is neither aneurysmal nor preaneurysmal. In contrast, Hassler and Saltzman,5,6 as well as Stehbens,15 found degenerative changes or defects in the muscular media and/or the elastica lamina of the infundibulum. These authors emphasized that this entity should be regarded as a preaneurysmal lesion.

In four of the lesions examined in the present study, red-dish bulges, suggestive of preaneurysmal change, were noted in the lateral wall of the dilated PCoA origin. We believe that an infundibular widening may often be enlarged and have a preaneurysmal bulge in part of its lateral wall. These changes may not be that unusual and their incidence may increase with age, although the incidence of transformation of a bulge into a true aneurysm is probably extremely low. At any rate, classification based simply on size and shape of this lesion may not be fruitful in discussing its clinicopathological features.

Angiographic Relationship of This Type of Lesion to the PCoA

Yaşargil20 reported that filling of the PCoA from the carotid artery was observed in approximately 60% of the ICA–PCoA junction aneurysms in his patients, although no filling was noted in the remaining 40%. He suggested that an angiographic assessment of the PCoA is not reliable in evaluating the characteristics of an aneurysm. In some studies, however, good filling of the PCoA has been found on carotid angiography in many patients with a ruptured ICA–PCoA junction aneurysm.1 In addition, a high incidence of good filling of the PCoA has been reported in patients with aneurysms that developed from an infundibular widening.7,10,16,17,21 Ebina and colleagues2 investigated the angiographic features of 67 infundibular dilations and reported that a well-developed PCoA may be one of the factors that contributes to the progression from an infundibulum to an aneurysm. Hemodynamic stress associated with the development and divergent angle of the PCoA may produce rupture or enlargement of the wall at the fragile site.

In all 10 lesions that proved to be true aneurysms or enlarged dilations with bulge the PCoA was clearly observed on carotid angiograms. In six of these cases the lesion was associated with a large fetal-type PCoA. In contrast, none of the 23 enlarged dilations was associated with a large PCoA, and filling of the PCoA was not observed in 15 of these cases. These results suggest that the development of the PCoA is an important diagnostic factor in determining whether an enlarged infundibular dilation is a true aneurysm or a preaneurysmal lesion with bulge. However, further study is required to examine this issue.

Diagnosis and Treatment of Enlarged PCoA Infundibular Dilation

In conclusion, it is difficult to determine whether an infundibular dilation of the PCoA is a true aneurysm or a preaneurysmal change, even if it is larger than 3 mm in diameter. In addition, clear follow-up data on the clinical course or angiographic changes of these lesions have not been presented until now. However, we believe that the possibility that the lesion is a true pathological lesion is great when a large PCoA is visualized on carotid angiography, and we would like to support aggressive surgery for the prophylactic treatment of these lesions. In contrast, the diagnosis of aneurysm should not be based simply on the size and shape of the dilation, especially in cases in which the PCoA does not fill or appears very small on carotid angiograms. Patients with these findings should be carefully monitored using angiography.

As to the surgical treatment selected in our patients, we regret having used clipping of the dilated origin of a small PCoA because it was not only risky but unnecessary. A sufficient coating procedure should be pursued as prophylactic treatment for an enlarged dilation with or without bulge, regardless of the angiographic and intraoperative findings, to ensure the development of the PCoA.

References


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Enlarged PCoA infundibular dilation


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