An Aneurysm Developing on the Infundibulum of Posterior Communicating Artery: Case Report and Literature Review

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Infundibular dilatation is funnel-shaped symmetrical enlargement that occurs at the origin of cerebral arteries and which is apparent on 7 to 25% of normal angiograms (9,10,12,13). Infundibular dilatation is frequently considered a normal anatomic variation of no pathologic significance. The authors report a case in which an aneurysm developed on an infundibular dilatation of the posterior communicating artery (PComA). A 72-year-old woman presented with severe headache, nausea, and vomiting. Digital subtraction angiography showed a saccular aneurysm arising from the origin of the left PComA. Operative findings revealed the aneurysm and infundibular widening of the right PComA. The aneurysm was successfully obliterated. Whether infundibular dilatation is a pre-aneurysmal state or a benign dilatation is controversial. However, we believe infundibular dilatation of the PComA in this case may have served as a pre-aneurysmal lesion.

KEY WORDS: Aneurysm · Infundibular dilatation · Posterior communicating artery

Introduction

Funnel-shaped dilatations most frequently affect the origins of the posterior communicating arteries (PComA) at their junctions with the internal carotid artery. This type of PComA dilatation is not a true aneurysm and has been referred to as an infundibular dilatation, a junctional dilatation, infundibular widening, or as infundibulum (9,10,12,13). It remains controversial whether this dilatation represents a preaneurysmal condition, and demonstrations of an aneurysm formed from it is rare (9,10,12,13,14,16). Only twelve such cases have been reported to the best of the author's knowledge (Table 1).

Case Report

A 72-year-old woman with hypertension and diabetes mellitus(DM) II was transferred to our department with a history of a sudden onset headache, nausea, and vomiting. She had no neurological symptoms or signs except nuchal rigidity. Computerized tomography(CT) showed a thick layer of subarachnoid hemorrhage in the basal cistern and Sylvian fissure, which was more prominent on the right (Fig. 1). Three-dimensional computed angiography(CTA) and cerebral angiography revealed an aneurysm with an or-

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- : Bilateral infundibula, - : Unknown

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igin at the right PComA (Fig. 2). The patient was operated on the day after admission using a right prefrontal approach. During the operation, we found a true aneurysm on the posterior-lateral wall of the PComA infundibulum. Marked atherosclerotic change was observed in the infundibulum and lateral wall of the internal carotid artery (ICA) (Fig. 3), and thus we clipped the aneurysm neck and wrapped the atherosclerotic infundibulum. Postoperative course was uneventful. The postoperative angiogram revealed obliteration of the aneurysm and a patent posterior communicating artery (Fig. 4).

**Discussion**

Most so-called 'true' PComA aneurysms are arising on the junction of the internal carotid artery and PComA unrelate with internal carotid artery or posterior cerebral artery. However, aneurysms originating directly from the PComA itself do not appear to attract much attention. The incidence of this 'true' PComA aneurysm varies from 0 to 3.3%.

The aneurysms developing on the infundibulum of the PComA is the verified case of 'true' PComA aneurysm.

Infundibular dilatation is defined as a funnel-shaped dilatation of the posterior communicating artery where the artery leaves the carotid siphon and the PComA emerges from the apex of the enlargement. The reported incidence of PComA infundibular dilatation detected by angiography ranges 6% to 17%, and this increases with age. The dilatation is roughly triangular and its diameter is no greater than 3mm. Although infundibular dilatation is a well-established angiographic diagnosis and is generally believed to be benign, it is sometimes difficult to differentiate angiographically an aneurysm from the PComA infundibulum.

Okuma et al., in their interesting angiographic study on infundibular dilatation of the PComA, pointed out that infundibular dilatations show a significant tendency to develop into aneurysms. They found that the incidence of infundibular dilatation was higher and the size of the infundibulum was larger in patients with PComA aneurysms. They also demonstrated that the incidence of a large infundibular dilatation was higher in young patients and in patients with a past history of hypertension.

There is still some controversy about the exact history and natural history of infundibular dilatation. Some histological studies that investigated the relationship between infundibular dilatation and aneurysms have furnished contradictory evidence. Hassler and Salzman looked at the histologies of 21 cases of infundibular dilatation and found that a significant

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**Fig. 1.** Computed tomographic scan on admission. High density is present in the subarachnoid space bilaterally, and is slightly more prominent on the right.

**Fig. 2.** Three-dimensional computed angiogram demonstrating an aneurysm (arrowhead) arising from the dilated infundibular portion of the posterior communicating artery (arrow).

**Fig. 3.** Operative findings demonstrating a true aneurysm on the posterolateral wall of the posterior communicating artery infundibulum (asterisk) with a reddish bulge at its origin (A). Marked atherosclerotic change is observed in the infundibulum and the lateral wall of the internal carotid artery (arrowhead: PComA).
proportion demonstrated typical aneurysmal defects in the internal elastic lamina and in the medial. However, similar studies by Ebstein et al disclosed no abnormality, and lead them to conclude that the condition is neither aneurysmal nor pre-aneurysmal.

Patrick et al compared the site distribution of aneurysms in familial aneurysm cases in the general population. Family cases showed a 30% incidence of PComA aneurysm and a 20% incidence of infundibular dilatation of the PComA, and supporting the hypothesis that infundibulum is indeed pre-aneurysmal.

Based on intra-operative findings, Endo et al reported atypical bulging at infundibular dilatations, especially in regions of the lateral wall, strongly suggestive of early aneurysm progression. They reported that a well-developed PComA may be a factor that contributes to progression from an infundibulum to a true aneurysm. They explained that hemodynamic stress at the PComA may contribute to such progression.

Several instances of aneurysms developing from infundibular dilatations have been reported, and the incidence of progression to aneurysm is greater in cases of multiple aneurysms, bilateral infundibular dilatations, and familial intracranial aneurysms.

Thus, it seems likely that infundibular dilatation is a pre-aneurysmal condition. There is a certain increased incidence of de novo aneurysm formation in patients with infundibular dilatations, i.e., in patients who have bilateral infundibula, large infundibula, multiple aneurysms, familial intracranial aneurysms, and who are young. Conceivably, hypertension, atherosclerosis at the infundibulum and an enlarged PComA would contribute to hemodynamic stress and repeated vessel wall distension, and could lead to aneurysm formation.

Conclusion

We present the case of a 72-year-old woman in whom an aneurysm developed at the site of an infundibular dilatation of the left PComA. Although infundibular dilatation may be frequent findings, and its incidence may increase with age, the incidence of the transformation of a bulge into a true aneurysm is probably extremely low. We believe that infundibular widening may be enlarged, and that such widening reflects a pre-aneurysmal tendency in patients with risk factors; such as bilateral infundibula, large infundibula, history of multiple aneurysms, a familial history of intracranial aneurysms, young age, hypertension, atherosclerosis, and an enlarged PComA.

References