Radiographic and histologic demonstration of an aneurysm developing on the infundibulum of the posterior communicating artery

Case report

J. Timothy Stuntz, M.D., George A. Ojemann, M.D., and Ellsworth C. Alvord, Jr., M.D.
Departments of Neurological Surgery and Pathology (Division of Neuropathology), University of Washington School of Medicine, Seattle, Washington

In this case an aneurysm of the posterior communicating artery developed 9 years after an infundibular dilatation of this posterior communicating artery, without aneurysm formation, had been radiographically demonstrated. Histological examination, after a fatal subarachnoid hemorrhage from the aneurysm, demonstrated that it arose exactly at the site of narrowing of the funnel-shaped infundibulum.

Key Words: intracranial aneurysm · posterior communicating artery · infundibulum · carotid ligation

Infundibular dilatation of the posterior communicating artery has been considered a "pre-aneurysmal" lesion by some authors. This belief rests on the increasing incidence of infundibular widening with age and on the histological demonstration of changes in the infundibulum similar to those characteristic of saccular aneurysms. However, the actual development of an aneurysm on a previously demonstrated infundibulum has rarely been reported. Drake presented one such case, documented by serial angiography, and mentioned that there was another in his series. This report concerns another such case, including histological examination of the aneurysm.

Case Report
A 38-year-old man was well and working when he suddenly vomited and became comatose. Nine years earlier the patient had suffered a subarachnoid hemorrhage from a right internal carotid artery aneurysm which had been treated with the placement of a Selverstone clamp on the right common carotid artery (Fig. 1 A). At that time no aneurysm had been seen on the left internal carotid circulation, but infundibular dilatation of the left posterior communicating artery had been demonstrated (Fig. 1 B).

Examination. Upon admission the patient was completely unresponsive, without localizing signs. Lumbar puncture revealed bloody cerebrospinal fluid. Within a week the patient spontaneously opened his eyes but remained an akinetic mute.

Course. A retrograde right vertebral angiogram revealed a large right vertebral artery with a large muscular branch that communi-
cated with the occipital branch of the right external carotid artery. There was retrograde flow of a large volume of contrast material into the right common carotid artery just distal to the point of clamping and thence into the internal carotid artery. There was partial filling of the previously demonstrated aneurysm (Fig. 1 C).

One month later the patient was transferred to Seattle Veterans' Administration Hospital where he was neurologically unchanged except for slight movement of the right arm in response to painful stimulus. A left percutaneous carotid angiogram revealed a 9-mm aneurysm of the left internal carotid artery proximal to its bifurcation into the anterior and middle cerebral arteries just at the point where infundibular dilatation had been seen 9 years previously. There was spasm of the internal carotid artery for several millimeters proximal and distal to the aneurysm (Fig. 1 D).
Aneurysm of the infundibulum of the PCA

The electroencephalogram showed intermittent delta activity in the left anterior temporal derivations. Communicating hydrocephalus with normal cerebrospinal fluid pressure was demonstrated by RISA cisternogram and pneumoencephalography and a ventriculoatrial shunt was performed. Nine weeks after his initial symptoms he suddenly became unresponsive with fixed midposition pupils and loss of oculocephaline reflexes. A flaccid right hemiplegia was present. Lumbar puncture revealed bloody cerebrospinal fluid with an opening pressure of over 600 mm of water. The patient remained unresponsive and died a few days later.

*Postmortem Examination.* Autopsy disclosed an unruptured 4-mm aneurysm at the junction of the right internal carotid artery and the right posterior communicating artery (this was the side on which the Selverstone clamp had been placed 9 years previously). Another unruptured minute aneurysm was also found at the junction of the right peri-callosal artery and the anterior communicating artery. A ruptured 9-mm aneurysm arose from the junction of the left internal carotid artery and left posterior communicating artery. A large left temporal lobe hematoma was present with transtentorial herniation and secondary brain stem hemorrhages. In addition, there was extensive pseudolaminar necrosis of the right cerebral hemisphere in the distribution of the right middle and anterior cerebral arteries. Moderate communicating hydrocephalus was present.

Serial microscopic sections of the left internal carotid artery aneurysm revealed it to arise on the infundibulum, 4 mm from the carotid artery itself, exactly at the narrowing of the infundibulum to form the posterior communicating artery (Fig. 2). The infundibulum of the posterior communicating artery could be identified proximal to the neck of the aneurysm both by its funnel shape and especially by superimposition of the serial sections identifying the level of the take-off of this branch. The internal elastic lamina of the infundibulum ended about 2 mm proximal to the neck of the aneurysm. Two smaller breaks in the elastica were present opposite the neck of the aneurysm. The aneurysm consisted of a thin layer of fibrous tissue to which a small amount of fibromuscular cushion was added at the neck. Rupture had occurred at the dome. There was no atherosclerosis within the aneurysm, but there was slight intimal fibrosis of the carotid artery and infundibulum. This fibrosis was more extensive and concentric in the posterior communicating artery throughout the 10 mm that were included in the sections.

Serial microscopic sections of the right internal carotid artery aneurysm revealed its origin to be at the distal angle of the internal carotid and posterior communicating arteries. No funnel-shaped dilatation of the origin of the right posterior communicating artery could be identified.

*Discussion*

This case demonstrates the formation of an internal carotid aneurysm subsequent to
infundibular dilatation of the posterior communicating artery. Saltzman reported that infundibular dilatation of the posterior communicating artery occurs in approximately 7% of angiograms and that the incidence increases with age. Hassler and Saltzman have described several cases in which histological changes in the infundibulum of the posterior communicating artery are similar to those found in aneurysms: defects in the media, splitting of the internal elastic lamina, and in one case entire absence of the internal elastic lamina. Taveras and Wood mentioned another case that appeared to confirm these findings, but no details were given. Differentiation of aneurysm from infundibular dilatation of the posterior communicating artery remains a difficult radiological problem.

Cerebral aneurysms typically occur at the apex of bifurcations. There Forbus and Carmichael demonstrated medial defects and believed that these "congenital" malformations predisposed to aneurysm formation. However, Glynn found medial defects in 80% of the bifurcations examined, an incidence far too high to permit implications of their etiological role in aneurysms. He demonstrated experimentally that the unsupported internal elastic lamina could withstand pressures of 600 mm Hg without bulging. He concluded that the medial defect plays no part in the development of an aneurysm and that lesions of the elastic layer are of greater significance. Just what these elastic changes are remains unclear. Hassler by observing pitting of silicone-lined plexiglass models of bifurcations concluded that the pitting, which represented the site of maximal hydrodynamic stress, corresponded to the sites of the medial defects. He suggested that the medial defects occurred first and these allowed elastica defects to develop from overstretching. Thus, medial defects, elastica defects, and aneurysms all occur at sites subjected to the greatest strain. Such hemodynamic factors may also account for the development of an aneurysm contralateral to the ligation of one carotid artery, as in the treatment of one aneurysm or in experiments on rabbits. If, indeed, infundibular widening of the posterior communicating artery is a "pre-aneurysmal" lesion, then contralateral carotid ligation may increase the risk of formation of a second aneurysm at that site by increasing the blood flow. Several small breaks in the internal elastica were found in the infundibulum, probably due to atherosclerosis, which may also have contributed to the production of the aneurysm. Carotid ligation was not involved in Drake's case of an aneurysm arising from an infundibulum.

Multiple aneurysms, as demonstrated here, occur in 10% to 20% of all cases of intracranial aneurysms. In one series of 17 multiple aneurysms, 42% were located bilaterally symmetrically. In a patient with one internal carotid artery aneurysm, the most probable site of the second aneurysm is the opposite internal carotid artery. Minute aneurysms measuring less than 2 mm in maximum diameter, such as that seen at the junction of the right pericallosal artery and the anterior communicating artery in this case, have also been well described, appear to be very common, and may enlarge to form larger aneurysms.

All of the above points are pertinent to our case: the initial bleeding from a typical saccular aneurysm arising at the apex of the bifurcation of the right posterior communicating and internal carotid arteries, and the subsequent formation of another aneurysm arising almost symmetrically at the point of narrowing of the infundibulum of the left posterior communicating artery contralateral to the previous ligation of the right common carotid artery. Drake's case similarly showed an initial internal carotid posterior communicating aneurysm, with a contralateral aneurysm arising in a symmetrical location from an infundibulum years later.

The partial thrombosis of the old right carotid aneurysm is interesting. This may have occurred spontaneously, but it would have been favored by the right common carotid artery ligation if subsequent to ligation there were at some time decreased blood flow or blood pressure at the aneurysmal orifice. Unfortunately we have no measurements of blood flow or blood pressure, either initially after ligation or later. However, the right retrograde vertebral angiogram 9 years after the carotid ligation demonstrated large collaterals and suggested that at that time there was brisk blood flow past the aneurysmal orifice. Despite this, the aneurysm was partially
Aneurysm of the infundibulum of the PCA

thrombosed and had not rebled. Such collateral channels around carotid occlusions have been well described by Fields, et al., and Somach and Shenkin.1,7

Summary

We have presented the case of a 38-year-old man in whom infundibular dilatation of the left posterior communicating artery had been demonstrated radiographically 9 years prior to a fatal subarachnoid hemorrhage from an aneurysm proven histologically to arise at exactly the site of narrowing of this funnel-shaped infundibulum. It has been suggested that infundibular widening is a "pre-aneurysmal" lesion, but documentation of formation of an aneurysm from an infundibulum has been rare. In this case, carotid ligation contralateral to the infundibular dilatation of the posterior communicating artery may have predisposed the formation of an aneurysm at that site.

References


Received for publication December 31, 1969.
Supported in part by U.S. Public Health Service grant NB 05211 and NB 05231.
Address reprint requests to: George A. Ojemann, M.D., Department of Neurological Surgery, University of Washington School of Medicine, Seattle, Washington 98105.