# Aneurysm of the Posterior Cerebral Artery with Unexpected Postoperative Neurological Deficit

## Case Report

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ongenital intracranial aneurysms of the posterior cerebral artery trunk are rare. We have seen but one case in our series of more than 300 aneurysms. The Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage has reported seven single posterior cerebral maintrunk aneurysms in 2,672 single aneurysm cases,<sup>3</sup> an occurrence of 0.26%. We know of no reports of obliterating such aneurysms by trapping.

This is the report of an aneurysm of the posterior cerebral artery trunk that was treated successfully by obliterating the parent vessel with very little permanent neurological deficit.

### Anatomy

Normally, the posterior cerebral arteries are the paired continuations of the basilar artery beyond its distal bifurcation. Each artery then passes laterally around the cerebral peduncle to the posterolateral aspect of the midbrain.4 Early in its course, it gives origin to the thalamogeniculate arteries and to the medial and lateral posterior choroidal arteries. The posterior cerebral is then contained in the tentorial incisura until it passes above the tentorium to the undersurface of the temporal lobe. There it divides into two terminal branches, the posterior temporal, and the internal occipital.<sup>4</sup> A variable and small anterior temporal branch supplies a portion of the anterior temporal lobe. The posterior temporal artery supplies the undersurface of the posterior temporal lobe (fusiform and lingual gyri). The internal occipital branch forms the calcarine and parieto-occipital arteries that supply the medial aspect of the occipital lobe and the precuneus.4 Some authors state that the cuneus and the splenium of the corpus callosum are fed by these parieto-occipital arteries. Branches of the posterior cerebral artery also supply the ependymal lining of the lateral ventricle, with the exception of its horns, and the caudal part of the choroid plexus.<sup>2</sup>

### Case Report

Approximately 1 year before admission, this 33-year-old white woman consulted a neurologist because of peculiar sensations of pain involving the left side of the body, except the face. She had no objective neurological deficit, and no further investigation was made. Four weeks before admission she had a sudden headache. There was no loss of consciousness, seizure, or sensory or visual disturbance. Fourteen days later, because the headache persisted, a lumbar puncture was done. The initial pressure was 139 mm of spinal fluid. The fluid was xanthochromic but contained no red cells. The total protein was 88 mg%.

Examination. Upon admission to the University Hospital on March 27, 1964, she was normally developed but thin. She was drowsy and appeared chronically ill. The pulse and respirations were 80 and 20 per minute respectively, the blood pressure 104/52 mm Hg, and the temperature 100°F. Aside from mild irritability, no neurological or physical abnormalities were noted. Visual fields were examined only by confrontation. A left transaxillary, right retrograde vertebral arteriogram demonstrated a large saccular aneurysm of the posterior cerebral artery trunk (Fig. 1).

Operation. Three days later, through a right temporoparieto-occipital craniotomy, the aneurysm was found with its dome buried within the transverse cerebral fissure and the infero-medial aspect of the temporal lobe. The pulvinar was not identified. The parent vessel was clipped both distally and proximally to the sac. The aneurysm was then aspirated and did not refill.

Postoperative Course. In the immediate

Received for publication October 31, 1966.

postoperative period, there was a right third nerve paralysis and minimal weakness of the left arm and leg. At 48 hours, although she did not have a demonstrable left homonymous hemianopsia, her visual attention and acuity seemed to be decreased in the left homonymous fields.

At 72 hours, the weakness of the left arm and leg was more pronounced and she appeared slightly more drowsy, but again no clear-cut hemianopsia was detected. In response to testing she reported a marked decrease in her awareness of the left side of her body. A very mild receptive dysphasia was present. Her ability to read could not be tested. Abdominal reflexes were absent on the left.

At 120 hours, there was no evidence of left hemianopsia, although the left hemiparesis was still manifest. She was mentally clear enough to deny the existence of the left side of her body.

By 10 days there had been a gradual return of the left body image, and the hemiparesis was almost undetectable.

On the 23rd postoperative day, there was still minimal disturbance of the left body image, an occasional tendency to fall to the left, and a right third nerve palsy. A repeat left transbrachial arteriogram showed no evidence of the aneurysm (Fig. 2). Twenty-seven days after surgery, the anterior cir-

culation was demonstrated to be free of aneurysm.

At the time of her discharge (36th postoperative day), she exhibited an improving right third nerve palsy, minimal left hemiparesis, and minimal impairment of the body image on the left.

Subsequently, the third nerve palsy and the sensorimotor hemiparesis recovered completely. Visual fields were plotted and were normal. The most persistent residual deficit was a severe disturbance in spelling which kept her from her job as a publisher's secretary for 14 months.

#### Discussion

The ligation of the posterior cerebral artery in this instance was deemed necessary as a life-saving measure. We fully expected a stormy postoperative course with a permanent left homonymous hemianopsia.

Most physicians describe severe transient sensorimotor deficit with permanent homonymous hemianopsia after occlusion of the posterior cerebral artery.<sup>2</sup> Thalamic softening with dysesthesia of the contralateral body and dysynergia due to dentato-rubrothalamic involvement are attributed to infarctions in the distribution of the thalamogeniculate arteries.<sup>1</sup> The syndrome of the retrolenticular capsule (hemiplegia, hemianesthesia, and hemianopsia) is also attrib-

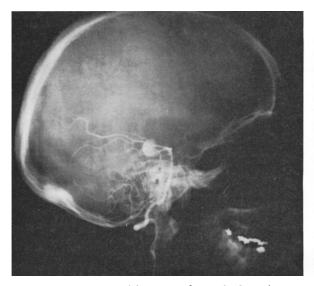




Fig. 1. Left transaxillary, right retrograde vertebral arteriograms demonstrating right main trunk posterior cerebral artery aneurysm.

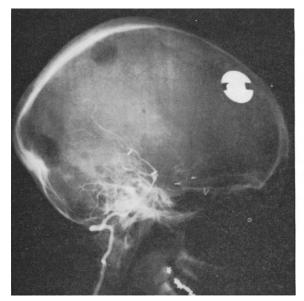




Fig. 2. Postoperative vertebral arteriograms showing non-filling of the aneurysm sac. Although it cannot be seen in the photograph, contrast media appeared in the distribution of the medial posterior choroidal artery.

uted to occlusion of branches of the posterior cerebral artery. The good recovery of function, except for hemianopsia sparing the macula, is ascribed to good collateral circulation.<sup>2</sup>

Presumably, collateral circulation for the internal occipital artery was, in this instance, adequate. The disturbance in body image must have reflected ischemia in the deep parietal lobe and the retrolenticular capsule. The gnostic disturbance with the peculiar defect in spelling remains unexplained, unless mixed dominance was present, or unless ischemia of the splenium of the corpus callosum could play some role in this complex syndrome.

It is noteworthy that the deficit was transient and that the risk of occluding the posterior cerebral artery was justified in this case. The postoperative angiogram showed the occlusion to be distal to both the posterolateral perforators (thalamogeniculate arteries) and the medial posterior choroidal artery (Fig. 2). The lateral posterior choroidal arteries were probably sacrificed. The deficit might have been much more severe had the parent trunk been occluded closer to its origin.

### Summary

We have reported a case of aneurysm of the main trunk of the posterior cerebral artery successfully treated by occlusion of that vessel. We have described the postoperative deficit which was less severe than anticipated and did not include homonymous hemianopsia.

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