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BACKGROUND AND PURPOSE: **P2 segment aneurysms develop between the junction of the posterior communicating artery with the posterior cerebral artery (PCA) and the posterior part of the midbrain in the ambient cistern. We reviewed our experience with parent artery occlusion in such aneurysms, looking for predictors of safety and effectiveness.**

METHODS: **Clinical and preprocedural data from 10 patients, referred for endovascular treatment of P2 segment aneurysms, were retrospectively studied for prognostic factors influencing postoperative neurologic deficits caused by ischemia of the PCA distal territory. Patient tolerance was assessed by using clinical or anatomic criteria. Embryologic and anatomic features of the PCA were reviewed.**

RESULTS: **Endovascular parent artery occlusion at the level of the aneurysmal neck was possible in nine cases. Control angiography after embolization showed that the aneurysm did not fill, and the distal PCA refilled via leptomeningeal anastomoses. One asymptomatic aneurysm could not be catheterized because of vascular tortuosity. No neurologic deficit occurred after treatment. Clinical presentations and grades were typical. No embryologic or anatomic configuration (eg, basilar tip arrangement, P2 position relative to the choroidal fissure, aneurysmal size or type [berry, fusiform, or serpentine]) was predictive of bad outcomes.**

CONCLUSION: **Acute parent artery occlusion appears to be safe in the treatment of P2 segment aneurysms, whatever the location of the occlusion. In our series, potential collateral supply and hemodynamic balance between the anterior and posterior choroidal arteries, pericallosal vessels, and anterior and middle cerebral vessels to the distal PCA made P2 occlusion safe, because the aneurysm occurred after the thalamoperforating vessels arose from the P1 segment.**

Aneurysms of the posterior cerebral artery (PCA) are rare, accounting for less than 1% of all intracranial aneurysms (1) and usually are reported along with vertebrobasilar or posterior circulation aneurysms (2, 3), although they do not have the same prognosis, especially when one considers basilar tip aneurysms. Only a few surgical reports discuss PCA aneurysms as a distinct subgroup (4–7); one article (8) on endovascular treatment has been published. Among PCA aneurysms, P2 segment aneurysms arise between the junction of the posterior communicating artery (PCoA) with the PCA and the posterior part of the midbrain. These aneurysms have been reported mostly as single cases (9), except in one article (10) that specifically addressed the different surgical approaches for P2 segment aneurysms.

P2 segment aneurysms may be difficult to reach during surgical procedures. The potential for morbidity and mortality is considerable with surgery, because the perforating branches are closely related to the cerebral peduncle. In addition, resection of the overlying parahippocampal gyrus is sometimes necessary. In the surgical series of Terasaka et al (10), four of 14 patients had a disability after surgery. Compared with surgical clip placement, endovascular treatment may have notable advantages in this subset of P2 segment aneurysms. Our purpose was to evaluate P2 segment aneurysms as a specific entity in terms of their clinical presentation, embryologic and anatomic considerations, and endovascular treatment. We retrospectively reviewed our experience in the treatment of a

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From the Neurosurgery Department, University Hospital Dupuytren, Limoges (P.H.) and the Department of Interventional Neuroradiology, Fondation A de Rothschild, Paris, France (M.P., J.M.).

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Address reprint requests to Paul Hallacq, MD, DSc, Neurosurgery Department, University Hospital Dupuytren, 2, Avenue Martin Luther King, 87042 Limoges, France.

series of 10 P2 segment aneurysms selected for endovascular therapy. We studied the clinical presentation, imaging studies, endovascular management, outcomes, and angiographic follow-up findings.

Methods

Patients

Between 1990 and 2000, 10 patients were referred to our institution for endovascular evaluation. Each had a P2 segment aneurysm. Five patients were female and five male, with an age range of 18–70 years and mean age of 43 years.

Clinical manifestations were the following: clinical history of subarachnoid hemorrhage (two cases, one accompanied by diplopia and one accompanied by a documented intracerebral hematoma), headaches (five cases), headaches and left lower quadrantanopia leading to complete left homonymous hemianopia (one case), headaches and phosphenes (one case), and Gerstmann syndrome and hand motor deficit (one case). Clinical grading at admission, according to the grade 0–V scale of Hunt and Kosmick (11), revealed a grade of 0 in eight patients and a grade of I in two patients. Symptoms were related to the aneurysm after the diagnosis was made. This diagnosis was sometimes suspected on the basis of either CT results or MR imaging findings because of the large or giant size of the lesions. The diagnosis was confirmed by means of conventional angiography in all cases. Five giant and five large aneurysms were diagnosed; they were located on the right side in four cases, and on the left side in six cases. One serpentine, four fusiform, and five berry aneurysms were noted. Five aneurysms were partially thrombosed (two fusiform, two berry, one serpentine). The characteristics of the patients and the aneurysms are summarized in the Table.

Endovascular Procedure

All procedures but one were performed with the patient under general endotracheal anesthesia. Femoral access was obtained by means of single-wall puncture with a 6F vascular sheath. A contralateral femoral puncture was used when a balloon test occlusion was performed. The procedures were performed with the administration of heparin. The patients received a heparin bolus of 5000 IU, followed by hourly bolus injections of 3000 IU. Heparin (5000 IU/L) was also administered in the flushing solutions. A 6F guiding catheter was advanced in the vertebral artery. Diagnostic angiograms were obtained to determine the optimal projections for viewing the neck of the aneurysm and for making baseline observations of the structures, especially leptomeningeal anastomoses.

Test occlusion was not performed in all cases. When the planned occlusion involved the distal P2 segment, no test was used (n = 4). Otherwise, test occlusion was performed by placing a balloon microcatheter in the parent vessel and then inflating the balloon over 30 minutes (n = 5). The patient's tolerance for the parent vessel occlusion was tested once by awakening the patient (n = 1). A staff neuroradiologist not involved in the embolization procedure performed a neurologic examination, looking for visual field defect and sensorimotor deficits in the fingers and both the upper and lower extremities. Testing was repeated three times over a half-hour. The patients' blood pressures was maintained at their normal level, and no testing other than clinical examination was performed. Control angiography was performed during balloon inflation, just before the patient was awakened, and repeated after 15 minutes and after 30 minutes of balloon occlusion. Vertebral and carotid injections were used each time, with the acquisition of late views, to evaluate the anatomy of the leptomeningeal collateral supply distal to the P2 occlusion, which arose mainly from choroidal and middle cerebral arteries on the same side. This anatomic evaluation was performed in the remaining four

Summary of data in 10 patients with P2 segment aneurysms

Patient No./Sex/ Age (y)	Aneurysm Type, Side, and Size	Presenting Symptom	Test	Treatment Type and Year	Clinical Outcome and Follow-Up Findings
1/43/M	Saccular, R, giant thrombosis	Headaches, phosphenes	Balloon occlusion	Balloon, 1990	Excellent, no headaches, phosphenes gone; at 11-mo angiography, no recanalization; MR imaging at 6 y, two-thirds reduction in aneurysmal mass
2/53/F	Saccular, R, giant thrombosis	Headaches, LHH	Balloon occlusion, clinical examination	Balloon, 1994	LHH, no headaches; at 6-mo angiography, no recanalization
3/20/M	Fusiform, L, large	Headaches	Angiography	GDC, 1996	Excellent, no headaches; at 1-y angiography, no recanalization
4/60/M	Fusiform, L, large	Headaches	Balloon occlusion	GDC, 1998	Excellent, no headaches; at 1-y angiography, no recanalization
5/49/F	Fusiform, L, giant thrombosis	Gertsmann syndrome	Angiography	GDC, 1998	Excellent, no deficit; at 1-y angiography, no recanalization
6/18/F	Saccular, L, large	SAH, hematoma	Angiography	Histoacryl, 1998	Excellent, no deficit; at 1-y angiography, no recanalization
7/49/F	Saccular, L, large	Headaches	Balloon occlusion	GDC, 1999	Excellent, no headaches; at 1-y angiography, no recanalization
8/47/M	Fusiform, L, giant thrombosis	SAH, diplopia	Balloon occlusion	GDC, 1999	Excellent, no deficit; at 1-y angiography, no recanalization
9/26/M	Serpentine, R, giant thrombosis	Headaches	Angiography	GDC, 1999	Excellent, no headaches; at 1-y angiography, no recanalization
10/70/F	Saccular, R, large	Headaches	None	None	Well 3 y after attempt

Note.—LHH indicates lateral homonymous hemianopsia; SAH, subarachnoid hemorrhage.

patients who were not awakened from their anesthesia (n = 4). The leptomeningeal collateral supply was judged adequate when the distal PCA territory was supplied, as determined with control carotid injection on early and late views.

When the test occlusion did not reveal any deficit or when the leptomeningeal anastomotic network was judged adequate, a microcatheter (Tracker-10; Target Therapeutics, Fremont, CA) was advanced through the guiding catheter and placed in the P2 segment. Nine of 10 P2 segment aneurysms were treated by endovascular means. Depending on the availability of embolic material at treatment, the parent vessel and aneurysmal neck were occluded by electrolytically detachable coils with a mixture of one-third histoacryl glue (n = 6) and two-thirds lipiodol (n = 1). Alternatively, occlusion was achieved by placing a detachable balloon in a proximal location in the artery and covering the neck of the aneurysm (n = 2).

After the endovascular procedure was completed, biplane views were obtained to detect any lack of filling in any of the branches. The patients were then awakened from the anesthesia, neurologically tested (visual field and sensorimotor testing of four limbs), and transferred to the intensive care unit. The heparin treatment was not reversed. Anticoagulation was maintained at twice the control level during 2 days; then, subcutaneous low-molecular-weight heparin was administered for 5 days. In every patient, clinical and angiographic follow-up was subsequently performed between 6 months and 1 year. Delayed control angiography was performed later. One patient underwent control MR imaging 6 years after treatment.

Results

Clinical Results

Endovascular parent artery and aneurysm occlusion was performed in nine cases. One 70-year-old woman was not treated because of tortuosity of the cervical vessels, and no further intervention was attempted in this patient. The patient was doing well without any neurologic deficit 3 years after treatment.

The nine other patients tolerated the parent artery occlusion well and had good neurologic recovery. Headaches resolved in all cases, and diplopia and phosphenes present in two patients resolved. No immediate postoperative complication occurred in any case. No delayed (at 1 year) deficit was associated with the procedure. No permanent complication related to the procedure was noted. One patient continued to have a homonymous visual field defect, which was present before the occlusion procedure was performed.

Clinical presentations did not differ from those usually reported for aneurysms in this location. Signs and symptoms included subarachnoid hemorrhage, mass effect, and visual field defects. Clinical grades, grades 0 and I, had the same meaning as they would in aneurysms at other locations.

Angiographic Results

In all cases, final angiograms obtained immediately after treatment showed occlusion of the aneurysm and retrograde filling of the distal PCA by leptomeningeal anastomoses. On follow-up angiograms obtained 6–12 months after the embolization, occlusion of the parent artery remained stable; no aneurysm refilled by means of a retrograde or leptomeningeal collateral supply.

No particular embryologic or anatomic configuration was predictive of the clinical result. In particular, the type of basilar tip arrangement, the position of P2 relative to the choroidal fissure, and the type of aneurysm (saccular, serpentine, or fusiform) were not predictors.

Illustrative Cases

Case 3.—A 20-year-old man had headaches lasting for a few months. His medical history was unremarkable. Findings from a neurologic examination were normal. CT depicted a partially thrombosed aneurysm in the left cistern. Angiography revealed a large fusiform aneurysm of the left P2 segment, with separate entry and exit points. Electrolytically detachable coils were placed through a microcatheter (Tracker; Target Therapeutics) inside the aneurysmal lumen without prior test occlusion. Control angiograms showed selective occlusion of the aneurysm and parent vessel. The leptomeningeal network from the anterior circulation supplied the distal PCA territory. The internal and middle temporal trunks served as an aspiration siphon for distal revascularization (Fig 1).

Case 7.—A 49-year-old woman complained of headaches lasting for 2 years, sometimes associated with nausea and photophobia. Findings from a neurologic examination were normal. CT, MR imaging, and angiography showed a large partially thrombosed aneurysm of the P2 segment on the left side. The circulating portion of the aneurysm was estimated to be 1 cm. Under general anesthesia, a bifemoral puncture was made, and a balloon test occlusion was performed with a number 1 nondetachable balloon inflated just proximal to the neck of the aneurysm. By using vertebral and left carotid artery injections and with the patient asleep, the leptomeningeal supply to the distal PCA was assessed and judged to be adequate on the left side. The balloon was deflated. A microcatheter (Tracker-10; Target Therapeutics) was advanced in the aneurysm, and Guglielmi detachable coils (GDCs; one 8×30, two 6×20, two 5×15, one 2×8, one 2×6) were detached. Postembolization assessment showed complete occlusion of the aneurysmal sac, along with the parent P2 segment. The distal territory of the PCA was vascularized by a leptomeningeal supply (Fig 2).

Case 9.—A 26-year-old man presented with sudden headaches and nausea. CT did not reveal any subarachnoid hemorrhage. Angiography showed a partially thrombosed giant serpentine aneurysm of the right P2 segment. Findings from a neurologic examination were normal. Under general anesthesia, a right femoral puncture was performed, and a microcatheter (Tracker-10; Target Therapeutics) was advanced in the aneurysm. GDC coils were detached from the center of the aneurysm to its origin in the parent P2 segment. Control angiography showed complete occlusion of the aneurysm and parent vessel. The distal PCA territory was fed by a leptomeningeal supply (Fig 3).

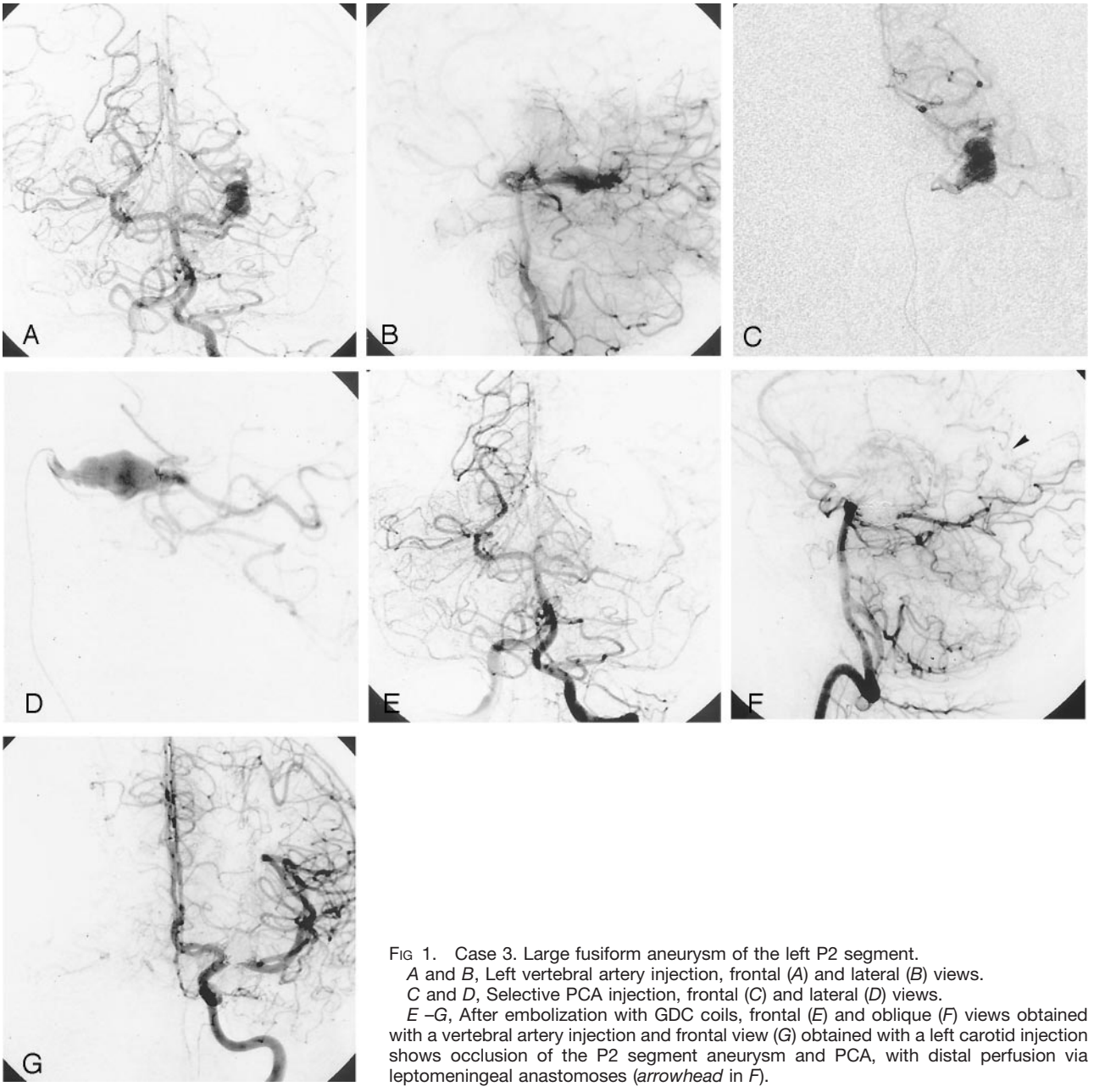

FIG 1. Case 3. Large fusiform aneurysm of the left P2 segment.
A and *B*, Left vertebral artery injection, frontal (*A*) and lateral (*B*) views.
C and *D*, Selective PCA injection, frontal (*C*) and lateral (*D*) views.
E –*G*, After embolization with GDC coils, frontal (*E*) and oblique (*F*) views obtained with a vertebral artery injection and frontal view (*G*) obtained with a left carotid injection shows occlusion of the P2 segment aneurysm and PCA, with distal perfusion via leptomeningeal anastomoses (*arrowhead* in *F*).

Discussion

Embryologic Considerations

The P2 segment represents the first segment of the true PCA system formed by P2, P3, and P4 segments (12). The PCA belongs to the internal carotid artery (ICA) system and constitutes its caudal terminal branch. The upper basilar artery distal to the trigeminal artery remnant, the P1 segment of the PCA and the PCoA, are included in this system. Embryologically, the PCA is a diencephalomesencephalic artery, which gathers its telencephalic supply by the distal annexation of the anterior choroidal artery (AChA) territory (13). In the so-called normal adult pattern, the P1 segment is larger than the PCoA. In the fetal-type configuration of the PCA, which is observed in as many as one third of cases (14), the PCA arises directly from the ICA. The diameter of the P1 segment is smaller than that of the PCoA, and Zeal and Rhoton (15) reported that the P1 segment is slightly longer than the P1 segment in the adult type.

Anatomic and Clinical Considerations

The PCA has been divided anatomically, and for surgical purposes related to the choice of the approaches, into three (7) or four segments (15, 16); the latter scheme is more commonly used (Fig 4). The P2 segment begins at the PCoA-PCA junction and courses within the distal peduncular and the ambient cisterns to end at the posterior part of the midbrain. It can be further subdivided into two parts, each 25 mm long. The anterior half, P2A, courses around the cerebral peduncle, inferior to the optic tract and basal

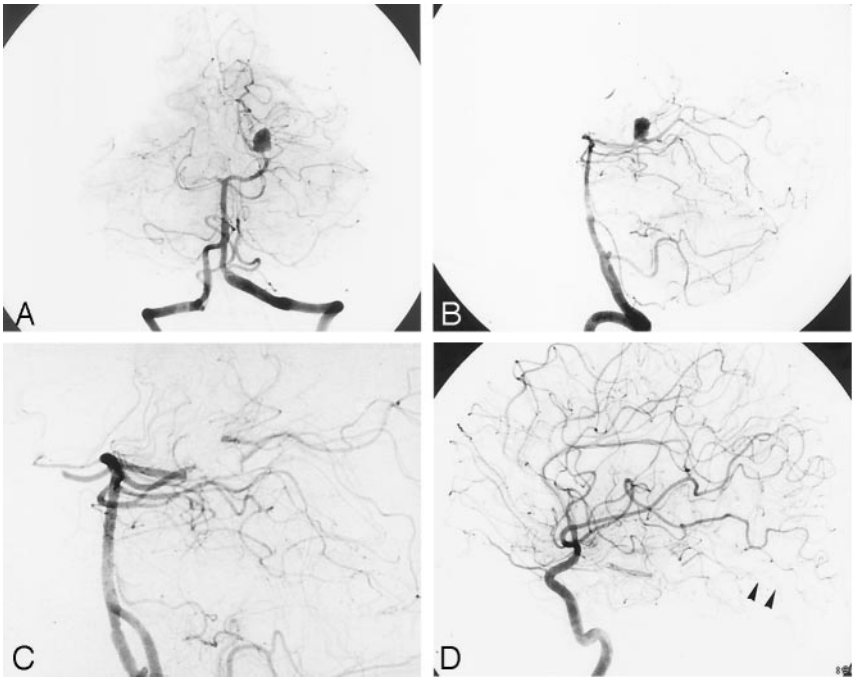

FIG 2. Case 7. Large saccular partially thrombosed aneurysm of the left P2 segment.

A and *B*, Vertebral artery injection, frontal (*A*) and lateral (*B*) projections.

C and *D*, After embolization, oblique view obtained with a vertebral artery injection (*C*) and lateral view obtained with a left carotid injection (*D*) show occlusion of the aneurysm and P2 segment, with distal perfusion of the PCA territory via a leptomeningeal supply. (*arrowheads* in *D*).

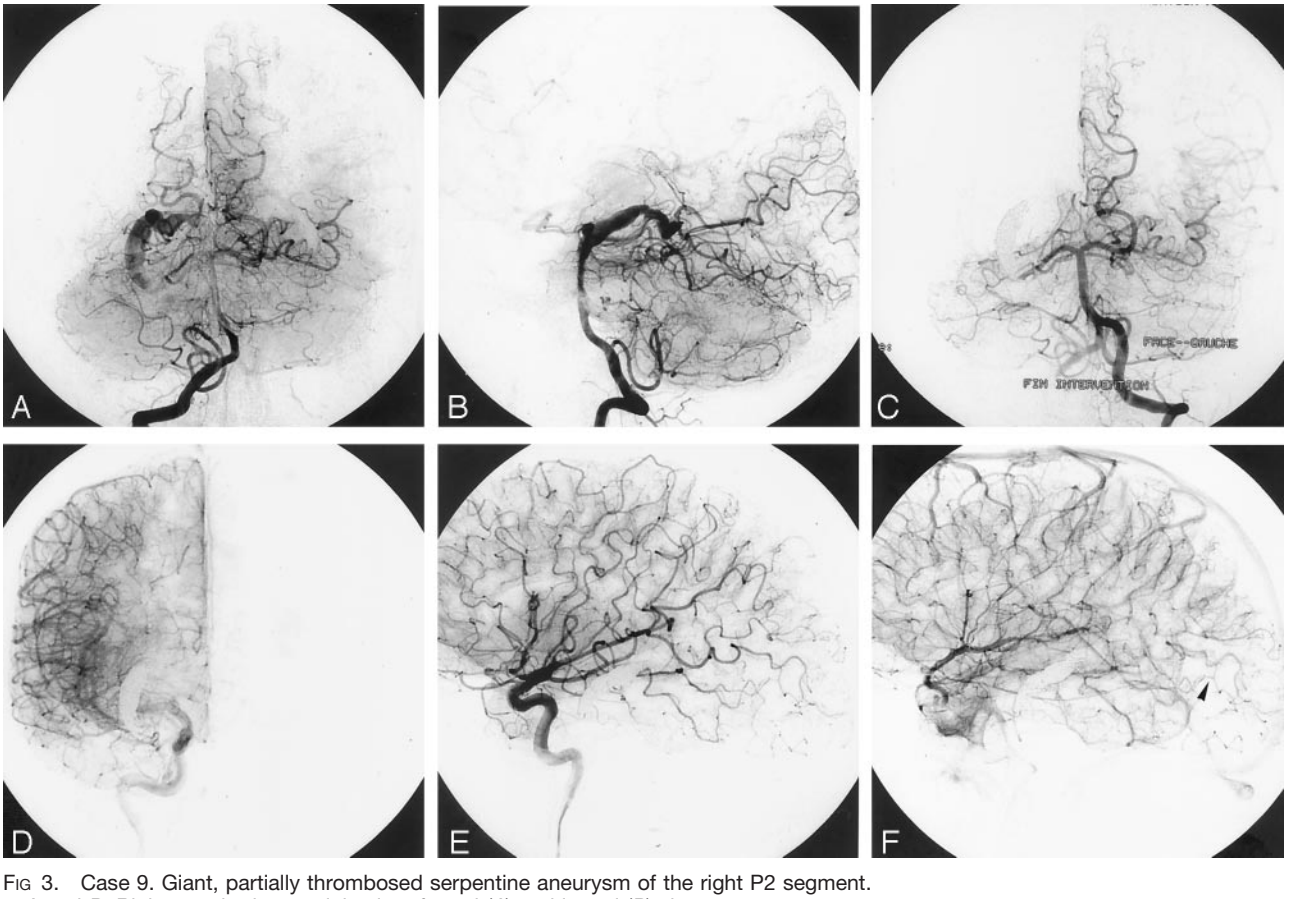

FIG 3. Case 9. Giant, partially thrombosed serpentine aneurysm of the right P2 segment.

A and *B*, Right vertebral artery injection, frontal (*A*) and lateral (*B*) views.

C, After embolization, frontal view obtained with a left vertebral artery injection shows complete occlusion of the P2 segment and aneurysm.

D–F, After embolization, frontal (*D*) and lateral early (*E*) and late (*F*) views obtained with a right carotid artery injection show the leptomeningeal supply to the right PCA territory (*arrowhead* in *F*).

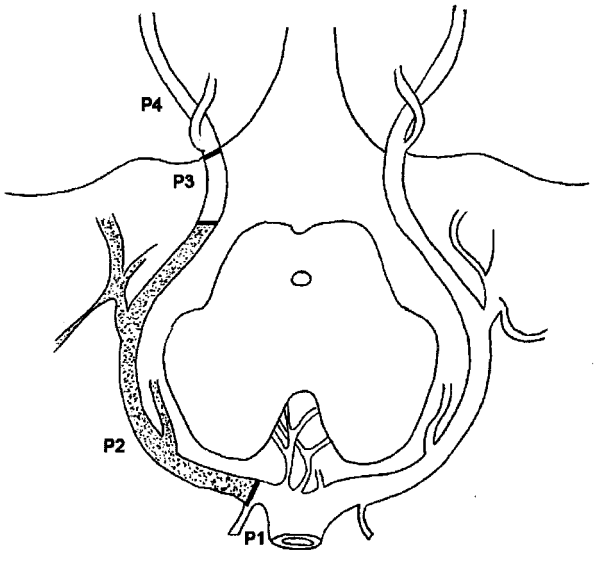

FIG 4. Schematic drawing of the midbrain surrounded by the PCA. The PCA is divided into four segments. The P2 segment begins at the PCoA-PCA junction and courses through the distal peduncular and ambient cisterns to the posterior part of the midbrain.

vein of Rosenthal and medial to the hippocampus to enter the proximal portion of the ambient cistern. The posterior half, P2P, begins at the posterior margin of the cerebral peduncle, courses along the posterolateral midbrain, parallel and inferior to the basal vein and optic tract, inferolateral to the geniculate bodies and pulvinar, and superomedial to the trochlear nerve and tentorial edge; P2P ends at its entrance into the quadrigeminal cistern. On anatomic specimens, the average diameter of the P2 segment is 2.9 mm, which allows endovascular navigation (15).

The P2 segment had three types of branches (13, 15). First, the P2 segment has central branches to the brain stem. Direct peduncular perforating arteries penetrate the cerebral peduncle and supply the corticospinal and corticobulbar pathways, the substantia nigra, and the red nucleus. Distal branches supply the midbrain, the posterior half of the lateral nucleus of the thalamus, the medial and centromedian nuclei, the pulvinar, the posterior limb of the internal capsule, the optic tract, and the geniculate bodies. These branches constitute the thalamogeniculate group (TGG) of Foix and Hillemand, or the posterior thalamic arteries of Lazorthes, which meet the thalamoperforating branches of the P1 segment near the middle of the thalamus and the premamillary branch of the PCoA anterior in the lateral nucleus. Infarction of the TGG territory causes the thalamic syndrome of Déjerine-Roussy. Long circumflex central branches (quadrigeminal arteries) usually arise from the P1 segment, but they may arise from P2A, supplying the peduncle, geniculate bodies, and tegmentum. They end in a rich arterial network over the superior and inferior colliculi, where they anastomose with branches of the superior cerebellar artery. Occlusion of circumflex arteries may result in Parinaud syndrome because of infarction of the posterior commissure or the nuclei of Darkschewitsh or Cajal.

Second, the P2 segment has ventricular branches. The medial posterior choroidal arteries (MPChA) may originate at the junction of P1, P2, and PCoA. They encircle the midbrain, distributing branches to the peduncle, tegmentum, geniculate bodies and colliculi, then turn forward and lateral to the pineal gland to enter the tela choroidea at its posterior end. The MPChA main trunk proceeds to the interventricular foramen and produces in a lateral branch that joins the arterial system of the choroid plexus of the lateral ventricle and anastomoses to branches of the AChA and lateral posterior choroidal arteries (LPChA). A medial branch has a recurrent course on the roof of the third ventricle, where it anastomoses with its counterpart of the opposite side. These branches supply the pulvinar, pineal gland, roof of the third ventricle, habenula, dorsal medial thalamus, and choroid plexus. The LPChA group originates mostly from P2P, passing laterally through the choroidal fissure over the pulvinar to enter the lateral ventricle.

Third, the P2 segment has inferior temporal branches that supply the cortical territory. These include the hippocampal artery, anterior temporal artery (ATA), middle temporal artery (MTA), and posterior temporal artery (PTA). The hippocampal arteries supply the hippocampus, the parahippocampal gyrus, and the uncus. In some instances, a true arterial ring supplies the limbic lobe with an anastomosis between the AChA; the anterior, middle, and posterior hippocampal arteries; the splenial arteries; and the pericallosal branch of the anterior cerebral artery (ACA). The ATA supplies the anteroinferior surface of the temporal lobe. The MTA and PTA supply the inferior surface of the temporal lobe, occipital pole, and lingual gyrus. The temporal arteries anastomose to the calcarine artery, or in about 10% of cases, the PTA may give off an accessory calcarine artery. The PTA has lateral cortical branches. Infarction in the PTA territory may induce a dysphasia or an amnestic syndrome with homonymous hemianopia in the dominant hemisphere. Splenial arteries or posterior pericallosal arteries may originate from the P2P, MPChA, LPChA, or PTA. The hippocampal artery, ATA, peduncular perforating artery, and MPChA most frequently arise from P2A. The MTA, PTA, common temporal artery, and LPChA most frequently arise from P2P. The TGG arises from either P2P or P2A.

P2 Segment Aneurysms

PCA aneurysms are unusual, accounting for approximately 1% of all intracranial aneurysms. Although Drake and Amacher (4) reported that the most common site of origin for PCA aneurysms is the first major branching point beyond the junction with the PCoA, the aneurysms may occur at any origin along the course of the main trunk of the PCA. According to the PCA segment involved, the aneu-

rysms are classified as those of the P1 segment, the P1-PCoA junction, the P2 segment, the P3 segment, or even the P4 segment. Aneurysms of the P2 segment have been reported either as single cases or as part of general reviews of vertebrobasilar aneurysms. Terasaka et al (10) presented a surgical series of 14 aneurysms in the P2 segment. Most of P2 aneurysms are saccular, arising at the origins of its central or cortical branches, but fusiform and serpentine forms have also been reported. Clinically, P2 segment aneurysms are discovered after a subarachnoid hemorrhage occurs (17), or they may cause a tumoral mass effect or even an ischemic stroke in the thalamic territory (18). They can also cause headaches and visual field defects (6). When the fetal type of PCA is encountered, aneurysmal formation is related to increased flow in the PCoA (19).

Surgical Treatment

For many years, treatment options were restricted to surgery. Direct interventions for aneurysms of the posterior circulation are more difficult than are those for aneurysms of the anterior circle of Willis. However, PCA aneurysms of P2, P3, and P4 segments (20) do not pose the same technical difficulties, nor do they have the same prognosis as that of basilar or P1 segment aneurysms. Yasargil (21) and Yasargil and Abdelrauf (22) recommend a pterional transylvian approach, sometimes expanded by making a 10–15-mm opening at the anterior inferior insular sulcus, allowing entrance into the temporal horn with subsequent opening of the choroidal fissure. Resection of part of the hippocampus and parahippocampus and sacrifice of a nondominant nonfetal PCoA may be necessary. Otherwise, P2 aneurysms are surgically approached by means of a subtemporal route without or with tentorium division or by means of a subtemporal transventricular transchoroidal route (7). The subtemporal approach may sometimes require excessive temporal lobe retraction, which can be complicated by postoperative swelling of special consequence on the dominant language hemisphere. Other potential complications are related to damage affecting cranial nerves III and IV. The use of intraoperative mannitol and the use of spinal or ventricular drainage for cerebrospinal fluid evacuation are usually sufficient to avoid hippocampal resection with its neuropsychological effects. Some authors advocate skull-base approaches, mainly the transzygomatic temporopolar approach to minimize temporal lobe retraction (5) with removal of the zygoma and the posterior wall of the orbit (exposure of which makes the middle cranial fossa flat). Further exposure is obtained by splitting the sylvian fissure and allowing the temporal lobe to fall backward after the anterior temporal veins are divided. Sindou and Fobé (23) propose removal of the roof of the external auditory meatus to gain access to P2-P3 segments without excessive temporal lobe retraction.

Surgical treatments differ, depending on the type of aneurysm (ie, berry or saccular, fusiform, or serpentine). For saccular aneurysms, even for giant ones, complete occlusion of the sac and neck should be attempted by placing clips in the aneurysmal necks. However, clip placement in the neck may sometimes be difficult or hazardous. Other surgical options available are clip placement in the proximal artery, excision of the aneurysmal sac without or with the restoration of distal flow via direct anastomosis or bypass, and use of a trapping procedure.

Serpentine aneurysms correspond to giant, partially thrombosed aneurysms containing tortuous vascular channels (24). They do not arise from arterial forking or at the site of vestigial vessels. Most of the time, they occur on a branch of the middle cerebral artery. Their presence on the PCA segments may contribute to the idea that the middle cerebral artery (MCA) and the PCA are embryologically similar and that the MCA is a branch from the ACA and the PCA is a branch from the ICA. In that particular type of aneurysm, the non-endothelialized intraaneurysmal channel is functional, meaning that it supplies part or all of the distal territory of the PCA. An intrathrombotic functional channel would result from the incorporation of the feeding artery in the aneurysmal sac. Most authors agree that giant serpentine aneurysms (GSAs) are derived from fusiform aneurysms or a fusiform configuration of the involved parent arteries (25). However, in a few cases, GSAs are related to associated saccular aneurysms and not atherosclerosis. Blood that enters GSAs then exit at a separate point; this point differentiates GSAs from true giant saccular aneurysms of the serpentine-like pattern that result from central clotting, because in the latter, the stream of blood enters and exits at the same site. In serpentine aneurysms of the saccular type, treatment options are similar to those proposed for true saccular aneurysms; that is, occluding the sac with parent vessel sparing by either surgical or endovascular means. When the neck cannot be defined, serpentine aneurysms are of the fusiform type. The sac cannot be obliterated. Surgical treatments for serpentine fusiform aneurysms and true fusiform aneurysms are the same, consisting of the removal of the sac with or without the restoration of distal flow by direct anastomosis or bypass, depending on the eloquence of the distal supplied area; proximal parent-artery occlusion or stent placement in the aneurysmal segment may be proposed. Current stents cannot be used in the P2 segment of the PCA at the present time.

Whatever the surgical option, treatment of P2 aneurysms poses a risk of infarction of the structures that are perfused by the perforating arteries or cortical branches at this level, either as a complication or consequence of voluntary occlusion of the parent P2 segment. To overcome any infarction from proximal occlusion or trapping of the parent vessel, some authors have advocated aneurysmal excision followed by end-to-end anastomosis of the parent artery (26) or bypass (27). In most surgical series or reports, however, occlusion of the PCA proximal to these branches did not result in a clinical syndrome, either transient or permanent, in all cases (6, 28, 29). This

result is certainly because of the rich anastomotic vascular network of this region (1). A collateral circulation is present between the posterior and AChAs. Splenial branches of both ACAs and PCAs contribute to the collateral vascular network. Leptomeningeal participation from the ACAs and MCAs provide a good collateral network to the occipital lobe (30). Ligation or exclusion of the P3 segment after it branches from the ATA (19) or PTA is reputed to be safe (30, 31). Collateral anastomotic channels contribute to the lack of any neurologic event, although the retrograde filling is not sufficient for injection into the aneurysmal formation. When occlusion is performed proximal to the origin of the MPChA, LPChA and splenial arteries, blood flow comes from the anterior circulation through the collateral network to the PCA distal to the occlusion. Occlusion may be safer in the proximal portion of the P2A than in the distal part, because the vascular network is spared in the former. The more proximal the occlusion, the smaller the likelihood of infarction.

Endovascular Treatment

Endovascular treatment is not associated with manipulation of the surrounding tissues. Therefore, the risks of brain infarction due to retraction or removal are theoretically reduced. However, specific thromboembolic events are related to endovascular navigation; these events should be considered when the treatment strategy is determined. We did not encounter any thromboembolic complications with the administration of heparin. Although our anticoagulation regimen for preventing thromboembolic events has not been scientifically proven, it may be supported with the absence of complications. In this particular subset of patients, no immediate or delayed ischemic event occurred in the distal PCA territory. In one patient, a homonymous hemianopia developed before the procedure; Lazinski et al (32) have reported this finding as a procedure-related complication.

The embolic material varied in our series, depending on the availability of the different devices at treatment. The six later cases were treated with GDCs. With all devices, occlusion of the parent vessel was accompanied by occluding the sac. In giant aneurysms, this combination ensured a reduction of the mass effect caused by the aneurysm, as others report (33). In large and giant fusiform serpentine aneurysms and true fusiform aneurysms, the combined occlusion of the sac and parent vessel appears to be the only option possible by endovascular means. For large and giant saccular P2 segment aneurysms, occlusion of the aneurysmal sac and neck may be difficult, especially in wide-necked aneurysms. Because well-developed collateral vascular pathways supply the distal PCA territory after its occlusion, our approach is to directly proceed to combined occlusion of the parent vessel and aneurysm.

In our series, the preoperative clinical grade was the only factor that influenced the prognosis. In all patients, the clinical grade was good or very good. We did not identify any other clinical or angiographic prognostic factor influencing the outcome; in particular, we assessed the basilar tip arrangement and the location of P2 segment relative to the choroidal fissure (34). Real-time angiography allowed us to evaluate the anastomotic supply to the desired occlusion point. When this network was sufficiently developed, we did not perform a test occlusion, because as Yamashita et al (36) reported, test occlusion may not be of any specific value. Of the three patients who tolerated aneurysmal occlusion, two had transient ischemic attacks. When the network was judged insufficient, the test occlusion showed the aperture of this collateral supply. In our series, a test occlusion was used in five patients. The only patient who was awakened from anesthesia had a preprocedural visual field defect. Analysis of the angiograms showed that, after the point of occlusion, distal temporal branches not occluded by the embolic material behave as sump aspirators for flow, allowing revascularization of the distal PCA via a leptomeningeal supply.

Conclusion

In our small series of 10 patients, acute parent artery occlusion in a P2 segment aneurysm was safe, regardless of whether the location was proximal or distal to the perforating, ventricular, or cortical branches. The potential for collateral supply and the hemodynamic balance between the AChAs and PChAs and the pericallosal and anterior and middle cerebral vessels to the distal PCA may account for the finding that aneurysmal and P2 segment occlusion was not accompanied by any adverse effect. The main reason for no adverse effect was that the aneurysm occurred after the origin of the thalamoperforating vessels arising from P1 segment. In our experience, endovascular parent vessel occlusion appears to be an appropriate procedure for treating P2 segment aneurysms.

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