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ORIGINAL RESEARCH

Giant and Large Peripheral Cerebral Aneurysms: Etiopathologic Considerations, Endovascular Treatment, and Long-Term Follow-Up

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BACKGROUND AND PURPOSE: Giant/large peripheral cerebral aneurysms beyond the circle of Willis and middle cerebral artery (MCA) bifurcation are rare lesions, their etiopathogenesis is not completely elucidated, and their treatment is often difficult. We reviewed the etiopathologic findings available in the literature and report the results and long-term follow-up in 10 patients with a giant/large peripheral aneurysm treated by parent artery occlusion.

METHODS: Four aneurysms were on the MCA, 2 on the anterior cerebral artery, and 4 on the posterior cerebral artery (PCA). Two patients presented with bleeding. An occlusion test was performed in 7 patients. Occlusion of the parent artery was performed by using coils in 8 patients; and liquid coils and glue, in 2. Imaging follow-up was available from 1.5 to 4.5 years; and clinical outcome, from 1.5 to 7 years.

RESULTS: All aneurysms were excluded. One patient developed a partial homonymous hemianopsia after PCA occlusion. A transient deficit was observed in 2 other patients. In the partially thrombosed aneurysms, follow-up imaging showed shrinkage of the thrombosed compartment of the aneurysm and disappearance of the mass effect. No patient experienced progression of symptoms and/or bleeding during the follow-up period. At long-term follow-up, 6 patients presented with a modified Rankin Scale score of 0; 3, with score of 1; and 1, with score of 2.

CONCLUSION: The endovascular occlusion of the parent artery appears a relatively safe and efficacious technique in the treatment of these lesions. Long-term follow-up studies confirm persistent exclusion of the aneurysm and good clinical tolerance to the vessel occlusion.

Intracranial aneurysms can be classified according to their shape and site of origin into saccular (named also "berry") aneurysms, arising from arterial bifurcations or an accentuated turn in the vessels, and nonsaccular aneurysms, arising from the arterial trunks unrelated to branching sites.[1,2] Peripheral cerebral aneurysms located distally along the cerebral arteries beyond the circle of Willis and middle cerebral artery (MCA) bifurcation may be included in the category of arterial trunk aneurysms. These "spontaneous" peripheral cerebral aneurysms must be differentiated from peripheral aneurysms with a known origin, such as flow-related aneurysms associated with an arteriovenous malformation (AVM), or infectious, oncotic, and traumatic aneurysms. Although most of the spontaneous arterial trunk aneurysms are presumed to be caused by spontaneous dissection,[1-4] the etiopathogenesis and natural history of these aneurysms not arising from a branch point remain not completely elucidated and have been rarely discussed.[2,3,5-7]

Spontaneous peripheral cerebral aneurysms located on the supratentorial arteries are often giant/large, and their management can be difficult.[2,8-13] The endovascular treatment of giant/large peripheral lesions has been reported in a few articles dealing with a relatively small series of aneurysms located more often on the posterior cerebral artery (PCA).[3,14-18] Selective treatment of these aneurysms appears ineffective,[14] and most of the lesions are treated by parent artery occlusion (PAO). In most reported cases, only short and midterm follow-ups after treatment are sometimes mentioned. In infratentorial locations, the results of the endovascular treatment of peripheral aneurysms located in the cerebellar arteries have also been reported.[19-21] In these reports, some of which included infectious, posttraumatic, and AVM flow-related aneurysms, the lesions were predominantly small and treatment of giant aneurysms was not reported.

The purpose of this study was to report our results, outcome, and long-term-follow-up in a series of patients with spontaneous giant/large peripheral cerebral aneurysms treated by elective PAO.

Methods

During a 6-year period between January 1998 and January 2004, 10 patients with supratentorial giant/large peripheral cerebral aneurysms were treated by endovascular occlusion of the parent artery. We excluded from this review 1) peripheral giant/large lesions treated by surgery or by selective endovascular occlusion of the aneurysm, 2) giant/large peripheral aneurysms with an obvious underlying cause or known origin, such as AVM flow-related aneurysms or infectious, oncotic, or traumatic aneurysms, 3) infratentorial giant/large peripheral lesions located on the cerebellar arteries (in our series, all of them were associated with a cerebellar AVM), and 4) lesions treated after January 2004 in order to include only patients with long-term follow-up (longer than 18 months) after PAO treatment. Retrospective chart analysis and radiologic studies were performed by 2 neuroradiologists (A.B., B.J.). There were 10 patients: 4 men and 6 women, ranging in age from 18 to 64 years (mean age, 40.6 years). All lesions

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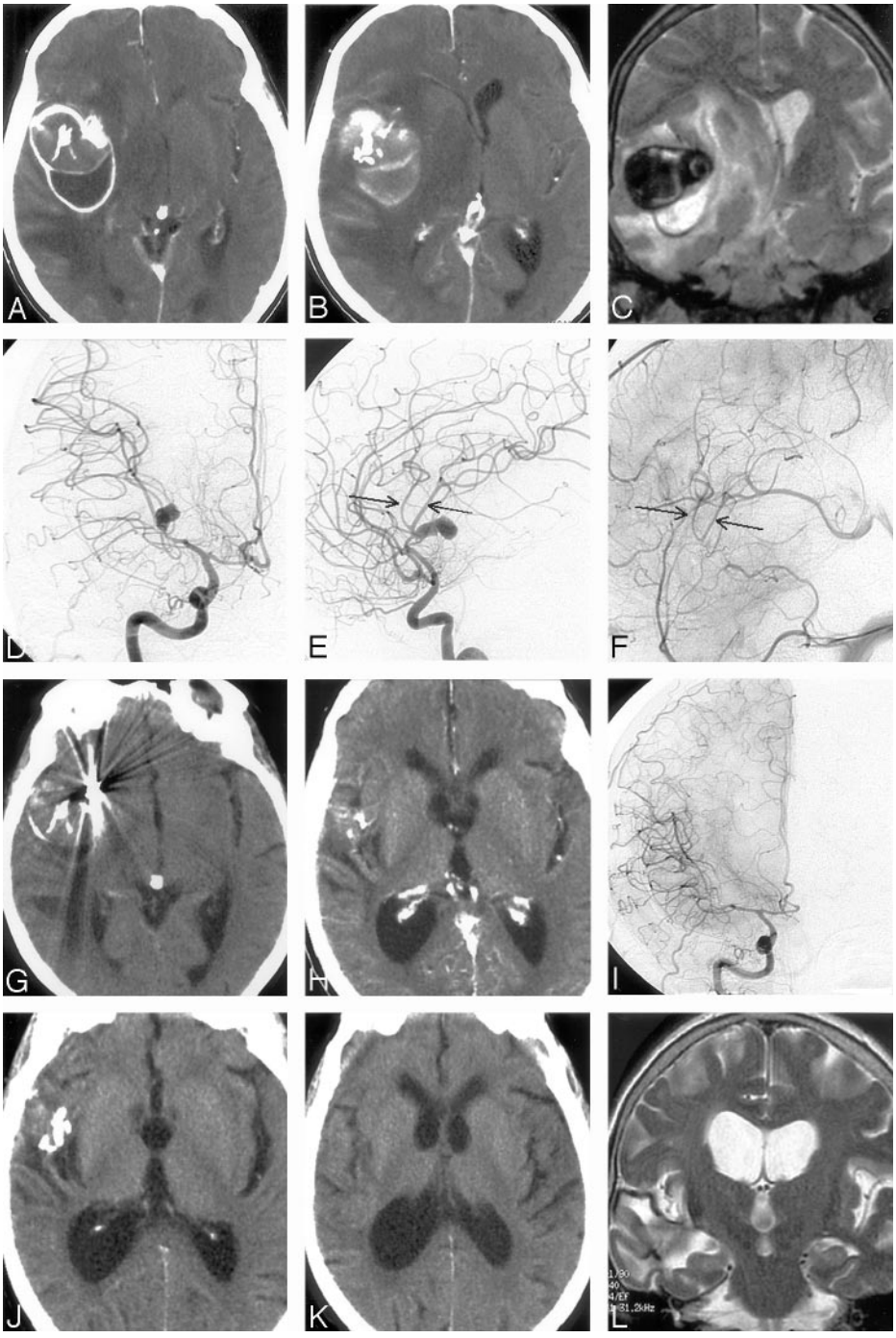

Fig 1. Partially thrombosed giant aneurysm of the superior branch of the right MCA bifurcation in a 64-year-old woman presenting with headache and paresis of the left arm.

A and *B*, CT scans with contrast injection show a giant heterogeneous right temporal lesion with a "cystic" compartment, calcifications, and severe mass effect. *C*, Coronal T2-weighted MR image shows an unusual aspect of a partially thrombosed giant aneurysm of a right MCA branch. The right internal carotid artery angiography, in anteroposterior (*D*) and lateral (*E*) views, depicts the patent compartment of the aneurysm. *F*, Occlusion test with a nondetachable balloon. On the angiographic venous phase in the lateral view, a retrograde filling of the arterial branches after the level of the occlusion (*arrows*) is observed via leptomeningeal anastomosis. *G*, Three days after the PAO was performed by using coils, a CT scan without injection of contrast shows shrinkage of the aneurysm and reduction of the mass effect. *H*, A CT scan with injection after 1.5 years shows persistent disappearance of the mass effect and reduction of the thrombosed compartment in relation to the CT examination performed after 6 months (not shown). *I*, Angiographic 1.5-year follow-up of the right internal carotid artery in the anteroposterior view shows persistent exclusion of the aneurysm and retrograde vascularization of the vessels beyond the occlusion via leptomeningeal anastomosis from ACA and MCA branches. Arterial supply from the PCA was also observed on the vertebral angiogram (not shown). In follow-up studies after 3.5 years, CT scans (*J* and *K*) and coronal T2-weighted MR image (*L*) show further shrinkage of the aneurysmal thrombosed compartment. Findings of the neurologic examination were normal.

were peripheral (distal to the circle of Willis and the MCA bifurcation). Six aneurysms were giant (>25 mm), and 4 were large (>10 mm). Four aneurysms were on the MCA; 2 aneurysms, on the anterior cerebral artery (ACA); and 4 aneurysms, on the PCA.

Four giant aneurysms were partially thrombosed (Figs 1 and 2). On angiography, 8 aneurysms showed a focal dilation, and 2 lesions were fusiform. Angiographic findings of the parent vessel suggesting a dissection were proximal and/or distal arterial stenosis in 3 patients, delayed and slow-filling of the parent artery beyond the aneurysm in 4 patients, irregular appearance of the vessel in 1 patient, and irregular

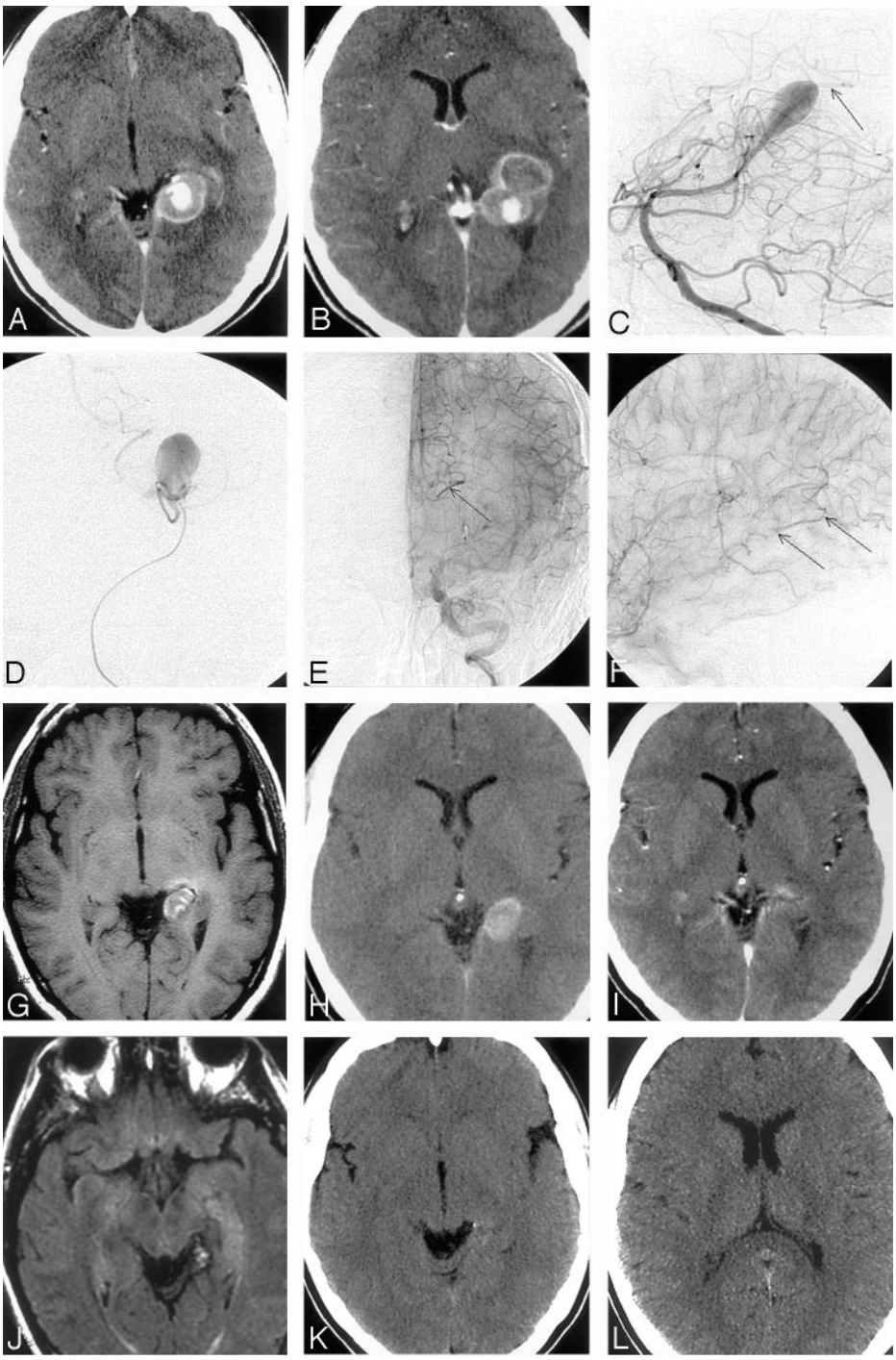

Fig 2. Partially thrombosed giant aneurysm of the P2-P3 segment of the left posterior cerebral artery in a 28-year-old woman presenting with headache and loss of consciousness.

A and *B*, CT scans with injection of contrast show a large lesion compatible with the diagnosis of a thrombosed aneurysm. *C*, Oblique view of the left vertebral artery angiography shows the circulating portion of the aneurysm associated with a slight stenosis of the parent artery. Slow filling of the parent artery beyond the aneurysm is noted (*arrow*). *D*, Intermediate step of the endovascular procedure. After occlusion of the P2-P3 segment of the posterior cerebral artery by using coils, anteroposterior (*E*) and lateral (*F*) views of the left internal carotid artery angiography show the retrograde vascularization of the PCA distal to the occlusion via the leptomeningeal anastomosis (*arrows*). Thirty-six hours after PAO, the patient presented with transient mild paresthesias and mild oculomotor paresis, which completely regressed in 3 days. Follow-up studies after 1 month (T1-weighted MR image, (*G*); and CT scan with injection of contrast, (*H*) show reduction of the aneurysmal thrombosed portion and reduction of the mass effect. Follow-up studies after 1 year (CT scan, (*I*) and after 4 years (fluid-attenuated inversion recovery MR image, (*J*); and CT scans, (*K* and *L*) show further shrinkage of the aneurysmal thrombosed compartment. The small left posterior thalamic infarct is well visualized (*L*). Findings of the neurologic examination were normal at 5-year follow-up.

appearance with distal subocclusion in another patient. In our series, clinical onset included intense headache in 6 patients (in 1 patient associated with a loss of consciousness), mass effect in 2, and subarachnoid hemorrhage in 2. One of the patients with subarachnoid hemorrhage and a clot in the right ambient cistern presented with left hemiplegia. One patient with a PCA aneurysm presenting with headache had a history of visual troubles compatible with embolic phenomena. In 2 other patients presenting also with headache, the aneurysm was diagnosed during pregnancy in 1 patient (case 2) and 3 weeks after delivery in the other (case 7, Fig 2). In all patients, there

were no findings suggesting an infectious or traumatic origin in the etiology of the aneurysm. We also evaluated the medical charts to eliminate other clinical contexts observed in association with aneurysms such as tumors, vasculopathies (systemic lupus erythematosus, Ehlers-Danlos disease, Takayasu arteritis, and fibromuscular dysplasia), and drug abuse. (Clinical and angiographic findings are summarized in the Table.)

In all procedures, the planned strategy was to obtain the occlusion of the parent artery. In 1 patient, the aneurysm had been previously treated electively with coils and had recurred twice.

Before the PAO, an occlusion test using a small undetachable balloon (Balt B1, Balt, Montmorency, France) was performed in 5 patients to evaluate the clinical and angiographic tolerance to the occlusion (Fig 1). For the clinical evaluation, the vessel occlusion by balloon lasted approximately 20 minutes. In 2 other patients with a P2-P3 aneurysm, the balloon test occlusion (BTO) was performed with the patients under general anesthesia and only the collateral circulation was evaluated without clinical examination. In 3 remaining patients (1 ACA and 2 P2-PCA aneurysms), the BTO was not performed. Control angiography during BTO showed retrograde filling of the branches distal to the parent vessel site of occlusion through leptomeningeal collateral circulation in all patients, and in the patients who were awake, no clinical deficit occurred. One patient underwent general anesthesia after the occlusion test; therefore, the endovascular treatment was performed with the patient under general anesthesia in 7 patients and under neuroleptoanalgesia in 3.

The PAO was obtained at the level of or very proximal to the aneurysm by using coils in 8 patients and liquid coils and glue in 2. Glue was added to liquid coils to obtain a more stable occlusion of the parent artery. In only 2 patients, coils were also partially positioned within the aneurysm. During the PAO procedure and also during the occlusion test, patients underwent systemic heparinization to obtain an activated clotting time 2- to 3-times baseline. After the procedure, systemic heparinization was maintained at twice the control level for 24 hours and never reversed with protamine sulfate. Arterial pressure was increased for 24–48 hours, usually to systolic values of 140–160 mm Hg, to facilitate the establishment of retrograde leptomeningeal circulation.

Before the procedure CT and/or MR imaging studies were available in all patients. MR angiography was also performed in 5 patients; and CT angiography, in 2. After the procedure, imaging follow-up was performed from 1.5 to 4.5 years (mean, 28 months) in all patients. Angiography was performed from 1 to 2 years (mean, 18 months) after the procedure. The patients with a partially thrombosed aneurysm underwent multiple CT and/or MR imaging follow-up studies to evaluate shrinkage of the lesion (Figs 1 and 2). In 3 patients, imaging follow-up was performed in another country or institution. Long-term outcome from 1.5 to 7 years (mean, 4.5 years) was obtained by clinical consultation or, more rarely, by telephone interview and was assessed by using the modified Rankin Scale (mRS).[22]

Results

After occlusion of the parent artery, a complete exclusion of the aneurysm was obtained in all cases. Retrograde collateral circulation never refilled the aneurysm. Transitory worsening was observed in 2 patients. In a patient with a right MCA aneurysm (case 4), despite the good clinical tolerance to BTO, a momentary left central facial palsy lasting 10 minutes was observed immediately after the procedure. In another patient (case 7), with a P2-P3 PCA aneurysm and PAO at the P2 seg-

Giant and large peripheral cerebral aneurysms treated by parent artery occlusion: angiographic and clinical results and follow-up

Patient No./ Sex/Age (y)	Clinical Onset	Side	Aneurysm Location	Size	Thrombosis	Angiographic Appearance	Occlusion Test	Complications After PAO	Angio FU (mo)	Angio Results: Aneurysm and PA	Clinical FU	mRS FU
1/F/64	Mass effect/L arm paresis	R	MCA, M2 proximal	Giant	Yes, Calcif.	Focal dilation	Negative		18	Occlusion	6 y 5 mo	0
2/F/34	Headache	L	MCA, M2 distal	Large		Fusiform	Negative		12	Occlusion	7 y	1
3/F/18	Headache	L	MCA, M2	Large		Fusiform	Negative		20	Occlusion	6 y 4 mo	0
4/M/44	Headache	R	MCA, M3	Large		Focal dilation	Negative	Transient facial palsy	17	Occlusion	4 y 4 mo	1
5/F/60	Mass effect/ visual deficit	R	ACA, A2	Giant	Yes	Focal dilation	Not performed		22	Occlusion	2 y	0
6/M/49	Hemorrhage	L	ACA, A3	Giant		Focal dilation	Negative		18	Occlusion	4 y 9 mo	0
7/F/28	Headache	L	PCA, P2–P3	Giant	Yes	Focal dilation	Yes, Angio. test	Transient paresthesia and oculomotor paresis	24	Occlusion	5 y 6 mo	0
8/M/34	Headache	L	PCA, P2–P3	Giant	Yes	Focal dilation	Yes, Angio, test		18	Occlusion	4 y 5 mo	0
9/F/25	Hemorrhage/L hemiplegia	R	PCA, P2 proximal	Giant		Focal dilation	Not performed	Partial HH	13	Occlusion	1 y 1 mo	2
10/M/50	Headache/visual troubles	L	PCA, P2 distal	Large		Focal dilation	Not performed		18	Occlusion	3 y	1

Note:—Angio. indicates angiographic; FU, follow-up; PA, parent artery; R, right; L, left; PAO, parent artery occlusion; HH, homonymous hemianopsia; mRS, modified Rankin scale (0 means no symptoms, 1 minor symptoms, 2 some restriction in lifestyle); Calcif, calcification; MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery.

ment, right hemiparesthesia and mild oculomotor paresis appeared 36 hours after the procedure. The onset of symptoms coincided with the interruption of the heparinization and the return to a normal low arterial pressure. After therapeutic subcutaneous low-molecular-weight heparin and aspirin and a moderate increase of arterial pressure were instituted, the patient recovered completely in a few days. CT and MR images showed a small left posterior thalamic infarct. Three years after, a second pregnancy was uneventful (Fig 2). We observed 1 permanent complication in the patient presenting with left hemiplegia due to bleeding of a PCA aneurysm (case 9). This patient developed a partial homonymous hemianopsia (HH) after PCA occlusion, and CT and MR imaging studies confirmed an occipital ischemic lesion. At 18-month follow-up, only a mild hyposthenia of the foot and a persistent slight visual field deficit were observed.

No patient presented progression of symptoms and/or bleeding during the follow-up period. Follow-up angiographic studies showed persistent occlusion of the artery and exclusion of the aneurysm in all cases (Fig 1). In the patients with a giant partially thrombosed aneurysm, CT and MR imaging follow-up studies showed a progressive shrinkage of the thrombosed compartment of the aneurysm and disappearance of the mass effect (Figs 1 and 2). Overall, at long-term follow-up, a good mRS was observed in our series: 6 patients with score of 0, 3 with score of 1, and 1 with a score of 2 (according to mRS, 0 means no symptoms; 1, minor symptoms; and 2, some restriction in lifestyle). Results are summarized in the Table.

Discussion

Intracranial aneurysms arising along the vessel trunk are uncommon lesions in contrast to intracranial saccular aneurysms arising at arterial bifurcations.[9,23,24] Peripheral aneurysms located along the cerebral arteries beyond the circle of Willis and the MCA bifurcation are rare lesions, and their origin and natural history are not completely elucidated.

Etiopathogenetic and Angiographic Aspects

Arterial trunk aneurysms unrelated to branching zones seem more likely to develop as a result of arterial dissection.[1-3] Although some authors[7,12,25,26] suggest atherosclerosis as the cause of lateral fusiform aneurysm, others[5,6,27,28] have demonstrated a lack of atherosclerosis in most surgical and pathologic studies. Drake and Peerless,[9] in a large review of giant fusiform aneurysms (also including distal aneurysms), found only 5% of patients affected by atherosclerosis. Day et al,[2] in a review of MCA aneurysms (most lesions were giant), proposed that essentially all fusiform aneurysms not associated with atherosclerosis are dissecting aneurysms. Although some experimental and computer models of lateral aneurysm have been developed,[29-32] few pathologic studies of arterial trunk aneurysms are available, most of them obtained in the vertebrobasilar system and ICA lesions[5,6,28,33,34] and, more rarely, in a distal location.[6,7,34] Consequently, even if dissection seems to be the most likely cause, the assumption that all arterial trunk aneurysms are dissecting in origin remains unproved.

In an animal model,[31] cerebral aneurysms arising at nonbranching sites and at branching sites were induced by the same factors: hemodynamic stress and hypertension. Available data suggest that fusiform aneurysms are lesions that start with internal elastic lamina (IEL) fragmentation due to hemodynamic stress followed by intimal hyperplasia, possibly as a reaction to the damage and successive dissection;[5-7,28] acute disruption of the IEL without intimal thickening has been observed in acute dissecting aneurysms.[5] Some authors[2,6] propose that the primary causative factor of the dissection is an intramural hematoma, most likely due to rupture of the vasa vasorum or of new vessels within the thickened intima. Others[35] seem to suggest that the rupture of the intima is the initial event that precedes the subsequent penetration of circulating blood into the vessel wall.

In our series, angiographic findings compatible with a dissecting origin were found in 8 patients. On angiography, dissecting aneurysms appear as elongated ovoid or saccular focal dilations, usually parallel to the axis of the artery. Saccular focal dilations, especially if giant, can mimic a true saccular aneurysm, and this aspect explains why some peripheral dissecting aneurysms are, at times, referred to as "saccular lesions." Angiographic findings of the parent vessel suggesting a dissecting aneurysm include vessel stenosis/occlusion, delayed filling of the parent artery beyond the aneurysm, and the pathognomonic, however rare, aspect of a double lumen with visualization of both true and false channels.[3,4,36-38] Morphologic changes in giant/large peripheral aneurysms have been shown in some lesions in which repeated angiographic studies were performed.[2,3,39] Lesions can enlarge and/or evolve from a focal dilation to a fusiform lesion. Multiple dissections of the intima, thrombus formation, recanalization, and further bleeding can be responsible for aneurysmal growth. In particular, the so-called giant serpentine aneurysms, often reported as a particular entity,[16,39-44] seem due to progressive enlargement of the dissection.[1,2,5] Arterial trunk lesions defined in the literature as fusiform, dolichoectatic, dissecting, and/or serpentine aneurysms, often reported as different entities, seem to be the same type of lesion or the evolution of an initial lesion. Aneurysms with a fusiform morphology are defined as lesions with separate inflow and outflow sites in contrast to saccular lesions. However, in dissecting aneurysms with a focal dilation, blood flow enters and exits at the same site. Extension of the dissecting phenomena, with appearance of another dissecting point, can permit the blood to exit back into the vessel and modify the morphology of the aneurysm from a focal dilation to a fusiform lesion.

In an animal model of dissecting aneurysm,[32] aneurysm formation and morphologic changes were strongly affected by the size of the intimal entry zone. However, it was not possible to induce formation of aneurysms with a fusiform morphology. We think that this seems to suggest that the dissecting phenomena can progress along the vessel if a long segment of the arterial wall is diseased and/or weakened. On the basis of a pathologic study,[35] it has been reported that aneurysms with 1 entrance along the arterial wall are associated with an unstable clinical course. Aneurysms with both an entrance and an exit back into the true lumen have a constant flow of blood through the pseudolumen and are clinically more stable. In our series, bleeding occurred only in aneurysms with a focal dilation.

Clinical Findings

A slight male preponderance is reported in series of giant arterial trunk aneurysms.[2,9,42] Other small series of peripheral lesions report an equal male/female ratio[14,15] or, as observed in our series, even a slight female preponderance.[17] Patients are younger compared with series of patients with saccular aneurysms. As observed in our patients, in most series, mean age ranges from 36 to 44 years, and these lesions can also observed in children and adolescents.[2,9,14,15,17,42] Regarding clinical presentation, headache and mass effect are more often responsible for clinical onset.[2,14,15,17,27,42] Hemorrhage was observed in 20% of patients in the series of Drake and Peerless.[9] Day et al,[2] in their series of MCA arterial trunk aneurysms, reported that small, large, and giant aneurysms with focal dilation had subarachnoid hemorrhage rates of 80%, 62%, and 23%, respectively, whereas serpentine aneurysms had a hemorrhage rate of 14%. In our series, only 2 of 10 patients presented with bleeding. In 2 of our patients, the aneurysm was diagnosed during or just after pregnancy. During pregnancy, increased laxity of vascular walls, fluctuations of blood pressure, and endocrinologic changes may predispose to both arterial dissection and aneurysm formation.[45]

Endovascular Treatment and Follow-Up

Despite progress in endovascular technique, the selective treatment of giant peripheral aneurysms remains impossible in lesions with a fusiform/serpentine morphology or is usually ineffective in the long term in lesions presenting a focal dilation.[14] As reported by other authors and observed in 1 case of our series, recanalization usually occurs in patients treated with selective occlusion.

The surgical treatment, which is a more-invasive procedure, is also challenging, and in most of cases, it consists of the sacrifice of the parent artery.[9,13,46-48] The PAO performed endovascularly on distal supratentorial branches appears to be a relatively safe treatment, and no mortality is reported in the recent literature. However, the incidence of neurologic deficits that may occur as a result of such treatment must be considered. Although the BTO permits clinical evaluation and angiographic assessment of collateral circulation, its value before the PAO is controversial. Regarding clinical aspects, Yamashita et al[49] reported that BTO may not be of any specific value, and of the 3 patients with PCA aneurysm who tolerated aneurysmal occlusion, 2 had transient ischemic attacks. Complications have also been reported, despite angiographic visualization of collateral filling of the vessels distal to the site of occlusion.[18] Conversely good outcome can be observed even without previous identified collateral circulation.[17]

It should be considered that giant/large aneurysms are often responsible for changes in the distal flow of the parent artery; consequently, some adaptive collateral circulation could have already been established. We do not perform an occlusion test if the parent artery endovascular occlusion is the only possible therapeutic approach and the patient has accepted the risk of the procedure (such as a potential HH). In all our test occlusions, it was possible to demonstrate a collateral circulation. However, in the presence of the lack of a retrograde circulation or if the occlusion test is clinically positive, alternative options and the need for arterial bypass should be considered.[50] Nevertheless, the risk of potential morbidity of bypass techniques should be also evaluated.[47]

Other techniques to evaluate tolerance of PAO have been reported. Kon et al[51] reported a combination of BTO monitored with single-photon emission CT. As reported in small peripheral infectious aneurysms,[52,53] the utility of Wada test with amobarbital should be considered in lesions located in functional territories. Selective Wada testing can also be performed to exclude the presence of perforating arteries not detected on angiography.[54] During the BTO, collateral supply and hemodynamic balance between the cortical branches of the MCA, ACA, and PCA can often be evaluated on angiography. This evaluation can be more difficult between the anterior and posterior choroidal arteries. The occlusion of the MCA beyond the bifurcation and of the ACA beyond A1 requires evaluation of cortical collateral circulation. Although rare, perforating arteries arising from the M2 segment have been described: The perforating arteries of MCA and ACA arise respectively from the M1 and A1 segments, and the artery of Heubner arises more often from the proximal A2 segment at the junction with the anterior communicating artery.[55,56]

The distal occlusion of the PCA (beyond P1) poses different problems due to a more complex anatomy, including perforating, ventricular, and cortical branches. Hodes et al[57] reported preliminary results of PAO by using balloon and autologous blood clot in 3 cases (1 MCA and 2 PCA) of distal giant aneurysms with test occlusion. In the series of Ross et al,[14] a BTO was performed in all patients before PAO, and only 2 of 9 patients experienced mild periprocedural ischemia. Drake et al[46] reported that the ACA usually has excellent leptomeningeal collateral flow, and some cases of PAO of the MCA branches with good outcome have been reported.[58,59] Mawad and Klucznik[16] reported 2 giant aneurysms of the left MCA treated by PAO, resulting in aphasia in 1 patient with a successful pretherapy Wada test and transient dysphasia in the second one, in which no occlusion test was performed. Our 4 patients with peripheral MCA aneurysms underwent a test occlusion; however, 1 patient presented with transient facial palsy after PAO, despite the preprocedural tolerance to BTO.

In case of PCA occlusion, sensory-motor and visual deficits can occur. Regarding the perforating branches of the PCA, most of them arise from P1; and some, from P2 (direct peduncular perforating arteries and the thalamogeniculate arteries). However, long circumflex (quadrigeminal) arteries may also arise from the P2 anterior segment, and the thalamogeniculate arteries can also arise from the P3 segment and even, in rare cases, from the calcarine and parietooccipital arteries. The anastomotic circulation is not well understood; it seems more frequent between the thalamogeniculate and thalamoperforating arteries at level of the thalamus and between the long circumflex arteries and the superior cerebellar artery at level of the quadrigeminal plate.[60-64] Hallacq et al[15] stated that PAO in a P2 segment, after the origin of the perforating branches arising from P1, is safe, and the authors did not use test occlusion when the planned occlusions involved the distal P2 segment. Nevertheless, they stated that occlusion may be safer in the proximal portion of the P2 segment than in the distal part because the vascular network is spared in the former. Ciceri et al[17] treated 5 giant aneurysms of the PCA by PAO, without a

preliminary test occlusion and reported hemiparesis and HH after PAO at P2 in 1 patient. Lazinski et al (2000)[3] treated 3 PCA dissecting aneurysms with PAO and observed HH in 1 patient. Arat et al,[18] in a multicentric study including 8 aneurysms (all treated without test occlusion), reported HH in 1 patient. One of our 4 patients who underwent the occlusion of the PCA presented a partial HH.

Overall, except for 1 series[15] with no reported complications, after endovascular treatment, the risk of HH ranges from 12.5% to 33%. In surgical series, Drake[65] reported 17.4% visual field deficits after occlusion or trapping of the PCA P2 segment for PCA aneurysms. However, mortality and morbidity of surgical treatment in distal PCA aneurysms are considerable (up to 36%).[66,67] Our patient (Fig 2), with a transient right hemiparesthesia and mild oculomotor paresis associated with a small posterior thalamic infarct after PCA occlusion, is very similar to a case of P2-P3 aneurysm reported by Arat et al,[18] in which hemiparesis resolved within weeks and hemihyperesthesia resolved within 1 year. As in our patient, this case was associated with a small posterior thalamic infarct (also observed in another patient of the same series). These thalamic lesions are probably due to occlusion of thalamogeniculate arteries supplying the posterior half of the lateral nucleus of the thalamus and arising at the P2 segment, where the PCA occlusion is performed. The transient oculomotor paresis observed in our patient is probably due to involvement of long circumflex (quadrigeminal) arteries, which can more rarely arise from the P2 segment instead of a common origin at P1. In our patient, transient deficits appeared after interruption of heparin therapy and concomitantly a return to a low blood pressure level. In our experience, moderate hypertension immediately and a few days after the PAO is recommended to promote development of collateral circulation.

In the case of aneurysms with a focal dilation, some authors[14,51] affirm that occlusion of the parent vessel should be accompanied by occlusion of the aneurysmal sac to ensure early thrombosis of the aneurysm. However, the elective occlusion of the parent artery at the level of the aneurysm without the presence of coils detached inside the aneurysm could increase shrinkage and resorption of the giant lesion (Figs 1 and 2).

In most of the articles reporting cases of giant/large peripheral aneurysms treated by PAO, mean clinical and radiologic follow-up is reported between 6 months and 1 year.[3,14,15,18] Our angiographic results were in accordance with the cases reported in the literature, with midterm follow-up showing that the occlusion of the parent artery remained stable without filling of the aneurysm.[3,14,15,18] In the literature, few cases of giant peripheral aneurysms with midterm follow-up MR imaging are mentioned.[17,51] Hallacq et al[15] reported only 1 case with long-term MR imaging follow-up at 6 years with a two-thirds reduction in the aneurysmal mass. In our cases of giant partially thrombosed lesions, subsequent MR imaging and CT studies showed a progressive shrinkage of the aneurysm continuing over the years (Figs 1 and 2). The good mRS outcome observed in our series at long-term follow-up (mean, 4.5 years) confirms the results at midterm follow-up reported by Ross et al.[14]

Conclusion

Giant/large aneurysms located distally on the supratentorial arteries are rare, and their infrequency has limited the understanding of these lesions. On the basis of pathologic studies and angiographic findings, most of lesions seem to be dissecting aneurysms. Although advances in micromaterials and endovascular techniques allow selective occlusion in most of intracranial aneurysms, including those located distally, the PAO remains an efficient and relatively safe strategy in case of peripheral giant/large aneurysms, which tend to recanalize if treated selectively, and the only endovascular treatment in lesions with a fusiform/serpiginous morphology. In case of giant partially thrombosed aneurysms, the elective PAO causes shrinkage of the aneurysm and reduction of the mass effect. Our results at long-term follow-up after PAO confirm persistent exclusion of the aneurysm, progressive shrinkage of the lesion, and good clinical tolerance to the vessel occlusion.

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