

MICROSURGICAL TREATMENT OF THE INTERHEMISPHERIC ARTERIOVENOUS MALFORMATIONS

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From a total of 364 patients who underwent surgery for AVMs, 46 (12.63%) had lesions located interhemispherically. The majority of patients have entered the 4th and 5th age decade. The youngest operated patient was 18 years old and the oldest was 64. The most frequent clinical signs encountered were headaches (13–28.2%), epilepsy (21–45.65%), motor deficits (12–26.08%), sensitivity disorders (8–17.39%), speech disabilities (4–8.69%), visual field deficits (3–6.52%), mental disorders (5–10.85%) and alteration of consciousness (4–8.69%). The initial imagistic examination consisted of a noncontrast computed tomographic scan, followed by a magnetic resonance angiography (MRA) and a digital subtraction angiography. We included the 46 patients into the Spatzler-Martin scale based on the location, the proximity to the eloquent areas, as well as on the type of the venous drainage. Next we show the number of patients included in every grade of the scale. Grade I–7 (15.21%) patients, Grade II–17 (36.95%) patients, Grade III–19 (41.30%) patients and Grade IV–3 (6.52%) patients. An interhemispheric surgical approach was used for these AVMs. Excellent and good postoperative results had been obtained in 37 (80.43%) patients, fair results in 5 (10.86%), poor in 2 (4.34%) and 2 (4.34%) patients have died. One of the 2 deaths was caused by a haematoma in the bed of the AVM and the other was caused by a pulmonary embolism.

Key words: brain, interhemispheric arteriovenous malformation, corpus callosum, microsurgical resection.

INTRODUCTION

The intracranial arteriovenous malformations (AVMs) represent abnormalities of vascular development with tangles of tortuous abnormal arteries and veins that permit single or multiple direct connections and high-flow shunting between them without intervening capillary beds. These AVMs look like a ball of worms that contains conspicuous gliotic nonfunctional neural tissue and vascular or interstitial calcification.

The aetiology of intracranial arteriovenous malformations remains unknown, but recent studies suggested a role for genetic factors in both susceptibility and disease progression¹. Nevertheless, sporadic AVMs are supposed to be most likely determined by the interaction between genetic and

environmental factors², and are capable of expanding by angiogenesis and rupture.

Interhemispheric AVMs are located at the levels of the medial parasagittal cortex, corpus callosum, basal ganglia, thalamus and pineal region and represent approximately 13% of all cerebral AVMs.

Due to the fact that after only endovascular embolisation and stereotactic radiosurgery the occluded vascular mass remains in place acting as a pseudotumoural process that leads to a pseudotumoural postocclusion syndrome, we have managed interhemispheric AVMs primarily and effectively by microsurgical excisions. After locating the AVM, the pseudotumoural postocclusion syndrome is characterised through headaches, mental disorders (affective, emotional, mnestic, motivational, behavioural etc.), epileptic seizures and/or neurological disorders.

Individualised treatment and patient selection for surgery was based on multiple variables, including age, medical status, and the grading scale that predicts the outcome.

PATIENTS AND METHODS

From a total of 364 patients suffering from AVMs, whom I performed surgery on between 1982-2011, 46 (12.63%) had interhemispheric AVMs.

Table 1 shows the location of those AVMs

Table 1

Classification of interhemispheric AVMs according to location

Location of interhemispheric AVMs	No. of patients
Frontal	19 (41.30%)
Parietal	13 (28.26%)
Frontoparietal	6 (13.04%)
Occipital	1 (2.17%)
Parieto-occipital	2 (4.34%)
Corpus callosum	2 (4.34%)
Pineal region	3 (6.52%)
Total	46

We noticed a slight dominance of male patients, because they represented 56.52% (26 patients) of the total.

The majority of patients have entered the 4th and 5th age decade because they added up to 50% (23 patients) of the total. The youngest operated patient was 18 years old and the oldest was 64.

The most frequent clinical signs encountered were headaches (13–28.2%), epilepsy (21–45.65%), motor deficits (12–26.08%), sensitivity disorders (8–17.39%), speech disorders (4–8.69%), visual field deficits (3–6.52%), mental disorders (5–10.85%) and alteration of consciousness (4–8.69%). Several patients showed multiple symptoms.

The initial imagistic examination consisted of a noncontrast computed tomographic (CT) scan, followed by a magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA).

In order to confirm the diagnosis and to correctly highlight the feeding arteries and the venous drainage, all patients underwent a digital subtraction angiography.

Thus, CT scans provided a more accurate view of the relationship of the AVM to the cortical surface and important subcortical structures, including the ventricular system and deep hemispheric nuclei. In addition, it can establish accurately the location of clots associated with recent haemorrhage from the AVM. The

combination of information provided by CT, MRI and MRA has made it possible to remove AVMs of significant size and those who involve eloquent areas of the brain with reduced surgical mortality and morbidity associated with total resection of these lesions.

In this study, I included the 46 patients into the Spetzler-Martin scale based on the location, the proximity to the eloquent areas, as well as on the type of venous drainage.

All these patients underwent microsurgical treatment.

Grade I – 7 patients (15.21%)

Grade II – 17 patients (36.95%)

Grade III – 19 patients (41.30%)

Grade IV – 3 patients (6.52%)

Grade V – no patient

MICROSURGICAL TREATMENT

The management options for AVMs available to the clinician include surgical excision, endovascular embolisation, stereotactic radiosurgery, a combination of two or more of these techniques, or, in some difficult cases, conservative treatment.

Standard practice advises surgery for every AVM, regardless of its location or patient's symptoms. It is excluded for all cases in which the risk of surgery exceeds that of the natural history of the disease. The primary goal in the treatment of interhemispheric AVMs is to eliminate disastrous haemorrhages, especially when surgery is performed after the initial bleed, by removing the malformation before it produces permanent neurologic deficits or death. Preoperative preparation, neuroanaesthesia with adequate cerebral protection, and careful microsurgical resection were the cornerstones of care. The barbiturate-induced electroencephalographic burst suppression was the important element in the neuroanaesthetic plan. Thiopental was the barbiturate we used for titration of burst suppression.

SURGICAL INDICATIONS

The interhemispheric arteriovenous malformations that are recommended for surgery are the ruptured ones with large haemorrhages, frequent seizures, obvious neurological deficits, and headache.

The presence of an important mass effect determined by a haematoma led to its emergency evacuation and a later approach of the malformation.

Therefore, it is recommended that a resection of interhemispheric AVMs does not take place under emergency conditions, but approximately 3–4 weeks after the haemorrhage, when the blood has liquefied and may facilitate the resection of the AVMs^{3,4,5}.

Small and medium interhemispheric AVMs may be resolved surgically, but the very large ones often need a multimodal therapeutic approach with embolisations, stereotactic radiosurgery and surgery.

A failure of endovascular treatment or stereotactic radiosurgery is an important indication for surgical approach in AVMs.

An incomplete radiosurgical treatment, checked after 2–3 years, which led only to a partial obliteration of the AVM, does not lower the risk of it bleeding again^{5,6,7,8}.

Endovascular embolisation, useful in the treatment of large supratentorial AVMs, is not as efficient in the case of deep malformations which generally receive their blood supply from the thalamoperforating, lenticulostriate or choroidal branches which are vessels with functional roles and a small lumen that is difficult to cannulate^{5,6,9}.

CONTRAINDICATIONS OF SURGICAL TREATMENT

The contraindications of surgical treatment similar in all types of AVMs are: altered neurological status of the patient, medical history that contraindicates surgical treatment, very old age, and the location in eloquent areas (sensorimotor cortex, visual and speech cortex).

In order to improve their neurological status these patients often benefit from palliative surgeries such as a ventriculostomy for an acute hydrocephalus or a ventriculoperitoneal shunt for a secondary hydrocephalus.

RISKS

The surgical risk is directly proportional to the size, location, complexity of the arterial supply and the venous drainage of the malformation.

The surgical risk and the risk of postoperative morbidity are higher for interhemispheric AVMs than for the ones on the cortical convexity.

Obvious risks show also the thalamic, basal ganglia or pineal region AVMs, as well as those located in the proximity of eloquent areas on the medial surface of the cortex (motor area, sensitive area and visual area).

Resection of critical brain or damage caused by intraoperative haemorrhage or its control is easily understood as a cause of new or permanent neurological deficits.

The size of the AVM, the presence of a deep venous drainage, and deep perforating arteries, each have been correlated with the development of new neurological deficits^{6,10,11,12}.

SURGICAL APPROACH

Interhemispheric arteriovenous malformations affect the medial cortical surface of the cerebral hemispheres, the structures of the median line in the proximity of the lateral ventricles, the corpus callosum, the pineal region, basal ganglia and thalamus.

These malformations show different anatomical and surgical characteristics depending on their location along the interhemispheric fissure.

For the AVMs located on the medial surface of the brain we used an interhemispheric surgical approach, because the majority was situated deep on the median line or on the medial surface of the cortex. The position of the patient's head depends on the location of the AVM. For frontal and parietal AVM the patient is placed in dorsal decubitus, with the head rotated to the opposite side of the lesion, and for posterior parietal and occipital AVMs the patient lies in ventral decubitus, with the lesion facing upward or in lateral decubitus with the affected hemisphere situated inferiorly. Some surgeons prefer the sitting position.

ANTERIOR LESIONS

The surgical approach to the anterior interhemispheric AVMs is made on the side of the major feeding vessels. These patients are positioned supine with the head slightly elevated.

We perform a laterally based U-shaped incision at the level of the scalp. The craniotomy should be made large enough and must extend the midline, in order to provide access to the interhemispheric fissure.

There follows a U-shaped incision with medial pedicle of the dura, meaning with the base along the sagittal sinus.

With the help of bipolar coagulation the median margin of the cortex is freed from the falx cerebri and then retracted with a spatula in order to isolate the malformation.

The cortically important bridging veins, between which the AVM is approached, must be maintained intact.

Placing the retracting spatula must be performed with care to avoid lesions of the corpus callosum,

cingulate gyrus, vessels afferent to the malformation or pericallosal and callosomarginal arteries.

The malformations located in the proximity of the gyrus rectus receive arterial supply from branches of the A1 segment of the anterior cerebral artery (Figs. 1 and 2).

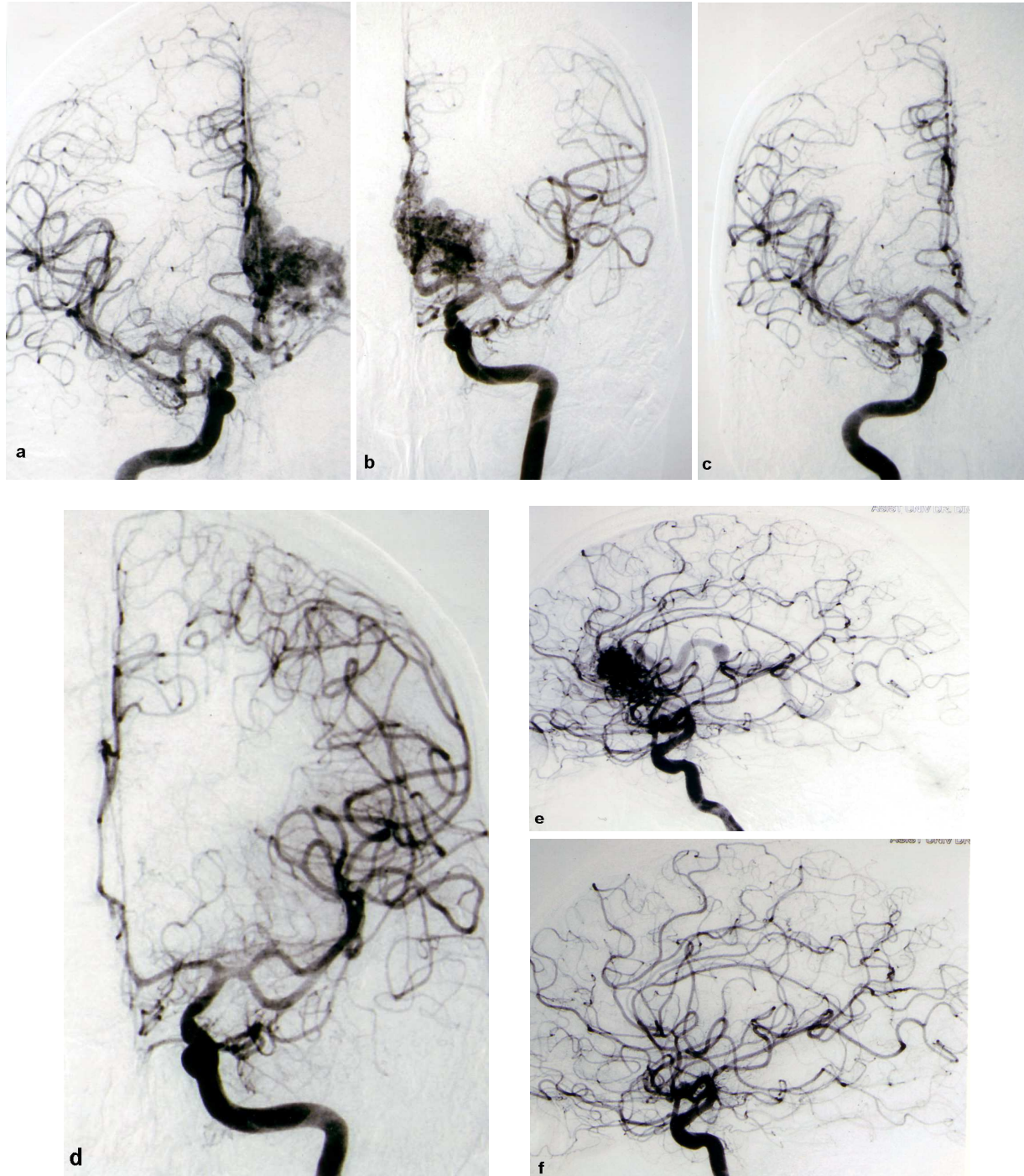


Fig. 1. Right and left anteroposterior (a, b) and lateral (e) angiography demonstrates a large, deep anterior interhemispheric AVM, involving especially the left side, the anterior commissure, the genu and rostrum of corpus callosum. The AVM is fed by multiple branches of anterior cerebral, and anterior choroidal arteries. The postoperative angiography demonstrates that a complete resection was achieved without residual nidus or early venous shunting (c, d, f). Postoperative, the patient remains in an excellent state.

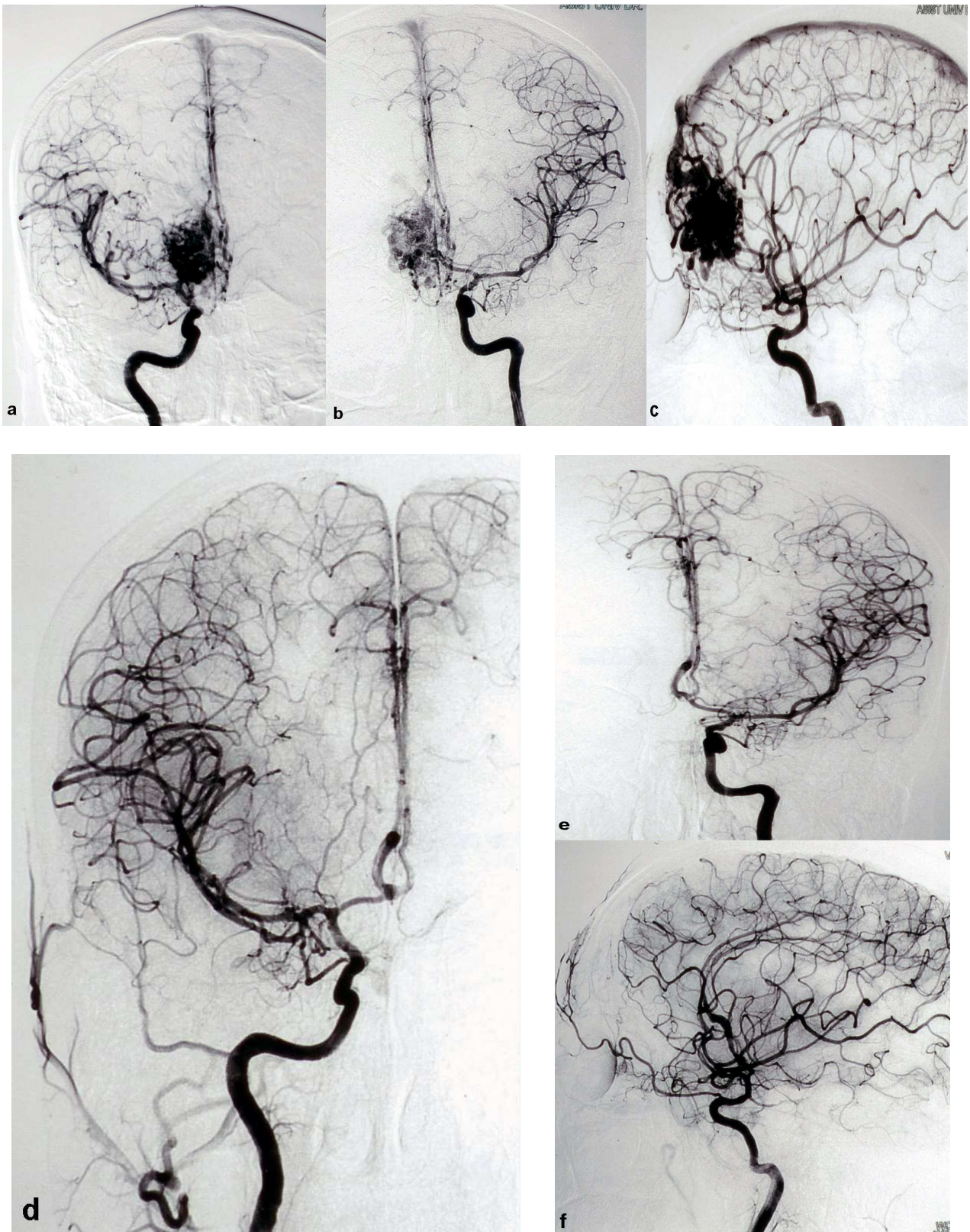


Fig.2. Right and left anteroposterior and lateral angiography shows a large anterior interhemispheric AVM involving especially the right frontal lobe. The AVM is fed by branches of the anterior cerebral arteries (a, b, c). Postoperative angiography demonstrates no residual malformation (d, e, f) and the patient remains in an excellent state.

Depending on their size, these malformations may develop laterally and may recruit vessels from branches of the middle cerebral artery. For larger and more extensive lesions the arterial supply comes from the medial lenticulostriate arteries and from the recurrent artery of Heubner. They may extend to the ventricle, caudate nucleus and the frontobasal region. The AVMs that have developed in the anterior third of the corpus callosum receive feeding arteries from the proximal region of the A2 segment of the anterior cerebral artery. For AVMs of the corpus callosum the arterial supply is usually bilateral. The venous drainage may be superficial in the superior and inferior sagittal sinus, or profound in the thalamostriate veins of the ventricles.

Red veins draining the AVM must be maintained intact until the feeding arteries are occluded.

For anterior lesions that extend into the medial striatum and hypothalamus, an initial subfrontal approach allows access to feeders from the anterior cerebral and anterior communicating arteries.

A subsequent interhemispheric approach can be made to gain control of pericallosal and callosomarginal branches (Figs. 3 and 4). So, during the operation, the vessels feeding the AVM are identified rostral to the malformation, are coagulated with bipolar forceps and are divided. When feeding arteries are not apparent on the cortical surface it is necessary to identify first the pericallosal artery which is followed proximally until the feeding vessels are identified.

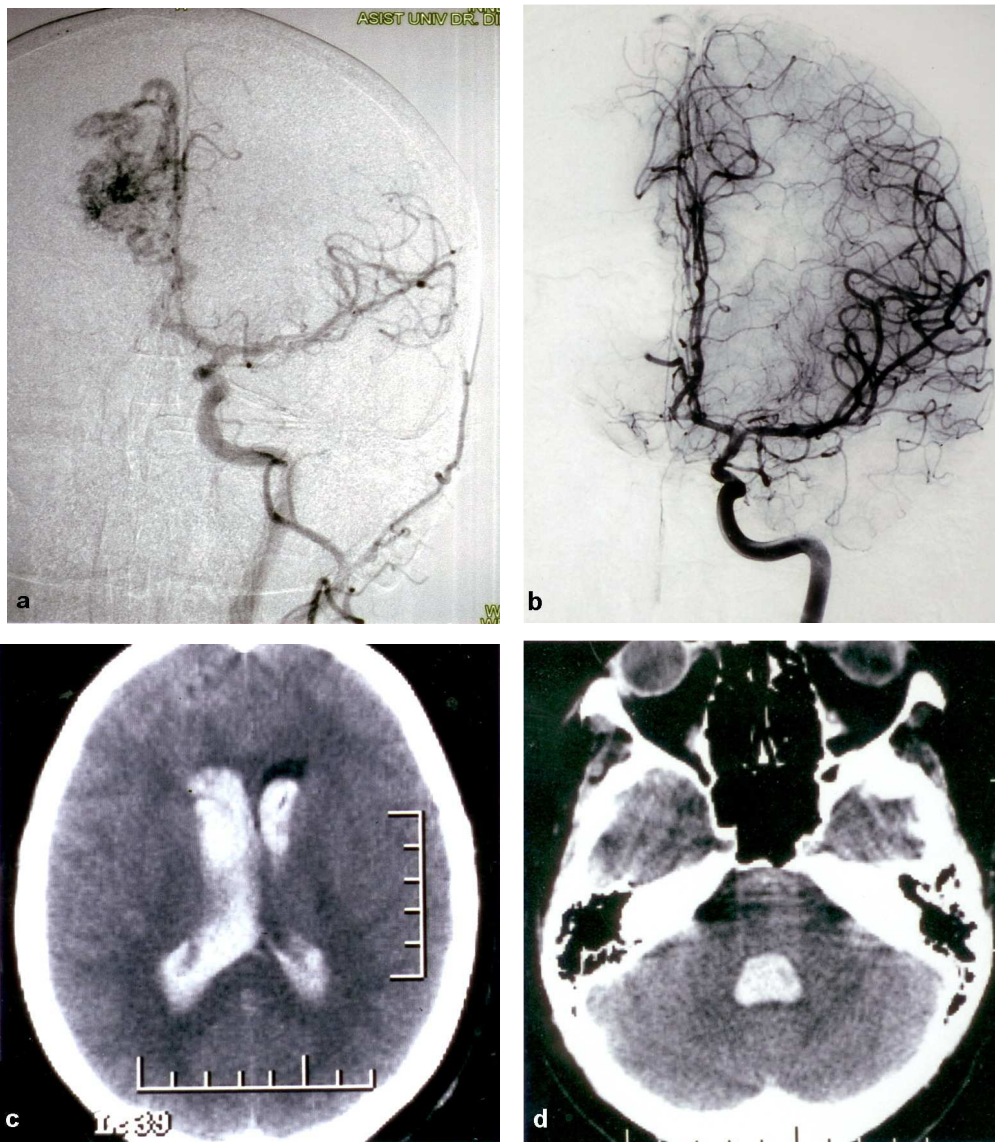


Fig.3. Left anteroposterior angiography demonstrates a large, interhemispheric AVM with multiple feeding branches from the pericallosal and callosomarginal arteries (a). Postoperative angiogram made after excision of the AVM, shows no residual malformation (b). The preoperative noncontrast computed tomographic scan showed intraventricular haemorrhage (c, d)

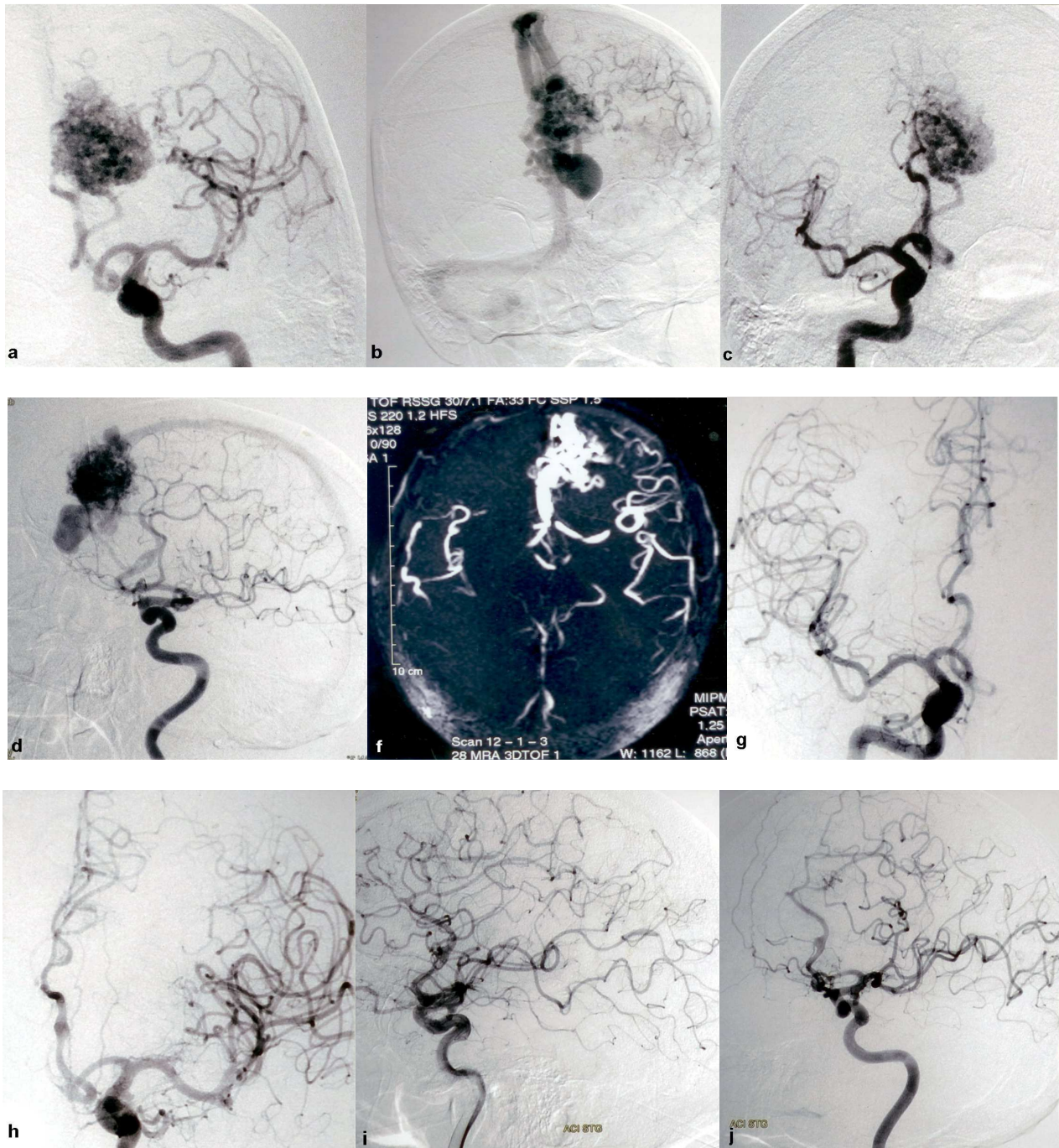


Fig. 4. Left and right, anteroposterior and lateral angiography (a, b, c, d, e) and angio- MRI (f) demonstrate an interhemispheric AVM with large aneurysms. Postoperative angiography demonstrates that complete resection of the AVM and the aneurysm was achieved without residual nidus (g, h, i, j). The patient remains in an excellent state.

In order to excise the callosal AVMs that drain into the internal cerebral veins, the approach must be carried into the ventricular system. When working within the ventricle, the internal cerebral veins should be spared to avoid the risk of venous infarction. Injury of the fornices must also be avoided to prevent the disabling memory deficit that results from bilateral damage^{6,13,14,15}.

The dissection and excision of the malformation was performed under the surgical microscope. I never used aneurysm clips for the occlusion of large feeding arteries.

It is important to identify and avoid coagulating normal arterial vessels that only pass through the malformation (en-passage vessels) but not participate to its blood supply.

The sacrifice of pericallosal and callosomarginal arteries carries a risk of lower limb weakness, so these arteries should be skeletonised as they pass through the malformation^{6,16}.

Once exposed, the ventricular wall must be protected with cottonoid patties in order to prevent blood accumulation in the ventricular system.

MIDDLE THIRD LESIONS

The AVMs from the middle third of the interhemispheric fissure are usually difficult to approach surgically, because of the important bridging veins, and venous circulation that drain the respective malformation and because of the normal sensorimotor cortex (Figs. 5 and 6).

The arterial supply comes from branches of the pericallosal or callosomarginal arteries, and when the malformation extends towards the ventricles, it comes from branches of the posterior choroidal arteries.

The venous drainage may be obtained through the superficial system, in the superior or inferior sagittal sinus and sometimes in the profound system in the ependymal veins and internal cerebral veins.

POSTERIOR LESIONS

The AVMs located in the posterior third of the interhemispheric fissure, that comprise malformations located in the posterior parietal and occipital regions, are approached through a large parieto-occipital craniotomy extended medially to the sagittal sinus and inferiorly to the transverse sinus. This allows a parafalcine approach.

The dural flap is reflected medially, and the occipital pole, which rarely has bridging veins, is retracted laterally and superiorly. The falcotentorial angle is followed to the incisura, where the terminal pericallosal artery and the posterior cerebral artery trunk, often involved in these AVMs, can be exposed.

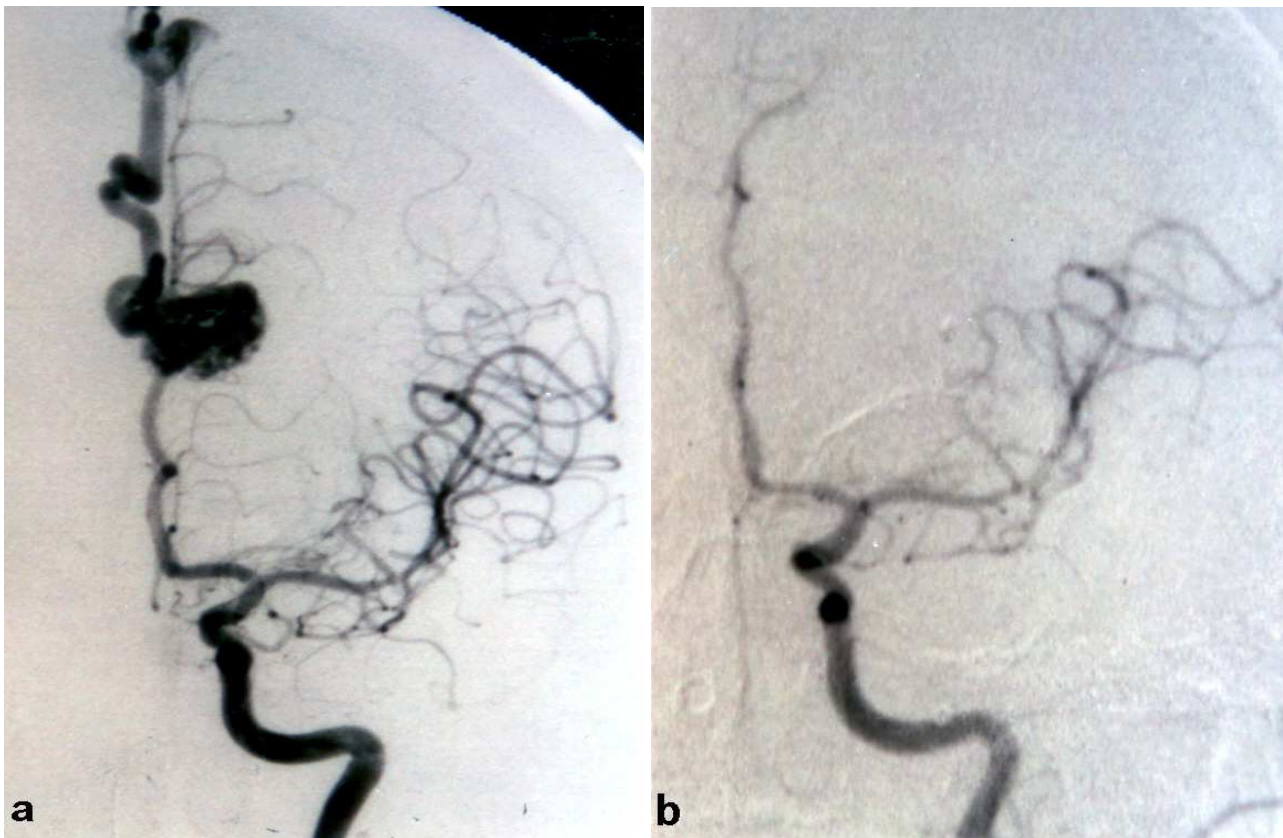


Fig. 5. Left anteroposterior carotid angiography (a) demonstrated an interhemispheric AVM fed by an enlarged segment A4 of the anterior cerebral artery. Postoperative anteroposterior left carotid angiography demonstrated complete resection of the AVM (b). The patient remains in an excellent state.

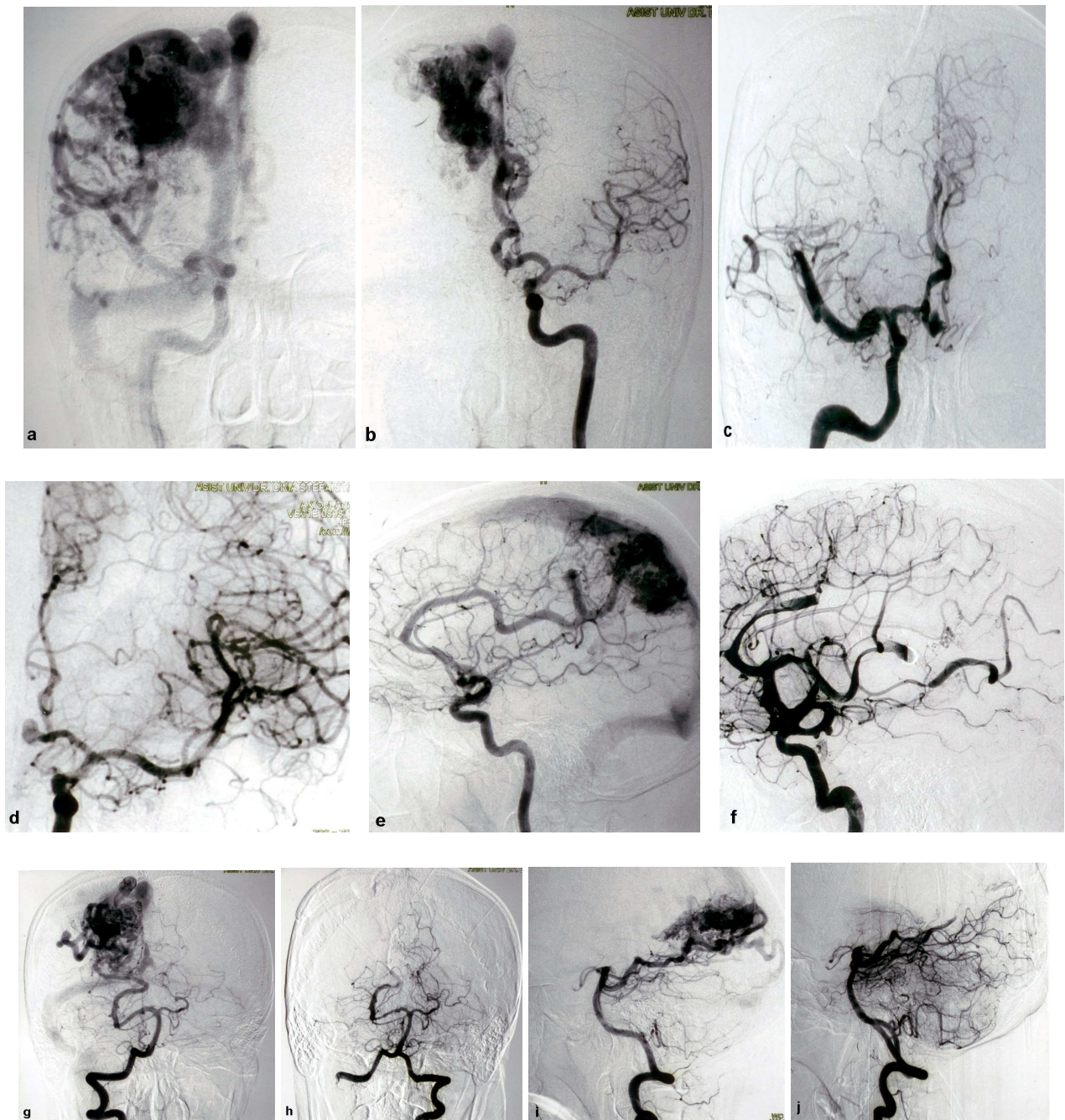


Fig. 6. Right and left anteroposterior and lateral carotid and right vertebral arteries angiography, demonstrates a large interhemispheric AVM, involving especially the right parietal lobe (a, b, e, g, i). This AVM receives multiple feeding arteries from anterior cerebral arteries, right middle cerebral and posterior cerebral arteries. Postoperative angiography (c,d, f, h, j) showing enlarged right anterior and middle cerebral arteries and no residual malformation. The patient remains only with a right homonymous hemianopsia.

The artery is traced distally until the feeding arteries of the AVM which are found ventrally, are identified, coagulated with bipolar forceps and divided.

The malformations at the level of the cuneus and precuneus that are adjacent to the parieto-occipital sulcus, and the ones that comprise the isthmus of the cingulate gyrus or the lingula,

receive arterial supply from branches of the posterior cerebral artery, the calcarine and the parieto-occipital artery, from branches of the middle cerebral artery and from posterior branches of the posterior cerebral artery.

Large malformations that reach the ventricular trigone, receive arterial supply from the posterolateral choroidal arteries.

Posterior parieto-occipital and occipital lesions have arterialised vein draining into the superior sagittal and inferior sagittal sinus or into the vein of Galen. After the feeding vessels are interrupted, the malformation is excised from the medial cortex.

The falcotentorial approach is also effective for the AVMs located far anterior at the level of the pineal region. During the immediate postoperative period 2 of 3 of my patients with parieto-occipital and occipital AVMs had transient homonymous hemianopsia sometimes accompanied by visual phenomena.

Thus, posterior interhemispheric AVMs occupy the medial occipital or inferomedial temporal cortex, hippocampus, posterior thalamus, splenium of the corpus callosum, or the pineal region.

These AVMs are fed by the posterior pericallosal branches of the anterior cerebral artery, anterior temporal, posterior choroidal, thalamoperforating, middle cerebral meningeal and distal branches of the posterior cerebral artery. Veins of the posterior AVMs drain into the galenic system, sagittal and lateral sinus.

SPLENIAL AND PINEAL REGION LESIONS

Splenium AVMs that are rarely symmetrical should be approached from the side of the greatest extension.

The surgical approach of these malformations is posterior interhemispheric with the patient in a semi-sitting position (slouch position) or in a lateral position and the dependent ipsilateral hemisphere allows gravity to assist with retraction^{6,16,17}.

By retracting the medial surface of the hemisphere, the falx cerebri and the pericallosal artery can be seen.

The pericallosal artery traced distally leads us to the feeding arteries of the AVM.

However, the AVMs at the level of the splenium of the corpus callosum receive arterial supply from the posterior portion of the pericallosal arteries, the posteromedian and posterolateral choroidal arteries, as well as from the direct branches of the posterior cerebral artery. The venous drainage goes towards the basal vein of Rosenthal and the vein of Galen. Pericallosal branches enter the malformation anteriorly, and posterior cerebral feeders enter from the quadrigeminal cistern. The feeders from lateral posterior choroidal branches

are difficult to expose. After performing a corticectomy at the level of the cingulate gyrus and after the coagulation and division of the feeding arteries, we can identify the anteroposterior margin of the malformation. The falx may be divided to improve access to the contralateral side. An incision into the cingulate gyrus and the division of the splenium in the direction of its fibres allows greater lateral exposure.

A section along the axons of the splenium allows the identification and coagulation of the feeding arteries from the posterolateral choroidal artery. The vascular supply from the posterolateral choroidal artery can be encountered in splenial AVMs with lateral extension towards the medial surface of the ventricle trigone.

The AVM lying in the pineal region adjacent to the collicular or geniculate region can be exposed through an interhemispheric approach which is a long one (Fig. 7). Ventrally located thalamic AVMs may be approached through an infratentorial, supracerebellar operation.

Frequently the surgeon encounters venous drainage to the internal cerebral veins or the vein of Galen before arriving at the arterial feeders.

Caution is required in managing the deep-draining venous system so that no injury occurs to the internal cerebral veins or the vein of Galen.

Partial section of the corpus callosum may be necessary. Apparently, partial section of the corpus callosum causes little neurologic deficit. However, when the splenium is completely divided there appears the possibility of causing dyslexia¹⁸.

The interhemispheric approach is the route farthest from the visual pathways, but the calcarine artery supply to the occipital lobe may be compromised inadvertently^{19,20}.

Microsurgery is the gold standard for the definitive treatment of AVMs.²⁰ compared the results of grade I to III AVMs treated microsurgically with stereotactic radiosurgery. With microsurgical treatment, there were statistically significantly fewer postoperative haemorrhages, neurological deficits and deaths and a higher incidence of obliteration. Additionally, these investigators reported a significant difference between the two groups in haemorrhage-free survival.

However, all treatment modalities have risks that must be weighted carefully against the natural history risk during the treatment and decision-making process.

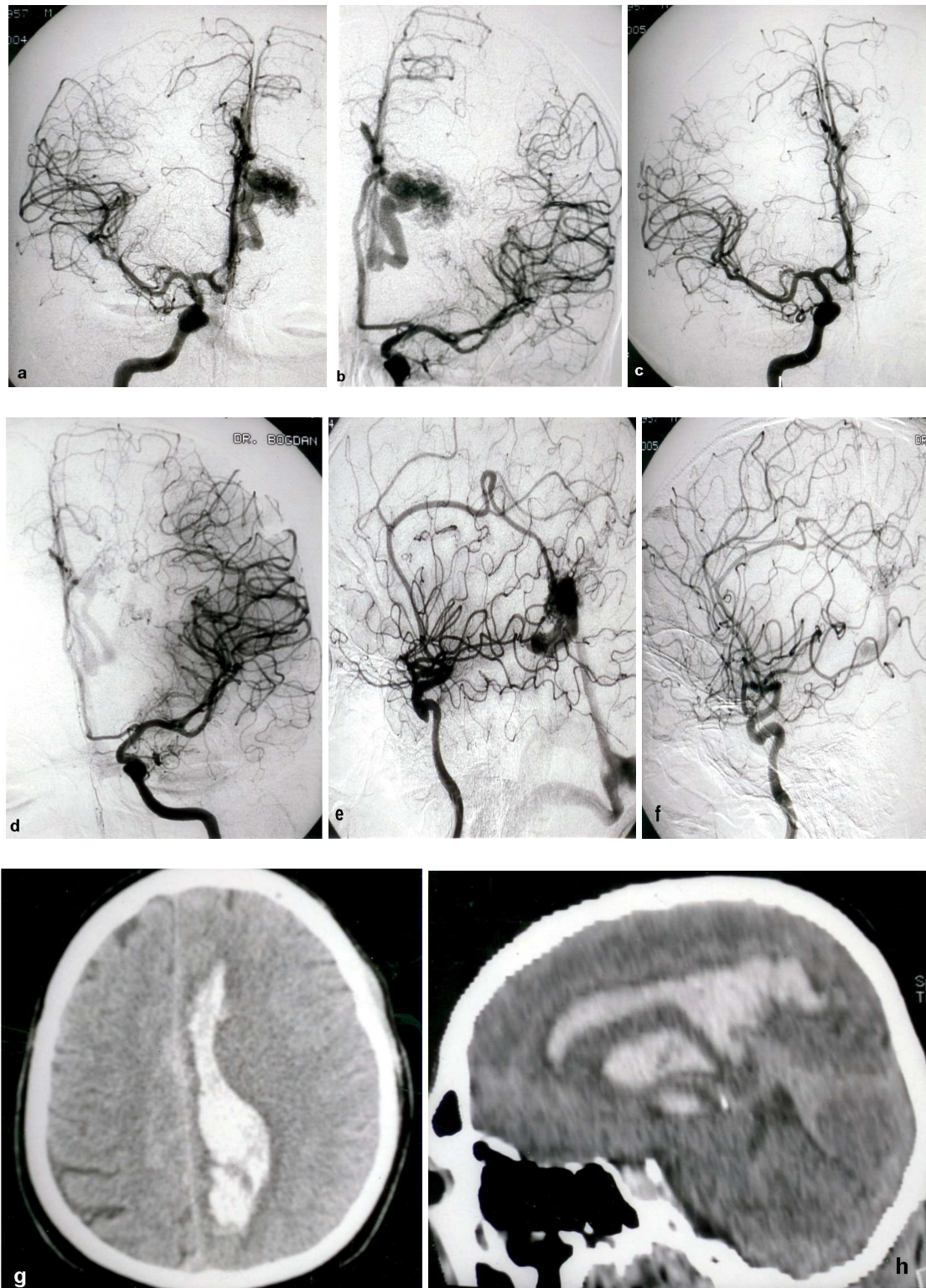


Fig. 7. Preoperative right and left anteroposterior (a, b) and lateral (e) carotid angiography showing a large pineal region AVM fed by anterior cerebral and posterior choroidal arteries and draining into the deep venous system. The postoperative left and right angiography (c, d, f) demonstrated complete resection of the AVM. The patient remains in a very good state. Preoperative axial and sagittal CT scan (g, h) shows intracerebral parietal and intraventricular haemorrhage.

POSTOPERATIVE CARE

Patients are generally extubated in operating room as soon as possible after surgery. By contrast, in dangerous situation such as large and deep AVMs, or when surgery was difficult and coating was necessary, sedation and ventilation should be maintained for 24 hours or longer if necessary. Postoperative computed tomography scans help in deciding when to reduce sedation and allow the patient to breathe spontaneously²¹.

Prophylactic antibiotics, dexamethasone, and anticonvulsants are continued during the immediate postoperative period. A smooth awakening and extubation are critical to avoid blood pressure elevations, Valsalva manoeuvres, or coughing and straining, which may cause immediate postoperative haemorrhage^{6,22}. If haemostasis is tenuous, maintaining the mild hypotension used during surgery may be wise in order to keep the blood pressure at a reduced systolic level for 24 hours postoperatively to prevent haemorrhagic complications caused by normal perfusion pressure breakthrough. The blood pressure control is absolutely necessary to avoid haemorrhage from the resection bed or adjacent brain. Steinberg and Stoodley (2000) maintain the mean arterial pressure at 65 to 75 mmHg for 1 to 2 days.

In case of residual AVMs or if there was troublesome bleeding during surgery, the mean arterial pressure is maintained at 55 to 65 mmHg for the first 2 days²².

One potential complication in the early postoperative period is venous thrombosis of the enlarged draining veins. This sudden reduction of the venous flow may result in parenchymal swelling, haemorrhage, and severe neurologic deficits.

Cerebral angiography was performed during the first few days after surgery to confirm complete excision of the AVM.

RESULTS

An angiographic control had been performed on 44 operated patients. In one (2.27%) of them, with a grade IV AVM on the Spatzler-Martin scale, we saw a residual nidus, so that complete resection of AVMs was proven in 97.73% of cases.

Our postoperative result evaluation scale comprises 5 categories: excellent, good, fair and poor results and deaths (Table 2).

In the category of excellent and good results we included the patients who did not show any neurological deficits or epileptic seizures late after surgery (6 months after surgery).

Patients with fair results showed minor or medium postoperative neurological deficits (moderate hemiparesis, homonymous hemianopsia, Parinaud syndrome) but could lead an independent life.

Patients with poor results remained dependent, due to severe neurological deficits (severe hemiparesis) and epileptic seizures.

Out of the 46 patients who underwent surgery for their interhemispheric AVMs, the postoperative results were excellent and good in 37 (80.43%) patients, fair in 5 (10.86%), poor in 2 (4.34%) and 2 (4.34%) patients have died. The deaths and poor results represented 8.68% of operated patients. The majority (42–95.45%) of operated patients did not present any new neurological deficits immediately after surgery. Neurological deficits were seen only in patients who already had neurological deficits prior to surgery.

Table 2

Postoperative results

Spetzler-Martin Grade	No. of patients	Excellent and good	Fair	Poor	Deaths
1	7	7 (100%)	0	0	0
2	17	16 (94.11)	1 (5.88%)	0	0
3	19	13 (68.42%)	4 (21%)	1 (5.25%)	1 (5.25%)
4	3	1 (33.3%)	0	1 (33.3%)	1 (33.3%)
5	0	0	0	0	0
Total	46	37 (80.43%)	5 (10.86%)	2 (4.34%)	2 (4.34%)

The highest rate of excellent and good results has been recorded in patients with grade I and II AVMs on the Spatzler-Martin scale (95.83%), fair results have been recorded especially in patients with grade III AVMs (21%), while poor results have been recorded in patients with grade IV AVMs (33.33%).

The postoperative angiography confirms the excellent surgical techniques employed. Out of 21 (45.65%) patients who presented with seizures and who underwent excision of AVM, 18 (85.71%) were cured by surgery. The remainder had persistent seizures.

Of the 2 deaths recorded during the postoperative interval of 30 days, one was caused by a complication related to the surgical act (the haematoma in the bed of the AVM) and the other one was caused by a pulmonary embolism.

DISCUSSION

The management of these complicated lesions can include open surgery, endovascular techniques, stereotactic radiosurgery, or some combination of these modalities.

Therapy varied from patient to patient, and careful planning and a meticulous technique were critical. An alternative to microsurgical resection and embolisation, **stereotactic radiosurgery**, has profoundly impacted on the management of cerebral arteriovenous malformations^{23,24}. The underlying mechanism of action for radiosurgery is thought to be a gradual endothelial hyperplasia of the abnormal vasculature, which, in turn, leads to progressive narrowing and vessel occlusion over a 2- to 3-year period^{25,26,27}. During that interval, the patient has no protection from haemorrhage because of the delay involved in achieving changes after radiation.

In the event of failure to completely obliterate the AVM nidus on the first attempt of radiosurgery, the options include microsurgery, endovascular therapy, conservative follow-up, and repeat radiosurgery^{24,26,28,29,30,31}.

Embolisation can be used before the removal of large AVMs to reduce flow to the AVM, to obliterate deep vascular pedicles that are not easily accessible through the planned surgical approach, and to reduce postoperative neurologic deficits^{21,24,32,33,34,35,36,37,38,39}.

So, it is used as an adjunct to surgery or radiosurgery in order to reduce the size of the lesion or its blood flow⁴³ and to contribute positively to the outcome.

As an alternative to stereotactic radiosurgery and embolisation, we used microsurgical resection on a large scale.

I decided to excise these malformations without preoperative embolisation. This decision was based on the fact that I felt I would have early surgical access to the major feeding branches that came especially from the anterior cerebral arteries bilaterally.

The morbidity associated with any treatment of large AVMs is high. For patients with AVMs greater than 3 cm the reported significant morbidity in the early postoperative period ranges from 40% to 75%, with approximately 30% of patients having a moderate to severe disability at late follow-up^{11,32,40}.

According to Fisher and Harrigan (2009)²⁷, surgical resection is an effective primary approach after haemorrhages of the AVM, when the control of seizures is a priority, for smaller AVMs and those involving noneloquent cortex, and for patients who would prefer a "fast cure".

In a review of surgical results, it was determined that the surgical risk of permanent neurological morbidity and mortality for grade I to grade III patients was low (0%). In higher grade patients, the risks were considerably higher: Grade IV patients incurred a 21.9% risk, and grade V patients incurred a 16.7% risk¹¹.

Thus grade IV and V AVMs present a difficult therapeutic challenge.

The result of surgical resection is, on the whole, disappointing, however, with a range of permanent severe new deficits or death in 9% to 44% of patients undergoing surgery^{11,12,40,41,42}. The great variation in outcome may reflect surgical triaging.

Complications leading to permanent or life-threatening disabilities can be attributed to embolisation, resection of eloquent brain, postretraction oedema and haemorrhage, AVM rupture, myocardial infarction, arterial-capillary-venous hypertensive syndrome, vasospasm, aneurysm rupture, new seizure development, extracerebral haematomas, complications of blood product replacement, infections, and deep venous thrombosis^{42,43}.

Associated aneurysms may be intranidal, on feeding arteries, on major arteries of the circle of Willis, or on arteries not related to the AVM. Intranidal aneurysms and aneurysms on feeding arteries close to the nidus carry the highest risk of haemorrhage⁶⁵.

Of the complications leading to **permanent neurological deficits**, 83% are present on emergence from anaesthesia, and 17% develop later but within the first 9 days after surgery⁴². This compares with an incidence of 1% to 5% in the population screened without AVMs⁴⁴.

Thus, the rates of the **new permanent neurological deficit** leading to a downgrade in the quality of life in grade IV-V AVMs is 44% to 57% and the rate of severe disability leading to loss of independence is 11% to 22%^{11,40,45}.

AVMs with a nidus size less than 3 cm have reported a complication rate of new permanent neurological deficits of 1.5% to 2.7 with an angiographically confirmed obliteration in 99% to 100%^{11,46,48}.

Of these, 4.3% have worsened in eloquent brain and 1.6% in noneloquent brain⁴⁸. Although 83% of deficits occur during surgery, 17% arise in a delayed fashion from the development of the arterial-capillary-venous hypertensive syndrome within the first 8 days after surgery⁴².

Vasospasm occurs as a complication of AVMs resection in only 2% of cases but is reported to occur in 27% of cases involving extensive dissection of the proximal middle and anterior cerebral arteries⁴⁵.

The new seizure disorder usually follows supratentorial brain surgery. Early authors reported an increase in seizure frequency after surgery for AVMs^{49,50,51,52}. Afterwards large series have found an incidence of postoperative seizures of less than 40% in patients with a history of preoperative seizures and less than 10% in patients without such a history^{53,54,55,56,57}.

CONCLUSION

AVMs have four components: feeding arteries, a nidus, draining veins and parenchymal gliotic nonfunctional elements.

Complex AVMs, including lesions in eloquent areas of the brain, have not been avoided in this surgical series, and our results indicate that a microsurgical approach to AVMs can result in a higher cure with an acceptable rate of morbidity and mortality.

As a general guide, open AVM resection is considered the gold treatment for small, medium and large interhemispheric AVMs, in ideal circumstance because microsurgery is superior to other modalities in terms of obliteration rate, post-

treatment haemorrhage rate, risk of neurologic morbidity, treatment-related death, and cost-effectiveness.

Outcome for grade III AVMs may be influenced strongly by the presence of deep perforating arterial supply⁵⁹. Thus, grade III AVMs without deep perforating supply should do well with surgery alone. For grade III AVMs with deep perforating arteries that are less than 3 cm in diameter, consideration must be given to radiosurgery⁴⁵.

In grade IV and V AVMs undergoing surgery, many are treated with preoperative embolisation or with stereotactic radiosurgery.

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