ARTERIOVENOUS MALFORMATIONS IN THE TEMPORAL LOBE: MICROSURGICAL TREATMENT AND RESULTS IN 89 CASES

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The current study is based on the analysis of 89 patients with surgically removed temporal lobe AVMs. In gender distribution we recorded a dominance of male patients with a sex ratio of approximately 1.5:1. As far as age is concerned we recorded incidence peaks in the 3rd and 4th decade. The most frequent onset pattern involved complex and generalised epileptic seizures as well as intracerebral haemorrhages. Hemiparesis, aphasia and hemianopsia were the neurological signs encountered most frequently. We recorded 48 (53.93%) AVMs at the level of the left temporal lobe, and 41 (46.06%) at the level of the right temporal lobe. 61 (68.53%) patients suffering from temporal lobe AVMs had a haemorrhagic imaging onset. The following is the classification of the 89 patients according to the Spetzler-Martin scale: Grade I - 21 (23.59%); Grade II - 38 (42.69%); Grade III - 24 (26.96%); Grade IV - 6 (6.74%); Grade V – 0. After the microsurgical removal of the AVMs in the above mentioned 89 patients we obtained excellent and good results in 82 (92.13%) patients, fair results in 3 (3.37%) patients, poor results in 3 (3.37%), and 1 (1.12%) patient died. We obtained our best results in patients suffering from Grade I and II AVMs on the Spetzler-Martin scale.

Key words: Arteriovenous malformation, temporal lobe, brain, 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 shutting between them without intervening capillary beds. These AVMs look like a ball of worms, and contain between them conspicuous gliotic nonfunctional neural tissue and vascular or interstitial calcifications.

Although the aetiology of intracranial arteriovenous malformations remains unknown, recent studies suggested a role for genetic factors in both susceptibility and disease progression (Young and Yong, 2004)

Nevertheless, sporadic AVMs are supposed to be most likely determined by the interaction between genetic and environmental factors (Fontanella *et al.*, 2012) and are capable of expanding by angiogenesis and rupture.

AVMs may increase in size, remain stable, decrease in size, completely regress or develop thrombosis over time (Hook and Johanson, 1958; Waltimo, 1973, Trumpy and Eldevik, 1977; Parkinson and Bachers, 1980, Wharen *et al.*, 1982, Ebeling *et al.*, 1988, Minikawa *et al.*, 1989, Pasqualin *et al.*, 1993, Guazzo and Zuered, 1994). Spontaneous regression can be acute or gradual and occurs in approximately 2% to 3% of patients with AVMs (Robinson *et al.*, 1993). Causes include low flow, occlusion of feeding vessels by atherosclerosis, embolisation, dissection or haemorrhage due to mass effect or vasospasm (Wharen *et al.*, 1982, Pasqualin *et al.*, 1993, Marconi et al., 1993, Abdulrauf *et al.*, 1999).

The risk of mortality or morbidity from an untreated AVM must be balanced against the risks associated with any treatment. Factors that enter into this decision include age, medical condition, neurologic condition, life style and psychological conditions (Steinberg and Stoodley, 2000) and experience of the surgeon.

The natural course of the arteriovenous malformations represents a risk of rebleeding of 2 to 4% annually, with a 1% risk of mortality and a 2.7% combined risk of major morbidity and mortality annually for these patients (Brown *et al.*, 1988; Ondra *et al.*, 1990).

As chief of the only vascular neurosurgical ward in Romania, which I grounded in 1982, I was bound to microsurgically remove many cerebral AVMs with very good results. Back then, but also currently, the possibility to treat these lesions through endovascular embolisation or radiosurgery were and remained extremely limited.

Because the AVMs treated only through embolisation or radiosurgery were not removed, a pseudotumoural occlusive syndrome characterised, depending on the location of the lesions, by headaches, asthenia, attention and memory deficits, mental and neurological disorders, appeared or persisted.

Microsurgery is superior to other modalities in term of obliteration rate, post-treatment haemorrhage rate, risk of treatment-related death or neurologic morbidity and cost-effectiveness.

MATERIALS AND METHODS

Patients and methods

I performed surgery on a number of 89 patients with arteriovenous malformations.

As far as the gender distribution in concerned we recorded a majority of male patients with a sex ration of approximately 1.5:1. Thus, out of the 89 patients with operated AVMs, 54 (60.67%) were males and 35 (39.32%) were females.

The age distribution recorded incidence peaks in the 3rd and 4th decade (Table 1).

Table 1

Age and sex distribution of 89 patients
with temporal lobe AVMs

Age	Male	Female	Total
10	0	0	0
11-20	7	2	9 (10.11%)
21-30	13	15	28 (31.46%)
31-40	22	12	34 (38.20%)
41-50	7	3	10 (11.23%)
51-60	3	3	6 (6.74%)
61-70	2	0	2 (2.24%)
Total	**54(60.67%)**	**35(39.32)**	**89**

I did not have patients in the extreme age groups. Therefore, I did not have any paediatric cases under the age of 10 years and no patient older than 70 years.

Out of the very young patients who underwent surgery for temporal lobe AVMs we would like to mention: 3 male patients aged 14, 17 respectively 19 years, two of whom with an AVM located at the level of the left temporal lobe (Spetzler-Martin Grade II), and the last one with a right temporal lobe AVM (Spetzler-Martin Grade I) and a female patient aged 20 years, who underwent surgery for a right temporal lobe AVM (Spetzler-Martin Grade I).

The symptomatology of the operated temporal lobe arteriovenous malformations was variable. Besides the intracranial hypertension syndrome, the most common presentations of AVMs were epilepsy, progressive neurologic deficit, headaches, migraine headaches and cardiac failure. Adding to the epileptic seizures, the symptomatology of ruptured AVMs had also been influenced by the location, left or right, and the extent of the haemorrhage. Hemiparesis, aphasia and hemianopsia represented the most frequently seen neurological signs.

Table 2 shows the main neurological and mental symptoms and signs of the operated 89 patients.

Table 2

Main neurological and mental symptoms and signs of 89 patients operated for temporal lobe AVMs

Symptoms	No. of patients
Intracranial hypertension	39 (43.82%)
Motor deficit (hemiparesis)	28 (31.46%)
Speech disorders (aphasia)	19 (21.34%)
Homonymous hemianopsia	22 (24.71%)
Facial paresis of the central type	6 (6.74%)
Epilepsy	44 (49.43%)
Mental disorders	9 (10.11%)
Balance disorders	10 (11.23%)
Changes of conscious state	9 (10.11%)

Out of the 44 (49.43%) patients with epilepsy, 24 (26.96%) presented with generalised seizures, 19 (49.43%) presented with psychomotor epileptic seizures (of the temporal lobe type) and 1 (1.12%) had simple Jacksonian seizures.

As far as the location of the 89 operated AVMs at the level of the hemispheres is concerned we point out the fact that we recorded 48 (53.93%) lesions at the level of the left temporal lobe and 41 (46.06%) at the level of the right temporal lobe

Based on an analysis of specialty literature, I consider that the increased incidence on the left is purely incidental and that there are no correlations with diverse aetiopathogenic or clinical factors or a predilection of AVMs in the left temporal lobe.

Imaging

According to their imagistic (computed tomography, magnetic resonance imaging and angiography) location, the temporal lobe AVMs were categorised as follows:

- Temporo-lateral AVMs - 72 (80.89%)
- Temporo-basal AVMs - 13 (14.60%)
- Temporo-medial AVMs - 0
- Mixed AVMs - 4 (4.49%)

Concerning the imaging onset of the operated temporal lobe AVMs, we point out the fact that we recorded 61

(68.53%) patients with intracranial haemorrhages and 28 (31.46%) with unruptured AVMs. In case of an onset with rupture of the malformation, the secondary intracranial haemorrhage was located as follows:

- Intraparenchymal haemorrhage - 41 (67.21%).
- Intraventricular haemorrhage (single) - 4 (6.55%).
- Intraparenchymal and intraventricular haemorrhage - 9 (14.75%).
- Intraparenchymal and subarachnoid haemorrhage - 3 (4.91%).
- Intraprenchymal haemorrhage with subdural haematoma - 2 (3.27%).
- Subdural haematoma - 2 (3.27%).

As far as discovering unruptured AVMs, for 17 patients the initial investigation consisted of a computed tomography (CT) which showed a tumoural formation, and for 11 patients the initial investigation consisted of magnetic resonance imaging (MRI), which showed a typical flow-void aspect for the vascular malformation.

To understand the angioarchitecture of the AVMs including the feeding arteries, draining veins and their juxtaposition, the preoperative evaluation of our patients included four-vessel cerebral angiography and MRI examination.

The Spetzler-Martin classification of the operated AVMs was the following:

- Grade I   21 (23.59%)
- Grade II   38 (42.69%)
- Grade III   24 (26.96%)
- Grade IV   6 (6.74%)
- Grade V   0

Treatment of cerebral AVMs

There are three techniques available to treat AVMs: conservative treatment, single modality treatment and multimodality treatment.

Defenders of surgery point out that immediate protection against rebleeding and the avoidance of delayed radiation-related brain injury offers distinct advantages over stereotactic radiosurgery (Deruty *et al.*, 2000).

Whenever surgery is considered, the potential risk of mortality or morbidity from the operation must be weighed against the risks posed by the natural course of the disease process.

It is excluded for all cases in which the risk of surgery exceeds that of the natural history of the disease. This is an annual rate of haemorrhage from 2% to 4%, rising to 6% during the year after an initial bleed (mean interval 7 to 8 years between two episodes of haemorrhage), an annual rate of morbidity of 3% and a mortality rate of 1% for each haemorrhagic episode (Sisti *et al.*, 1993, Nusbaum *et al.*, 1995, Friedman, 1995, Kondziolka *et al.*, 1995, Pollock *et al.*, 1996).

These figures should be kept in mind when considering aggressive treatment for difficult malformations (Deruty *et al.*, 2000). The neurosurgeon must interpret the known data towards keeping mortality and morbidity from surgery on AVM to a minimum.

The lesions that are small or modest in size and those located outside of eloquent areas of the brain are easy to operate. If left untreated, these lesions are just as dangerous in term of recurrent haemorrhages or neurologic deficit as are those located in the more inaccessible or critical regions of the brain.

Microsurgical resection

The use of preoperative steroids, lumbar drainage, and manitol depends on the specific lesion and the condition of the patient. Maintenance of normotension during surgery is recommended. The head is fixed in a rigid device.

In all cases, the bony flap should encompass the entire cortical aspect of the malformation. A well-placed and wide enough craniotomy contributes to minimise brain retraction.

The opening of the dural flap should be performed very carefully because there are often thickened arachnoid strands attached to the external aspect of the AVM, which must be coagulated and divided.

In addition, the surgeon should be aware that large draining veins may be adherent to the dura or even incorporated in it before draining into the dural sinuses (Yasargil, 1988).

Based on MRI and angiography the microsurgical extirpation of temporal lobe AVMs consists first of a meticulous dissection of fissures, sulci, and subarachnoid cisterns to control the feeding arteries, preservation of vessels en passant and surrounding neural tissue, skeletonisation of venous drainage, followed by isolation and resection of the nidus and finally, transection of venous drainage. Feeding arteries should be transected as near to the nidus as possible to allow any surrounding parenchyma supplied by the vessel en route to the nidus to continue receiving blood (Clatterbuck *et al.*, 2005).

In order to isolate and keep the vessels en passant it is important to have a wide opening of the sylvian fissure and to dissect the middle cerebral artery past the AVM.

So, if it is clear that the artery enters the AVM and is not a vessel en passant, it can be coagulated and cut.

If the nidus does not reach the cortical surface, a red arterialised vein may be present and can be followed back to the AVM. However, microsurgical resection of temporal lobe AVMs is sometimes difficult and is still associated with a significant risk.

The care with which draining veins need to be approached is obvious. Otherwise, an early sacrifice of the venous drainage of an AVM may have disastrous consequences.

The resection of anterior AVMs needs a large opening of the sylvian fissure in order to follow the anterior choroidal artery to the level of the choroid fissure (Figs. 1 and 2).

Malformations located in the medial part of the temporal lobe categorise into anteromedial and posteromedial AVMs. For the anteromedial temporal lobe AVMs, the approach is performed through a pterinoal craniotomy and through a large opening of the sylvian fissure (Figs. 3 and 4). For the posteromedial temporal lobe AVMs, I most often prefer a transtemporal approach through the inferior temporal gyrus, over the subtemporal one because it is more direct and decreases the risk of injuring the vein of Labbe (Fig. 5). Two male patients (28 and 36 years) and a female patient (31 years) had aneurysms associated to the arteriovenous malformation. In two cases, the aneurysm was located on the main feeding vessel of the AVM, respectively on the right middle cerebral artery, and in the last case we had an intranidal aneurysm. In all three patients we performed a surgical cure for both the aneurysm and the arteriovenous malformation.

Another patient showed a left temporal lobe AVM which was removed and a right parietal venous malformation which we did not operate on.

Partial resection of the AVM does not protect against the risk of fresh haemorrhage.

Gabriel *et al.* (1996) reported a rare case of regrowth of a malformation and subsequent bleeding, after an angiographically confirmed complete excision.

RESULTS

The following are the evaluation criteria for the operated patients:

- **Excellent** – no neurological deficits, able to work.
- **Good** – with minimal mental and neurological deficits, able to work.
- **Fair** – in order to work they require assistance because of the impossibility to walk on their own and because of the mental and physical symptoms.
- **Poor** – is not able to work, even if assisted.
- **Dead** – we recorded deaths after the surgery, during hospitalisation

Starting with these criteria we show the postoperative results of the 89 patients with AVMs of the temporal lobe, in Table 3.

Table 3

Surgical management and results in 89 cases

Spetzler-Martin Grade of the AVM	Excellent and Good	Fair	Poor	Dead	Total
	21	0	0	0	21
I	37	1	0	0	38
II	22	1	1	0	24
V	2	1	2	1	6
	0	0	0	0	0
Total	82 (92.13%)	3 (3.3%)	3 (3.37%)	1 (1.12%)	89

From Table 3 it is obvious that we obtained the best results in patients with Grade I and II AVMs on the Spetzler-Martin scale, whose excellent and good results of the surgery summed up to 98,30% of the patients, recording no major deficits or deaths.

For Grade III we obtained excellent and good results in 22 (91.66%) patients, fair results in 1 (4.54%) and poor results in 1 patient. For patients with grade IV AVMs we recorded 2 (33.33%) poor results (mixed aphasia and right hemiplegia), 1 (16.66%) fair result (mixed aphasia) and 1 death.

The death was caused by an important postoperative haematoma in the operative bed. The female patient, aged 46 years, operated for a left temporal lobe AVM, bled in the first 24 hours after surgery. We operated again but the evolution was rapid and not favourable, leading to the death of the patient. We must mention that the neurological status before the surgery was altered. However, the size, location, and vascular anatomy are the critical factors in predicting good outcomes in AVM resection.

Nevertheless, the results depend not only on the size, location and microarchitecture of the lesion, but also on the talent, tenacity, perspicacity of mind and accuracy of the microneurosurgeon.

None of the patients who had proven total resection have subsequently had a recurrent haemorrhage, and a number of these have been followed for a decade.

The overall morbidity among my 89 patients was related to the location of the lesion, but not necessarily to its size.

So, those lesions that extended deep into the central portion of the brain and were fed by deep arteries were associated with a higher rate of morbidity and permanent complications.

Seizure disorders present before surgery were neither aggravated nor significantly ameliorated after operation.

Yeh *et al.* (1990) demonstrate that a complete cure of epilepsy may be achieved in 70% of cases provided surgery is directed not only at the resection of the AVM but also at the removal of epileptic foci remote from the AVM area.

Headaches present preoperatively were alleviated after the resection of the AVM.

An ischemic deficit does not seem to be greatly influenced by resection. In general, surgery increases such deficits in cases of large malformations.

On the other hand, Piepgras *et al.* (1993) find on follow-up a good capacity for recovery of previous or new deficits.

Deruty *et al.* (2000) found considerable variations in outcome in the published series. Roughly, the results were good outcome (or no deterioration), 52% to 82%; fair outcome (or minor deterioration), 4% to 29%; poor outcome (major deterioration) 2% to 8% and death, 1 to 14%.

Nusbaum *et al.* (1995) evaluate surgical outcome separately in two different groups of AVMs: (1) small and medium-sized AVMs, as well as large malformations located in silent areas; and (2) large malformations with deep extension, located in or adjacent to critical areas.
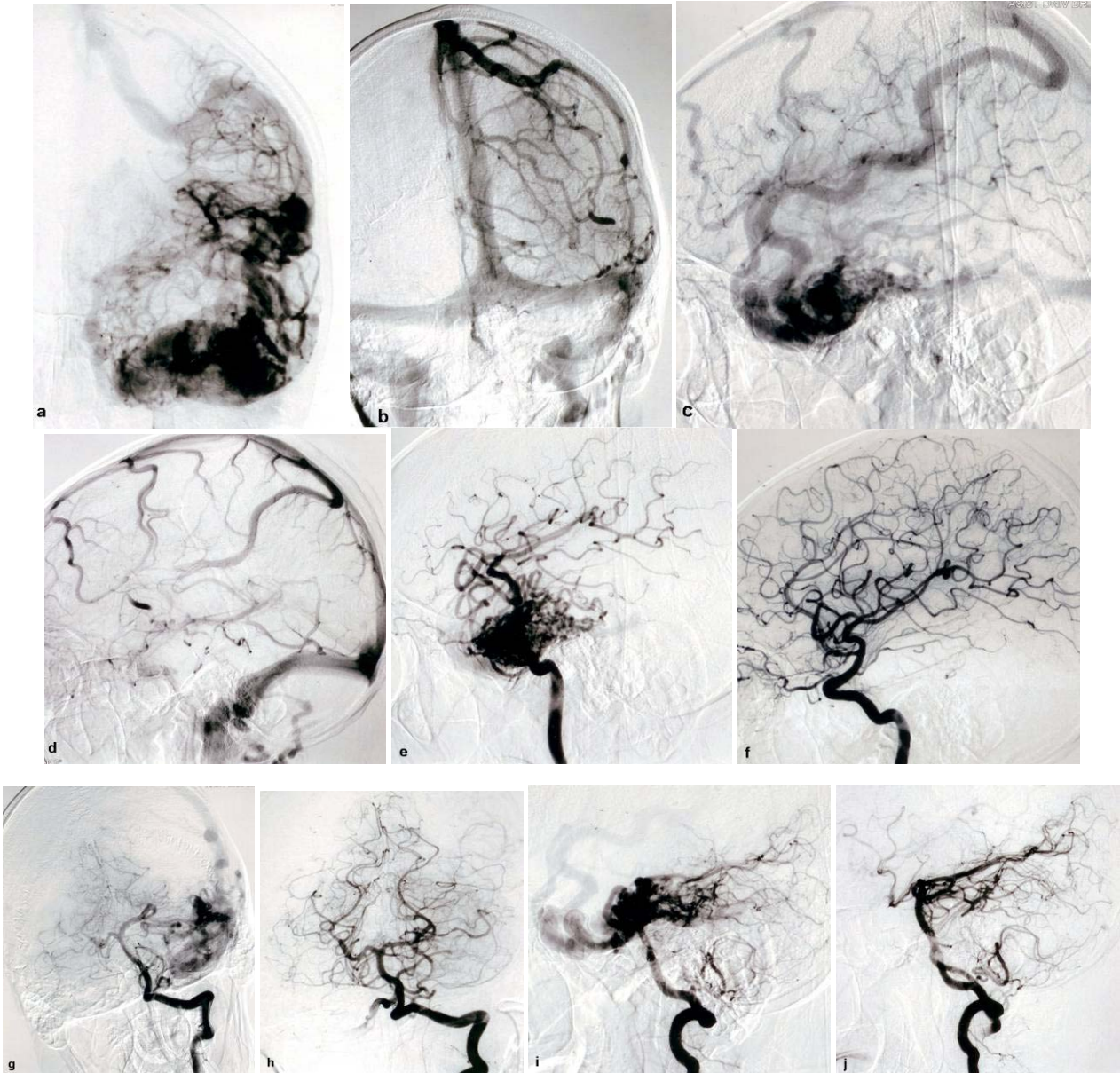
Fig. 1. Anteroposterior (a) (early-phase) and lateral (c, e) (early- and late-phase) of the left internal carotid angiography, and early-phase on the anteroposterior (g) and lateral (i) vertebral artery angiography reveals a very large AVM situated in the anterior left temporal lobe, fed by multiple branches from carotid artery, and middle cerebral artery. The superficial draining veins are very large. Postoperative internal carotid and vertebral angiography (b, d, f, h, j) demonstrate complete reaction of the AVM. The patient remains in an excellent state.
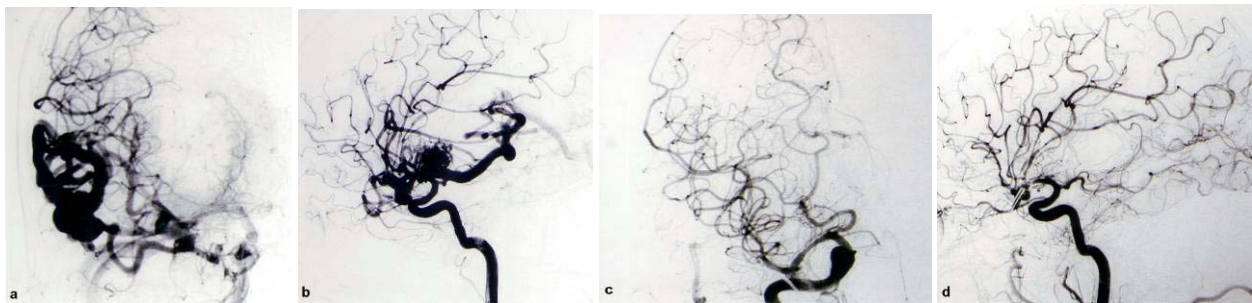

Fig. 2. Anteroposterior (a) and lateral (b) right internal carotid angiography showed a large AVM situated in the anterior right temporal lobe and a large middle cerebral artery aneurysms. Postoperative, right internal carotid angiography (c, d) demonstrated complete resection of the AVM and completely clipped of the aneurysm.
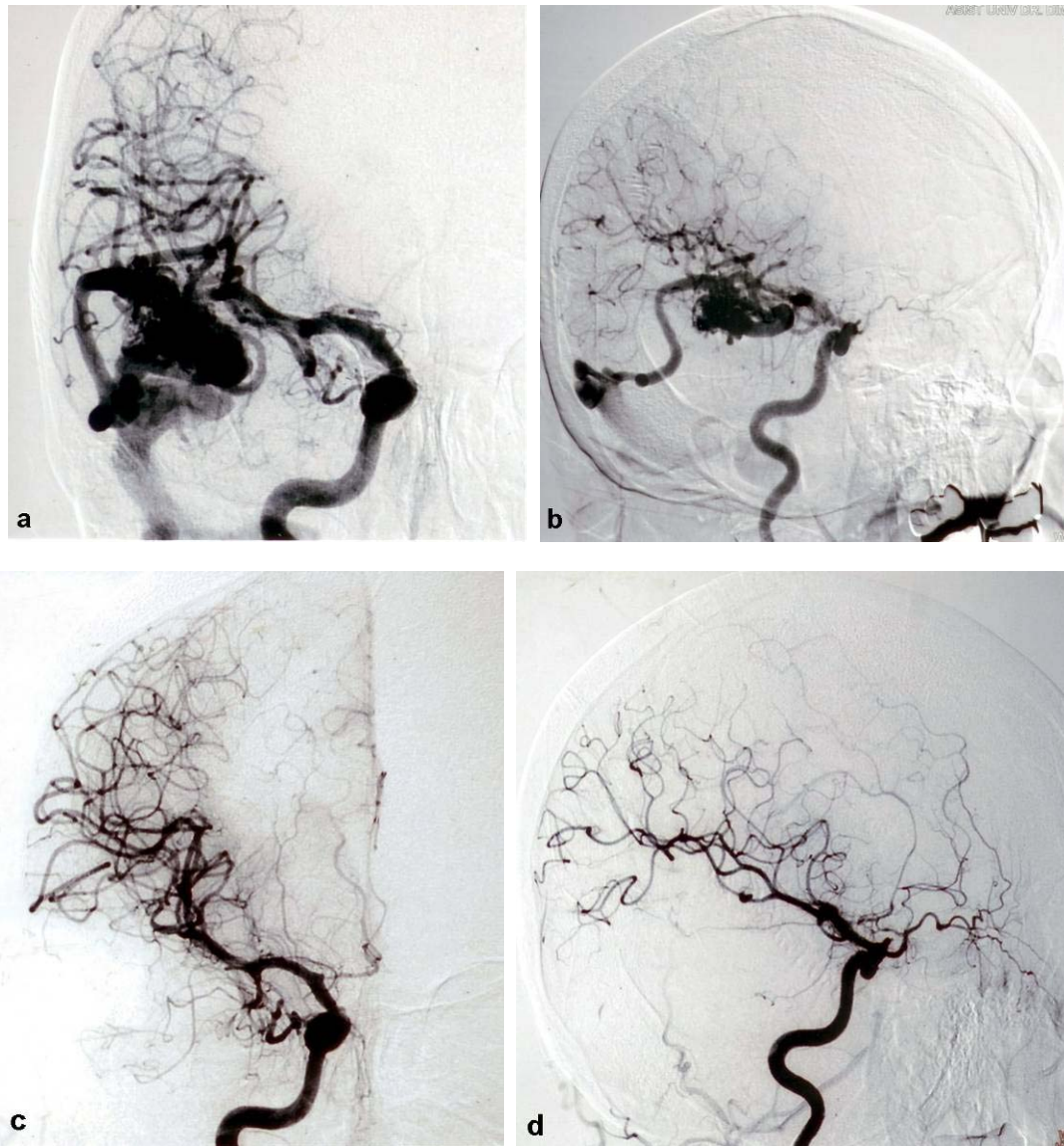
Fig. 3. The anteroposterior and lateral of the right internal carotid artery (a, b) show a temporal AVM fed by branches of middle cerebral artery. Postoperative angiography (c, d) demonstrates no residual malformation.
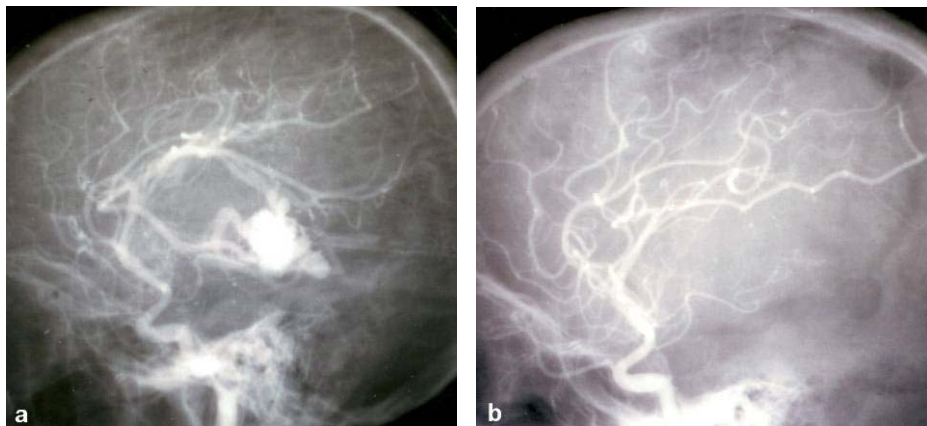

Fig. 4. The early arterial phase of the lateral angiogram of the right internal carotid artery, show a small middle cerebral artery AVM, supplied by the right middle cerebral artery (a). The early phase of the lateral postoperative angiogram of the right internal carotid artery show that complete resection of the AVM was achieved (b). The patient remains in a very good state.
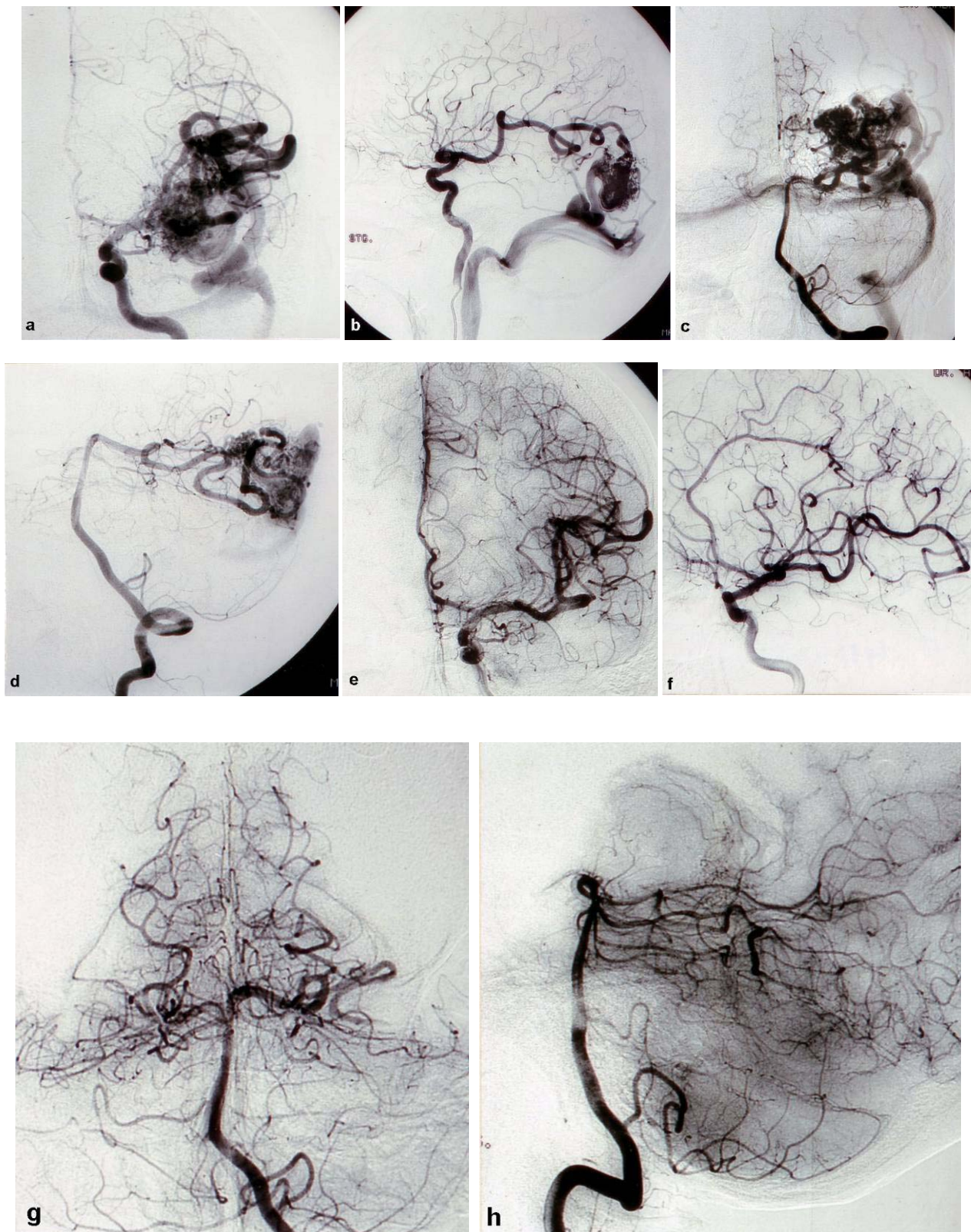
Fig. 5. The ateroposterior and lateral of the left internal carotid artery and vertebral artery (a, b, c, and d) show a giant arteriovenous malformation in the left temporo-occipital region. The AVM is supplied by branches of the posterior middle cerebral artery and posterior cerebral artery who are enlarged. Postoperative carotid (e, f) and vertebral (g, h) angiography demonstrates complete excision of the AVM. The patient remains in an excellent state.

In the first group, the combined mortality and morbidity rate with surgery was approximately 10% (Nusbaum *et al.*, 1995). In a series consisting only of microsurgical resection of small malformations (<3 cm in diameter), the morbidity and mortality rates dropped as low as 1.5% and 0% respectively (Sisti *et al.*, 1993). In the second group, the rate of serious morbidity/mortality was as high as 50% (Nusbaum *et al.*, 1995).

Aneurysms associated with AVMs occur in 3% to 23% of the cases and are commonly multiple (Perata *et al.*, 1994, Thompson *et al.*, 1998, Shen and Wang, 1998).

Aneurysms are at risk of bleeding before, during or after treatment of the AVM (Thompson *et al.*, 1998). Aneurysms may disappear after resection of the AVM (Perata *et al.*, 1994, Kondziolka *et al.*, 1995).

Small aneurysms that are not on feeding vessels and are not exposed to higher pressures after surgery can be treated similar to asymptomatic aneurysm not associated with AVMs.

DISCUSSION

As with any surgical procedure, careful patient selection is crucial to ensuring optimal treatment outcomes. Patient selection for microsurgical treatment of AVMs is based on multiple variables including age, medical status and a grading scale that predicts the outcome of surgical intervention (Spetzler and Martin, 1986; Han *et al.*, 2003; Dănăilă *et al.*, 2010).

Presurgical plans may include cortical functional mapping, such as functional magnetic resonance imaging, and subcortical mapping, such as tractography, to predict and inform the patients of the risk of neurological deficits (Nozaki and Hashimoto, 2005).

Direct microsurgery which is recommended for small, medium-sized and sometimes for large AVMs, depends on the experience of the physician and on the patient's wish.

For large malformations (>3 cm in diameter), especially those located in eloquent or deep areas, multimodality treatment (embolisation, surgical resection or stereotactic radiosurgery), is increasingly the treatment of choice (Deruty *et al.*, 2000; Dănăilă *et al.*, 2010). In 1960, Luessenhop and Spence first described embolisation as treatment of cerebral AVMs. The benefit of embolisation before surgical resection or

stereotactic radiosurgery is mainly of reducing the size and volume of the lesion.

However, the risk of the treatment should not be greater than that of the disease's natural history.

There are many controversies about the treatment of very large and complex malformations, those located in eloquent brain areas, or those with deep extensions.

Controversy remains over whether to undertake any treatment at all (Standard and Hopkin, 1996).

However, there are two goals in the treatment of brain AVMs: complete removal of the malformation and preservation of neurological function (Sisti *et al.*, 1993, Lawton *et al.*, 1995). Partial excision is unsatisfactory because it does not reduce the danger of recurrent haemorrhage.

AVMs should be totally removed when there are progressive neurologic deficits, incapacitating seizures, recurrent haemorrhages, mental deterioration or intractable headaches (French, 1976). Standard practice advises surgery for every AVM, regardless of its location or patient's symptoms.

Microsurgical resection of cerebral AVMs can be difficult and associated with a significant risk of injury to the arterial branches of the normal brain, and neuronal structures. Hence, various technical strategies to image cerebral vascular malformations and to reduce the surgical complication rate have been proposed such as intraoperative digital subtraction angiography (DSA) colour-coded, duplex sonography, spectral Doppler sonography, microscope-based intraoperative near-infrared indocyanine green (ICG) videoangiography and navigation (Hashimoto *et al.*, 2007; Hänggi *et al.*, 2010, Takagi *et al.*, 2012).

The main difficulties with AVMs lie with the deeper portion of the nidus and the vessels that are not directly visible under the operating microscope (Sekhar, 2010).

Embolisation of deep or dominant feeding arteries can be used before removal of large AVMs because it results in a reduced operating time and less blood loss, allows clear delineation of the AVM margins, simplifying its resection and improving the safety of surgery.

Since the first published description by Steiner (1985) stereotactic radiosurgery has been used to treat more than 7000 patients worldwide.

The underlying mechanism of action for radiosurgery is thought to be gradual endothelial hyperplasia of the abnormal vasculature, which, in turn, leads to progressive narrowing and eventual

vessel occlusion (Chang *et al.*, 1997). When smaller than 2,5 cm in diameter, subsequent obliteration occurs in 80 to 85% of AVMs over a 2- to 3-year period (Steiner, 1985; Colombo *et al.*, 1989; Spiegelmann *et al.*, 1992; Colombo *et al.*, 1994; Pollock *et al.*, 1996; Maruyama *et al.*, 2004; Sinclair *et al.*, 2006, Liscák *et al.*, 2007). According to Lišcák *et al.* (2007), complications after radiosurgery are manifested as acute, subacute and delayed.

Referring to our cases we point out that the postoperative evolution of the 88 living patients has generally been very good and good. The mean total length of both clinical and radiosurgical follow-up in this study was 26.8 months (range 2 to 61 mo).

In one patient appeared a new postoperative left hemiparesis. Except for one case, the motor deficits before the surgery and the new ones improved very much due to neuromotor rehabilitation procedures. That one patient, aged 33 years, presented with a very large left fronto-temporal intracerebral haematoma.

The DSA showed a Grade IV left temporal lobe AVM on the Spetzler-Martin scale. Postoperative, both the right hemiplegia and the mixed aphasia were also present at the 12 months follow up after surgery. The predominance of left temporal lobe AVMs led to an increased percentage of aphasias.

Thus, out of the 19 (21.34%) patients with postoperative speech disorders, 11 (12.5%) had aphasias for a period of over 3 months. Even though the majority of speech disorders manifested through moderate and medium expressive aphasias determined by intracerebral haematomas extending to the motor area of speech, there were a few cases with severe aphasias and discrete recovery afterwards.

The homonymous hemianopsia present at onset in 22 (24.71%) patients was recorded postoperative in 10 (11.36%) cases. The respective symptoms appeared in patients with ruptured temporal lobe AVMs with secondary intracerebral haematomas.

It is important to notice that 44 (49.43%) patients had onset epilepsy. Postoperative, the epileptic seizures were present in 9 (10.22%) patients. In 7 patients the epileptic seizures were generalised and 2 had temporal lobe epilepsy. For these patients we revised the antiepileptic therapy. However, the computed tomography showed a right temporal haemorrhage at the level of the old AVM in one patient. By repeating the digital subtraction angiography we could see a remaining malformative nidus. In order to resolve remaining AVMs we performed a second surgery. The control cerebral angiography showed a complete removal of the nidus.

Postoperative mental disorders persisted in 5 (5.68%) patients who had left temporal lobe AVMs.

They were evident in all postoperative controls. Imagistic investigation did not show any neuro-surgical causes for the psychiatric symptomatology.

We considered as being permanent, those neurological deficits that remained unchanged after at least 1 year of follow-ups. The general status of the patients had been evaluated at discharge after surgery and also in the postoperative follow-up period which normally extended to 3 months, 6 months, 12 months and 24 months. Out of the total 89 patients with operated temporal lobe AVMs, 84 could be followed over a period of at least 2 years. The exceptions were 1 death, 3 patients who did not participate at follow-ups and 1 recently operated patient (2011) who could not be included in the minimal 2 year follow-up period.

CONCLUSIONS AND FUTURE PROSPECTS

The majority of patients with temporal lobe AVMs presented epilepsy, hemiparesis, homonymous hemianopsia, and speech disorders determined by the intracranial haemorrhage present in 61 (68.53%) cases.

MRI and four-vessel angiography was important for preoperative evaluation. The surgical approach of the AVMs with such location had been performed through a large opening of the sylvian sulcus. We interrupted the feeding arteries as close as possible to the malformation.

The experience and talent of the surgeon play the most important role during surgery.

Arteriovenous malformation surgery runs the scale from the fairly simple surgical excision of a small superficial cortical lesion to an extremely complex, demanding, and dangerous operation to remove deep-seated large lesions (Solomon, 2005).

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