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Clinical Study

Endovascular treatment of posterior fossa arteriovenous malformations

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ABSTRACT

Infratentorial arteriovenous malformations (AVM) are rare, representing only 7–15% of cerebral AVM. The concentration of eloquent neurological structures and the high rate of bleeding presentation of AVM in this location complicate the management of such lesions. New therapeutic options, especially in endovascular therapy, have fundamentally modified the treatment strategy and also the outcome of posterior fossa AVM. Between 1999 and 2013, baseline, clinical and angiographic data of cerebral AVM were prospectively collected. We analyzed data from patients treated for a posterior fossa AVM, focusing on risk factors for bleeding, and clinical and angiographic outcomes. Sixty-nine patients (mean age 34 years, male to female ratio 2:1) were consecutively treated for an infratentorial AVM. Fifty-seven presented with hemorrhage, six with focal neurologic deficits, and the remaining six patients were diagnosed incidentally. The Spetzler–Martin grade was <3 in 39 (56.5%) patients. Associated aneurysms were noted in 43.5% of patients. All patients were treated using endovascular procedures, associated with microsurgical resection in nine patients and with stereotactic radiosurgery in six. Mean follow-up was 28.5 months, with angiographic exclusion of the AVM in 72.5% of patients; 21.7% of patients presented a modified Rankin Score $\geqslant$3 at follow-up. Endovascular embolization seems to be a secure approach for posterior fossa AVM although a large number of sessions are necessary to achieve complete obliteration. Multidisciplinary discussion and management is crucial to obtain the best cure rate without increasing procedural risks.

1. Introduction

Infratentorial arteriovenous malformations (AVM) were first reported by Clingenstein [1] in 1908 and the first successful resection of such a lesion was accomplished by Olivecrona and Riives [2] in 1932. Posterior fossa AVM are rare lesions, accounting for 7–15% of cerebral AVM in most large clinical series [3–5]. The management of these neurovascular lesions is complicated by the concentration of highly eloquent neurological structures within the small space of the posterior fossa [6]. Despite advances in microsurgical techniques and the introduction of radiosurgery and endovascular embolization in recent decades, treatment of posterior fossa AVM remains a challenge. This subtype of cerebral AVM has an aggressive natural history, and reports in the literature [4,7–11] are heterogeneous in terms of the treatment strategies followed. We present our clinical and angiographic results of the management of posterior fossa AVM treated with an endovascular technique, combined with microsurgery or radiosurgery when necessary.

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2. Material and methods

2.1. Demographic data

Since 1995, we have maintained a prospective database in which the baseline clinical data including patient age, sex, clinical presentation and angiographic characteristics of the AVM have been collected. From 1995 to 2013, of the 794 patients treated for an AVM in our department, 69 patients with infratentorial AVM were treated using an endovascular approach. The treatment strategy was discussed in a multi-disciplinary manner including neurosurgeons and interventional neuroradiologists.

2.2. Preprocedural management

The diagnosis of posterior fossa AVM was confirmed by digital subtraction angiography for all patients. Cavernomas, developmental venous anomalies, vein of Galen and dural AVM were not included. All patients underwent preoperative MRI or CT imaging of the brain, to diagnose possible hemorrhage (subarachnoid hem-

orrhage or parenchymal hematoma) and confirm the infratentorial location of the AVM. The need for external ventricular drainage or suboccipital decompressive craniectomy was examined for each patient with a bleeding presentation. The treatment strategy was planned for each patient before starting the first session of embolization. In general, in patients with hemorrhagic presentation, we waited for decreases in both parenchymal edema and hematoma size before starting the first session of treatment.

2.3. Endovascular procedures

Endovascular procedures were performed under general anesthesia. After diagnostic angiography, superselective catheterization of the arterial supply of the AVM was undertaken. Treatment options included vessel occlusion or embolization of the AVM nidus with liquid embolic agent, either Onyx (ev3 Neurovascular, Irvine, CA, USA) or cyanoacrylate synthetic glue (Histoacryl, Braun, Germany or Glubran, GEM, Viareggio, Italy) and coiling for occlusion of aneurysms.

2.4. Postoperative follow-up

Follow-up started at the time of diagnosis and finished with the last visit or at angiography. Angiographic follow-up after complete exclusion of the AVM involved digital subtraction angiography at 1 year and at 5 years. The modified Rankin Score (mRS) and Glasgow Outcome Score (GOS) were recorded at each visit and at angiography. Poor outcome was defined as mRS $\geqslant 3$ or GOS $\leqslant 3$.

3. Results

3.1. Demographic data and AVM characteristics

Between 1995 and 2013, 69 patients with angiographically visible AVM were consecutively treated by endovascular treatment at our institution. Patient baseline data and AVM characteristics are described in Table 1. Mean age was 34 years (range, 4–67 years) with a male-to-female ratio of 2:1. The majority of patients (n = 57, 82.6%) presented with bleeding at admission; 21 of these patients (30.4%) had a subarachnoid hemorrhage, 23 (33.4%) a parenchymal hematoma and 13 (18.8%) had both. The clinical presentation was a focal neurological sign in six patients (8.7%); five patients presented with a trigeminal neuralgia secondary to a pontine AVM and one patient with cerebellar dysmetria secondary to an ipsilateral cerebellar hemispheric AVM. The other six patients (8.7%) had an incidental discovery of the AVM. Locations of the AVM included the cerebellar hemisphere in 42 patients (60.1%), cerebellar vermis in 22 (31.9%), cerebellar nuclei in 13 (18.8%) and brainstem in 11 (15.9%). As described by Spetzler and Martin [12], eloquent areas in the posterior fossa were considered as the brainstem, cerebellar nuclei and cerebellar penduncles. In our series, 30 AVM (43.5%) were in eloquent areas, 14 (20.3%) involved cerebellar peduncles, 14 (20.3%) involved cerebellar nuclei and 11 (15.9%) involved the brainstem. The AVM were <30 mm in size in 48 cases (69.6%) and between 30 mm and 60 mm in 21 cases (30.4%). The Spetzler–Martin grade [12] was I in 20 cases (29%), II in 19 (27.5%), III in 18 (26.1%) and IV in 12 (17.4%). No patient had a grade V AVM.

Table 1
Baseline data and arteriovenous malformation characteristics

Variable	Patients (n=69)
Age in years, mean (range)	34 (4–67)
Males	46 (66.7%)
Presentation	
Bleeding	57 (82.6%)
Subarachnoid hemorrhage	34 (49.3%)
Parenchymal hematoma	36 (52.2%)
Compression signs	6 (8.7%)
Incidental	6 (8.7%)
Localization	
Cerebellar hemisphere	42 (60.1%)
Cerebellar vermis	22 (31.9%)
Cerebellar nuclei	13 (18.8%)
Brainstem	11 (15.9%)
Eloquent area	30 (43.5%)
AVM size (mm)	
<30	48 (69.6%)
30–60	21 (30.4%)
>60	0 (0%)
Spetzler–Martin grade	
Grade I	20 (29.0%)
Grade II	19 (27.5%)
Grade III	18 (26.1%)
Grade IV	12 (17.4%)
Grade V	0 (0%)

AVM = arteriovenous malformation.

3.2. AVM angioarchitecture

The AVM angioarchitecture is given in Table 2. The mean number of arterial feeders was 2.5 (range 1–5). Arterial supply was from the superior cerebellar artery in 49 patients (71.0%), the postero-inferior cerebellar artery in 34 patients (49.3%), the antero-inferior cerebellar artery in 27 patients (39.1%) and the posterior cerebral artery in six patients (8.7%). Dural feeders from the external carotid artery were noted in four patients (5.8%). Thirty associated intracranial aneurysms were found and classified as nidal (10 cases, 14.5%), inflow (18 cases, 26.1%) and unrelated to the AVM (two cases, 2.9%). The AVM nidus was classified as compact in 50 cases (72.5%). The mean number of draining veins was 1.8 (range, 1–5). Thirty AVM (43.5%) had superficial venous drainage only, 23 (33.3%) deep venous drainage only and 16 (23.2%) had both. Venous ectasia was noted in 25 AVM (36.2%).

3.3. Treatment modality

At the acute phase of intracranial bleeding, the need for external ventricular drainage or suboccipital decompressive craniectomy was considered for each patient with a bleeding presentation. A suboccipital decompressive craniectomy was necessary in six patients (8.7%) and three (4.3%) of them required hematoma evacuation. External ventricular drainage was performed in 16 patients (23.2%) with secondary ventriculoperitoneal shunting in six patients (8.7%).

Table 2
Arteriovenous malformation angioarchitecture

Variable	AVM (n=69)
Arterial supply, mean (range)	2.5 (1–5)
Posterior cerebral artery	6 (8.7%)
Superior cerebellar artery	49 (71.0%)
Antero-inferior cerebellar artery	27 (39.1%)
Postero-inferior cerebellar artery	34 (49.3%)
Vertebral artery	3 (4.3%)
Dural artery	4 (5.8%)
Intracranial aneurysm	
Inflow aneurysm	18 (26.1%)
Intranidal aneurysm	10 (14.5%)
Unrelated to AVM	2 (2.9%)
Compact nidus	50 (72.5%)
Superficial venous drainage only	30 (43.5%)
Deep venous drainage only	23 (33.3%)
Deep and superficial venous drainage	16 (23.2%)
Number of draining veins, mean (range)	1.8 (1–5)
Venous ectasia	25 (36.2%)

AVM = arteriovenous malformation.