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Anatomic and angiographic findings of cerebellar arteriovenous malformations: Report of a single center experience

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ABSTRACT

Cerebellar arteriovenous malformations (AVM) are rare and comprise only 5–10% of cerebral AVM. The concentration of eloquent neurological systems and the high rate of bleeding presentation of AVMs particularly in this location complicate the management of such lesions. New therapeutic options, especially in endovascular neurosurgery, have fundamentally modified the strategy and, also, the outcome of cerebellar AVMs. Between 1995 and 2013, demographic, clinical and angiographic data of cerebral AVMs have been prospectively collected. We analyzed data of patients treated for a cerebellar AVM, focusing on the angiographic anatomy. Fifty-nine patients (mean age : 35 years, male to female ratio : 2) were consecutively treated for a cerebellar AVM. 81.4% of them presented bleeding at admission. 20 AVMs (33.9%) were in eloquent areas. The Spetzler–Martin grade was I or II in 36 cases (51%). An associated aneurysm was noted in more than 40% of cases and a venous drainage anomaly in 70%. The vast majority of cases of this series presented an anatomical risk factor of bleeding. Patients with cerebellar AVMs presented with bleeding more often than patients with supratentorial AVMs, justifying an aggressive management. The analysis of angio-architecture highlighted the high rate of associated aneurysm and/or venous drainage anomalies that could explain the tendency to bleed of such lesion.

1. Introduction

Infratentorial arteriovenous malformations (AVMs) were first reported by Clingenstein [23] in 1908 and the first successful resection of such lesion was accomplished by Olivecrona and Riives [12] in 1932. Posterior fossa AVMs are rare lesions accounting for 7–15% of cerebral AVMs in most large clinical series [1,4,6]. The management of these neurovascular lesions is complicated by the concentration of highly eloquent neurological structures within the small content of the posterior fossa [23]. The aggressive natural history [1,3,16,22] of this subtype of cerebral AVMs highlighted the importance of pertinent anatomical and angiographic knowledge of the cerebellum. We described anatomic and angiographic particularity of our series of cerebellar AVMs and highlighted the importance of arterial and venous changes in relation to the AVM which were known to be independent risk factors for bleeding or rebleeding [3–12]. See Table 1

2. Materials and methods

2.1. Demographic data

We have maintained an ongoing prospective database of demographic, clinical and angiographic information regarding 794 patients harboring a cerebral arteriovenous malformation. From 1995 to 2013, 59 patients with cerebellar AVM have been evaluated in our institution. Demographic data recorded for each patient included age, sex, and clinical presentation. The diagnosis of cerebellar AVM was confirmed by digital subtraction angiography (DSA) for all patients. Cavernomas, developmental venous anomalies, vein of Galen and dural arteriovenous malformations were not included. Every patient has undergone preoperative magnetic resonance (MR) or computerized tomography (CT) imaging of the brain which diagnosed a possible bleeding (subarachnoid hemorrhage or parenchymal hematoma) and confirmed the cerebellar location of the AVM.

2.2. Types of cerebellar AVMs

To classify AVM of the cerebellum, we used anatomical parts of the cerebellum. We identified four different locations: cerebellar hemisphere, deep cerebellar nuclei, cerebellar peduncles and vermis. This classification permitted us to know directly if the AVM was located in

Abbreviations: AP, antero-posterior; AICA, antero-inferior cerebellar artery; AVM, arteriovenous malformation; CT, computerized tomography; DSA, digital subtraction angiography; MRI, magnetic resonance imaging; PICA, postero-inferior cerebellar artery; WFNS, World Federation Neurosurgical Society.

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Table 1
Angioarchitectural details of the AVMs.

Variable	AVM (n = 59)
Arterial supply: mean (range)	2.41 [1–5]
Superior cerebellar artery	48 (81.4%)
Antero-inferior cerebellar artery	23 (39.0%)
Postero-inferior cerebellar artery	32 (54.2%)
Dural artery	5 (8.5%)
Intra-cranial aneurysm	
Inflow aneurysm	18 (30.5%)
Intra-nidal aneurysm	7 (11.9%)
Without relation to AVM	2 (3.4%)
Pial-to-pial collateral	5 (8.5%)
Superficial venous drainage only	26 (44.1%)
Deep venous drainage only	17 (28.8%)
Deep and superficial venous drainage	16 (27.1%)
Periventricular drainage	15 (25.4%)
Number of draining veins: mean (range)	1.83 [1–5]
Venous stenosis	19 (32.2%)
Venous occlusion	1 (1.7%)
Venous ectasia	23 (39.0%)
Venous reflux	18 (30.5%)
Sinus thrombosis	2 (3.4%)
Pseudo phlebitis pattern	26 (44.1%)

eloquent area which were defined by Spetzler [18] as cerebellar nuclei and peduncles in cerebellar locations.

2.3. Arterial feeder anatomy and variants

We have noted, helped by selective arterial catheterism, the number of arterial feeders, which arteries were implicated and the presence of associated aneurysms. Associated aneurysms were classified as flow-related aneurysms (in relation with the AVM but without the nidus), intra-nidal aneurysms or aneurysms without relation to the AVM. We also noted the presence of pial-to-pial collateralization because it may be considered related to the degree of pressure reduction and, indirectly, to the hemorrhagic risk of the AVM.

2.4. Venous drainage anatomy and anomalies

We also analyzed in detail the anatomy of the venous drainage of the AVM. Primarily, the number of veins reaching sinus and the quality of superficial or deep venous drainage were noted. All anomalies of the venous drainage known to influence the hemorrhagic risk of the AVM have been carefully analyzed. So, the following data were considered important and recorded: the presence of periventricular drainage, venous stenosis, thrombosis, occlusion, reflux or ectasia. Associated venous sinus thrombosis and pseudo-phlebitis pattern have also been examined.

3. Results

3.1. Demographic data and AVM characteristics

Between 1995 and 2013, 59 patients with angiographically visible cerebellar AVM were consecutively evaluated at our institution. Mean age was 35 years (range, 4–67 years) with a male-to-female ratio of 2:1. The majority of patients (n = 48, 81.4%) presented with bleeding at admission: 18 of these patients (30.5%) had a subarachnoid hemorrhage, 21 (35.6%) a parenchymal hematoma and 9 (15.3%) both. We used the Fisher CT scale [5] to quantify the blood in the subarachnoid space; 12 patients had grade II, 7 patients grade III and 8 grade IV. All intra-cerebellar hematoma encountered were of small size, less than 30 mm [3]. We used the World Federation Neurosurgical Society score to quantify the clinical gravity of patients who presented bleeding. Among the 48 patients who have bled, 24 had a grade of I, 15 grade II, 5 grade III, 2 grade IV and 2 grade V. The clinical presentation was a focal neurological sign in 5 patients (8.7%). Two of them presented a six nerve palsy which could be explained by a mass effect of the nidus. The three other patients presented a dynamic cerebellar syndrome which was more explained by a regional decrease in blood flow. The other 6 patients (8.7%) had an incidental discovery of the AVM. Locations of the AVM included the cerebellar hemisphere in 42 patients (71.2%), cerebellar vemis in 21 (35.6%) and cerebellar nuclei in 13 (22.0%). As described by Spetzler and Martin [18], eloquent areas in the cerebellum were considered as cerebellar nuclei and cerebellar peduncles. In our series, 20 AVMs (33.9%) were in eloquent areas, 14 (23.7%) involved cerebellar peduncles and 14 (23.7%) cerebellar nuclei. The AVMs were <30 mm in size in 39 cases (66.1%) and between 30 mm and 60 mm in 20 cases (33.9%). The Spetzler–Martin grade [18] was I in 20 cases (33.9%), II in 16 (27.1%), III in 11 (18.6%) and IV in 12 (20.3%). No patient had a grade V AVM. The AVM nidus was classified as compact in 43 cases (72.9%).

3.2. Arterial supplies

The AVM angio-architecture was given in the table. Mean number of arterial feeders was 2.4 (range, 1–5). Arterial supply was from the superior cerebellar artery in 48 patients (81.4%), the postero-inferior cerebellar artery in 32 patients (54.2%) and the antero-inferior cerebellar artery in 23 patients (39.0%). External carotid artery feeders were noted in 5 patients (8.5%). Associated intracranial aneurysms were classified as nidal (7 cases, 11.9%), inflow (18 cases, 30.5%) and unrelated to the AVM (2 cases, 3.4%). A pial-to-pial collateralization was presented in 5 cases (8.5%). Arterial anatomy of AVMs was illustrated in Fig. 1.

3.3. Venous drainage

The mean number of draining veins was 1.8 (range, 1–5). Twenty-six AVMs (44.1%) had superficial venous drainage only, 17 (28.8%) deep venous drainage only and 16 (27.1%) had both. A single venous drainage was presented in 27 cases (45.6%). We examined the presence of venous drainage anomaly as venous stenosis (n :19, 32.2%), venous (n :1, 1.7%) or sinus (n :2, 3.4%) occlusion, venous ectasia (n :23, 39%)and venous reflux (n :18, 30.5%). Fig. 2 showed examples of AVM venous drainage anomalies.

4. Discussion

Cerebellar AVMs are complex neurovascular lesions that, as opposed to their supratentorial counterparts [2,3,7–9,12,20,21,23], pose an increased risk of bleeding presentation [1,10] as well as increased morbidity [1,15,17,23,24] due to their presence in the narrow confines of the posterior fossa and in proximity to many eloquent structures. The first successful extirpation of a cerebellar AVM was reported in 1932 [12] by Olivecrona and Riives. Despite the evolution of microsurgical techniques [3,11,18,23], the introduction of the radiosurgery [14,16,22] and endovascular embolization [7] during the last decades, treatment of cerebellar AVMs remains a challenging quest.

Unlike other brain arteriovenous malformations, cerebellar AVMs are more likely to present with hemorrhage [1,2,16,20,21]. In our series, 81% of cases presented with a bleeding event. A series of posterior fossa [1,3,4,7,13,16] published in the literature, show a rate of bleeding presentation comprised between 72 and 92%. Fults and Kelly [7] note that less than half of their patients survived after initial hemorrhage and other authors [2,3,9] have reported a hemorrhagic mortality of more than 66.7% for posterior fossa AVM rupture.

Analyzing the angio-architecture of cerebellar AVMs, we highlight the rate of associated aneurysm as high as 45.3%. We have noted in our series that 35% of bleeding was from inflow aneurysm. The Toronto Study Group [3] shows a high rate of associated aneurysm to posterior

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