



Solid vs. cystic predominance in posterior fossa hemangioblastomas: implications for cerebrovascular risks and patient outcome

Samuel Moscovici¹ · Carlos Candanedo¹ · Sergey Spektor¹ · José E. Cohen¹ · Andrew H. Kaye¹

Received: 22 December 2020 / Accepted: 23 March 2021

© The Author(s), under exclusive licence to Springer-Verlag GmbH Austria, part of Springer Nature 2021

Abstract

Background Hemangioblastomas (HGBs) are highly vascular benign tumors, commonly located in the posterior fossa, and 80% of them are sporadic. Patients usually present with features of raised intracranial pressure and cerebellar symptoms. HGB can be classified as either mostly cystic or solids. Although the solid component is highly vascularized, aneurysm or hemorrhagic presentation is rarely described, having catastrophic results.

Methods We identified 32 consecutive patients with posterior fossa HGB who underwent surgery from 2008 through 2020 at our medical center. Tumors were classified as predominantly cystic or solid according to radiological features. Resection was defined as gross total (GTR) or subtotal (STR).

Results During the study period, 32 posterior fossa HGBs were resected. There were 26 cerebellar lesions and 4 medullar lesions, and in 2 patients, both structures were affected. Predominant cystic tumors were seen in 15 patients and solids in 17. Preoperative digital subtraction angiography (DSA) was performed in 8 patients with solid tumors, and 4 showed tumor-related aneurysms. Embolization of the tumors was performed in 6 patients, including the four tumor-related aneurysms. GTR was achieved in 29 tumors (91%), and subtotal resection in 3 (9%). Three patients had postoperative lower cranial nerve palsy. Functional status was stable in 5 patients (16%), improved in 24 (75%), and 3 patients (9%) deteriorated. One patient died 2 months after the surgery. Two tumors recurred and underwent a second surgery achieving GTR. The mean follow-up was 42.7 months (SD ± 51.0 months).

Conclusions Predominant cystic HGB is usually easily treated as the surgery is straightforward. Those with a solid predominance present a more complex challenge sharing features similar to arteriovenous malformations. Given the important vascular association of solid predominance HGB with these added risk factors, the preoperative assessment should include DSA, as in arteriovenous malformations, and endovascular intervention should be considered before surgery.

Samuel Moscovici and Carlos Candanedo contributed equally to this work.

This article is part of the Topical Collection on *Tumor — Other*

✉ Samuel Moscovici
samuelm@hadassah.org.il

Carlos Candanedo
ccandanedo@hotmai.com

Sergey Spektor
sergeyspektor@gmail.com

José E. Cohen
jcohenns@yahoo.com

Andrew H. Kaye
andrewk@hadassah.org.il

¹ Department of Neurosurgery, Hadassah-Hebrew University Medical Center, P.O. Box 12000, 91120 Jerusalem, Israel

Keywords Aneurysm · Embolization · Hemangioblastoma · Subarachnoid hemorrhage · Von Hippel Lindau disease

Introduction

Hemangioblastomas (HGBs) are uncommon benign WHO I lesion, representing 1.5–2.5% of all intracranial tumors. The most common location for HGBs is the posterior fossa, representing 7–8% of all the tumors in the region [11, 12]. Twenty percent of cases are associated with Von Hippel Lindau disease (VHL) [7, 9, 23]. The peak incidence is in the fifth and sixth decades of life [23]. They may become large and may have either a prominent cystic or solid component. Consequently, the common clinical presentation is related to increased intracranial pressure in more than half of cases with cerebellar signs in one-third of the patients [9].

Table 1 Reported cases of aneurysm-associated posterior fossa hemangioblastomas

Author year	Age/ gender	Presentation/cerebrovascular ictus	Aneurysm location	Type of tumor	Treatment	Complication	Outcome
Yoshii, et al. (1976) [28]	50/F	Tumor	BA bifurcation Lt ICA bifurcation	----	Resection	---	Aneurysm found 23 months after tumor extirpation
Ueno, et al. (1977) [25]	50/F	Tumor	Lt PICA Lt ICA	----	----	----	----
Guzman, et al. (1999) [5]	53/M	Tumor/ICH	Feeder vessel aneurysm	Solid/cystic	Tumor resection/hematoma drainage/clipping	Intraoperative aneurysm rupture	----
Menovsky, et al. (2002) [16]	52/F	Tumor	Left BA-AICA	Solid	Resection/aneurysm not adequately clipped/ wrapped	----	After 5 years — SAH AICA aneurysm enlargement
Zager, et al. (2002) [29]	53/-	Tumor	Distal branch of the AICA	Solid	Embolization and resection	----	Good
Murai, et al. (2006) [17]	72/M	tumor	PPTA	Solid	Resection/aneurysm bypass was offered, patient refused	----	----
Seong, et al. (2011) [21]	----	Tumor	Left PICA	----	Embolization, resection	----	----
Suzuki, et al. (2014) [24]	36/F	Tumor/SAH	PICA/feeder	Solid	NBCA embolization, resection	----	----
Ju, et al. (2017) [8]	70/M	Tumor/SAH	PICA Intratumoral	Solid	Resection/polyvinyl alcohol particle tumor and aneurysm embolization	LCN deficit Severe vasospasm	Death

AICA, anterior inferior cerebellar artery, *BA*, basilar artery, *ICA*, internal carotid artery, *ICH*, intracerebellar hemorrhage, *LCN*, lower cranial nerve, *Lt*, left, *NBCA*, N-butyl cyanoacrylate, *PICA*, posterior inferior cerebellar artery, *PPTA*, persistent primitive trigeminal artery, *SAH*, subarachnoid hemorrhage

HGBs are, by nature, highly vascularized tumors. Cerebrovascular insults and aneurysms are unusually associated with posterior fossa HGBs [5, 8, 16, 17, 21, 25, 26, 29, 30], but, although uncommon, subarachnoid or intratumoral hemorrhage resulting from the tumor's high vascularity could be potentially life-threatening [5, 8, 25, 29]. In the literature, we found 9 cases of aneurysms associated with HGBs, where only 3 had acute bleeding [5, 8, 16, 17, 21, 25, 26, 29, 30] (Table 1).

Angiography and angioarchitecture definition studies before surgery are not always included in the preoperative evaluation process unless it is the surgeon's preference. In general, HGB workup is similar to an intracranial tumor. Surgical resection is the preferred management strategy due to the combination of posterior fossa location, large volume, and high vascularization. Tumor embolization prior resection has also not been established as a standard practice [1, 29].

The authors present their experience in the surgical management of 32 posterior fossae HGBs. We give special attention to the anatomical tumor features particularly related to tumor vascularity, cerebrovascular presentation, and preoperative endovascular angioarchitecture.

Methods

We identified consecutive patients with posterior fossa HGB who underwent surgery from 2008 through 2020 at our medical center. Data from the retrospective review of records was supplemented by the surgeon's notes recorded before and immediately after the surgery. Patients with HGBs in other locations, those whose tumors were not resected, and those with other tumors involving the posterior fossa were excluded from this study.

Data summarizing patients' medical histories, clinical and neurological signs and symptoms, findings on radiological studies and digital subtraction angiography (DSA), neuroendovascular therapy and cerebrospinal fluid (CSF) diversion when relevant, details of the surgical procedure,

pathology reports, complications, morbidity and mortality, adjuvant radiotherapy, and clinical and radiological long-term follow-up to identify tumor progression or recurrence were recorded. Our Institutional Review Board formally waived the requirement for informed consent for this retrospective review.

Preoperative magnetic resonance imaging (MRI) was performed in all patients. For this study, MRI studies were reviewed, the presence of hydrocephalus was recorded, and tumors were classified into two subtypes according to radiological features:

- Predominantly cystic, including cystic nodular in cases where intraparenchymal cyst appeared as a mural enhancing nodule and the cyst is more than 50% of total tumor volume (Fig. 1a).
- Predominantly solid, including solid cystic, in cases where there was a predominance of a solid tumor being more than 50% of total tumor volume (Fig. 1b) and solid when no cystic areas were seen in the tumor mass (Fig. 1c).

Preoperative DSA was performed when the surgeon considered it was relevant for surgical planning. When embolization was performed, the technique was selected by the neuroendovascular surgeon. Based on peri- and intratumoral angioarchitecture, embolization of intratumoral regions, aneurysms, and feeder arteries was performed when indicated and feasible.

CSF diversion was performed before, during, or after surgery when necessary.

Resection was defined as gross total (GTR) when no residual tumor was evident on postoperative MRI, subtotal (STR) when less than 5% of tumor residual remained on the postoperative MRI, or partial tumor resection (PTR) when more than 5% of tumor residual was left. Initial tumor volume and residual tumor volume were measured using the simple formula $V = A*B*C/2$, as an accepted alternative for tumor volume measurement for routine clinical use [24].

Fig. 1 Preoperative axial T1-weighted gadolinium/enhanced magnetic resonance imaging (MRI) showing patients with **a** predominant cystic, **b** solid predominance, and **c** purely solid hemangioblastomas

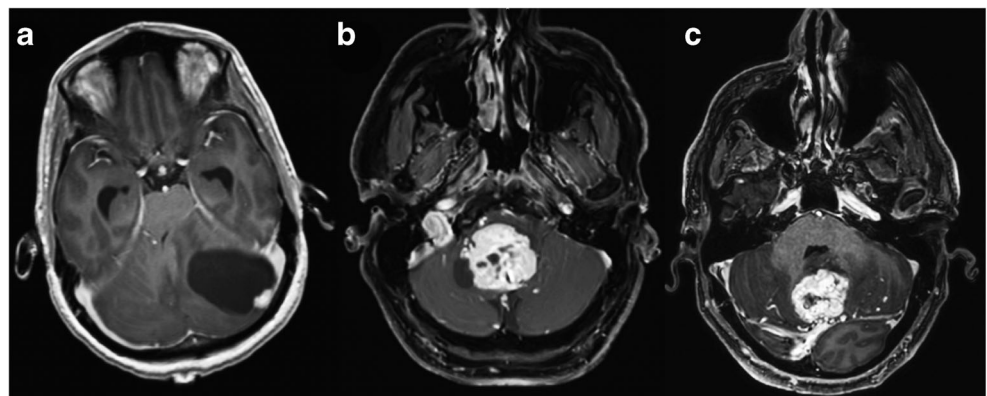


Table 2 Characteristics in 32 posterior fossa hemangioblastoma surgically treated

Variable	No (%)
Age (years)	45 ± 15
Gender	
Male	14/27 (52%)
Female	13/27 (48%)
Months of symptoms prior surgery	10 ± 14
Preoperative KFS	
100	25/30
80	1/30
70	1/30
60	3/30
Von Hippel syndrome	4/27 (15%)
De novo diagnosis	21/32 (66%)
Tumor recurrence/regrowth	11/32 (34%)
Subarachnoid/intraparenchymal hemorrhage	1/30 (3%)
Hydrocephalus	17/30 (57%)
Tumor location ¹	
Left side	12/32 (38%)
Midline	10/32 (31%)
Right side	10/32 (31%)
Volume (cc)	16.7 ± 12.2
Tumor type ¹	
Predominantly cystic ²	15 (47%)
Predominantly solid ^{3,4}	17 (53%)
Preoperative management	
Diagnostic DSA	8 (27%)
Endovascular embolization	6 (20%)
VP Shunt	2 (7%)
Ventriculostomy	2 (7%)
Approaches	
Suboccipital midline	17/30 (57%)
Suboccipital paramidline	7/30 (23%)
Suboccipital retrosigmoid	6/30 (20%)
Extent of resection	
Gross total resection	29/32 (91%)
Subtotal resection	3/32 (9%)
Days of hospitalization	12.8 ± 11
Months of follow-up	38 ± 50

¹ Four patients presented with multiple tumors; there were a total of 32 tumors in 27 patients

² Includes cystic nodular in cases where intraparenchymal cyst appeared as a mural enhancing nodule

³ Includes solid cystic in cases where there was a predominance of solid tumor with small associated cysts

⁴ Solid tumor with no cystic areas seen in the tumor mass

DSA, digital subtraction angiography; KFS, Kamofsky score; SAH, subarachnoid hemorrhage

Table 3 Symptoms at presentation

Symptom	No. patients
Headache	24/30 (80%)
Ataxia	18/30 (60%)
Dizziness	14/30 (47%)
Vomits	10/30 (33%)
Visual deficit	5/30 (17%)
Diplopia	1/30 (3%)
Cognitive impairment	1/30 (3%)
Paresthesia	1/30 (3%)

Tumor pathology was reported according to WHO classification. The tumor was not subclassified as either cellular or reticular type.

MRI scans were performed 3 months postoperation to determine the extent of resection and residual tumor and at follow-up 6 months, 12 months, and annually thereafter. Clinical follow-up was scheduled at the same intervals. Tumor progression was defined as if a new enhancing tumor is evidenced on follow-up MRI, or in case of STR or PTR if the residual tumor growth.

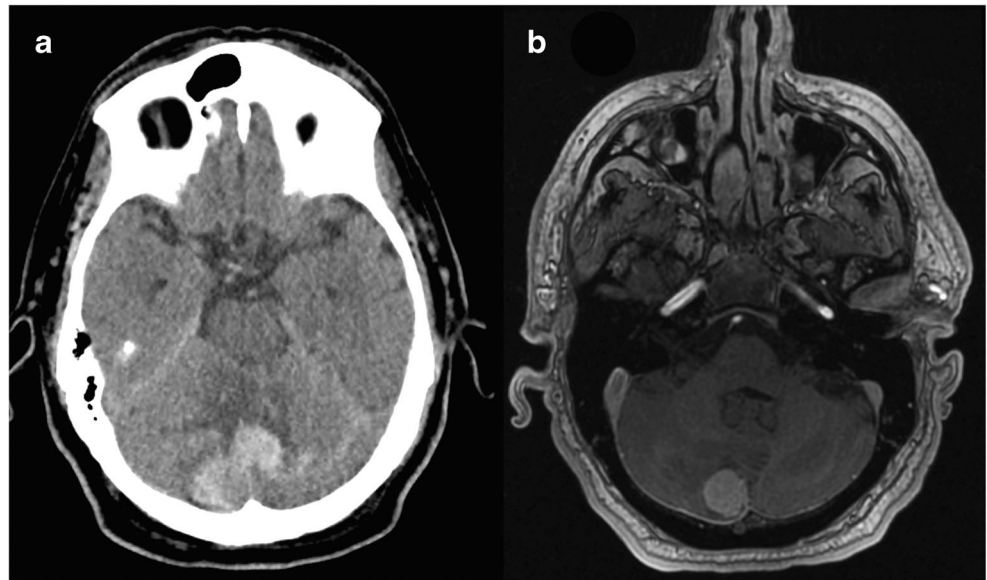
All continuous parameters are reported as mean and standard deviation. Non-continuous parameters are reported as median and interquartile range. The analysis was performed using GraphPad Prism version 7.0a.

Results

Overall, 27 patients met the inclusion criteria for the study. There were 13 women (48%) and 14 men (52%), with a mean age of 45 years (SD ± 15 years). These patients underwent a total of 30 operations for the resection of 32 tumors. A single tumor was detected in 23 patients, while 4 patients had multiple lesions. Surgery was performed following a primary diagnosis in 20 patients (74%), and in seven, this surgery followed a previous resection performed elsewhere or at our medical center before the study period. Ten operations (33%) were performed due to tumor recurrence or regrowth. There were 26 cerebellar lesions, 4 medulla lesions, and 2 cases where both structures were affected. Patient characteristics are summarized in Table 2.

Four patients (15%) had VHL. Headache was the most common complaint in 24 (80%) patients. Cerebellar signs were the most common physical findings, being present in 21 (70%) patients. Hydrocephalus was detected at presentation in 17 (57%) patients, and one (3%) presented with acute subarachnoid hemorrhage (SAH)/intraparenchymal cerebellar hemorrhage (ICH) (Fig. 2a, b). The details of the patient presentation are summarized in Table 3.

Fig. 2 **a** Axial non-contrast head computed tomography (CT) showing a posterior fossa subarachnoid/intraparenchymal hemorrhage, being clinical presentation of a right cerebellar solid hemangioblastoma. **b** Axial T1-weighted gadolinium/enhanced magnetic resonance imaging (MRI) showing the right cerebellar solid hemangioblastoma 2 weeks after the previous bleeding



Diagnostic DSA was performed in 8 patients (27%). In four patients (13%), tumor-related aneurysms were identified, all of them in the predominant solid group 4/17 (24%). The findings were one right superior cerebellar artery (SCA) aneurysm (Fig. 3a, b), one right posterior inferior cerebellar artery (PICA) aneurysm (Fig. 4), and two intratumoral aneurysms (Fig. 5a, b). Embolization of the tumor was performed before 6 operations, using Onyx in 3 patients and N-butyl cyanoacrylate (NBCA) in 3. All four aneurysms found were embolized preoperatively. In two cases, only a diagnostic DSA was performed, without the need for embolization. One of these patients had presented with subarachnoid hemorrhage.

Ventriculoperitoneal (VP) shunts were placed in two patients before surgery, and two underwent preoperative ventriculostomy. Following surgery, cystoperitoneal shunting

was indicated in one patient, and temporary ventriculostomy (10 days duration) was performed in one case. No permanent CSF diversion was required after surgery.

GTR was achieved in 29 tumors (91%), and STR in 3 (9%). The pathology of all tumors was WHO grade I HGB. Adjuvant SRS was recommended to one patient, but he declined the treatment. His tumor was stable at 18-month follow-up. The mean hospitalization period was 12.8 days (SD \pm 11 days).

All tumors were WHO I hemangioblastomas.

Three patients had lower cranial nerve palsies (10%). All required permanent tracheostomy and percutaneous endoscopic gastrostomy (PEG). One patient (3%) had transient diplopia due to IV CN weakness. Two (7%) patients developed pneumonia. Of the 30 surgeries at late follow-up, functional status was stable in 5 (17%), improved in 24 (80%), and

Fig. 3 Right vertebral artery digital subtraction angiography (DSA) showing **a** pre-embolization image of a right superior cerebellar artery (SCA) feeder vessel aneurysm (red arrow). **b** Postembolization images showing the occluded aneurysm and decreased in the tumor vascular flush



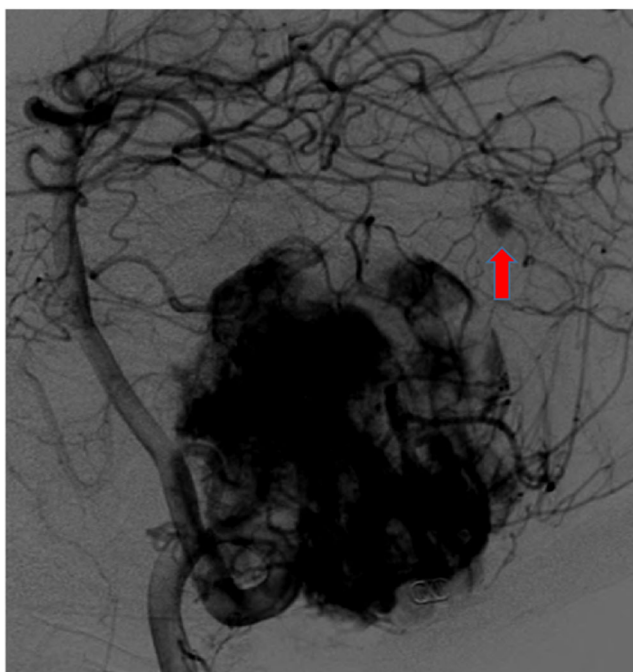
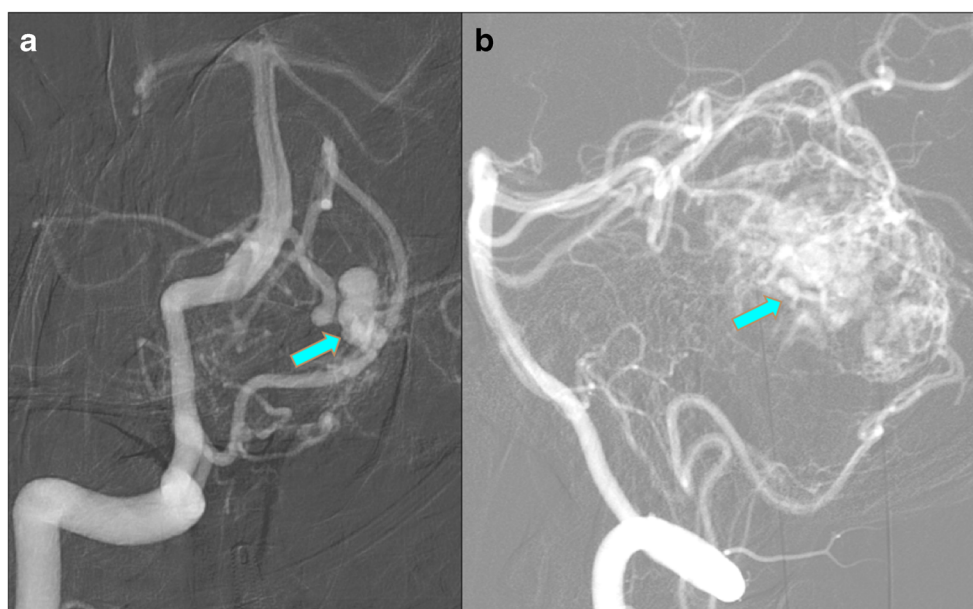


Fig. 4 Preoperative lateral view digital subtraction angiography (DSA) showing a right posterior inferior cerebellar artery (PICA) aneurysm (red arrow)

3 (10%) patients deteriorated their pre-op status, all of whom had permanent lower cranial nerve palsies. One 50-year-old patient who received anticoagulation due to pulmonary embolism 3 days after surgery died following an intratumoral and subarachnoid hemorrhage in the residual tumor 2 months later. Two (6%) tumors recurred, one 90 days after surgery and one 5 years later.

Both patients underwent a second surgery with GTR. The mean follow-up time was 42.7 months (SD \pm 51.0 months).

Fig. 5 a, b Preoperative digital subtraction angiographies (DSA) showing two different patients with intratumoral aneurysms (red arrows)



Discussion

Our results suggest that solid and cystic HGBs need to be considered as two separate entities. Those with a predominant cystic component present a straightforward management approach due to a relatively small solid component, a cyst that gave ready access to the tumor, and drainage of the cyst at the commencement of the operation resulted in a relaxation of the cerebellum. However, those with a predominantly solid component are a much more complex neurosurgical challenge, and in these, the cerebrovascular implications need to be carefully considered. This relates to the surgical challenge, which can be similar to the treatment of an arteriovenous malformation in the posterior fossa, with the intimate relationships to the brainstem and cranial nerves, and the possibility of an associated cerebral aneurysm, either intratumoral or on feeding vessels.

We recognize a high rate of aneurysm in our study compared with previous reports, which may be related to a lower threshold of DSA in our institution for pre-operative assessment of solid HB. Some of these aneurysms are probably “flow aneurysms” associated with the hyperdynamic local circulation that could resolve spontaneously following resection of the tumor. However, we suggest that given the crucial vascular association of solid predominant HGB with these added surgical risk factors, consideration should be given to including pre-operative DSA in the surgical assessment and, if considered, appropriate endovascular intervention. Follow-up radiological assessment should be undertaken in those patients whose aneurysms are not directly treated to confirm their resolution following surgery.

The hypervascular nature of solid HGB mirrors an AVMs, with an angioarchitecture consisting of feeder arteries to a

nidus that resembles the tumor and its draining veins as such surgery involves obliteration of the feeding arteries and then resection of the tumor on block, leaving obliteration of the draining veins as the last step, as in AVM surgery.

AVMs have a reported incidence of intracranial hemorrhage of 41% to 79%. AVM-associated aneurysms are found in 10.9% to 30.7% of patients, reaching approximately 80% in those presented with bleeding [13, 15, 18]. Moreover, when the AVM is located infratentorial, pedicle aneurysms on feeding vessels occur more frequently and have a higher bleeding rate [20, 27]. By contrast, hemorrhage presentation is low in patients with HGB, with an incidence of only 0.024% per person per year [4]. SAH at presentation is seen in only 1% of cases [10, 29], and intraparenchymal hemorrhage is even less common [5]. In our series, there was one patient with SAH and intraparenchymal hemorrhage. Only nine reported aneurysms were associated with HGBs; three aneurysms bled, and six were found incidentally during angiographic imaging performed for other indications (Table 1) [5, 8, 16, 17, 21, 25, 26, 29, 30]. Connie et al. reported a case showing an intratumoral HGB aneurysm causing SAH, followed by severe vasospasm [8]. Another 2 patients had acute bleeding, one of them suffered an ICH caused by a feeder-related aneurysm [5], and a 36-year-old woman bled from an HGB flow-related distal PICA aneurysm [25]. Four patients had aneurysms in our series, two were in feeder vessels, an SCA and a PICA, and two were intratumoral. All of them were found during DSA and treated by an endovascular procedure before surgery.

The pathogenesis of coexisting AVMs and aneurysms could be relevant to understand the relation between HGBs and aneurysms better. In the case of AVM, it appears to be related to increased flow velocity in the feeding artery due to loss of capillary bed resistance, a vessel wall defect, or a simple coincidence [2, 3, 14]. Altered vascular hemodynamics may cause developmental anomalies, leading to enhanced activity of hypoxia-inducible factor (HIF). Upregulation of VEGF has also been hypothesized as an etiology for the development of HGB-related aneurysms [5, 6, 8, 19, 21, 22, 28].

Knowing the angioarchitecture of the tumor and the feeding vessels is crucial for patient treatment. In two of our cases, aneurysms were on the feeding arteries (Figs. 2 and 3) that were not in the surgical field. Preoperative embolization of the tumor is not mandatory, and it is noted that some studies have found no decrease in intraoperative complications and even worse postoperative outcomes [29]. However, preoperative obliteration of aneurysms associated with HGB is considered highly advisable. Because our sample is small, and there was no comparison group, the significance of the tumor's embolization was not calculated. However, we felt that a successful embolization improved surgical conditions significantly.

HGBs are generally classified into four groups according to their radiological features: cystic, cystic nodular, solid cystic,

and solid (Fig. 1a–c). We suggest that they could be divided into two different entities, one group with predominantly cystic content and a second group that is predominantly solid. There were 15 tumors with a predominant cystic component in our series, and all of these patients had a benign course after GTR. There were no permanent deficits and no tumor recurrence. However, there were two acute bleeding cases among patients with the 17 predominantly solid tumors, and preoperative images demonstrated a tumor-related aneurysm in 23.5% of the patients. Moreover, patients with predominantly solid tumors had a higher rate of permanent deficits related to low cranial nerve injury. One patient who anticoagulated due to PE died 2 months after surgery due to subarachnoid and intratumoral hemorrhage in the residual tumor. In our experience, posterior fossa HGB could not be considered as one entity since the predominance of cystic or solid components will be a key feature defining surgical complexity and outcome.

Conclusions

HGBs that are predominantly solid may have complex vascular associations. We recognize that the high incidence of aneurysms in our study could be due to a low threshold of pre-operative DSA in our institution. Besides, at least some of the aneurysms identified may be “flow aneurysms” that could resolve after resection of the predominant solid component. Nevertheless, we consider that in patients with predominantly solid tumors, consideration should be given to perform a pre-operative DSA. Appropriate endovascular obliteration of the aneurysm could be undertaken before tumor resection. If the aneurysms are not treated, then a follow-up radiological assessment of the aneurysms is essential.

Declarations

Informed consent Informed consent was obtained from all individual participants included in the study.

Conflict of interest The authors declare no competing interests.

References

1. Ampie L, Choy W, Lamano JB, Kesavabhotla K, Kaur R, Parsa AT, Bloch O (2016) Safety and outcomes of preoperative embolization of intracranial hemangioblastomas: a systematic review. *Clin Neurol Neurosurg* 150:143–151. <https://doi.org/10.1016/j.clineuro.2016.09.008>
2. Cunha e Sa MJ, Stein BM, Solomon RA, McCormick PC (1992) The treatment of associated intracranial aneurysms and arteriovenous malformations. *J Neurosurg* 77:853–859. <https://doi.org/10.3171/jns.1992.77.6.0853>

3. Gao E, Young WL, Pile-Spellman J, Joshi S, Duong H, Stieg PE, Ma Q (1997) Cerebral arteriovenous malformation feeding artery aneurysms: a theoretical model of intravascular pressure changes after treatment. *Neurosurgery* 41:1345–1356; discussion 1356–1348. <https://doi.org/10.1097/00006123-199712000-00020>
4. Glasker S, Van Velthoven V (2005) Risk of hemorrhage in hemangioblastomas of the central nervous system. *Neurosurgery* 57:71–76; discussion 71–76. <https://doi.org/10.1227/01.neu.0000163250.71951.18>
5. Guzman R, Grady MS (1999) An intracranial aneurysm on the feeding artery of a cerebellar hemangioblastoma. Case report. *J Neurosurg* 91:136–138. <https://doi.org/10.3171/jns.1999.91.1.0136>
6. Hatva E, Bohling T, Jaaskelainen J, Persico MG, Haltia M, Alitalo K (1996) Vascular growth factors and receptors in capillary hemangioblastomas and hemangiopericytomas. *Am J Pathol* 148:763–775
7. Jagannathan J, Lonser RR, Smith R, DeVroom HL, Oldfield EH (2008) Surgical management of cerebellar hemangioblastomas in patients with von Hippel-Lindau disease. *J Neurosurg* 108:210–222. <https://doi.org/10.3171/JNS.2008.108.2.0210>
8. Ju C, Wright CH, Wright J, Duan Y, Bambakidis NC (2017) Subarachnoid hemorrhage associated with intratumoral aneurysm rupture within a posterior fossa hemangioblastoma: the importance of continued surveillance for cerebral vasospasm. *Cureus* 9:e1606. <https://doi.org/10.7759/cureus.1606>
9. Kuharic M, Jankovic D, Splavski B, Boop FA, Arnautovic KI (2018) Hemangioblastomas of the posterior cranial fossa in adults: demographics, clinical, morphologic, pathologic, surgical features, and outcomes. A systematic review. *World Neurosurg* 110:e1049–e1062. <https://doi.org/10.1016/j.wneu.2017.11.173>
10. Lee JY, Dong SM, Park WS, Yoo NJ, Kim CS, Jang JJ, Chi JG, Zbar B, Lubensky IA, Linehan WM, Vortmeyer AO, Zhuang Z (1998) Loss of heterozygosity and somatic mutations of the VHL tumor suppressor gene in sporadic cerebellar hemangioblastomas. *Cancer Res* 58:504–508
11. Liu X, Zhang Y, Hui X, You C, Yuan F, Chen W, Zhang S (2015) Surgical management of medulla oblongata hemangioblastomas in one institution: an analysis of 62 cases. *Int J Clin Exp Med* 8:5576–5590
12. Lonser RR, Butman JA, Huntoon K, Asthagiri AR, Wu T, Bakhtian KD, Chew EY, Zhuang Z, Linehan WM, Oldfield EH (2014) Prospective natural history study of central nervous system hemangioblastomas in von Hippel-Lindau disease. *J Neurosurg* 120:1055–1062. <https://doi.org/10.3171/2014.1.JNS131431>
13. Lv X, Wu Z, Li Y, Jiang C, Yang X, Zhang J (2011) Cerebral arteriovenous malformations associated with flow-related and circle of Willis aneurysms. *World Neurosurg* 76:455–458. <https://doi.org/10.1016/j.wneu.2011.04.015>
14. McKissock W, Paterson JH (1956) A clinical survey of intracranial angiomas with special reference to their mode of progression and surgical treatment: a report of 110 cases. *Brain* 79:233–266. <https://doi.org/10.1093/brain/79.2.233>
15. Meisel HJ, Mansmann U, Alvarez H, Rodesch G, Brock M, Lasjaunias P (2000) Cerebral arteriovenous malformations and associated aneurysms: analysis of 305 cases from a series of 662 patients. *Neurosurgery* 46:793–800; discussion 800–792. <https://doi.org/10.1097/00006123-200004000-00004>
16. Menovsky T, Andre Grotenhuis J, Bartels RH (2002) Aneurysm of the anterior inferior cerebellar artery (AICA) associated with high-flow lesion: report of two cases and review of literature. *J Clin Neurosci* 9:207–211. <https://doi.org/10.1054/jocn.2001.0945>
17. Murai Y, Kobayashi S, Tateyama K, Teramoto A (2006) Persistent primitive trigeminal artery aneurysm associated with cerebellar hemangioblastoma. Case report. *Neurol Med Chir (Tokyo)* 46:143–146. <https://doi.org/10.2176/nmc.46.143>
18. Redekop G, TerBrugge K, Montanera W, Willinsky R (1998) Arterial aneurysms associated with cerebral arteriovenous malformations: classification, incidence, and risk of hemorrhage. *J Neurosurg* 89:539–546. <https://doi.org/10.3171/jns.1998.89.4.0539>
19. Rothbart D, Awad IA, Lee J, Kim J, Harbaugh R, Criscuolo GR (1996) Expression of angiogenic factors and structural proteins in central nervous system vascular malformations. *Neurosurgery* 38:915–924; discussion 924–915. <https://doi.org/10.1097/00006123-199605000-00011>
20. Schmidt NO, Reitz M, Raimund F, Treszl A, Grzyska U, Westphal M, Regelsberger J (2011) Clinical relevance of associated aneurysms with arteriovenous malformations of the posterior fossa. *Acta Neurochir Suppl* 112:131–135. https://doi.org/10.1007/978-3-7091-0661-7_23
21. Seong Eom K, Won Kim D, Sung Choi S, Ha Choi K, Young Kim T (2011) Preoperative embolization of a cerebellar haemangioblastoma using Onyx: case report and literature review. *Neurol Neurochir Pol* 45:292–296. [https://doi.org/10.1016/s0028-3843\(14\)60082-7](https://doi.org/10.1016/s0028-3843(14)60082-7)
22. Skirgaudas M, Awad IA, Kim J, Rothbart D, Criscuolo G (1996) Expression of angiogenesis factors and selected vascular wall matrix proteins in intracranial saccular aneurysms. *Neurosurgery* 39:537–545; discussion 545–537. <https://doi.org/10.1097/00006123-199609000-00021>
23. Slater A, Moore NR, Huson SM (2003) The natural history of cerebellar hemangioblastomas in von Hippel-Lindau disease. *AJNR Am J Neuroradiol* 24:1570–1574
24. Sreenivasan SA, Madhugiri VS, Sasidharan GM, Kumar RV (2016) Measuring glioma volumes: a comparison of linear measurement based formulae with the manual image segmentation technique. *J Cancer Res Ther* 12:161–168. <https://doi.org/10.4103/0973-1482.153999>
25. Suzuki M, Umeoka K, Kominami S, Morita A (2014) Successful treatment of a ruptured flow-related aneurysm in a patient with hemangioblastoma: case report and review of literature. *Surg Neurol Int* 5:S430–S433. <https://doi.org/10.4103/2152-7806.141887>
26. Ueno K, Mabuchi S, Echizenya K, Isu T, Goto S (1977) Incidentally-discovered aneurysms—a report of eight cases (author's transl). *No Shinkei Geka* 5:183–188
27. Westphal M, Grzyska U (2000) Clinical significance of pedicle aneurysms on feeding vessels, especially those located in infratentorial arteriovenous malformations. *J Neurosurg* 92:995–1001. <https://doi.org/10.3171/jns.2000.92.6.0995>
28. Wizigmann-Voos S, Breier G, Risau W, Plate KH (1995) Up-regulation of vascular endothelial growth factor and its receptors in von Hippel-Lindau disease-associated and sporadic hemangioblastomas. *Cancer Res* 55:1358–1364
29. Yoshii Y, Maki Y, Tomono Y, Nakamura T (1976) Cerebellar hemangioblastoma with multiple aneurysms. *No To Shinkei* 28:703–708
30. Zager EL, Shaver EG, Hurst RW, Flamm ES (2002) Distal anterior inferior cerebellar artery aneurysms. Report of four cases. *J Neurosurg* 97:692–696. <https://doi.org/10.3171/jns.2002.97.3.0692>

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.