

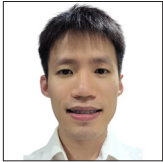


Case Report **Neuroradiology/Head and Neck Imaging**

Primary cerebellar angiosarcoma: A rare case of posterior fossa vascular tumor with hemorrhagic presentation and favorable surgical outcome

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ABSTRACT

Primary cerebellar angiosarcoma is an exceptionally rare intracranial malignancy, with only 22 cases reported in the literature. We report the case of a 67-year-old male who presented with a severe headache, unsteady gait, and giddiness. Computed tomography of the brain demonstrated a hemorrhagic lesion in the right cerebellar hemisphere with associated vasogenic edema and early hydrocephalus. Magnetic resonance imaging revealed a right cerebellar intra-axial lesion with mixed signal intensities and a characteristic peripheral “bull’s eye” enhancement pattern. Histopathological evaluation showed a vasoformative neoplasm with atypical endothelial cells, high mitotic activity, and immunopositivity for CD31 and erythroblast transformation specific (ETS)-related gene (ERG), consistent with low-grade angiosarcoma. The patient underwent gross total resection and remained recurrence-free 16 months postoperatively. This case underscores the importance of multi-modality imaging in the early recognition of hemorrhagic cerebellar tumors and reinforces the role of radiological-pathological correlation in diagnosis and treatment planning. Given its rarity, diagnosis of cerebellar angiosarcoma requires a high index of suspicion, supported by advanced imaging and immunohistochemical profiling. Multidisciplinary management is essential, and further case documentation is necessary to guide therapeutic strategies and prognostication.

Keywords: Case report, Hemorrhagic cerebellar mass, Posterior fossa tumor, Primary central nervous system angiosarcoma, Surgical resection

INTRODUCTION

Angiosarcomas are rare and aggressive soft-tissue masses originating from vascular or lymphatic endothelial cells. Comprising <2% of all soft-tissue sarcomas, they can occur in the skin, liver, and other soft tissue.^[1] Primary angiosarcoma of the central nervous system (CNS) is rare and usually located in the supratentorial brain, more frequently involving the parietal lobes.^[2] The incidence of primary angiosarcoma in the cerebellum is exceedingly rare.^[2] Here, we report an unusual case of a man who presented with a solitary lesion in the right cerebellum, which was confirmed on histopathology to be an angiosarcoma. Given the current paucity of data on CNS angiosarcoma, we review the imaging characteristics and histopathological evidence of primary cerebellar angiosarcoma.

CASE REPORT

A 67-year-old Chinese male with a past medical history of hypertension and hyperlipidemia presented to the emergency department with a 1-week history of severe headache. The headache

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was accompanied by unsteady gait, vomiting, and giddiness that worsened on standing. On examination, his vital signs were stable, and his Glasgow coma scale score was 15. Neurological assessment revealed right-sided dysmetria, while limb power and reflexes were normal in all four extremities. Initial blood tests were unremarkable.

Non-contrast computed tomography (CT) of the brain revealed a well-defined hyperdense intra-axial mass in the right cerebellar hemisphere, measuring approximately 2.2 cm × 1.9 cm × 2.1 cm [Figure 1]. Thin bilateral acute tentorial subdural hemorrhages were also noted. Subsequent magnetic resonance imaging (MRI) demonstrated a right cerebellar lesion with heterogeneous signal intensities on both T1- and T2-weighted sequences. Post-contrast images showed heterogeneous enhancement, including a peripheral rim of low-signal intensity that produced a characteristic “bull’s eye” or “target” appearance. These mixed signal intensities were suggestive of blood product degradation at various stages [Figure 2]. Diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) maps revealed patchy central areas of restricted diffusion, consistent with regions of high cellularity. The lesion exerted a significant mass effect, with effacement of the fourth ventricle, early hydrocephalus, and tonsillar herniation [Figure 2]. Time-of-flight magnetic resonance (MR) angiography showed no evidence of aneurysm or vascular malformation.

To reduce the perilesional vasogenic edema and mass effect, anti-edema therapy with dexamethasone was administered along with analgesia. The CT thorax, abdomen, and pelvis did not show any malignancy or significant lymphadenopathy. The positron emission tomography (PET)-CT scan did not reveal any hypermetabolic focus. The patient subsequently

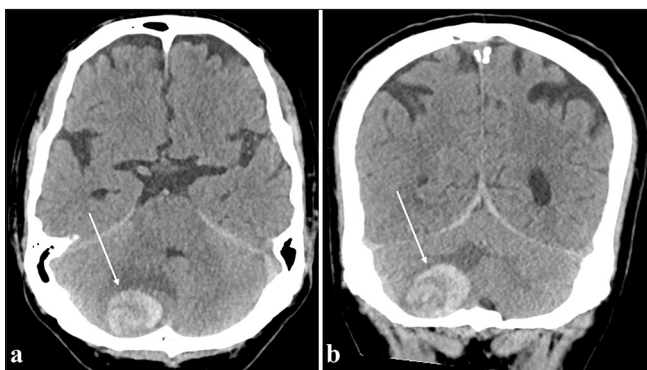


Figure 1: A 67-year-old male presented to the emergency department with a severe headache. (a) Axial non-enhanced computed tomography demonstrates a hyperdense hemorrhagic lesion in the right cerebellar hemisphere (white arrow), also seen on (b) coronal view (white arrow), surrounded by a moderate amount of vasogenic edema. It is associated with mass effect with effacement of the fourth ventricle and secondary dilation of the lateral ventricles.

underwent suboccipital craniotomy and complete tumor resection. Microscopic examination revealed a vasoformative neoplasm with nuclear atypia and raised mitosis as well as a 30–40% elevation of Ki67 [Figure 3]. Immunohistochemistry (IHC) showed expression of CD31 and erythroblast transformation specific (ETS)-related gene [Figure 3]. These were features consistent with a low-grade angiosarcoma.

After a multidisciplinary team suggested follow-up brain imaging, the patient was not recommended for adjuvant therapy, given the complete tumor resection. The patient was discharged from the hospital on the 3rd post-operative day and had remained asymptomatic with no clinical evidence of recurrence at outpatient clinic follow-up 24 months after surgery. A follow-up MRI brain about 16 months after surgery showed no evidence of tumor remnant or recurrence. In summary, the patient had a low-grade angiosarcoma that was successfully treated through suboccipital craniotomy and complete tumor resection.

DISCUSSION

Primary intracranial angiosarcoma is an extremely rare malignancy, with only 22 cases reported in the literature to date [Table 1].^[1-14] It affects a wide range of age groups, from a 12-day-old infant to a 72-year-old male, with a mean age of 42 years [Table 1]. The neurological symptoms experienced by patients vary based on the tumor’s location and growth rate, with most patients presenting with acute symptoms associated with hemorrhage. In general, acute worsening of clinical symptoms has often been related to either tumor growth or intratumoral hemorrhage.

As summarized in Table 1, most reported cases involved supratentorial lesions, with cerebellar angiosarcomas being exceedingly rare. According to our review, most primary CNS angiosarcomas are located in the supratentorial brain, and more frequently in the parietal lobes. Only three patients were reported to have infratentorial tumors; one in the cerebellopontine angle and two within the cerebellum, including our current case. Other locations of tumors include the sphenoid wing/sinus, pineal gland, extra-axial meningeal, and retro-orbital regions [Table 1]. A few other cases of radiation-associated intracranial angiosarcoma have also been reported in the literature associated with the use of Thorotrast as well as radiation therapy.^[15]

The diagnosis and management of primary CNS angiosarcoma require a multidisciplinary approach in a specialist soft-tissue sarcoma setting due to the complexity of the diagnosis and aggressive behavior of the tumor.^[16]

Role of imaging

The imaging characteristics of CNS angiosarcoma vary depending on the tumor content and composition, including

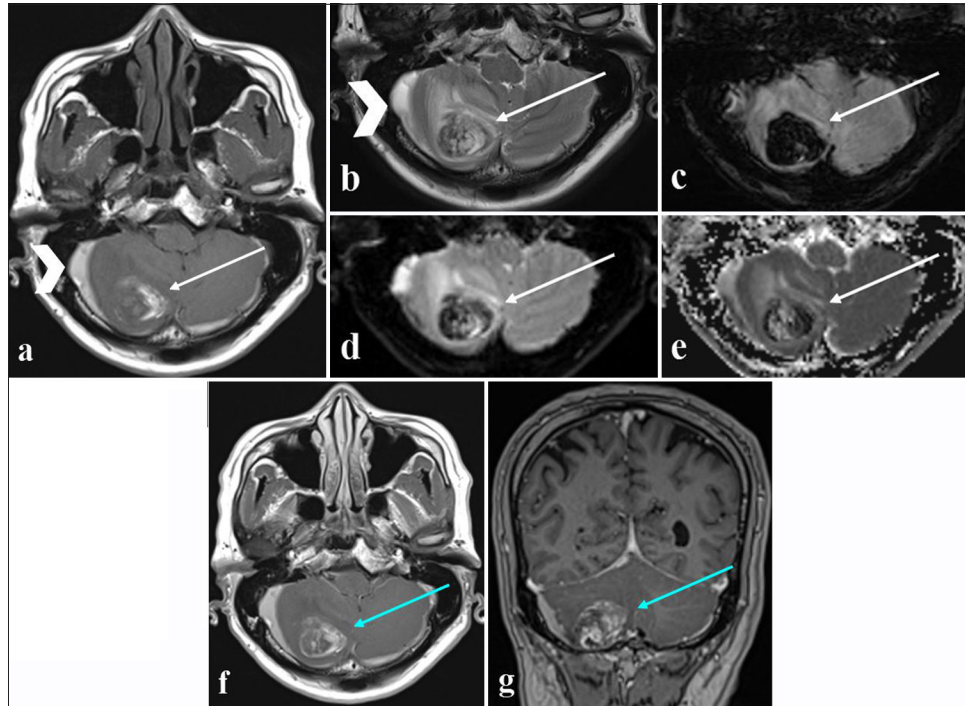


Figure 2: Multi-modality magnetic resonance imaging of a 67-year-old man with severe headache. (a) Axial T1-weighted image showing lesion with eccentric hyperintense signal in the right cerebellar lesion (white arrow) with hyperintense signal along the bilateral cerebellar convexities (white arrowhead) from bilateral subdural hemorrhages in the posterior fossa. (b) T2-weighted imaging showing hyperintense right cerebellar lesion (white arrow) with similar hyperintense signal along the bilateral cerebellar convexities (white arrowhead). (c) Susceptibility-weighted images showing marked susceptibility artifact in the right cerebellar hemisphere (white arrow). The lesion contains a large amount of hemosiderin and is consistent with a hemorrhagic lesion. (d) Axial diffusion-weighted imaging with hyperintense foci within the right cerebellar lesion (white arrow) and (e) Apparent diffusion coefficient images with corresponding hypointense foci within the right cerebellar lesion (white arrow), in keeping with restricted diffusion within the medial aspect of the lesion. It indicates an area of high cellularity within the lesion. (f) Axial T1-weighted post-contrast images demonstrate the lesion having an eccentric peripheral enhancement and areas of hypoenhancement within its center (light blue arrow), also seen on (g) coronal T1-weighted post-contrast images (light blue arrow); the latter is due to the presence of hemosiderin substance. The pattern of enhancement resembles a “bull eye” or “target” in appearance.

intratumoral bleeding.^[17] These tumors originate from vascular endothelial cells and can occur in the CNS as a primary brain tumor or more commonly as metastases. CT can be useful as an initial diagnostic modality. However, CT features are not typical for angiosarcoma. In the event of metastatic disease, CT with intravenous contrast is useful for staging and assessing the disease response to treatment. The disadvantages of using CT include exposure to ionizing radiation and poorer soft-tissue resolution compared to MRI.

Multi-modality MRI is the preferred imaging modality for assessing soft-tissue sarcomas, including primary CNS angiosarcomas. Besides having high spatial resolution, MRI can demonstrate anatomical information, disease extent, and tumor composition.^[16] With increased tumor vascularity, there are often areas of hemorrhage and necrosis within the

lesion, giving a heterogeneous signal intensity and patchy enhancement. Depending on the age of the hemorrhage, variable T1- and T2-weighted signal intensities can be present within the center of the lesion. The presence of a peripheral rim of enhancement with a relatively well-demarcated border of the tumor associated with perilesional edema resembles a “target” or “bullseye” appearance on the post-contrast images [Figure 2]. Other authors have reported the presence of cystic areas within the lesion.^[18] The solid component of the tumor often demonstrates restricted diffusivity on DWI and ADC images due to the high tumor cellularity, while MR perfusion imaging may show high relative blood volume within the tumor due to its hypervascular nature. The mass effect in the posterior fossa, with effacement of the fourth ventricle, may lead to obstructive hydrocephalus. MR spectroscopy is

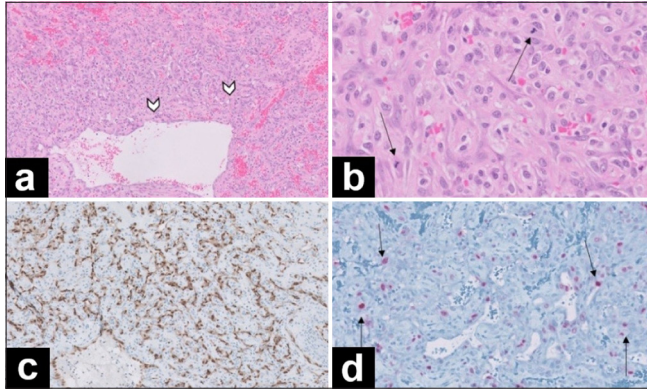


Figure 3: Histopathological features of cerebellar angiosarcoma. (a) Photomicrograph of histologic specimen [Haematoxylin and Eosin (H&E) Stain, original magnification x 100] reveals a vasoformative neoplasm with vascular channels lined by sheet-like atypical endothelial cells (arrowheads). (b) At the high power field (H&E Stain, original magnification x 400), there are pleomorphic tumor cells with raised mitosis (black arrows). These cells are scattered within a myxohyaline stroma. (c) Immunohistochemistry (original magnification x 100) shows expression of CD 31 and ERG, highlighting the vasoproliferative nature of the tumor. (d) (original magnification x 200) Ki-67 (Red Chromagen) proliferation index is markedly raised around 30-40% with increased mitosis (black arrows).

seldom used for imaging angiosarcomas because they do not show high cellular turnover with raised choline levels, unlike gliomas.

Typical differential diagnosis for CNS angiosarcoma on imaging includes hemangioblastoma, glioblastoma, and hemorrhagic metastases (usually associated with choriocarcinoma, thyroid cancer, melanoma, and renal cell carcinoma but also from other primaries, e.g., breast and lung carcinoma in view of their prevalence). However, the presence of atypical imaging features may cause a diagnostic dilemma. Table 2 compares the typical imaging characteristics of CNS angiosarcoma with those of its common hemorrhagic mimics, aiding in radiological differentiation.

Histopathology

The histological diagnosis of angiosarcomas can be challenging due to their highly variable features. The spectrum of findings can range from malignant endothelial cells to solid sheets of spindle or epithelioid cells without vasoformation.^[16] Epithelioid angiosarcomas are poorly differentiated tumors with areas of hemorrhage and necrosis and are associated with an unfavorable prognosis.^[16] It is notable that primary CNS angiosarcoma is not specifically classified in the 2021 World Health Organization (WHO) Classification of CNS Tumors, likely due to its extreme rarity and mesenchymal origin, which aligns it more closely with soft-tissue sarcomas than with primary glial or neuronal neoplasms.^[19]

Due to the challenges in histological diagnosis, IHC plays an important role in confirming the diagnosis. According to the 2020 WHO classification of soft-tissue tumors, the essential histopathological criteria for angiosarcomas include vasoformative or sheet-like growth or multilayering of endothelial cells, nuclear atypia, elevated mitosis, and necrosis.^[16] Other essential diagnostic criteria include the expression of CD31 and ERG on IHC.^[16] ERG, a nuclear stain, has been shown to complement CD31, a cytoplasmic/membranous stain, in the cytological diagnosis of angiosarcoma, both of which have strong sensitivity for angiosarcoma compared to other stains such as CD34.^[20] Other markers, for example, vascular endothelial growth factor receptor-3 and von Willebrand factor, have lower sensitivities for angiosarcomas, while friend leukemia integration 1 (FLI-1) is also seen in Ewing's sarcoma, synovial sarcoma, melanoma, and pulmonary adenocarcinoma.^[21]

It is also important to exclude other possible histological differential diagnoses. A subset of CIC-rearranged sarcomas has been found by Kojima *et al.* to express ERG and CD31 as an inherent phenotypic variation.^[22] This may represent a diagnostic pitfall just based on ERG and CD31 co-expression, which may result in inappropriate/ineffective treatment. Nonetheless, they can still be distinguished histologically based on focal myxoid change and the total lack of vasoformative architecture, as well as other stains such as ETV4 and nuclear WT1, absent in angiosarcomas. Hence, histopathological diagnosis of CNS angiosarcomas requires a comprehensive assessment of both histomorphology and IHC characteristics.

Treatment

There are currently no established treatment guidelines specific to primary CNS angiosarcoma due to its rarity. Management strategies are typically extrapolated from soft-tissue angiosarcoma protocols and depend on individual patient factors, tumor resectability, and institutional expertise. Surgical resection followed by adjuvant radiotherapy is the cornerstone of treatment for cases of localized angiosarcomas.^[23] However, these cases should still be managed by a sarcoma multidisciplinary team involving surgery, medical oncology, radiation oncology, and sarcoma specialists for consideration of neoadjuvant/adjuvant therapies.^[24,25]

Total surgical resection is usually the treatment of choice for primary CNS angiosarcomas, while adjuvant radiotherapy and chemotherapy are recommended due to the high risk of local recurrence. Among the 22 reported cases, 18 had total tumor resection, while two had incomplete resection, and two others did not undergo surgical resection. Most reported cases had either adjuvant chemotherapy or radiation, or a combination of both. Chemotherapeutic agents such as ifosfamide, temozolomide, and bevacizumab were used [Table 1]. However, the use of chemotherapy may be limited

Table 1: Literature review of cases of primary CNS angiosarcoma.

First author, year	Age/gender	Location	Key imaging features	Treatment	Pathology and IHC	Prognosis
La Corte <i>et al.</i> , 2015 ^[1]	35 y/F	L frontal (posterior) lobe	MRI: Partially hemorrhagic, moderately enhancing lesion with peripheral edema	TTR, adjuvant Chemo RT	Epithelioid AS	37 months RFS
Sari <i>et al.</i> , 2018 ^[2]	42 y/M	Multiple lesions in the basal ganglia, bilateral cerebral and R cerebellar hemispheres	MRI: Multiple hemorrhagic lesions in supra- and infra-tentorial brain with T2 hyperintensities resembling a “target” appearance with peripheral enhancement	Stereotactic biopsy of cerebellar lesion	Malignant cells with variable levels of differentiation and vascular structures covered with clustered or blister-like papillary malignant endothelial cells, compatible with AS. IHC: CD34+and CD31+	† 4 days after biopsy
Hackney <i>et al.</i> , 2012 ^[3]	35 y/F	L retro-orbital region	MRI: Homogeneous T1W enhancing extra-axial lesion with dural thickening	Sub-TTR (due to hemorrhage) with RT and bevacizumab	Vascular tumour with polygonal and spindle cells, and abundant hemorrhage. Epithelioid AS. IHC: CD31+	18 months RFS
	47 y/M	L sphenoid wing lesion extending into the sphenoid sinus.	MRI: Heterogeneous enhancing lesion with dural involvement and extension to the sphenoid sinus.	Near TTR with RT, Chemo (temozolomide and bevacizumab)	Abortive vascular structures, spindle cells within myxoid stroma. IHC: CD31+	Unknown
Charman <i>et al.</i> , 1988 ^[4]	65 y/M	L parietal and occipital lobes	No MRI	TTR, postop Chemo	AS cells with IHC factor VIII-related antigen positivity, and a high labeling index with bromodeoxyuridine	13 months RFS
Jerjir <i>et al.</i> , 2016 ^[5]	61 y/M	L frontal and temporal lobes	CT: Hyperattenuating lesion with cystic components and surrounding vasogenic edema. MRI: Mixed T1W and T2W isointensity with T2 hyperintense cystic components within and periphery of the lesion, associated with diffuse and accentuated rim enhancement after gadolinium administration. The posterior part of the lesion shows T2 hypointensity without enhancement, representing an acute hemorrhage.	TTR with postop RT	AS	105 months RFS
Cai <i>et al.</i> , 2016 ^[6]	30 y/F	R frontoparietal region	MRI: Meningeal AS with broad-based T1 hypointense lesion with “dural tail sign.” Dotted T2 hyperintense signal with irregular necrotic areas and multiple flow voids associated with extensive perilesional edema.	TTR with aggressive ChemoRT	AS with spindle-shaped and epithelioid cells showing marked cellular atypia. IHC: CD31+, CD34+, factor VIII (FVIII)+, FLI-1+, CD117+, ERG+and Nestin+.	4 months RFS

(Contd...)

Table 1: (Continued).

First author, year	Age/gender	Location	Key imaging features	Treatment	Pathology and IHC	Prognosis
Guode <i>et al.</i> , 2008 ^[7]	16 y/F	R CPA	MRI: Hemorrhagic lesion in the CPA	Suboccipital craniectomy, TTR, postop RT	AS	6 months RFS
Fuse <i>et al.</i> , 1995 ^[8]	39 y/M	R parietal lobe	MRI: Hyperintense and enhancing lesion. Cerebral angiography showed a vascular lesion.	TTR and RT	Epithelioid AS with positive IHC for CD31 and vimentin	Re-operated 2x but † 28 months after first surgery
Cookston <i>et al.</i> , 1991 ^[9]	32 y/F	R occipital lobe	CT and MRI: Discrete mass with enhancement and surrounding edema	TTR	Atypical endothelial cells with hyperchromatic nuclei and interconnecting vascular spaces. Hemorrhage and necrosis within the tumor.	42 months RFS
Antoniadis <i>et al.</i> , 1996 ^[10]	41 y/M	L parietal lobe	CT: Marked enhancing lesion with hypodense areas	TTR, adjuvant Chemo (ifosfamide) and whole brain RT at 45 Gy (+10 Gy to the tumor) 30 daily fractions of 1.8 Gy.	Poorly differentiated AS with positive IHC for both Factor VIII and lectin ULEX	41 months RFS
Mühlau <i>et al.</i> , 2003 ^[11]	72 y/M	Multiple bilateral cerebrum	Multiple intracranial hemorrhagic lesions.	No treatment	Post-mortem: Atypical vessels lined by poorly differentiated AS cells within the hemorrhagic lesions. IHC: CD31+, vimentin+with positive GFAP, HMB45, and cytokeratin.	† soon after admission, before surgery
Mena <i>et al.</i> , 1991 ^[12]	Ranging 2 wks–72 y (8 patients; 5/M and 3/F)	6 cases in the cerebral hemispheres, one meningeal, one unknown	Unknown	All underwent surgical resection	AS with irregular vascular channels and intraluminal papillae. IHC: Factor VIII-related antigen and Ulex europaeus agglutinin I performed in 5 tumors of which 4 were positive.	3/8 postop RFS between 39 and 102 months but 4/8 † within 4 months and 1/8 † after 30 months
Gao <i>et al.</i> , 2019 ^[13]	68 y/M	Left frontal lobe	CT and MRI: Hemorrhagic lesion with slightly enhancing margin and surrounding edema.	Exploratory surgery	AS	† 4 weeks PO
Kurian <i>et al.</i> , 2006 ^[14]	56 y/F	Pineal gland	CT: Localized intraventricular hemorrhage around the posterior third ventricle consistent with a pineal lesion. MRI: Superficial hemosiderosis in the brain stem and cerebellum.	Craniotomy, removal of the pineal mass	Vasoformative tumor with elongated bi-Olaf cells, epithelioid cells, and vacuolated cells in a loose matrix. Increased blood vessel formation throughout the lesion. This is consistent with low-grade AS. IHC: CD 31+and CD4+	Rec 48 months after initial surgery.

(†): Death, (+): Positive. AS: Angiosarcoma, Betw: Between, Chemo: Chemotherapy, Diff: Differentiated, CT: Computed tomography, D: Day, F: Female, IHC: Immunohistochemistry, L: left, M: Male, Mths: Months, MRI: Magnetic resonance imaging, PO: Postoperative, Pts: Patients, R: Right, Rec: Recurrence, RT: Radiotherapy, RFS: Recurrence-free survival, TTR: Total tumor resection, WI: Weighted images, Wks: Weeks, Y: Year-old, CPA: Cerebellopontine angle, GFAP: Glial fibrillary acidic protein

Table 2: Typical imaging features of primary CNS angiosarcoma and common differential diagnoses.

Appearance	Primary central nervous system angiosarcoma	Hemangioblastoma	Glioblastoma	Hemorrhagic metastasis
Hemorrhage within lesion	Yes	Uncommon except in large tumors	Possible from microvascular proliferation	Yes
Calcification	Uncommon	No	Uncommon	No
Perilesional edema	Yes	Uncommon	Variable	Yes
Cystic component	Uncommon (if present, small cystic areas)	Yes, with mural nodule	May have central necrosis	Uncommon
Flow voids	Commonly seen within a tumor	Commonly seen around the tumor	Occasionally seen	Not seen
Enhancement	Heterogeneous enhancement, mainly peripheral with “bull’s eye” or “target” appearance	Nodule has avid post-contrast enhancement	Peripheral irregular enhancement with nodular components	Usually homogeneous enhancement unless central necrosis/hemorrhage
Location	Variable	Mostly in the cerebellum and the spinal cord	Usually centered in the subcortical white matter	Usually at the gray-white matter junction
Other features	-	Associated with Von Hippel-Lindau disease	Multiple foci in multicentric glioblastoma multiforme	Maybe multiple

as many angiosarcoma patients are elderly with comorbidities and may be at risk of developing chemotherapy-related toxicity.

Prognosis

Primary CNS angiosarcoma appears to have a relatively better prognosis compared to glioblastoma, which has a mean survival of only 15 months.^[26] Although much of the literature does not describe the long-term prognosis of primary CNS angiosarcomas, we found that the majority of patients have survived beyond 6 months with adequate treatment [Table 1]. Recurrence-free survival among these cases ranged between 4 and 105 months [Table 1]. Seven deaths (31.8%) occurred within 30 months after the initial diagnosis [Table 1].

CONCLUSION

Primary CNS angiosarcoma is an exceptionally rare and aggressive malignancy that presents diagnostic challenges due to its varied histological features. Multi-modality MRI remains the imaging method of choice due to its superior spatial resolution and ability to characterize lesion anatomy, extent, and internal composition. Characteristic features include heterogeneous signal intensities, patchy enhancement with hemorrhagic and necrotic areas, and a peripheral rim-enhancement with perilesional edema, producing a “target” or “bull’s eye” appearance.

Definitive diagnosis relies heavily on IHC. Total surgical resection is the mainstay of treatment, often followed by

adjuvant radiotherapy and/or chemotherapy to mitigate the high recurrence risk. Given its rarity and biological complexity, further research is critical to enhance understanding and management of this disease. Future studies should aim for larger sample sizes and long-term follow-up to determine optimal treatment protocols and improve patient outcomes.

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