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CASE REPORT

# Multimodal imaging for the diagnosis of oligodendroglioma associated with arteriovenous malformation: A case report

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## Abstract

### *BACKGROUND*

The rare co-occurrence of oligodendroglioma and arteriovenous malformation (AVM) in the same intracranial location.

### *CASE SUMMARY*

In a 61-year-old man presenting with progressive headaches, is described in this case study. Preoperative multimodal imaging techniques (computed tomography, magnetic resonance imaging, magnetic resonance spectroscopy, digital subtraction angiography, and computed tomography angiography) were employed to detect hemorrhage, cystic and solid lesions, and arteriovenous shunting in the right temporal lobe. The patient underwent right temporal craniotomy for lesion removal, and postoperative pathological analysis confirmed the presence of oligodendroglioma (World Health Organization grade II, not otherwise specified) and AVM.

### *CONCLUSION*

The preoperative utilization of multimodal imaging examination can help clinicians reduce the likelihood of misdiagnosis or oversight of these conditions, and provides important information for subsequent treatment. This case supports the feasibility of craniotomy for the removal of glioma with AVM.

**Key Words:** Oligodendroglioma; Arteriovenous malformation; Angioglioma; Multimodal imaging; Case report

**Core Tip:** This case report details the rare co-occurrence of oligodendroglioma and arteriovenous malformation in a 61-year-old male. Preoperative multimodal imaging, including computed tomography, magnetic resonance imaging, magnetic resonance spectroscopy, digital subtraction angiography, and computed tomography angiography, was crucial in accurately diagnosing the combined pathology, guiding the successful surgical removal. This study underscores the importance of comprehensive imaging to prevent misdiagnosis and highlights the feasibility of craniotomy for treating this rare condition.

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## INTRODUCTION

The coexistence of oligodendroglioma and arteriovenous malformation (AVM) in the brain is a rare phenomenon, with a limited number of cases documented in the literature, primarily in case reports. Although oligodendroglioma and AVM are typically considered to be distinct diseases, the presence of both within a single lesion raises questions regarding the nature of this combined pathology[1,2]. Various hypotheses have been proposed to elucidate the potential mechanisms underlying the association of glioma with AVM in the same patient, including fortuitous coexistence, genetic predisposition, and a unique entity. Further investigation is required to better understand the pathophysiology of this rare occurrence[3,4]. Here, we describe a patient with an oligodendroglioma associated with major arteriovenous shunting and a vascular nidus mimicking an AVM, and highlight the use of multimodal imaging for the diagnosis of this type of disease.

## CASE PRESENTATION

### *Chief complaints*

A 61-year-old man presented with a progressive headache for 4 days.

### *History of present illness*

The patient presented to our neurology outpatient clinic four days ago with the acute onset of headache, without any apparent precipitating factors. The patient denied experiencing dizziness, nausea, vomiting, unilateral limb numbness, unilateral limb weakness, or altered mental status.

### *History of past illness*

The patient had no history of trauma, hypertension, or other medical conditions.

### *Personal and family history*

His history, personal history, and family history were unremarkable.

### *Physical examination*

A neurological examination was performed on admission and yielded normal findings.

### *Laboratory examinations*

Laboratory examinations were normal.

### *Imaging examinations*

Computed tomography (CT) examination revealed a significant area of hemorrhage, edema, and calcification in the right temporal lobe, resulting in a midline shift to the left (Figure 1A and B). Multimodal imaging was conducted and yielded findings consistent with right middle cerebral artery elevation and an early lesion drainage vein [detected by CT angiography (CTA)] (Figure 1C). CT perfusion (CTP) imaging demonstrated increases in the regional cerebral blood flow (rCBF) and mean transit time in the lesion area (Figure 1D and E). Contrast-enhanced magnetic resonance imaging (MRI) revealed a 4.5 cm × 6.3 cm × 4.6 cm necrotic cystic lesion in the right hemisphere involving the basal ganglia, with a midline shift and uncal herniation. The lesion exhibited dark signaling and edema belts on T2-weighted images, and heterogeneous enhancement on contrast-enhanced T1-weighted images (Figure 2A–D). Magnetic resonance spectroscopy (MRS) demonstrated aberrant metabolic function (Figure 2E). Specifically, an elevated choline/creatine (Cr) peak ratio of

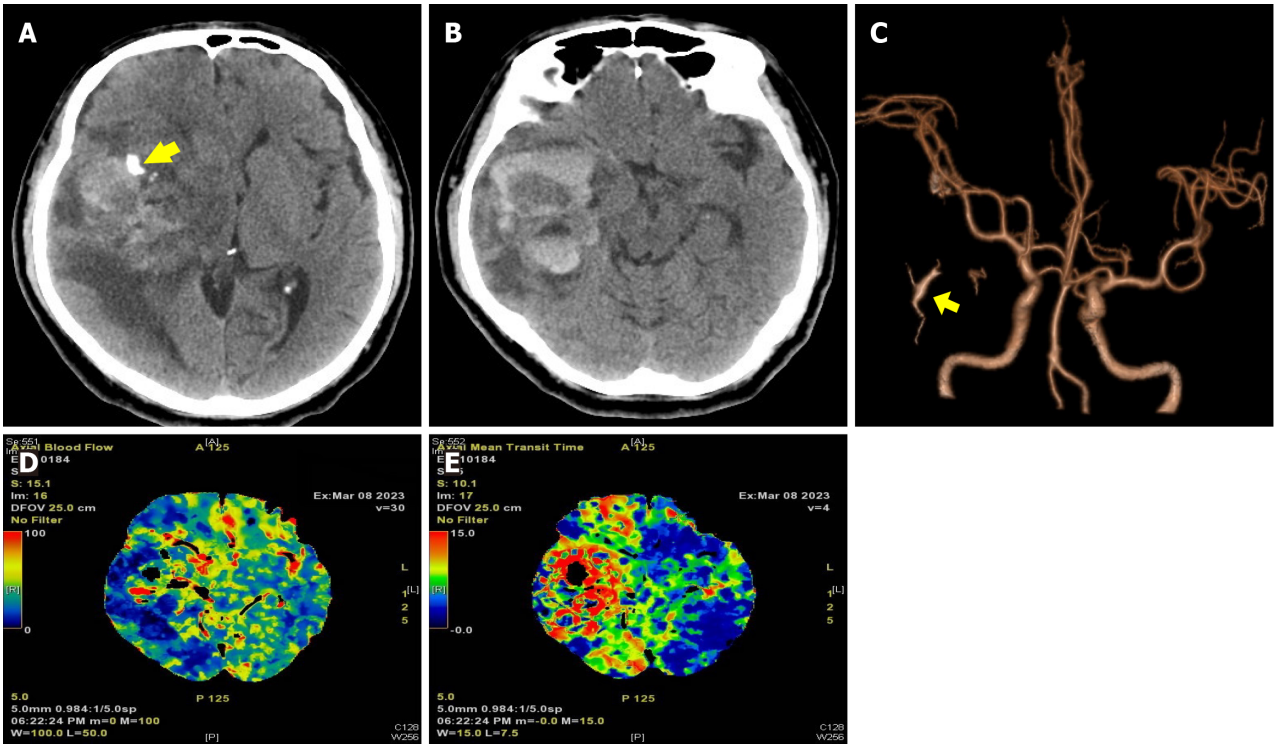


**Figure 1 Preoperative neuroimaging.** A and B: Cranial computed tomography revealed hemorrhage and edema in the right temporal lobe, calcification in the anterior portion of the mass (arrow), and a midline shift to the left; C: Computed tomography angiography revealed right middle cerebral artery elevation and an early drainage vein (arrow); D and E: Computed tomography perfusion imaging showed increased cerebral blood flow and mean transit time in the lesion area.

1.88 ppm and a severely decreased N-acetyl aspartate/Cr ratio of 1.49 ppm were observed within the lesion, consistent with the metabolic profile of glioma. Although the majority of the imaging findings supported the diagnosis of glioma, the presence of an early drainage vein on CTA images and dark vessel-like signals on T2-weighted images raised the possibility that a vascular malformation was present within the lesion, necessitating further angiographic evaluation.

Cerebral digital subtraction angiography (DSA) was utilized to confirm the presence of an AVM. It revealed the presence of two primary feeding vessels originating from branches of the right middle artery. Additionally, two large drainage veins with a caput medusae distribution characteristic of a developmental venous anomaly were also identified. One vein drained into the great cerebral vein leading to the sinuses rectus, and the other drained through the superior petrosal sinus into the ipsilateral transverse sinus. Numerous small, tortuous blood vessels were observed to form a nidus between the feeding artery and the draining vein (Figure 3).

## FINAL DIAGNOSIS

Pathological examination of the tumor revealed the diffuse and invasive growth of neoplastic glial cells with a single-cell morphology, round nuclei, clear borders, moderate atypia, perinuclear haloes, rare mitotic figures, branched proliferation of interstitial blood vessels, focal calcification, and patchy hemorrhage (Figure 4A and B). Immunohistochemical analyses demonstrated positive staining for glial fibrillary acidic protein (Figure 4C), S-100, and SRY-related HMG-box 10; negative staining for cytokeratins; scattered positive staining for p53; and a Ki-67 index of 30%. The final pathological diagnosis was oligodendroglioma (World Health Organization Grade II) associated with an AVM.

## TREATMENT

The patient underwent right temporal craniotomy, during which the tumor was observed to be grayish red with soft and tough consistencies, an uneven texture, and indistinct boundaries. It contained numerous slender blood vessels. The blood supply artery originating from the middle cerebral artery was occluded, and the tumor was removed in a piecemeal manner. Intraoperative fresh-frozen analysis confirmed the presence of low-grade gliomas, and microscopic examination indicated that the lesion had been nearly completely excised. As no preoperative embolization was conducted due to the lack of a facility for interventional embolization at our institution, the intraoperative bleeding volume was significant, at about 2000 mL. To mitigate the risks of intracranial hemorrhage and cerebral edema, the post-craniotomy removal of the tracheal intubation was delayed and the patient was transferred back to the surgical intensive care unit for further management.

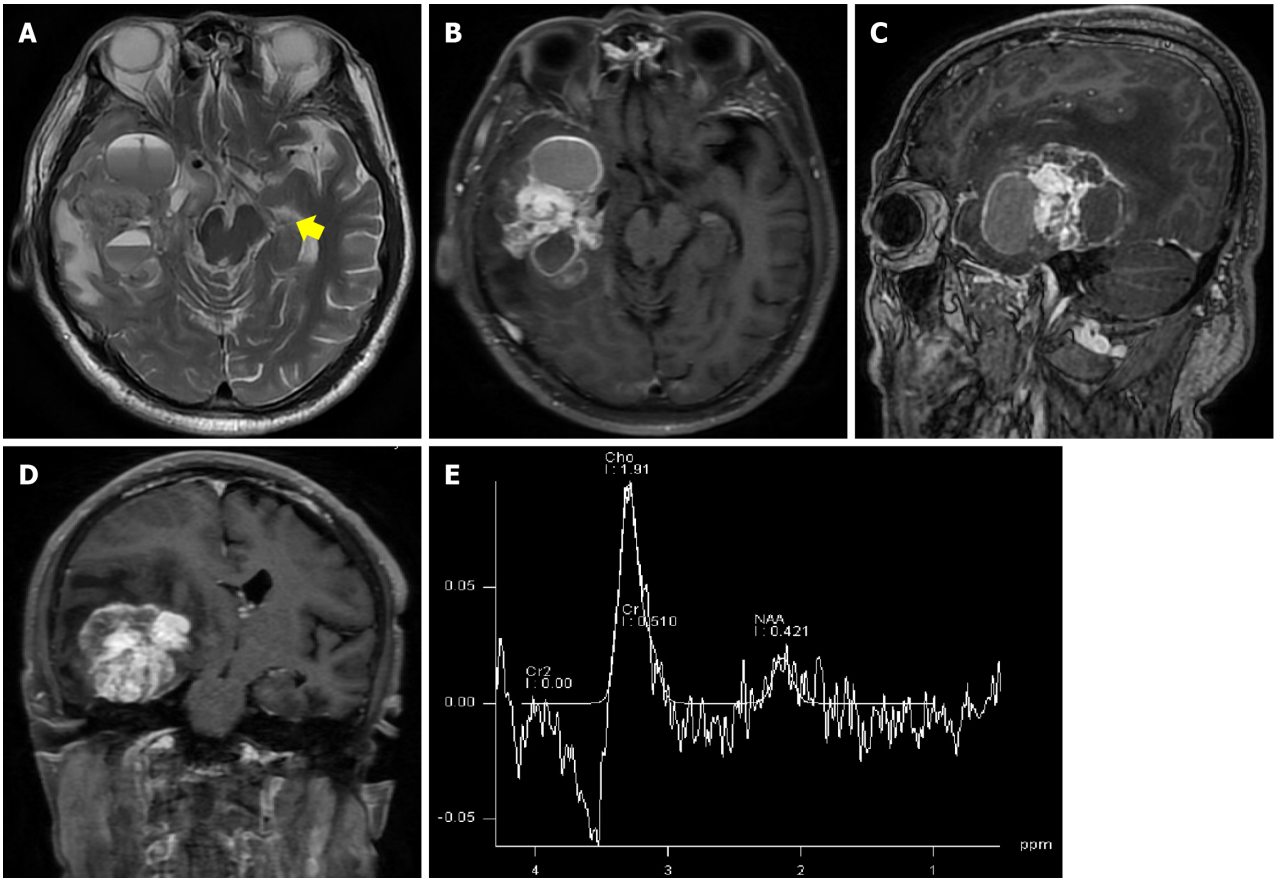


**Figure 2 Preoperative neuroimaging.** A: T2-weighted axial magnetic resonance imaging demonstrated cystic and solid space-occupying lesions, a visible air-fluid level in the cyst, and flow void signs within the lesion (arrow); B–D: Gadolinium-enhanced T1-weighted magnetic resonance imaging of the brain revealed a 4.5 cm × 6.3 cm × 4.6 cm necrotic cystic lesion with heterogeneous enhancement involving the basal ganglia, with a midline shift and uncal herniation; E: Magnetic resonance spectroscopy revealed aberrant metabolic function. An increased choline/creatine (Cr) peak ratio and a decreased N-acetyl aspartate/Cr ratio, matching the metabolic signature of glioma, were detected within the lesion.

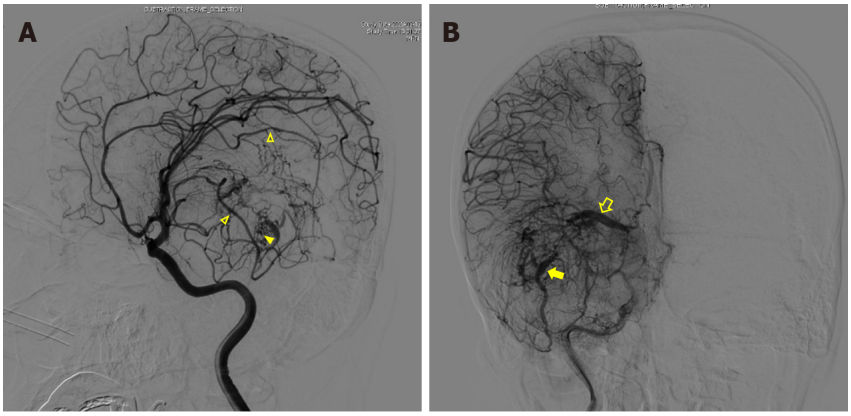


**Figure 3 Preoperative cerebral angiography.** A: Two feeding vessels originating from the branches of the right middle artery (open triangles) and two large draining veins were seen in the early arterial phase. Numerous small tortuous blood vessels were observed to form a nidus between the feeding artery and the draining vein (solid triangle); B: In the late arterial phase, both draining veins exhibited a caput medusae distribution consistent with a developmental venous anomaly; one drained into the great cerebral vein and to the sinuses rectus (open arrow), and the other drained through the superior petrosal sinus to the ipsilateral transverse sinus (solid arrow).

## OUTCOME AND FOLLOW-UP

Postoperative head CT examination revealed minor hemorrhaging in the surgical cavity and resolution of the midline shift (Figure 5A). At the time of discharge, the patient exhibited no apparent neurological deficit; he had a modified Rankin Scale score of 5, a Glasgow Coma Scale score of 15, and a National Institutes of Health Stroke Scale score of 0. Following discharge, the patient underwent radiotherapy and chemotherapy as planned. A follow-up enhanced MRI

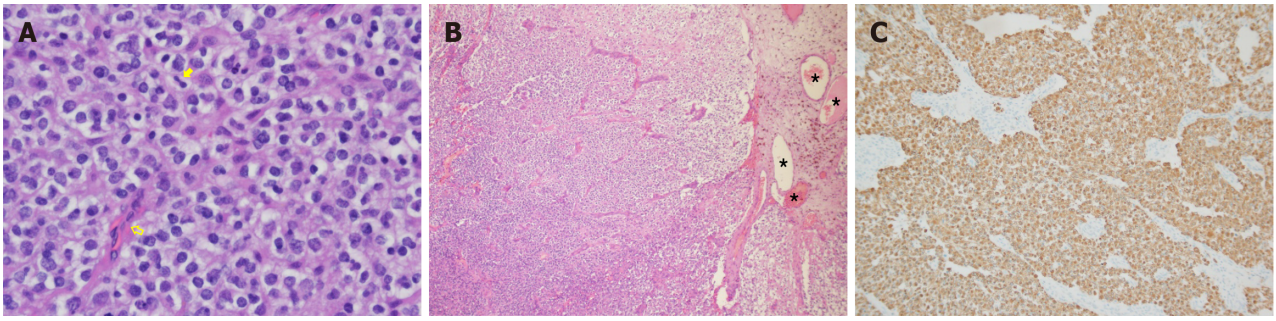


**Figure 4 Histological findings.** A: Examination of the neoplastic glial cells revealed a single-cell morphology, round nuclei, clear borders, moderate atypia, perinuclear haloes, rare mitotic figures (solid arrow), endothelial cell hyperplasia (open arrow), focal calcification, and patchy hemorrhage [hematoxylin and eosin (HE) stain, magnification 400 ×]; B: The tumor contained a large number of blood vessels of various sizes (asterisks; HE stain, magnification 40 ×); C: Immunohistochemical analysis showed strong cytoplasm staining for glial fibrillary acidic protein (magnification 100 ×).

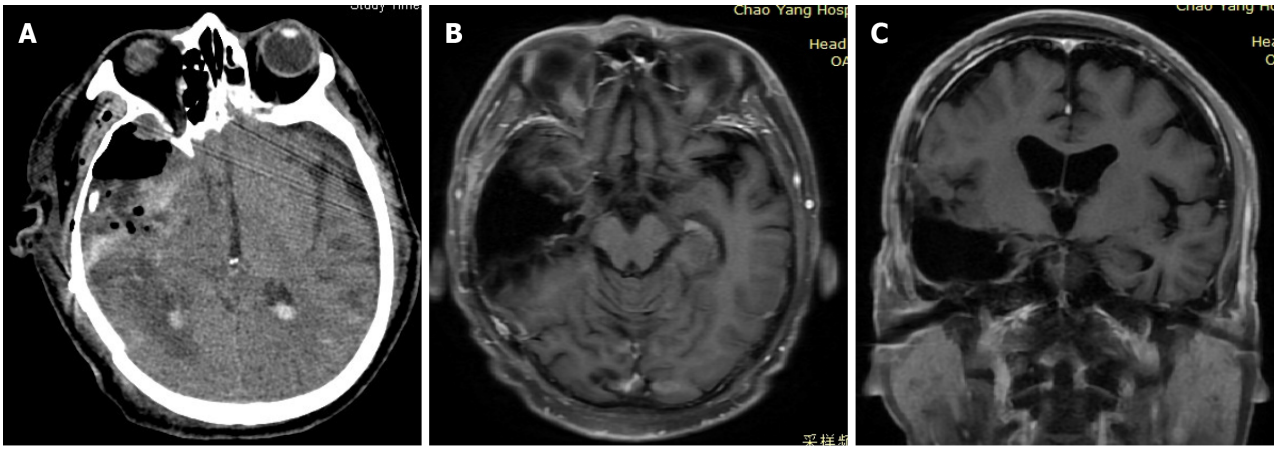


**Figure 5 Postoperative neuroimaging.** A: Postoperative head computed tomography showed a small amount of bleeding in the surgical cavity and the midline return; B and C: Enhanced magnetic resonance imaging performed at 6 months postoperatively revealed no definitive sign of recurrence.

examination performed 6 months postoperatively showed no definitive sign of recurrence (Figure 5B and C).

## DISCUSSION

The coexistence of glioma and AVM has been rarely reported in the literature, primarily in case reports[5-21]. These lesions may present simultaneously in the same part or distinct regions of the brain, or occur sequentially in the same area[5,9,15]. Additionally, the literature contains isolated descriptions of the occurrence of de novo gliomas coexisting with AVMs in the same part of the brain[7,12]. In most cases, however, these mixed lesions are discovered simultaneously in the same cerebral region[4,6,8,10,11,13,14,16-21]. Vascular malformations can be associated with various glial components, including oligodendroglioma[4,7], pilocytic astrocytoma[10], pleomorphic xanthoastrocytoma[9,14], and the predominant type, glioblastoma[6,8,11-13,16,18-21]. Although the precise relationship between these lesions remains unclear, numerous studies suggest that vascular formation in gliomas and the development of AVMs are related to vascular endothelial growth factor (VEGF)[5]. Angiogenesis is stimulated by the upregulation of VEGF and other pro-angiogenic factors. VEGF overexpression in tumors, attributed to the upregulation of hypoxia-associated factors, induces aberrant vascular proliferation that is unable to adequately oxygenate the rapidly expanding tumor tissue, creating a cycle of hypoxia and dysfunctional angiogenesis[11]. Tumor-secreted VEGF binds to endothelial cells expressing VEGF receptors, promoting neoangiogenesis[11]. Contemporary research suggests that the rapid growth of gliomas necessitates a substantial increase in blood supply. Angiogenic factor levels are high in and around these tumors, facilitating the proliferation of new blood vessels. However, the convoluted nature of these vessels hinders their ability to adequately supply the growing neoplasms, resulting in a detrimental cycle that culminates in the development of vascular malformations. Some researchers have proposed that the brain tissues surrounding AVMs experience ischemia, prompting the proliferation of glial cells and ultimately contributing to glioma formation. The histological term "angioglioma" has been used to denote a low-grade, highly vascular glioma with a favorable prognosis[14]. Such lesions must not be confused with high-grade malignant gliomas[6]. The angiographic pattern plays a crucial role in distinguishing conditions; true AVMs and AVM-associated neoplasms are typically visible on angiography, whereas angiogliomas are avascular or exhibit only a "tumor blush" with no evidence of shunting[4]. In the present case, the tumor and arteriovenous fistula

were identified simultaneously. Cerebral arteriography can verify the presence of an arteriovenous fistula within a tumor, although the sequence of lesion development is challenging to ascertain. Imaging modalities such as MRI and DSA can convincingly reveal the presence of an arteriovenous fistula in a tumor.

The incidence of intracranial AVMs coexisting with brain tumors (most commonly gliomas) is 0.1%[6]. The radiographic features differentiating AVM from glioma are frequently obscured, especially in the presence of acute or subacute hemorrhage[21], which often leads to misdiagnosis or missed diagnosis, posing significant challenges and risks for subsequent treatment. Reports document four cases of the preoperative misdiagnosis of AVMs[6,13,14,17]; in three of these cases, no intracranial bleeding was present and no preoperative CTA or DSA examination was performed[6,13,14]. In the fourth case, DSA findings were negative and postoperative pathological examination led to the diagnosis of pleomorphic xanthoastrocytoma with AVM[17]. In addition, three cases of the preoperative misdiagnosis of glioma in the absence of intracranial bleeding have been reported[8,10,12]; no preoperative MRI examination was performed in two of these cases[8,12]. In the third case, preoperative MRI examination was performed, but the presence of bleeding signals obscured the tumor signal, resulting in misdiagnosis[10]. In all of these cases, postoperative pathological examination or autopsy confirmed the diagnosis of glioma combined with vascular malformation[8,10,12]. The remaining 10 cases described in the literature were accurately diagnosed as gliomas combined with AVMs through preoperative MRI, CTA, or DSA examination[4,5,7,9,11,16,18-21]. Cases with intracranial bleeding are more prone to missed diagnosis gliomas, whereas those without bleeding tend to be missed diagnosis AVMs. Thus, the utilization of both MRI and DSA is essential for the accurate diagnosis of these lesions. Contrast-enhanced MRI plays a critical role in diagnosis because it enables the detection of intracranial tumors, elucidation of their connections with vascular malformations, and identification of the need for CTA or DSA examination to exclude the possibility of such malformations upon the identification of vascular lacunae in or around the tumor on T2-weighted images[11,12,17]. The diagnosis of occult AVM that are DSA-negative is advantageous compared to other examinations[17]. The primary factor contributing to most misdiagnoses and missed diagnoses is the lack of comprehensive multimodal imaging assessment. Clinicians must maintain a vigilant attitude toward examination and treatment. For cases without intracranial bleeding, the presence of abundant blood flow lacunae on T2-weighted MRI images necessitates further evaluation with DSA to rule out the presence of concurrent intracranial vascular malformations. In cases of intracranial bleeding in atypical locations, comprehensive multimodal (*i.e.*, head CTA, DSA, and MRI) imaging assessment to identify intracranial vascular abnormalities or neoplastic lesions is recommended. For cases in which the diagnosis remains unclear after MRI and DSA examination, auxiliary examinations such as MRS and CTP can be performed to reduce the potential for misdiagnosis or missed diagnosis.

In the present case, the patient was admitted after a single-phase stroke CT examination revealed intracerebral hemorrhage in the right temporal lobe and basal ganglia. CTP demonstrated increased perfusion with elevated rCBF in the right temporal lobe. Subsequent contrast-enhanced MRI displayed heterogeneous enhancement within the hemorrhagic lesion, characterized by cystic necrotic regions and the limited presence of vascular lacunae. To mitigate ambiguity, additional MRS and DSA examinations were performed. MRS indicated the presence of a glioma, and DSA confirmed the existence of arteriovenous drainage within the lesion. Consequently, the preoperative utilization of multimodal imaging techniques can help healthcare providers to make the diagnosis of glioma coexisting with AVM, thereby facilitating subsequent therapeutic interventions.

Most existing reports on gliomas combined with AVMs are case studies. No standardized treatment protocol or guideline for this condition has been established. However, a review of the existing literature suggests that treatment planning in such cases should prioritize the addressing of the lesion responsible for the patient's presenting symptoms, particularly in cases of non-adjacent diseases. When the tumor is malignant, resection of the malformation may not be recommended due to the short median survival time and the risk of hemorrhage is approximately 2% *per* year[6,9]. The optimal treatment for mixed lesions in the same location is surgical resection, but this procedure is associated with significant challenges, especially regarding the intraoperative maintenance of hemostasis[4,7,8,11,13,14,16,17,19,20]. Consequently, preoperative embolization using methods similar to those applied for AVMs has been recommended to mitigate the risk of intraoperative bleeding[7,13,16,19,21]. Imai *et al*[16] first reported the use of a low concentration (20%) of n-butyl cyanoacrylate and a detachable coil to embolize the tortuous and dilated vessels inside the tumor. After embolization, the lesion was resected completely, with an intraoperative bleeding volume of 60 mL[16]. The authors advocated for the performance of preoperative embolization as a precautionary measure for tumors situated in eloquent regions and noted the need for preoperative provocation testing[16]. Although embolization is a technically routine procedure, the high vascular density, tumor infiltration of vessel walls, and absence of the glial plane typically found in AVM make vessel coagulation more challenging than with AVM in these cases[7]. Despite preoperative embolization, McKinney *et al*[7] experienced significant intraoperative bleeding and brain tissue protrusion, leading to the patient's death on the sixth postoperative day despite complete tumor resection. In the present case, preoperative embolization was not performed. During surgery, the tumor's rich vascularization and ample blood supply complicated hemostasis, resulting in approximately 2000 mL of blood loss during total excision. Hence, although craniotomy enables the concurrent resection of neoplastic and AVM components, the procedure is associated with significant intraoperative bleeding and hemostasis challenges. Interventional embolization before craniotomy for tumor removal is thus recommended[22].

## CONCLUSION

The combination of glioma and AVM is an exceedingly rare condition, and the two lesions may mask each other when they are located at the same site in the brain. Multimodal preoperative examination can help clinicians minimize the risk

of misdiagnosis or missed diagnosis, and provides valuable insights for subsequent treatment. Craniotomy for the removal of glioma and AVM is feasible, and preoperative interventional embolization is recommended to reduce intraoperative bleeding and associated risks.

## FOOTNOTES

**Author contributions:** Guo P and Li JP were responsible for the conception, design, and coordination of the study; Guo P conducted the literature search and drafted the manuscript; Cao WY assisted with data analysis; Guo P, Sun W, and Song LX performed data collection and manuscript revision. All authors reviewed and approved the final version of the manuscript.

**Informed consent statement:** Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. The patient have been informed that their identity will remain confidential, and all identifying information will be anonymized to ensure privacy.

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