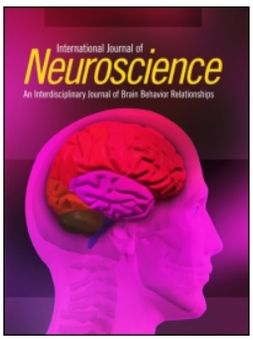

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Secondary cerebellopontine angle oligodendroglioma after cranial irradiation: A case report and literature review

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Abstract

Purpose/Aim: Cerebellopontine angle (CPA) oligodendrogliomas are very rare, and only 3 preoperative cases have been confirmed. Secondary cerebellopontine angle oligodendrogliomas after radiation therapy are exceptionally rare, and no other cases have been reported.

Case report: We present a case of a 25-year-old male with CPA oligodendroglioma who experienced hearing loss in right ear with walking instability for more than 2 months. The patient underwent craniotomy in our hospital because of grade II astrocytoma of the right temporal lobe 10 years ago. Postoperative radiotherapy lasted for 30 days, and six rounds of chemotherapy were performed. Magnetic resonance imaging (MRI) of the head revealed a cystic lesion located in the right CPA. The patient underwent surgery without obvious complications, and the tumor was subtotally removed. Histopathological examination revealed a diagnosis of oligodendroglioma, World Health Organization (WHO) grade II. The patient was discharged on the tenth postoperative day with a good recovery. Two weeks after discharge, chemotherapy with temozolomide and radiotherapy were performed. The patient remained well at 8 months follow-up.

Conclusions: To the best of our knowledge, no other cases of secondary CPA oligodendroglioma after cranial irradiation have been reported in the literature. Compared with general oligodendroglioma, the tumor has no typical calcification and is more aggressive. The cranial nerves in the CPA area are closely adhered, and the blood supply is abnormally rich. It is difficult to completely remove the tumor. Postoperative radiotherapy and chemotherapy should be carried out as soon as possible.

KEYWORDS Cerebellopontine angle; Oligodendroglioma; Radiation-induced glioma

1. Introduction

Oligodendrogliomas account for 5-10% of gliomas and arise from oligodendrocyte malignant tumors[1]. It is a diffuse, infiltrating, and slow-growing glioma that usually occurs in the supratentorial cerebral hemispheres, especially in the frontal and temporal lobes[2]. Oligodendrogliomas localized in the posterior fossa are rarely observed, with the vast majority arising in the cerebral hemispheres[1, 3-6]. Oligodendrogliomas localized in the CPA are exceptionally rare; to date, only 3 cases have been reported in the English literature[7, 8]. One case was primary oligodendroglioma in a child[8], and the other two cases were not described and discussed in detail[7]. We report an unusual case of an adult patient with oligodendroglioma arising in the CPA. The patient was diagnosed with grade II primary astrocytoma localized in the left temporal lobe 10 years ago. After surgical resection and concurrent chemoradiotherapy, the patient developed oligodendroglioma in the CPA. To our knowledge, this is the first report of such a case. We aimed to discuss the clinical characteristics, diagnosis and treatment of our case in relation to the literature to improve the understanding of this disease.

2. Case report

A 25-year-old male presented to our clinic with a more than two-month history of hearing loss in the right ear with walking instability. He had no vomiting, fever, headache, facial numbness, inclination of the mouth, cough after drinking water, hoarseness, or dysphagia. Ten years ago, the patient presented to our institution with epilepsy, cranial MRI revealed a 1.5 cm × 3.0 cm × 3.5 cm solid lesion located in the depths of the right temporal lobe. Precontrast MRI demonstrated that the tumor was hypointensity on T1-weighted images and hyperintense on T2-weighted images, contrast-enhanced MRI demonstrated slightly heterogeneous postcontrast enhancement (Figures 1A-F). The patient underwent craniotomy, intraoperative findings were that the tumor was found in the right medial temporal lobe, the boundary with the surrounding brain tissue was unclear, the tumour was soft and reddish grey, some tumor tissues were gelatinous and have abundant blood supply. The tumor was completely removed under microscope. Histopathological examination revealed a diagnosis of astrocytoma, WHO grade II (Figures 2A-D). Postoperative radiotherapy lasted for 30 days, and six rounds of chemotherapy were performed. The total dose of radiotherapy was 54 Gy. Neurological examination revealed that the patient had a clear mind, equal size and equal circle of bilateral pupils, symmetrical facial sensation, symmetry of nasolabial groove, no deviation of mouth angle, middle extension of tongue, and normal eye movement, and he was sensitive to light reflection. Myodynamia and muscular tension of all extremities were normal with a negative Barthel's sign and positive dyskinesia sign. Cranial MRI performed with and without intravenous contrast revealed a 2.0 cm × 2.0 cm × 3.0 cm solid lesion located in the right cerebellopontine angle. Precontrast MRI showed that the tumor was isointense on T1-weighted images and slightly hyperintense on T2-weighted images. Contrast-enhanced MRI showed homogeneous postcontrast enhancement with a clear boundary. No obvious enhancement lesion was found in the right temporal lobe (Figures 3C-F). Thin slice computed tomography (CT) of the middle ear and mastoid showed bilateral internal auditory canal symmetry and no obvious enlargement of the right internal auditory canal (Figure 3B). Thus, the presumed diagnosis was meningioma in the right CPA. On March 18, 2020, the patient underwent surgery in the lateral position under general anesthesia. The right retrosigmoid sinus approach was performed. When cerebrospinal fluid was fully released and the cerebellar hemisphere was distracted, a reddish brown, soft, and relatively tough tumor without

calcification was observed in the right CPA. The tumor was located outside the cerebellar cortex, and a small amount of tumor protruded into the internal auditory canal, which was not closely connected with the facial auditory nerve and trigeminal nerve, and it was significantly adhered to the lower cranial nerve. Near-total excision of the tumor was achieved during surgery, but a small amount of tumor remained at the lower cranial nerve. The tumor blood supply was abundant, and hemostasis was extremely difficult, resulting in an operation time of 11 h. The postoperative CT scan showed that there was still a small amount of bleeding in the operation area (Figure 5A), which was absorbed after conservative treatment. Histopathological examination revealed a diagnosis of oligodendroglioma, WHO grade II (Figures 4A-H). The patient was discharged on the tenth postoperative day. Postoperatively, the patient made a good recovery. Walking instability was improved, and no new neurological deficits occurred, but hearing loss of the right ear did not improve. Two weeks after discharge, chemotherapy with temozolomide and radiotherapy were performed. Post-operative gadolinium-enhanced MRI at 1 month after the surgery showed near-total tumor resection (Figure 5B). The patient remained well at 8 months follow-up. Follow-up MRI scan 6 months after surgery indicated that the lesions were well controlled (Figure 5C).

3. Discussion

Oligodendroglioma is a neuroepithelial tumor that can be divided into oligodendroglioma (grade II) and anaplastic oligodendroglioma (grade III) according to WHO classification[9]. It often occurs in the subcortical white matter and extends to the cortex. The clinical course and prognosis of oligodendroglioma (grade II) are better than those of other gliomas. Grade III anaplastic oligodendrogliomas are more malignant tumors, which indicates a poor prognosis[10]. In our case, the pathological diagnosis was a grade II tumor. Immunohistochemically, CD34, Olig2, and synaptophysin were positive, Ki67 was 15%, and the prognosis was relatively good. However, infratentorial oligodendrogliomas may be more malignant than supratentorial oligodendrogliomas[5], tumor was not completely removed, and the recurrence time may be short, which all affect clinical course and prognosis. Further follow-up is needed.

Epilepsy was the most common first symptom in oligodendroglioma patients, followed by focal neurological deficit and cognitive impairment[11]. The symptoms of focal neurological deficits vary according to the location of the tumor, such as hemiplegia, aphasia, limb numbness, blurred vision, and abnormal smell, among others[2]. Our patient's symptoms are similar to those of acoustic neuroma, which is characterized by hearing loss. Thus, it is easily misdiagnosed as acoustic neuroma. During the operation, it was found that the tumor invaded the internal auditory canal, compressed the cochlear nerve, and caused hearing loss. At the same time, ataxia caused by cerebellar damage, such as walking instability, was found. The majority of oligodendrogliomas in the cerebellopontine angle described by Ellenbogen were mostly due to tumors located in the cerebellar hemisphere, and the patient's symptoms were mainly intracranial hypertension and ataxia[8].

On CT scan, oligodendrogliomas primarily exhibit isodense or slightly low densities, uneven densities, calcified cystic necrosis, and intratumoral hemorrhage. Enhanced CT scans demonstrate mild, moderate, or obvious heterogeneous enhancement. There are various enhancement modes, such as spot, patchy, nodular, or annular enhancement. On MRI, there are isointense or hypointense

signals on T1-weighted images and hyperintense signals on T2-weighted images without enhancement[12]. Our patient's cranial MRI showed obvious homogeneous enhancement in the CPA. Oligodendrogliomas easily invade the pia mater and cause obvious connective tissue reactions. The characteristics of oligodendrogliomas are strip and mass calcification inside the lesion. The incidence rate is as high as 50%-80%[2, 13]. In general, low-grade oligodendrogliomas (grade II) often have calcification, while anaplastic oligodendrogliomas (grade III) do not calcify[14]. In our case, although it was a low-grade oligodendroglioma, there was no typical calcification on CT scan (Figure 1A), and the enhancement of the tumor was obvious on MRI. The CPA oligodendroglioma described by Ellenbogen had calcification[8], and the other 2 cases were not described in detail. Therefore, CPA oligodendrogliomas may not have calcification, which makes them easy to confuse with meningiomas in the CPA. Our patient was misdiagnosed with meningioma before the operation.

Oligodendrogliomas can occur in any age group, but they are rarely seen in children and young people. They occur more often in older patients (approximately 40-50 years of age)[2]. Both our patient and the patient reported by Ellenbogen were young. The cause of oligodendrogliomas is the same as that of other tumors, the basic reason is that gene mutation caused by internal and external factors causes normal cells to mutate into tumor cells, resulting in uncontrolled cell growth and tumor evolution. External factors include viral infection, chemical pollution, and environmental factors, including ionizing radiation. This patient suffered from grade II right temporal lobe astrocytoma 10 years ago. After surgery, 54 Gy total dose radiotherapy was performed. The oligodendroglioma occurred on the same side of the cerebellopontine angle. The distance between the cerebellopontine angle and the temporal lobe was relatively small. We considered that the secondary tumor may have been related to the patient's previous radiotherapy. Many previous articles have reported that a total dose of radiotherapy more than 10 Gy may cause secondary tumors[15, 16]. The secondary tumor caused by radiotherapy must be consistent with the following: (1) the tumor occurs in or near the previous irradiation area; (2) the incubation period is more than 6 years; (3) there is no genetic tendency; and (4) there is a histological difference between the primary and secondary tumors. Our patient developed a rare secondary oligodendroglioma that m*et al*l these criteria. Therefore, when considering oligodendroglioma secondary to radiotherapy, one must remember that the most common intracranial tumors secondary to radiotherapy are meningioma, glioma, and sarcoma. Anaplastic astrocytoma and glioblastoma are more common among gliomas, and secondary oligodendroglioma is rare. He *et al.* [17] reported a case of oligodendroglioma in the frontal lobe caused by radiotherapy for medulloblastoma. Doskaliyev *et al.*[18] reported a case of oligodendroglioma in the temporal lobe caused by radiotherapy for a pineal region tumor. We report a rare case of secondary oligodendroglioma arising after cranial irradiation in the CPA. It has not been reported thus far.

Oligodendrogliomas are usually characterized by soft texture, calcification, unclear boundary, abundant blood supply, and gray-white appearance on operation. In contrast, this case of oligodendroglioma located in the CPA had an extremely abundant blood supply, and the tumor was aggressive. During the operation, it was found that the tumor grew into the internal auditory canal, similar to an acoustic neuroma. In contrast to acoustic neuroma and meningioma, the tumor adhered closely to the lower cranial nerve and cerebellar hemisphere brainstem, which made it difficult to completely remove the tumor and may lead to poor prognosis and a short tumor recurrence time. Therefore, timely radiotherapy and chemotherapy were needed to delay the growth rate of the tumor.

4. Conclusion

In summary, we described a patient with a secondary CPA oligodendroglioma that developed after cranial irradiation. To the best of our knowledge, this is the first report of such a case. It should be kept in mind that oligodendrogliomas may present as CPA-localized tumors. CPA oligodendroglioma is very easy to confuse with acoustic neuroma and meningioma. Compared with general oligodendroglioma, the tumor has no typical calcification and is more aggressive. The cranial nerves in the CPA area are closely adhered, and the blood supply is abnormally rich. It is difficult to completely remove the tumor. Postoperative radiotherapy and chemotherapy should be carried out as soon as possible.

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Ethics approval and consent to participate

The study was approved by the ethical committee of the Second Affiliated Hospital of Anhui Medical University.

Consent to publication

Informed written consent was obtained from the patient's kin for publication of this case report and accompanying images.

Competing interests

The authors report no conflict of interest.

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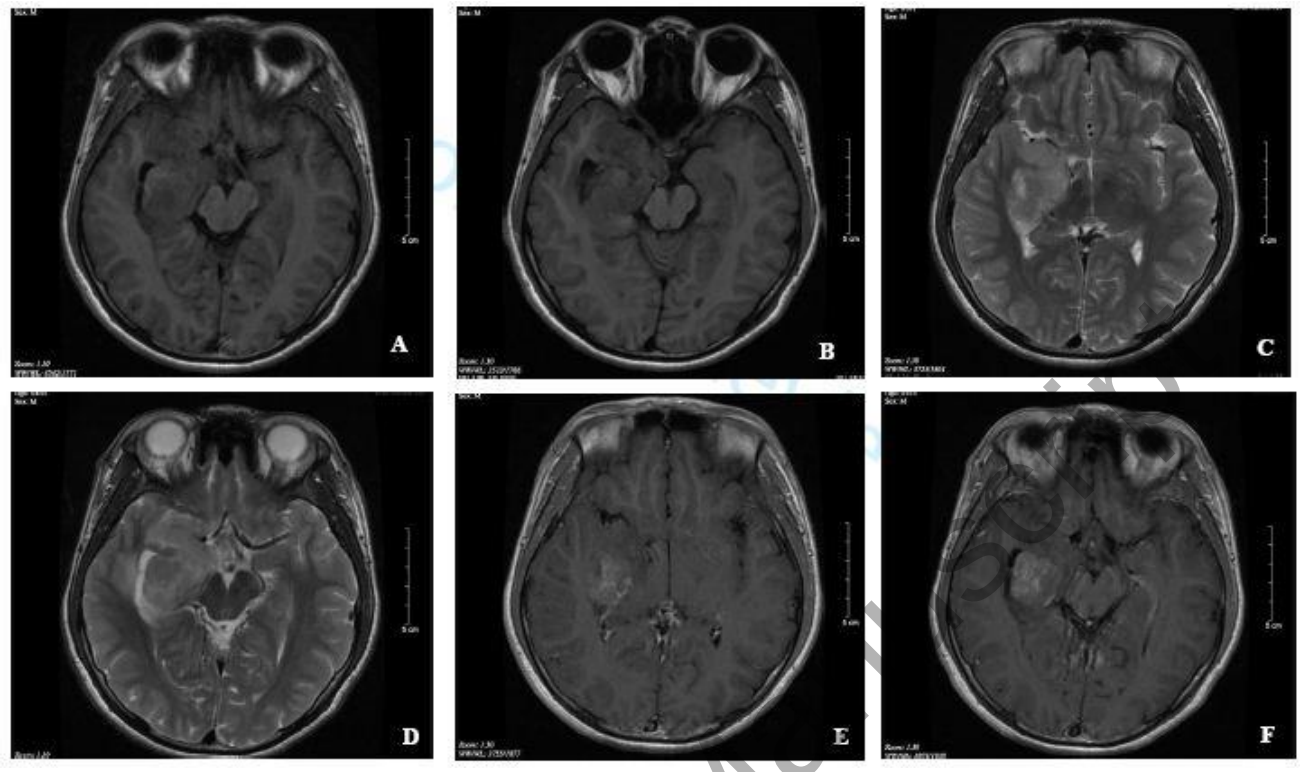

Figure 1. The first preoperative MRI of brain 10 years ago. Axial T1-weighted (A-B) and T2-weighted (C-D) images reveal the solid lesion of medial right temporal lobe with hypointensity on T1-weighted images and hyperintense on T2-weighted images. Axial T1-weighted post-gadolinium images (E-F) show slightly heterogeneous post-contrast enhancement of the lesion located in the right temporal lobe.

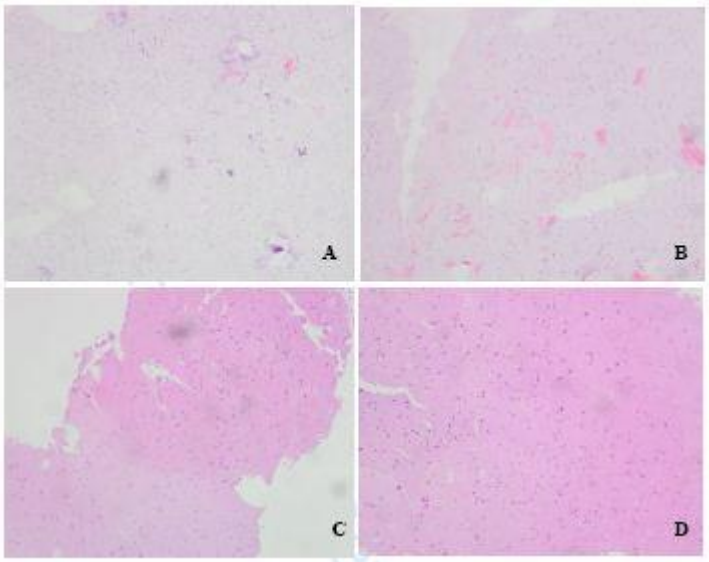

Figure 2. Histological findings of the first pathological sample (in the temporal lobe). The tumour cells were diffusely distributed with medium density and calcification and slightly heteromorphic nuclei without mitotic figures. Haematoxylin–eosin staining (A-B: ×40, C-D: ×100).

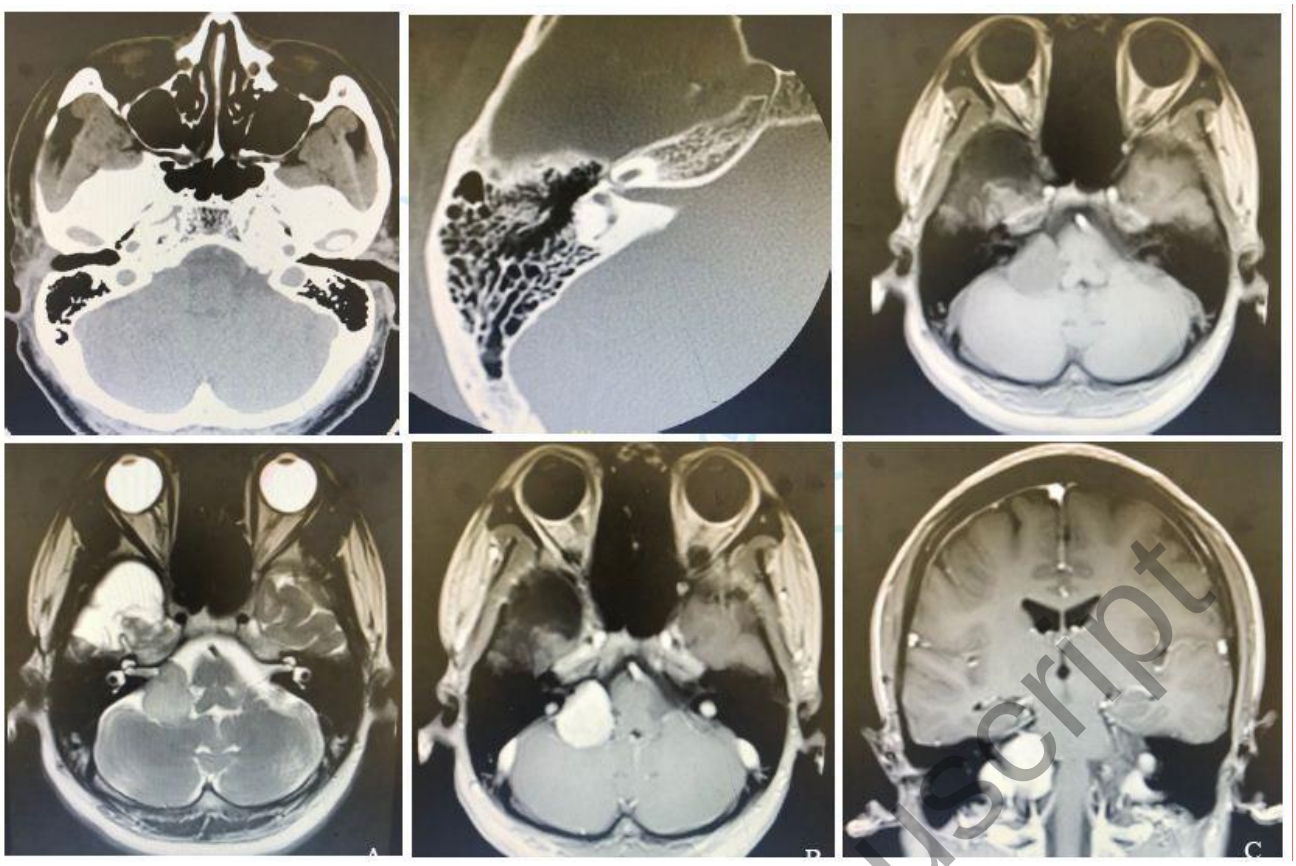

Figure 3. Preoperative CT and MRI of brain. (A) Axial non-enhanced CT brain demonstrating a heterogeneous cystic with no calcification in the right CPA. (B) Thin slice CT of middle ear and mastoid showed no obvious enlargement of right internal auditory canal. Axial T1-weighted (C) and T2-weighted (D) images reveal the solid lesion of right CPA with equal signals on T1-weighted images and slightly increased signals on T2-weighted images. (E) Axial and (F) coronal T1-weighted post-gadolinium images show avid enhancement of the lesion located in the right CPA.

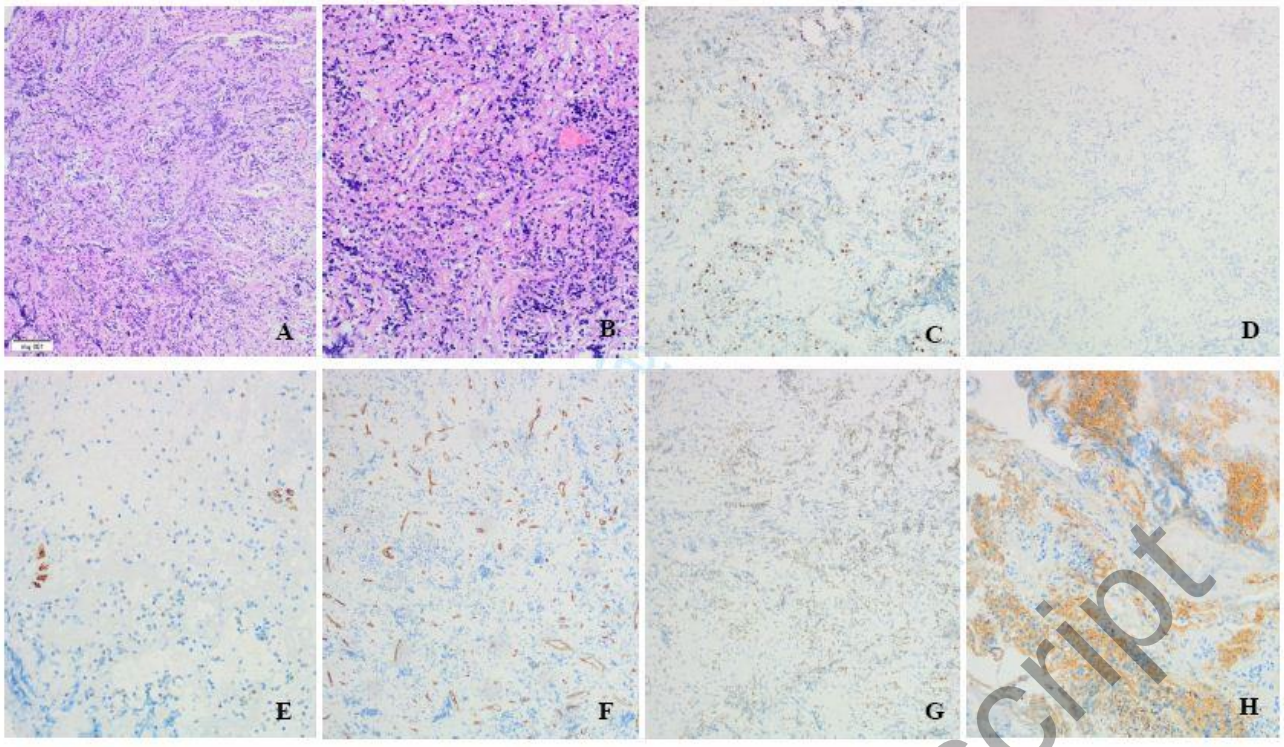

Figure 4. Histological findings. (A, B) Photomicrograph showing small round cells proliferated. Some areas were rich in cells. The shapes of proliferative cells were simple, and the nuclei were round. There were perinuclear halos and networks of branching capillaries in the stroma. Hematoxylin-eosin × 200 (A and B: ×100 and ×200, respectively). (C) Tumor cells show a Ki-67 labeling index of approximately 15%. Ki67 × 100. (D, E) IDH1 and vimentin immunostaining were negative. ×100. (F) Blood vessels showed CD 34 positivity. CD 34 × 100. (G, H) Olig2, and synaptophysin were positive.

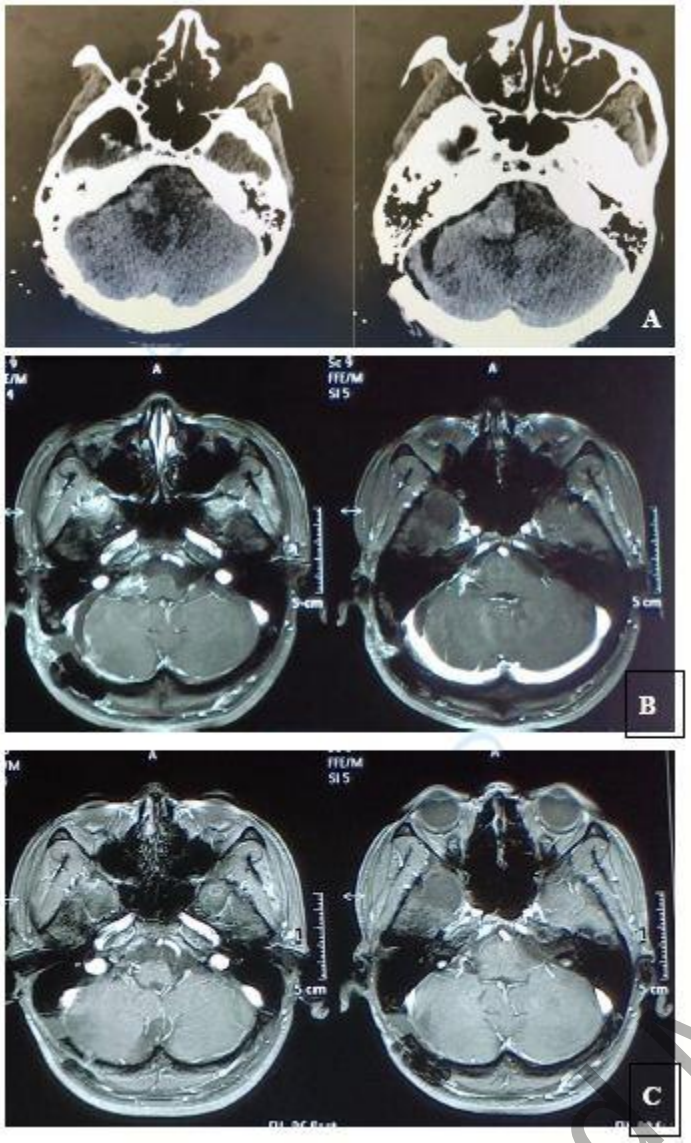

Figure 5. Postoperative CT and MRI. (A) The postoperative axial CT at 2 days after the surgery showed that there was still a small amount of bleeding in the operation area. (B) Postoperative gadolinium-enhanced MRI at 1 month after the surgery showed a few irregular enhancement lesions at the CPA in T1-weighted axial sections. (C) Follow-up T1-weighted gadolinium-enhanced axial MRI scan 6 months after surgery indicated no obvious enhancement lesions in the right CPA.