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Case report

Collision brain tumor with atypical meningioma and glioblastoma: Case report

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ABSTRACT

Introduction: Intracranial collision tumors are rare diseases in which two distinct neoplasms are found at the same location. We present an unusual case of an intracranial collision tumor composed of atypical meningioma (WHO grade II) and glioblastoma.

Presentation of case: The case was a 56-year-old woman hospitalized due to generalized weakness and dizziness. Imaging survey revealed a right frontal lobe extra-axial mass with significant perilesional edema. The patient underwent a one-stage craniotomy for tumor removal. The pathology revealed collision brain tumors of clear cell atypical meningioma (WHO grade II) and glioblastoma. The patient had an uneventful postoperative recovery.

Clinical discussion: The mechanism behind collision brain tumors remains unclear, and some experts consider these tumors sporadic events. Further research is needed to optimize preoperative diagnosis and surgical strategy for collision brain tumor patients.

Conclusion: Surgeon should consider intracranial collision tumors when brain image indicated unusual perilesional edema of meningioma. Though there is no standard treatment for these patients, it seems one-staged surgical treatment is feasible. To our knowledge this is the first case of collision tumors with clear cell atypical meningioma (WHO grade II) and glioblastoma.

1. Introduction and importance

It is very rare that two histologically distinct brain tumors appear at the same time in patients without a prior history of radiation therapy. In the literature, only about 20 cases of simultaneously occurring meningioma and glioma have been reported [1,2]. The pathogenesis of concurrent or collision brain tumors remains unknown [3].

We present here the case of a 56-year-old woman without a previous history of malignancy who had an unusual presentation of two different types of intracranial brain lesions – atypical meningioma (WHO grade II, clear cell type) and glioblastoma. To our knowledge, this is the first case an intracranial collision tumor consisting of these two distinct types of malignancies. This case report has been reported in line with the SCARE 2020 criteria [4].

2. Case presentation

A 56-year-old woman with prior history of hypertension presented to our emergency department for complaints of generalized weakness, dizziness, and severe headache. The clinical assessment indicated no significant neurologic deficits. Other symptoms included difficulty walking and frequent falling. A computed tomography (CT) (Fig. 1A) scan showed a large left frontal brain mass causing cerebral edema and mass effects. Preoperative gadolinium-enhanced MRI revealed that an approximately 8 cm (Fig. 1B, C, D) extra-axial mass with a homogeneously eccentric enhancing solid component and heterogeneous cystic parts was causing significant perilesional cerebral edema. Mass effects and subfalcine herniation were noted. Based on the MRI findings, we first considered the possibility of atypical meningioma.

The patient's meningioma was removed using a bicoronal craniotomy. The enhancing extradural solid tumor was removed intraoperatively. Beyond the tumor border, there was a yellowish necrotic tumor that was distinct from the original tumor. As expected, the highly enhanced lesion on MRI was meningioma, and was proved by the frozen section sent to our institute pathology lab. However, due to the unusual presentation of the excision margin of the tumor, the frozen biopsy was re-sent to pathologist, and the result was inconclusive for diagnosis.

The final histology and immunohistochemistry reports revealed two

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different tumors, one of which was clear cell type atypical meningioma (WHO grade II), composed of meningeal cells with clear cytoplasm in a syncytial pattern and the other was isocitrate dehydrogenase 1(IDH-1) mutant glioblastoma. The meningioma foci did not show mitotic figures, but there was brain tissue invasion to the surrounding high-grade glioma (Figs. 2 & 3).

Postoperative, the patient recovered without major complications and was extubated smoothly. A follow up MRI scan grossly showed total removal of the tumor. The patient then underwent adjuvant concurrent chemoradiation (CCRT), which she tolerated well. Postoperative follow up MR imaging showed glioblastoma recurrence at six months.

3. Clinical discussion

Though glioblastoma is the most common primary malignant brain tumor in adults [5], and meningioma is the most common brain tumor, interestingly, the coincidence of these two distinct type of tumor occurrence at the same location are extremely rare. In the literature, only about 20 cases of simultaneously occurring meningioma and astrocytoma have been reported, and most of the astrocytoma cases were high-grade [6,7]. We hereby report another case of coexistence of meningioma and astrocytoma, and to the best of our knowledge it is the first case of clear cell atypical meningioma (WHO grade II) with IDH-mutant glioblastoma.

For collision brain tumors, it is difficult to observe the natural history of disease progression, and the exact mechanism of collision brain tumors remain elusive. Some authors believe that collision brain tumors are merely coincidental [8]. Others suggest that the initial tumor can stimulate the growth of the second tumor by secretion of growth factors such as Platelet-derived growth factor (PDGF) [9,10]. Therefore, there are three hypotheses: (1) the glioma promotes the growth of the meningioma. Zhang et al. (2015) observed the dynamic development of a collision tumor of WHO grade II astrocytoma and WHO grade II anaplastic meningioma six years after removal of a benign meningioma [2]. This may indicate the stimulation of the astrocytoma promotes the growth of the meningioma; (2) the original meningioma induced the oncogenesis of the glioma [6,11]; (3) the two distinct tumors are co-stimulated by each other. Based on our limited clinical data, we could not conclude which tumor developed first in our case. But histological invasion of each tumor (Fig. 2C, D) hints that the collision tumor growth comes from each other's stimulation. In other case series, however, they have reported no evidence for collision brain tumors invading each other under histology exam [6]. Glioblastomas with IDH mutations are known to develop from precursors of the lower-grade glioma that escaped from initial diagnosis [12]. We consider the glioma may incubate in the patient for a while and induce the transformation of arachnoid cap cells to meningiomas [2].

As lower-grade glioma evolved to fast-growing glioblastoma, clinical symptoms occur in the patient. Of note, clear cell meningioma is an extremely rare type of WHO grade II tumor, which accounts only 0.2–0.8% of all meningiomas with high recurrence rate and more aggressive clinical behavior [13,14].

The treatment of patients with coincident glioblastoma and meningioma is controversial and requires special consideration. Firstly, we

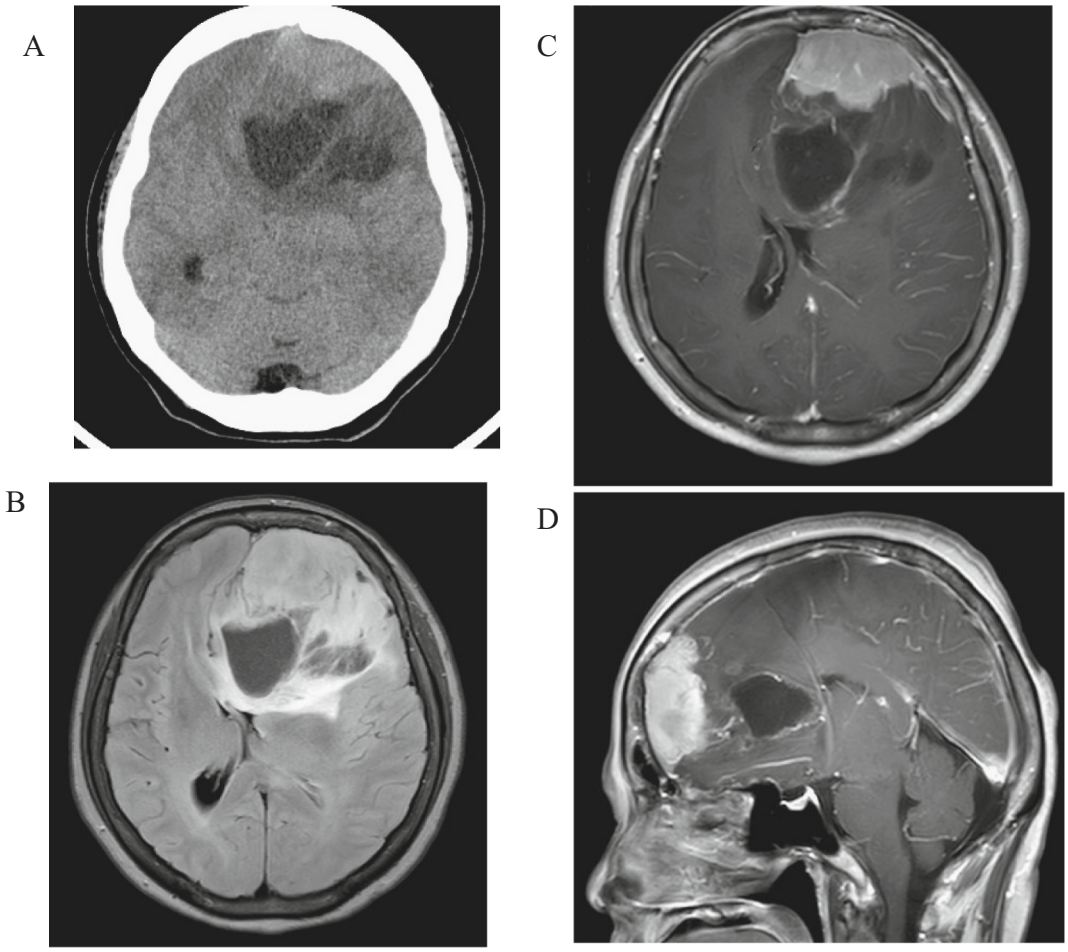

Fig. 1. Pre-operative computed tomography (CT) (A) and magnetic resonance imaging (MRI) T2 FLAIR (B) and T1 gadolinium-enhanced (C) showed an extra-axial tumor with a homogeneously eccentric enhanced lesion with cystic parts, which caused significant perilesional cerebral edema. T1 post-gadolinium imaging (C, axial; D, coronal) showed strongly enhanced dural lesions with unclear border of non-enhancing lesion.

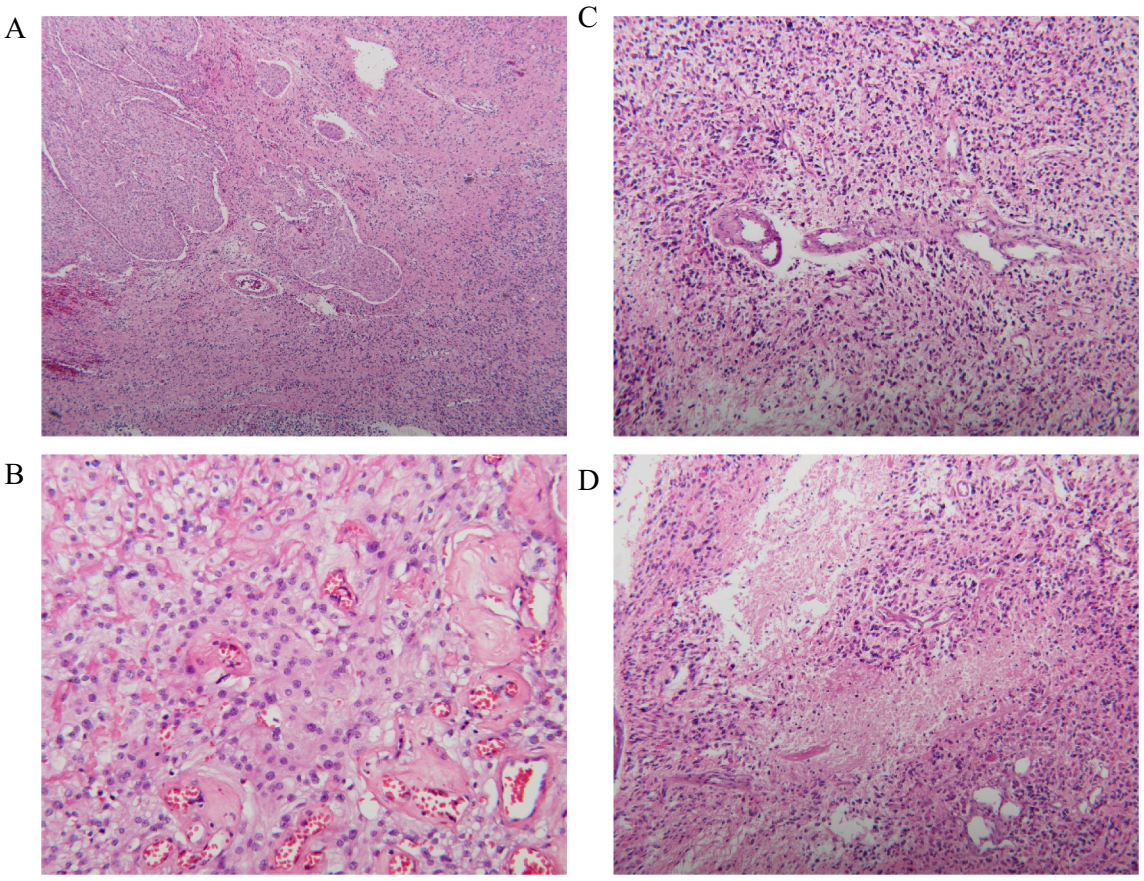

Fig. 2. Pathology image of hematoxylin and eosin (H&E) stained collision brain tumor consisting of World Health Organization (WHO) class II clear cell meningioma and glioblastoma. (A) The tumor comprised of clear cell meningioma (left upper of slide) with finger-like invasions to the surrounding glioblastoma (H&E 40×). (B) The clear cell meningioma component, showing cells with abundant cytoplasm and prominent perivascular and interstitial collagen (H&E 200×). (C, D) Images of the atypical astrocytes proliferating into meningioma tumor, with central necrosis.

believe that preoperative imaging is valuable for diagnosis and surgical management, but due to the rarity, the pathologist or surgeon may be unaware of this intracranial diagnosis. In current clinical practice, brain imaging cannot identify the collision tumors, and most cases are diagnosed postoperatively [1].

Secondly, an intraoperative biopsy of the suspected meningioma and the surrounding brain tissue is essential, and it is best to maintain appropriate perioperative communication with pathologists to alleviate discrepancies. In our case, although the initial intraoperative frozen histology section indicated meningioma, it was unusual that the resected margin contained necrotic brain tissue and possibly represented other types of brain lesions. It is possible that diagnoses of simultaneous brain pathologies from an initial frozen biopsy are missed due to lack of awareness.

Finally, as surgical removal is the definitive treatment, most case series have shown that one-stage tumor resection is feasible when two lesions are adjacent [3,6]. In our case, it was reasonable and straightforward to remove the contiguous lesions. The recurrence of glioblastoma occurred at the sixth month postoperative.

4. Conclusion

We report an extremely rare case of a collision brain tumor with clear cell type atypical meningioma, WHO grade II and IDH-mutant glioblastoma. We suggest that for patients with MRI showing an extradural lesion with significant perilesional cerebral edema, the possibility of a collision brain tumor should be considered, and this may alter clinical management of the patient.

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Ethical approval

Not applicable.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Research registration

Not applicable.

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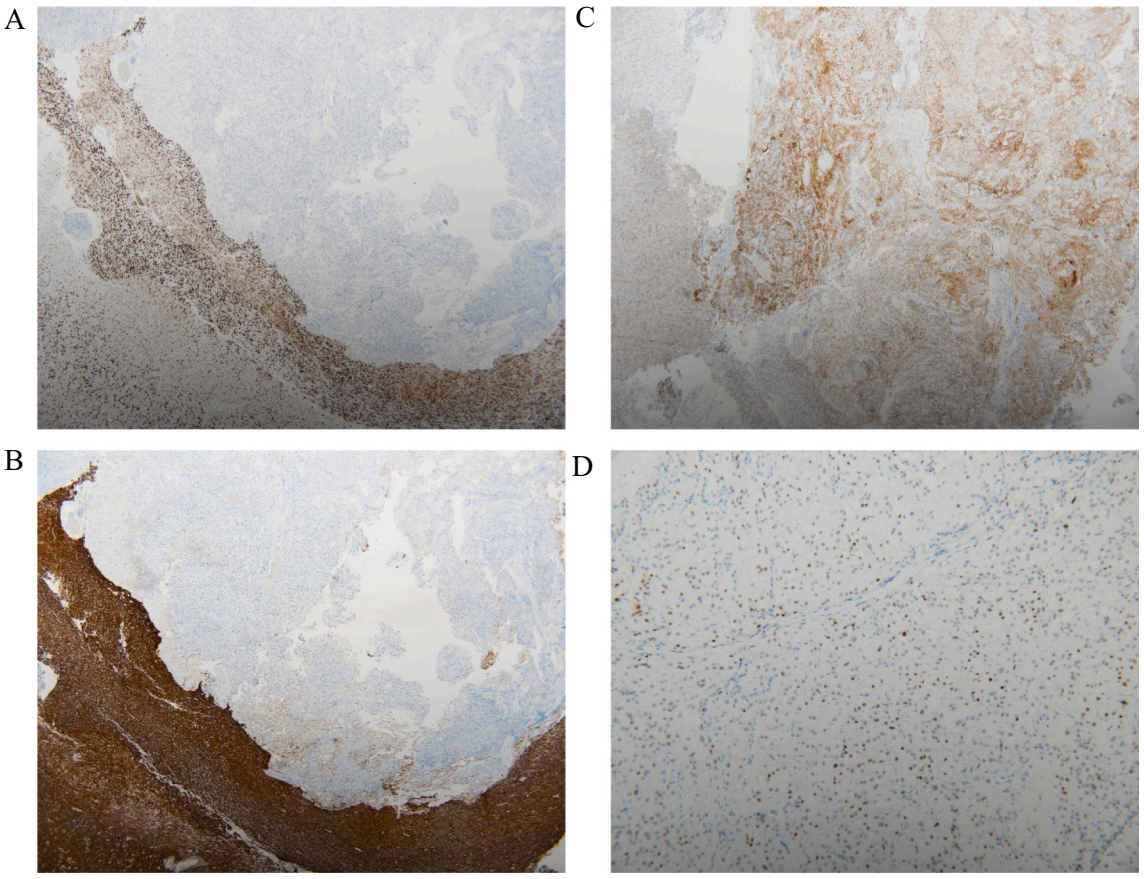

Fig. 3. Immunohistochemistry results for OLIG2 (positive on glioblastoma) (A), GFAP (positive on glioblastoma) (B), EMA (positive on both tumors and more strongly expressed by the meningioma) (C), PR (positive on meningioma) (D). EMA: epithelial membrane antigen; GFAP: glial fibrillary acidic protein; PR: progesterone receptor.

CRediT authorship contribution statement

MS Lin, resident, first author, writing the paper.
CH Lee, surgeon, background research, edit the manuscript.
SY Chen, literature review.
CC Shen, supervision.

Declaration of competing interest

All authors declare that they have no competing interests.

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