Frontal lobe epilepsy and hibernoma: a case report

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Case presentation

A 69-year-old female presented to the clinic with a 2-year history of intermittent headaches. Cranial computed tomography (CT) and magnetic resonance imaging (MRI) examination (Fig 1a and b) revealed a space-occupying lesion in the right frontal lobe, suggestive of a meningioma. Preoperatively, the patient experienced a single episode of epilepsy lasting for 2 minutes, relieved by antiepileptic medication.

Cranial surgery was performed based on the preoperative CT localisation. The tumour was completely separated and excised, measuring 4 × 3 × 2 cm, greyish-yellow in colour, and encapsulated (Fig 1e). The encapsulated surface was rich in blood vessels. Upon opening, the tumour was yellow, and lipid droplet-like fluid was observed on the surface (Fig 1f).

Postoperative pathology indicated a hibernoma. Haematoxylin and eosin staining revealed a complete thin capsule attached to the outside of the tumour that was lobulated. A network of capillaries could be seen inside. Under the microscope, polygonal/round cells with eosinophilic granular cytoplasm were seen. The cell nuclei were round and centrally located. In addition, many small vacuolated brown adipocytes and unilocular adipocytes were seen, with a considerable amount of blood vessels, spindle cells, and collagen fibres infiltrating around the tumour cells (Fig 2).

Immunohistochemistry showed CD34 to be positive in the vessels, and Ki-67 scattered positive with a proportion of <1%. Immunohistochemistry results were as follows: CK (-), S-100 (-), MDM2 (-), CDK4 (-), P53 (-), CD34 (vascular +), CD117 (-), and Ki -67 (+, <1%) [Fig 2].

Postoperative re-examination confirmed complete removal of the tumour, and the patient had no further epileptic seizures. Three years later, cranial MRI follow-up revealed no tumour recurrence (Fig 1c and d).

Discussion

Hibernomas are rare benign tumours composed of brown adipose tissue.[1] They are asymptomatic and slow-growing. Compared with lipomas originating from white adipose tissue, hibernomas are exceedingly rare, with fewer than 200 cases reported in the literature.[2] These tumours most commonly occur in the proximal axial skeleton, where fetal brown adipose tissue exists and continues into adulthood. They are most frequently found in the interscapular region, upper mediastinum, axilla, retroperitoneum, and neck. According to literature reports, hibernomas are extremely rare in the cranial cavity. In 1972, Vagn-Hansen et al[3] reported a case of intracranial hibernoma. Due to medical constraints at the time, detailed case descriptions of CT and MRI images were not available. Therefore, the diagnosis of this patient has significant clinical significance and academic value. The rarity of intracranial hibernomas in clinical diagnosis can easily be overlooked or misdiagnosed as other more common intracranial tumours such as meningiomas. Clinicians and pathologists need to remain highly vigilant for this rare tumour to ensure timely and accurate diagnosis and treatment.

Imaging characteristics of hibernomas

In radiology, differentiation between hibernomas and meningiomas is challenging. Hibernomas typically present as low- to medium-intensity on T1-weighted MRI and high intensity on T2-weighted MRI. Due to their origin from adipose tissue, hibernomas may show signal suppression in the fat-suppressed sequence of MRI. Due to the rich vascular supply inside the tumour, MRI post-contrast enhancement is evident. Also, due to the high fat content of hibernomas, they may appear as low density or iso-density lesions on CT scans.

Meningiomas typically present as iso- to high-intensity on both T1- and T2-weighted MRI and show significant uniform enhancement following contrast administration. Meningiomas often accompany the dural tail sign, an important distinguishing feature in imaging. Furthermore, the density of meningiomas on CT scans is quite uniform, with significant enhancement following contrast administration. Therefore, while there is overlap in imaging between hibernomas and meningiomas, detailed radiological analysis, especially the combination of fat-suppressed

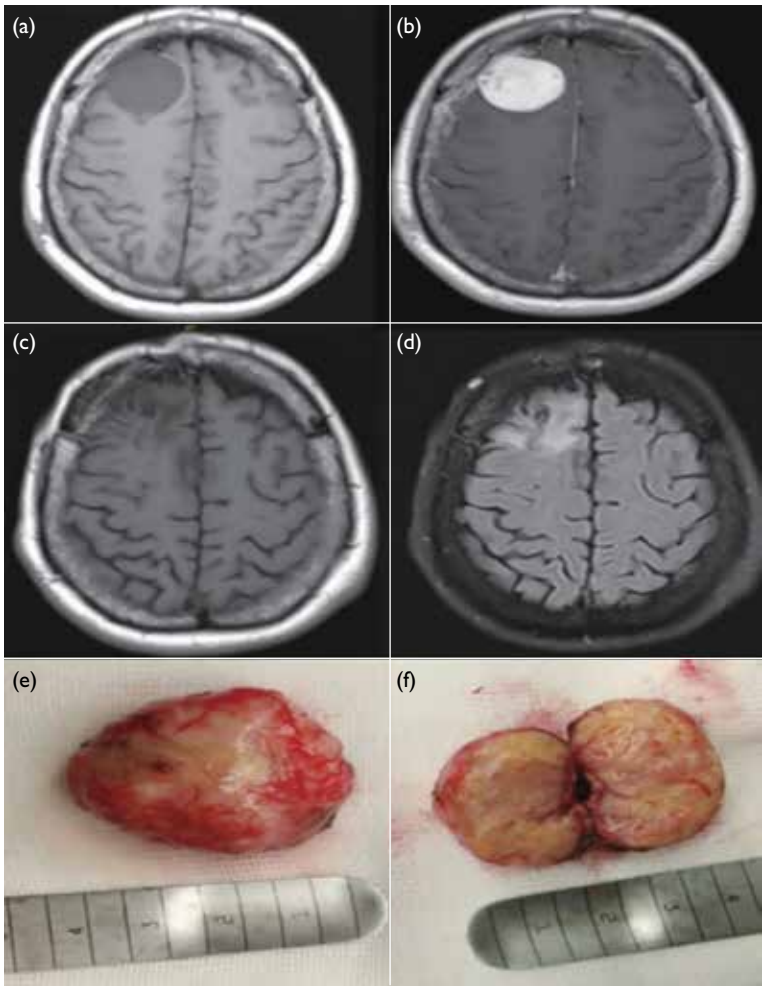

FIG 1. (a, b) Preoperative magnetic resonance imaging (MRI) scan in the transverse position. The space-occupying lesion appears slightly hypointense on T1-weighted imaging, with significant and uniform enhancement on the contrast-enhanced scan. It seems to have a broad base connected to the dura mater. The adjacent brain parenchyma shows mild compression and displacement. (c, d) Postoperative MRI scan: transverse T1-weighted and contrast-enhanced images at 3 years postoperatively. The T1-weighted scan shows mixed, slightly hypointense signals in the surgical area. The contrast-enhanced scan reveals patchy enhancements around the surgical site, likely indicating gliosis. (e, f) Specimen images show the tumour is greyish-yellow in colour with a complete capsule. Numerous tiny blood vessels are distributed across the surface of the capsule. When cut open, lipid droplet-like fluid flows out, and scattered tiny vascular sections are visible within the sectioned tissue

sequences and tumour enhancement characteristics, can help distinguish the two and increase diagnostic accuracy.

Pathological characteristics of hibernomas

Pathological diagnosis is the gold standard for confirming hibernomas. Histological features include small multilocular brown adipocytes that have a rich granular cytoplasm and central or eccentric round or oval nuclei. Unlike the single large lipid droplet in white adipocytes, brown adipocytes contain multiple small lipid droplets and abundant mitochondria, giving the cytoplasm a granular appearance. In addition, hibernomas usually have a rich vascular network. Immunohistochemical staining showing vascular CD34 positivity can further confirm the diagnosis. Hibernoma cells have varying degrees of S-100 protein and CD34 expression according to the literature.[4,5] Combining these pathological features can effectively distinguish hibernomas from other intracranial tumours such as meningiomas.

Conclusion

The diagnostic process of this case of intracranial hibernoma emphasises the importance of clinicians and pathologists when facing uncommon intracranial tumours. Detailed imaging analysis and pathological examination facilitate accurate differential diagnosis, providing the best treatment plan for patients. More case reports and research are needed to further enrich the understanding and treatment strategies of intracranial hibernomas.

Author contributions

All authors contributed to the concept or design, drafting of the manuscript, and critical revision of the manuscript for important intellectual content. All authors had full access to the data, contributed to the study, approved the final version for publication, and took responsibility for its accuracy and integrity.

Conflicts of interest

The authors declared no conflicts of interest.

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Ethics approval

The study was approved by the Ethics Committee of Suzhou TCM Hospital Affiliated to Nanjing University of Chinese Medicine (Ref No. 2024-005). The patient was treated in accordance with the Declaration of Helsinki. The patient provided written consent for publication of this case report.

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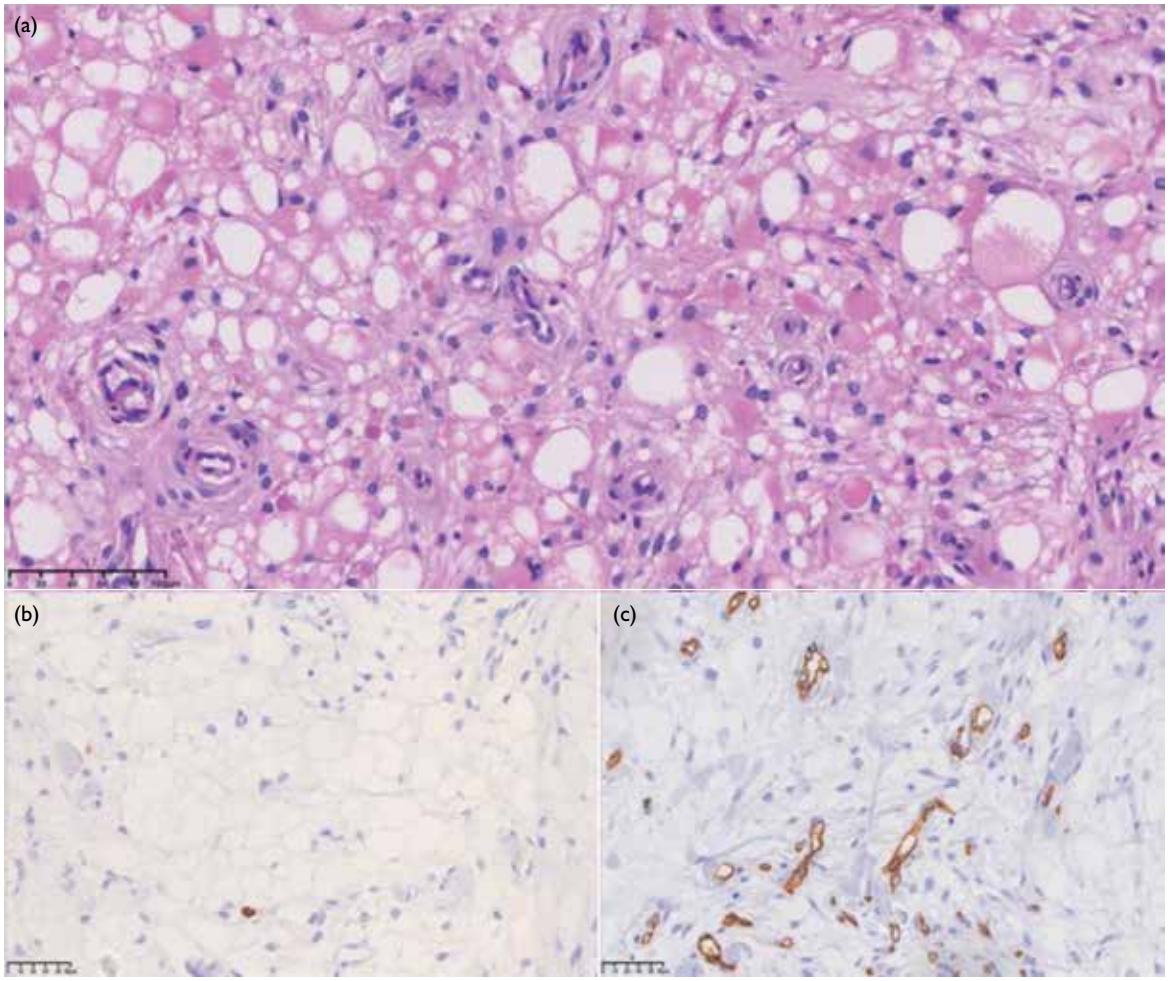

FIG 2. (a) Microscopy following haematoxylin and eosin staining (100 μm) reveals polygonal/round cells with eosinophilic granular cytoplasm, and round nuclei centrally located in the cells under the microscope. Additionally, numerous small vacuolated brown adipocytes and unilocular adipocytes can be seen, along with a considerable amount of blood vessels, spindle cells, and collagen fibres infiltrating around the tumour cells. (b) Immunohistochemistry (50 μm) shows scattered positivity for Ki-67. (c) Immunohistochemistry (50 μm) shows CD34 positivity in the vessels

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