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CLINICAL CASE REPORT

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Rare Parenchyma Meningioma in an Adolescent Female With Cheek Tingling

A Case Report

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Abstract: The following is a report on a rare parenchyma meningioma and the computed tomography (CT) and magnetic resonance imaging (MRI) findings. To our knowledge, this was the first characterization of magnetic resonance spectroscopy (MRS) in a parenchyma meningioma.

Three days after initial presentation, a 14-year-old female student reported feeling tingling in her cheek, grading 3 to 4 points. Two hours later, the tingling had disappeared. The patient was admitted to hospital with stable vital signs and no abnormal presentations upon physical examination. A routine CT scan of the brain showed a quasicircular region of the left occipital lobe was homogenous hyperdense and an arcualia calcification was found on the lesion's margin and the boundary was ill-defined. Further MRI and contrast-enhanced scanning of the brain showed that a lobulated nidus with abnormal signaling was present in the left occipital lobe and was approximately 1.9 × 2.0 cm. Hypointensity on T1-weighted imaging and a slight hyperintensity on T2-weighted imaging was also observed. A short T2 signal appeared on the margin and a few longer T2 edema zones appeared around the nidus, whereas the lesion showed homogenous enhancement. MRS was characterized by a slight or moderate increase of a choline (Cho) peak and a small reduction of the N-acetyl aspartate (NAA) peak. After completing the preoperative preparation, the excision of the supratentorial deep lesions was performed on the patient. The pathology led to a diagnosis of a left occipital lobe meningioma, WHO I. The patient was followed-up for 14 months postoperation, and had no reoccurrences.

Intraparenchymal meningioma rarely occurs in brain parenchyma, and is characterized by lesions with abundant blood supply and requires a glioma to be identified. MRS is a potential tool for preoperative diagnosis of intraparenchymal meningioma.

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Abbreviations: Ala = alanine, Cho = choline, Cr = creatine, CT = computed tomography, DWI = diffusion weighted imaging, Glx = glutamine and glutamate, Lac = lactic acid, Lip = lipid, mI = myo-inositol, MRI = magnetic resonance imaging, MRS = magnetic resonance spectroscopy, NAA = N-acetyl aspartate, T1WI = T1-weighted imaging, T2WI = T2-weighted imaging, TE = time echo.

INTRODUCTION

Meningioma is the second most common intracranial tumor, accounting for approximately 20% of male and 38% of female intracranial tumors.[1] Tumors originate from the meningothelial cells on the arachnoid layer and are typically benign. Meningioma can be grouped into fibrous, syncytial, and transitional tumors.[1] Epidemiological research shows the incidence of meningioma in both males and females increases with advancing age,[1,2] but rarely occurs in juveniles, accounting for only 5% of intracranial tumors in this age group.[1,2] Pediatric meningioma is characterized by a lack of meningeal adhesions, cystic and sarcomatous changes, and high intraventricular incidence with a high prevalence among males.[3] Furthermore, intraventricular meningiomas and parenchyma meningiomas (very rare) may appear if meningeal adhesion is lacking. The following study reports a case of pediatric meningioma occurring in the left occipital lobe, and the clinical and imaging presentations. To our knowledge, this is the first report of MRS findings for parenchyma meningioma.

CONSENT

Written, informed consent was obtained from the patient's father to use the content and imaging material for publication.

CASE REPORT

A 14-year-old female student presented with cheek tingling grading 3 to 4 points, which self-resolved 2 hours later with no radiating pain. The patient was admitted to the people's hospital for treatment and a routine magnetic resonance imaging (MRI) scan of the brain suggested a nidus in the left occipital lobe, which was initially thought to be a glioma. The patient was then admitted to the First Affiliated Hospital, College of Medicine, Zhejiang University, Zhejiang, China, for further treatment. When the patient was admitted, she had no obvious discomfort, stable vital signs, and her physical examination was normal. A routine computed tomography (CT) scan of the brain revealed a quasicircular mass in the left occipital lobe with a homogenous hyperdense. Arcualia calcification was also found on the lesion's margin and the boundary was ill-defined (Figure 1). Moreover, a routine MRI scan and diffusion-weighted imaging (DWI) sequence suggested a lobulated nidus with an abnormal signal in the left occipital lobe approximately 1.9 × 2.0 cm in size. A slight hypointensity was observed on T1-weighted images (T1WI) and a slight hyperintensity on T2-weighted images (T2WI). A short T2 signal appeared on the margin and, a few long T2 edema zones appeared around the nidus, with a slight hypointensity on the DWI sequence. The

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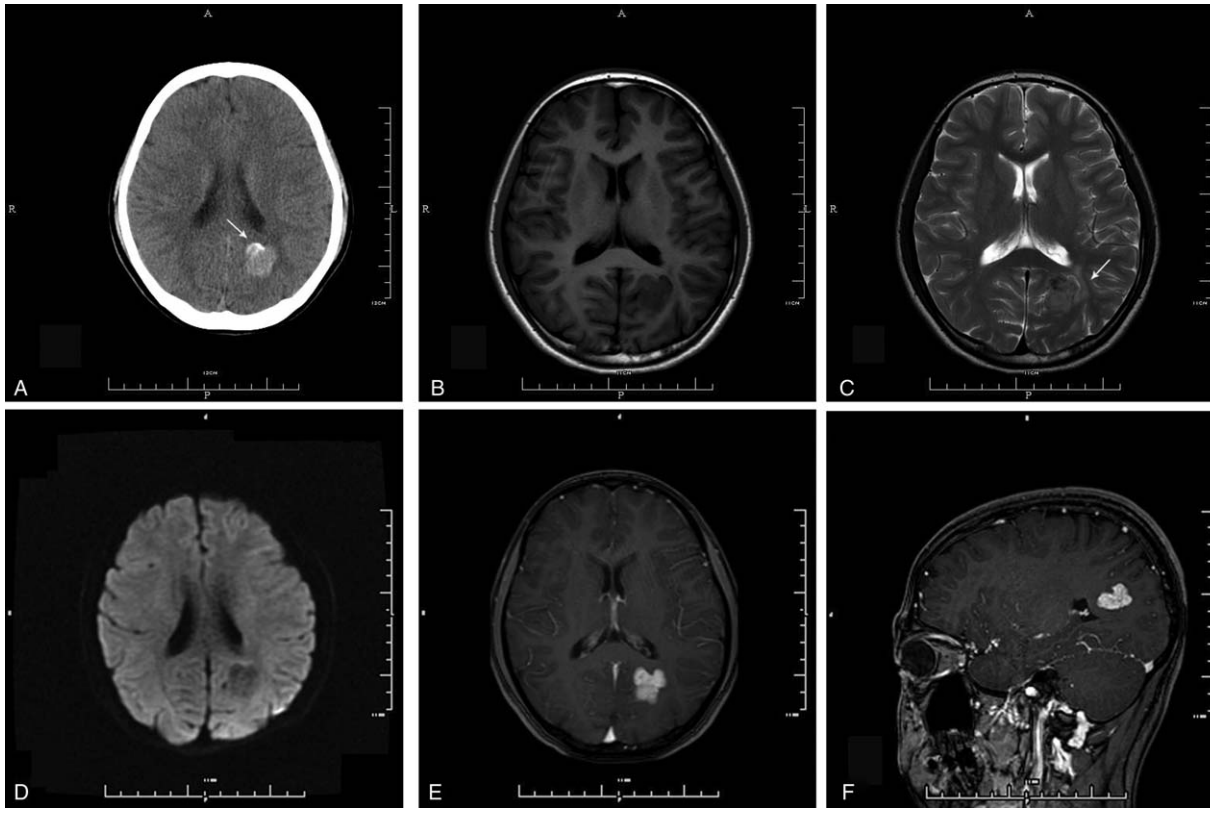

FIGURE 1. A routine CT scan of the brain showed the left occipital lobe had a hyperdense mass, and arcualia calcification was found on the lesion's margin (A, arrow). A routine and contrast-enhanced MRI scan of the brain showed a lobulated nidus with an abnormal signal occurring in the left occipital lobe and slight hypointensity in T1WI (B) and slight hyperintensity in T2WI; a short T2 signal appeared on the margin, with some longer T2 edema zones with a stripe shape appearing around the nidus (C, arrow), and slight hypointensity in the DWI sequence (D). The lesion was homogenous with significant enhancement (E, F). CT = computed tomography, DWI = diffusion weighted imaging, MRI = magnetic resonance imaging, T1WI = T1-weighted imaging, T2WI = T2-weighted imaging.

contrast-enhanced scan of the lesion found significant homogenous enhancement (Figure 1).

Further characterization of the lesion through magnetic resonance spectroscopy (MRS) indicated the agglomerate nidus had abnormal signaling occurring in the left occipital lobe. Short time echo (TE) spectrum suggested a small reduction of the *N*-acetyl aspartate (NAA) peak and a slight or moderate increase of the choline (Cho) peak; the values, including Creatine (Cr), were as follows: Cho/Cr = 1.19, NAA/Cr = 2.09, no increase of myo-inositol (mI) peak, and mI/Cr = 0.53. Lactic acid (Lac) or lipid (Lip) peaks in 0.9 to 1.4 ppm could be found. The long TE suggested an increase of the Cho peak, Cho/Cr = 1.60, NAA/Cr = 2.28. The short TE, glutamine, and glutamate (Glx) peak did not increase, but the alanine (Ala) peak did increase (Figure 2). Multivoxel proton MRS including the nidus and normal structure area suggested the Cho peak in the central zone was 1.5 to 2.1 times greater than normal; no obvious increase of the Cho peak occurred near the edema area, and pseudocolor images showed a specific value of 1.3. Pseudocolor images also showed a slight increase of the Cho peak in the nidus area and a relevant area of another side (Figure 3). The abundant blood supply to the lesion in the left occipital lobe and MRS was characterized by a slight or moderate increase in the Cho peak and small reduction of the NAA peak. The imaging diagnosis did not exclude the possibility of a benign neoplastic lesion. The following structures required further identification: outer ventricle ependymoma; ganglioneuroma; and part of the neurogliocyte mixed tumors derived from oligodendroglioma.

After completing the preoperative preparation, the excision of the supratentorial deep lesions was performed. The tumor was found on a deep surface of the left occipital lobe and had a hard texture, abundant blood supply, and there was a boundary with the brain tissue. An intraoperative frozen-section showed the tumor was rich in spindle cell tumors with psammoma bodies. In pathologic histology, the tumor cell appeared as a long fusiform, some were also observed as sarciniform and swirling, with more disseminated intravascular coagulation in a thick wall, and the potential presence of collagen and psammoma bodies. Immunohistochemistry included: GFAP(−), S-100(−), epithelial membrane antigen (partial+), progesterone receptor (small nidus+), Ki-67 (<5%,+), E-cadherin(−), β-Catenin(+), CD34(−), SMA(−), and Desmin(−) (Figure 4). The pathological findings led to the diagnosis of a left occipital lobe meningioma, WHO I. The patient was followed-up for 14 months after the operation, with no reoccurrence.

DISCUSSION

At present, the origin of parenchyma meningiomas is controversial. Some research suggest parenchyma meningiomas originate from arachnoid cells near the pia mater, and travel

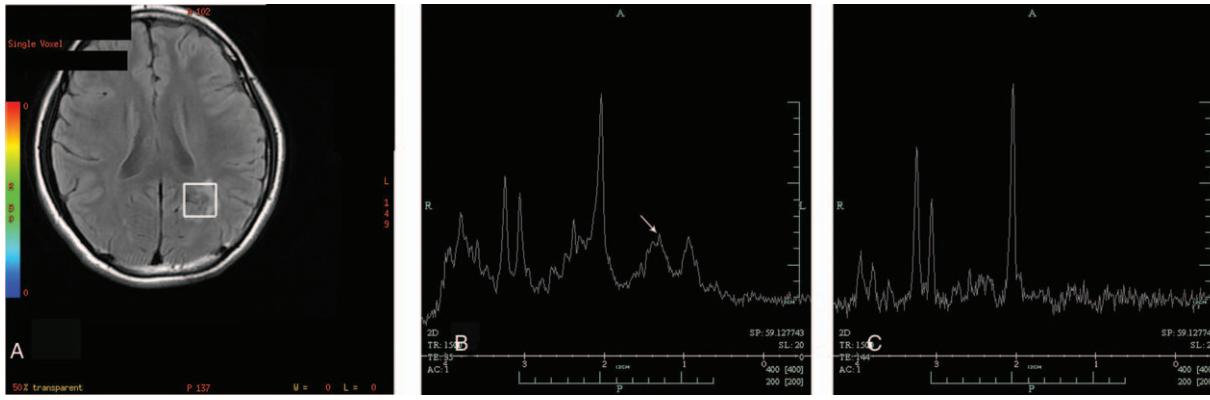

FIGURE 2. MRS of parenchyma meningioma single-voxel (A–C). No obvious reduction of the NAA peak, but a slight increase in the Cho peak in the complete lesion. The high alanine (Ala) peak was to the right of the NAA (B arrow). MRS = magnetic resonance spectroscopy, NAA = *N*-acetyl aspartate.

deep into the brain along blood vessels.[4] Conversely, it has been suggested that they originate from arachnoid cells that stop at the transport process.[5] The morbidity of parenchyma meningioma is unknown and is primarily reported by East Asians. The age of onset ranges from 5 months to 60 years with an average age of onset of 21 years.[4] Incidence of parenchyma meningioma is higher in men with the male to female ratio being 3:2.[4] Approximately 90% of cases occur in the frontal supratentorium.[4] The most common symptom of parenchyma meningioma is seizures. Other symptoms include: motor weakness,

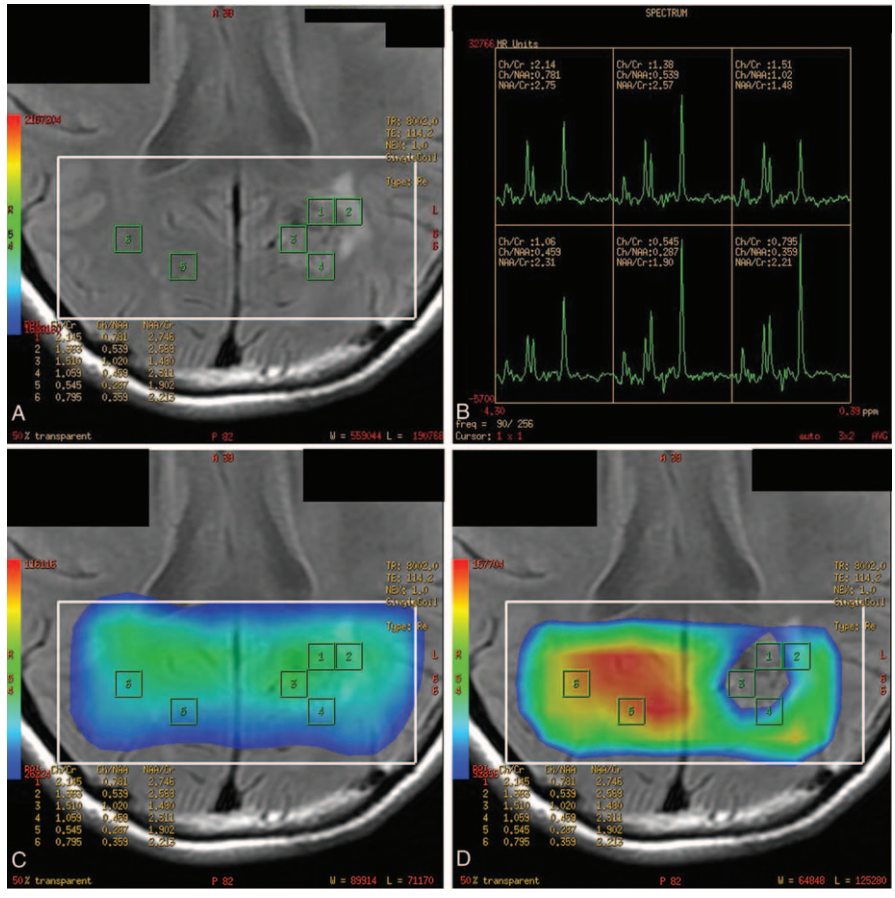

FIGURE 3. MRS of the parenchyma meningioma multivoxel (A–D): in the central zone of the nidus (1.3) the Cho peak is slightly increased, NAA peak is slightly reduced, and the specific values are 1.5 to 2.1, with no obvious increase of the Cho peak near the edema area (2), the specific value reaches 1.3; pseudocolor images (C, D) Slight increase of the Cho concentration in the nidus area and normal brain tissue with the NAA concentration slightly reduced. MRS = magnetic resonance spectroscopy, NAA = *N*-acetyl aspartate.

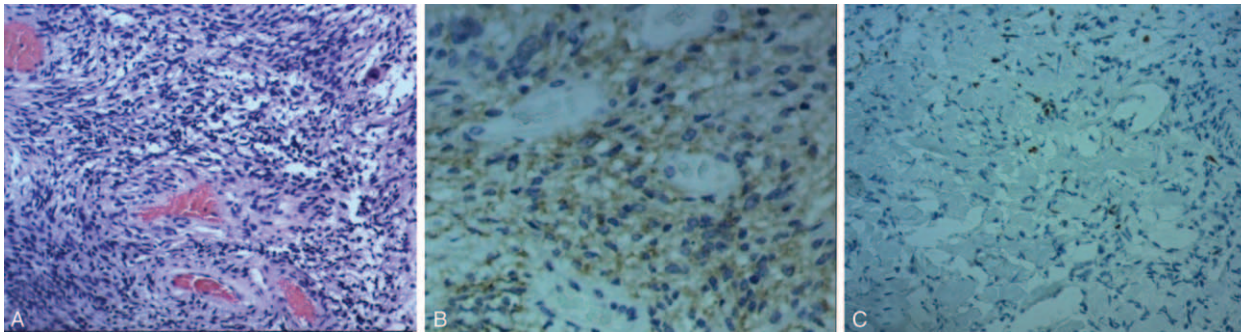

FIGURE 4. In pathologic histology, a tumor cell is fusiform under the microscope; it is shaped as a sarciniform and swirling, with disseminated intravascular coagulation, collagen and psammoma bodies can also be seen (H&E staining, ×100) (A). Some of the tumor cells have positive EMA staining (×400) (B). A small number of the tumor cells are PR-positive (×100) (C).

headache, motor disorder, macrocephaly, and hemiparesis.[4,6] In this case, the parenchyma meningioma occurred in the supratentorium of the occipital lobe in a young female patient; this has only been reported once before.[7] The manifestation of the parenchyma meningioma as tingling in the cheek is completely different from clinical features that have previously been reported. The most common pathological pattern is fibrous, and the second is psammomatous, which accounts for 30% and 13% of pathological patterns, respectively. Other pathological patterns include: anaplastic, fibroblastic, meningothelial, transitional, sarcomatous, clear cell, chordoid, atypical, rhabdoid, papillary WHO III.[4,8,9] The pathological and differential diagnosis are chordoma, extraskeletal myxoid chondrosarcoma, low-grade chondrosarcoma, myxopapillary ependymoma, and chordoid glioma.[10,11]

Typically parenchyma meningiomas are round, quasicircular, or wedge-shaped, and are lobulated with clear boundaries.[4–18] On routine CT scans, parenchyma meningiomas are primarily hyperdense and on rare occasion can be isodense.[4–18] Furthermore, punctate calcification or overall tumor calcification can be found, but typically there is no mass effect or peritumoral edema.[4–6,8,12,13,15] In contrast-enhanced CT scanning, lesions of the parenchyma meningioma are significantly enhanced, and can present with homogeneous enhancement or no enhancement.[5,6,12,13,15] When using MRI, parenchyma meningiomas appear as a solid mass and hypointensity or equal signal strength is observed in T1WI lesions. In T2WI lesions, hypointensity, hyperintensity, or high-low mixed signals relative to the brain alba signal can be observed. Signals may be uneven, with no mass effect or edema zone of the tumor.[4–6,8,12–14,17] However, the edema zone can be found in some parenchyma meningiomas.[4,6,8,9,11,16,17] In contrast-enhanced MRI scanning, significant homogeneous or heterogeneous enhancement of the tumor can be observed; however, some tumors do not demonstrate enhancement.[4–9,11,13,14,16,17] Parenchyma meningiomas can be characterized by a mixed solid cystic mass and edema zone, with significant enhancement of the solid component.[9,10,16,18] In contrast, some cases of cystic and solid meningiomas have demonstrated significant enhancement of the solid component and others minimal enhancement.[7,15] Before surgery, the parenchyma meningioma could be misdiagnosed as a glioma, as it is similar to metastatic tumors if peritumoral edema is present.[5,11,13,16] In this case, the routine CT and MRI scans and contrast-enhanced imaging findings indicated abundant blood supply to the lesions and mass effect, which is similar to past parenchyma meningioma image reports that fail to determine the nature of the lesion. To our knowledge, this is the first report of MRS findings of a parenchyma meningioma, which included a slight or moderate increase of the Cho peak, a small reduction of the NAA peak, a slight increase of the Glx peak, and a high Ala peak. This is different from gliomas that have obvious reductions of the NAA peak and an obvious increase of the Cho peak; therefore, a metastatic tumor was ruled out. Conventional imaging findings of brain parenchyma meningioma are similar to those of extracerebral meningioma; however, the MRS of these has significant differences, beyond positioning. In this study, the MRS contained a high Ala peak similar to those of extracerebral meningioma; however, the peak of NAA was present, which is different from extracerebral meningioma, which lacks a NAA peak. Further investigation into the presence of the NAA peak is needed.

Parenchyma meningioma is typically removed surgically or followed by chemoradiotherapy.[4]The complete removal of the tumor typically results in no relapses and has an excellent prognosis.[4] If a relapse occurs, prognosis is poor.[11]

CONCLUSIONS

Parenchyma meningioma is characterized by a young age of onset (approximately 21 years) and patients typically present with seizures. Imaging indicates abundant blood supply to lesions lacking mass effect, with hyperdense via CT, hypointensity of T1WI and T2WI, and occasionally calcification or cystic lesions. MRS of parenchyma meningioma showed that the Cho peak and the Ala peak are increased, but the NAA peak has no obvious reduction. This observation could contribute to identifying it from other brain tumors.

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