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Incidental pediatric intraparenchymal meningioma: illustrative case

Adriano M. Lino-Filho, MD,[1] Bárbara A. Morais, MD,[1,2] Mateus Neves Faria Fernandes,[3] Jairo Porfirio Oliveira-Junior, MD,[1] Bruna Estrozi, MD, PhD,[4] Weder Silva Borges-Junior,[3] and Paulo Ronaldo J. Ribeiro, MD, MSc[1,2]

[1]Division of Neurosurgery, Department of Surgery, Clinics Hospital, Federal University of Goiás, Goiânia, Goiás, Brazil; [2]Department of Neurological Surgery, Children's Hospital, Goiânia, Goiás, Brazil; [3]School of Medicine, Federal University of Goiás, Goiânia, Goiás, Brazil; and [4]Department of Pathology, Clinics Hospital, Federal University of Goiás, Goiânia, Goiás, Brazil

BACKGROUND Meningiomas are the most common benign tumors among CNS neoplasms. In the pediatric population, however, they account for only 0.4%–4.6% of all intracranial neoplasms; they are rare inside the brain parenchyma and are frequently confused with other entities, such as glioneuronal tumors and cavernomas, among others.

OBSERVATIONS The authors describe the case of a 4-year-old male who presented to the emergency department for evaluation of periorbital cellulitis and was incidentally diagnosed with a brain tumor. MRI demonstrated an expansive heterogeneous lesion, 2.2 × 1.9 × 1.8 cm, in the left lingual gyrus. Spectroscopy and perfusion imaging suggested a low-grade glioneuronal tumor. After thorough discussion, the family and medical team elected to pursue surgical treatment. The patient had an uneventful postoperative recovery, and subsequent pathological and immunohistochemical analysis confirmed the diagnosis of a fibrous meningioma (WHO grade 1).

LESSONS Intraparenchymal meningiomas are a rare and misdiagnosed tumor, especially in the pediatric age group, and therefore are not usually considered in the differential diagnosis of intra-axial neoplasms in children. When suspected, surgery may be encouraged due to the tendency of these tumors to exhibit more aggressive behavior compared with adult meningiomas.

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KEYWORDS intraparenchymal meningioma; intra-axial meningioma; pediatric tumor; oncology; case report

Meningiomas are the most prevalent benign CNS tumor in adults, accounting for 35% of all brain tumors, with multiple studies reporting an increasing incidence in the general population.[1] However, in the pediatric population they are rare, accounting for only 0.4%–4.6% of all intracranial neoplasms in this age group.[2]

A typical meningioma has several imaging features that allow an accurate diagnosis; they usually consist of a clearly defined, extra-axial, uniformly enhancing mass with dura mater attachments, and in many cases they present with the dural tail sign.[3,4] In the pediatric age group, they tend to occur in atypical locations, including the intraventricular and infratentorial regions.[5] When located within the brain parenchyma, they are frequently misdiagnosed as cavernous malformations, gliomas, metastases, and other tumors.[6]

The literature on intraparenchymal meningiomas—defined as lesions entirely surrounded by brain parenchyma without dural attachment—in children and adolescents is extremely scarce, with only a few published reports and case series available. We report the case of an intra-axial meningioma in a 4-year-old child with clinical, radiological, and histopathological features. Moreover, because of their sporadic nature and limited documentation, we undertook a review of the literature on intraparenchymal meningiomas restricted to the pediatric population.

Illustrative Case

We describe the case of a 4-year-old male who presented to the emergency department with left eyelid swelling and edema, diagnosed as periorbital cellulitis. He did not have any medical conditions (including phakomatoses or other hereditary diseases) or a history of radiation exposure and before this had otherwise been healthy. On physical examination, he had no neurological deficits or skin lesions. CT of the head was performed to evaluate the extent of the infection and, in addition to confirming the localized cellulitis, revealed an incidental finding of a left occipital and parasagittal well-defined mass with surrounding vasogenic edema and calcifications (Fig. 1A). Further MRI demonstrated an expansive heterogeneous lesion, 2.2 × 1.9 × 1.8 cm, without a dural tail (Fig. 1B), located in the left lingual gyrus. Spectroscopy and perfusion imaging suggested a low-grade glioneuronal tumor.

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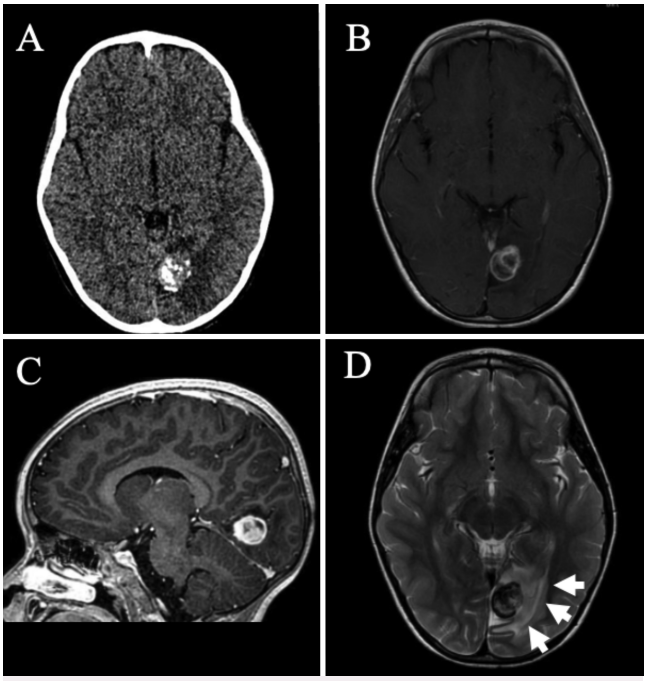

FIG. 1. Preoperative imaging. **A:** Axial CT scan without contrast demonstrating calcified left occipital and parasagittal lesion. **B:** Axial T1-weighted MR image with contrast demonstrating a tumor with heterogeneous enhancement by gadolinium. **C:** Sagittal T1-weighted MR image with contrast showing the lesion in the lingual gyrus without the dural tail sign. **D:** Axial T2-weighted MR image demonstrating the surrounding edema (*arrows*).

Treatment and Follow-Up

After resolution of the periorbital cellulitis, the patient was transferred to our service for treatment. Observation was considered given the lack of tumor-related symptoms; however, after thorough discussion, the family and medical team opted for surgical treatment to better understand the nature of the lesion. Dexamethasone was given perioperatively to decrease brain edema. A horseshoe-shaped incision was performed on the left occipital parasagittal region, followed by a left occipital craniotomy. The dura was incised in a C-shaped fashion. As expected, there were no dural attachments, and the overlying cortex was grossly abnormal. Using a microsurgical technique, we resected the tumor en bloc, revealing a rubbery and fibrous mass. Postoperatively, the patient recovered well with no neurological deficits. Postoperative MRI showed no evidence of residual tumor. He was discharged on the 3rd postoperative day.

Pathology

Pathology and immunohistochemistry confirmed a fibrous meningioma, a WHO grade 1 tumor (Fig. 2). Genetic analysis was requested but was not available in the public health system.

Follow-Up

At follow-up, the patient had a normal neurological examination and no symptoms. MRI after 3 months revealed no evidence of residual tumor (Fig. 3). The patient remains in outpatient follow-up.

Informed Consent

The necessary informed consent was obtained in this study.

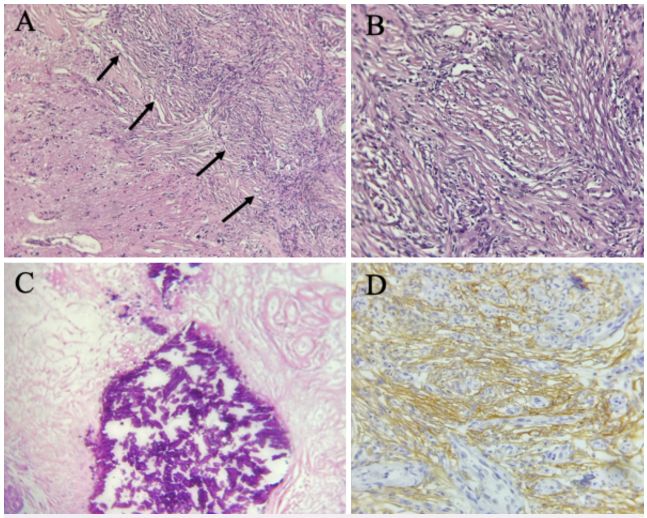

FIG. 2. A and B: Histological sections reveal well-defined neoplasm, not encapsulated, characterized by the proliferation of spindle cells, with elongated nuclei, inconspicuous nucleoli, and indistinct cytoplasm, permeated by a large amount of collagen, sometimes thick. The *arrows* show the interface between the meningioma and brain parenchyma. **C:** Presence of psammomatous and other dystrophic pattern calcifications. Mitosis figures were not detected. **D:** An immunohistochemical study reveals epithelial membrane antigen expression. Original magnification ×40.

Discussion

In our review, after screening three different databases (PubMed, Embase, and LILACS), only 33 documented cases (Table 1) were identified (including the present report) in patients younger than 19 years of age, with insufficient details available for the majority of these cases. Meningiomas are, by definition, extra-axial tumors. The prevailing hypothesis is that intraparenchymal meningiomas originate from cap cells situated in the pia mater. These cap cells are believed to infiltrate the brain or sulci with the penetrating blood vessels during the developmental process.[7]

In our analysis, patients had a mean age of 9 ± 5.6 years at the time of diagnosis. Males were more affected than females, accounting

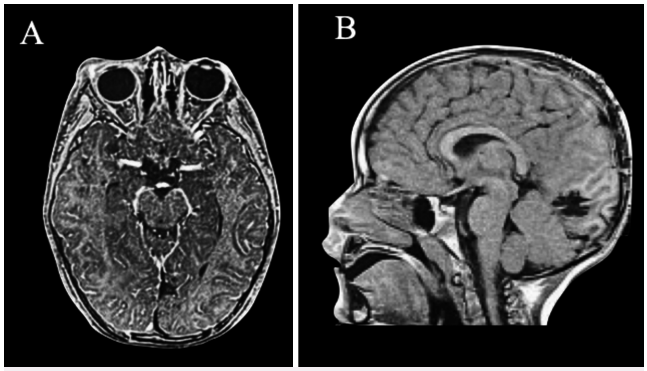

FIG. 3. Postoperative MR images. **A:** Axial T1-weighted postgadolinium image. **B:** Sagittal T2-weighted FLAIR image revealing no residual tumor.

TABLE 1. List of childhood intraparenchymal meningiomas described in the literature

Authors & Year	Age, yrs	Sex	Clinical Presentation	Localization	Histology	WHO Grade	Resection	Adjuvant Therapy	FU, mos	Outcome	Differential Diagnosis
Dong et al., 1980[9]	0.3	M	Macrocephaly	Temporal	Fibromatous	1	GTR	NA	NA	Death	NA
Drake et al., 1985[12]								NA	72		NA
Case 11	12	M	Seizure	Temporal	Transitional	1	STR	No	36	NR	NA
Case 13	3	M	Raised ICP	Sylvian fissure	Malignant	3	GTR	No	5	NR	NA
Legius et al., 1985[13]	1.16	M	Seizure	Parietal	Fibromatous	1	NA	NA	12	NR	NA
Sakaki et al., 1987[14]	1	M	Seizure	Frontal	Fibromatous	1	GTR	NA	5	NR	NA
Schroeder et al., 1987[15]	7	M	Seizure	Frontal	Fibromatous	1	NA	NA	NA	NA	Oligodendroglioma, postinflammatory granuloma
Mamourian et al., 1991[16]	2	F	Nausea/vomiting	Frontal	Fibromatous	1	GTR	NA	0.5	NR	Oligodendroglioma/teratoma
Schut et al., 1994[17]	5	M	Focal deficits	Temporal	Fibromatous	1	GTR	NA	1	NR	Ganglioglioma/oligodendroglioma
Kohama et al., 1996[18]	1.8	F	Seizure	Frontal	Fibromatous	1	GTR	NA	24	NR	Glial tumor
Teo et al., 1998[19]	2	F	Focal deficits	Brainstem	Clear cell	2	STR	NA	NA	NR	NA
Karadereler et al., 2004[20]	14	M	Seizure	Temporal	Mixed/no definition	NA	GTR	NA	36	NR	NA
Zhang et al., 2007[21]	16	M	Seizure	Frontal	Atypical	1	GTR	NA	NA	NR	NA
Shimbo et al., 2011[22]	10	M	Seizure	Frontal	Chordoid	1	GTR	NA	5	NR	NA
Jung et al., 2012[23]	1.6	M	Seizure	Frontoparietal	Transitional	1	GTR	NA	9	NR	Pilocytic astrocytoma/dysembryoplastic neuroepithelial tumor
Pinto et al., 2012[24]	17	F	Seizure	Temporal	NA	1	NA	NA	NA	NA	NA
Werbrouck et al., 2014[25]	13	M	Seizure	Temporal	Fibromatous	1	NA	NA	NA	NA	Ganglioglioma/gangliocytoma
Jia et al., 2014[26]	11	F	Headache, nausea/ vomiting	Frontal	Rhabdoid	3	GTR	Yes	NA	NA	Primitive euroectodermal tumor
Nayil et al., 2015[27]	3	M	Seizure, nausea/ vomiting	Frontal	Anaplastic	3	GTR	NA	6	NR	Neuroectodermal tumor
Dash et al., 2016[28]	16	M	Seizure	Frontal	Transitional	1	GTR	No	3	NA	NA
Reynolds et al., 2016[29]	15	M	Focal deficits	Basal ganglia	Chordoid	1	STR	Yes	3	NR	
Liang et al., 2016[30]	14	F	Tingling	Occipital	Mixed/no definition	1	NA	NA	14	NA	Ependymoma/ganglioneuroma/ oligodendroglioma
Donovan & Thavapalan, 2016[31]											
Case 1	11	M	Nausea/vomiting, seizure	Sylvian fissure	Mixed/no definition	1	GTR	No	120	NR	NA
Case 2	7	M	Seizure, focal deficits	Sylvian fissure	Fibromatous	1	STR	Yes	84	Recurrence	NA
Larrew & Eskandari, 2016[32]	14	F	Seizure	Frontal	Mixed/no definition	1	GTR	NA	3	NR	Oligodendroglioma/mixed oligoastrocytoma/ependymoma
Huntoon et al., 2017[3]									NA		NA
Case 7	11	M	NA	NA	Chordoid	2	NA	No	NA	NA	NA
Case 9	12	M	NA	NA	Transitional	2	NA	No	NA	NA	NA

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Authors & Year	Age, yrs	Sex	Clinical Presentation	Localization	Histology	WHO Grade	Resection	Adjuvant Therapy	FU, mos	Outcome	Differential Diagnosis
Case 12	18	M	NA	NA	Chordoid	2	NA	No	NA	NA	NA
Case 13	16	F	NA	NA	Anaplastic	3	NA	Yes	NA	NA	NA
Liu et al., 2018[33]	8	M	Headache, nausea/vomiting	Basal ganglia	Atypical	NA	STR	NA	3	NR	NA
Tan & Tan, 2018[34]	5	M	Seizure, headache	Temporal	Fibromatous	1	STR	No	3	Recurrence	NA
Vescovi et al., 2018[35]	7	M	Blurred vision/focal deficit	Parietal	Meningothelial	2	GTR	NA	4	NR	Astrocytoma/ependymoma/ependymoblastoma
Guo et al., 2021[36]	12	F	Seizure	Temporal	Fibromatous	1	GTR	NA	6	NR	NA
Present case	4	F	No symptoms	Occipital	Fibromatous	1	GTR	No	3	NR	NA

GTR = gross-total resection; ICP = intracranial pressure; NA = not available; NR = no recurrence; STR = subtotal resection.

for 69.7% and 30.3%, respectively. Epileptic seizure is the most common symptom (54.5%), followed by nausea/vomiting (15.2%) and focal deficit (15.2%).

The most reported histological type was fibromatous meningioma, corresponding to more than one-third of the cases (33%). Two-thirds (66%) were WHO grade 1, 15.2% were grade 2, and 12.1% were grade 3.

Observations

The 2021 WHO classification of tumors of the CNS classifies meningiomas as grade 1, 2, or 3, with a broad spectrum of 15 histological subtypes: 9 benign (grade 1), 3 intermediate-grade (grade 2), and 3 malignant (grade 3) variants,[8] with approximately 93% corresponding to grade 1, 4.6% to grade 2, and 2.4% to grade 3 in the general population.[9] In our analysis, grade 2 and 3 meningiomas were more prevalent than expected (27.3%), representing a poor prognosis of the disease in the pediatric age group.

An interesting finding is the predominance of male patients affected in this population (male/female ratio: 2.3/1). In adults, meningiomas are much more prevalent in women (60%–80%).[7] It is known that a significant number of these tumors exhibit hormonal receptors for various hormones, notably progesterone, which could potentially influence tumor growth and explain the disparities noted before the onset of puberty, but the exact pathophysiology remains unknown.[10,11]

Childhood meningiomas tend to arise in atypical locations.[10] In our review, the lesions were more located on the lobes and central core. Some series found headache to be the predominant symptom,[10] probably due to intracranial hypertension. In our analysis, however, the most common clinical presentation was epileptic seizures, followed by nausea/vomiting and focal deficits, with symptoms varying by location of the tumor and the child's age. We hypothesize that headache may not have prompted further investigation until the child presented with other symptoms, for which they were referred for brain imaging. To our knowledge, this is the first reported case in which no symptoms were directly related to the tumor itself, as it was discovered incidentally.

Lessons

Intraparenchymal meningiomas are a rare and misdiagnosed tumor, especially in the pediatric age group, and are not usually considered in the differential diagnosis of intra-axial neoplasms in children. This study highlighted differences in the tumor in the pediatric age group when compared with adults: in children, the tumor has a predilection for boys and is more frequently a grade 2 or 3 meningioma. When suspected, surgery may be encouraged, due to the tendency of these tumors to exhibit more aggressive behavior. Our study has some limitations. Genetic evaluation of the tumor was not feasible because of constraints within the public health system. Additionally, the heterogeneity of information collected from case reports may have impacted our results. Additional investigation is necessary to unravel the molecular intricacies and signaling pathways implicated in the migration of cells and the establishment of meningiomas within intraparenchymal sites.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Lino-Filho, Morais, Estrozi, Borges-Junior, Ribeiro. Acquisition of data: Lino-Filho, Morais, Fernandes, Estrozi, Borges-Junior, Ribeiro. Analysis and interpretation of data: Lino-Filho, Estrozi. Drafting the article: Lino-Filho, Morais, Fernandes, Oliveira-Junior, Borges-Junior. Critically revising the article: Lino-Filho, Morais, Fernandes, Oliveira-Junior, Ribeiro. Reviewed submitted version of manuscript: Lino-Filho, Fernandes. Approved the final version of the manuscript on behalf of all authors: Lino-Filho. Statistical analysis: Lino-Filho. Administrative/technical/material support: Lino-Filho. Study supervision: Morais, Ribeiro.

Correspondence

Adriano M. Lino-Filho: Medical School, Federal University of Goiás, Goiânia, Goiás, Brazil. adrianomlf.filho@gmail.com.