Case Report

High-grade astroblastoma in a child: Report of one case and review of literature

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Abstract

Background: Astroblastoma is a rare glial neoplastic lesion that affects children and adolescents; its histogenesis remains uncertain. It is considered to account for 0.5% of all glial neoplasms, and two different subtypes have been defined based upon histologic characteristics.

Case Description: We present the case of a 9-year-old girl who presented with headache, motor symptoms, and seizures a few days before she was admitted to our institution. Computed tomography (CT) and magnetic resonance imaging (MRI) scans showed an intra-axial heterogeneous frontoparietal lesion with a striking “bubbly” appearance in MRI T2-weighted sequences and features of intracranial hypertension. Gross total resection of the tumor was achieved and the histopathologic diagnosis revealed high-grade astroblastoma. We reviewed the current published cases of astroblastoma to highlight the demographic, clinical, radiologic, and pathologic data.

Conclusion: Astroblastomas are a distinct clinicopathologic entity, with well-described radiologic, pathologic, and cytogenetic features. Its recurrence is high and efforts must be made to elucidate the role and usefulness of radiotherapy and chemotherapy in these tumors.

Key Words: Astroblastoma, glial tumors, pediatric tumors, supratentorial tumor

INTRODUCTION

Astroblastoma is a rare glial tumor affecting children and adolescents whose histogenesis remains uncertain. It accounts for 0.5% of all glial neoplasms. Two different subtypes of astroblastoma have been defined based upon histologic characteristics. These tumors are usually located in the cerebral hemispheres, but have also been described in the cerebellum, brainstem, corpus callosum, hypothalamus, and the ventricular system. Their neuroradiological appearance is characteristic; typically they present as supratentorial multilobulated lesions with both solid and cystic components. Here, we present the clinical features of a child treated at our institution with a supratentorial case of astroblastoma.

CASE REPORT

History and presentation

We present the history of a 9-year-old girl who presented with headache of progressive severity with nausea
and vomiting for 20 days, weeklong hemiparesis, and tonic-clonic seizures 24 h prior to presentation in the emergency room. She had an unremarkable medical history. On medical examination, she was found to have motor aphasia, dysnomia, right hemiparesis and right-side hemihypoesthesia, and right corticospinal signs. Complete blood count, serum biochemistry, and urine analysis were within normal limits.

**Neuroimaging findings**

A computed tomography (CT) scan without contrast was performed which revealed a supratentorial, multilobulated left frontoparietal lesion, relatively well demarcated, with a solid and cystic appearance, with mixed densities within the cysts. The solid portion of the tumor was hyperdense to white matter and minimal peritumoral edema was seen. There was midline shift observed and collapse of the ipsilateral ventricular system as well [Figure 1].

Magnetic resonance imaging (MRI) findings revealed a neoplastic well-demarcated intra-axial lesion whose size was 6.1 × 5.6 cm. On T1-weighted images it was heterogeneous, but predominantly isointense to white matter [Figure 2]. The T2-weighted images revealed a multicystic hyperintense lesion with a striking “bubbly” heterogeneous pattern inside the tumor [Figure 3]. Fluid attenuated inversion recovery (FLAIR) sequences demonstrated isointensity within the lesion, but minimal peritumoral edema. Contrast images revealed a mixed solid and peripheral rim enhancement [Figure 2]. Spectroscopy showed a high choline peak.

**Surgery and adjuvant treatment**

The tumor was surgically resected. A left frontoparietotemporal craniectomy was performed and the tumor excised with ultrasonic aspirator. A trans-sulcal approach through the left intraparietal sulcus was used. The intraoperative finding was a solid, soft, and white-pearly lesion with hemorrhagic areas and a visible arachnoid plane with cerebral edema. The lesion was resected completely. The patient’s postoperative course was uneventful. There was improvement of the hemiparesis and language disturbances by 1 month following surgery.

**Histopathology**

Pathologic examination of the tumor was consistent with astroblastoma. Light microscopy demonstrated a papillary neoplasm composed of mildly pleomorphic cells with evident nucleoli, pleomorphic nuclei, and atypical mitoses. A striking perivascular array of pseudorosettes was found [Figure 4]. The nuclei were generally round to oval in shape. Prominent endothelial hyperplasia and hyalinized vessel walls were found in this hypervascularized tumor. The tumor cells exhibited weak and focal staining for epithelial membrane antigen (EMA) and diffuse staining for vimentin throughout the tissue section. Gliob fibrillary acidic protein (GFAP) was also diffusely positive in the epithelioid cells and was mostly marked in the perivascular areas. Analysis of Ki-67 immunoreactivity within the tumor showed a Ki-67 labeling index of approximately (or up to) 40%.

**DISCUSSION**

Astroblastomas still remain as rare and controversial tumors with variable clinical outcomes and unknown cellular origin. Bailey and Cushing, who defined it as a separate type of glioma, initially used the term “astroblastoma” in 1924. They characterized the entity as a unique type of astrocytic glioma with glial fibrillary acidic protein (GFAP)-positive reactive cells and the histologic feature of perivascular pseudorosettes. In 1930, Bucy and Bailey described different tumoral macro- and microscopic features in the first large series published (25 patients), highlighting the individual features of astroblasts (unipolar cells with broad “feet” adjacent to the blood vessels). In 1933 and 1937, Cox classified astroblastomas as a transitional entity between astrocytoma and glioblastoma multiforme.
There is still some controversy in terms of tumor classification, histogenesis, diagnosis, and therapeutics for astroblastoma. According to the last World Health Organization (WHO) classification of tumors of central nervous system, 0.45-2.8% of neuroglial tumors corresponds to astroblastomas. Navarro et al. described in their series a prevalence of 0.92% of all glial tumors.

Although Salvati et al. described a mean age of 36 years, this entity appears to affect young adults and children. We found a mean age of 18.5 years in our review of 28 different series and case reports. We found a striking predominance of female patients (71.15%) in our review. Classically, a predominance of supratentorial tumors has been described, with the cerebral hemispheres being the main location; however, infiltration to corpus callosum, cerebellum, pineal gland, and brain stem, and intraventricular tumors have been previously described. In concordance with this information, lobar tumors, specifically frontal and parietal areas, were the most important locations found in 44.2 and 26.9% cases, respectively, in our review. The third most important location was the temporal lobe in 8.6% cases.

The cardinal symptoms found in our review were the triad of headache, focal neurologic deficits, and seizures. These are correlated with the important mass effect of the tumors in the supratentorial compartment. Salvati et al. described the time of diagnosis to be between 1 week and 18 months of the beginning of the first symptoms, which suggests a slow tumoral growth. Most tumors are diagnosed when they have grown enormously.

Most of these lesions are encountered by imaging in the cerebral lobes. Frequently, they are peripheral (in the convexity vicinity) and well demarcated, with nodular, expansive growth rather than infiltrative or invasive and rich vasculature. CT scans usually reveal classic heterogeneous multilobar images with cystic and solid components; punctate calcifications are not uncommon findings. In MRI, solid portions are hypo/isointense to gray matter in T1-weighted sequences and hyper/isointense to gray matter in T2-weighted sequences. In contrast-enhanced sequences, there is a classic ring enhancement of the cystic portion, and in T2-weighted images, there is a striking “bubbly” pattern.

Differential diagnoses in imaging are ependymoma, primitive neuroectodermic tumor, and atypical rhabdoid-teratoid tumor.

The cytogenesis of astroblastomas is not known. Bailey and Cushing proposed that astroblasts are embryonic cells destined to become astrocytes, more specifically, cells consisting of an intermediate stage in development between unipolar spongioblasts and astrocytes. Other authors have suggested a possible cellular origin derived from tanyocytes based on ultrastructural similarities observed in electronic microscope between tanyocytes and astroblasts.

Macroscopically, astroblastomas are well-circumscribed soft lobulated lesions with foci of necrosis and hemorrhage. Microscopically, some features such as perivascular pseudorosettes, prominent perivascular hyalinization, and lack of stromal fibrillation are important for the histopathologic diagnosis. Astroblastic cells are commonly polarized and monopolar; they have single cytoplasmic processes attached to blood vessels, lack a free epithelial surface differentiation, and are poorly cohesive among themselves. Pseudorosettes consist of glial cells forming a corona around the capillary lumen composed of flat endothelial cells and thickened vessel walls.
| Series                                      | Gender | Age (years) | Symptoms                          | Radiologic features | Localization | Surgery | Radiotherapy | Chemotherapy | Histologic Grade | Grade | Recurrence |
|---------------------------------------------|--------|-------------|-----------------------------------|--------------------|--------------|---------|--------------|--------------|----------------|--------|-------------|
| De Reuk et al., 1975[10]                    | M      | 61          | Cognitive disturbance             | Solid              | Frontal      | -       | -            | -            | Low            | No     |             |
| Husain et al., 1986[14]                     | M      | 3           | Seizures, hemiparesis             | Solid              | Frontal      | Subtotal | -            | Vincristine, methotrexate | Low    | Yes         |
| Bonnin and Rubinstein, 1989[5]              | 10     | 13          | Headache, vomiting, hemiparesis, seizures | Solid+cystic      | 17 lobar 2 pineal 1 suprasellar 1 subcortical 1 cerebellar 1 IV ventricle | 12 total 11 subtotal | 11 patients | 5 patients | 13 low 8 high 2 intermediate |
| Pizer et al., 1995[26]                      | 1      | 17 days     | Irritability, vomiting            | Solid+cystic      | Frontal      | Subtotal | Vincristine, etoposid | Low        | No            |
| Thiessen et al., 1998[30]                   | 1      | 6           | 1.25-51 (mean 12.67) Headache, hemiparesis, seizures | -                  | 3 frontal 3 parietal 1 temporal | 4 total 3 subtotal | 3 patients | (5940 cGy) | 3 low 4 high |
| Brat et al., 2000[61]                       | 4      | 16          | 3-46 (mean 14) Headache, seizures, vomiting | Solid+cystic      | 9 frontal 7 parietal 2 temporal 1 occipital 1 midbrain | 18 total 2 subtotal | 10 patients | 3800-7200 cGy | (mean 5250) | 10 low 10 high |
| Port et al., 2002[27]                       | 1      | 5           | 3-46 (mean 20.5) Headache, vomiting, motor disturbances | Solid+cystic      | 3 frontal 1 temporal 1 parietal-occipital 1 corpus callosum | 5 total 1 subtotal | 3 patients | (5400 cGy) | 3 low 3 high |
| Catalán-Uribarrena et al., 2002[7]         | 1      | 17          | Headache, seizures                | Solid+cystic      | Frontal      | Total    | -            | Low          |               |        |             |
| Kim et al., 2004[19]                        | 1      | 7           | Headache, vomiting                | Solid              | Brainstem    | Total    | Yes          | Low          |               |        |             |
| Kim et al., 2004[20]                        | 1      | 15          | Headache, diplopia                | Solid+cystic      | Frontal      | Total    | 4500 cGy    | High         |               |        |             |
| Navarro et al., 2005[24]                    | 3      | 5           | 1.8-14.5 (mean 7) Headache, vomiting, seizures | 4 solid 4 cystic | 4 frontal 1 parietal 1 temporal 1 III ventricle 1 IV ventricle | 6 total 2 subtotal | 6 patients | 5 patients | 4 low 4 high |
| Kaji et al., 2006[15]                       | 1      | 17          | Headache, hemiparesis             | Solid              | Frontal      | Total    | Initial dose 60 Gy | Etoposid vincristine | High  | Yes         |
| Kubota et al., 2006[22]                     | 1      | 8           | Headache                           | Hemorrhagic solid+cystic | Frontal | Total | Initial dose 40 Gy | Hig         | Yes          |        |             |
| Series                        | Gender | Age (years) | Symptoms               | Radiologic features | Localization | Surgery | Radiotherapy | Chemotherapy | Histologic Grade | Recurrence |
|------------------------------|--------|-------------|------------------------|---------------------|--------------|---------|--------------|--------------|------------------|------------|
| Miranda et al., 2006[23]     | 1      | 43          | Headache, seizures     | Solid               | Frontal      | Total   |              |              | Low              |            |
| Alaraj et al., 2007[7]       | 1      | 33          | Headache, nausea       | Hemorrhagic solid   | Temporal     | Total   | 5400 cGy     |              | Low              |            |
| Tumialán et al., 2007[31]    | 1      | 33          | Headache, nausea       | Hemorrhagic solid   | Frontal      | Subtotal | 3600 cGy     |              | Low              |            |
| Bell et al., 2007[4]         | 1      | 11          | Headache, seizures     | 9 solid+cystic      | 4 frontal    | 7 total | 2 patients   | 1 patient     | -                | 4 patients |
| Miranda et al., 2006[23]     | 1      | 43          | Headache, seizures     | Solid               | Frontal      | Total   |              |              | Low              |            |
| Alaraj et al., 2007[7]       | 1      | 33          | Headache, nausea       | Hemorrhagic solid   | Temporal     | Total   | 5400 cGy     |              | Low              |            |
| Tumialán et al., 2007[31]    | 1      | 33          | Headache, nausea       | Hemorrhagic solid   | Frontal      | Subtotal | 3600 cGy     |              | Low              |            |
| Bell et al., 2007[4]         | 1      | 11          | Headache, seizures     | 9 solid+cystic      | 4 frontal    | 7 total | 2 patients   | 1 patient     | -                | 4 patients |
| Fathi et al., 2008[15]       | 1      | 53          | Headache, vomiting     | Solid+cystic        | Parietal     | Total   | Initial dose 66 Gy | Temozolamide | High             | Yes 6 years |
| Denaro et al., 2008[9]       | 1      | 6           | Headache, seizures     | Solid+cystic        | Intraventricular | Total |              |              | Low              |            |
| Eom et al., 2008[11]         | 1      | 20          | Headache               | Solid+cystic        | Temporal     | Total   |              |              | Low              |            |
| Unal et al., 2008[32]        | 1      | 4           | Hemiparesis            | Solid+cystic        | Parietal     | Total   | 5400 cGy     | Cisplatin, etoposid | High         |            |
| Ganapathy et al., 2008[13]   | 1      | 12          | Headache, vomiting     | Solid               | IV ventricle (spinal metastases) | Total | Yes | Low |
| Notarianni et al., 2008[20]  | 1      | 20          | Diplopia, ataxia       | Cystic              | Brainstem   | Total   |              |              | Low              |            |
| Kantar et al., 2009[19]      | 1      | 7           | Headache, vomiting, seizures | Solid+cystic      | Parietal     | Subtotal | 5940 cGy | Cisplatin, etoposid, vincristine | High | Yes |
| Kemerdere et al., 2009[17]   | 2      | 6-7         | (mean 6.5)             | Headache, vomiting, seizures, motor disturbances | Solid+cystic | 1 frontal+parietal | 2 total | 2 high |            |
| Salvati et al., 2009[28]     | 2      | 4           | 27-50                  | (mean 37)           | Motor disturbances, headache, vomiting | Solid+cystic | 2 frontal | 4 total | 4 initial dose 60 Gy | 2 patients with temozolamide | 3 low | 3 patients |
| Khosla et al., 2012[18]      | 1      | 11          | Headache, vomiting     | Solid+cystic        | 1 frontal+parietal | Total | Yes | High |
Table 1: Contd...

| Recurrence | Gender | Age (years) | Symptoms               | Radiologic features | Location |
|------------|--------|-------------|------------------------|---------------------|----------|
| 2 years    |        | 30          | Headache, vision deficit, vomiting, hemiparesis | Solid+cystic        | Frontal+parietal |
|            |        | 74          |                        | Total               | 74 total (71.15%) |
|            |        | (28.84%)    |                        | 29 subtotal (27.8%) |
|            |        | 10          |                        | Solid + cystic      | 84 total (44.2%) |
|            |        | 18.5        |                        | 12 solid (11.5%)    |
|            |        |             |                        | 5 cystic (4.8%)     |
|            |        |             |                        | 3 hemorrhagic       |
|            |        |             |                        | 2 extracranial (2.8%) |
|            |        |             |                        | 2 pial (1.9%)       |
|            |        |             |                        | 1 suprasellar (0.96%) |
|            |        |             |                        | 1 cerebellar (0.96%) |
|            |        |             |                        | 1 subcortical (0.96%) |
|            |        |             |                        | 1 corpus callosum (0.96%) |

Astroblastomas can be considered a distinct clinicopathologic entity, with well-described radiologic, basal membrane. Angioarchitecture shows a papillary pattern, and in 60% of cases reported by Bonnin and Rubinstein, important collagen deposits and mural hyalinization were found.

The electronic microscopy images have shown irregular cytoplasm, prominent nucleolus, cytoplasmic interdigitations on the cellular lateral borders, and inconstant and poor intercellular junctions. Based on histologic features, Bonnin and Rubinstein divided this entity into two groups: low-grade tumors with low to moderate mitotic figures, little cellular atypia, uniform perivascular arrangement, minimal or no proliferation of vascular endothelium, and prominent sclerosis of vascular walls, and high-grade tumors related to cytological atypia. They commonly have perivascular cells arranged in multiple layers, a high mitotic rate, and hypertrophy/hyperplasia of vascular endothelium. Our review reveals 47.11% of cases classified as low-grade tumors versus 32.6% classified as anaplastic ones [Table 1]. According to Bonnin, there is a risk of anaplastic transformation with each recurrence. The immunohistochemical profile of these tumors shows positivity to GFAP, S-100 protein, and vimentin. There is a focal immunoreactivity to EMA. Brat et al. described frequent genomic abnormalities among these tumors, including gain in chromosomes 20q and 19 and deletions in 10 and X. Loss of heterozygosity in 9p has been described as a predictor of malignant transition in astroblastomas.

The ideal treatment of these lesions is complete surgical resection, which in nearly all cases is feasible because they have well-demarcated arachnoid planes visible in imaging and an expansive nature, rather than infiltrative. Near-total or total resections were described in 71.15% of the tumors reviewed in the present article, which implicitly indicates the relative ease for complete excision [Table 1].

Natural history of astroblastomas depends on the duration of symptoms before diagnosis and the resection grade. Adjuvant therapy is effective for high-grade lesions, and there is still a need to define its use for low-grade tumors. In our review, we found recurrence in 30.7%, mostly in patients with anaplastic tumors; however, as indicated by Bonnin and Rubinstein, still in the presence of histologic features strongly related to malignancy, clinical course may mislead the microscopic appearance. The role of chemotherapy has not been elucidated; there are neither protocols nor evidences that support its use or favor any specific agent nowadays.

**CONCLUSION**
pathologic, and cytogenetic features. Its recurrence is high and efforts must be taken to elucidate the role and usefulness of radiotherapy and chemotherapy to treat high-/low-grade lesions.

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