Surgical Neurology International

OPEN ACCESS

For entire Editorial Board visit : http://www.surgicalneurologyint.com

Editor: James I. Ausman, MD, PhD University of California, Los Angeles, CA, USA

Case Report

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

Víctor Hugo Escobar de la Garma, Arturo Ayala Arcipreste, Felipe Padilla Vázquez, Ricardo Ramírez Aguilar, Uriel Oliva Castruita, Rafael Mendizábal Guerra

Department of Neurological Surgery, Hospital Juarez de México, Mexico City, México

E-mail: *Víctor Hugo Escobar de la Garma - tataboxboy@yahoo.com; Arturo Ayala Arcipreste - arcipreste@hotmail.com; Felipe Padilla Vázquez - fepavar@hotmail.com; Ricardo Ramírez Aguilar - ramirezricardo2002@yahoo.com.mx; Uriel Oliva Castruita - urieloliva@hotmail.com; Rafael Mendizábal Guerra - neurozabal@hotmail.com
*Corresponding author

Received: 12 April 14 Accepted: 19 May 14 Published: 24 July 14

This article may be cited as:
de la Garma VH, Arcipreste AA, Vazquez FP, Aguilar RR, Castruita UO, Guerra RM. High-grade astroblastoma in a child: Report of one case and review of literature. Surg Neurol Int 2014;5:111.

Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2014/5/1/111/137532

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 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

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.137532

Quick Response Code:

INTRODUCTION

Astroblastoma is a rare glial tumor affecting children and adolescents whose histogenesis remains uncertain. It accounts for 0.5% of all glial neoplasms.[5] Two different subtypes of astroblastoma have been defined based upon histologic characteristics.[5] These tumors are usually located in the cerebral hemispheres, but have also been described in the cerebellum,[5] brainstem,[19,25] corpus callosum,[30] hypothalamus,[5] and the ventricular system.[9] 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. Glial fibrillary acid 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.[3] 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).[2] In 1933 and 1937, Cox classified astroblastomas as a transitional entity between astrocytoma and glioblastoma multiforme.[8]

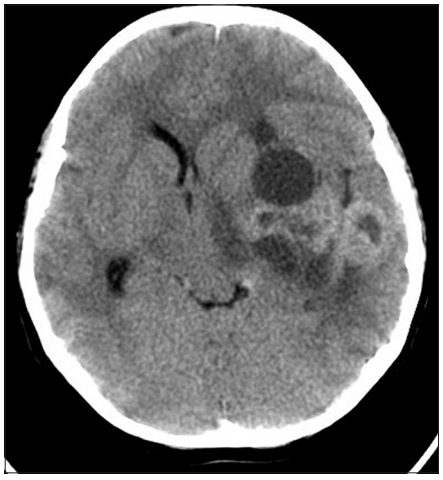

Figure 1: Axial non-contrast CT scan reveals a relatively well-demarcated frontoparietal tumor with mixed densities, with solid and cystic areas and minimal peripheral edema, but displacement of midline structures

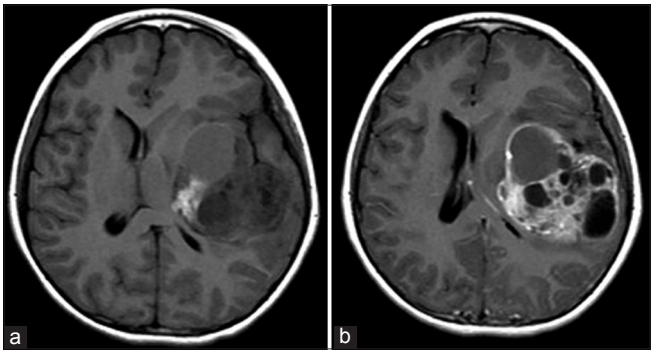

Figure 2: Axial T1-weighted (a) and contrast (b) MRI demonstrates a heterogeneous well-circumscribed frontoparietal tumor with rim enhancement and enhancement of the solid and cystic areas of the tumor

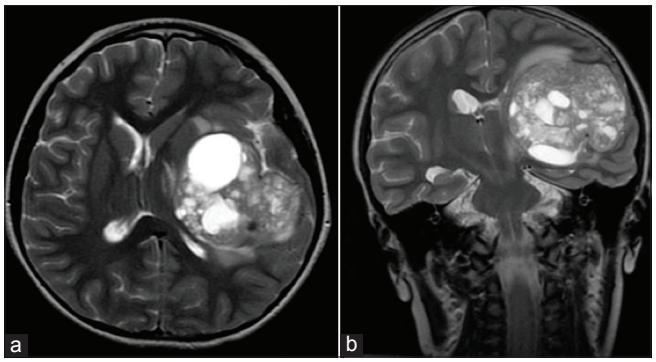

Figure 3: Axial T2-weighted MRI shows a heterogeneous, predominantly hyperintense lesion with white matter, with minimal surrounding edema and "bubbly" appearance (a). Coronal T2-weighted MRI reveals a strikingly "bubbly" appearance and midline shift (b)

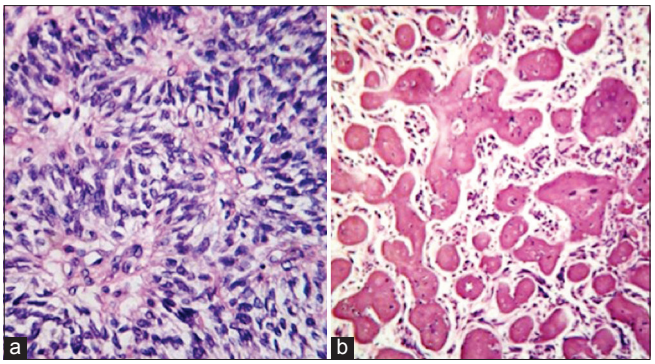

Figure 4: Histopathologic analysis revealed a papillary neoplasm composed of mildly pleomorphic cells with a striking perivascular array of pseudorosettes (a, 10×). Also, prominent endothelial hyperplasia and hyalinized vessel walls were found (b, 4×) (hematoxylin/eosin)

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.[24] Although Salvati *et al*. described a mean age of 36 years,[29] 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 [Table 1]. Female patients are predominantly affected; in the series of Navarro *et al*., 63% of patients were female.[24] We found a striking predominance of female patients (71.15%) [Table 1]. Classically, a predominance of supratentorial tumors has been described, with the cerebral hemispheres being the main location; however, infiltration to corpus callosum,[30] cerebellum,[5] pineal gland,[5] and brain stem,[19,25,30] and intraventricular[4,5,9,13] 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 [Table 1].

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.[24] 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.[4] 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.[4] Most of the series in our review present combined cystic and solid component tumors (80.7%), followed by solid (11.5%) and cystic tumors (4.8%). Hemorrhagic appearance is not uncommon; there are three reported cases in which radiologic presentation was confused with vascular etiologies because the initial presentation was acute hemorrhage.[1,22,31] Differential diagnoses in imaging are ependymoma, primitive neuroectodermic tumor, and atypical rhabdoid-teratoid tumor.[4]

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.[3] Other authors have suggested a possible cellular origin derived from tanycytes based on ultrastructural similarities observed in electronic microscope between tanycytes and astroblasts.[21,28]

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.[21,25] 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

Table 1: Features of patients diagnosed with astroblastoma described in different series

Series	Gender		Age (years)	Symptoms	Radiologic features	Localization	Surgery	Radiotherapy	Chemotherapy	Histologic Grade	Recurrence
	M	F									
De Reuk *et al.*, 1975[10]	1		61	Cognitive disturbance	Solid	Frontal	-	-	-	Low	No
Husain *et al.*, 1986[14]		1	3	Seizures, hemiparesis	Solid	Frontal	Subtotal	-	Vincristine, methotrexate	Low	Yes
Bonnin and Rubinstein, 1989[5]	10	13	21.2 (mean 5-58)	Headache, vomiting, hemiparesis, seizures	Solid+cystic	17 lobar 2 pineal 1 supraselar 1 subcortical 1 cerebellar 1 IV ventricle	12 total 11 subtotal	11 patients	5 patients	13 low 8 high 2 intermediate	7 patients
Pizer *et al.*, 1995[26]	1		17 days	Irritability, vomiting	Solid+cystic	Frontal	Subtotal		Vincritine, 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	2 patients
Brat *et al.*, 2000[6]	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	3 patients
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	2 patients
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	7 patients
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

Cont...

Table 1: Contd....

Series	Gender		Age (years)	Symptoms	Radiologic features	Localization	Surgery	Radiotherapy	Chemotherapy	Histologic Grade	Recurrence
	M	F									
Miranda *et al.*, 2006[23]		1	43	Headache, seizures	Solid	Frontal	Total			Low	
Alaraj *et al.*, 2007[1]	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	0-50 (mean 20)	Headache, seizures	9 solid+cystic 2 solid	4 frontal 5 parietal 3 extraaxial 1 temporal 2 intraventricular	7 total 4 subtotal	2 patients	1 patient	-	4 patients
Fathi *et al.*, 2008[12]	1		53	Headache, vomiting	Solid+cystic	Parietal	Total	Initial dose 66 Gy	Temozolemide	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	Frontal+parietal	Total	5400 cGy	Cisplatine, etoposid	High	
Ganapathy *et al.*, 2008[13]		1	12	Headache, vomiting	Solid	IV ventricle (spinal metastases)	Total	Yes		Low	
Notarianni *et al.*, 2008[25]		1	20	Diplopia, ataxia	Cystic	Brainstem	Total			Low	
Kantar *et al.*, 2009[16]		1	7	Headache, vomiting, seizures	Solid+cystic	Parietal	Subtotal	5940 cGy	Cisplatine 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 1 parietal	2 total			2 high	
Salvati *et al.*, 2009[29]	2	4	27-50 (mean 37)	Motor disturbances, headache, vomiting	Solid+cystic	2 frontal 2 occipital 2 temporal	4 total 2 subtotal	4 initial dose 60Gy 2 with Co 60	2 patients with temozolemide	3 low 3 high	3 patients
Khosla *et al.*, 2012[18]		1	11	Headache, vomiting	Solid+cystic	1 frontal+parietal	Total	Yes		High	

Cont...

Table 1: Contd...

Series	Gender		Age (years)	Symptoms	Radiologic features	Localization	Surgery	Radiotherapy	Chemotherapy	Histologic Grade	Recurrence
	M	F									
Escobar *et al.*, 2013	1		10	Headache, vision deficit, vomiting, hemiparesis	Solid+cystic	Frontal+parietal	Total	5400 cGy		High	2 years
Total	30 (28.84%)	74 (71.15%)	18.5		84 solid+cystic (80.7%) 12 solid (11.5%) 5 cystic (4.8%) 3 hemorrhagic (2.8%)	46 frontal (44.2%) 28 parietal (26.9%) 9 temporal (8.6%) 6 intraventricular (5.7%) 3 occipital (2.8%) 3 extraaxial (2.8%) 3 brainstem (2.8%) 2 pineal (1.9%) 1 suprasellar (0.96%) 1 cerebellar (0.96%) 1 subcortical (0.96%) 1 corpus callosum (0.96%)	74 total (71.15%) 29 subtotal (27.8%)	53 patients (50.96%)	19 patients (18.26%)	49 low grade (47.11%) 34 high grade (32.6%)	32 patients (30.7%)

basal membrane.[29] Angioarchitecture shows a papillary pattern, and in 60% of cases reported by Bonnin and Rubinstein, important collagen deposits and mural hyalinization were found.[5]

The electronic microscopy images have shown irregular cytoplasm, prominent nucleolus, cytoplasmic interdigitations on the cellular lateral borders, and inconstant and poor intercellular junctions.[22]

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.[5] 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.[5] The immunohistochemical profile of these tumors shows positivity to GFAP, S-100 protein, and vimentin.[22] There is a focal immunoreactivity to EMA.[14] 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.[6]

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.[29] 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.[5] 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

Astroblastomas can be considered a distinct clinicopathologic entity, with well-described radiologic,

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.

REFERENCES

1. Alaraj A, Chan M, Oh S, Michals E, Valyi-Nagy T, Hersonsky T. Astroblastoma presenting with intracerebral hemorrhage misdiagnosed as dural arteriovenous fistula: Review of a rare entity. Surg Neurol 2007;67:308-13.
2. Bailey P, Bucy PC. Astroblastomas of the brain. Acta Psychiat Neurol 1930;5:439-61.
3. Bailey P, Cushing H. A classification of the tumors of the gliomas group on a histogenic basis with a correlation study of prognosis. Philadelphia: JB Lippincott; 1926.
4. Bell JW, Osborn AG, Salzman KL, Blaser SI, Jones BV, Chin SS. Neuroradiologic characteristics of astroblastoma. Neuroradiology 2007;49:203-9.
5. Bonnin JM, Rubinstein LJ. Astroblastomas: A pathological study of 23 tumors, with a postoperative follow-up in 13 patients. Neurosurgery 1989;25:6-13.
6. Brat DJ, Hirose Y, Cohen KJ, Feuerstein BG, Burger PC. Astroblastoma: Clinicopathologic features and chromosomal abnormalities defined by comparative genomic hybridization. Brain Pathol 2000;10:342-52.
7. Catalan-Uribarrena G, De Las Heras-Echeverria P, Caton-Santaren B, Martinez-Soto LJ, Torrecilla-Sardon MV, Ramos-Gonzalez A. Cerebral astroblastoma: report of a case and literature review. Neurocirugia (Astur) 2002;13:378-84.
8. Cox LB. The cytology of the glioma group: With special reference to the inclusion of cells derived from the invaded tissue. Am J Pathol 1933;9:839-98.
9. Denaro L, Gardiman M, Calderone M, Rossetto M, Ciccarino P, Giangaspero F, *et al*. Intraventricular astroblastoma. Case report. J Neurosurg Pediatr 2008;1:152-5.
10. De Reuck J, Van de Velde E, vander Eecken H. The angioarchitecture of the astroblastoma. Clin Neurol Neurosurg 1975;78:89-98.
11. Eom KS, Kim JM, Kim TY. A cerebral astroblastoma mimicking an extra-axial neoplasm. J Korean Neurosurg Soc 2008;43:205-8.
12. Fathi AR, Novoa E, El-Koussy M, Kappeler A, Mariani L, Vajtai I. Astroblastoma with rhabdoid features and favorable long-term outcome: Report of a case with a 12-year follow-up. Pathol Res Pract 2008;204:345-51.
13. Ganapathy S, Kleiner LI, Mirkin DL, Broxson E. Unusual manifestations of astroblastoma: A radiologic-pathologic analysis. Pediatr Radiol 2009;39:168-71.
14. Husain AN, Leestma JE. Cerebral astroblastoma: Immunohistochemical and ultrastructural features. Case report. J Neurosurg 1986;64:657-61.
15. Kaji M, Takeshima H, Nakazato Y, Kuratsu J. Low-grade astroblastoma recurring with extensive invasion. Neurol Med Chir (Tokyo) 2006;46:450-4.
16. Kantar M, Ertan Y, Turhan T, Kitis O, Anacak Y, Akalin T, *et al*. Anaplastic astroblastoma of childhood: Aggressive behavior. Childs Nerv Syst 2009;25:1125-9.
17. Kemerdere R, Dashti R, Ulu MO, Biceroglu H, Demiroz AS, Albayram S, *et al*. Supratentorial high grade astroblastoma: Report of two cases and review of the literature. Turk Neurosurg 2009;19:149-52.
18. Khosla D, Yadav BS, Kumar R, Agrawal P, Kakkar N, Patel FD, *et al*. Pediatric astroblastoma: A rare case with a review of the literature. Pediatr Neurosurg 2012;48:122-5.
19. Kim BS, Kothbauer K, Jallo G. Brainstem astroblastoma. Pediatr Neurosurg 2004;40:145-6.
20. Kim DS, Park SY, Lee SP. Astroblastoma: A case report. J Korean Med Sci 2004;19:772-6.
21. Kubota T, Hirano A, Sato K, Yamamoto S. The fine structure of astroblastoma. Cancer 1985;55:745-50.
22. Kubota T, Sato K, Arishima H, Takeuchi H, Kitai R, Nakagawa T. Astroblastoma: Immunohistochemical and ultrastructural study of distinctive epithelial and probable tanycytic differentiation. Neuropathology 2006;26:72-81.
23. Miranda P, Lobato RD, Cabello A, Gomez PA, Martinez de Aragon A. Complete surgical resection of high-grade astroblastoma with long time survival: Case report and review of the literature. Neurocirugia (Astur) 2006;17:60-3.
24. Navarro R, Reitman AJ, de Leon GA, Goldman S, Marymont M, Tomita T. Astroblastoma in childhood: Pathological and clinical analysis. Childs Nerv Syst 2005;21:211-20.
25. Notarianni C, Akin M, Fowler M, Nanda A. Brainstem astroblastoma: A case report and review of the literature. Surg Neurol 2008;69:201-5.
26. Pizer BL, Moss T, Oakhill A, Webb D, Coakham HB. Congenital astroblastoma: An immunohistochemical study. Case report. J Neurosurg 1995;83:550-5.
27. Port JD, Brat DJ, Burger PC, Pomper MG. Astroblastoma: Radiologic-pathologic correlation and distinction from ependymoma. AJNR Am J Neuroradiol 2002;23:243-7.
28. Rubinstein LJ, Herman MM. The astroblastoma and its possible cytogenic relationship to the tanycyte. An electron microscopic, immunohistochemical, tissue- and organ-culture study. Acta Neuropathol 1989;78:472-83.
29. Salvati M, D'Elia A, Brogna C, Frati A, Antonelli M, Giangaspero F, *et al*. Cerebral astroblastoma: Analysis of six cases and critical review of treatment options. J Neurooncol 2009;93:369-78.
30. Thiessen B, Finlay J, Kulkarni R, Rosenblum MK. Astroblastoma: Does histology predict biologic behavior? J Neurooncol 1998;40:59-65.
31. Tumialan LM, Brat DJ, Fountain AJ, Barrow DL. An astroblastoma mimicking a cavernous malformation: Case report. Neurosurgery 2007;60:E569-70.
32. Unal E, Koksal Y, Vajtai I, Toy H, Kocaogullar Y, Paksoy Y. Astroblastoma in a child. Childs Nerv Syst 2008;24:165-8.