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Well-differentiated and anaplastic astroblastoma in the same patient: a case report and review of the literature

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Abstract. Astroblastoma is a rare brain tumor occurring in children and adults, rarely in the elderly. It constitutes up to 3% of all brain tumors. We report a case of a 14-year-old girl who presented with recurrent seizures and minimal right hemiparesis. Magnetic resonance imaging (MRI) revealed a left fronto-parietal brain tumor. It was managed with subtotal resection in a local hospital. Subsequently, she was referred to Princess Nora Oncology Center for further characterization and management. Pathology slide revision revealed well-differentiated astroblastoma. Upon follow up, the patient had multiple recurrences of the same tumor and emergence of a new lesion at the area of Sylvian fissure. Excision of the emerging tumor revealed anaplastic astroblastoma. Astroblastoma is a glial tumor that predominantly affects females. Its clinical progression is unpredictable, with high recurrence rate. Surgical intervention is considered the mainstay of treatment, while radiotherapy and chemotherapy effectiveness is debatable. To our knowledge, this is the first reported case of well-differentiated and anaplastic astroblastoma as two separate neoplastic lesions in the same patient with its clinical, radiological, and pathological features.

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Introduction

Astroblastoma is a rare glial tumor of uncertain origin occurring in children and adults. However, it rarely emerges as a congenital lesion or among the elderly [1, 2]. Astroblastoma makes up to 3% of all glioma types [2]. Pathologically, it is characterized by a pseudorosette pattern of glial fibrillary acidic protein (GFAP)-positive astrocytic cells around the vasculature. This tumor is predominantly supratentorial and cerebral, but rarely affects other central nervous system (CNS) components [3, 4]. Astroblastoma is classified as either low- or high-grade (well-differentiated or anaplastic/malignant) based on the cellularity, presence of necrosis, and mitotic figures [3]. Although Baily and Cushing described it early in the 1920s, and it was further characterized by Baily and Bucy, astroblastoma carries with it a large uncertainty due to having no definite histogensis or clear criteria for diagnosis [5, 6]. Moreover, this tumor has some morphological features of both astrocytoma and ependymoma, but it does not belong to either [7]. Generally, gross total resection is considered the mainstay of treatment, however, chemotherapy and radiotherapy have been shown to be beneficial, but their role has not been fully defined [8]. We describe the first occurrence of well-differentiated and anaplastic astroblastoma as two separate lesions in the same patient along with a recent review of the literature.

Case report

A 14-year-old girl presented with focal seizure and mild right hemiparesis. The family history was negative for malignancies, and physical examination revealed no neurocutaneous stigmata. She was found to have a left fronto-parital mass on magnetic resonance (MR) scan images, and the mass was subtotally resected. Seven months later, she was referred to Princess Nora Oncology Center for further management and characterization of the tumor. Pathology slides, revision revealed well-differentiated astroblas-

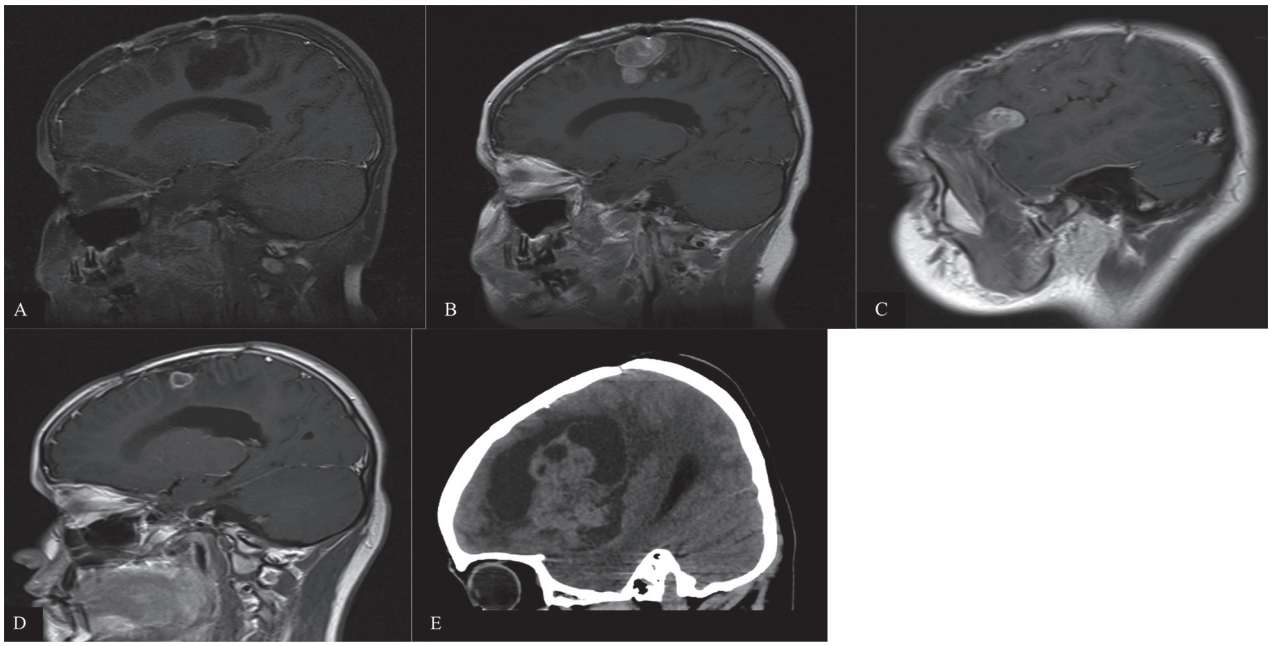

Figure 1. A: Parasagittal T1W MR image with fat saturation and postcontrast administration demonstrating the first postsurgical cavity in the left frontal area. B: A few months later, MR images obtained in sagittal plane of T1WI with contrast show the recurrent enhancing nodules in the surgical cavity. D: Follow up MRI 1 year after the second resection shows the residual ring-enhancing lesion at the surgical cavity of the left frontal lobe. C: A new intra/extra axial enhancing mass at the left perisylvian region is identified. E: Extensively enlarged left perisylvian mass with solid-cystic components seen on parasagittal post enhanced CT scan reformatted image.

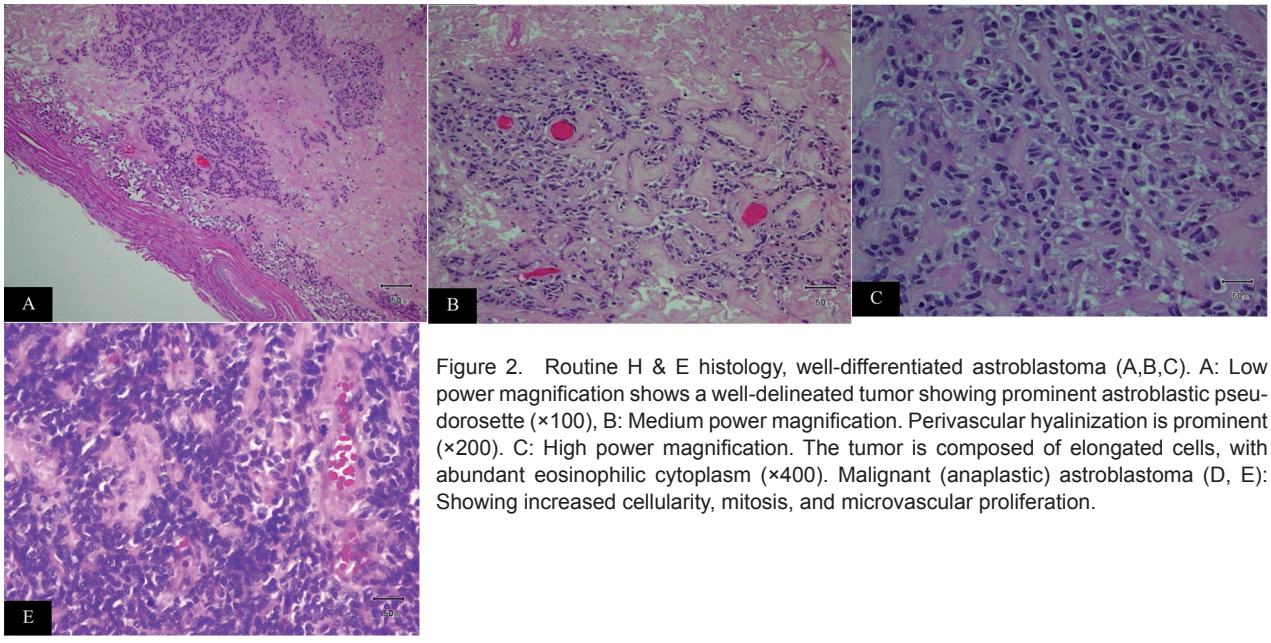

Figure 2. Routine H & E histology, well-differentiated astroblastoma (A,B,C). A: Low power magnification shows a well-delineated tumor showing prominent astroblastic pseudorosette (×100), B: Medium power magnification. Perivascular hyalinization is prominent (×200). C: High power magnification. The tumor is composed of elongated cells, with abundant eosinophilic cytoplasm (×400). Malignant (anaplastic) astroblastoma (D, E): Showing increased cellularity, mitosis, and microvascular proliferation.

toma. Repeated MRIs were performed at our center, showing multiple recurrent nodules at the surgical bed and an emerging new lesion at the area of Sylvian fissure later. Over a period of 5 and half years she experienced multiple local recurrences despite exposure to multiple treatment modalities, including 2 near-total surgical excisions and 2 sets of radiotherapy of 60 Gy concurrent with temozolmide. Then 2 sets of chemotherapy were used, which were Temozolmide in 8 cycles and vincristine/carboplatine for 4 months.

Table 1. Astroblastoma cases.

Study	Male-to-female	Age (mean years)	Symptoms	Anatomic/radiologic features	Localization	Surgery	Grade	Radiation	Chemotherapy	Recurrence	Follow-up of living patients (mean)
Kubota et al. 1985 [22]	0:1	30	Headache, vomiting, hemiparesis	Cystic	Frontal	Total	Low	None	Bleomycin	No	
Hoag et al. 1986 [9]	2:1	51.3	Confusion, urinary incontinence, seizure	3 solid	3 frontal	None	3 low	Case 3	None	–	Died
Husain et al. 1986 [10]	0:1	3	Seizures, hemiparesis	Solid	Frontal	Subtotal	Low	None	Vincristine, methotrexate	Yes	47 months
Bonnin et al. 1989 [3]	10:13	21.2	Headache, vomiting, hemiparesis, seizures	Solid + cystic	17 lobar 2 pineal 1 suprasellar 1 cerebellar 1 IV ventricle 1 subcortical	12 total 11 subtotal	13 low 2 intermediate 8 high	11 patients	5 patients	7 patients	–
Pizer et al. 1995 [2]	1:0	30 days	Irritability, vomiting	Solid + cystic	Frontal	Subtotal	Low	None	Vincristine, etoposide	No	30 months
Thiessan et al. 1998 [23]	1:6	12.7	Headache, hemiparesis, seizure	–	3 frontal 3 parietal 1 temporal	4 total 3 subtotal	3 low 4 high	3 patients (5,940 cGy)	None	2 patients	40.34 months
Brat et al. 2000 [20]	4:16	11.7	Headache, seizure, vomiting	Solid + cystic	9 frontal 7 parietal 1 midbrain	18 total 2 subtotal	10 low 10 high	10 patients 3,800 – 7,200 cGy	None	3 patients	24.4 months
Sener et al. 2001 [24]	0:1	5	Headaches, seizures	Cystic	Temporal	Total	Low	None	None	–	–
Port et al. 2002 [7]	1:5	20.5	–	Solid + cystic	3 frontal 1 temporal 1 parietal + occipital 1 corpus callosum	5 total 1 subtotal	3 low 3 high	3 patients (5,400 cGy)	None	2 patients	19.3 months
Sugita et al. 2002 [25]	0:1	33	Partial seizure of the face	Solid	Frontal	Total	Low	50 Gy	None	None	96 months
Kim et al. 2004 [26]	1:0	7	Headache, vomiting	Solid	Brainstem	Total	Low	Yes	None	–	–
Kim et al. 2004 [27]	0:1	15	Headache, diplopia	Solid + cystic	Frontal	Total	High	4,500 cGy	None	None	Died
Caroli et al. 2004 [28]	1:0	30	Coma	Cystic	Temporal	Total	High	Whole brain ^{60}Co	Temozolomide	No	60 months
Huhn et al. 2005 [53]	0:1	11	Seizures	Solid	Frontal	Total	Low	None	None	No	12 months
Navarro et al. 2005 [29]	3:5	7	Headache, vomiting, seizures	4 solid 4 cystic	4 frontal 1 parietal 1 temporal 1 III ventricle 1 IV ventricle	6 total 2 subtotal	4 low 4 high	6 patients	5 patients	7 patients	64 months
Mangano et al. 2006 [8]	1:0	8	Headache, vomiting, hemiparesis	Solid + cyctic	Frontal	Total	High	5,940 cGy	None	No	54 months

Table 1. Continuation.

Study	Male-to-female	Age (mean years)	Symptoms	Anatomic/ radiologic features	Localization	Surgery	Grade	Radiation	Chemotherapy	Recurrence	Follow-up of living patients (mean)
Kubota et al. 2006 [19]	0:1	8	Headache	Hemorrhagic solid + cystic	Frontal	Total	High	Initial 40 Gy	ACNU	No	Almost 24 months after discharge
Kaji et al. 2006 [30]	1:0	17	Headache, diplopia	Solid + cystic	Frontal	Total	Low	Initial 60 Gy	ACNU, etoposide, vincristine, interferone Beta	Yes	5 months
Miranda et al. 2006 [31]	0:1	42	Headache, seizures	Solid	Frontal	Total	Low	None	None	No	18 months
Hata et al. 2006 [32]	0:1	16	Headache	Cystic	Parietal	Total	Low	None	None	No	24 months
Alaraj et al. 2007 [33]	1:0	33	Headache, nausea	Hemorrhagic solid	Frontal	Total	Considered as high	5,400 cGy	None	No	3 months
Bannykh et al. 2007 [34]	1:0	33	Headache, weakness, blurred vision	–	Frontal	Total	High	–	–	–	–
Bell et al. 2007 [35]	1:11	20	Headache, seizures	9 solid + cystic 2 solid	4 frontal 5 parietal 3 extra-axial 1 temporal 2i ntraventricular	7 total 4 subtotal	–	2 patients	1 patient	4 patients	11.5 months
Tumialan et al. 2007 [36]	0:1	33	Headache, nausea, imbalance	Hemorrhagic solid	Frontal	Subtotal	Low	3,600 cGy	None	No	24 months
Fathi et al. 2008 [37]	1:0	53	Headache, vomiting	Solid + cystic	Parietal	Total	High	Initial 66cGy	Temozolmide	Yes	–
Denaro et al. 2008 [38]	0:1	6	Headache, seizure	Solid + cystic	Intraventricular	Total	Low	–	–	–	–
Eom et al. 2008 [39]	0:1	20	Headache	Solid + cystic	Temporal	Total	Low	None	None	No	24 months
Unal et al. 2008 [40]	1:0	4	Hemiparesis	Solid + cystic	Frontal + parietal	Total	High	5,400 cGy	Cisplatin, etoposid	No	8 months
Ganapathy et al. 2008 [41]	0:1	12	Headache, vomiting	Solid	IV ventricle with spinal involevement (spinal and thoracic)	Total	Low	Yes	None	No	Recurrence-free after 18 months
Notarianni et al.2008 [42]	0:1	20	Diplopia, numbness ataxia	Cystic	Brainstem	Total	Low	None	None	No	No residual tumor in her 3-month follow-up
Kantar et al.2009 [43]	0:1	7	Vomiting, seizure	Solid + cystic	Parietal	Subtotal	High	5940 cGy	Cisplatin, etoposid, vincristine	Yes	Died
Kemerdere et al.2009 [4]	0:2	6.5	Headache, Vomiting, seizure	Solid + cystic	1 frontal + parietal 1 frontal	2 total	2 high	Yes (both)	None	No	–
Salvati et al. 2009 [44]	2:4	36	Seizures, aphasia, hemiparesis	Solid + cystic	2 frontal 2 occipital 2 temporal	4 total 2 subtotal		4 with 60Gy initially 2 with Co 60	2 patients with temozolmide	3 patients	–

Table 1. Continuation.

Study	Male-to-female	Age (mean years)	Symptoms	Anatomic/ radiologic features	Localization	Surgery	Grade	Radiation	Chemotherapy	Recurrence	Follow-up of living patients (mean)
Johnson et al. 2010 [45]	0:1	12	Sudden headache, coma	Hemorrhagic solid	Frontal	Total	High	None	None	–	Died
Bergkasa et al. 2011 [46]	0:1	50	Seizure	Solid	Frontal	Total	High	54 Gy	Procarbazine, CCNU, vincristine	No	84 months
Weintraub et al. 2011 [47]	0:1	58	Headache, nausea, cognitive decline	Solid	Parietal + occipital	Total	Low	Gamma knife (initial 18 Gy)	None	No	17 months
Binesh et al. 2011 [1]	0:1	25	Headache, diplopia	Solid	Frontal	Subtotal	High	60 Gy	None	No	Did not accept chemotherapy
Agarwal et al. 2012 [48]	0:1	12	Headache, diplopia	Cystic	Parietal	Total	Low	None	None	No	14 months
Khosla et al. 2012 [49]	0:1	11	Headache, vomiting	Solid + cystic	Frontal + parietal	Total	High	Yes	None	No	23 months
Eschobar et al. 2013 [13]	0:1	10	Headache, hemiparesis, vomiting	Solid + cystic	Frontal + parietal	Total	High	54,00 cGy	None	Yes	No
Yao et al. 2013 [50]	1:0	36	Headache, nausea, vomiting	Hemorrhagic	Temporal + occipital	Total	Low	None	None	No	5 months
Fu et al. 2013 [51]	0:1	60	Hemiparesis	Cystic	Frontal	Total	High	None	None	No	24 months
Janz et al. 2014 [21]	0:2	16	Headache, vomiting, seizure	Solid + cystic	1 parietal + occipital 1 temporal	Total	1 low transformed to high 1 high	None	None	1 yes 1 failed to appear for MRI follow-up	30 months
Singh et al. 2014 [52]	0:1	12	Headache, vomiting, seizure	Cystic	Parietal	Total	Low	Yes	None	None	–
Samkari et al. 2015	0:1	14	Seizures, hemiparesis	Solid + cystic	Frontal + parietal	Subtotal/ Near total	Low + High	60 Gy	Temozolmide, vincristine/ carboplatine	Yes	48 months
Results	Female: 90 (72.6%) Male: 34 (27.4%) Total: 124	Mean: 7.7 years Upper: 60 years Lower: 30 days	Most common symptoms: headache, vomiting, seizure, hemiparesis	Solid and cystic: 80 (69%) Solid: 19 (16.3%) Cystic:12 (10.3%) Hemorrhagic: 5 (4.3%) :	Frontal: 59 (47.6%) Parietal: 28 (22.6%) Temporal: 9 (7.23%) Frontal + parietal: 5 (4%) Occipital: 3 (2.4%) Intraventricular: 7 (5.65%) Brainstem: 3 (2.4%) Extra-axial: 3 (2.4%) Pineal: 2 (1.6%) Temporal + occipital: 1 (0.8%) Suprasellar: 1 (0.8%) Cerebellar: 1 (0.8%) Subcortical: 1 (0.8%) Corpus callosum: 1 (0.8%)	Total: 91 (72.8%) Subtotal: 31 (24.8%) Neartotal: 2 (1.6%) None: 1 (0.8 %)	Low: 56 (45%) High: 49 (39%)	65 patients (51%)	24 patients (18%)	34 patients (27%)	9.2 months

ACNU = Nimusine; CCNU = Lomustine

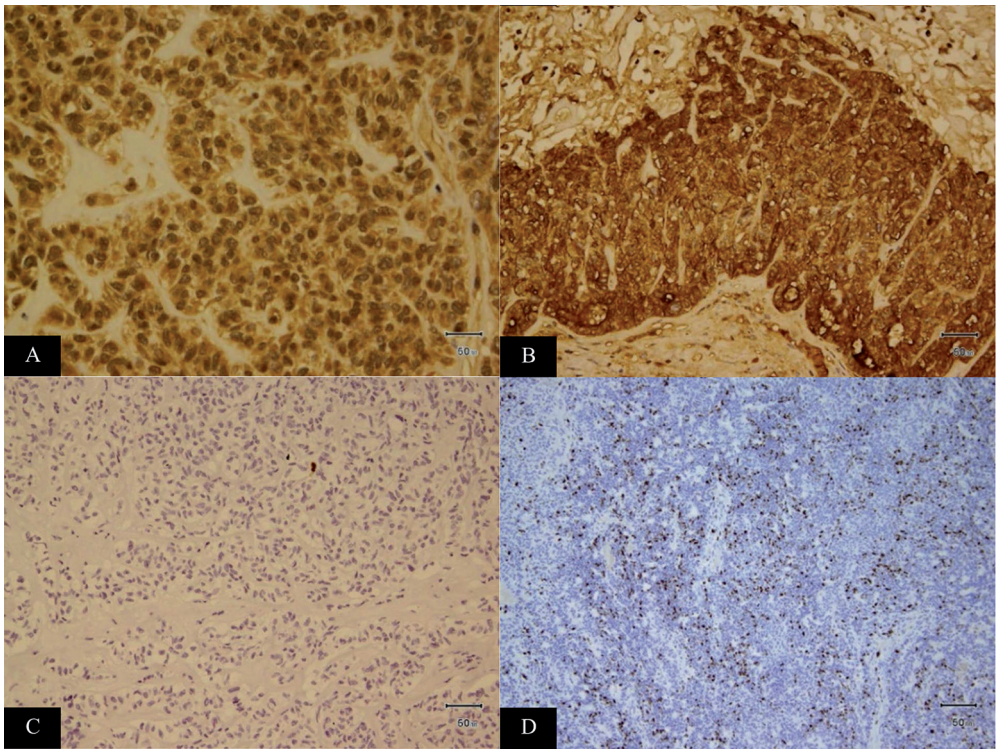

Figure 3. Immunohistochemistry. A: GFAP positive staining (×200); B: EMA positive staining (×200); C: Ki67 of the first tumor (< 2%); D: Ki67 of the second tumor (~ 20%).

Currently, the patient is progressing both clinically and radiologically.

Radiology

The first MRI scan performed at our center demonstrated a large surgical cavity in the left frontal lobe with no evidence of residual nodule. A few months later, the follow-up MRI scan showed left frontal surgical cavity with two recurrent nodules. The largest was seen at the superior aspect of the surgical bed, measuring 1 × 1.4 cm with direct contact to the meninges and the adjacent clavarium. The smaller lesion measured 1.2 × 0.6 cm and was identified at the anterior aspect of the surgical cavity. These lesions appeared to be isointense on T1 and T2 weighted images with heterogenous intense enhancement after IV gadolinium contrast administration. This was the first recurrence incidence.

In the second recurrence, the patient MRI scan showed residual tumor tissue that was previously noted in the anterior margin of the left frontal surgical cavity with interval progressive increase in size. In addition, there was a newly developed dural-based extra-axial lesion that was identified in the left Sylvian fissure area and demonstrated marked interval increase in size on multiple consequent MR images. The mass was growing extensively even after the second resection with intra- and extra-axial involvement. It showed a mixed solid component with a large cystic component, and the solid part demonstrates heterogeneous intense enhancement. Marked peritumoral edema was identified with significant mass effect, midline shift, and hydrocephalus.

Pathology

The histopathology examination of the first tumor showed a well-delineated tumor with prominent astroblastic pseudorosette (Figure 2a, b). These structures are composed of elongated tumor cells containing abundant eosinophilia with tapering processes extending to a central blood vessel. Perivascular hyalinization was prominent. However, no mitosis, infarction-like necrosis was identified. The perivascular hyalin-

ization was diagnosed as well-differentiated astroblastoma.

The histopathology examination of the emerging tumor showed similar features to the first tumor. However, increased cellularity, high anaplastic features, and mitotic index (7 mitoses/10 hpf), microvascular proliferation, and necrosis were evident. Immunohistochemistry showed that both tumors were positive for GFAP and EMA. Ki67 showed a low proliferative index (1 – 2%) for the first tumor; however, for the emerging tumor it was ~ 20%, which was subsequently diagnosed as anaplastic astroblastoma.

Discussion

Astroblastoma is a rare glial tumor of a distinctive entity [9, 10, 11]. Recently, it has been elucidated that astroblastoma occurs mainly in children and adults, rarely in the elderly [2]. The mean age in our review of 124 cases was 7.7 years, ranging from 30 days to 60 years (Table 1). We found a female predominance of 72.6% (Table 1). However, a recent study done by Ahmed et al. [12, 13] concluded that the majority of the patients (54%) are males.

Most patients in our review presented with headache, nausea, vomiting, seizures, and diplopia. Cognitive impairment, behavioral changes, and coma were found to be rare (Table 1). Regarding the location of the tumor, astroblastoma tends to affect the cerebral lobes mainly the frontal lobe (47%), the parietal lobe (22.6%), and the temporal lobe (7.23%) (Table 1).

On computed tomography (CT) scan images, the astroblastoma mass has a variable attenuation density with calcification in the majority of cases [14]. It is usually a well-delineated mass with lobular outline. It is often superficial with cortical involvement; however, it can be extra-axial or even intraventricular [14]. It appears as a bubbly lesion on T2 weighted images on MRI due to mixed solid cystic components. The solid component is usually dominant, and it shows intense enhancement postcontrast agent administration [14]. Lack of peritumoral edema is not uncommon. Although astroblastoma in most circumstances shows the aforementioned features, it can be confused with other brain tumors, including neuroectodermal, ependymoma, and other gliomas [1]. Moreover, depending solely upon the morphological features does not allow for differentiation between high- or low-grade tumor, which necessitates a pathological diagnosis [1, 6].

Since the first description of astroblastoma by Baily and Cushing and further characterization by Baily and Bucy, the criteria of diagnosis and cellular origin has not been agreed upon [5, 6]. In 1926, Baily and Bucy believed that astroblasts were precursors for astrocytes [5, 15]. Recent studies by Raff et al. [16] do not support the aforementioned theory. In 1989, Russel and Rubinstein suggested the theory of dedifferentiation from mature astroglial cells. In another paper, Rubinstein and Herman proposed that astroblastic cells are intermediate cells between astrocytes and ependymal cells [17, 18].

Astroblastoma pathological features include short processes forming perivascular pseudorosettes, vessel hyalinization, and a fibrillar pattern [1]. The differentiation between astroblastoma and nonglial tumors can be done by immunohistochemical features that show different degrees of positivity to S-100, vimentin, and GFAP [2, 10, 19]. Low-grade tumors are characterized by presence of pseudorosettes, few mitotic figures, low cellularity, and absence of vascular proliferation, whereas the high-grade tumors display higher cellularity, high mitotic index, and vascular proliferation with necrosis [3]. Electron microscopy images of astroblastic cells show cytoplasmic irregularity, prominent nucleoli, and roughly absent intercellular junctions [19]. Genomic abnormalities have been elucidated as gain in chromosome 20q and 19, deletions in 10 and X, and heterozygosity in 9p as a predictor of high-grade transformation [20].

The possible theory behind the development of the malignant astroblastoma could be due to cerebrospinal fluid (CSF) seeding. However, it is was only reported once in the literature [47]. Another point against CSF seeding are the malignant features that the second tumor displayed. The theory of transformation is not possible in this case since the second malignant focus had a different location and was growing rapidly.

Surgical intervention is regarded as the mainstay of treatment since astroblastoma,

in most instances, manifests as a well-circumscribed mass without evidence of infiltration [8, 13, 21]. Approximately 73% of the reviewed cases underwent total resection, while 25% underwent subtotal resection (Table 1).

The tumor progression was reported in many cases as unpredictable; however, well-differentiated forms are less infiltrative and show lower clinical progression in terms of symptoms and complications [1]. Some authors support the use of radiation in high-grade tumors to retard their aggressive behavior although the role of radiation is not yet well-defined in astroblastoma management [8, 13, 21]. However, chemotherapy effectiveness is not well-established, but promising outcomes were evident in high-grade tumors [8, 13, 21]. Almost 27% of patients go through a recurrence regardless of their tumor grade, and most of the living patients were followed for 9 months as recurrence-free (Table 1).

Conflict of interest

The authors have no conflict of interest to report.

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