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Clinical and imaging features of subependymal giant cell astrocytoma: report of 20 cases

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Abstract

Background: Subependymal giant cell astrocytoma (SEGA) is a clinically benign brain tumor associated with tuberous sclerosis complex (TSC). There are still controversies on early diagnosis of the tumor.

Methods: CT and MR imaging of 20 patients with pathologically confirmed SEGA were retrospectively reviewed. Two radiologists evaluated the location, shape, size, number, edge, cerebral edema, homogeneous or heterogeneous appearance, attenuation and signal intensity, degree of enhancement and calcification of lesions. Their prognoses were based on clinical observations.

Results: SEGA showed similar features in imaging: an extra-axial, well-circumscribed, periventricular mass, isodense or slightly hyperdense on CT, hypointensity on T1-weighted imaging and isointensity to hyperintensity on T2-weighted imaging. The mass enhanced markedly and heterogeneously after the administration of contrast agent. Subependymal nodules were demonstrated in 5 cases. Remarkably, 17 patients (85%) showed ventricular dilatation and 14 patients (70%) showed calcification in CT and MR imaging. Moreover, perifocal edema was not significantly near the masses. Four cases are associated with tuberous sclerosis complex (TSC).

Conclusions: Although there are no pathognomonic imaging findings for SEGA, the following clinical and imaging features might be helpful for the diagnosis, such as the initial age of first or second decade, typical location in the periventricular regions adjacent to the foramen of Monro, hydrocephalus accompanied with raised intracranial pressure, TSC and marked heterogeneous enhancement.

Keywords: Subependymal giant cell astrocytoma, Tuberous sclerosis complex, Central nervous system, Computed tomography, Magnetic resonance imaging

Background

Subependymal giant cell astrocytoma (SEGA) is a World Health Organization (WHO) Grade I tumor that typically occurs in the lateral ventricle near the foramen of Monro and rarely in the third ventricle [1]. Nevertheless, their outcome may be poor due to obstructive hydrocephalus or intratumoral hemorrhage [2]. The association of this tumor with tuberous sclerosis complex (TSC) is well known; however, its histogenesis is poorly understood. Its pure astrocytic nature has been doubted and several recent reports demonstrate it is mixed, glio-neuronal nature [3]. The purpose of this study was to delineate the basic clinical and imaging features of SEGA in a series of 20 histologically verified consecutive patients treated at our institution and to analyze previously reported cases.

Methods

Patient data

We reviewed cross-referenced records from Sep. 2002 to Dec. 2010 in the departments of radiology, neurosurgery and pathology of our hospital and identified 20 patients with pathologically confirmed SEGA, including 11 males (55%) and 9 females (45%). Computed tomography (CT) images were available in 14 patients and MRI images

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were available in all the 20 patients. All cases underwent craniotomy.

CT and MRI technique

CT scanning was performed for 14 patients using cranial CT scanner (LightSpeed, GE Medical Systems, US or Somatom Plus, Siemens, Germany). Eleven patients had non-enhanced CT images while three patients (patient 7, 8 and 15) had both non-enhanced CT and contrast material–enhanced CT images. They received 1.5 ml/kg of iohexol (Omnipaque 300; Amersham, Shanghai, China) before they underwent CT scans.

For each patient, non-enhanced MRI and contrast material–enhanced MRI scanning was performed using 1.5-T MRI scanner (Signa, GE Medical Systems, US) equipped with the standard head coil with a 240 × 240 mm field of view.

Findings were recorded by consensus. We investigated CT and MR imaging characteristics, with emphasis on the location, size, internal content, margin of the lesion, pattern of enhancement and change of the adjacent structures. The size of the lesion was measured at its greatest diameter. As for the internal content, the lesions were divided into solid and cystic. At non-enhanced CT and MRI, attenuation (signal intensity) was classified as hypodense (hypointensity), isodense (isointensity), or hyperdense (hyperintensity) compared with the adjacent cerebral parenchyma. And after contrast-enhancement, degree of CT enhancement was classified as no enhancement, mild (10-20Hu), moderate (20–50 Hu), or marked (>50 Hu) while degree of MRI enhancement was classified as no enhancement, mild (slightly higher than that of cerebral parenchyma), moderate (lower than that of dura), or marked (equal to that of dura).

Survival analysis

Survival analysis was done by SPSS software for Windows (version 16.0). The Kaplan–Meier method was used to measured Recurrence-free survival (RFS). And the assessments of Kaplan–Meier plots were tested using log rank. P values <0.05 were considered to be statistically significant.

Results

Demographics

This series included 11 males and nine females, with ages ranging from 5 to 32 years (mean age, 16.4 years, fifteen at <18).

Clinical characters

The most common symptom was increased intracranial pressure (n = 16, 80%) in accompany with headache (n = 16, 80%), vomiting (n = 5, 25%) and visual disorder (n = 3, 15%), and other presentations included paroxysmal epilepsy (n = 9, 45%) and facial angiofibromas (n = 4, 20%). Among 9 patients with epilepsy, three (patient 5, 9 and 12) were also associated with mental retardation. The duration from the appearance of the initial symptom(s) to diagnosis varied from 3 days to 10 years. The appearance of epilepsy was in smaller tumors in early stage. Headache and visual disorder was in bigger tumors in later stage. Four cases are associated with tuberous sclerosis complex (TSC) in pathology. The data of clinical characters of the 20 patients were recorded in Table 1.

Lesion locations

The most common location was lateral ventricle (n = 19, 95%). Among the 19 cases, one patient with two lesions located in both lateral ventricles. The remaining one was predominantly in the third ventricle. The lesion size ranged from 2 cm to 10 cm, with a mean of 4.1 cm. The margins of all masses were well- defined with a lobulate contour.

Radiological findings

As shown in Fig. 1, similar imaging features of SEGA were an extra-axial, typically around the region of Monro foramen, well-circumscribed mass. CT imaging data were available in 14 cases. CT scan showed isodense (n = 7) or slightly hyperdense (n = 7) masses with markedly heterogeneous enhancement after administration of the contrast agent, and cystic degeneration within lesions was noted in 3 patients (patient 9, 13 and 17). In addition, amorphous calcification was seen in all the 14 cases, and 5 associated with small tubers on either side of the ventricular wall. MRI imaging data were available in all cases. Most of the tumors were hypointense on T1WI and isointense to hyperintense on T2WI. Remarkably, ten patients (case 3–6, 8, 10, 11, 14–16) showed strong hypointensity, five patients (2, 9, 12, 13 and 18) showed isointensity, while the remaining 5 patients (1, 7, 17, 19 and 20) showed hypointensity within isointensity on T1-weighted or fluid attenuated inversion recovery (FLAIR) images. On T2-weighted or FLAIR images, 13 patients (case 2, 4, 6, 9–13, 15–18 and 20) showed strong hyperintensity; hypointensity (case 3) and isointensity (case 5) were seen in one case each; the remaining 5 patients (1, 7, 8, 14 and 19) showed hyperintensity within hypointensity. All tumors demonstrated moderate to strongly heterogenous enhancement after administration of gadolinium, and marked peritumoral edema was not obvious in our study. Hyperintensity and hypointensity on diffusion-weighted imaging (DWI) were observed in 2 cases each (case 5, 6, 10 and 19). Seventeen patients (case 1–8, 10, 12–14 and 16–20) involved ventricular dilatation.

Table 1 Grade, age, symptom, location and follow-up of the investigated SEGAs in 20 eligible subjects

Subject no	Sex	Age	Symptom	Diameter (cm)	Tumor location	associated with tuberous sclerosis complex	treatment	Follow-up time (months)/ Symptoms	Survival status
1	M	7	Headache	3	Right lateral ventricle	No	Total removal	24 / Non	Non-recurrence
2	F	16	Headache	4	Right lateral ventricle	No	Total removal	13 / Non	Non-recurrence
3	F	26	Headache, Epilepsy	3	Right lateral ventricle	No	Total removal	13 / Epilepsy	Non-recurrence
4	M	17	Headache, vomiting	6	Left lateral ventricle	No	Total removal	16 / hemiplegia	Non-recurrence
5	F	5	Epilepsy, mental retardation	4	Left lateral ventricle	Yes	Total removal	35 / Epilepsy, mental retardation	Non-recurrence
6	M	13	Headache, Epilepsy	3	Right lateral ventricle	No	Total removal	26 / Non	Non-recurrence
7	F	30	Headache, vomiting, visual disorder	7	Right lateral ventricle	No	Subtotal removal & radiotherapy	25 / Headache, visual disorder	Recurrence
8	M	16	Headache, Epilepsy	3	Right lateral ventricle	No	Total removal	27 / Non	Non-recurrence
9	F	6	Epilepsy, mental retardation	3	Left lateral ventricle	Yes	Total removal	22 / Epilepsy, mental retardation	Non-recurrence
10	F	12	Headache, vomiting	4	Left lateral ventricle	No	Total removal	8 / Non	Non-recurrence
11	M	16	Epilepsy,	2	Right lateral ventricle	No	Total removal	15 / Non	Non-recurrence
12	M	6	Headache, Epilepsy, mental retardation	4	Left lateral ventricles	Yes	Total removal	18 / Epilepsy, mental retardation	Non-recurrence
13	F	13	Headache, vomiting, visual disorder	10	Left lateral ventricle	No	Partial removal & radiotherapy	26 /Headache, visual disorder	Recurrence
14	F	26	Headache, Epilepsy	3	Both lateral ventricle	No	Total removal	13 / Epilepsy	Non-recurrence
15	M	16	Epilepsy,	2	Right lateral ventricle	No	Total removal	15 / Non	Non-recurrence
16	F	22	Headache	4	Right lateral ventricle	No	Total removal	17 / Non	Non-recurrence
17	M	15	Headache, vomiting, visual disorder	7	Right lateral ventricle	Yes	Subtotal removal & radiotherapy	12 / visual disorder	Non-recurrence
18	M	32	Headache	2	Third ventricle	No	Total removal	23/ Headache	Recurrence
19	M	17	Headache	5	Right lateral ventricle	No	Total removal	32 / Non	Non-recurrence
20	M	17	Headache	3	Left lateral ventricle	No	Total removal	25 / Non	Non-recurrence

Follow-up

In this study, total macroscopic removal was achieved in 17 patients (85%), subtotal removal in 2 patients (10%), and partial removal in one patient (5%). The follow-up ranged from 8 to 35 months. Most of the patients have exhibited similar physical and mental conditions to preoperative. There has been evidence of recurrences in 3 cases (15%). The details of follow-up were recorded in Table 1.

To compare the recurrence-free survival (RFS) for patients with bigger tumors (diameter > = 4cm) versus those with smaller tumors (diameter < 4cm). RFS was determined using the Kaplan–Meier method and assessments of Kaplan–Meier plots were tested using log rank. As shown in Fig. 2, Kaplan–Meier plots and log rank of SEGA shows that there was no significant difference between patients with bigger tumors and those with smaller tumors ($P = 0.059$). This may be due to too few cases.

Discussion

Clinical presentation

SEGA is a WHO Grade I tumor that often occurs in the lateral ventricle near the foramen of Monro and rarely

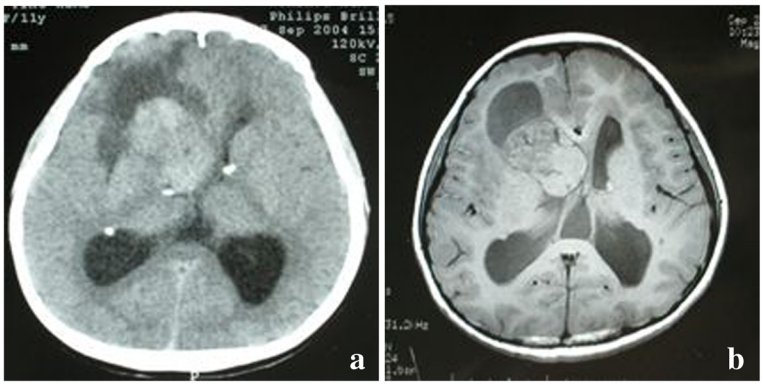

Fig. 1 **a** Nonenhanced CT image shows slightly hyperdense lesion with calcification at the right lateral ventricle adjacent to the foramen of Monro. Axial T1-weighted (**b**) MR images show the mass is well-defined with the ventricular dilatation, and the mass enhances strongly and homogeneously after the administration of Gd-based contrast agent

in the third ventricle, the fourth ventricle and pineal region [1–3]. However, the outcome of SEGA may be poor due to obstructive hydrocephalus or intratumoral hemorrhage [4]. Tuberous sclerosis complex has an association with sudden death. Sudden death may be due to epilepsy, cardiac arrhythmia, and intra-tumoral hemorrhage [5, 6]. Some authors reported that SEGA has been no evidence of any recurrence even after subtotal removal. However, some other authors considered that residual lesions tended to develop [3, 7].

SEGA accounts for 1.5% of all pediatric brain tumors [8]. In our study, the mean initial age was 16.4 years (range: 5–32years), and 15 patients were less than 18 years upon initial diagnosis, indicating that SEGA tended to occur in young patients. This was similar to the previously reported cases where the age distribution was in the first two decades of life, with the highest frequency in those 5–13 years old [9–11]. The male to female ratio in our series was 11: 9, which did not show obvious sex bias. However, in the group below 18 years, the male: female was 2:1, which showed a prominent male predilection. One of the previous reports described a slight male predominance, which was the same with our result [12]. These demographic discrepancies between the studies were probably caused by a selection bias because of the small number of cases.

It was generally believed that SEGA had a tendency to arise within periventricular regions often adjacent to the foramen of Monro [13]. In our study, the lateral ventricular location was much more frequent (19/20) than the other

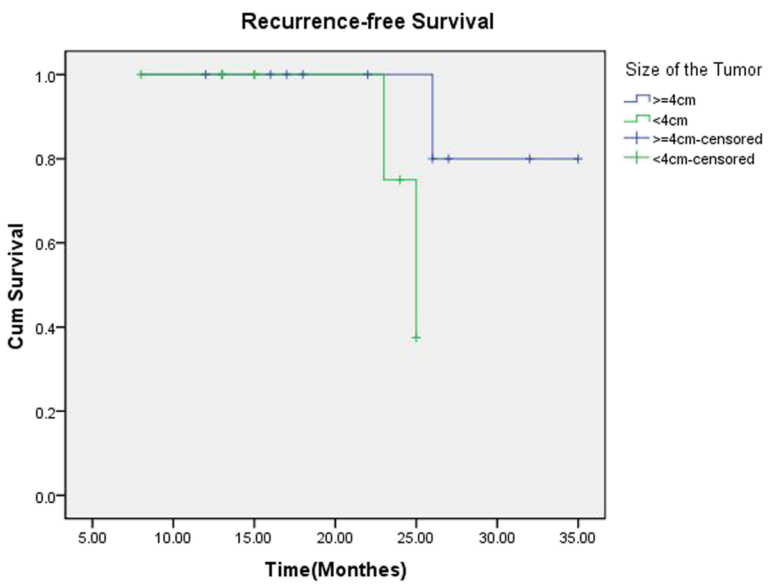

Fig. 2 Kaplan–Meier plots and log rank of SEGA shows that there was no significant difference between patients with bigger tumors (diameter > = 4cm) and those with smaller tumors (diameter < 4cm) ($P = 0.059$)

ventricular location. The remaining one SEGA was in the third ventricle. This distribution pattern was similar to conventional SEGA [1]. Rarely, parenchymal locations have been described in the brain (Basal ganglia and frontal lobe) [8, 14]. Depending on the location of tumors, the most common symptom was increased intracranial pressure. Although SEGA was thought slowly growing, it always deteriorated due to elevated intracranial pressure [15].

Associations with tuberous sclerosis complex

Other symptom, such as abnormal dermatosis was reported to be associated with TSC. When the clinical sign of Tuberous sclerosis (TS) was obvious, an early diagnosis of SEGA was common. Nevertheless, in cases with no evidence of TS, diagnosis and treatment of SEGA in adult seems to be a challenge. A rare case of an intratumoral and a small intraventricular hemorrhage complicating a SEGA without any signs of TS was reported [16]. Thus, a high index of suspicion and urgent treatment should be also required, when SEGA outside the clinical setting of the TS complex and with some urgent presentations. TSC was an autosomal disease and about 60% cases arise as spontaneous mutations as one feature for SEGA. None of our patients had positive family history for TSC. Before the advances in neuroimaging, the diagnosis of TSC was based on clinical manifestation and histopathological findings (Fig. 3). The classic clinical triad of Vogt included facial angiofibroma, mental retardation, and seizures [17].

Earnest L et al.'s study has been shown that 45% of TSC-affected patients had normal intelligence, 29% had features of "Vogt's triad" and 6% hadn't any of three features [18]. In our analysis, only one patient presented with classical Vogt's triad (case 12). TSC was a disorder of cellular migration and differentiation involving all of the germinal layers. It was manifested by hamartomas formed in skin, CNS and viscera. Facial angiofibromas were presented in approximately 85% of older children and adolescents. Multiple peri/subungual fibromas, pathognomonic of were seen in 15–20% of cases [19], as seen in our cases that all these 4 patients with facial angiofibroma were over 3 years old.

TSC has a high incidence for mental retardation, especially when associated with seizures in children. The earlier the seizures occured, the poorer was the intellectual outcome of the patient [19]. In present series, 2 patients with epilepsy showed poor intellectual development at the age of 4 months and 3 years respectively. Therefore, it seemed some correlations between seizure and intellection.

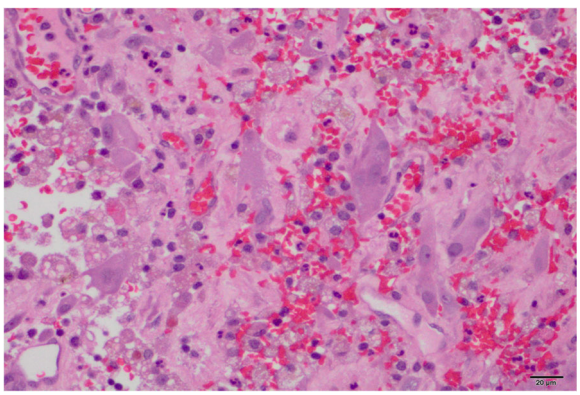

Fig. 3 Histopathological image shows that there are numerous giant cells with eccentric nuclei and tumor cells line up around the blood vessel. (original magnification, ×400)

Imaging features

The major neuroradiological lesions are tubers, SEN and SEGA. Nodules and tumors were both located at the subependymal region. A pathogenetic link between SEN and SEGA has been confirmed. Several studies have showed that there was a continuum from SEN to SEGA. The risk factors of the SEN transformed into a tumor primarily included a diameter above 5 mm, calcification and enhancement after gadolinium administration. Moreover, some studies have showed that all SENs were clonal, had the capacity to proliferate, and behaved as true neoplasms [9]. How to distinguish a tumor from SEN is still difficult especially at an earlier stage when the lesion is very small [20]. There were many studies about signal intensity or dense and contrast-enhancing characteristics suggesting correlations between SEN and SEGA [20]. The uniquely detectable difference between SEN and SEGA was the tendency of the latter lesion to enlarge and to obviously enhance with contrast administration in both CT and MRI image [21]. Cortical tubers were better diagnosed on MRI imaging than on CT scan, and they were low signal intensity in T1-weighted imaging and high signal intensity in T2-weighted imaging [20]. However, some authors suggested that signal intensity and contrast enhancement were not useful in detecting the difference between SEN and SEGA, because they found that some SENs also had slight enhancement on radiological imaging [22]. Therefore, the clinical criteria were more important than radiological images in diagnosis. If patients had hydrocephalus or high intracranial pressure, the perimonro lesion could be diagnosed as a tumor [6]. When a lesion near monro was not big enough to diagnose as a tumor, serial follow-up was essential [20]. With the growth of lesion on serial MRI image, the diagnosis of tumor could be made easily. We agreed with the criteria put forward by Cuccia at 2003 which included presence of hydrocephalus, interval increase in tumor size, new focal neurological deficit attributable to tumor, and/or symptoms of intracranial pressure [20].

SEGA appeared to arise from SEN on serial imaging, which was present in 88–95% of patients with TSC [13]. The nodule would grow up even with the treatment of gamma knife after 2 years follow-up [6]. Moreover, the lesion seemed more like a tumor because of its obvious enhancement and absence of calcification.

The common sites of SEGA were cerebral ventricles, including third ventricle, fourth ventricle and lateral ventricle. Only a few cases have been reported of SEGA with intra-axial extension in the literature. To our knowledge, only three cases were reported located at cerebral hemisphere [10, 22, 23].

On CT scan, they were well circumscribed, isodense or hyperdense, and were obviously and homogeneously enhanced after injection of contrast medium; on MRI finding, tumors were isointense, hypointense or hypointense within isointense on T1-weighted or FLAIR images, and hyperintense, isointense or hypointense on T2-weighted image or FLAIR images. Being moderate to strongly heterogenous enhancement could be seen on all of the MRI images. Moreover, frequent calcification and ventricular dilatation could be noted.

In differentiating lateral ventricular tumours, the CT density and morphology characteristics were non-specific, and MR signal intensity patterns, which varied from case to case, were also unhelpful in predicting tumour histology. Currently, data available in the literature showed that exact diagnosis of ventricular neoplasms (neurocytoma, ependymoma, subependymoma, SEGA) was hardly possible on the CT and MRI images since nearly every variation of imaging characteristics (hypo- or hyperintense, contrast enhancement, cyst, etc.) may be present or entirely absent in a single case [12]. Ventricular ependymomas were mostly hyperdense with pronounced contrast uptake. On the contrary, subependymomas were hypodense, mostly without enhancement, but occasionally slight or moderate enhancement was noted, and SEGA also displayed hypodense, rarely hyperdense or mixed imaging characteristics and always in significant degree of contrast enhancement [12]. In accordance with previous reports, all showed hypo- or mixed density, but mostly slight to moderate enhancement. Ependymomas, anaplastic astrocytomas and glioblastomas followed the characteristics of the similar extraventricular ones [21]. The imaging features of ventricular neoplasms were non-specific and usually the diagnosis was made after taking the patient's clinical condition, age and the location of tumors into consideration. We supposed that the diversity of CT or MR image might due to the different histologic contents of the tumor, such as calcification, necrosis or hemorrhage. We thought that more SEGA cases are needed to further evaluate this problem.

Overall, although the appearance of the tumors on CT or MR imaging scans in our patients was initially indistinguishable from that of other lateral ventricle gliomas and central neurocytomas, clinical features including presence in the first or second decade of life, typical location in the periventricular regions adjacent to the foramen of Monro, hydrocephalus in company with raised intracranial pressure and TSC might be a characteristic finding for SEGA.

Conclusions

Although there are no pathognomonic imaging findings for SEGA, some clinical and imaging features such as presence in the first or second decade, typical location in the periventricular regions adjacent to the foramen of Monro, hydrocephalus in company with raised intracranial pressure, TSC and marked heterogeneous enhancement might be helpful for the diagnosis.

Abbreviations

CT: Computed tomography; DWI: Diffusion-weighted imaging; FLAIR: Fluid-attenuated inversion recovery; MRI: Magnetic resonance imaging; SE: Subependymomas; SEGA: Subependymal giant cell astrocytoma; SEN: Subependymal nodules; TS: Tuberous sclerosis; TSC: Tuberous sclerosis complex

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Availability of data and materials

All data generated or analysed during this study are included in this published article.

Authors' contributions

GHM, PZ and XXL carried out the studies, participated in collecting data, and drafted the manuscript. MS and GHM performed the statistical analysis and participated in its design. All authors read and approved the final manuscript.

Competing interests

The authors report no potential conflicts of interest concerning the materials or methods used in this study or the findings presented.

Consent for publication

Written informed consent was obtained from patient (patient was over 16 year's old) or his/her patients (patient was under 16 year's old).

Ethics approval and consent to participate

This retrospective study was undertaken at the Huashan Hospital (Shanghai, China) with approval from the Huashan Institutional Review Board.

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