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SUBEPENDYMAL GIANT CELL ASTROCYTOMA: A RARE CENTRAL NERVOUS SYSTEM TUMOUR WITH A COMPLEX HISTOLOGY

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INTRODUCTION & AIM

Subependymal giant cell astrocytomas (SEGAs) are rare, low-grade neuroglial tumours that are strongly associated with the tuberous sclerosis complex (TSC). They are preferentially located in the walls of the lateral ventricles, and are characterised by marked cellular heterogeneity within the same lesion, which may complicate diagnosis, particularly for less experienced pathologists. This work aims to highlight the main histological and immunohistochemical characteristics of this entity.

METHODS & RESULTS

A 13-year-old male patient with no relevant medical history presented with seizures, persistent vomiting, and severe headaches during the span of a week. Laboratory investigations were unremarkable. An initial cranial CT scan was performed, showing a solid-cystic lesion occupying the lateral ventricles. To further categorise the lesion, an encephalic MRI revealed a 4-cm solid-cystic mass bulging intraventricularly, associated with hydrocephalus. Complete surgical resection was performed for symptomatic relief.

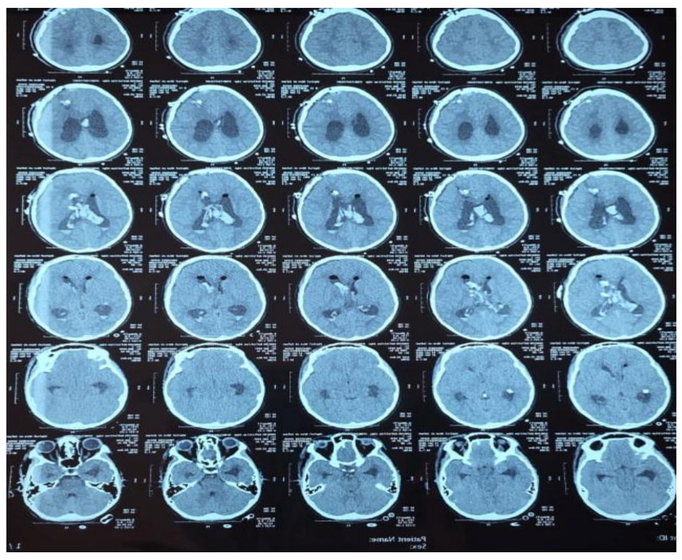

Figure 1: cranial CT scan showing a solid-cystic lesion pushing into the lateral ventricles

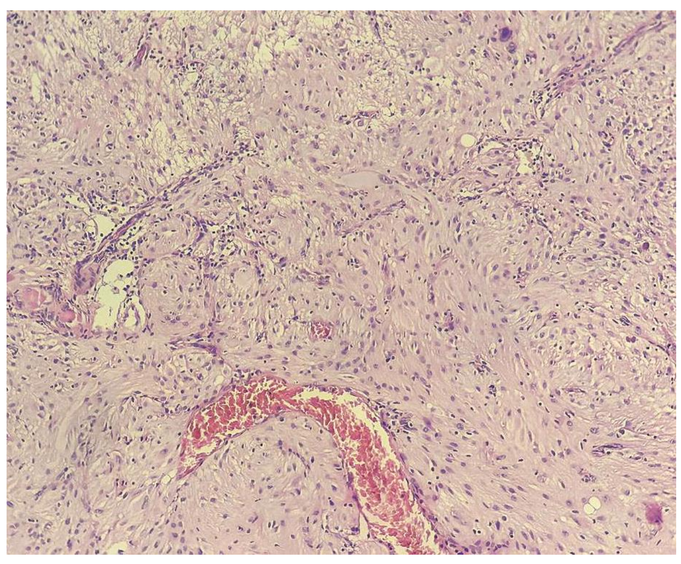

Figure 2: moderately cellular tumour proliferation organised in whorls and short fascicles

Histological examination showed a well-demarcated tumour with moderate to high cellularity arranged in nodules and short fascicles, composed of spindle, gemistocyte-like, and neuronal-like cells. No necrosis or microvascular proliferation was observed, and mitotic activity was low. Additionally, regions of widespread calcifications were noted.

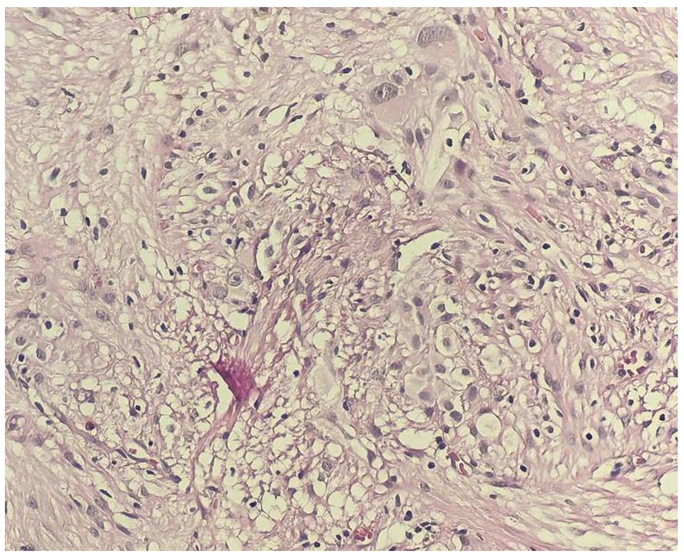

Figure 3: higher magnification showed cells with different morphologies, astrocytic, neuronal, oligodendrocytic and gemistocytic.

Immunohistochemically, tumour cells showed dual positivity for glial and neuronal markers, with strong positivity for GFAP, S100, NSE and neurofilament protein, while OLIG2 remained negative, and synaptophysin staining was focal. The Ki-67 index was approximately 5%. A reticuline stain highlighted the nodular architecture but did not show individual circumscription of the tumour cells.

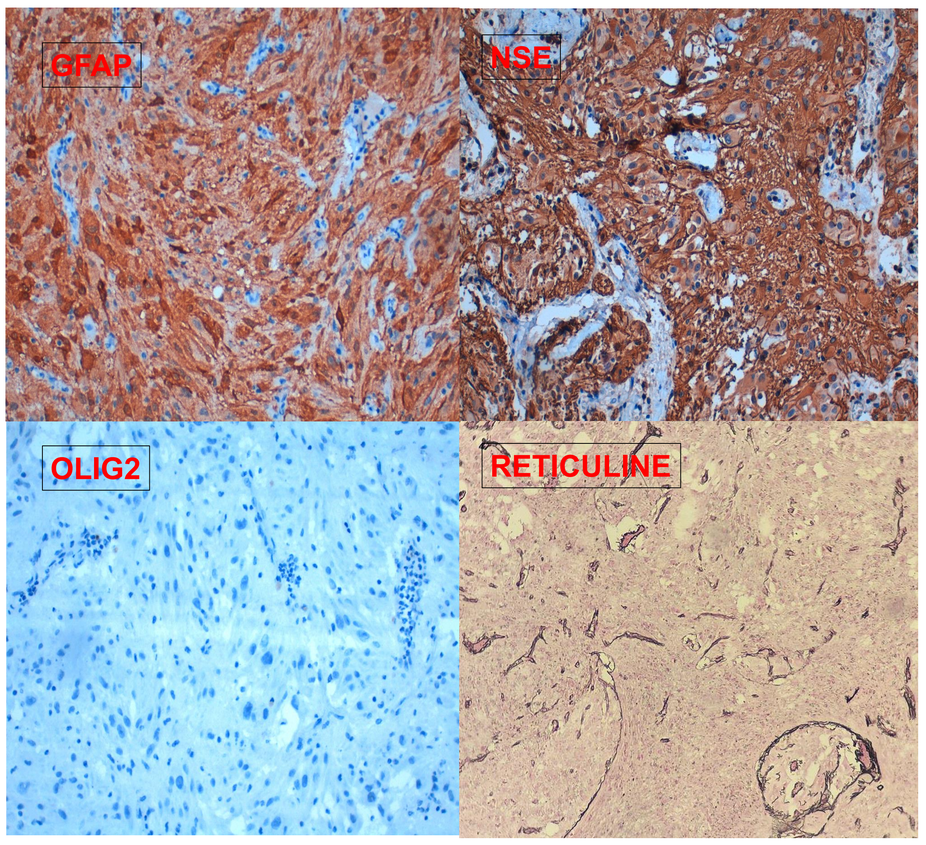

Figure 4: tumour cells stained positive for GFAP and NSE, showing neuro-glial differentiation, but were negative for OLIG 2. Reticulin staining did not show individual circumscription of tumour cells

<u>Based on morphological features, immunoprofile, tumour location and size, a diagnosis of CNS WHO grade 1 SEGA was established, and evaluation for TSC was recommended. The patient showed rapid clinical improvement following surgery.</u>

DISCUSSION

SEGA is a rare, slow-growing neuroglial tumour that is classically associated with tuberous sclerosis complex. It is classified as a CNS WHO grade 1 tumour and usually arises from the ventricular walls, most often near the foramen of Monro. Despite its benign histological features, it may cause significant morbidity, mainly due to the development of obstructive hydrocephalus.
SEGA mainly affects children and young adults, with no clear sex predominance. The reported prevalence among patients with TSC ranges from 6% to 25%. While the majority of SEGA cases occur within the context of TSC, the prevalence of non-TSC-related cases remains unclear owing to their rarity.
Clinically, symptoms are generally related to increased intracranial pressure and include headache, nausea, vomiting, seizures, and visual disturbances.
On computed tomography, SEGA typically appears as a hyperdense intraventricular mass, frequently located near the foramen of Monro. MRI usually demonstrates a T1 hypointense to isointense and T2 hyperintense lesion, often associated with calcifications, cystic changes, and hydrocephalus.
Macroscopically, the tumour is usually well circumscribed, with a rubbery consistency and a yellowish appearance. Calcifications may be encountered on sectioning.
Histologically, SEGA is characterised by a well-demarcated proliferation composed of solid sheets, nests, and short fascicles of morphologically diverse cells. These include spindle cells, gemistocyte-like cells with abundant eosinophilic cytoplasm, neuronal-like cells, and occasionally multinucleated cells. An angiocentric pattern may sometimes be observed. Nuclear pseudo-inclusions, calcifications, and nuclear pleomorphism are relatively frequent, whereas mitotic figures and necrosis remain rare.
Immunohistochemically, SEGA shows dual glioneuronal differentiation. Tumour cells are positive for GFAP and S100 but are negative for OLIG2. Variable expression of neuronal markers, including NSE, neurofilament protein, synaptophysin, and NeuN, may also be observed. In addition, TTF1 expression has been reported, while CD34 is typically negative.
The main differential diagnosis includes subependymal nodules, which are considered precursor lesions of SEGA and require careful clinicoradiological correlation. Other intraventricular tumours, such as ependymoma and central neurocytoma, should also be considered. In most cases, the marked cellular heterogeneity and immunohistochemical profile of SEGA allow a reliable distinction.
Treatment is mainly based on surgical resection in symptomatic patients, with complete resection being associated with a good prognosis. Additionally, mTOR inhibitors have shown significant efficacy in reducing tumour volume.

CONCLUSIONS

Subependymal giant cell astrocytoma is a rare CNS WHO grade 1 tumour that is strongly associated with tuberous sclerosis complex and is typically located near the foramen of Monro. Clinical manifestations are mainly related to obstructive hydrocephalus. Diagnosis relies on recognition of the characteristic morphological variability and dual glioneuronal immunophenotype. Complete surgical resection remains the main treatment and is associated with favourable outcomes.