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Letter to Editor Glioblastoma in lateral ventricle: A case report^{\star}

To the editor,

We present a 34-year-old male with convulsions and consciousness disorder. MRI scan revealed a solid-cystic mass in the right lateral ventricle and involved the corpus callosum growing towards the contralateral ventricle (Fig. 1). The final pathological diagnosis revealed a high-grade glioma, conforming to glioblastoma, WHO grade IV.

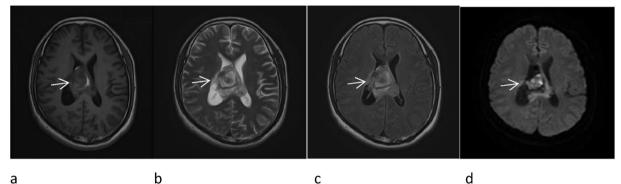
About 50% of astrocytomas are GBM, including primary glioblastomas and secondary glioblastomas which may progress from lowgrade diffuse astrocytoma, anaplastic astrocytoma, oligodendroglioma or ependymoma. Histologically, primary and secondary glioblastomas are largely indistinguishable, but their genetic and epigenetic profiles differ. The dominant genetic signposts of secondary glioblastoma are IDH1 mutations, which are absent in primary glioblastomas.¹ The immunohistochemistry in this case showed IDH1 (–), suggesting it might be primary glioblastoma.

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GBMs classically present in the cerebrum, localized to the deep white matter of the frontal, parietal, and temporal lobes. A few of them can be located in the basal ganglia region, corpus callosum,





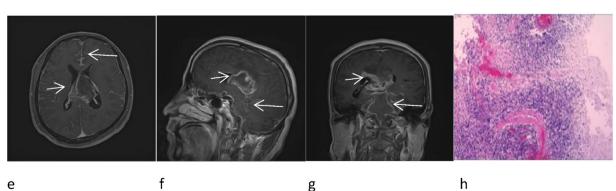


Fig. 1. Pathologically confirmed lateral ventricular glioblastoma in a 34-year-old male. (a) Plain axial T₁WI showed an iso-to a hypo-intense lesion in the lateral ventricle and involved the corpus callosum (short arrow)with strip-like hyper-intensity in it. The lesion was heterogeneously hyper-intense on axial T₂WI(b) and FIAIR(c) (short arrow). (d)DWI showed slightly hyperintensity (short arrow). (e)-(g) Post-contrast T₁WI showed irregular peripheral enhancement (short arrow) and diffuse leptomeningeal enhancement (long arrow), indicating cerebrospinal fluid seeding and sub-ependymal spread. (f)Microscopic examination (HE, 100 \times).

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spinal cord, pineal gland area, cerebellum and cerebellopontine angle area. However, although described in the literature, intraventricular location of glioblastoma is an exceptional entity, and very few cases are reported,² can affect younger individuals including children. It hypothesized that the tumor may grow into the ventricle by transependymal invasion or that the origin of intraventricular glioblastomas are the neuroglial cells of the septum pellucidum or, more likely, the fornix.³ In this case, the tumor mainly localized in the body of the right lateral ventricle and involved the corpus callosum growing towards the left lateral ventricle without any periventricular infiltrated structure identifiable. The most probable origin of this tumor should be located at any of the structures that surround this cavity, or could developed at the subependymal level, breaking early in their growth through the ependymal layer towards the ventricle cavity.

Intraventricular GBMs, even though their location is not very typical, can have typical imaging characteristics of high-grade gliomas.⁴ It can be extended to both cerebral hemispheres through the corpus callosum, anterior commissure and posterior commissure, and also can metastasize to the leptomeninges through the subarachnoid space. The imaging findings of this case are consistent with GBMs, and diffuse leptomeningeal enhancement indicated cerebrospinal fluid seeding and sub-ependymal spread (Fig. 1).

This malignant tumor should be included in the differential diagnosis of intraventricular lesions especially in the lateral ventricles, including choroid plexus papilloma, meningioma, ependymoma, central neurocytoma and so on. In this case, the preoperative MRI imaging was misdiagnosed as ependymoma. The patient's age and location of the tumor were one of the reasons for the misdiagnosis. Then, their imaging findings are similar, which is another difficulty to distinguish the two. Calcification can be seen more often in ependymomas than in GBMs, and most of the ependymomas are connected broadly with the base of the lateral ventricle wall, and due to their plastic nature, may grow along the ventricle, which are the characteristics to differentiate.⁵

Authorship

Y.L. Chang: Conception and design of study, Data analysis and/or interpretation, Drafting of manuscript and/or critical revision, Approval of final version of manuscript.

Declaration of competing interest

A conflict of interest occurs when an individual's objectivity is

potentially compromised by a desire for financial gain, prominence, professional advancement or a successful outcome. *ASJSUR* Editors strive to ensure that what is published in the Journal is as balanced, objective and evidence-based as possible. Since it can be difficult to distinguish between an actual conflict of interest and a perceived conflict of interest, the Journal requires authors to disclose all and any potential conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.asjsur.2022.03.070.

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