Primary central nervous system lymphoma differential diagnosis

Despite some characteristic conventional MR imaging findings, it may be difficult or even impossible to distinguish cerebral lymphomas from glioblastoma multiforme (Glioblastoma)\(^1\).

Primary cerebral lymphoma shows the FA decrease compared with NAWM. The FA and ADC of primary cerebral lymphoma were significantly lower than those of Glioblastoma. DTI is able to differentiate lymphomas from Glioblastoma\(^2\).

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For general imaging appearances on CT and MRI consider:

Secondary CNS lymphoma: indistinguishable on imaging, however, it tends to involve more leptomeninges (~2/3 of cases)

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Cerebral toxoplasmosis:

Toxoplasmosis does not exhibit subependymal spread

More likely to lie in basal ganglia, corticomedullary junction

CNS lymphoma is thallium/PET avid, whereas toxoplasmosis is not.

Topographic analysis using voxel-based morphometry (VBM) provides useful information for differentiating PCNSL from Glioblastoma\(^3\).

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tumefactive MS/ADEM

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Cerebral abscess

Peripheral enhancement of PCNSL is thicker

Central restricted diffusion

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Neurosarcoidosis\(^4\).
Glioblastoma

**Butterfly glioma/Glioblastoma**

more commonly centrally necrotic

more commonly demonstrates evidence of haemorrhage

Multiparametric MRI evaluation using **DWI**, **T1 perfusion MRI**, and **SWI** enabled reliable differentiation of PCNSL and Glioblastoma in the majority patients, and these results support an integration of advanced MRI techniques for the diagnostic work-up of patients with these tumours.\(^5\)

**Case from HGUA**

![MRI images](image)

The findings are compatible with a primary neoplastic lesion with **subependymal** and **subarachnoid** extension, suggesting as a first possibility a **high-grade glioma** or less likely a **lymphoma**.

**Space occupying lesion** of probable **extra-axial** location, centered on the left **lateral ventricle** (frontal horn, body, atrium) and part of the **temporal horn**, which crosses the midline, extending to the **septum pellucidum**, to the contralateral lateral ventricle (part of the frontal horn, body and atrium) and to the **floor of the third ventricle**. The most voluminous portion of the mass (centered in the **atrium** of the left lateral ventricle) is approximately 7.5 x 5.8 x 4.5cm. This **lesion** shows alteration of the **intensity** of
the heterogeneous signal in all sequences, with restriction to the predominant heterogeneous diffusion in peripheral areas. There is a central area of hypointense necrosis in all sequences and some foci of hemorrhage / calcifications within the lesion. There is no edema in the white matter. Signs are noted of growth of the lesion through the fornix until reaching the region of both mammillary body. In the diffusion and postcontrast sequences in addition, foci of signal and enhancement alteration are appreciated, some of probable subarachnoid seat on the left surface of the pons, and midbrain, in both trigeminal bones, cais, as well as on the surface of the medulla and perilesional medulla. Another lesion can be seen, intra-axial and probably dependent on the optic chiasm, isointense on T2 and with a hypointense focus on T2 * suggestive of a hemorrhagic focus and another alteration in signal intensity with restriction to diffusion in the posterior region of the bridge and medulla. The findings are compatible with a primary neoplastic lesion with subependymal and subarachnoid extension, suggesting as a first possibility a high-grade glioma or less likely a lymphoma. Left lateral ventricular bulging due to intraventricular injury.

2) http://www.ajnr.org/content/29/3/471.full
4) https://radiopaedia.org/articles/primary-cns-lymphoma