Dissemination pathways of glioblastoma multiforme.

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Learning objectives

To illustrate how *gliobastomas multiformes* (GBMs) may extend, not only locally but seeding. To make radiologists aware of the important role in accurate and timely diagnosis of GBM and its spread pathways.
Background

Glioblastoma multiforme (GBM) is an extremely aggressive diffuse astrocytic tumor commonly found in the supratentorial white matter of the cerebral hemispheres. It is the most common primary brain tumor in adults, accounting for 25% of all cases.

Attending to the pathogenetic mechanism, there are three kinds of GBM: primary GBMs, that develop de novo (without pre-existing lower grade tumor), secondary GBMs (degeneration from a lower grade astrocytoma) and the ones that occur sporadically or as a part of a heritable tumor syndrome (NF1, Li-Fraumeni syndrome, Maffucci syndrome,...)

The most consistent prognostic variables in malignant gliomas are patient age, Karnofsky Performance Status (KPS) score, tumor grade, and treatment (extent of resection and postoperative radiation therapy). However, clinical parameters do not fully account for the observed variation in survival rates. Therefore, additional indicators are needed to more accurately determine the diagnosis and the prognosis of patients with anaplastic gliomas. According to this, the relationship between survival and the appearance of tumor on imaging studies is important. Several imaging properties of GBM, such as noncontrast-enhancing tumor (nCET), edema, and multifocality/satellite lesions, have been studied in order to establish proper characterization of the tumor and survival rates. Neuroradiologists should have a firm understanding of the issues involved in imaging brain tumors spread; and this paper focus on the dissemination pathways of GBM.

Dissemination of GBM occurs most commonly by local extension from their original location by direct extension along white matters tracts; however, cerebrospinal fluid, subependymal, and haematogenous spread also can occur. Supratentorial tumors usually spread in an anterior-posterior direction; yet, crossing over to the opposite hemisphere was seen frequently in the area of the corpus callosum and the thalamus. When GBM appears in an infratentorial location, it has a 50% incidence of spinal seeding. The development of extraneural metastases may be indirectly related to previous surgery or radiation therapy, but there is also increasing evidence that distant metastases may occur in the absence of previous operation or radiotherapy.
Imaging findings OR Procedure details

DISSEMINATION PATHWAYS:

The most frequent imaging appearance of GBM is a large heterogeneous mass in the supratentorial white matter that exerts considerable mass effect. Less frequently, GBM can occur near the dura mater or in the corpus callosum, posterior fossa, and spinal cord. Ex. of GBM on page

Attending to classical GBM spread:

1-White matter tracts extension:

It is the most common mechanism of dissemination. Supratentorial tumors usually spread in an anterior-posterior direction (Fig. 1 on page 6), and infratentorial neoplasms more often extend in a cranio-caudal direction.

Crossing over to the opposite hemisphere was seen frequently in the area of the corpus callosum:

- The dense compact nature of the white matter tracts of the corpus callosum, relative to the adjacent hemispheric white matter, makes it a barrier to the flow of interstitial edema and tumor spread. Due to the aggressive behavior of GBM it should be considered for any lesion crossing the corpus callosum. Fig. 2 on page 6
- In these cases, GBM commonly display a characteristic bihemispheric involvement, resulting in a classic butterfly pattern. Fig. 3 on page
- On MR imaging, these tumors typically enhance solidly and intensely in the corpus callosum, although occasionally no enhancement is seen. Fig. 4 on page 8

White matter tracts extension has also been described through anterior and posterior commisures, thalamus, internal and external capsule and spinotalamic tracts. Fig. 5 on page 9

2- Satellite tumor nodules. Multifocal and multicentric GBM:
Up to 20% of the GBM are **multifocal** (microscopically demonstrated connections between the multiple areas of the tumor) (Fig. 6 on page 10), and up to 5% are **multicentric** (without pathological microscopic contact) (Fig. 7 on page 11).

It’s often difficult to distinguish between true metastatic forms from these types of central nervous system GBM.

**3- Ependymal, subpial and intramedullary spinal cord metastasis:**

These disseminations uncommonly occur, but, when an infratentorial location of GBM takes place, it has a 50% incidence of spinal seeding, and may develop in association with leptomeningeal tumor dissemination.

The interval between the intracerebral GBM and these metastasis is 12-14 months and the mean survival time, under this condition, never exceeds 6 months.

Ependymal dissemination: Fig. 8 on page 12.

**4- Meningeal spread:**

Either leptomeningeal (Fig. 9 on page 13) or paquimeningeal gliomatosis may be very difficult to document, even when clinically suspected. MRI and biopsy of meningeal tissue can play an important role in confirming the diagnosis.

The most common sites for spinal GBM metastases are the lower thoracic, upper lumbar and lumbosacral regions.

**5- Extraneural metastases:**

Non-nervous central system metastases rarely take place but at any part of the body (principally lung, liver, nodes and bones). They may be indirectly related to previous surgery or radiation therapy by virtue of the increasing length of survival from which the patient has benefited. There is also increasing evidence that they may occur in the absence of previous operation or radiotherapy.
Fig. 0: Axial MRI. FLAIR sequence. GBM in frontal lobe.

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**Fig. 0:** Axial CT after IV contrast administration. GBM in left lobe with corpus callosum extension.

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**Fig. 0:** Basal (A) and after IV contrast administration (B) axial CT. Butterfly pattern of GBM.

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**Fig. 0:** CT after IV contrast administration. GBM affecting splenium of corpus callosum.

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Fig. 0: FLAIR(A) and T1 weighted after IV contrast administration(B) axial MRI. GBM disseminating through mesencephalon.

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**Fig. 0:** FLAIR(A) and T1 weighted after IV contrast administration(B) axial MRI. Multifocal GBM.

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Fig. 0: T1 weighted after IV contrast administration axial MRI. Multicentric GBM.
Fig. 0: After IV contrast administration T1 weighted axial MRI (A) and after IV contrast administration CT (B). Ependymal spread of GBM.

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Fig. 0: FLAIR axial MRI. Leptomeningeal dissemination of GBM.

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Conclusion

Over 80% of all GBM spread either by contiguity or seeding. Knowing the dissemination pathways of GBM, diagnosis and monitoring turn faster and more precise.

Radiologists should be familiarized with these ways of spread to improve patients management and to make a more accurate diagnosis approach.
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References


