Glioblastoma

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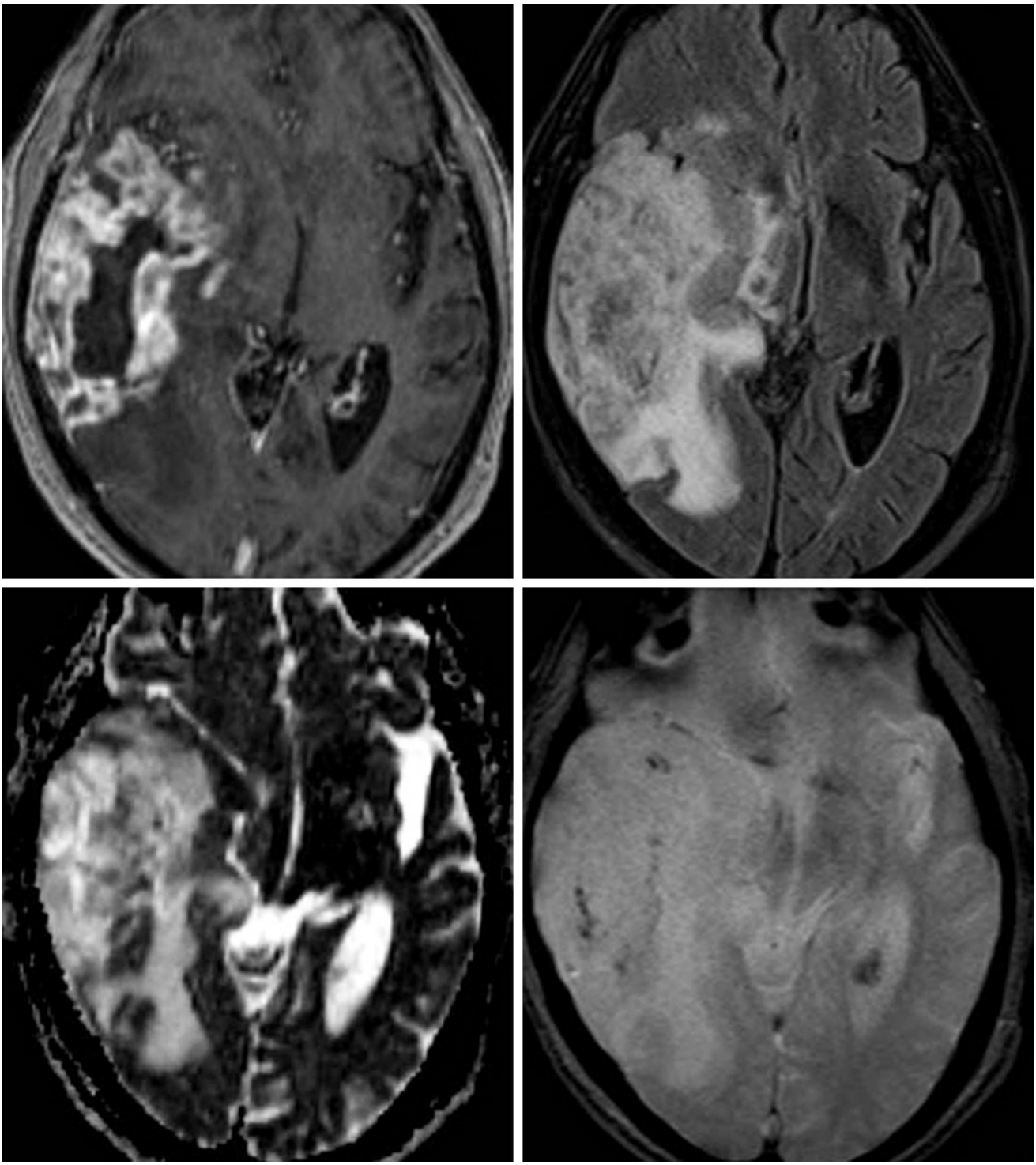

Figure 1: (Top Left) The avid peripheral enhancement and central necrosis seen in this lesion on postcontrast T1WI is typical of a GBM. (Top Right) Infiltrative FLAIR hyperintensity extending beyond the

enhancing margins usually represents a combination of edema and nonenhancing infiltrative tumor. (Bottom Left) Areas of low signal intensity in regions of known tumor on ADC imaging usually indicate hypercellularity. (Bottom Right) Hemorrhagic foci are also not uncommonly seen on GRE or SWI, as in this patient's GBM.

BASIC DESCRIPTION

- Malignant, rapidly enlarging astrocytic tumor with microvascular proliferation and necrosis
- Most common primary intracranial neoplasm

PATHOLOGY

- WHO grade IV
- Necrosis and microvascular proliferation are characteristic features
- Develops de novo (primary) or from malignant dedifferentiation of anaplastic astrocytomas (secondary)
    - Uncommonly secondary to previous radiation
    - Primary glioblastoma (GBM) more aggressive, more common in elderly patients
    - Secondary GBM more common in younger patients
- Associated with neurofibromatosis type 1 (NF-1), Turcot, Li-Fraumeni, and Maffucci syndromes and Ollier disease

CLINICAL FEATURES

- All ages affected (ages 45–75 years most common)
- Slight male gender predilection
- Median survival <1 year
    - Better prognosis with younger age, greater extent of resection, and *O*(6)-methylguanine-DNA methyltransferase (MGMT)-positive genetics
- Presenting symptoms depend on tumor location

    - Seizures, focal neurologic deficits
- Treatment: tumor debulking, radiotherapy, chemotherapy (temozolomide, antiangiogenesis agents)

IMAGING

- General
    - Poorly defined, infiltrating supratentorial white matter mass
        - Neoplastic cells extend beyond areas of signal abnormality
        - Frontal, temporal, and parietal lobes most common
        - Brainstem and cerebellum more common in children
    - Usually solitary but can be multifocal (synchronous tumors) in up to 20% of cases
    - Neovascularity and necrosis are common features
    - Thick, irregularly enhancing rind with central necrosis
    - Cysts, hemorrhage, fluid-debris levels, and vascular flow voids are common features
    - Commonly spreads along white matter tracts and crosses midline via the corpus callosum, anterior and posterior commissures (“butterfly” glioma)
    - Occasional cerebrospinal fluid dissemination
- CT
    - Irregular hypodense to isodense mass with central hypodensity (necrosis)
    - Surrounding vasogenic edema/tumor with mass effect on adjacent structures
    - Radiodense hemorrhage could be present, calcification rare
    - Irregular, heterogeneous rind of enhancement on contrast-enhanced CT
- MRI
    - T1WI: irregular, hypointense white matter mass; areas of

hyperintensity could represent subacute hemorrhage

- T2WI: heterogeneously hyperintense; surrounding vasogenic edema and tumor infiltration; presence of flow voids suggests neovascularity
- FLAIR: heterogeneously hyperintense
- T2*GRE: susceptibility artifact related to blood products/hemorrhage
- DWI: diffusion restriction reflecting hypercellularity is common in solid tumor components
- T1WI+C: thick, irregular rind of peripheral enhancement; enhancement can be ring, nodular, homogenous, or patchy
- MRS/MR perfusion: decreased NAA and myoinositol, increased Cho, lipid/lactate peak (1.3 ppm) indicating anaerobic metabolism of necrosis, increased relative cerebral blood volume (rCBV) compared with rCBV associated with lower-grade astrocytomas
- Diffusion tensor imaging (DTI) tractography and functional MRI (fMRI) may assist in surgical planning

IMAGING RECOMMENDATIONS

- MRI with contrast; MRS, MR perfusion, DTI, and fMRI are often useful adjuncts

For more information, please see the corresponding chapter in Radiopaedia.

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