

### 3. Metastatic Brain Disease

Metastases comprise about half of all intracranial tumors. Lung cancer followed by breast cancer then melanoma are the most common tumors to metastasize to the brain. Metastatic lesions are confirmed by the presence of multiple parenchymal mass lesions (Fig 3.1 A-D), although the differential diagnosis also includes less common entities such as multifocal primary brain tumors and abscesses. Solitary brain metastases—seen frequently in breast, uterine, and gastrointestinal cancer—comprise about half of all brain metastases and pose a diagnostic challenge. Other imaging features suggestive of metastatic brain cancer include well-defined lesions (although pathologically metastases are less well defined than they appear on imaging) and a location at the gray-white matter junction. The predilection for this region likely relates to the inability of hematogenously spreading tumors to freely move through the smaller vascular structures found there. This hematogenous spread may form the basis for the preponderance of metastatic cancer found in the supratentorial region, reflecting the dominance of the carotid over the vertebrobasilar system.

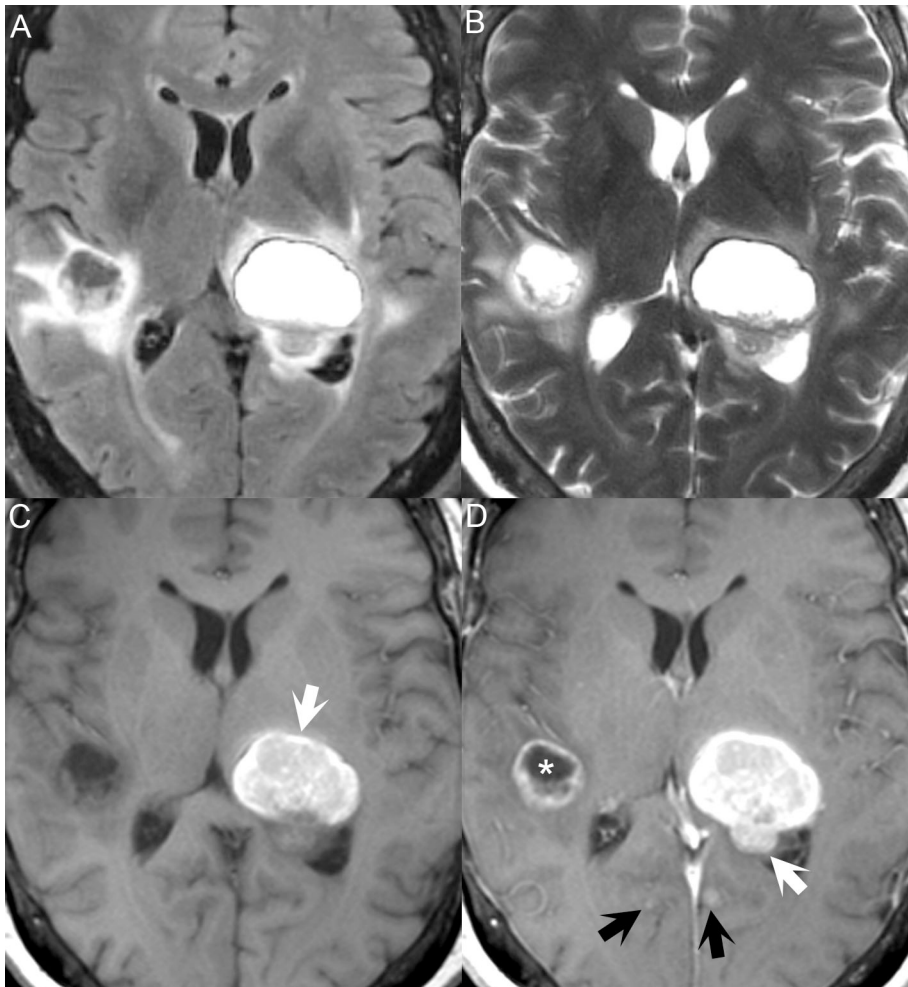


Fig. 3.1

Of unenhanced scan sequences, T2WI (and in particular FLAIR, Fig. 3.1 A) is preferred for the visualization of metastatic cancer due to the high SI of the associated edema. Vasogenic edema from metastatic lesions tends to follow white-matter tracts without crossing the corpus callosum (in distinction to a glioblastoma) and tends to be disproportionately large/prominent in extent when compared to the size of the tumor foci. Unfortunately, metastatic lesions at the cortical gray-white matter junction (and in other locations) may lack sufficient edema to be visualized using T2WI alone. This is illustrated in Figure 3.1 D by the two small occipital lesions (black arrows), which are visualized post-contrast but are inconspicuous on FLAIR and T2WI (Fig. 3.1 A, B). The number of lesions detected, and their location, dictate surgical versus non-surgical (chemotherapy and/or radiation) management. Contrast administration is mandated for ease of diagnosis and improved lesion detection (the latter essential for therapy planning). If only a solitary lesion is apparent on first review, a careful search of the enhanced scans for a second metastatic lesion must be made. Contrast-enhanced MRI detects far more lesions than enhanced CT, with the latter modality no longer employed for this diagnosis/evaluation.

It should be noted, irrespective of the sensitivity of MR for detection of metastases, that some lesions can be difficult to visualize even on post-contrast T1WI. There are many ways to improve lesion detectability on MR. These include thin section imaging and the use of high field (3 T) MR. Due both to the higher available SNR and the improved sensitivity to intravenous contrast enhancement, preferential use of 3 T in screening for intracranial metastatic disease is suggested. Triple-dose (0.3 mmol/kg) contrast administration further improves the number of visualized metastases (by  $\approx 30\%$ ). However, cost of the contrast agent has led to high-dose brain MR not being widely used.

Contrast administration also aids in distinguishing benign hemorrhagic disease (which may appear similar to the lesion denoted by the white arrow in Figure 3.1 C) from hemorrhagic metastases by more clearly demonstrating the enhancing tumor focus (Fig. 3.1 D, white arrow). On nonenhanced studies, hemorrhagic metastases—most common in melanoma, renal cell carcinoma, and choriocarcinoma—demonstrate signal on T1 and T2WI more heterogeneous and delayed in evolution than the expected signal changes of hemorrhage (see chapter 8). Hemorrhagic metastases also lack the uniform, hypointense hemosiderin-rim seen in the later stages of benign hemorrhage.

Metastases may appear as solid or ring-enhancing lesions. Ring-enhancing metastatic lesions (Fig. 3.1 D, asterisk) may be distinguished, to some extent, from those of more benign entities (e.g. abscess) by the thickness and irregularity of the enhancing wall. A cystic appearance is also common for metastatic lesions (Fig 3.1 D, asterisk). These tend to be of CSF-like intensity on T2 and T1WI (Fig 3.1 B, C), but differentiated from CSF on

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FLAIR scans (Fig 3.1 A) due to the presence of protein. Finally, certain metastatic tumors demonstrate signal characteristics on MR that provide hints to their origin. For example, (nonhemorrhagic) melanotic melanoma is characteristically high SI on T1WI due to the paramagnetic effects of melanin, while mucinous adenocarcinoma of the colon is suggested by hypointensity on T2WI. Leptomeningeal metastatic disease, a much less common finding, presents on imaging as abnormal enhancement of the leptomeninges, often focal and somewhat nodular. Bacterial, viral, or tuberculous meningitis, as well as sarcoidosis can mimic leptomeningeal metastases on post-contrast MR.