Metastasis

Last Updated: May 3, 2021

PARENCHYMAL METASTASES

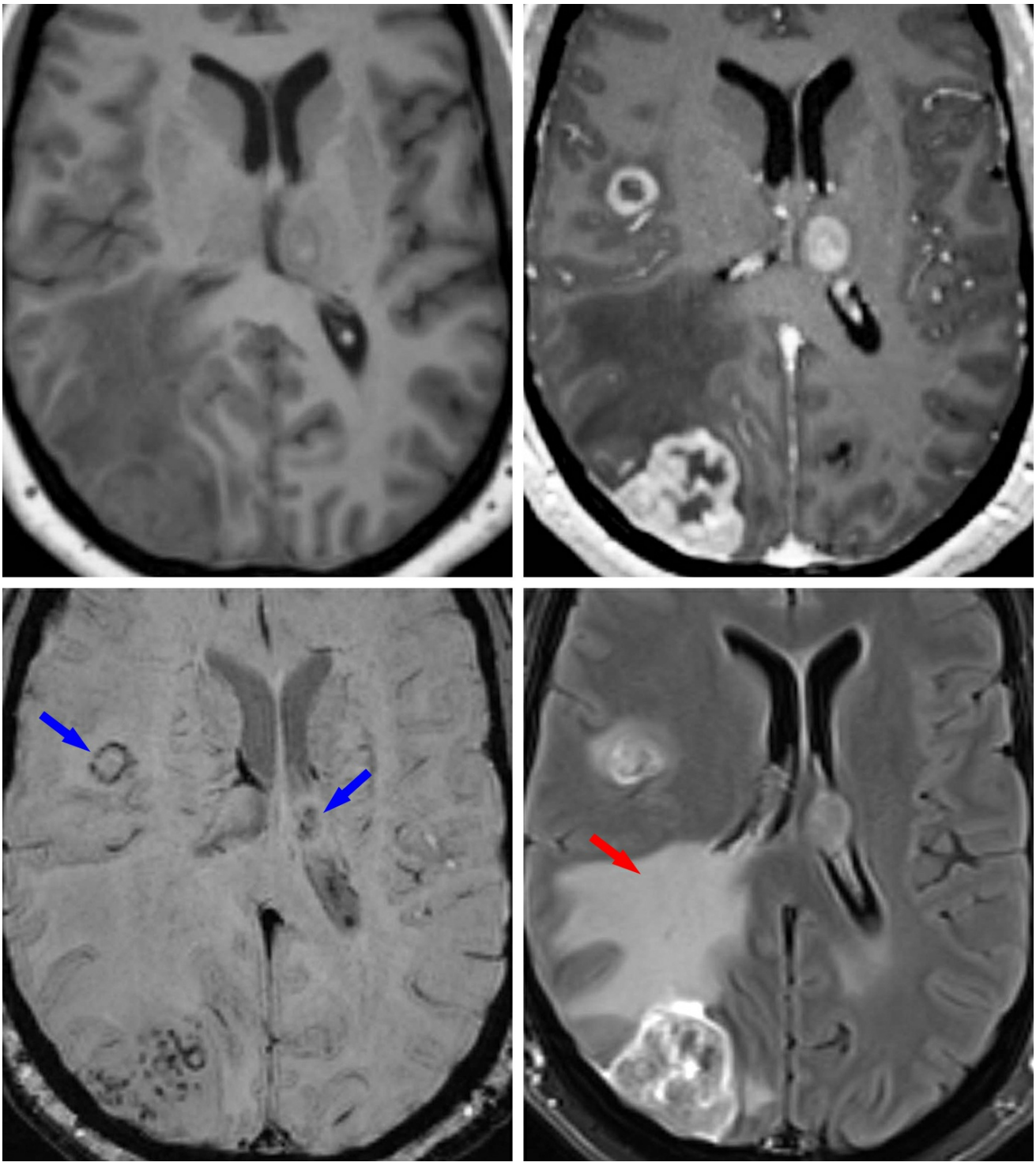


Figure 1: Parenchymal metastases. In this patient with a history of lung cancer presenting with headache, there are parenchymal metastases

located with the left thalamus at the gray–white junction of the right frontotemporal and right parietooccipital lobes. The lesions demonstrate predominantly low signal intensity on axial T1-weighted precontrast imaging (top left) but avid enhancement on postcontrast T1 imaging (top right). (Bottom Left) The lesions demonstrate foci of low signal on SWI, suggesting associated tumoral microhemorrhage (blue arrows). (Bottom Right) The lesions demonstrate characteristic marked surrounding T2 FLAIR signal, compatible with vasogenic edema that is most conspicuous around the right parietal lesion (red arrow).

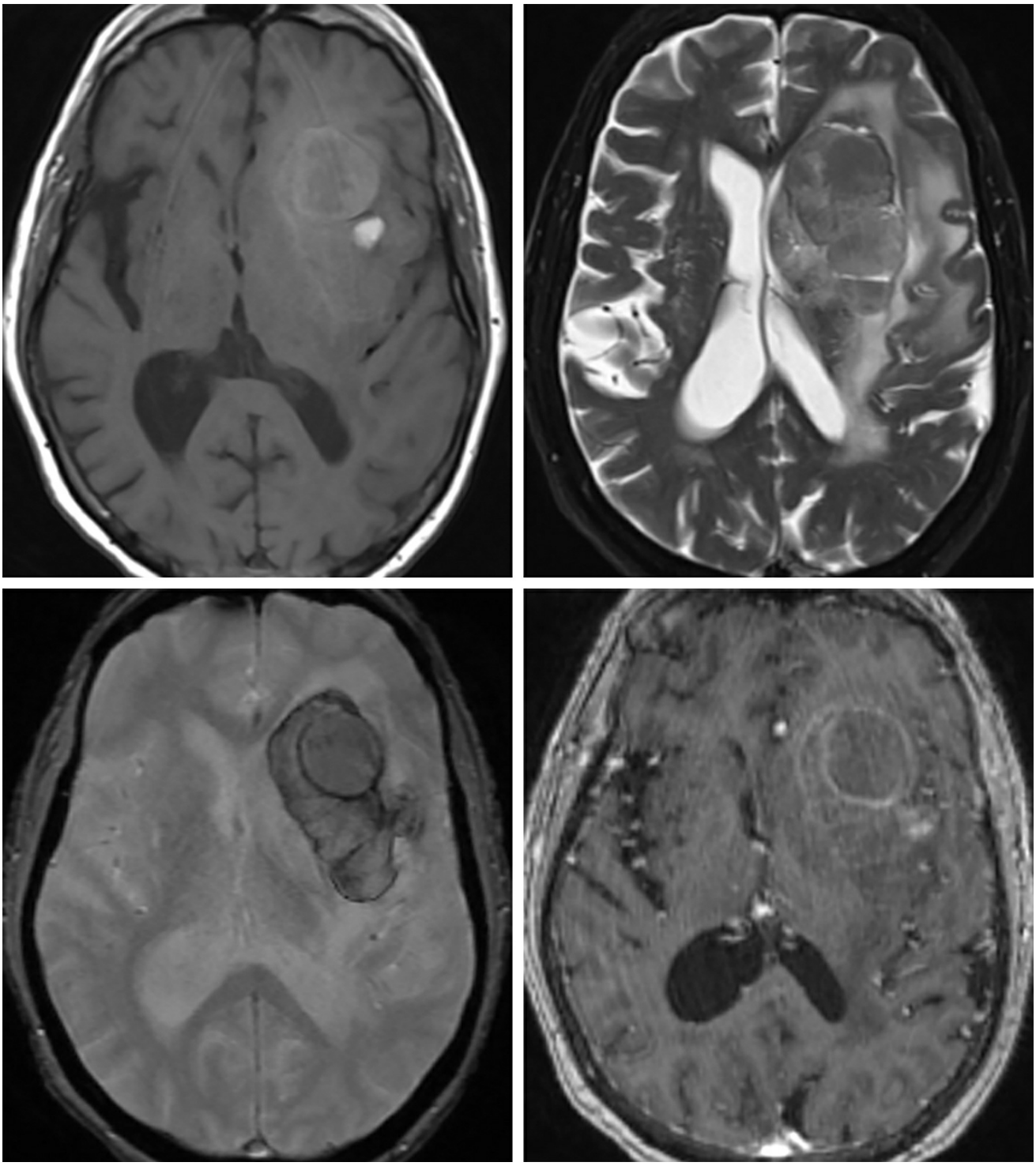


Figure 2: A single large hemorrhagic lesion can often be a diagnostic conundrum with a large differential. (Top Left) This melanoma metastasis demonstrated evidence of extensive hemorrhage, with areas of T1 hyperintensity. Low T2 signal intensity and T2-hyperintense surrounding edema (top right) and dark signal on GRE (bottom left) can be seen. Only faint enhancement was visible in some portions of this lesion (bottom right) when compared with the precontrast image (top left), much of which could have been obscured by the tumoral hemorrhage.

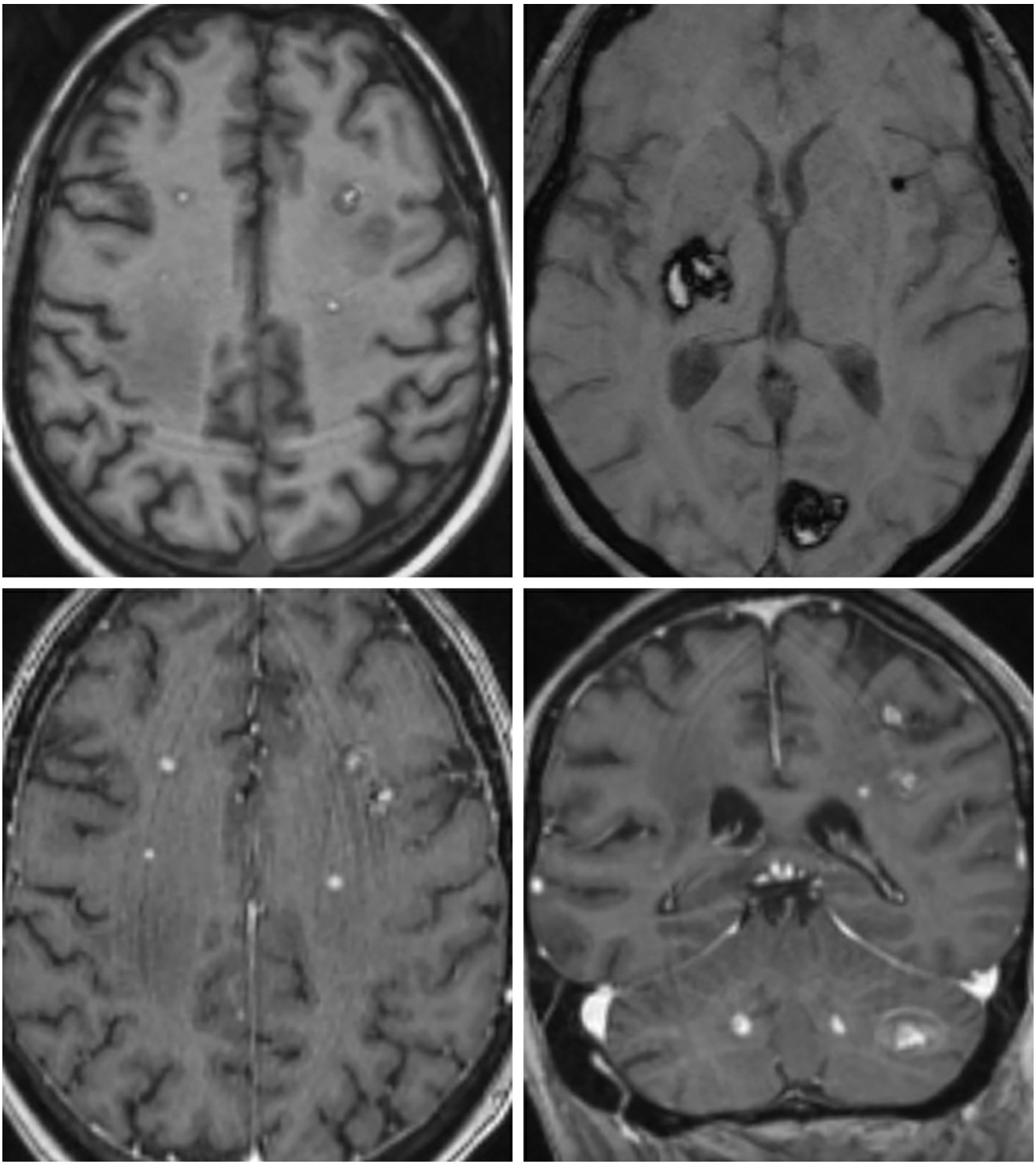


Figure 3: (Top Left) Even without intravenous contrast, melanotic

melanoma metastases are often bright on T1-weighted images, either because of the melanin or the high rate of hemorrhage of these lesions. (Top Right) These features also often make melanoma highly visible on SWI as black lesions. Contrast-enhanced imaging is still of great value for these patients (bottom left, axial; bottom right, coronal), often showing lesions that are otherwise not clearly visible.

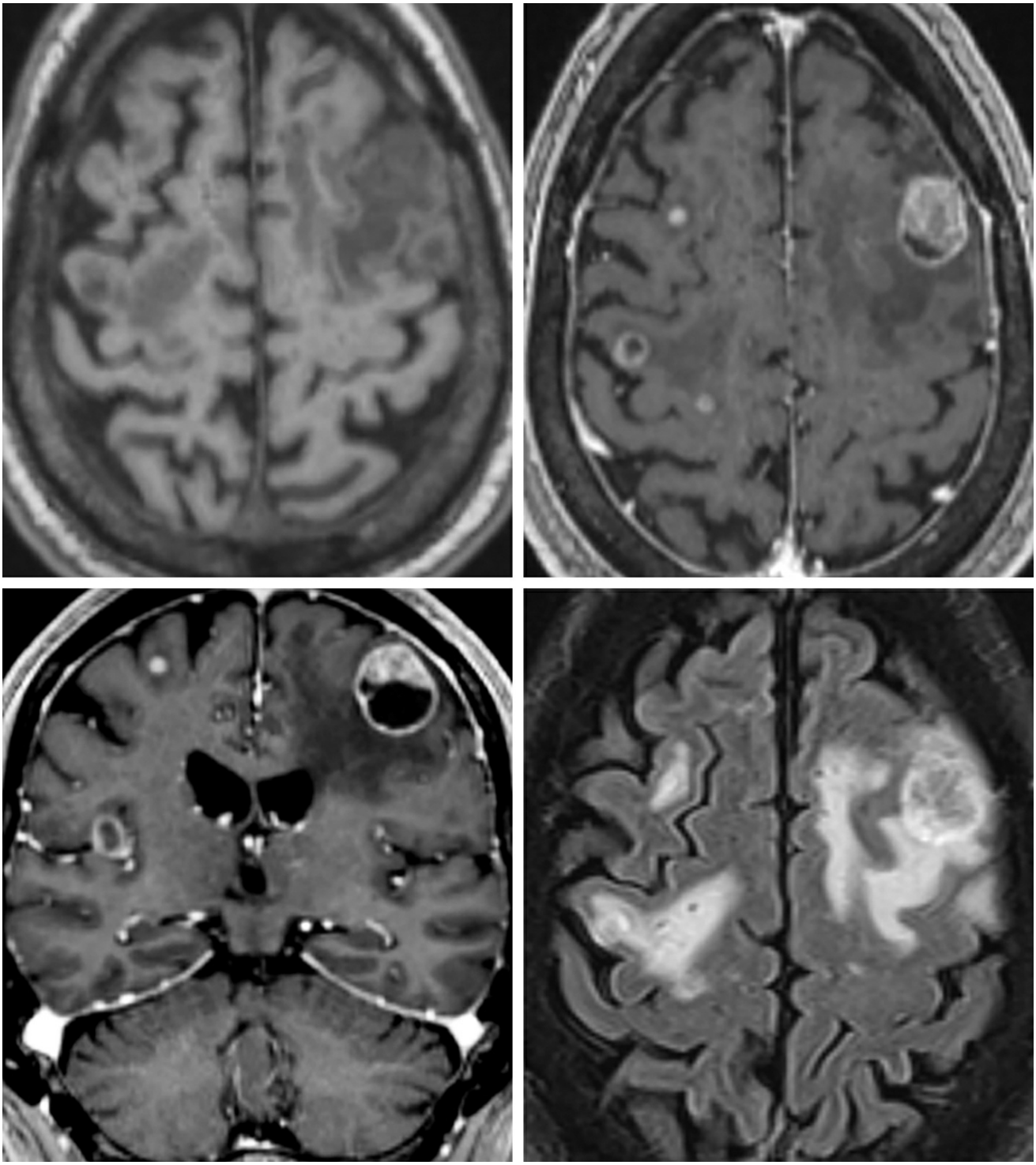


Figure 4: (Top Left) This example of melanoma metastases demonstrates no intrinsic T1 hyperintensity. The lesions demonstrate bright enhancement on contrast-enhanced T1-weighted sequences (top right,

axial; bottom left, coronal). (Bottom Right) FLAIR imagine also illustrates the degree of hyperintense vasogenic edema associated with these metastases.

Pathology

- Typically secondary to hematogenous dissemination
- Lung, breast, melanoma, renal, and colorectal cancer represent the most common primary sites
- Although less common, parenchymal metastases can arise in the setting of direct extension from head and neck primary tumors
- Gross specimen appears as round, circumscribed, tan or grayish-white nodules
- Associated features, including hemorrhage, peritumoral edema, necrosis, and mucinous material, and specific histology vary depending on the primary tumor

Clinical Features

- Symptoms
    - Presenting symptoms are highly variable, ranging from asymptomatic (up to 75% of the time) to severe neurological deficits
    - Patients with symptoms typically present with headache, seizure, and/or focal neurological deficits
    - Specific neurological deficits directly related to the particular anatomic site of parenchymal metastases
- Prognosis
    - Parenchymal metastatic disease overall portends a very poor outcome
    - Detection of parenchymal metastases is of critical clinical importance, because treatment strategy often drastically changes based on presence of metastatic disease

Imaging

- General
    - Parenchymal metastases typically located at the gray–white junction
    - Majority occur within the cerebral hemispheres (80%), with the cerebellum (15%) and basal ganglia (3%) less commonly involved
    - A few primary malignancies, however, such as uterine and prostate cancer, may show predilection for the posterior fossa
    - Parenchymal metastases can be solitary (50%), occur as 2 lesions (20%), or present as 3 or more lesions (30%)
    - Variable size; most are <1.5 cm
- Modality specific
    - CT
        - Predominant feature of parenchymal metastases is marked surrounding vasogenic edema (white matter hypodensity abutting adjacent peripheral gray matter) and can be the only visible feature
        - Majority of metastases, if visualized, are hypodense to isodense relative to adjacent brain parenchyma
        - Metastases with a high nuclear-to-cytoplasmic ratio can appear hyperdense
        - Associated hyperdense hemorrhage can be seen, particularly with melanoma, thyroid, renal cell, choriocarcinoma, and breast primaries
        - Avid enhancement is a typical feature on contrast-enhanced CT imaging
    - MRI
        - T1WI
            - Metastases are usually mildly hypointense to isointense on T1WI
            - Exceptions include T1 hyperintense melanotic

melanoma metastases and hemorrhagic metastases with variably increased signal depending on the age of the blood products

        - T2WI/FLAIR
            - Typically T2 hyperintense, although tumors with a high nuclear-to-cytoplasmic ratio can be hypointense
            - Associated vasogenic edema appears as white matter T2/FLAIR hyperintensity surrounding metastasis
        - DWI
            - Parenchymal metastases have variable appearance on DWI, with high-cellularity tumors more likely to show restricted diffusion (high DWI/low ADC signal)
        - SWI
            - Melanoma and hemorrhagic metastases appear as low signal “blooming” on SWI
        - Contrast
            - Most parenchymal metastases demonstrate avid enhancement, particularly compared to the surrounding brain parenchyma with its intact blood–brain barrier
            - Enhancement patterns are usually solid, nodular, or ring-enhancing
- Imaging recommendations
    - Modality of choice depends on the clinical scenario and the acuity of symptoms
    - Noncontrast CT is often performed in emergent situations to evaluate for hemorrhage and to assess sequelae of mass effect
    - If nonemergent, MRI without and with intravenous contrast is the preferred imaging modality for completely characterizing the extent of metastatic disease and monitoring for treatment response or progression

- Mimics
  - Infectious
    - Focal nodular and ring-enhancing lesions at the gray–white junction also often seen with infectious etiologies, such as cerebral abscesses and septic emboli
    - Central restricted diffusion more commonly seen with abscesses/emboli
  - Neoplastic
    - Solitary metastases in particular can be difficult to differentiate from primary central nervous system neoplasms such as glioblastoma
    - Glioblastomas more likely centered in deep white matter and demonstrate infiltrative appearance versus the demarcated appearance and peripheral gray–white location of metastases
  - Demyelinating
    - Demyelinating lesions, such as in patients with multiple sclerosis, more likely to be located in deep and periventricular white matter
    - Incomplete ring enhancement is classic pattern for demyelinating lesions
  - Ischemic
    - Small subacute strokes might enhance, mimicking metastatic lesion
    - Enhancement pattern with ischemia more likely to be nodular or ring-enhancing

DURAL/PACHYMENINGEAL METASTASES

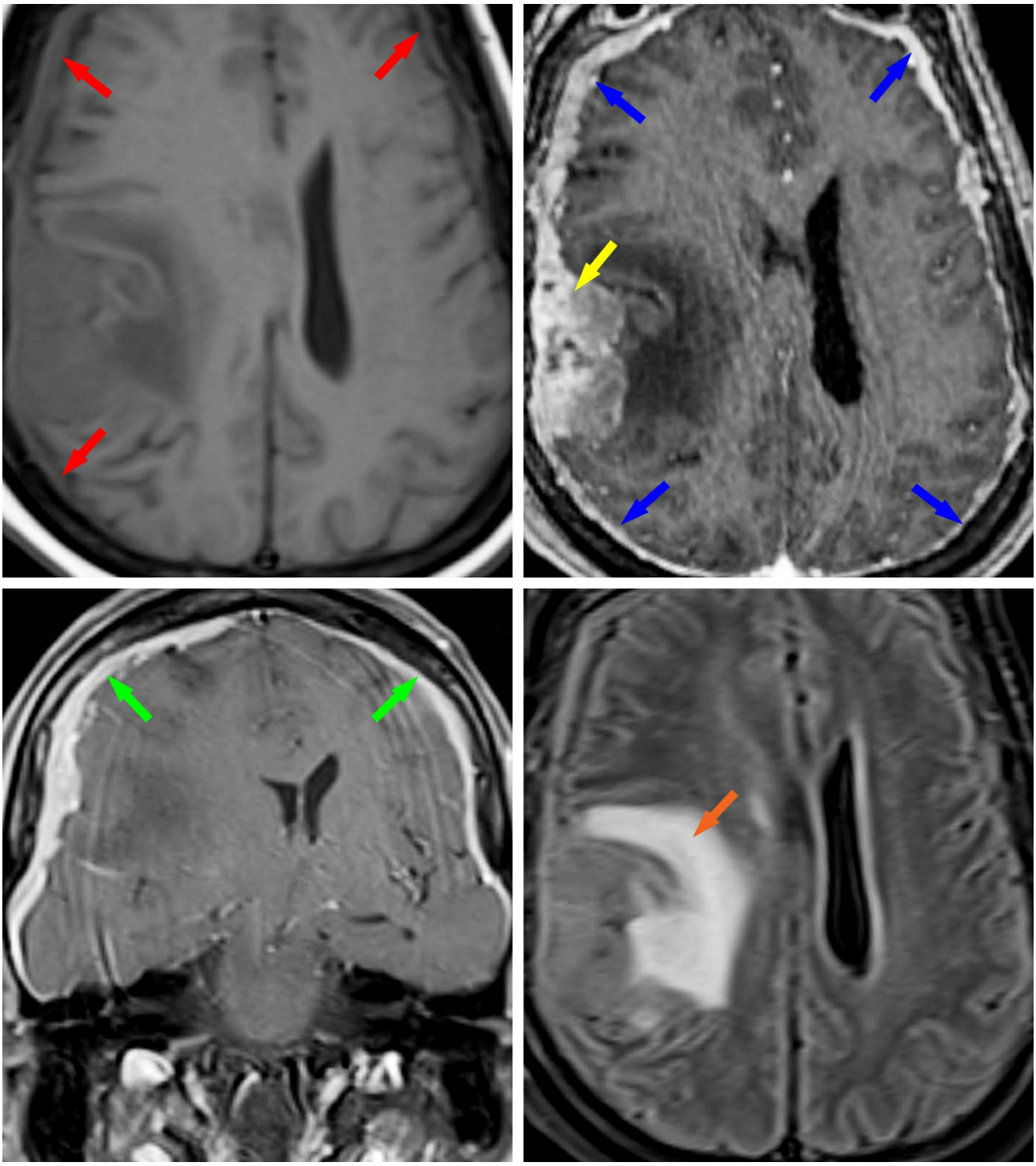


Figure 5: Dural/pachymeningeal carcinomatosis. In this patient with a history of prostate cancer presenting with right facial weakness and left leg numbness, there is diffuse pachymeningeal carcinomatosis. There is extensive extra-axial thickening within the supratentorial compartment, which is intermediate in signal on T1 (top left, red arrows) and avidly enhancing on axial (top right, blue arrows) and coronal (bottom left, green arrows) postcontrast images. (Top Right) More mass-like enhancing metastatic dural thickening also protrudes into the right hemisphere (yellow arrow). (Bottom Right) Axial FLAIR images more clearly demonstrate the extent of surrounding hyperintense edema in the

cerebral parenchyma adjacent to the larger dural metastatic lesion (orange arrow).

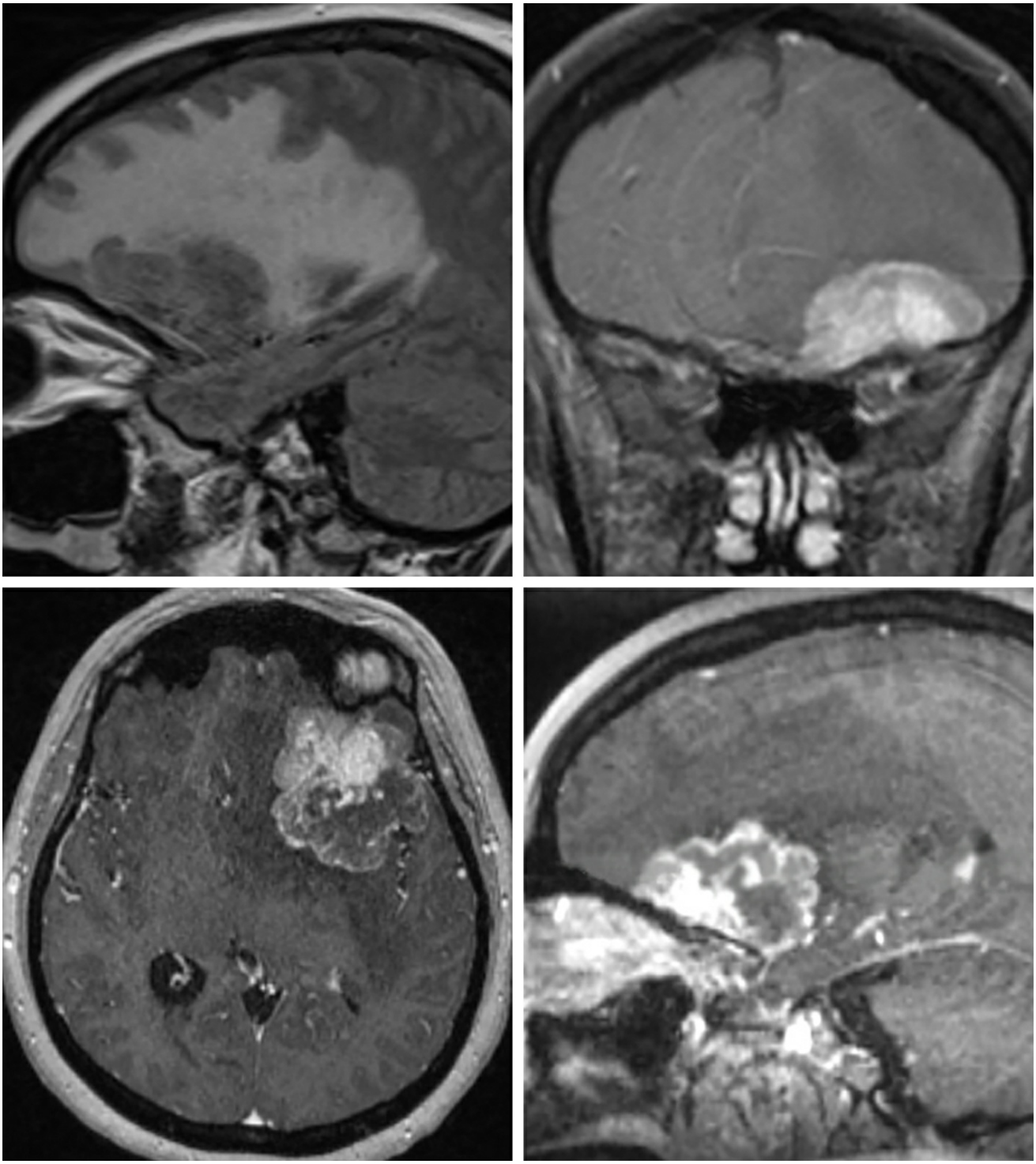


Figure 6: (Top Left) This dura-based metastasis along the inferior aspect of the left frontal lobe is causing a tremendous amount of hyperintense edema on this sagittal FLAIR image. Coronal (top right), axial (bottom left), and sagittal (bottom right) postcontrast T1-weighted images show this lesion to have bright enhancement and a broad dural base, a finding more typically seen with meningioma, a common mimic of metastatic disease.

Pathology

- Typically occurs in setting of direct extension from adjacent calvarial metastases
- Additional proposed mechanisms include retrograde seeding through valveless vertebral venous plexus, hematogenous dissemination, and lymphatic seeding
- Most common primary sites include breast, prostate, head and neck, lung, and hematologic malignancies
- Dural metastases appear as focal nodules or plaquelike sheets

Clinical Features

- Symptoms
  - Range from asymptomatic, with dural metastases incidentally discovered on imaging, to those with significant neurological symptoms
  - Patients with symptoms typically present with headaches, cranial neuropathy, seizures, altered mentation, and/or visual disturbances
- Prognosis
  - Presence of dural metastatic disease forebodes a poor prognosis with most patients eventually demonstrating disease progression
  - Disease progression typically manifests as local dural progression, distant dural spread, and leptomeningeal or intraparenchymal extension

Imaging

- General
  - Dural metastases involve dura-arachnoid immediately subjacent to the calvarium
  - May occur from direct extension from an adjacent calvarial metastasis or hematogenous spread

    - Presence of single dura-based metastasis usually due to extension from calvarial metastatic disease
    - Most commonly, hematogenous spread to the dura presents as multiple dura-enhancing lesions
- Modality specific
    - CT
        - Limited assessment for dura-based metastases
        - Bone algorithm helpful for evaluating presence of associated calvarial metastases
    - MRI
        - T1WI
            - Focal nodular or diffuse dural thickening with usually hypointense or isointense signal relative to adjacent cortex
            - Associated calvarial metastases are T1 hypointense, replacing the normal bright marrow fat
        - T2WI/FLAIR
            - Dural metastases have variable T2/FLAIR signal
            - T2/FLAIR hyperintensity of the adjacent brain parenchyma can occur with presence of vasogenic edema secondary to compression, direct invasion, or impaired venous drainage
        - DWI
            - High-cellularity metastases can show restricted diffusion (high DWI/low ADC signal)
        - Contrast
            - Foci of dural thickening demonstrate avid enhancement
            - Enhancement pattern can be smooth, nodular, or a combination of both
- Imaging recommendations
    - Modality of choice depends on the clinical scenario and the acuity of symptoms

    - Noncontrast CT is often performed in emergent situations
    - If nonemergent, MRI without and with intravenous contrast is the preferred imaging modality
- Mimics
    - Meningioma
        - Focal dural metastasis can have an essentially identical appearance to a meningioma on imaging
        - Presence of associated skull metastasis and bony erosive changes and parenchymal lesions suggest metastatic disease
        - Previous imaging very helpful for evaluating stability
    - Diffuse dura-arachnoid thickening
        - Many nonneoplastic processes can result in diffuse pachymeningeal thickening, such as chronic subdural hematomas, intracranial hypotension, autoimmune dural thickening, and infectious meningitis
        - Clinical context is often of critical importance

LEPTOMENINGEAL METASTASES

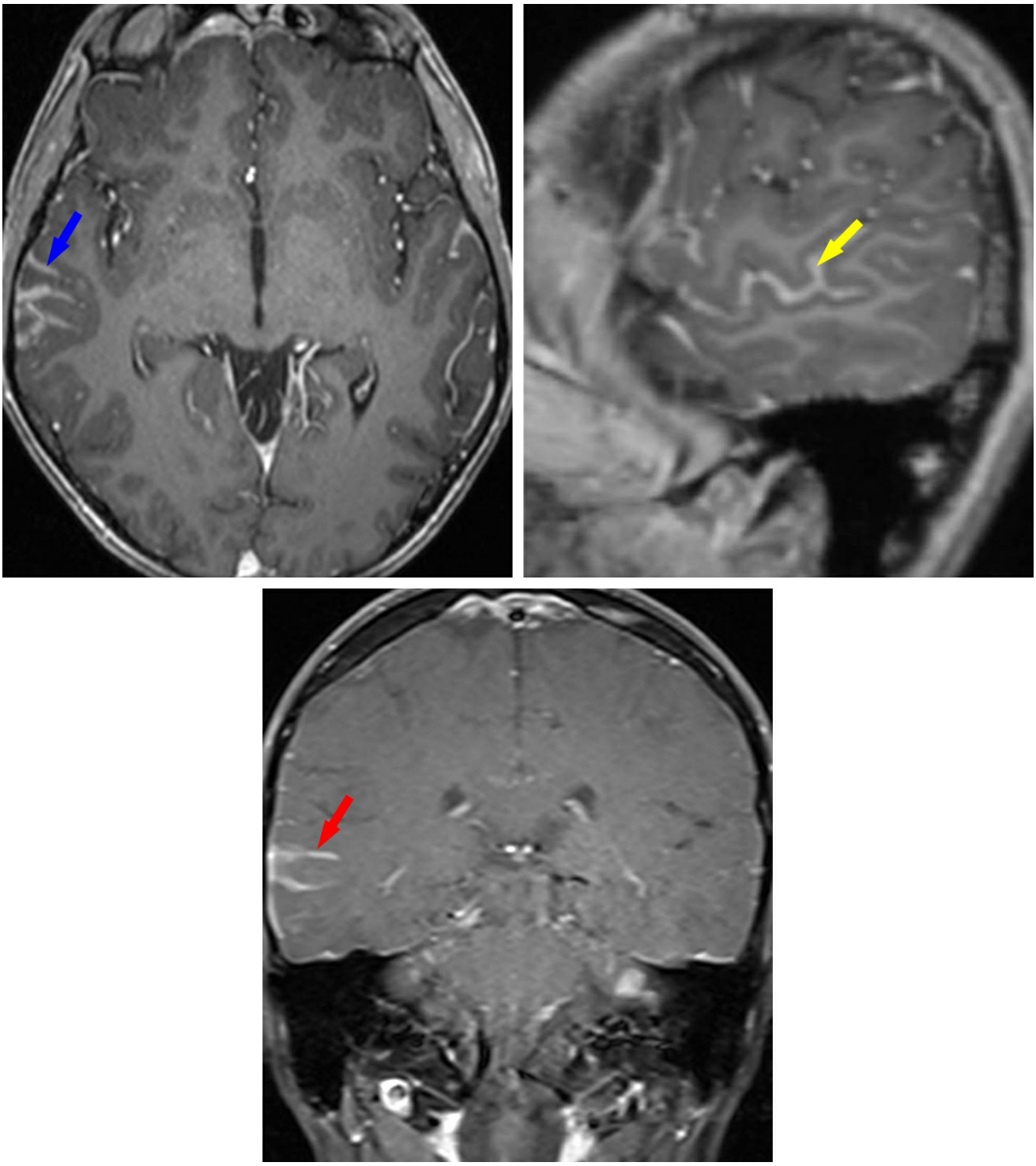
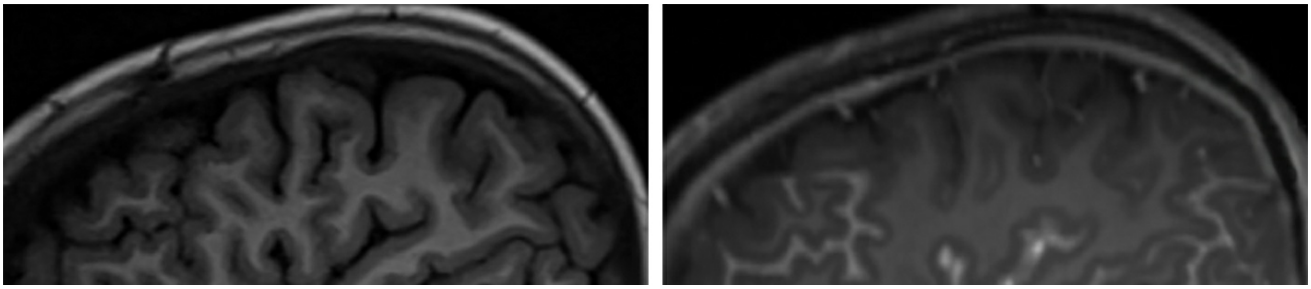


Figure 7: Leptomeningeal metastases. In this pediatric patient with a history of medulloblastoma, there is leptomeningeal thickening and enhancement noted within the right temporal sulci on axial (top left), sagittal (top right), and coronal (bottom) postcontrast T1-weighted images (arrows), compatible with leptomeningeal metastatic disease.



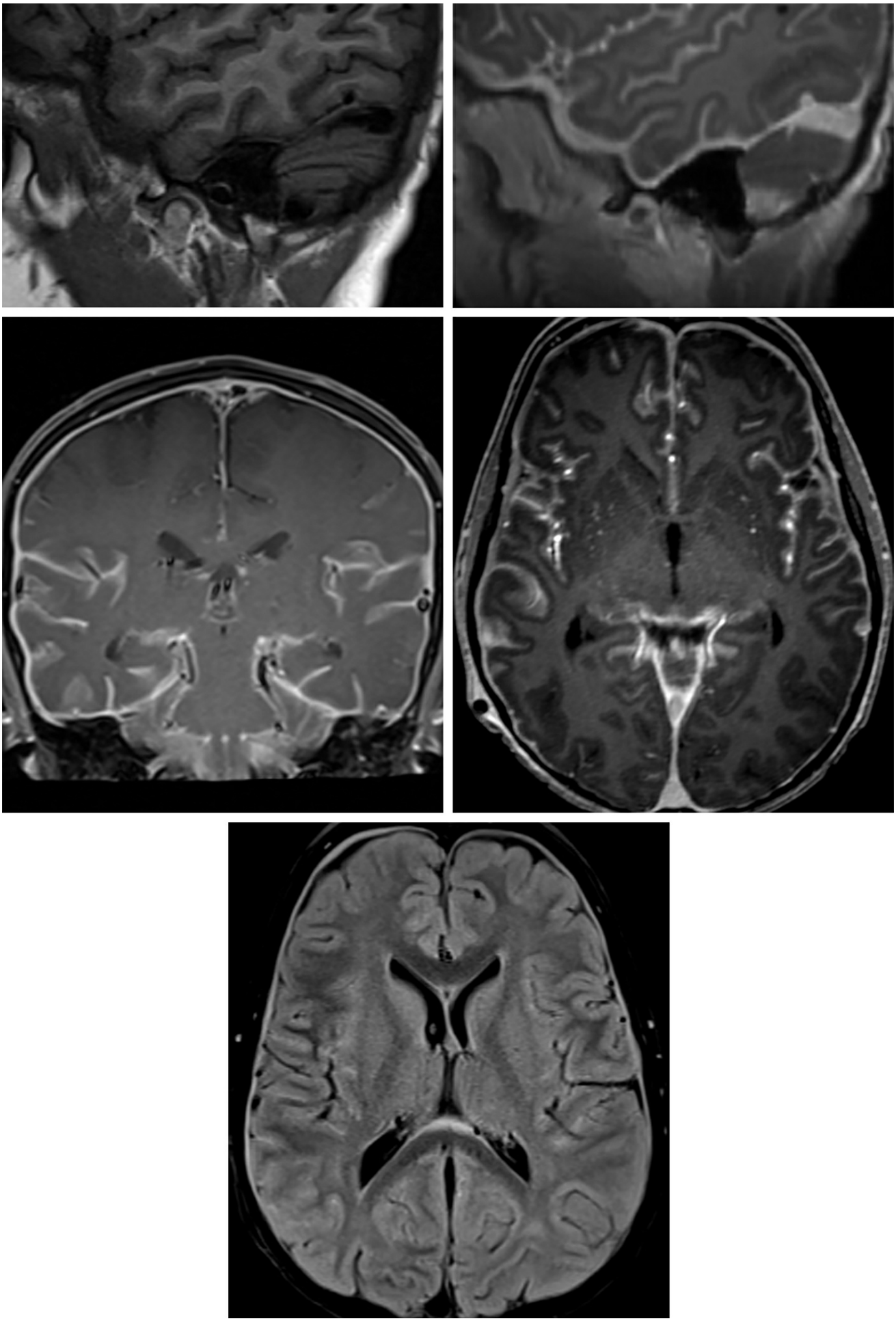


Figure 8: The difference between sagittal T1-weighted precontrast (top left) and postcontrast (top right) images is striking in this patient with

leptomeningeal metastatic disease, with diffuse enhancement throughout the cerebral sulci that is also visible on coronal (middle left) and axial (middle right) contrast-enhanced images. Scattered hyperintense sulci are also visible on FLAIR imaging (bottom), along with reactive or metastatic thickening of the dura. Leptomeningeal metastatic disease is often very nodular but can sometimes be smooth, as in this patient.

Pathology

- Often occur secondary to hematogenous dissemination; breast cancer, lung cancer, and melanoma represent the most common primary tumor types
- Leptomeningeal metastases from intracranial primary tumors
    - In adults, most commonly seen with glioblastoma
    - In children, most commonly seen with medulloblastoma and other embryonal tumors
- Multifocal tumoral deposits coating the pia mater and filling the subarachnoid spaces

Clinical Features

- Symptoms
    - Highly variable depending on particular site of involvement
    - Typical general symptoms include headache, confusion, nausea, and vomiting
    - Cranial and spinal neuropathies are common, with diplopia, facial weakness, and hearing impairment frequent symptoms secondary to involvement of cranial nerves VI, VII, and VIII (abducens, facial, and vestibulocochlear nerves)
- Prognosis
    - Leptomeningeal metastatic disease portends a poor prognosis; average survival, 2 to 4 months

Imaging

- General
  - Leptomeningeal metastases involve the subarachnoid spaces and pial layer along the parenchymal surface following the gyri and sulci
  - Hydrocephalus can be present as a complication of leptomeningeal metastatic debris or secretions obstructing at the level of the arachnoid granulations
- Modality specific
  - CT
    - Often normal but may see hydrocephalus and subtle effacement of the cerebral sulci and cisterns
    - With more pronounced involvement, can see enhancement of the cerebral sulci and cisterns
  - MRI
    - T1WI
      - Often normal, but subtle increased signal in the cerebral sulci might be seen
    - T2WI/FLAIR
      - Leptomeningeal metastases often similar in signal to cerebrospinal fluid (CSF) on T2WI
      - Unlike CSF, leptomeningeal metastases will not lose signal on FLAIR, resulting in increased signal within the sulci and cisterns
    - Contrast
      - Postcontrast T1WI will demonstrate miliary, sheet-like, or nodular enhancement of the leptomeninges resulting in enhancement of the sulci and cisterns
      - Contrast-enhanced T2 FLAIR imaging
        - Highly sensitive for detection of leptomeningeal metastases
        - Demonstrates hyperintense “enhancement” in the involved CSF spaces due to incomplete CSF signal suppression
- Imaging recommendations

    - Modality of choice depends on clinical scenario and acuity of symptoms
    - Noncontrast CT often performed in emergent situations
    - If nonemergent, MRI without and with intravenous contrast is the preferred imaging modality
- Mimics
    - Diffuse leptomeningeal thickening and enhancement
        - Many nonneoplastic processes can demonstrate diffuse leptomeningeal thickening and enhancement, such as infectious meningitis and neurosarcoidosis
        - Clinical context, history, and CSF analysis are critical for distinguishing etiologies, although CSF analysis is often falsely negative for leptomeningeal disease
    - Diffusely increased signal within the subarachnoid spaces on FLAIR; many conditions can contribute to diffusely increased signal within the sulci on FLAIR, mimicking diffuse leptomeningeal metastatic disease
        - Propofol for anesthesia
        - Hyperoxygenation
        - Previous intravenous gadolinium administration
        - Subarachnoid hemorrhage
        - Infectious meningitis
    - Clinical context and history critical for distinguishing etiologies

INTRAVENTRICULAR/CHOROID PLEXUS METASTASES

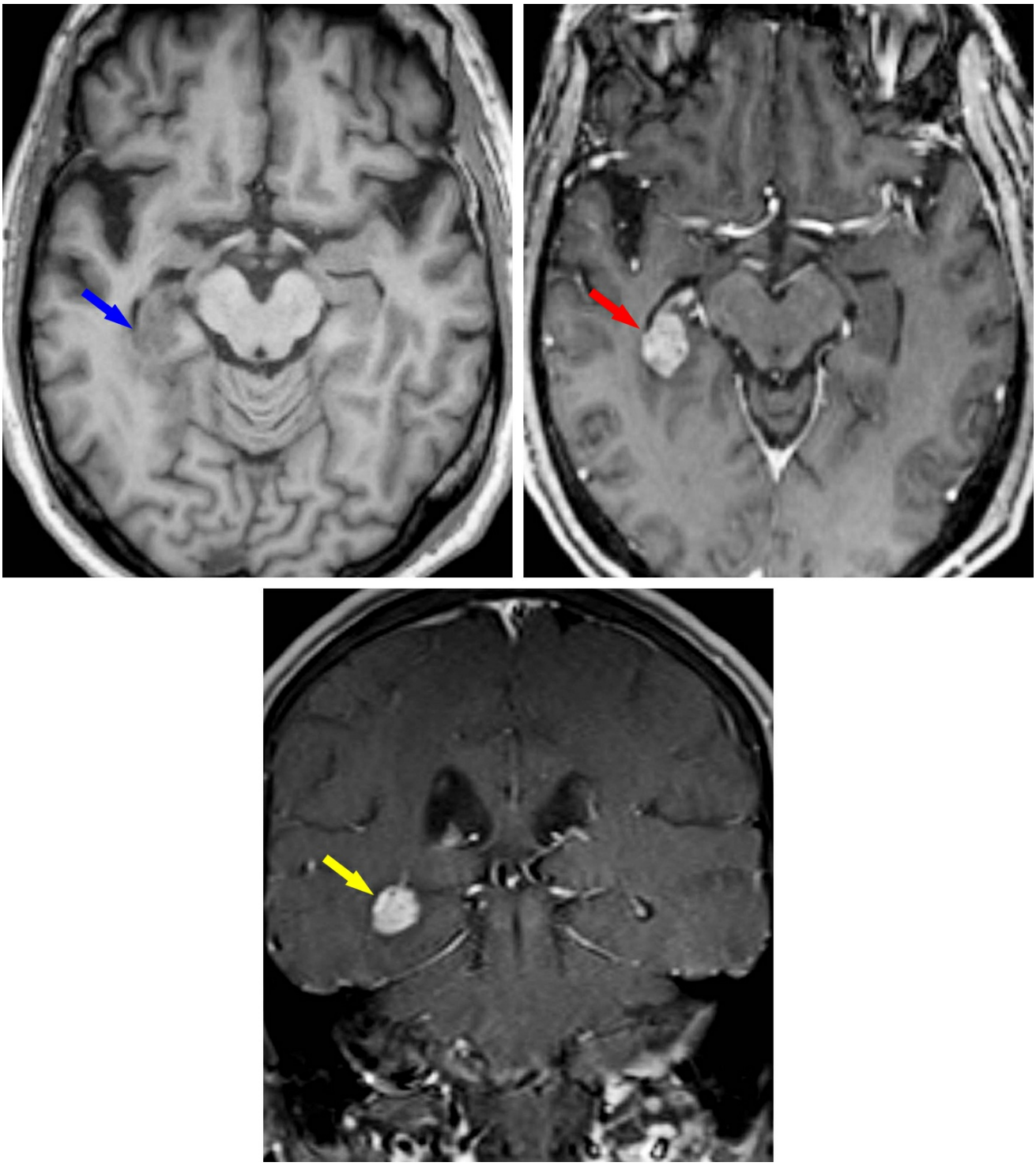


Figure 9: Intraventricular/choroid plexus metastasis. In this patient with a history of metastatic lung cancer, there is evidence of a right choroid plexus metastasis. (Top Left) Axial T1-weighted image without contrast, demonstrating a right choroid plexus lesion with intermediate T1 signal (blue arrow). After the administration of contrast, the lesion is much more clearly visible on axial (top right) and coronal (bottom) images as an avidly enhancing lesion with close association with the temporal horn choroid plexus.

Pathology

- Typically secondary to hematogenous spread to the choroid plexus
- Lung and kidney represent the most common primary sites in adults, whereas neuroblastoma, Wilms tumor, and retinoblastoma are the most common etiologies in children

Clinical Features

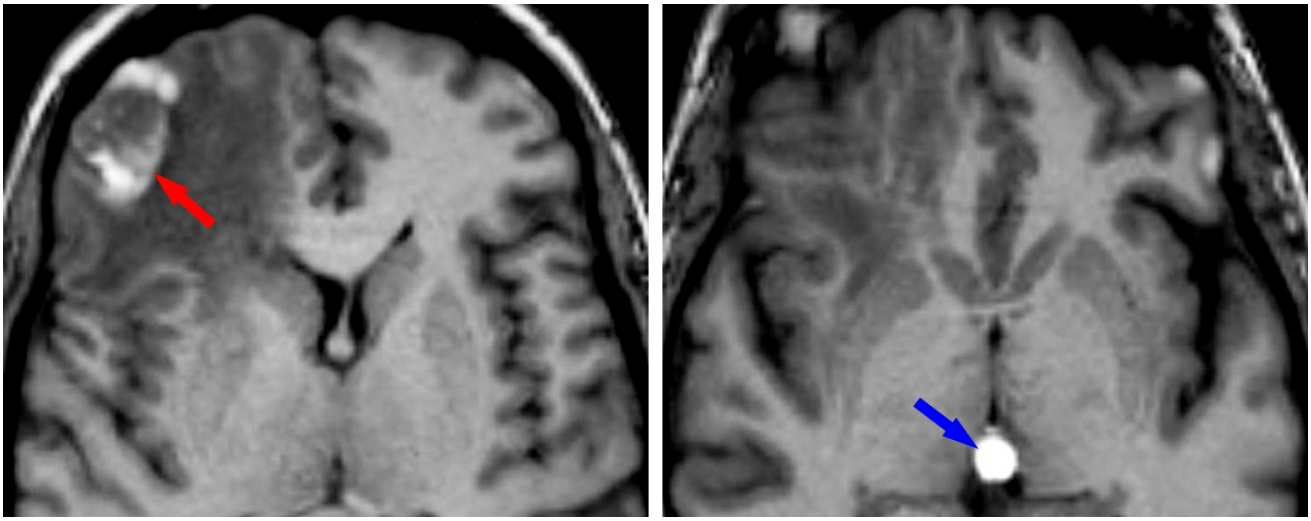
- Intraventricular metastases are relatively uncommon, representing only 0.9% to 4.6% of cerebral metastases
- Symptoms
    - Intraventricular neoplasms in general result in various presentations based on particular location
    - Nausea, vomiting, and headache are more likely in the setting of hydrocephalus and elevated intracranial pressure
- Prognosis
    - Presence of intraventricular metastatic disease generally indicates a poor prognosis

Imaging

- General
    - Mass-like enlargement of the choroid plexus
    - Involvement is most common in the lateral ventricle and second most common in the third ventricle
    - Choroid plexus metastases are often hemorrhagic
- Modality specific
    - CT
        - Isodense to hyperdense enlargement of the choroid plexus
        - Avid, heterogeneous enhancement post-contrast
    - MRI
        - T1WI
            - Usually hypointense on T1WI
            - Variable signal with hemorrhagic metastases

        - T2WI/FLAIR
            - Typically T2/FLAIR hyperintense
            - Might show edema of the adjacent brain parenchyma
        - SWI
            - Hemorrhagic metastases demonstrate hypointense signal on SWI
        - Contrast
            - Avid, heterogenous enhancement
- Imaging recommendations
    - Modality of choice depends on the clinical scenario and on the acuity of symptoms
    - Noncontrast CT is often performed in emergent situations
    - If nonemergent, MRI without and with intravenous contrast is the preferred imaging modality
- Mimics
    - Choroid plexus/intraventricular metastases are often indistinguishable from intraventricular meningiomas and from primary choroid plexus papillomas and carcinomas
    - Age of the patient and presence of a known primary malignancy should influence concern for metastatic disease
    - Previous imaging very helpful for documenting stability of nonmetastatic lesions

PINEAL METASTASES



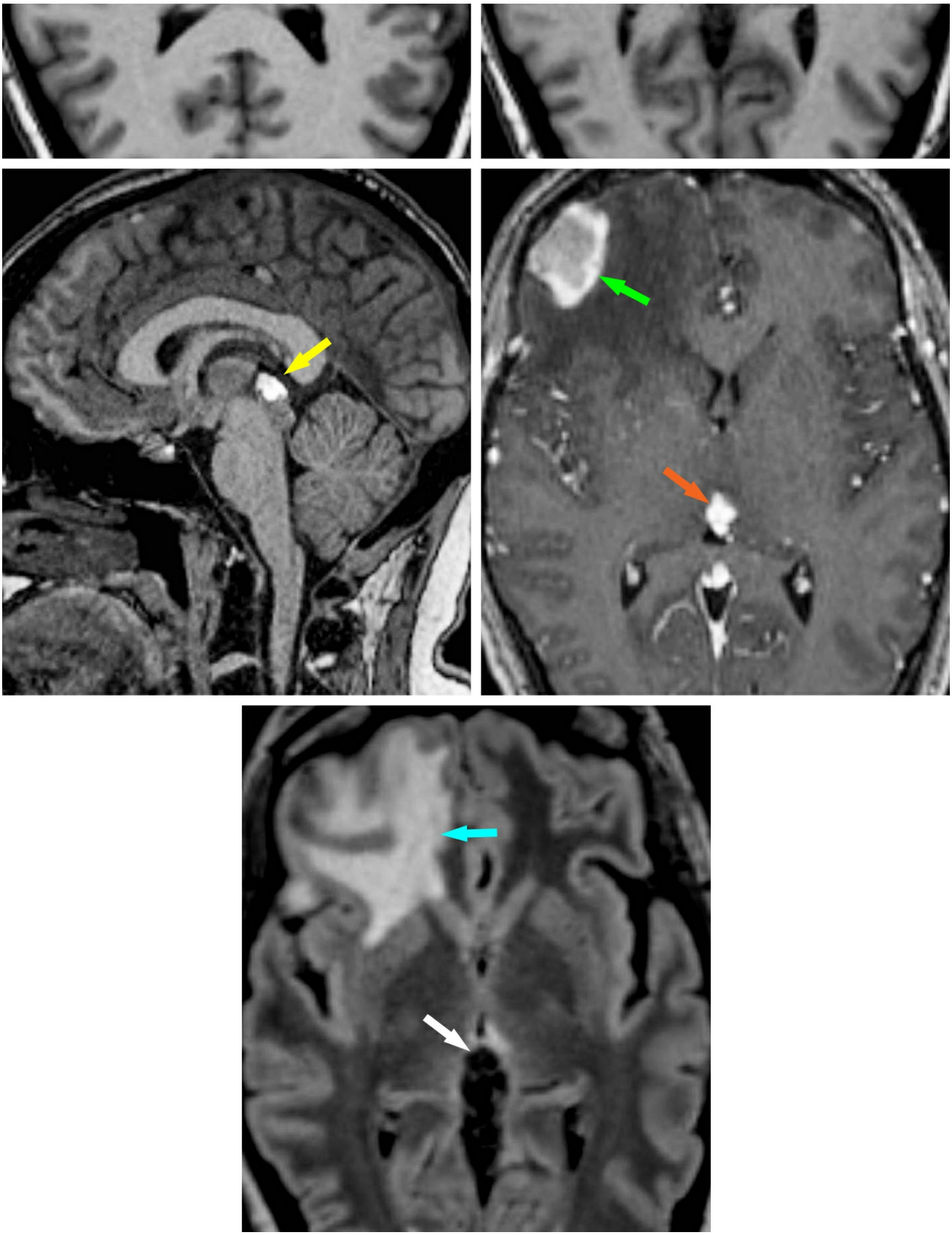


Figure 10: Pineal metastasis. In this patient with a history of melanoma presenting with headache, there is evidence of right frontal parenchymal and pineal metastases. Noncontrast T1-weighted axial images demonstrate the hyperintense right frontal metastasis (top left, red arrow) and the hyperintense pineal metastasis (top right, blue arrow). (Middle Left) The pineal metastasis is also visible on a sagittal T1-

weighted image (yellow arrow). The high T1-weighted signal is a common feature of melanoma metastases. (Middle Right) An axial T1-weighted postcontrast image demonstrates these right frontal parenchymal (green) and pineal (orange) lesions as having additional hyperintense enhancement. (Bottom) On an axial T2 FLAIR image, the pineal lesion demonstrates low signal (white arrow), while the right frontal parenchymal lesion demonstrates characteristic associated subcortical vasogenic edema (teal arrow).

Pathology

- Occurs most commonly from hematogenous spread of extracranial malignancies, with lung (most common), breast, kidney, and gastrointestinal tract representing the most common primary sites

Clinical Features

- Symptoms
    - Pineal neoplasms in general result in symptoms arising from mass effect on nearby structures
    - Compression of the tectal plate can result in Parinaud syndrome (vertical gaze paralysis, pupillary dysfunction, blepharospasm)
    - Obstruction of cerebral aqueduct contributes to hydrocephalus and increased intracranial pressure with headache, nausea, and vomiting

Imaging

- General
    - Mass involving the pineal gland
    - Often solitary without additional intracranial metastases
    - Variable appearance on CT and MR images but likely to demonstrate avid enhancement
    - Associated obstructive hydrocephalus commonly seen

- Imaging recommendations
    - Modality of choice depends on clinical scenario and acuity of symptoms
    - Noncontrast CT often performed in emergent situations
    - If nonemergent, MRI without and with intravenous contrast is the preferred imaging modality to completely characterize the extent of metastatic disease and to monitor for treatment response or progression
- Mimics
    - Pineal metastases having similar imaging characteristics of other primary pineal neoplasms (eg, pineoblastoma or pineocytoma), making clinical history of critical importance

PITUITARY METASTASES

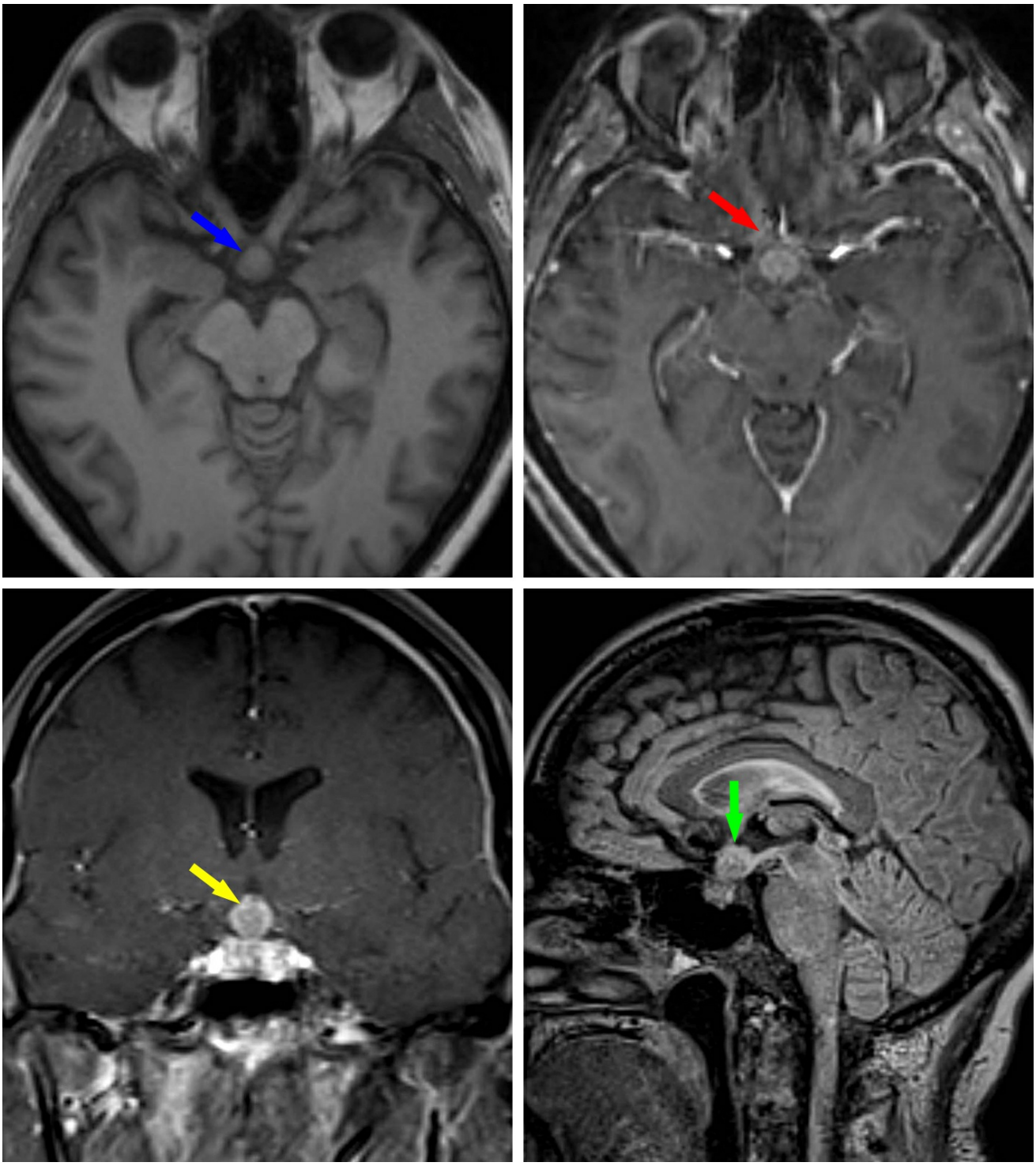


Figure 11: Pituitary infundibulum metastasis. In this patient with a history of breast cancer, there is evidence of a pituitary infundibulum metastasis. (Top Left) Mass-like enlargement of the pituitary infundibulum is noted on precontrast axial T1-weighted imaging (blue arrow). This lesion also demonstrates avid enhancement typical of metastatic disease on postcontrast T1-weighted axial (top right, red arrow) and coronal (bottom left, yellow arrow) images. (Bottom Right) This lesion demonstrates mild FLAIR hyperintensity (green arrow), but a metastatic lesion in this location has less continuity with surrounding parenchyma, which often renders the associated vasogenic edema less conspicuous.

Pathology

- Usually occurs secondary to hematogenous spread of extracranial primary malignancies, with breast and lung representing the most common primary sites

Clinical Features

- Symptoms
    - Patients often present with diabetes insipidus due to predilection for metastases to involve the posterior pituitary lobe and infundibulum
    - Anterior hypopituitarism can also occur, although it is less likely
    - Visual deficits can be seen secondary to compression of the optic chiasm

Imaging

- General
    - Sellar-based mass with variable associated mass effect on adjacent suprasellar or parasellar structures, such as the optic chiasm, and with bony changes
    - Predilection for local invasion and rapid growth
- Modality specific
    - CT
        - May see erosive bony changes of the sella turcica
    - MRI
        - Sella-based mass with usually avid enhancement
        - Can have thickening and enhancement of the infundibulum
        - Often have loss of normal T1 bright spot of the posterior pituitary
        - Can see cavernous sinus invasion

- Imaging recommendations
  - Modality of choice depends on clinical scenario and acuity of symptoms
  - Noncontrast CT often performed in emergent situations
  - If nonemergent, MRI without and with intravenous contrast is the preferred imaging modality
  - Dynamic sella protocol MRI could be helpful
- Mimics
  - Pituitary and infundibular metastases can be difficult to distinguish from other primary neoplastic processes such as a macroadenoma, and nonneoplastic processes such as sarcoidosis and lymphocytic hypophysitis
  - Presence of diabetes insipidus and rapid growth are more suggestive of metastasis
  - Clinical context and presence of known systemic malignancy are of critical importance

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DOI: https://doi.org/10.18791/nsatlas.v1.03.01.29

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