Neuroradiology as a Tool in Neuropathologic Diagnosis of Intracranial Masses

Zoran Rumboldt

Introduction

Basics of CT and MRI

This chapter discusses characteristics of intracranial and intraspinal masses on the primary neuroradiological imaging studies – magnetic resonance imaging (MRI) and computerized tomography (CT). First, a very brief description of these imaging techniques – CT uses x-rays, while MRI shows the magnetic properties of tissues, without ionizing radiation. Both of these techniques operate with digital images – during the actual scanning a huge amount of digital data is being acquired, which is then processed by a powerful computer and converted into images on the scanner. MRI is generally the preferred modality, offering higher contrast resolution between tissues and lesions, and an increased amount of information. CT may offer a more reliable visualization of calcifications and better depiction of osseous morphology. Intravenous contrast agents (iodine-based for CT and gadolinium-based for MRI) are frequently used with both modalities.

CT Terminology

The acquired digital data are converted to images in different ways to make them sharper or smoother (by changing the spatial resolution and noise), and these manipulations are known as algorithms (or filters). The best way to evaluate osseous structures is to use a “bone algorithm,” which offers the highest spatial resolution (and highest noise, which is however of minimal significance thanks to a very high contrast between the bright white bone and everything else). On the other hand, brain is best visualized with a “soft tissue algorithm,” which minimizes noise at the expense of spatial resolution (pixels are combined to improve image quality, as the images would otherwise be extremely grainy). Both of these sets of images are obtained from the same scan.

The images are then sent to other computers (or printed on film), where they are reviewed by radiologists. While viewing the images, windowing is adjusted to best show the pertinent anatomy and pathology. Windowing is somewhat similar to adjusting contrast and brightness on TV or monitor screens, and certain preset combinations are regularly used: “bone window” is best for images reconstructed with bone algorithm, while “brain window” is generally used for visualization of the intracranial structures (Fig. 2.1a, b). In contrast to MRI, CT scans are in the axial plane only, however, high quality reconstructed images in other planes are readily available with modern scanners.

Lesion description on CT uses the terms density or attenuation; compared to the adjacent normal tissue an abnormality may be darker (hypodense, of lower attenuation) or brighter (hyperdense, of increased attenuation). Isodense lesions are of the same brightness as the surrounding structures.

If intravenous contrast is administered, pre- and postcontrast images need to be compared. Enhancement is increased brightness (density, attenuation) of a normal structure (Fig. 2.1c) or a lesion (Fig. 2.2b) on postcontrast images; a nonenhancing abnormality stays the same.

MRI Terminology

Clinical MR imaging is based on protons in the nuclei of hydrogen atoms. The two main sources of signal are water and “fat” (a collective name for long-chained organic molecules containing fat). MRI studies include a number of different sets of images, known as pulse sequences, requiring a separate acquisition of each sequence. The main magnetic properties of tissues are T1 and T2 and so the basic MR sequences are T1-weighted (T1w) and T2-weighted (T2w). Water (cerebrospinal fluid) is dark on T1w (Fig. 2.3a) and very bright on T2w images (Fig. 2.3b). With additional manipulations either water or fat can be suppressed. Standard
Fig. 2.1 Normal brain CT in the axial plane at the level of the pons. (a) Image with soft tissue (brain) filter (algorithm) and brain window, without intravenous contrast (nonenhanced). The bones are white, while the air is black. The CSF is very dark (hypodense), the white matter is brighter, and the gray matter slightly brighter still. The skin and the muscles (arrowheads) are brighter (hyperdense) compared to the brain, while the subcutaneous fat (arrow) is very dark, approaching the appearance of air. (b) Corresponding image with bone filter and bone window. Note that the bones are still very bright, and the air is black, while all the soft tissues are gray with little difference among them. (c) Corresponding contrast enhanced CT image with brain filter, the window is slightly wider (less contrast) than in (a). Note the enhancement (increased brightness) of the vascular structures – middle cerebral arteries (long arrows), basilar artery (short arrow), and choroid plexus (arrowheads).

Fig. 2.2 Brain metastases – contrast enhancement. (a) Axial nonenhanced brain CT at the level of the centrum semiovale. Multiple bilateral slightly darker (hypodense, of decreased attenuation) areas are limited to the white matter, consistent with vasogenic edema (arrows on the larger ones). (b) Corresponding postcontrast CT image shows multiple bright enhancing lesions, mostly within the areas of vasogenic edema. Note that the larger masses show peripheral (rim) enhancement.
brain MRI includes fluid attenuated inversion recovery (FLAIR) images, which is basically a T2w sequence with water suppression (Fig. 2.4a). There are also T2*-weighted images, which are acquired to accentuate artifacts from inhomogeneous magnetic fields, leading to a blooming black out appearance of blood products, calcifications, gas, and metal.

Lesion description terminology is slightly different from CT – brighter is hyperintense (of increased signal), and darker is hypointense (with decreased signal) compared to normal anatomic structures. T1w sequences are also used with intravenous contrast agents. Similar to CT, enhancement is increased brightness on postcontrast images. Frequently used is fat suppression, that eliminates bright signal from fat, for both T2w and postcontrast T1w imaging.

Standard brain MRI also includes diffusion imaging – measuring motion of water molecules. Diffusion-weighted images (DWIs) are a combination of T2-weighting and water diffusion imaging; and water (CSF) is of hypointense signal (Fig. 2.4b). Apparent diffusion coefficient (ADC) maps show water diffusion only and CSF is depicted as very bright (Fig. 2.4c).

MR spectroscopy and MR perfusion are also performed for evaluation of brain masses. These techniques are, however, not widely accepted in routine clinical practice.

**Fig. 2.3** Normal brain MRI in the axial plane at the level of the lateral ventricles and basal ganglia. (a) On T1-weighted image (T1WI) the white matter is brighter than the gray matter and the CSF is very dark (hypointense). (b) On T2-weighted (T2WI) the white matter is darker than the gray matter, and the CSF is very bright (hyperintense). Very dark lines and dots (arrows) are flow voids consistent with vasculature. Fat is bright on both T1WI and T2WI. This is the appearance in adults and children over 2 years of age, the intensity of the gray and white matter is predominantly reversed in neonates and then undergoes continuous changes to reach the adult appearance.

**Distinguishing Imaging Features**

After becoming familiar with the basic terminology, we should proceed to major distinguishing features on neuroimaging studies. Lesion location, primarily intra-axial versus extra-axial is usually clearly evident on imaging studies, resembles gross pathology, and is the first step in evaluation. Helpful signs for extra-axial location are meniscus sign (expansion of the adjacent subarachnoid spaces around the convex mass), inward displacement of subarachnoid vessels (veins), and buckling of the gray–white matter interface. Extra-axial masses commonly also demonstrate a broad dural base (Fig. 2.5), may at times be completely outlined by the CSF, and may cause reactive changes in the adjacent bone. Intra-axial lesions do not expand the subarachnoid space, are surrounded by the brain parenchyma, and may expand the cortex. At times this distinction may be difficult or even impossible, usually when aggressive lesions invade the other compartment (more commonly seen with extra-axial tumors extending into the brain).

The patient’s age, while frequently of substantial importance in the differential diagnosis, will not be mentioned in this chapter. Out of many possible imaging characteristics, only the ones that are helpful for discrimination of intracranial masses will be discussed.
Edema

Many space-occupying disease processes present with edema, seen as darker on CT and T1w MR images and brighter (hyperintense) on T2w and FLAIR images. Two main types can be distinguished: vasogenic and cytotoxic. Vasogenic edema surrounds masses and extends into the white matter, sparing the (cortical) gray matter, and hence having a finger-like appearance (Figs. 2.2 and 2.6). In addition, there is increased diffusion of water molecules in this area, seen as bright signal on ADC maps. In contrast, cytotoxic edema, which is characteristic for infarctions, homogenously involves a well-delineated area including the gray and white matter and extending to the brain surface (Fig. 2.7); the diffusion of water is decreased and infarcts are dark on ADC maps. The term “infiltrative edema” has also been used to describe the pattern frequently present with primary brain tumors – it resembles vasogenic edema, however, the overlying cortical gray matter (and/or the deep gray matter) is also abnormal, at least in some focal areas (Fig. 2.8). Differentiation of edema types is crucial in interpretation of space-occupying lesions.
Prominent vasogenic edema (Figs. 2.2 and 2.6):
- Metastases
- Primary CNS lymphoma (PCNSL)
- High-grade gliomas (infiltrative edema)
- Abscesses
- Granulomas
- Toxoplasmosis

Progressive multifocal leukoencephalopathy (PML) (no to minimal mass effect)
- Absent to minimal edema (Fig. 2.9):
- Dysembryoplastic neuroepithelial tumor (DNET)
- Low-grade gliomas
- Tumefactive demyelination
- Focal cortical dysplasia
Lesions may be well-defined with clear margins on imaging, which is true for many benign disease processes (Figs. 2.7 and 2.9), but also occurs with a number of aggressive neoplasms:

Abscesses
Granulomas
Cysticercosis
Tumefactive demyelination
Infarcts
DNET
Low-grade gliomas
Metastases
PCNSL

**Density/Signal Intensity**

The disease processes can be further differentiated by their internal structure, most notably by the predominant density (CT) or signal intensity (MRI). A number of masses are characteristically bright on CT and not bright (hypointense to isointense to the brain) on T2w MRI (Figs. 2.10 and 2.11): PCNSL
Medulloblastoma
All other small blue cell tumors

Granulomas
Adenocarcinoma metastases
Meningioma

Among other causes, hemorrhage within a lesion also leads to a bright appearance on CT and dark on T2w MRI, as is commonly the case with melanoma metastases (Fig. 2.12). A dark appearance is much more prominent on T2*-weighted images, due to signal loss from blood products (caused by their magnetic properties).

Oligodendrogliomas frequently contain even brighter areas corresponding to calcifications on CT (bone-like), which also may be present with cysticercosis, meningiomas, and vascular malformations.

Infarcts are characteristically dark (hypodense) on CT (Fig. 2.13), which is also a feature of pilocytic astrocytomas (including solid portion) and all cystic lesions.

**Flow-Voids**

Vascular structures known as “flow-voids” are readily visualized on T2w MR images (Fig. 2.14). These are typically present with hemangioblastomas, arterio-venous malformations (AVMs), paragangliomas, hemangiopericytomas, and, in rare cases, with very vascular metastatic tumors.
2 Neuroradiology as a Tool in Neuropathologic Diagnosis of Intracranial Masses

Fig. 2.10 Primary CNS B-cell lymphoma (PCNSBCL) – hyperdense mass. Axial nonenhanced CT image shows an oval bright mass in the left cerebellar hemisphere. There is associated mass effect with displacement of the fourth ventricle (arrow) and hypodense vasogenic edema (arrowheads). Lymphoma and other small blue cell neoplasms are characteristically hyperdense without contrast administration.

Fig. 2.11 Adenocarcinoma metastasis – low T2 signal mass. Axial T2WI shows a somewhat irregular and heterogeneous right cerebellar mass (arrows) that is predominantly darker than the contralateral normal brain parenchyma. Note bright perifocal vasogenic edema (arrowheads).

Fig. 2.12 Melanoma metastases – hemorrhage on T2*-weighted images (T2*WI). Multiple lesions with signal loss (arrows) are noted on T2*WI axial MR image at the level of the pons, consistent with hemorrhagic masses. T2*WI is prone to artifacts and is visually less appealing than T2WI, however, they are extremely sensitive for blood products, which lead to artifactual signal loss primarily due to magnetic properties of iron-containing hemoglobin.

Fig. 2.13 Subacute infarction – hypodense lesion. Axial CT image at the level of the pons reveals a well-demarcated large dark area in the left occipital and mesial temporal lobes that extends to the surface of the brain involving both gray and white matter. This is consistent with infarction in the left posterior cerebral artery territory. Compare to the MRI findings of acute infarction in Fig. 2.7.
Intracranial masses may or may not enhance with intravenous contrast agents and, when present, different types of contrast enhancement may be observed:

- Nonenhancing (sometimes minimally enhancing):
  - DNET
  - PML
  - Focal cortical dysplasia
  - Gliomas (even GBM)

- Smooth peripheral enhancement (Fig. 2.15):
  - Abscesses
  - Metastases
  - Cysticercosis
  - Some granulomas

- Eccentric “target” enhancement (Fig. 2.16):
  - Toxoplasmosis

- Incomplete ring of enhancement (Fig. 2.17):
  - Tumefactive demyelination

- Homogenous solid enhancement (Figs. 2.5b and 2.18):
  - PCNSL (immunocompetent patients)
  - Some granulomas
  - Some metastases (without necrosis)
  - Meningiomas

**Diffusion**

Diffusion MR imaging is now routinely included in brain MR imaging protocols and low ADC value (dark, hypointense to normal brain) consistent with decreased diffusion of water molecules is a very helpful feature of a number of diverse disease processes (Figs. 2.7c and 2.19). These are listed

---

**Fig. 2.14** Arterio-venous malformation (AVM) – flow-voids. Coronal T2WI shows a large cluster of linear and punctate flow-voids in the right temporal lobe (arrows), with the “bag of worms” appearance, consistent with AVM. Compare to the flow-voids of normal vessels in Fig. 2.3b.

**Fig. 2.15** Abscess – smooth peripheral enhancement. Axial contrast-enhanced CT image at the level of the third ventricle and midbrain shows thin and smooth peripheral rounded enhancement (arrows) in the right frontal lobe. The central portion of the mass is darker (of lower attenuation). Note also darker perifocal vasogenic edema posterior to the abscess.

**Fig. 2.16** Toxoplasmosis – eccentric “target” enhancement. Coronal postcontrast T1WI demonstrates two enhancing lesions in the left cerebral hemisphere. In addition to the peripheral enhancement of the masses there is also an internal bright area (arrows) located off center and adjacent to a portion of the enhancing ring.
Neuroradiology as a Tool in Neuropathologic Diagnosis of Intracranial Masses

according to the underlying mechanism responsible for the decreased (restricted) diffusion:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Densely packed cells</td>
<td>PCNSL (solid)</td>
</tr>
<tr>
<td></td>
<td>Medulloblastoma (and other small blue cell tumors)</td>
</tr>
<tr>
<td></td>
<td>High-grade glioma</td>
</tr>
<tr>
<td>Pus</td>
<td>Abscess (necrotic core)</td>
</tr>
<tr>
<td>Acute inflammation</td>
<td>Tumefactive MS (rim)</td>
</tr>
<tr>
<td>Cytotoxic edema</td>
<td>Encephalitis</td>
</tr>
<tr>
<td></td>
<td>Acute infarct</td>
</tr>
</tbody>
</table>

Increased diffusion (bright on ADC maps) is found in a number of tumors, primarily those with a large amount of extracellular space, such as pilocytic astrocytomas (the solid portions), hemangioblastomas, and schwannomas (Fig. 2.20).

**Perfusion**

Perfusion MR (or CT) imaging is becoming progressively more utilized in evaluation of intracranial masses. Infectious and inflammatory disease processes show decrease in cerebral blood volume (CBV) relative to the normal brain (Fig. 2.21), while metastatic tumors, high-grade gliomas and PCNSL have high CBV (Fig. 2.22); low-grade gliomas
Generally range from slightly decreased to slightly increased perfusion.

Typical imaging characteristics of individual intracranial masses are listed in the following chapters and illustrated with corresponding figures. The constellation of features is highly indicative for a corresponding lesion; however, there are exceptions to the rule and not every mass will show all the listed characteristics.

### Intra-axial Supratentorial Masses

#### Low-Grade Astrocytoma (Fig. 2.23)

Well delineated, mild mass effect, no edema, centered in white matter – may involve the gray matter, hypodense on CT, bright on T2w and FLAIR MRI, no contrast enhancement, increased diffusion (bright ADC).

#### Oligodendroglioma (Fig. 2.24)

Involves both gray and white matter, well delineated, mild mass effect, no edema, calcifications common, otherwise hypodense on CT, bright on T2w and FLAIR MRI, contrast enhancement variable, increased diffusion (bright ADC).
Irregular/ill-defined margins, prominent mass effect, infiltrative edema, heterogeneous, multiple lesions possible, predominantly hypodense on CT, bright on T2w and FLAIR MRI, heterogeneous enhancement, areas of decreased diffusion within the lesion. Minimal vasogenic edema is noted anteriorly (arrowheads). (c) Corresponding CBV MR image shows similar perfusion within the tumor (arrows) and the contralateral cerebral hemisphere (dark ADC), high CBV on perfusion imaging [necrotic areas do not enhance, show high diffusion and low CBV].

**Ganglioglioma (Fig. 2.27)**

Cystic component common, temporal lobe, well delineated, mild mass effect, no edema, calcifications possible, otherwise hypodense on CT, bright on T2w and FLAIR MRI, no contrast enhancement, increased diffusion (bright ADC).

**DNET (Fig. 2.28)**

Well delineated, minimal mass effect, no edema, involves gray and white matter, characteristic “bubbly” appearance with multiple small cysts, hypodense on CT, bright on T2w and FLAIR MRI, no contrast enhancement, increased diffusion (bright ADC).

**Central Neurocytoma (Fig. 2.29)**

Multiseptated/polycystic, within lateral ventricles around septum pellucidum.

**PCNSBCL (Figs. 2.10, 2.18, and 2.30)**

Well delineated, homogenous, within white matter, prominent mass effect and edema, multiple lesions possible, hyperdense
Fig. 2.25  “Butterfly” GBM. (a) Axial FLAIR image at the level of the lateral ventricles reveals a lesion centered at the splenium of the corpus callosum involving both cerebral hemispheres. Note the features of infiltrative edema – the margin of the abnormal signal in the white matter is indistinct (arrowheads) and the cortical gray matter is involved (arrows). (b) Postcontrast axial T1WI at a slightly higher level shows heterogeneous enhancement in the central portion of the lesion (arrow) and subtle hazy enhancement (arrowheads) along the more peripheral areas.

Fig. 2.26  Cystic GBM. (a) T2WI in the axial plane shows a large right frontal mass with predominantly very bright internal signal, similar to the CSF, suggestive of fluid. There is finger-like vasogenic edema (arrowheads) spreading into the subcortical white matter posterior to the lesion. The lateral portion of the tumor is of different signal, consistent with abnormal solid tissue, involving the white and gray matter (arrow), indicative of infiltrative edema. (b) Coronal postcontrast T1WI reveals predominantly smooth and thin peripheral contrast enhancement, however, there is much thicker solid enhancement at the lateral aspect of the lesion (arrows), corresponding to the solid tissue, which spreads to the brain surface involving the cortical gray matter. Note displaced right lateral ventricle (arrowhead).
on CT, not bright on T2w MRI, dense homogenous enhancement, low diffusion (dark ADC), increased CBV [may be necrotic/heterogeneous in immunocompromised patients].

**Metastasis (Figs. 2.2, 2.11, 2.12, and 2.31)**

Well delineated, homogenous, or centrally cystic/necrotic, at the cortico-subcortical junction, prominent edema, multiple lesions common, peripheral enhancement (if necrotic) or homogenous enhancement, high diffusion (bright ADC) in the central necrotic portion, increased CBV of the enhancing portion [adenocarcinoma not bright on T2w MRI, hyper-dense on CT].

**Abscess (Figs. 2.6, 2.15, 2.21, and 2.32)**

Well delineated, thin rim of high T1 and low T2 signal, centrally cystic/necrotic, at the cortico-subcortical junction, prominent edema, multiple lesions common, smooth peripheral enhancement, low diffusion (dark ADC) in the central necrotic portion, low CBV.

**Infarct (Figs. 2.7 and 2.13)**

Well delineated, involves gray and white matter with cytotoxic edema, no vasogenic edema, hypodense on CT, bright on T2w and FLAIR MRI, enhancement possible (usually peripheral “gyriform”), low diffusion (dark ADC), low CBV.

---

**Fig. 2.27** Ganglioglioma. Coronal FLAIR image shows a right temporal well-defined lesion (arrow) of central low signal intensity similar to the CSF. There is minimal hyperintensity around this cyst-like mass (arrowhead) and no notable mass effect.

**Fig. 2.28** Dysembryoplastic neuroepithelial tumor (DNET). (a) Axial FLAIR image at the level of the midbrain shows a right temporal lesion (arrows) that is slightly brighter and of heterogeneous “bubbly” appearance. There is no significant mass effect and no surrounding edema. (b) Axial nonenhanced T1WI in a different patient again shows the “bubbly” appearance of the left cerebral hemisphere lesion (arrows), which extends from the brain surface to the lateral ventricle wall.
Tumefactive Demyelinating Lesion
(Figs. 2.9, 2.17, and 2.33)

Well delineated, within white matter, minimal edema, multiple lesions possible, hypodense on CT, centrally bright on T2w and FLAIR MRI, centrally high diffusion and peripheral low diffusion, low CBV.

PML (Fig. 2.34)

Prominent vasogenic edema, absent to minimal mass effect, no focal lesion within the edema, absent to minimal contrast enhancement, multiple lesions possible, increased diffusion (bright ADC), low CBV.

Intra-axial Infratentorial Masses

PA (Figs. 2.20 and 2.35)

Well delineated, hypodense on CT, contrast enhancement, high diffusion (bright ADC) of enhancing/solid portion.
**Fig. 2.31** Metastasis. (a) There are four heterogeneous, round to oval masses (arrows) on this axial T2WI image at the level of the lateral ventricles. Most lesions contain large central areas of very high signal (asterisk), indicative of fluid and/or necrosis. Prominent vasogenic edema is found around large lesions. (b) Corresponding DWI reveals very low signal of the central areas that is similar to the CSF. This is consistent with free motion of water molecules within the necrotic fluid and not to viscous pus.

**Fig. 2.32** Abscess. (a) T2WI in the axial plane at the level of the lateral ventricles shows two masses (arrows) in the right cerebral hemisphere. Both lesions exhibit dark rim and central increased signal, similar to Fig. 2.31a. Also note prominent perifocal vasogenic edema. (b) On the corresponding DWI image both masses are bright “light bulbs,” consistent with a prominent reduction in diffusion of water, as found in dense, viscous pus. Compare to Fig. 2.31b.

**Fig. 2.33** Tumefactive MS. (a) Postcontrast T1WI in the axial plane shows a rim enhancing mass (arrow) in the right centrum semiovale. The central portion of the lesion is of low signal intensity while the enhancing ring is incomplete and absent in its lateral aspect (arrowheads). (b) Corresponding DWI also shows peripheral brightness and central low signal. This constellation of findings is characteristic for tumefactive demyelinating lesions.
Medulloblastoma (Figs. 2.19 and 2.36)

Within fourth ventricle, hyperdense on CT, contrast enhancement, low diffusion (dark ADC).

Ependymoma (Fig. 2.37)

Heterogeneous, calcifications and hemorrhage common, within fourth ventricle, extending through the foramina, contrast enhancement, heterogeneous diffusion.

Hemangioblastoma (Fig. 2.38)

Well delineated, hypodense on CT, prominent flow-voids on T2w MRI, contrast enhancement, high diffusion (bright ADC).

Subependymoma (Fig. 2.39)

Well delineated, within fourth ventricle, absent to minimal enhancement, high diffusion (bright ADC).
Metastasis (Figs. 2.11 and 2.31)

Well delineated, homogenous or centrally cystic/necrotic, prominent edema, multiple lesions common, peripheral enhancement (if necrotic) or homogenous enhancement, high diffusion (bright ADC) in the central necrotic portion, increased CBV of the enhancing portion [adenocarcinoma not bright on T2w MRI, hyperdense on CT].

Extra-axial Supra/Infratentorial Masses

Meningioma (Figs. 2.5 and 2.40)

Well delineated, homogenous, “dural tail,” hyperdense on CT, not bright on T2w MRI, dense homogenous enhancement, underlying bone not invaded [unless intraosseus meningioma – epicenter within the bone, sphenoid wing].
Systemic Lymphoma (Fig. 2.41)

Well delineated, homogenous, hyperdense on CT, not bright on T2w MRI, dense homogenous enhancement, adjacent bone commonly involved.

Extra-axial Metastasis (Fig. 2.42)

Well delineated, heterogeneous/necrotic or homogenous, primary prostate cancer, contrast enhancement, adjacent bone/scalp commonly involved.

Choroid Plexus Papilloma (Fig. 2.43)

Although choroid plexus papilloma is an intra-axial lesion, on imaging studies it typically appears as an intraventricular extra-axial mass.

Multilobular, heterogeneous, calcifications common on CT, hemorrhage possible, within ventricles, flow-voids present on T2w MRI, contrast enhancement.

Schwannoma (Fig. 2.44)

Well delineated, along the cranial nerves (IAC), homogenous when small, heterogeneous and bright on T2w MRI when large, contrast enhancement, high diffusion (bright ADC).

Extra-axial Sella/Skull Base/Pineal Masses

Pituitary Adenoma (Fig. 2.45)

Intrasellar, suprasellar extension common, enhancement less than the normal pituitary gland, cystic, and hemorrhagic changes possible.
**Fig. 2.40** Meningioma. (a) Postcontrast T1WI in the coronal plane shows a large homogenously enhancing bright mass (arrows). The lesion has a broad base along the falx (white arrowheads), which is displaced to the left, consistent with a parafalcine meningioma. Note the normal bright signal of the fatty bone marrow (black arrowheads) in the calvarium adjacent to the tumor and on the other side. (b) Sagittal postcontrast T1WI in a different patient demonstrates a bright homogenously enhancing mass (arrows) with a broad base (arrowhead) along the dura at the anterior aspect of the foramen magnum.

**Fig. 2.41** Systemic lymphoma. Postcontrast coronal T1WI shows a homogenously enhancing dural-based mass (arrows) in the right cerebral hemisphere. This appearance may be indistinguishable from a meningioma. However, the adjacent bone marrow (black arrowheads) has lost its normal bright signal, consistent with infiltration. Note that the contralateral bone marrow has normal brightness (white arrowheads). Compare to Fig. 2.40a.

**Fig. 2.42** Skull base metastasis. Axial CT image with bone filter and window shows a destructive lesion (arrows) at the left skull base. Note the irregular lytic appearance along the lesion margins.
**Fig. 2.43** Choroid plexus papilloma. (a) Nonenhanced axial CT image at the level of the midbrain shows a very bright mass (arrow), similar to bones, within the temporal horn of the left lateral ventricle. Large calcifications as in this case are not common in choroid plexus papillomas. The tip of the temporal horn (arrowhead) is dilated. (b) Sagittal postcontrast T1WI through the lesion (arrow) demonstrates enhancement of the tumor. Again note the dilated temporal horn tip (arrowhead).

**Fig. 2.44** Schwannoma. (a) Axial T2WI at the level of the pons shows a heterogeneous predominantly bright mass (arrows) at the left cerebello-pontine angle. The internal auditory canal (IAC) is expanded by the tumor – compare to the normal right IAC (arrowhead). (b) Postcontrast axial FLAIR image in a different patient demonstrates a large heterogeneously enhancing mass (arrow) at the left cerebello-pontine angle. There is trapped CSF (white arrowhead) adjacent to the lesion, without peripheral contrast enhancement. Note the prominent mass effect with compression and displacement of the fourth ventricle (black arrowhead). FLAIR images may also be acquired (as in this case) following contrast administration, similar to T1WI.

**Parasellar Meningioma (Fig. 2.46)**

Parasellar, commonly centered at cavernous sinus, not bright on T2w MRI, hyperdense on CT, homogenous dense enhancement, dural tail, narrows the internal carotid artery caliber.

**Cavernous Sinus Hemangioma (Fig. 2.47)**

Parasellar, at cavernous sinus, calcifications possible, otherwise hypodense on CT, very bright on T2w MRI, dense contrast enhancement.
Neuroradiology as a Tool in Neuropathologic Diagnosis of Intracranial Masses

Fig. 2.45 Pituitary adenoma. (a) Coronal postcontrast T1WI through the sella shows a large sellar mass with suprasellar extension (arrows), especially on the right. The tumor also extends inferiorly into the sphenoid sinus (asterisk). The lesion is immediately adjacent to the internal carotid arteries (ICAs, white arrowheads). Normal Meckel’s caves (black arrowheads) are also visualized. (b) Coronal postcontrast T1WI through the sella in a different patient reveals a mass (arrows) that is centered at the left side of the pituitary gland and shows absent to minimal enhancement. The tumor extends laterally (white arrow) past the ICA (arrowheads), indicating cavernous sinus invasion.

Parasellar Schwannoma (Fig. 2.48)

Well delineated, along the cranial nerves (Meckel’s cave, “dumbbell shape” common), homogenous when small, heterogeneous and bright on T2w MRI when large, hypodense on CT, contrast enhancement.

Craniopharyngioma (Fig. 2.49)

Suprasellar, calcifications very common on CT, (poly)cystic appearance common, otherwise hypodense on CT and bright on T2w MRI, contrast enhancement of cyst walls is sometimes very subtle.
Cavernous sinus hemangioma (Fig. 2.47)

(a) Coronal postcontrast T1WI through the sella shows a densely enhancing bright mass (arrow), which enhances more than the displaced pituitary gland (arrowhead). (b) Corresponding T2WI image with fat suppression reveals marked brightness of the lesion, approaching the CSF signal – compare to Fig. 2.47b showing much darker meningioma. Also note that the right ICA is displaced but without any narrowing of the lumen.

Parasellar schwannoma (Fig. 2.48)

Axial postcontrast T1WI with fat saturation shows a heterogeneously enhancing mass extending along the course of the left trigeminal nerve, with one portion lying adjacent to the pons (arrowhead) and the other part filling the Meckel’s cave (arrow).

Plasmacytoma/Multiple Myeloma (Fig. 2.50)

Centered at bone, osteolytic on CT, replacement of normal bright bone marrow on T1w MRI, contrast enhancement.

Chordoma (Fig. 2.51)

At midline, arising from clivus,lobulated, and septated, very bright on T2w MRI, enhancement mild to absent, high diffusion (bright ADC).

Chondrosarcoma (Fig. 2.52)

Off midline (petroclival junction), lobulated and septated, very bright on T2w MRI, calcifications common on CT (chondroid matrix pattern), enhancement mild to absent, high diffusion (bright ADC).

Olfactory Neuroblastoma (Fig. 2.53)

At cribriform plate, sinonasal and intracranial extension, cysts common and characteristic, contrast enhancement, dural metastases possible.

Pineoblastoma (Fig. 2.54)

At pineal gland, not bright on T2w MRI, hyperdense on CT with “exploded” scattered pineal calcifications, contrast enhancement, low diffusion (dark ADC).
**Fig. 2.49** Craniopharyngioma. (a) Nonenhanced axial CT image at the level of the suprasellar cisterns shows a very bright right paramedial lesion (*arrow*), consistent with calcification. The vast majority of craniopharyngiomas contain calcifications on CT studies. (b) Sagittal postcontrast T1WI shows thin peripheral increased brightness of the rounded mass (*arrow*), consistent with subtle enhancement. The tumor is suprasellar in location; note the normal enhancing pituitary gland (*arrowhead*) within the sella.

**Fig. 2.50** Plasmacytoma/multiple myeloma. Axial CT image with bone algorithm and window shows a destructive lesion of the clivus (*arrows*). Note absence of mass effect and smooth regular margins of the abnormality. Compare to metastasis in Fig. 2.42.
**Fig. 2.51** Chordoma. (a) T2WI in the axial plane at the level of the clivus and pons shows a bright mass (arrows) arising from the clivus at the midline and to the left. (b) Sagittal postcontrast T1WI reveals only mild and heterogeneous enhancement of the tumor (arrows).

**Fig. 2.52** Chondrosarcoma. (a) Axial CT image with bone filter and window shows bright coarse calcifications (arrows) anterior to and above the right petro-civil junction (arrowhead). (b) Coronal postcontrast T1WI through the sellar region demonstrates markedly heterogeneous enhancement of the mass (arrows) in a linear and punctuate fashion. Note normally enhancing pituitary gland (arrowhead).
Neuroradiology as a Tool in Neuropathologic Diagnosis of Intracranial Masses

Germ Cell Tumor (Fig. 2.55)

At pineal gland, not bright on T2w MRI, hyperdense on CT surrounding preserved pineal calcifications, contrast enhancement, low diffusion (dark ADC).

Spinal Masses

Spinal masses are best differentiated on imaging studies based on their location and patient’s age. The primary division is into extramedullary and intramedullary lesions. Schwannomas, meningiomas, and myxopapillary ependymomas are the most common extramedullary neoplasms, while ependymomas, astrocytomas, and hemangioblastomas are located within the spinal cord.

Schwannoma (Fig. 2.56)

Along nerves (neural foramina, cauda equina), oval, well delineated, bright on T2w MRI, contrast enhancement.

Fig. 2.53 Olfactory neuroblastoma (Esthesioneuroblastoma). Postcontrast T1WI in the sagittal plane reveals a large enhancing mass (arrows) with intracranial and extracranial components. The lesion extends from the nasal cavity and nasopharynx to the frontal lobe, with the epicenter and waist at the level of the cribriform plate. The intracranial portion of the tumor contains a few rounded nonenhancing areas (arrowheads), consistent with cysts, which are highly suggestive of an olfactory neuroblastoma.

Fig. 2.54 Pineoblastoma. (a) Midsagittal T1WI following contrast administration shows a large enhancing mass (arrows) in the pineal region. (b) Axial nonenhanced CT image through the lesion (arrow) reveals its mild hyperdensity. The normal pineal calcifications (arrowheads) are separated, “exploded” by the tumor. This is a typical appearance of a pineoblastoma.
**Fig. 2.55** Germ cell tumor. (a) Axial nonenhanced CT image at the level of the pineal gland and basal ganglia shows an isodense mass (arrow) surrounding intact pineal calcification (arrowhead), indicative of a germ cell tumor. Compare to Fig. 2.54b. (b) Following treatment the enhancing tumor recurred in the parasellar location (arrow), as seen on this sagittal postcontrast T1WI. Note the absence of lesions in the pineal region, which is filled with CSF (arrowhead).

**Fig. 2.56** Spinal schwannoma. (a) Postcontrast T1WI in the axial plane through the lower cervical spine shows an enhancing mass (black asterisk) that is expanding the left neural foramen (arrows). The spinal cord (white asterisk) is mildly displaced to the right by the tumor. (b) Axial T2WI through the lower thoracic spine in a different patient demonstrates a bright mass (arrows) within the spinal canal on the right side, which displaces the spinal cord (asterisk) to the left. (c) Corresponding T1WI with fat saturation reveals bright slightly heterogeneous enhancement of the lesion (arrow).
Myxopapillary Ependymoma (Fig. 2.57)

At/adjacent to conus medullaris, lobular when large, bright on T2w MRI, contrast enhancement [when small at cauda equine may not be distinguished from Schwannoma].

Meningioma (Fig. 2.58)

Dural-based, “en plaque” appearance, not bright on T2w MRI, dense homogenous enhancement.

Ependymoma (Fig. 2.59)

Central intramedullary, peritumoral cysts possible, hemorrhage possible, contrast enhancement variable (absent to prominent), occurs in children only with NF-2, multiple lesions common with NF-2.

Astrocytoma (Fig. 2.60)

Eccentric intramedullary, enhancement variable, pediatric population.

Hemangioblastoma (Fig. 2.61)

Along spinal cord surface, contrast enhancement, multiple lesions possible [with VHL].

Fig. 2.57 Myxopapillary ependymoma. Sagittal T2WI through the thoraco-lumbar region shows a heterogeneous predominantly bright mass (arrows), which is located just inferior to the conus medullaris. There is also a small amount of bright edema (arrowhead) within the conus. This is a typical location of myxopapillary ependymoma.

Fig. 2.58 Spinal meningioma. (a) Sagittal T2WI through the cervical and upper thoracic spine shows an extramedullary mass with a broad base along the ventral dura (arrows) – compare to Fig. 2.40b. The lesion is of similar signal as the spinal cord, which is displaced. (b) Postcontrast axial T1WI through the tumor (asterisk) shows its homogenous enhancement. The mass occupies most of the spinal canal at this level.
**References**


Intra-Operative Neuropathology for the Non-Neuropathologist
A Case-Based Approach
Welsh, C.T. (Ed.)
2012, IX, 170 p., Hardcover
ISBN: 978-1-4419-1166-7