Patterns of contrast enhancement in the brain and meninges. A Pictorial Review.

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Learning objectives

- To describe patterns and mechanisms of contrast enhancement in the brain and meninges.
- To review and depict the brain and meninges disorders related to contrast material.
- To focus a differential diagnosis depending on the pattern of enhancement.
- To emphasize imaging key features which can help in the main differentials.

Background

1. Contrast material enhancement in the central nervous system: intavascular and interstitial enhancement, with functional drawing schemes and radiological correspondences.

2. Meningeal enhancement: pachymeningeal and leptomeningeal based on pathologic conditions. primary neoplasms, granulomatous disease, metastases, meningitis and meningoencephalitis.

3. Intraaxial enhancement: gyral, nodular cortical and subcortical, and ring enhancement.

Imaging findings OR Procedure details

INTRODUCTION

Contrast material enhancement for cross-sectional imaging has been used since the 1970s for computed tomography and the 1980s for magnetic resonance imaging. Knowledge of the patterns and mechanisms of contrast enhancement facilitate radiologic differential diagnosis.

There are many tools for analyzing magnetic resonance (MR) or computed tomography (CT) images to produce differential diagnosis.

Contrast material enhancement in the central nervous system (CNS) is a combination of two primary processes. fig.1 on page 30
1. **Vascular enhancement (intravascular):** The blood-brain barrier is a selectively semipermeable capillary membrane that blocks lipophobic compounds (creates a unique interstitial fluid environment for the neural tissues), and may cross the blood-brain barrier. **Refers to abnormal retention of contrast medium in the brain vessels and is proportional to increases in blood flow.** May reflect: neovascularity, vasodilatation-hyperemia and shortened transit time or shunting. Is commonly seen in the infarcted territory during the first week. Reflect leptomeningeal inflammation, encephalitis, or perivenular compromise of the blood-brain-barrier with alterations in cerebellar blood flow. Rapid dynamic CT images shows intravascular enhancement.

2. **Interstitial enhancement (extravascular):** Is related to alterations in the permeability of the blood-brain-barrier. Delayed 10-15 minutes contrast enhancement CT images shows interstitial enhancement. Examples of interstitial enhancement include leakage of fluid into the interstitium from low-grade fluid secreting neoplasms (hemangioblastoma, ganglioglioma, pleomorphic xanthoastrocytoma) and venous perivascular inflammation in demyelation from multiple sclerosis (MS). Abscess enhancement results from both increased vascularity and abnormal permeability.

**Extraaxial enhancement** fig. 2 on page 31, fig.3 on page 10

- Pachymeningeal- dura-arachnoid enhancement.
- Leptomeningeal- pia-arachnoid enhancement.

**Intraaxial enhancement**

- Gyral - Necrotic
- Nodular: cortical and subcortical - Fluid-secreting
- Deep Ring - Demyelination
- Cerebritis and abscess - Periventricular

**Extraaxial enhancement**

- Pachymeningeal- dura-arachnoid enhancement.
- Leptomeningeal- pia-arachnoid enhancement.
The meninges are developed from a precursor layer known as the meninx primitive. The meninges comprise the dura and the underlying pia-arachnoid. fig.4 on page 11

- **The dura, consists of two layers**: an outer layer: endosteal layer, is directly apposed to the inner table of the skull and forms the periosteum of the inner table. The outer layer is tightly fused for most of its length to an inner layer, or meningeal layer. These inner layers join to form the falx and tentonium. Between the inner layer of the dura and the arachnoid is the subdural space, which contains a thin film of fluid. Between the arachnoid and the pia is the subarachnoid space, filled with CSF. **The arachnoid crosses over the sulci.** The pia is closely applied to the surface of the brain and extends into the deepest sulci. **The arachnoid and pia together are called the leptomeninges.** In normal meningeal enhancement, enhancement of the meninges is visualized as a thin, markedly discontinuous rim covering the surface of the brain. It is seen primarily in the dura and venous structures. The arachnoid is thin and avascular.

MR imaging is substantially more sensitive than CT for visualization of both the normal and abnormal meninges.

The appearance of the meninges on MR images is dependent on numerous factors: amount of contrast and concentration in the meninges at the time of imaging, the type of pulse sequence performed, and the specifics of the selected pulse sequence parameters. Short repetition time (TR) and echo time (TE), large flip angle (gradient echo), coronal images are preferable, use of high dose (0.3 mmol/kg) intravenous gadopentetate dimeglumine, and fat saturation. Fluid attenuated inversion recovery (FLAIR) is useful in the settings of subarachnoid pathology, Gradient echo (GRE) is useful for blood products, iron and calcium. **Multiplanar contrast-enhanced T1 axial and coronal sequences are also very helpful.**

fig. 5 on page 12

**Pachymeningeal- dura-arachnoid enhancement** fig.6 on page 13

The vessels within the dura mater do not produce a blood-brain-barrier.

- On CT is well seen normal dural enhancement in the dural reflections of the falx cerebri, tentorium and falx cerebelli.

- On T1-weighted MR images: normal duramater and inner table bone are uniformly hypointense. After contrast normal dura shows thin, linear, and discontinuos enhancement.

**CAUSES: fig.7 on page 14**

1. **Postoperative changes.** It typically lasts 3-4 weeks on CT and can persist up to 1 year on MR. Less 5% after uncomplicated lumbar puncture.
2. **Intracranial hypotension**: benign cause that may be localized or diffuse.

After surgery, lumbar puncture, skull fracture with leakage of cerebrospinal fluid or with idiopathic loss of cerebrospinal fluid pressure. Vasocongestion and interstitial edema in the dura mater. Is characterized by a postural headache that responds to treatment with an epidural blood patch. **fig.8** on page 25

**MR findings- Intracranial hypotension**

- Thick linear enhancement of the pachymeninges.
- No enhancement of the sulci.
- Enhancement above and below the tentorium.
- Enlargement of the pituitary gland.
- Descent of the brain.
- Subdural effusions or hemorrhage.

3. **Neoplasms**-meningioma. The typical meningioma is a localized lesion with broad base of dural attachment. Arises from the arachnoid membrane that is attached to the inner layer of the dura mater. **fig. 9** on page 26, **fig.10.** on page 27

**MR findings- Meningioma**

- *Dural Tail.* Is a curvilinear region of dural enhancement adjacent to the bulky hemispheric tumor, seen in 2 consecutive 5 mm sections.
- > 1 cm away from the tumor is caused by a reactive process, dural capillaries are nonneural, and they do not form a blood-brain-barrier and enhancement occurs.

4. **Metastasic disease**: breast, lung, prostate cancer, malignant melanoma. **fig. 12** on page 29

5. **Secondary CNS lymphoma**: usually extraaxial.

6. **Granulomatous disease**: sarcoid, tuberculosis, Wegener granulomatous, luetic gumas, rheumatoid nodules, fungal disease.

**MR findings- Granulomatous disease**

- *Dural masses*: in tuberculosis (isointense to brain parenchyma on T1 weighted-imaging (WI), iso to hyperintense on T2 weighted-imaging (WI) and hyperdense on unenhanced CT). In Sarcoidosis (isointense to cortex on T1w, hypointense on T2 WI and enhance homogeneously with similar appearance to calcified meningiomas), 10% of patient with systemic sarcoidosis have neurological symptoms. **fig.11** on page 28
- *Basilar meninges* > convexities of the cerebral hemispheres.
Leptomeningal- pia-arachnoid enhancement fig.13 on page 32

Enhancement of the sulci and cisterns is leptomeningeal enhancement.

Breakdown of the blood-brain-barrier without angiogenesis. Allows contrast material to leak from vessels into cerebrospinal fluid.

Mechanisms: Hematogenous spread, direct extension.

CAUSES:

1. Neoplasms: "carcinomatous meningitis": spread into the subarachnoid space. fig. 14 on page 33, fig. 15 on page 34


   Medulloblastoma: are the most common nervous system malignancy in children. Metastatic spread of medulloblastoma via drop metastasis which occurs in approximately 40% of cases. Spread is via CSF pathway to the spinal cord, cerebral convexities, sylvian fissure, suprasellar cistern, and into lateral and 3rd ventricle. Subarachnoid metastasis and "drop mets", "frosting" of tumor on pia.

2. Meningitis: - Bacterial and viral: thin and linear enhancement. Infiltration with inflammatory cells, and increase of the permeability in the meninges.

   - Viral encephalitis: enhancement along the cranial nerves and brain surface.

   - Fungal: thicker, nodular enhancement in the subarachnoid space.

3. Superficial siderosis: is caused by the deposition of hemosiderin along the leptomeninges, subpial, and subependymal tissues. Classically affected sites are the brainstem and cerebellar vermis. It is the result of chronic repeated subarachnoid hemorrhage. GRE is very sensitive MR sequence for hemosiderin, due to the blooming effect.

Intraaxial enhancement

   - Gyral - Necrotic

   - Nodular: cortical and subcortical - Fluid-secreting
- Deep Ring - Demyelination

- Cerebritis and abscess - Periventricular

**Gyral** (serpentine)

1. **Vascular**: fig. 16 on page 24, fig. 17 on page 23
   - Acute symptoms.
   - +freq. Single artery territory. Middle cerebral artery territory 60%, posterior cerebral artery territory in posterior reversible encephalopathy (PRES).
   - Mechanisms: **variable time courses**. Earliest enhancement: reversible blood-brain-barrier changes. Early reperfusion: vasodilatation, dynamic changes "luxury perfusion". Increases blood flow caused by autoregulation mechanisms. Healing phases: from 5-7 days to several weeks after the event there will be vascular proliferation. After 4 months the enhancement is replaced by encephalomalacia.

   - **Postictal states**: may mimic cerebral infarction: gyral swelling and enhancement, sulcal effacement, increased signal intensity on T2WI, decreased signal on T1WI.

2. **Inflammatory**: encephalitis, meningitis.
   - Indolent history, headache, lethargy.
   - Multiple territories.

   - Herpes virus encephalitis: superficial gray matter disease. Begin: medial temporal lobes (uncus), and cingulate gyrus of the medial frontal and parietal lobes.

**Nodular**: cortical and subcortical

- Typical: **hematogenous dissemination of metastasic** neoplasms and clot emboli. fig.18 on page 15

- 40-60% CT-MR solitary metastatic lesion. Increasing dose of contrast agent, delayed imaging may reveal additional metastatic lesions.

- Circumscribed lesions near the gray matter-white matter junction, subcortical (primary tumors usually deeper).

- Small (<2cm). First identified while they are solid nodular lesions 0.5-2.5 cm, in contrast with primary glial tumors.
- Artery pathway: + freq. majority supratentorial: middle cerebral artery.

- Venous pathway: primary pelvic malignancy and travels through the prevertebral veins of the Batson venous plexus to posterior fossa.

- Angiogenesis allows the metastases to grow larger than 5 mm, and blood-brain-barrier abnormality which results in contrast enhancement and perilesional vasogenic edema.

**Deep Ring**

Contrast enhancement of ring-enhancing lesions results from either or both of two processes: intravascular (vascular) and extravascular (interstitial) enhancement. The breakdown of the blood-brain-barrier results in interstitial enhancement while vascular enhancement relies on increases in blood flow or blood volume.

Ring-enhancement of necrotic neoplasms from neovascularity as well as luxury perfusion from increased circulation through an area of infarcted brain are forms of vascular enhancement.

The useful pneumonic **MAGICAL DR** is commonly used for the differential diagnosis ring-enhancing lesions of the brain.

**M** Metastasis: cortical and subcortical lesions with cavitation. Solid nodular lesions that may become ring-enhancing because of necrosis.

**A** Abscess: cortical and subcortical lesions with cavitation. Deep white matter ring enhancing lesions, mass effect, surrounding vasogenic edema.

**G** Glioma: deep white matter ring enhancing lesions, mass effect, surrounding vasogenic edema

Granuloma.

**I** Infarct/Infection: multiple cortical and subcortical. (subacute bacterial endocarditis, indwelling catheters…)

**C** Contusion.

**A** AIDS.

**L** Lymphoma.

**D** Demyelinating disease.

**R** Radiation necrosis/Resolving hematoma.
Cerebritis and abscess

-Causes: septic emboli transmitted hematogenously, transdural spread from sinus infections.

- Formation:

1. Early stage: cerebritis: acute inflammatory reaction with altered permeability of the native vessels, without angiogenesis. Minimal vasogenic edema. In immunocompetent patient cerebritis progresses to form an organized abscess.

MR: Ring-enhancing lesion. Images obtained over 20-40 minutes show "fill in" of the ring center.

2. Abscess: Typically hypointense rim on T2WI, and high signal intensity on diffusion-weighted images because of the viscosity of the pus and liquefaction necrosis organization and partial neutralization of an infection. Well-formed capsule 2-4 weeks. Ring enhancement reflects the granulation tissue in its wall that has both increased vascularity and collagen deposition. The rim is thin (2-7 mm), uniformly convex, smooth on both the outer and inner aspects. Thinner margin of the deep or medial aspect that predisposes to daughter abscess formation and deep rupture into the ventricle. The organizing infection forms concentric layers. fig.19 on page 16, fig.20 on page 17, fig. 21 on page 18

Necrotic

-Necrotic neoplasms usually malignant; primary or metastatic.

-Neovascularity is prominent in high-grade-tumors.

- Thick irregular ring (contains the greatest concentration of neovascularity) with a shaggy inner margin, multilocular and complex ring patterns, wall thicker than 10 mm.

-Delayed images: progressive enhancement inside the rim, patchy pattern, reflects the presence of islands of surviving tumor cells within regions of necrosis. fig.22 on page 35, fig.23 on page 19

Fluid-secreting

-Fluid-secreting primary neoplasms are typically well marginated and usually low histologic grade.
-Incomplete ring of enhancement (part is nonneoplastic: compressed or gliotic brain tissue). Thin (<2cm).

-"Cyst-with-nodule".

-Infratentorial: Familiar pilocytic astrocytoma, hemangioblastoma (more complex pattern).

-Supratentorial: pilocytic astrocytoma fig.24 on page 20(most common cyst-with-nodule neoplasm), pleomorphic xanthoastrocytoma, ganglioglioma…

**Demyelination**

-Most common cause: multiple sclerosis.

-Multiple sclerosis plaques enhance during: "active phase" due to inflammation, no neovascularity, no angiogenesis, no necrosis. No vasogenic edema.

-2-6 weeks, **incomplete enhancing rim**.

-Additional lesions of the spinal cord help to support the diagnosis. fig.25 on page 21

**Periventricular**

-Primary CNS lymphoma: Intaaxial (secondary CNS lymphoma more frequent meningeal involvement), solitary supratentorial mass or multiple, sharply demarcated, deep cerebral hemisphere with surrounding edema. Can involve: corpus callosum, periventricular white matter, thalamus or basal ganglia location.

-"Lamb's wool" appearance on contrast-enhanced images.

-Heterogenenity or ring enhancement is more common in patients immunosupression: Hypo or isointense on T1WI and Iso to hyperintense on T2WI.

-Infectious ependymitis: ependymitis and ventriculitis may cause linear and thin (<2mm) enhancement along the ventricular (inferior) surface of the corpus callosum. fig.26 on page 22

**Images for this section:**
Extraaxial enhancement

Meningeal layers

Normal dura mater, which is extraaxial nonneural connective tissue, does not have a blood-brain-barrier. The arachnoid mater provide a the layer with a barrier function that prevents the movement of fluid across it. The pia is highly vascular, connective tissue envelope surrounding the brain and spinal cord.

Fig. 1: fig.3
Extraaxial enhancement

• Normal Meningeal Enhancement Pattern:

  - THIN (maximum 2 mm).
  - Smooth.
  - Discontinuous.
  - Best detected on coronal MR sequences
  - Marked in the anterior portions of the temporal lobes, parasagittally, and at sites of dural reflections of the superior sagittal sinus.

Axial T1WI + contrast  Coronal T1WI + contrast

Fig. 2: fig.4
Extraaxial enhancement

- Abnormal meningeal enhancement

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Fig. 3: fig.5
**Extraaxial enhancement**

**Pachymeningeal:** Dura-arachnoid enhancement. The vessels within the dura mater do not produce a blood-brain barrier.

**Leptomeningeal:** Pia-arachnoid enhancement. Extends into the subarachnoid spaces of the sulci and cisterns. Usually associated with meningitis.

**Fig. 4:** fig.6
Extraaxial enhancement

Pachymeningeal

- The vessels within de dura mater do not produce a blood-brain-barrier. (Nonneural extraaxial connective tissue).
- Normal dural enhancement is well seen on CT: in the dural reflections of the falx cerebri, tentorium cerebelli, and falx cerebelli.
- Usually not recognized because appears: “white on white”.
- RM T1WI: the normal dura mater and inner table bone are uniformly hypointense.
- Contrast: thin, linear, discontinuous enhancement.

- BENIGN
  - Post-operative
  - Intracranial hypotension
  - Meningioma.
  - Granulomatous disease

- MALIGN
  - Metastases (mama/prostata)
  - Secondary CNS lymphoma.

Fig. 5: fig.7
Intraaxial enhancement

Nodular cortical-subcortical

- Metastases

Axial CT with contrast scan (a), (b). Patient 60 year old male with lung cancer. Subcortical nodular-enhancement. Multiple nodular lesions that are hyperattenuating enhancement because of microscopic hemorrhages, and ring enhancement due to necrosis of the metastases.

Fig. 6: fig.18
Intraaxial enhancement

CONCENTRIC ZONES OF ORGANIZING INFECTION
An abscess is a result of organization and partial neutralization of an infection. Develops a well-formed capsule in 2-4 weeks.

Fig. 7: fig.19
**Fig. 8:**

Axial (a) T2WIMRI image shows a circular mass with mass effect and extensive perilesional vasogenic edema that surrounds a dark rim: abscess wall. Axial (b) T1WIMRI + contrast: the rim of cerebral abscess have a smooth inner and outer margin. Coronal (c) FLAIR shows a ringed lesion with perilesional edema. Axial DiffusionWI MRI (d) shows the lesion with a markedly restricted diffusion (hyperintensity) due to viscous pus and necrotic brain tissue in the abscess core.

**Intraaxial enhancement**

**Abscess**

Smooth ring-enhancing pattern in late cerebritis and subsequent cerebral abscess.
Intraaxial enhancement

- NECROTIC NEOPLASMS
  - Thick-irregular ring.
  - Wall thicker than 10 mm.
  - Thick medially.
  - Multilocular and complex ring.

- ABSCESS
  - Smooth ring.
  - Smooth inner and outer margin.
  - Typically hypointense rim on T2WI.
  - Hyperintense on diffusionWI.
  - Thinner margin of the deep aspect.

Fig. 9: fig.21
Intraaxial enhancement

Necrotic neoplasms

- Glioblastoma

Coronal T1 WI MRI + contrast. 60 year old male with glioblastoma multiforme. MRI shows a mass with a thick irregular rim with a shaggy inner margin (7) (typical of a glioblastoma multiforme).

Fig. 10: fig.23
Intraaxial enhancement

Fluid secreting neoplasms

- Pilocytic astrocytoma

Axial nonenhanced T1WI MRI (a), axial T1WI MRI+ contrast. (a) shows a smooth-margined mass in the cerebellum surrounded by a cyst with fluid that is higher intensity than the cerebrospinal fluid in the fourth ventricle, there is a intense enhancement of the mural nodule in (b).

Fig. 11: fig.24
Intraaxial enhancement

Demyelination  ▪ Multiple sclerosis

Sagittal T1 WI MRI+ contrast in 34 year old female with multiple sclerosis. Incompletely rim cervical spinal cord lesion in a patient with multiple sclerosis during «active phase».

Fig. 12: fig.25
Axial CT with contrast. Thin periventricular enhancement in ventriculitis and ependymitis. Abnormal enhancement surrounding left lateral ventricle, thin and uniform.

**Fig. 13:** fig.26
Axial (a), (b) CT + contrast. Serpentine enhancement affecting a single artery territory (right cerebral middle artery) after 2 weeks of cerebral infarction.

**Fig. 14:** fig.17
Intraaxial enhancement

Cortical-Gyral  •  Encephalitis

Axial (a) T1 WI MRI+ contrast and Coronal (b) Flair WI MRI. In case of herpes meningoencephalitis that shows in (a) intraaxial, curvilinear, cortical gyriform enhancement that involves left temporal lobe. In (b) image reflect left temporal lobe hyperintensity due to herpes encephalitis.

Fig. 15: fig.16
Extraaxial enhancement

Pachymeningeal

- Intracranial hypotension

Axial (a), Coronal (b), (c) MRI T1WI + contrast: Dura-arachnoid pachymeningeal enhancement in a patient with intracranial hypotension. In (b) is shown cerebellar infarction.

Fig. 16: fig.8
Extraaxial enhancement

Meningioma.

Axial MRI T1WI + contrast: Meningioma with dural tail (arrow). Caused by vasocongestion and edema that reflects inflammatory changes in the dura.

Fig. 17: fig.9
Fig. 18: fig.10

Axial MRI T1WI + contrast: Meningioma with dural tail (arrow). Caused by vasocongestion and edema.
Coronal MRI T1 WI: focal and nodular pachymeningeal enhancement pattern, like dural masses. Granulomatous processes affect the basilar meninges, more than the convexities of the cerebral hemispheres.

**Fig. 19:** fig.11
Fig. 20: fig.12

Axial CT shows pachymeningeal enhancement. Smooth thickening of the dura-arachnoid (*) of the convexities, due to metastatic disease from breast carcinoma.
**Introduction**

**Mechanism of contrast**

**Intravascular**
- Reflect **neovascularity**, vasodilatation or hyperemia and shunting.
- The semipermeable blood-brain barrier **blocks lipophobic compounds** and creates a unique interstitial fluid environment for the neural tissues.
- In the brain, spinal cord, and proximal cranial and spinal nerves, the intact blood-brain barrier will **prevent leakage of contrast material**.

**Extravascular**
- Related to alterations in the **permeability** of the blood-brain-barrier.
- Proportional to **increases** in blood flow or blood volume.
- Interstitial enhancement when CT imaging is **delayed** 10-15 minutes.

**Fig. 21: fig.1**
- The arterial blood supply to the dura mater is through vessels that run along its superficial surface. These vessels arise from branches of the external and internal carotid and the vertebral arteries forming an anastomotic plexus, superficial in location. Most of the supratentorial dura is supplied by the **middle meningeal artery** (a branch of the internal maxillary artery).

**Fig. 22: fig.2**
Extraaxial enhancement

- Usually associated with meningitis
  - Bacterial
  - Viral
  - Fungal

- Breakdown of the blood-brain barrier without angiogenesis.
- Bacterial and viral meningitis: typically thin and linear enhancement.
- Infiltration of subarachnoid space with inflammatory cells.
- Fungal meningitis: thicker, nodular enhancement.
- Neoplasms: «Carcinomatous meningitis»: spread into subarachnoid space.
- Primary tumors: medulloblastoma, ependymoma, secondary tumors (lymphoma).
- Superficial siderosis.

Fig. 23: fig.13
Extraaxial enhancement

Leptomeningeal ▪ Medulloblastoma

Sagittal MRI T1+contrast (a), Coronal T1+contrast (b): shows leptomeningeal enhancement. (a) Leptomeningeal metastases with "sugar coating" enhancement [7]. (b) Drop metastasis in a case of medulloblastoma. MRI T1 postcontrast images showing densely enhanced masses extending and compressing the spinal cord in a case of recurrent medulloblastoma.

Fig. 24: fig.14
**Fig. 25:** fig.15

Axial (a) and Coronal (b) T1 WI MR + contrast. Pia-arachnoid leptomeningeal enhancement with multiple meningeal-based nodular metastasis from ovarian cancer.
**Fig. 26: fig.22**

Axial (a) and Coronal (b) T1 WI MRI + contrast. 60 year old male with glioblastoma multiforme. MRI shows a mass with a complex appearance, with a thick irregular rim which contains the greatest concentration of neovascularity, inside the rim there is a patchy pattern which reflects the presence of islands of surviving tumor with regions of necrosis. The mass is located in corpus callosum.
Conclusion

Understanding the classic patterns of lesions enhancement and the radiologic-pathologic mechanisms that produce them, can improve image assessment, differential diagnosis and follow up of neoplastic and nonneoplastic disorders affecting the brain and meninges.

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