Department of Radiology
Division of Nuclear Medicine
Didactic
Outline

• Key concepts: MRI sequences, brain edema.
• Parenchymal infections: cerebritis vs Abscess, aunt minnie appearances of certains entities.
• Extra-axial infections: Meningitis vs Empyema
• AIDS associated infections: HIV encephalopathy, PML, Toxoplasmosis.
## Key Concept 1

<table>
<thead>
<tr>
<th>Sequence</th>
<th>T&lt;sub&gt;1&lt;/sub&gt;/CT</th>
<th>T&lt;sub&gt;2&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSF</td>
<td>Low signal</td>
<td>High Signal</td>
</tr>
<tr>
<td>Edema/ fluids</td>
<td>Low signal</td>
<td>High Signal</td>
</tr>
<tr>
<td>Collagen/ scarring/ Hemosiderin</td>
<td>Low signal</td>
<td>Low signal</td>
</tr>
<tr>
<td>Cerebral edema</td>
<td>Vasogenic</td>
<td>Cytotoxic</td>
</tr>
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<td>----------------</td>
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</tr>
<tr>
<td><strong>Pathology</strong></td>
<td>Disruption of BBB with fluid leakage from capillaries</td>
<td>Cellular swelling secondary to Lack of ATP</td>
</tr>
<tr>
<td><strong>Entities</strong></td>
<td>Brain tumors, Abscesses, Cerebral contusions</td>
<td>Infarct, cerebral hypoxia</td>
</tr>
<tr>
<td><strong>Key imaging finding</strong></td>
<td>Involves white matter only</td>
<td>Involves grey and white matter</td>
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</table>
Vasogenic or cytotoxic edema?

Case 1

Case 2
Key concept 3: Diffusion DWI

Detect small restrictions in the movement of water molecules inside the injured areas. These small changes, which are commonly referred to as "areas of restricted diffusion," appear **as bright spots** on DWI, **dark spots** on ADC map.

Examples: Acute stroke (toxic edema), Abscess, some tumors with high N/C ratio.
Parenchymal infections

- Bacterial
- TB
- Fungal
- Parasitic
- Viral
Bacterial infections

• Route
  • Direct Extension
    • Sinusitis (frontal), otomastoiditis (temporal), trauma, surgery, dental infections
  • Hematogenous
    • Lung infection, endocarditis, congenital heart disease
    • MCA territory, Gray-white jxn
Symptoms

• Headache, malaise
• Fever is absent more than 50% of the time
• Meningeal signs present in only 30% of the time
• Focal neuro deficits, papilledema
Impact of Imaging on CNS infections

- Death secondary to CNS pyogenic abscess has dropped from 30-70% to 5% in recent years. This drop is believed to be related to ability of CT to diagnose brain abscesses and monitor treatment.
4 stages of brain infection:

- Early cerebritis
- Late cerebritis
- Early capsule
- Late capsule
Early cerebritis

- Days
- Path: Areas of necrosis filled with PMN, lymphocytes and plasma cells. Organisms are present at the center and periphery of lesion.
- CT: normal vs edema
- MRI: High T2 signal, Low T1 signal.
- Mimics: infarct.
- Tx: Antibiotics
Late cerebritis

- 1-2 weeks
- Central necrosis is increased, fewer organisms in center—**Thick irregular** contrast enhancement on edges of lesion.
- Vasogenic edema surrounding ring enhancement.
- Tx: Antibiotics.
Early Capsule

- 2 weeks
- Capsule of reticulin and collagen - both low on T2
- Ring enhancement on contrast CT with low T2 signal on MRI
- Mild DWI restriction
- Vasogenic edema
Late capsule

- On T1: isointense or hyperintense to white matter
- T2: hypointense to white matter
- Increased DWI
- Medial aspect is usually thinner of peripheral aspect in 50% of times
<table>
<thead>
<tr>
<th>Time</th>
<th>Path</th>
<th>Imaging</th>
<th>TX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Cerebritis</td>
<td>Days</td>
<td>PMN, Plasma, lymph</td>
<td>Diffuse Edema</td>
</tr>
<tr>
<td>Late Cerebritis</td>
<td>1-2 wks</td>
<td>Necrosis in center, PMN and macro in periphery</td>
<td>Thick ring enhancement</td>
</tr>
<tr>
<td>Early Capsule</td>
<td>2 wks</td>
<td>Reticulin deposition</td>
<td>Low T2 rim</td>
</tr>
<tr>
<td>Late Capsule</td>
<td>2-3 wks</td>
<td>Increased maturation of collagen formation</td>
<td>Diffusion restriction</td>
</tr>
</tbody>
</table>
Role of imaging

- Complications
  - Ventriculitis
  - Infarction
  - Hydrocephalus
Not all ring enhancing lesions are Abscesses!
Long Differential Diagnosis for ring enhancing lesions

- Metastasis
- Infarct
- Abscess
- Glioblastoma
- Radiation necrosis
- Demyelination
- Contusion
Mnemonic: MAGIC DR

M - metastasis
A - abcess
G - gliobastoma
I - infarct (subacute phase)
C - contusion
D - demielyinating disease
R - radiation necrosis
TB

- Tuberculoma: caseating granuloma
- CT: One or more isodense or slightly hyperdense nodules or small mass lesions
- Very little edema
- CSF with elevated protein and low glucose.
- Do not always show restricted diffusion
- Solid nodular enhancement
- Ddx: small abscesses, fungal/parasitic infections, sarcoidosis.
<table>
<thead>
<tr>
<th>Fungal Infections</th>
<th>Examples</th>
<th>Target Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endemic</td>
<td>Coccidio Histo Blasto</td>
<td>Geographically restricted, Both IS and IC.</td>
</tr>
<tr>
<td>Cosmopolitan</td>
<td>Aspegillus Mucor Candida Crypto</td>
<td>IS. (except for cryptococcus)</td>
</tr>
</tbody>
</table>
Aunt Minnie appearances of certain infections

-Viral
-Parasites
HSV Encephalitis

Sparing of putamen helps differentiate from MCA infarct.

Reactivation of latent HSV1 virus in Trigeminal/ Gasserian ganglion in temporal lobe.

Death in 70 % of cases if not treated.
HSV Encephalitis

- Encephalitis frequently manifests as FDG-PET hypermetabolism, but focal hypometabolism can also be observed.
- During the acute phase of a viral encephalitis the Tc99m-HMPAO exam typically demonstrates an area of increased perfusion (i.e.: a 'hot spot') in up to 94% of cases.
- During the subacute phase (15 days after presentation) a followup exam may demonstrate either normal or decreased tracer uptake at the site of infection.
- Patients with a normal perfusion pattern during the subacute phase have a very good clinical prognosis.

B.Y. Lee et al. FDG PET findings in patients with encephalitis.
Viral Encephalitis

**FIGURE 1.** Matched level transverse slices showing acute (A–D) and chronic (E–H) phase morphofunctional imaging in human herpes virus 6 limbic encephalitis: postcontrast T1-weighted (A, E) and fluid-attenuated inversion recovery (B, F) 3-T magnetic resonance imaging (MRI), fused MRI PET images (C, G), and FDG PET (D, H). On acute phase imaging acquired 10 days after the beginning of neurologic signs and symptoms, FDG PET shows bilateral intense FDG uptake in both hippocampi and amygdalae (C, D).1–7 At the same time, magnetic resonance imaging showed only a mild increased signal on fluid-attenuated inversion recovery sequence (B) without contrast enhancement (A). The follow-up study, done after 3 months, showed FDG hypometabolism (G, H) associated with a hippocampal sclerosis (E, F).8,9
ADEM-Acute Disseminated Encephalomyelitis
CMV – Periventricular calcifications
Arbovirus
Cysticercosis
Extra-axial infections

• Normal vs slight increase T2 signal in meninges +/- enhancement.

• Imaging main role is to look for complications:
  • Hydrocephalus
  • Cerebritis/ abscess
  • Ventriculitis
  • Subdural and epidural empyema/ abscess
  • Venous / arterial infarcts
Extra-axial infections

Subdural Empyema
Racemose Cysticercosis
Subdural Empyema
Basilar meningitis: TB vs Fungal vs Sarcoidosis vs meningeal carcinomatosis
AIDS associated infections

- 2/3 of AIDS patients develop some kind of CNS disease—
infection, neoplasm
- Infection:
  - HIV encephalopathy
  - PML
  - Toxoplasmosis---need to differentiate from lymphoma
  - Cryptococcus and other fungal infections
  - CMV and herpes meningoencephalitis
HIV encephalopathy

- HIV is neurotopic, infects brain in 90% of autopsies
- Path: vacuolation of white matter. Gray matter is spared.
- Early phase: patchy punctate white matter lesion. MRI more sensitive.
- Late phase: Extensive white mater disease. Progression can lead to AIDS Dementia Complex (ADC) characterized by dementia, behavioral and motor deterioration
- Clinical and imaging abnormalities often respond to HAART
Progressive multifocal leukoencephalopathy

• 8% incidence in AIDS patients
• JC papovavirus
• Sx include blindness, aphasia, hemiparesis, ataxia.
• Progressive course to death within months, although treatment with HAART significantly prolongs survival.
• High T2 and FLAIR in suocortical and deep WM----No mass effect, no contrast enhancement.
• In non-AIDS (leukemics, transplant), there is predilection for occipital lobes.
Toxoplasmosis

- Most common opportunistic infection in AIDS (13-30%)
- CD4 less than 100
- Cause: reactivation of previously acquired infection
- Imaging typical features: multiple enhancing masses with vasogenic edema, small lesions (1-4 cm), ring enhancement is seen in larger lesions, no diffusion restriction, basal ganglia are favored.
- Respond to antitoxoplasma antibiotics
- Main differential based on imaging features: lymphoma
- NO RESTRICTED DIFFUSION.
Toxoplasmosis

- abnormal $^{18}$F-FDG PET/CT brain scan demonstrating focal or multifocal low-grade $^{18}$F-FDG uptake

Scarlett Lewitschnig et al 2013. EJNMMI
Cryptococcus

- Dilation of Virchow-Robin spaces filled with cryptococcus organisms
- Rounded structures in basal ganglia, iso-intense to CSF.
- No enhancement following contrast
- What about other lesions?
Case 1:

Thallium 201 brain Scan

Case 2

Palestro et al., The quarterly journal of nuclear medicine and molecular imaging, 2009; 53: 105-23
Labeled leukocyte imaging: current status and future directions.
Suggested Articles


• Palestro et al., The quarterly journal of nuclear medicine and molecular imaging, Feb;2009; 53: 105-23.

• Scarlett Lewitschnig et al. $^{18}$F-FDG PET/CT in HIV-related central nervous system pathology. 2013.

• B.Y. Lee et al. FDG - PET findings in patients with encephalitis.
THANK YOU