UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE



Department of Radiology Division of Nuclear Medicine Didactic



Brain Infections

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Outline

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

Key Concept 1



Sequence	T1/CT	Τ2
CSF	Low signal	High Signal
Edema/ fluids	Low signal	High Signal
Collagen/ scarring/ Hemosiderin		Low signal

Key Concept 2

Cerebral edema	Vasogenic	Cytotoxic
Pathology	Disruption of BBB with fluid leakage from capillaries	Cellular swelling secondary to Lack of ATP
Entities	Brain tumors, Abscesses, Cerebral contusions	Infarct, cerebral hypoxia
Key imaging finding	Involves white matter only	Involves grey and white matter

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---<u>Thick irregular</u> contrast enhancement on edges of lesion.
- Vasogenic edema surrounding ring enhancement.
- Tx: Antibiotics.



FIGURE 6.2. Late Cerebritis. This contrast-enhanced CT scan demonstrates irregular enhancement peripherally and low density centrally. There is surrounding low-density vasogenic edema. This is typical of the late cerebritis stage of pyogenic infection.

Copyright © 2007 by Lippincott Williams & Wilkins, a Wolters Kluwer company. Fundamentals of Diagnostic Radiology, Third Edition by William E. Brant and Clyde A. Helms.

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





	Time	Path	Imaging	ТХ
Early Cerebritis	Days	PMN, Plasma, lymph	Diffuse Edema	Antibx
Late Cerebritis	1-2 wks	Necrosis in center, PMN and macro in periphery	Thick ring enhancement	Antibx
Early Capsule	2 wks	Reticulin deposition	Low T2 rim	Antibx +/- Surgery
Late Capsule	2-3 wks	Increased maturation of collagen formation	Diffusion restriction	Antibx +/- Surgery

Role of imaging

- Complications
 - Ventriculitis
 - Infarction
 - Hydrocephalus



Not all ring enhancing lesions are Abscesses !









Long Differential Diagnosis for ring enhancing lesions















Mnemonic:

MAGIC DR









Μ













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

С

D



ΤB

- 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.



Fungal Infections

Fungal Infections	Examples	Target Population
Endemic	Coccidio Histo Blasto	Geographically restricted, Both IS and IC.
Cosmopolitan	Aspegillus Mucor Candida Crypto	IS. (except for cryptcoccus)



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 encepalitis 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.



FDG-PET increased medial temporal lobe and vermis in a patient with encephalitis

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}

Hubele et al Clinical Nuclear Medicine 2012

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 sucortical 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 ¹⁸F-FDG PET/CT brain scan demonstrating focal or multifocal lowgrade ¹⁸F-FDG uptake



Fig 1

A typical ¹⁸F-FDG PET/CT scan positive for toxoplasmosis with multiple central photopoenic lesions (**a**). The corresponding T2-weighted MRI scan at the same level shows multifocal mass lesions (**b**). ¹⁸F-FDG PET/CT was requested, as there was no initial clinical or imaging improvement under empirical therapy

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

GARCIA-MORALES, CLINICAL NUCLEAR MEDICINE November 2001, 26: 981-2 .Nocardia Brain Abscess Identification with TI-201 SPECT.

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

- GARCIA-MORALES, CLINICAL NUCLEAR MEDICINE November 2001, 26: 981-2.
- Palestro et al., The quarterly journal of nuclear medicine and molecular imaging, Feb;2009; 53: 105-23.
- <u>Scarlett Lewitschnig</u> et al. ¹⁸F-FDG PET/CT in HIV-related central nervous system pathology. 2013.
- B.Y. Lee et al. FDG PET findings in patients with encephalitis.

THANKYOU

