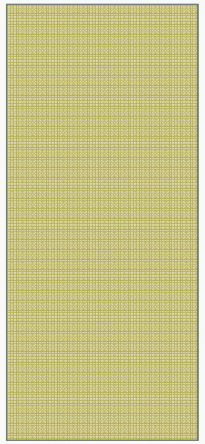
NEUROIMAGING IN ENCEPHALITIS

NADINE GIRARD
DEPARTMENT OF NEURORADIOLOGY MARSEILLE
AMU FRANCE



LAY OUT

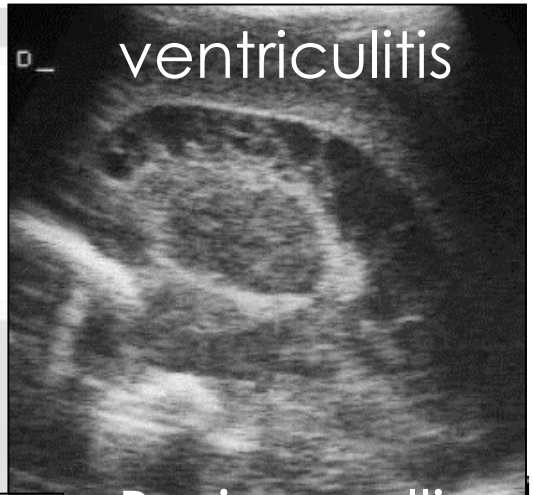
- **1. Imaging techniques**
  - US scan: neonates
  - CT
  - MRI
- **2. Acute Encephalitis**
  - and related disorders: Autoimmune response to viral infections
- **3. Subacute, chronic, progressive infections**

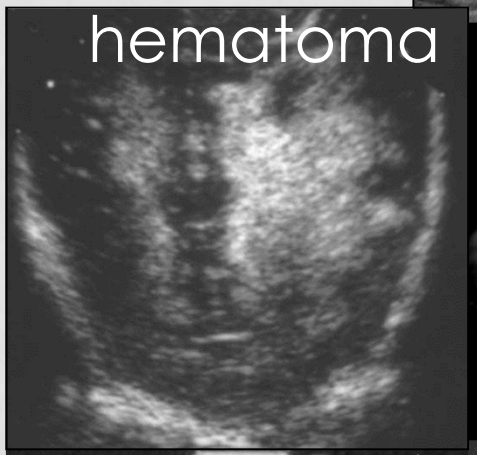
1. IMAGING TECHNIQUES

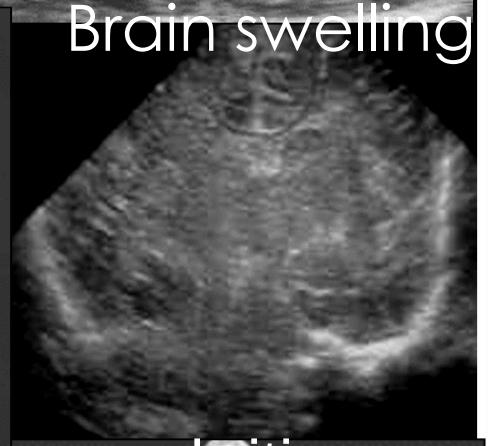
US SCAN: NEONATES

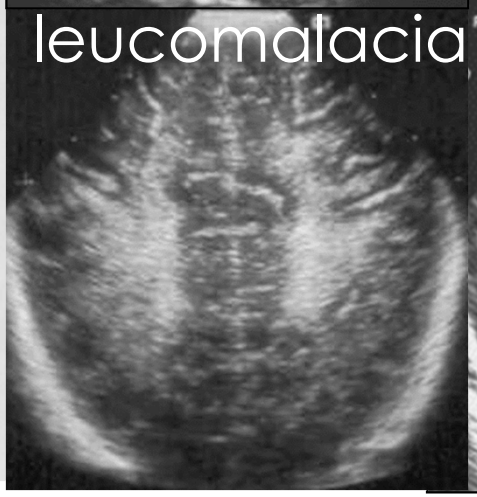
- Ventriculitis
- Cerebritis
- Abscess
- Encephalitis
  - brain swelling

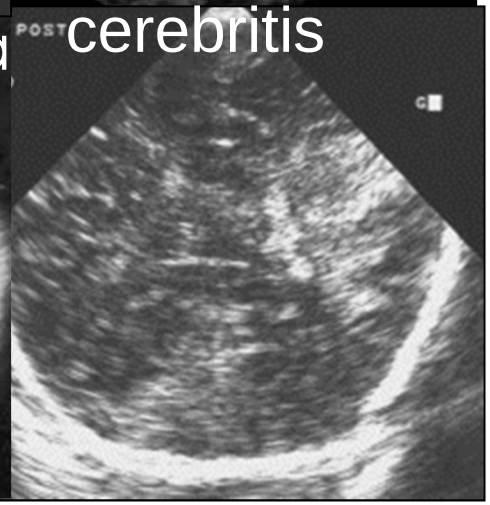
- Hyperechoic lesion: non specific
- Differential diagnosis
  - Ischemia
  - hemorrhage

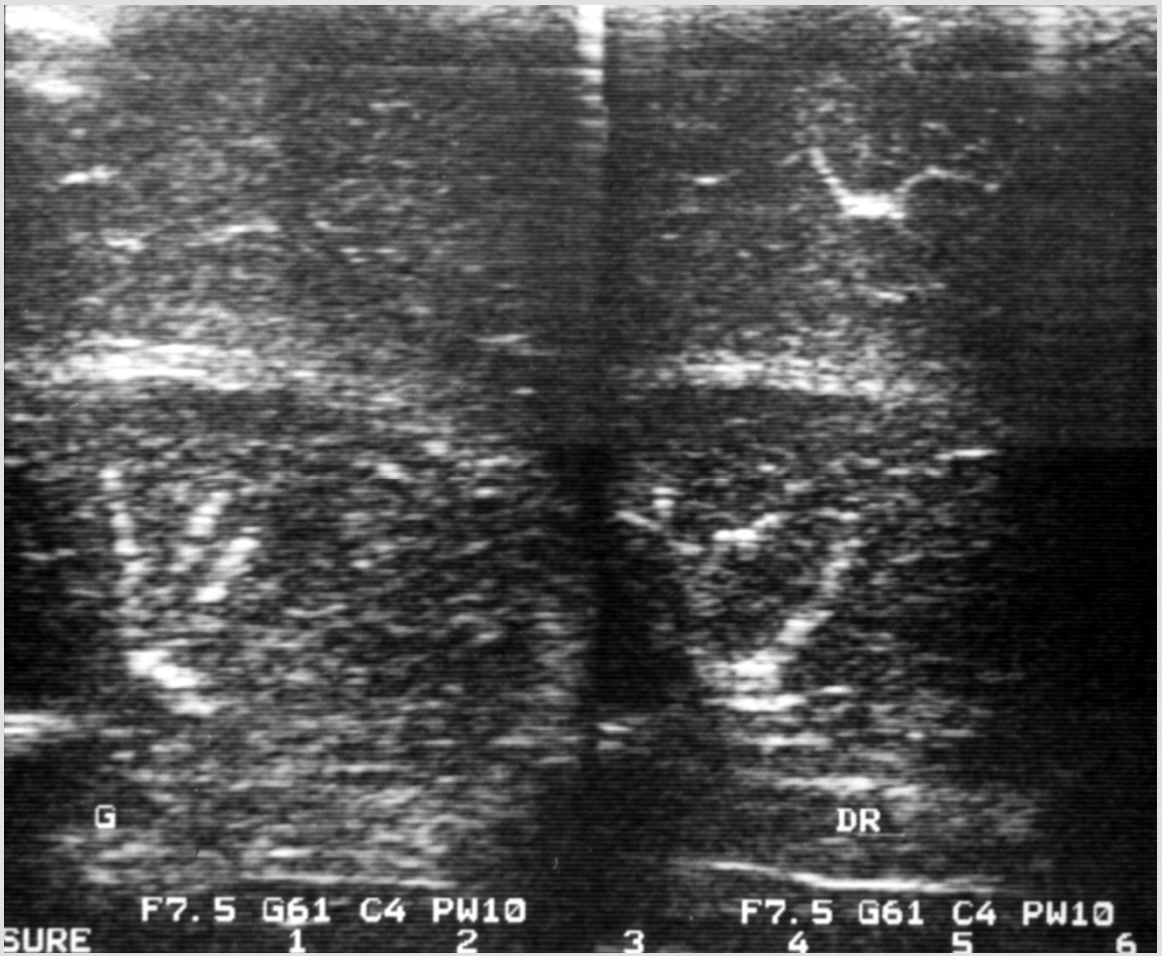










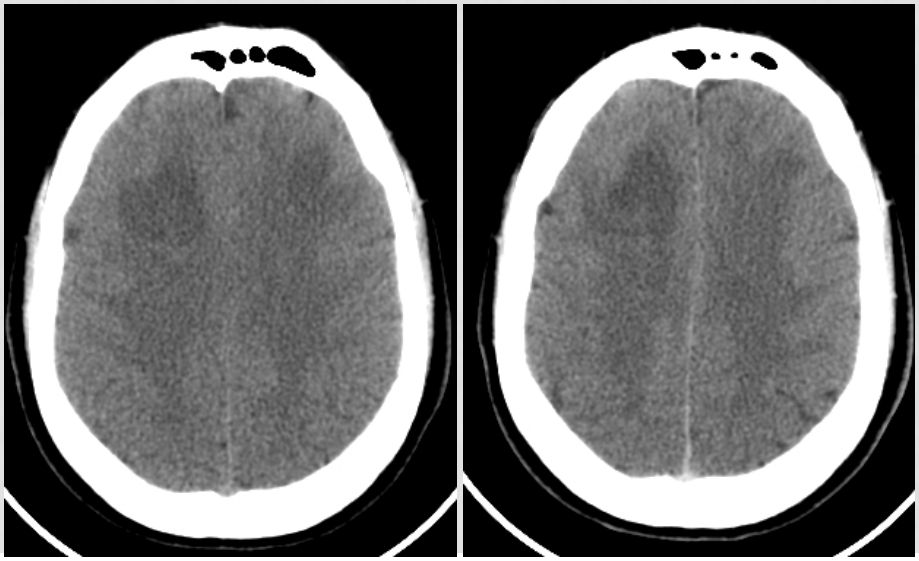


Transfontanel US : « lenticulostriate vasculopathy »

trisomy 13, 21, anoxic/toxic injury, CMV infection

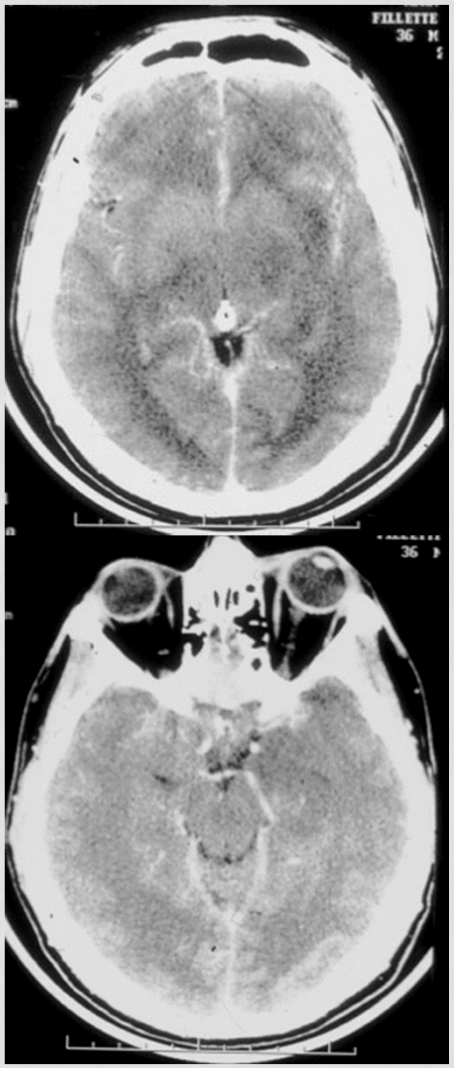
CT SCAN

- Depends on the local access to CT & MRI
- CT
  - emergency
  - before & after contrast +/- angioCT
  - Rule out brain shift, diffuse brain swelling, tonsils prolapse before LP



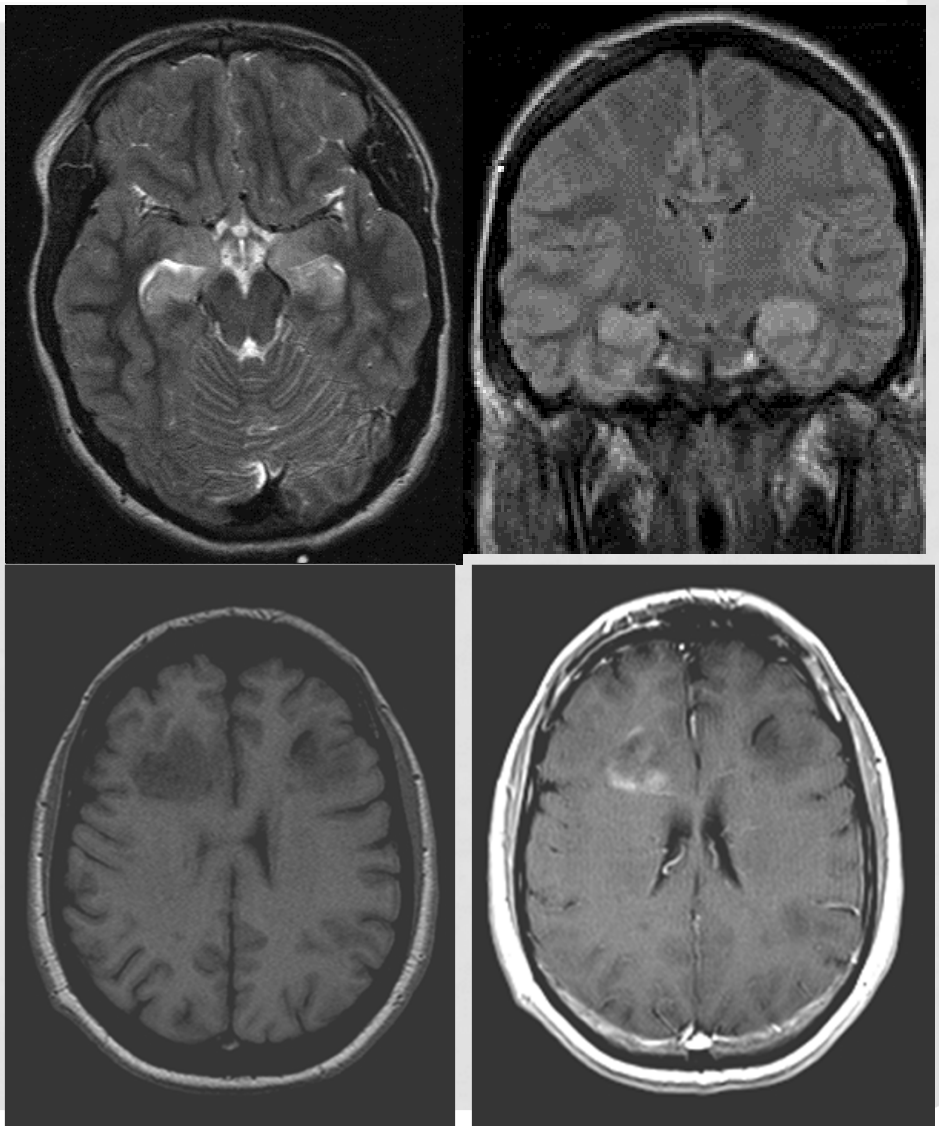
ADEM

Herpes encephalitis



MRI

- most sensitive
  - Diffusion
  - FLAIR
  - T1, T2, T2*
  - IV injection
  - Perfusion
    - Abscess, inflammation
  - MRS
    - Differential diagnosis with tumours
    - Especially in the posterior fossa
  - +/- angioMR, dynamic angioMR

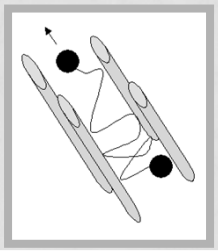


DIFFUSION - PRINCIPLES

- Evaluate mobility (diffusion) of water molecules within tissues
- Displacements of water molecules are modified by structural & physiologic factors in a medium
- Random direction = isotropy; diffusion of water molecules is identical in all directions (CSF)
- In contrast: anisotropy; the process is dependent on direction; the water molecules are spatially distributed (within an ellipsoid) (white matter axons-myelin)

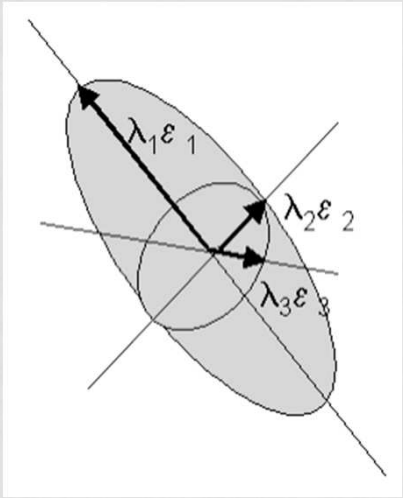
Isotropic media

Anisotropic media



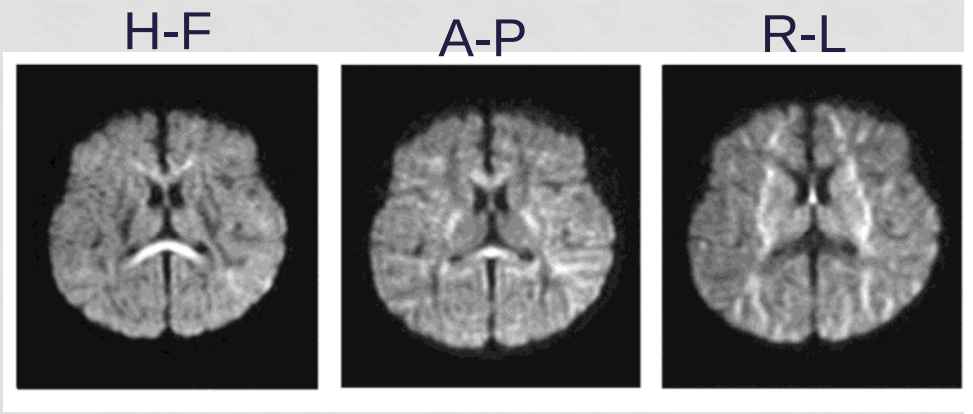
- Diffusion is not the same in all directions of 3D space (anisotropy)
- Molecules are moving in a preferential direction
- Characterized by:
  - coefficient of diffusion (ADC apparent diffusion coefficient)
  - effective diffusion tensor (DT MR imaging with at least 6 directions)
    - 3 eigen values
    - Radial diffusivity: λ2+ λ 3
    - Longitudinal diffusivity: λ1

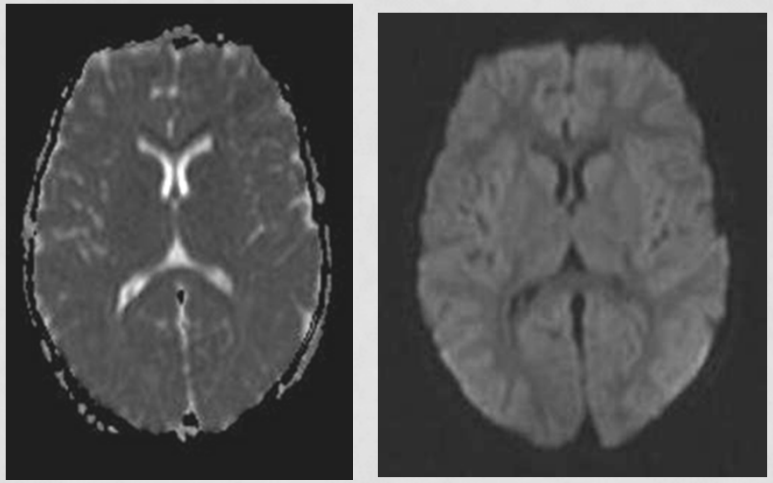
Tensor model



GENERATED IMAGES

- Anisotropic images: 3D space
  - Anatomy
  - Direction of WM bundles
- Diffusion WI
  - Trace (combination of 3 directions)
- ADC map
  - slope of b0, 500, 1000
  - measurement of ADC
    - Structural information
    - Quantitative
    - Dependent on structure

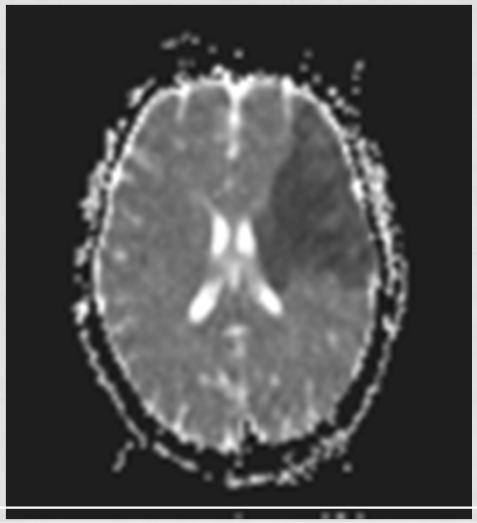


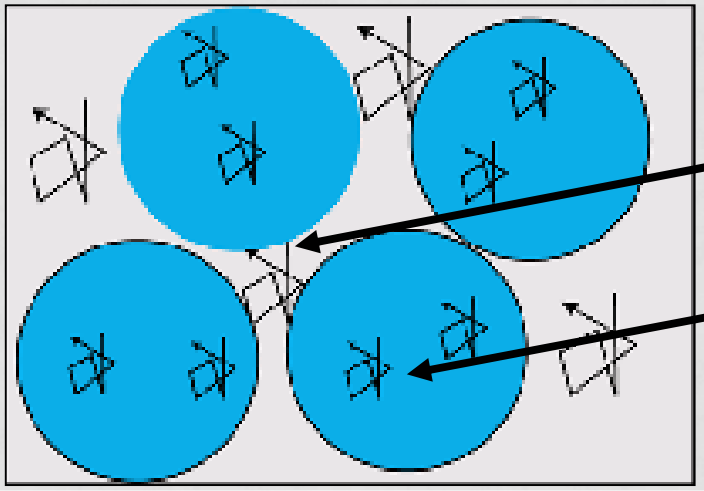


PATHOPHYSIOLOGY OF ADC CHANGES

Cellular edema (cytotoxic) => Decreased ADC

(for b values between 800 and 2000s/mm^2)

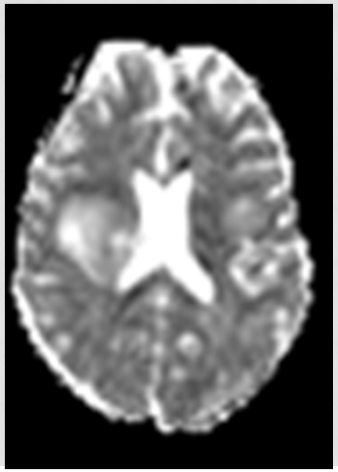


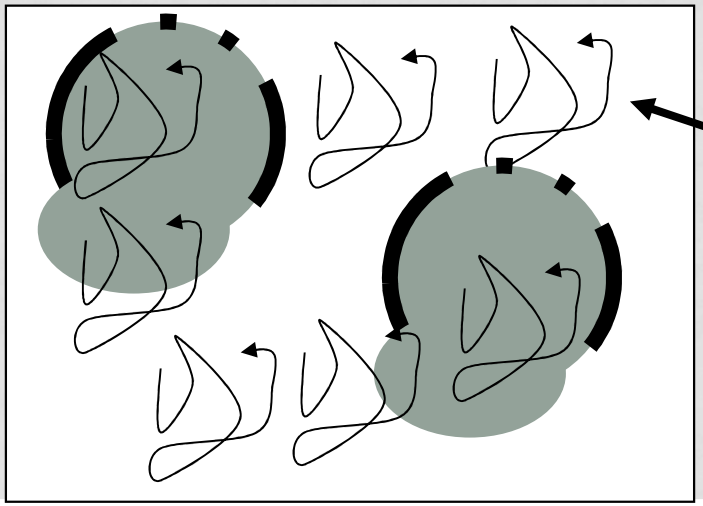


Restricted diffusion in the extracellular space

Increased diffusion in the intracellular space

Vasogenic edema => increased ADC



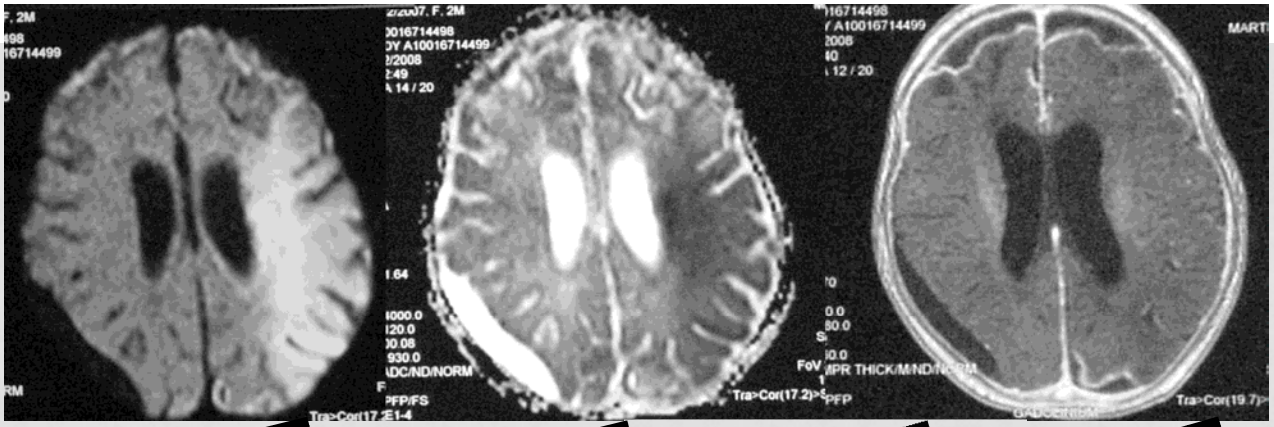


Less restriction of water motion
Membrane breakdown
Expansion of extracellular space

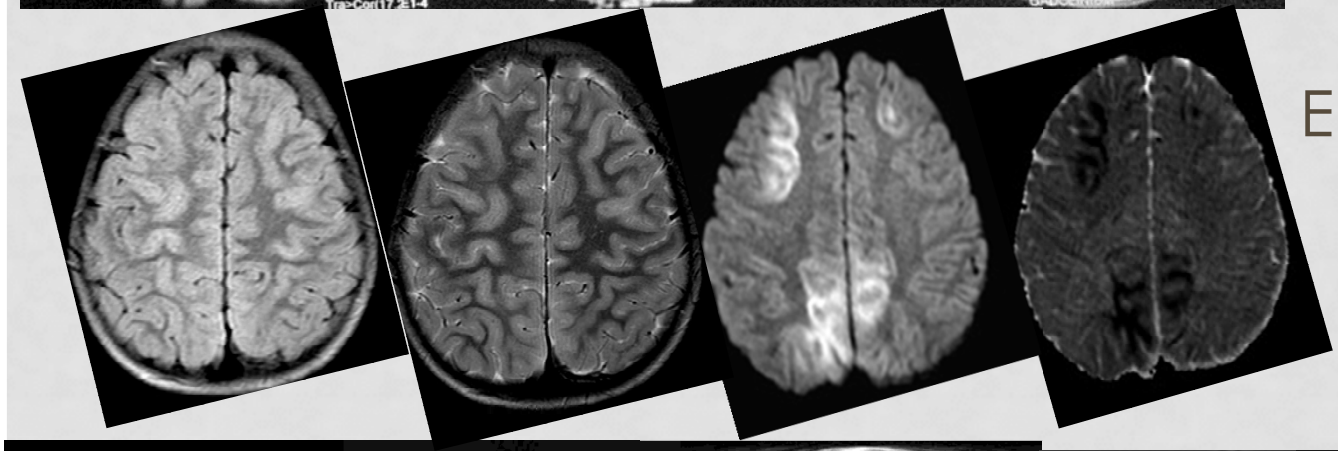
DIFFUSION: APPLICATION

- Factors causing **restriction of diffusion**: increased signal on DWI & low ADC
  - Hypoxia-ischemia, Stroke
  - Trauma (acute axonal shear injury)
  - Compact tumoral tissue
  - Abscess (bacterial origin, fungal origin)
  - Metabolic
- Factors causing **expansion of extracellular space**: decreased or increased signal on DWI & normal or increased ADC
  - Acute demyelination (MS, ADEM, encephalitis)
  - Peritumoral edema
  - Loose tumoral tissue
  - Toxicity of chemotherapy
  - PRES (posterior reversible encephalopathy syndrome)
- **Combination of both**: trauma, metabolic injury, encephalitis, ADEM

RESTRICTION OF DIFFUSION - INFECTIONS

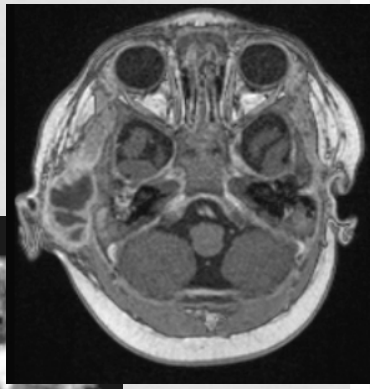


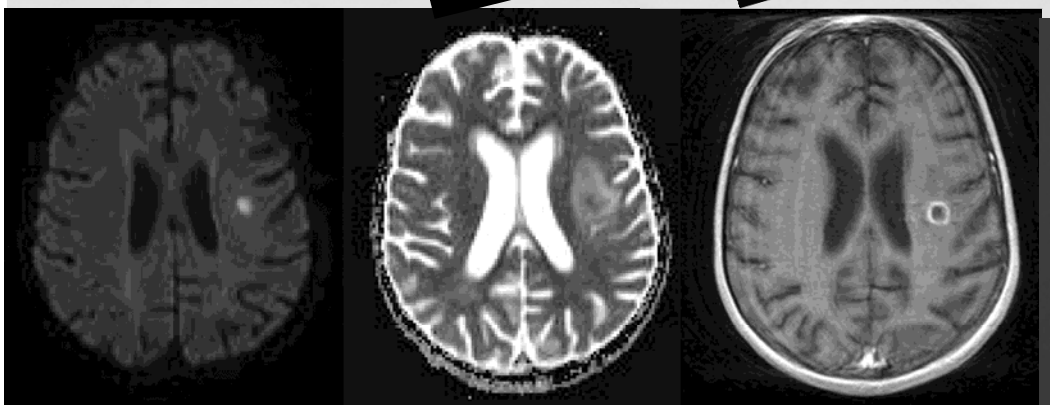
Empyema

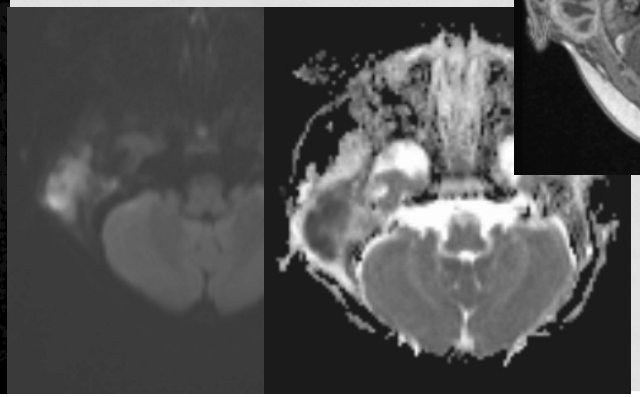


Encephalitis

Abscess



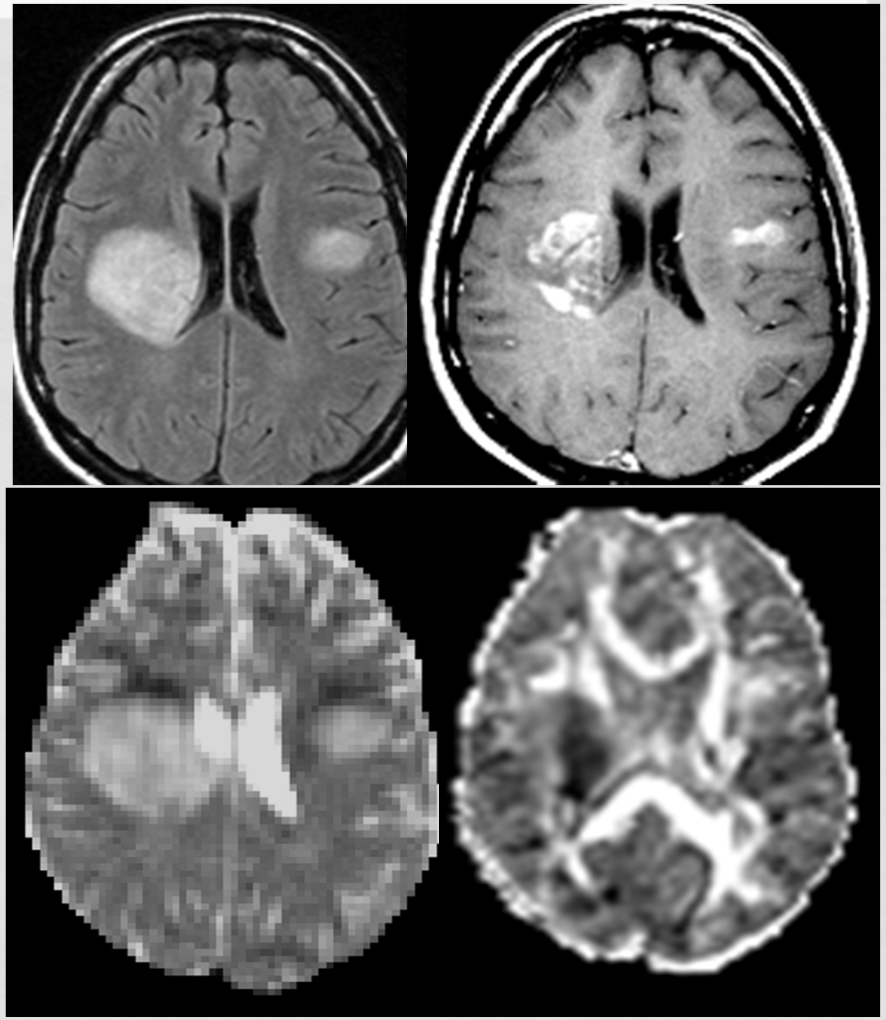




NO RESTRICTION OF WATER MOTION INFLAMMATORY LESIONS - MS

=> Increased MD

⇒ ADC decrease can be related to a mass effect

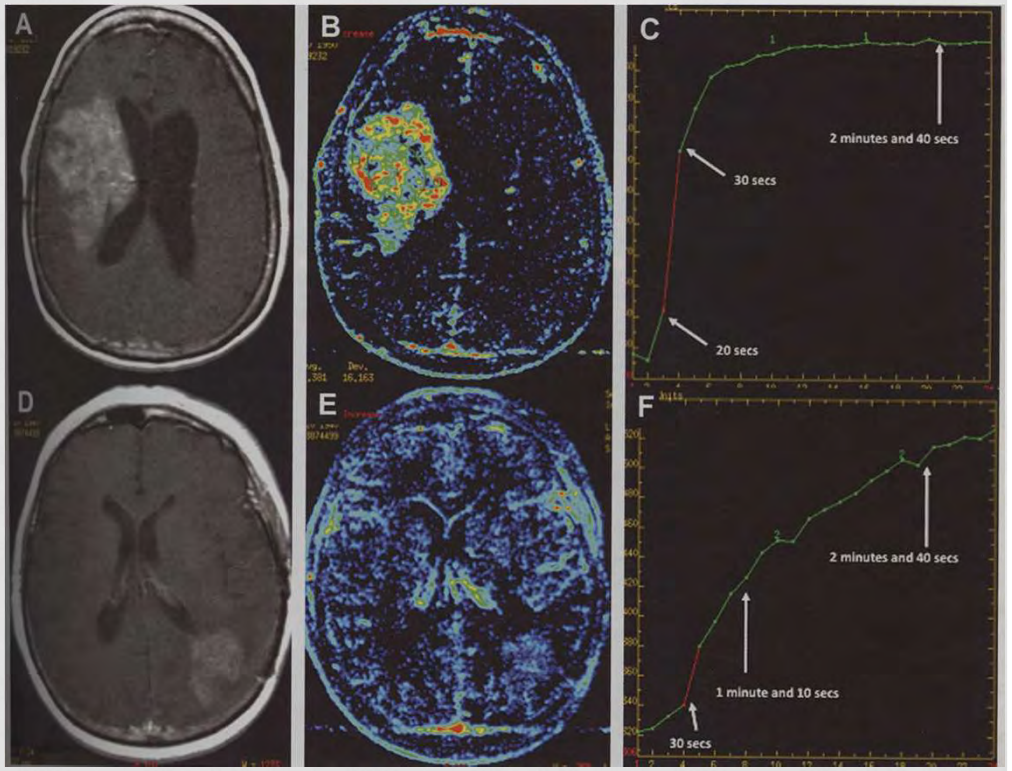


MD FA

PERFUSION MR IMAGING

- **T2* weighted dynamic susceptibility technique (DSC)**
- T1 WI: DCE (dynamic contrast enhancement), leakage (ENT & breast imaging)
- ASL (Arterial spin labeling):, without contrast injection, not used in routine practice

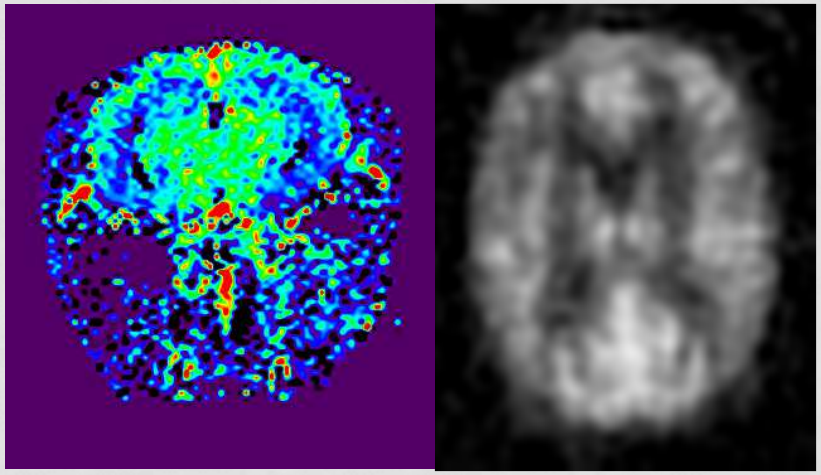
- T1 gadolinium: rarely used in neuroradiology
- used to be the only sequence: ischemia, tumor
- No map available
- Is becoming again fashionable
- Skull base



- Arterial spin labeling: ASL, without contrast injection, not used in routine practice: CBF only

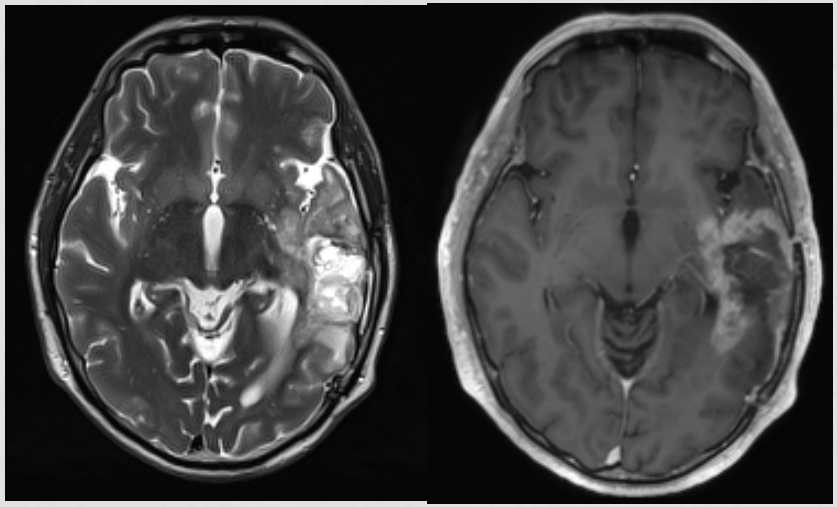
Mouse @ 11.75T

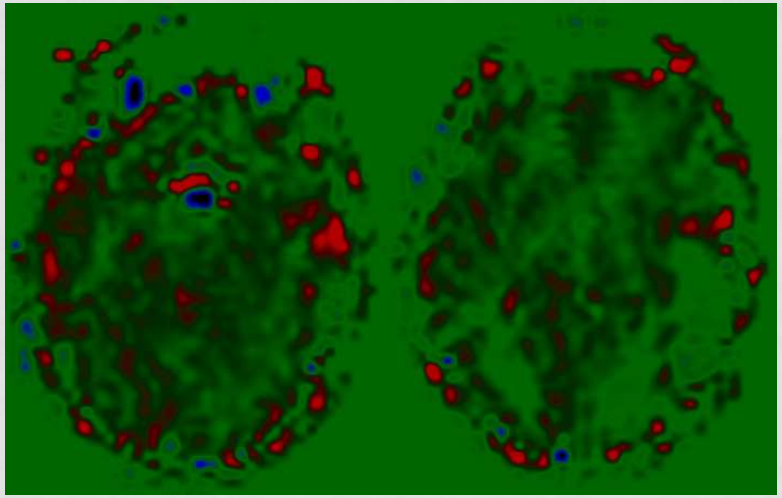
CBF ~ 300 mL/100g/min



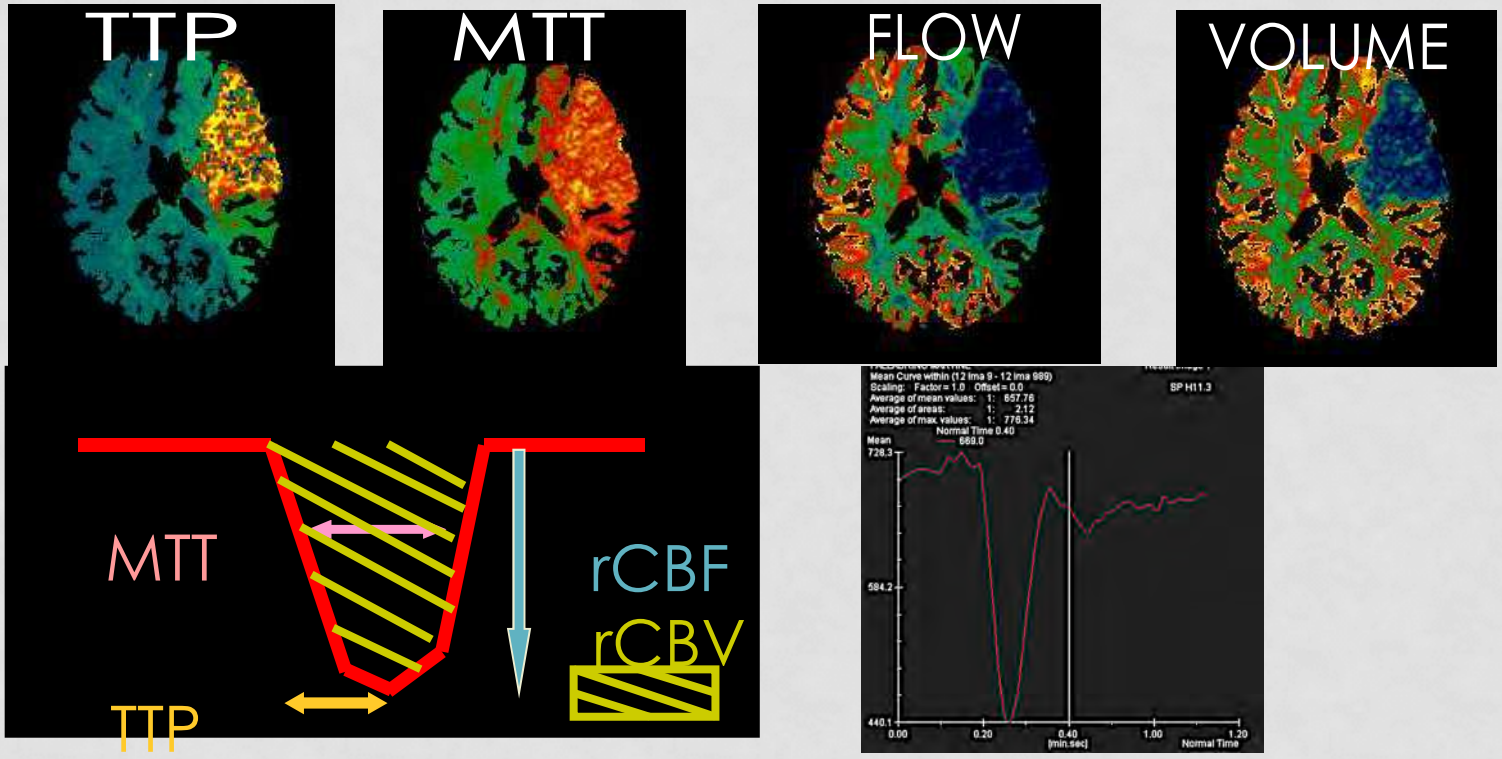
Human @ 3T

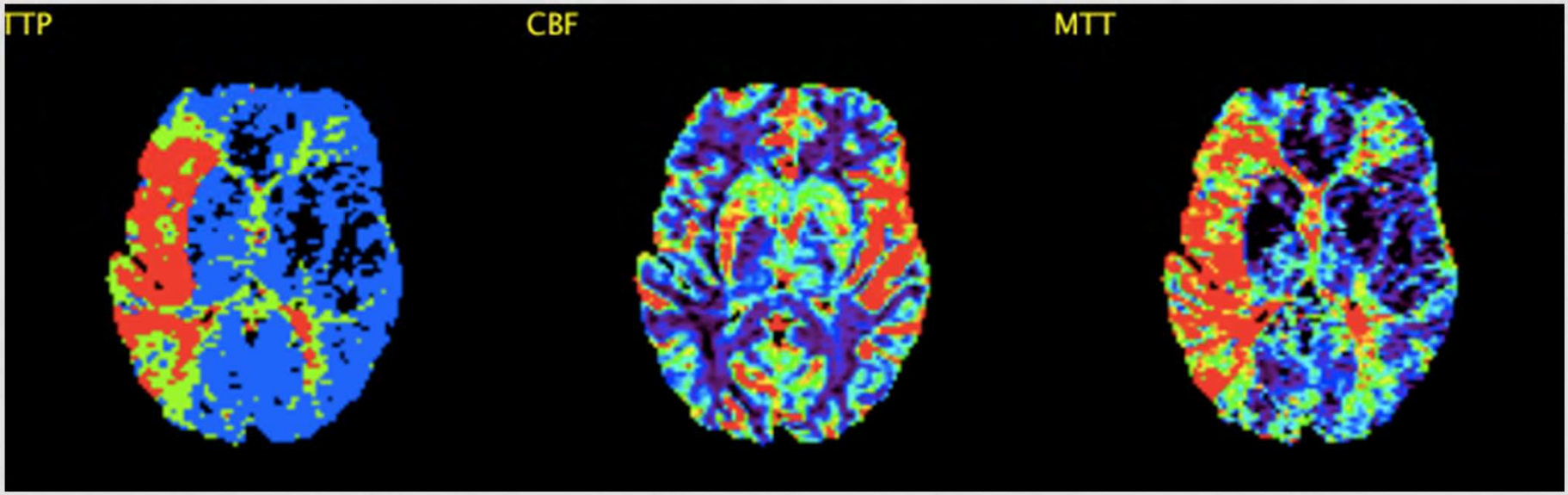
CBF ~ 50 mL/100g/min

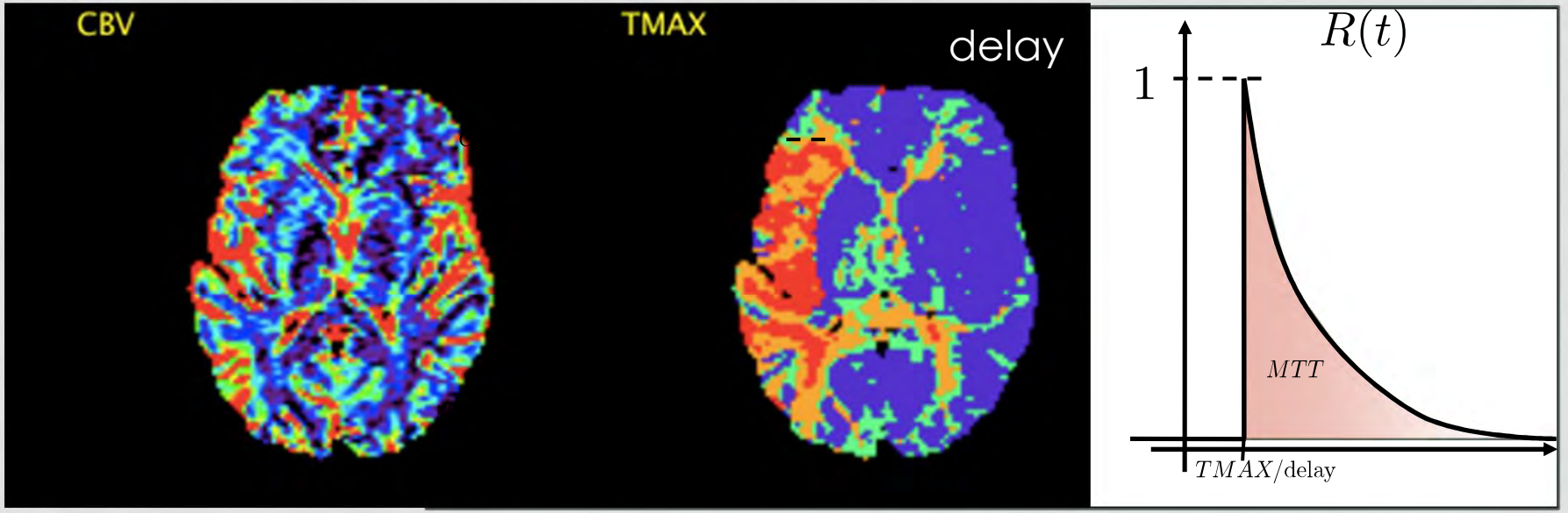




- T2* weighted dynamic susceptibility technique
  - Exploit signal changes that accompany the passage of a tracer through the cerebrovascular system
  - Non diffusible Gd-DTPA/DOTA
  - Large transient signal loss of 25% in normal white matter after contrast injection
  - 6 color maps: MTT, TTP, rCBF, rCBV, K2, Tmax

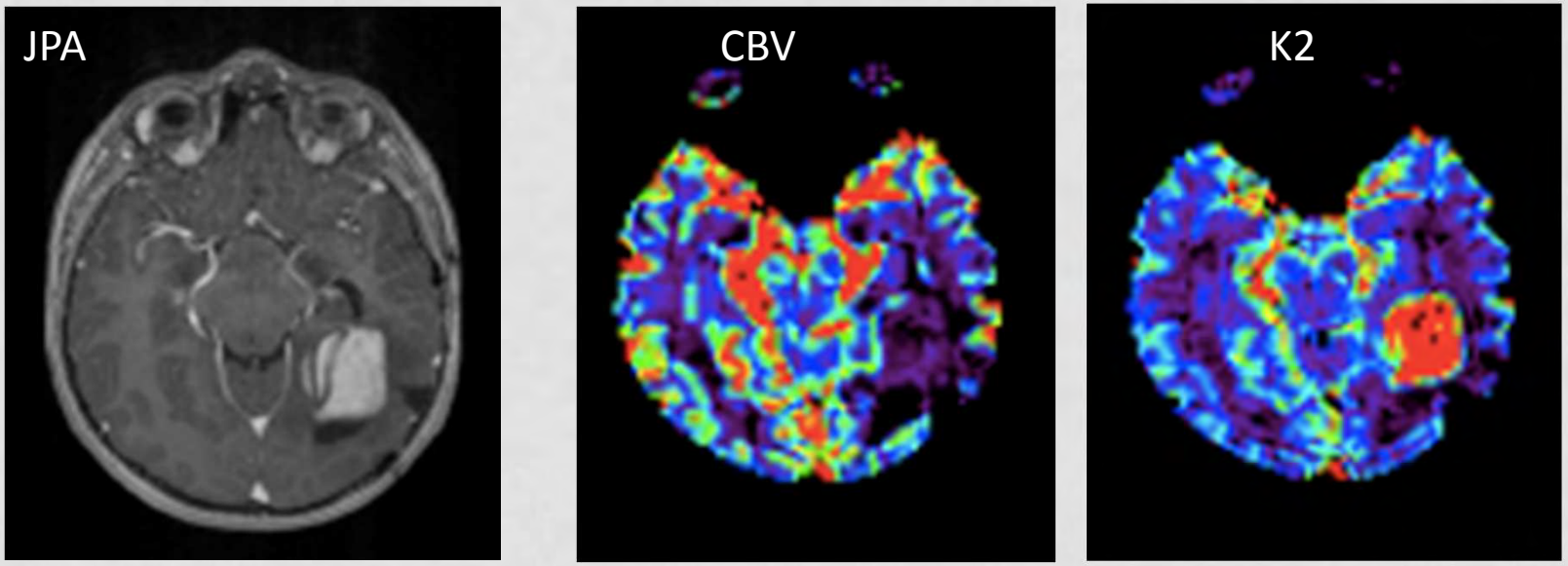






Stroke

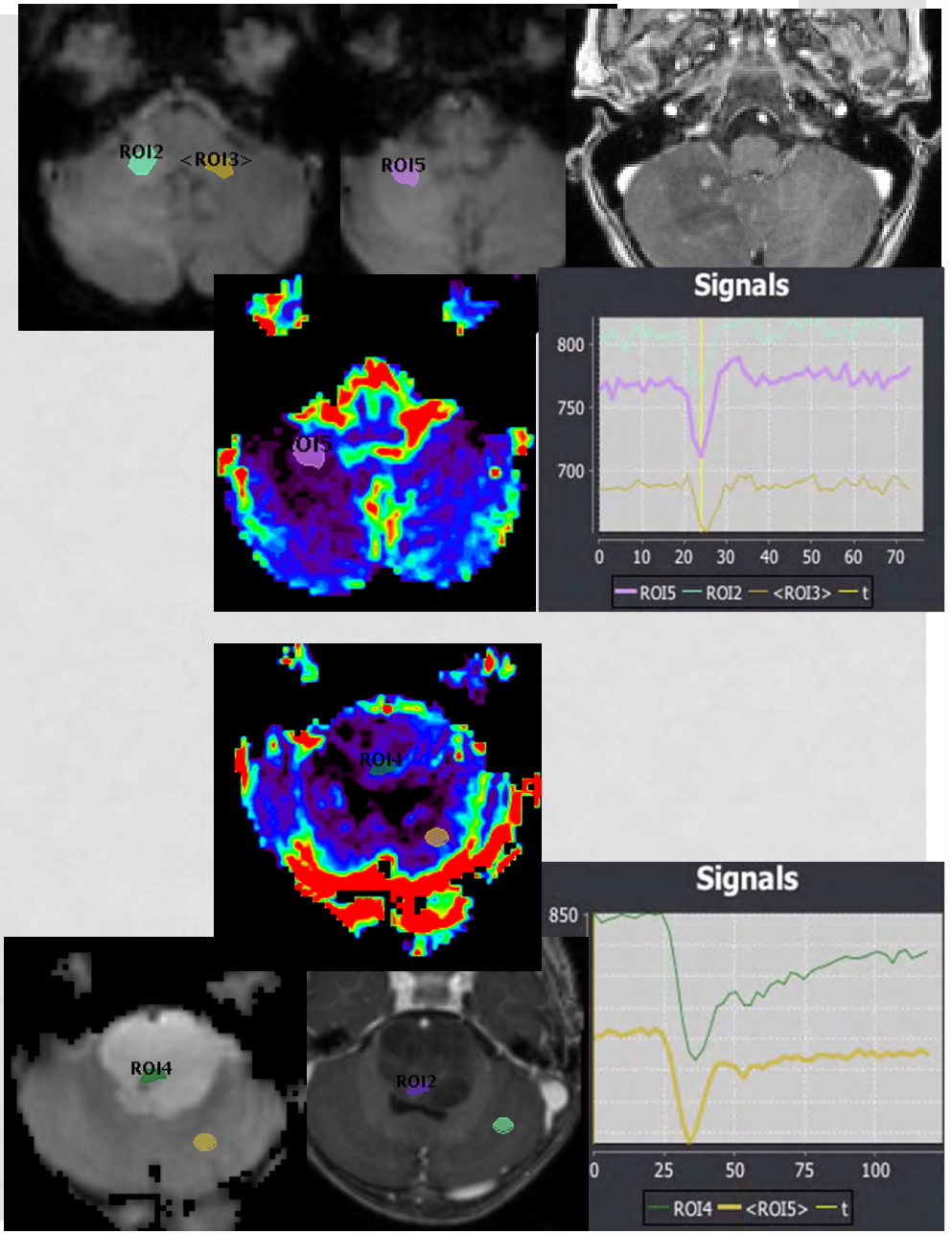
permeability



Brain masses

APPLICATIONS OF PERFUSION MR

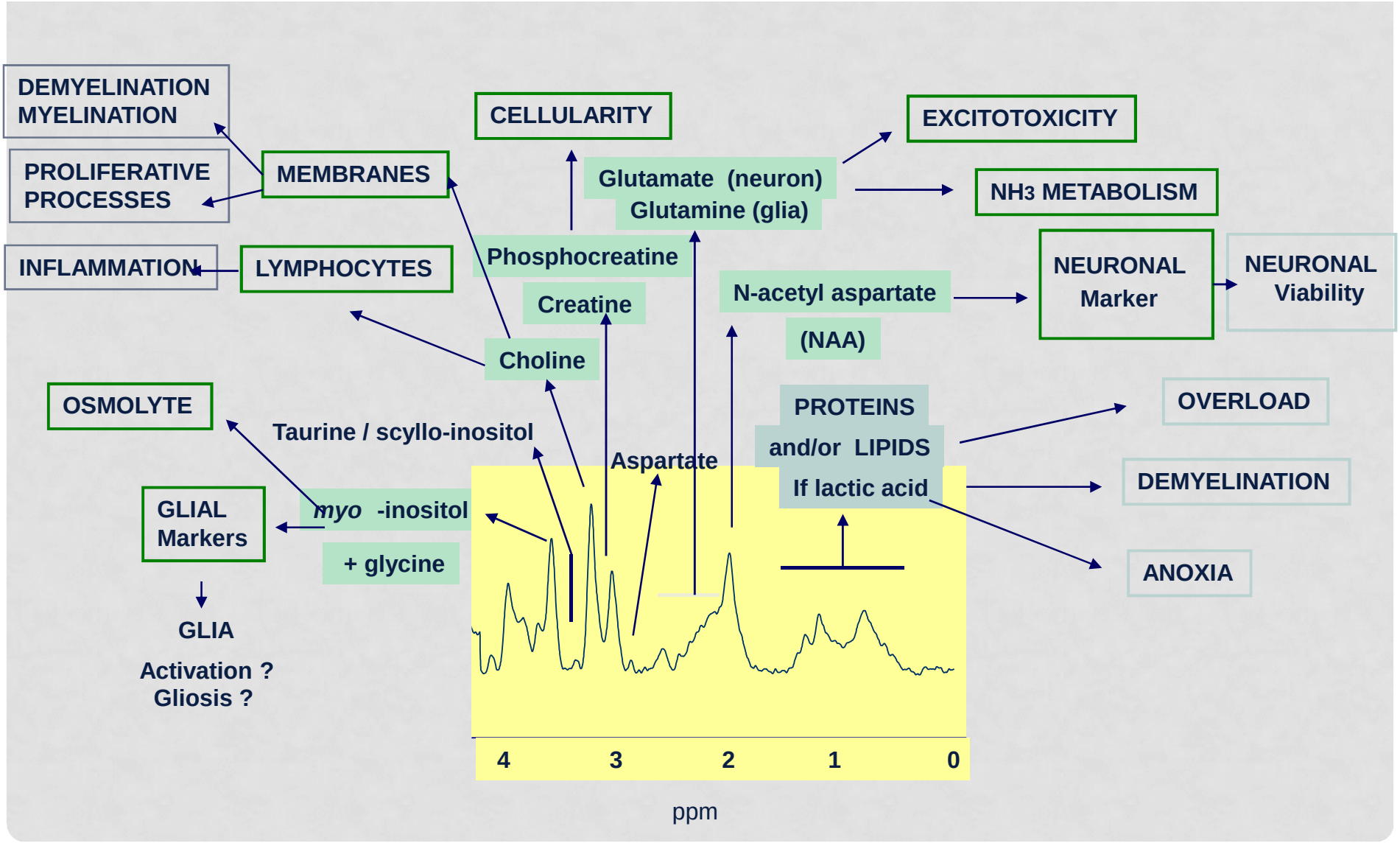
- Stroke: adult population, selection of patient for thrombolysis
- Tumors & non neoplastic mass: adult & pediatric population
- Contrast enhancement
  - Rupture of BBB
    - Capillary extravasation
    - Overshoot above the baseline
    - Tumors, inflammation, infection
  - Neoangiogenesis
    - Increased CBV
    - tumors



PROTON MR SPECTROSCOPY

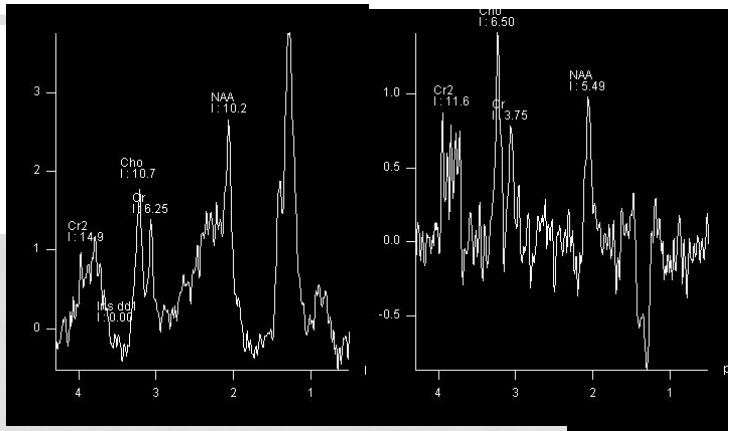
- MR imaging: signal obtained in the time domain is used to generate an image
- MR spectroscopy: signal obtained is used to generate a frequency domain with spectrum of components (that make up the image)
- At each frequency corresponds metabolite or water with a chemical shift to express a peak position
- Reference: NAA set at 2 ppm from the center radiofrequency of the spectrometer

Metabolic information extracted from ^{1}H brain spectrum

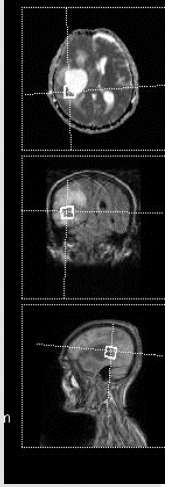


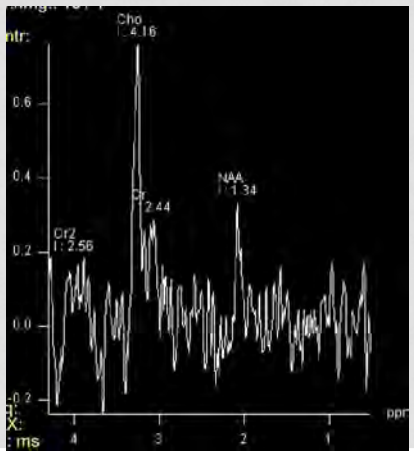
Technical issues of MRS

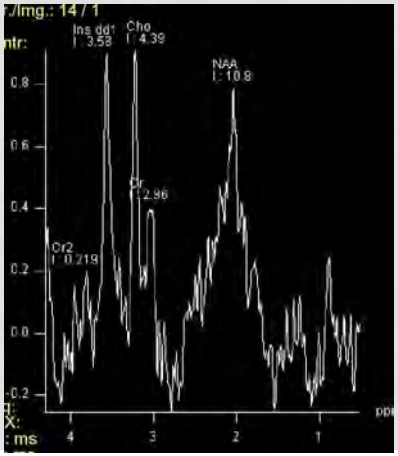
- Monovoxel (SVS single voxel)
  - PRESS
  - 30 to 128 measures (depending on VOI location)
  - short (30ms) and long (135ms) TE
  - Acceptable AT: 51s to 3min (*ex utero*)
  - More easy than CSI
- Enemy
  - hemorrhage, calcification, fat
  - CT scan+++ before MRS

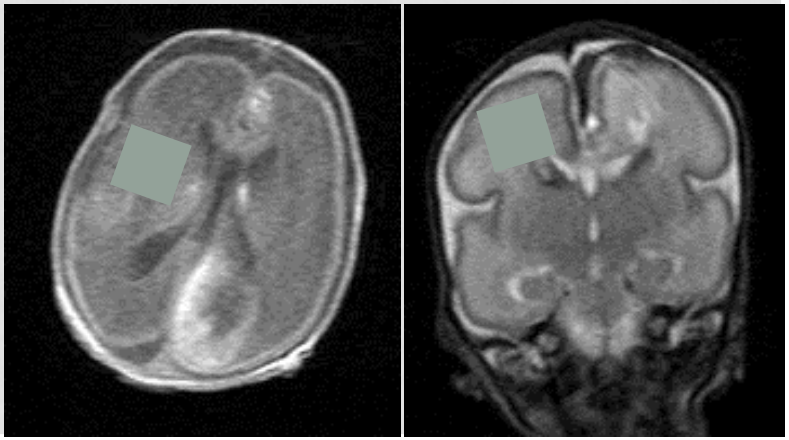


30 measures
51 seconds







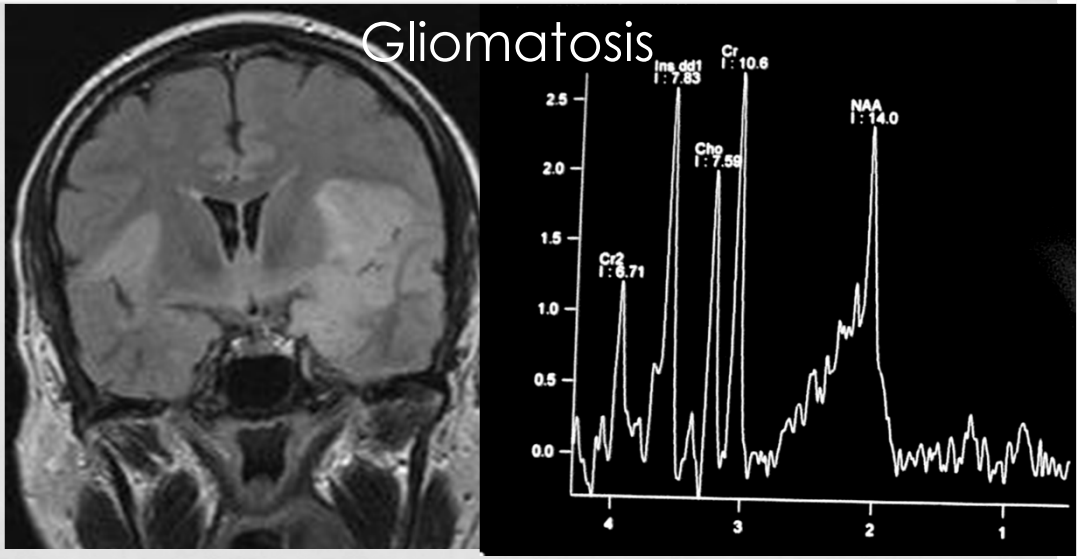


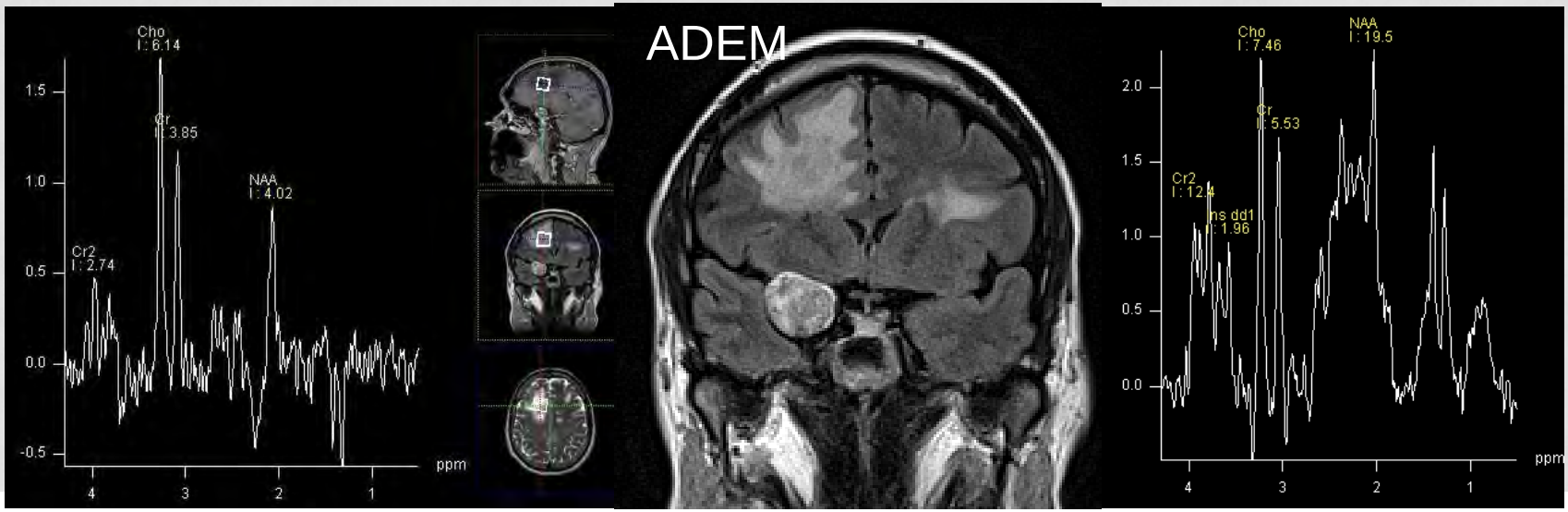
General rules of MRS

- **Brain metabolism is age dependent**
- **With regional variations during maturation and in adult population**
- Specific pattern: few diseases display specific pattern as abnormal peak or lack of normal peak
- Metabolic changes reflect extension of a disease: pathogenic approach
- Focal disease: normal tissue of each patient is a control
- Diffuse disease: references of normality do not exist (age, location, T1 & T2 parameters dependent)

MRS APPLICATIONS

- Inborn error of metabolism
- Neoplastic mass
- Non neoplastic mass
- Trauma

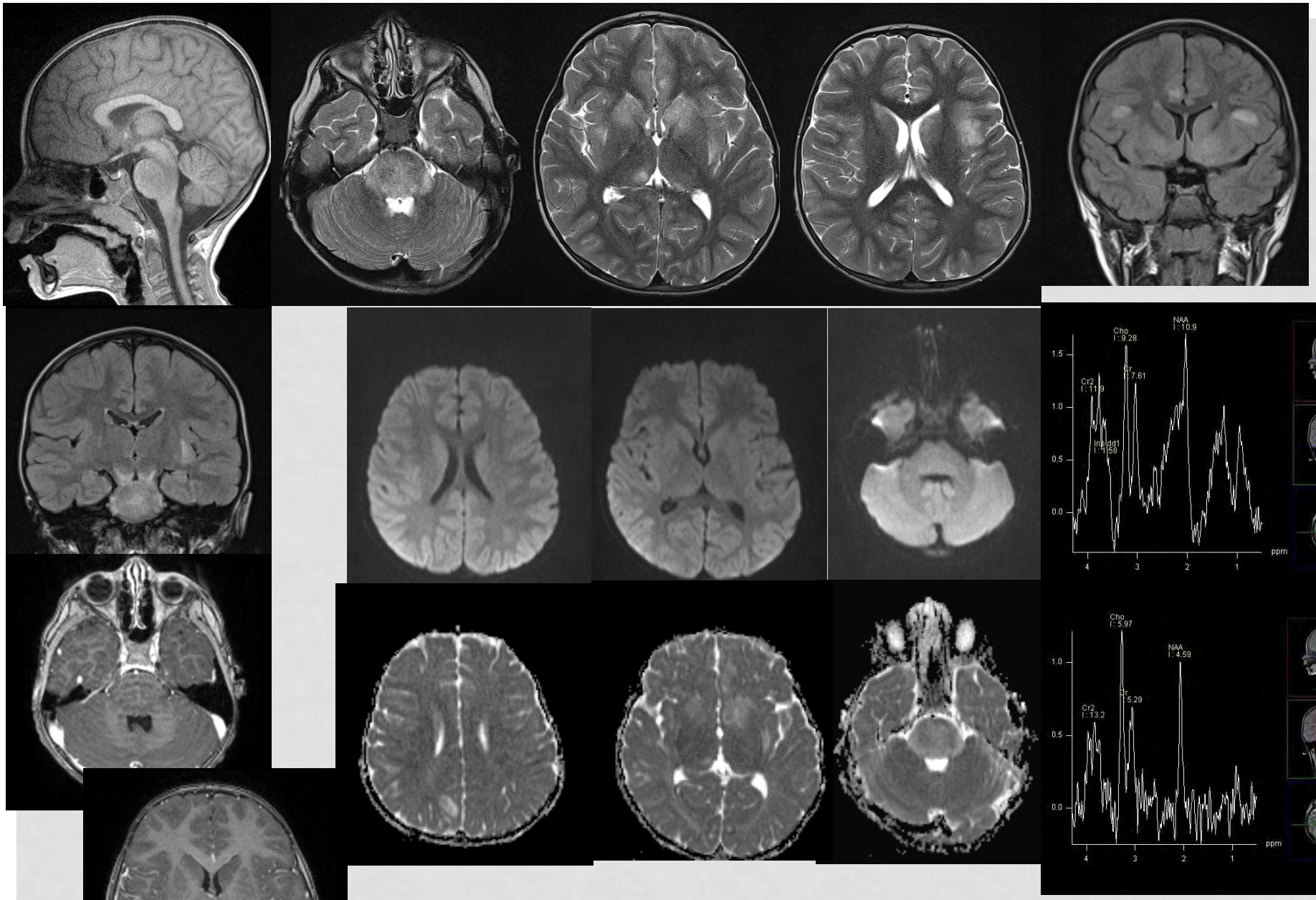




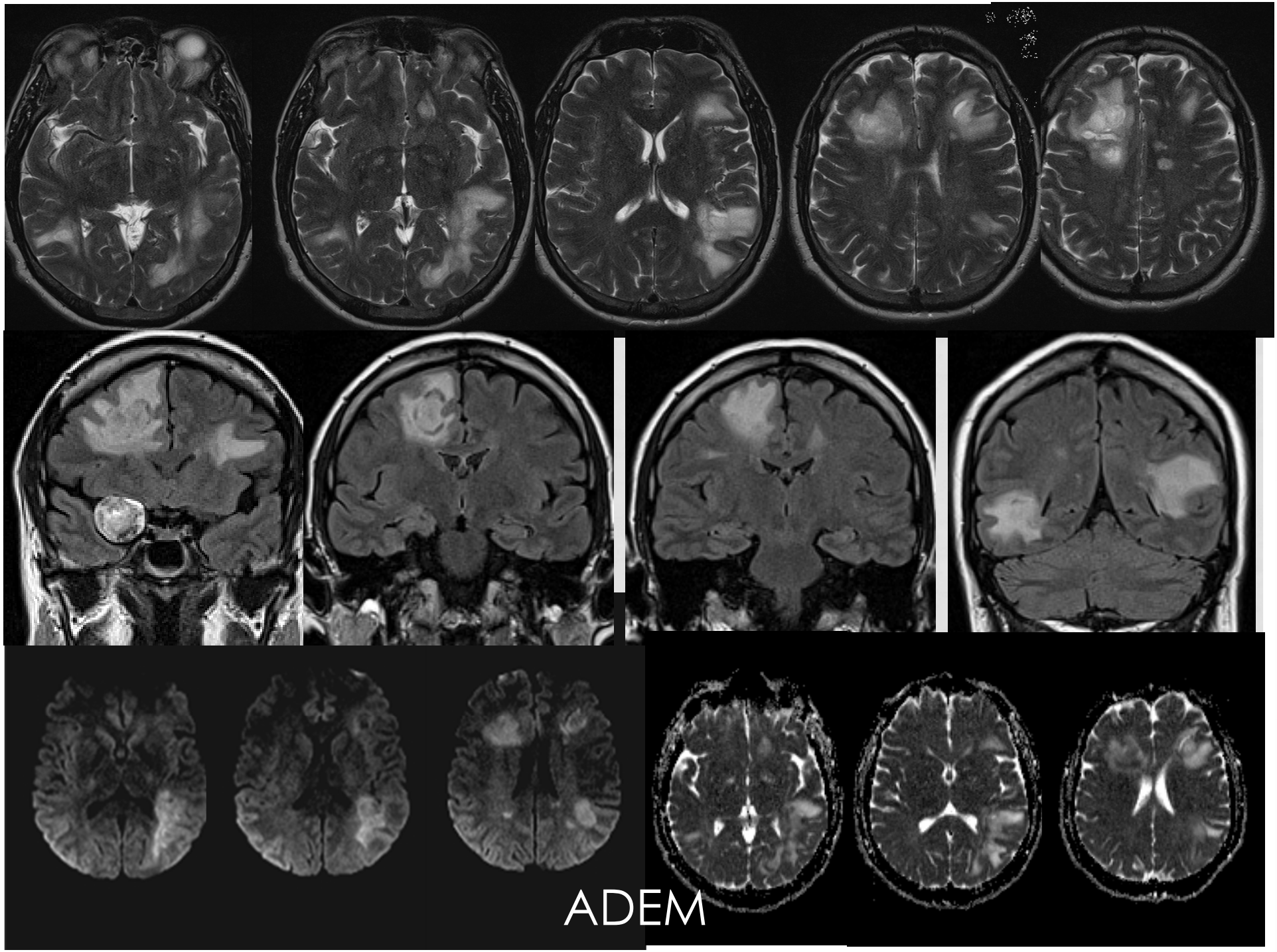
2. ACUTE VIRAL INFECTIONS OF THE BRAIN & OTHER ENCEPHALITIS

- differences between Europe, USA, Asia
  - Herpes encephalitis still the most frequent worldwilde
  - Japanese encephalitis in Asia
  - West-Nile virus in Africa and Middle East
  - St-Louis encephalitis in USA

- **Complications of a viral infection (or a vaccination) such as Acute Disseminated Encephalomyelitis (ADEM), an autoimmune reaction to the virus**
- **Immune mediated encephalitis**
- **can mimic viral infections on imaging**


Cho
I: 9.28
NAA
I: 10.9
Cr2
I: 11.9
Br
I: 7.61
Inositol1
I: 9.58
1.5
1.0
0.5
0.0
4
3
2
1
ppm
Cho
I: 5.97
NAA
I: 4.59
Cr2
I: 13.2
Br
I: 5.29
1.0
0.5
0.0
4
3
2
1
ppm

ADEM

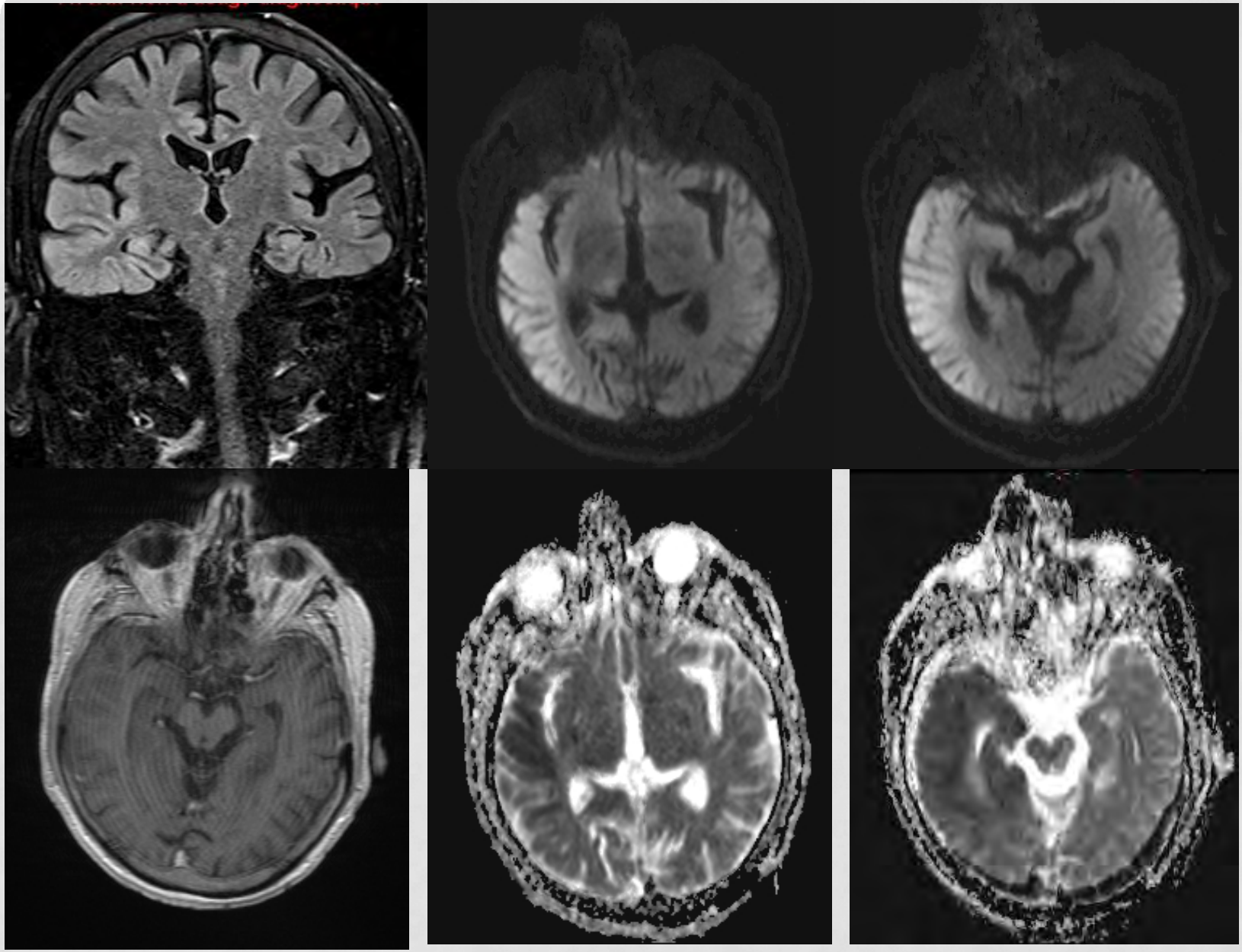

ADEM

CASE 1

- 53 years male
- Confusion, agitation
- Fever 38°5
- ICU: Intubated & ventilated

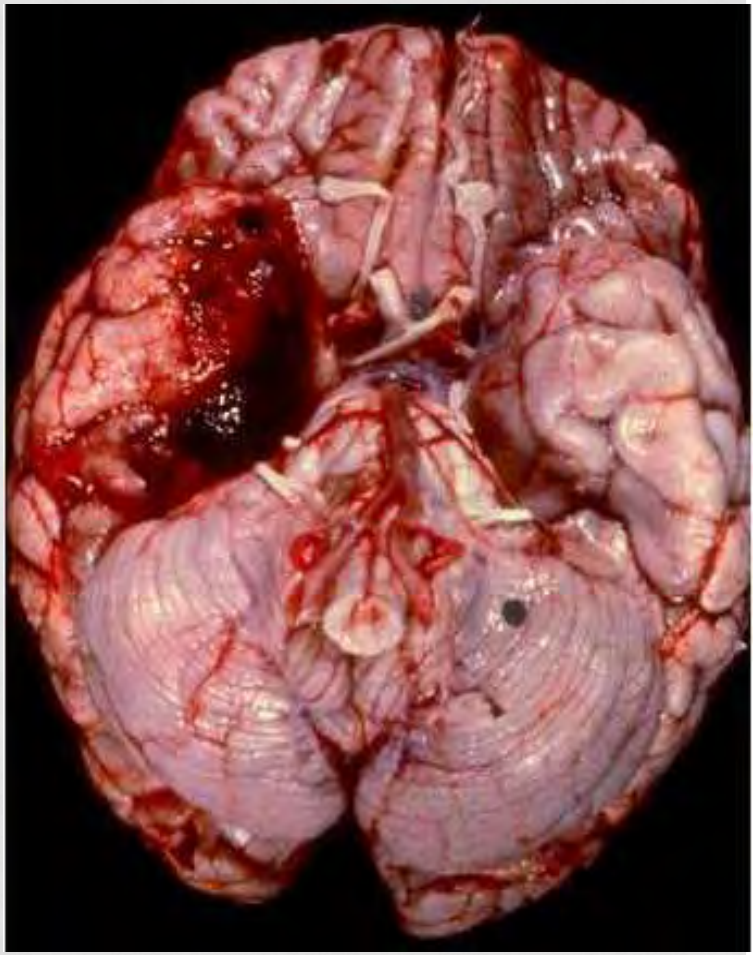
- Questions?

- 1. stroke
- 2. ADEM
- 3. vasculitis
- 4. herpes encephalitis
- 5. MELAS

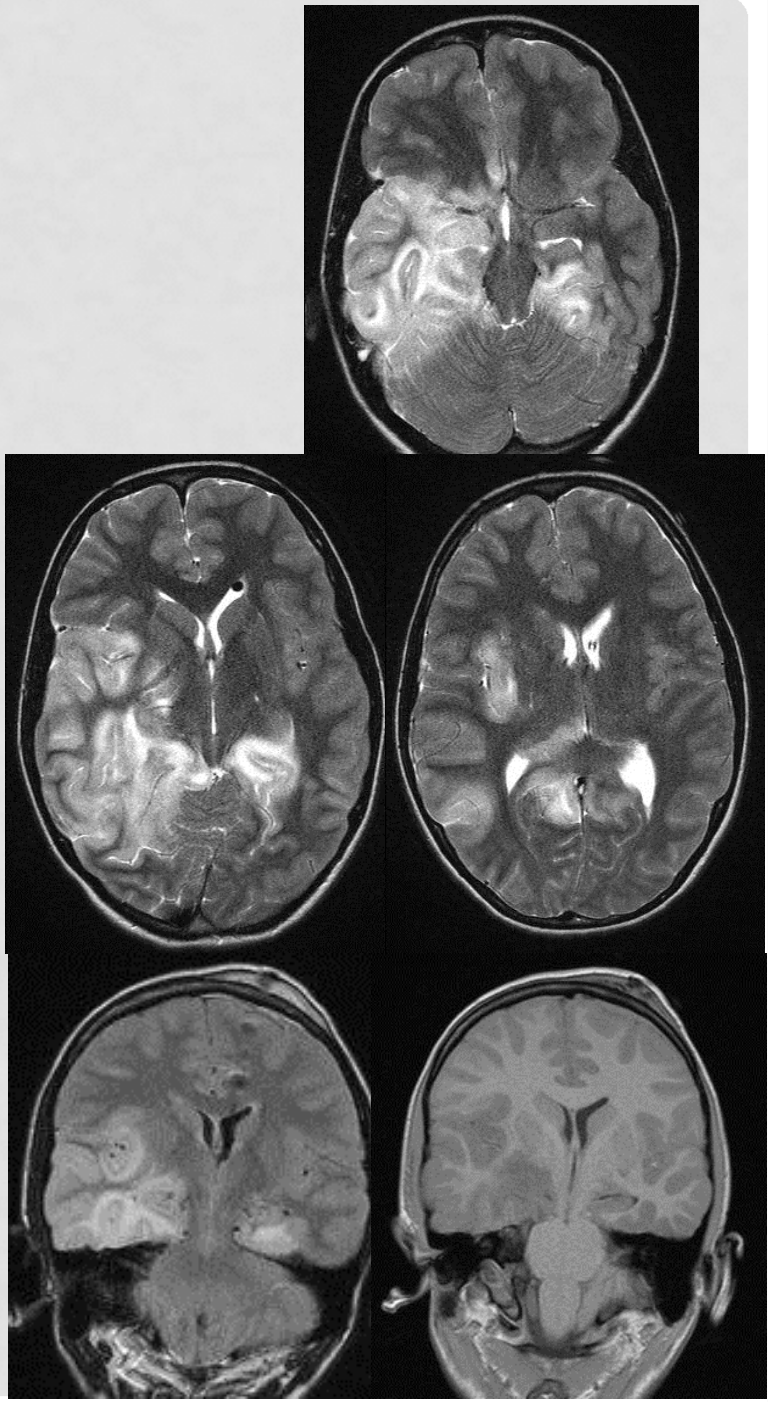


HERPES SIMPLEX ENCEPHALITIS

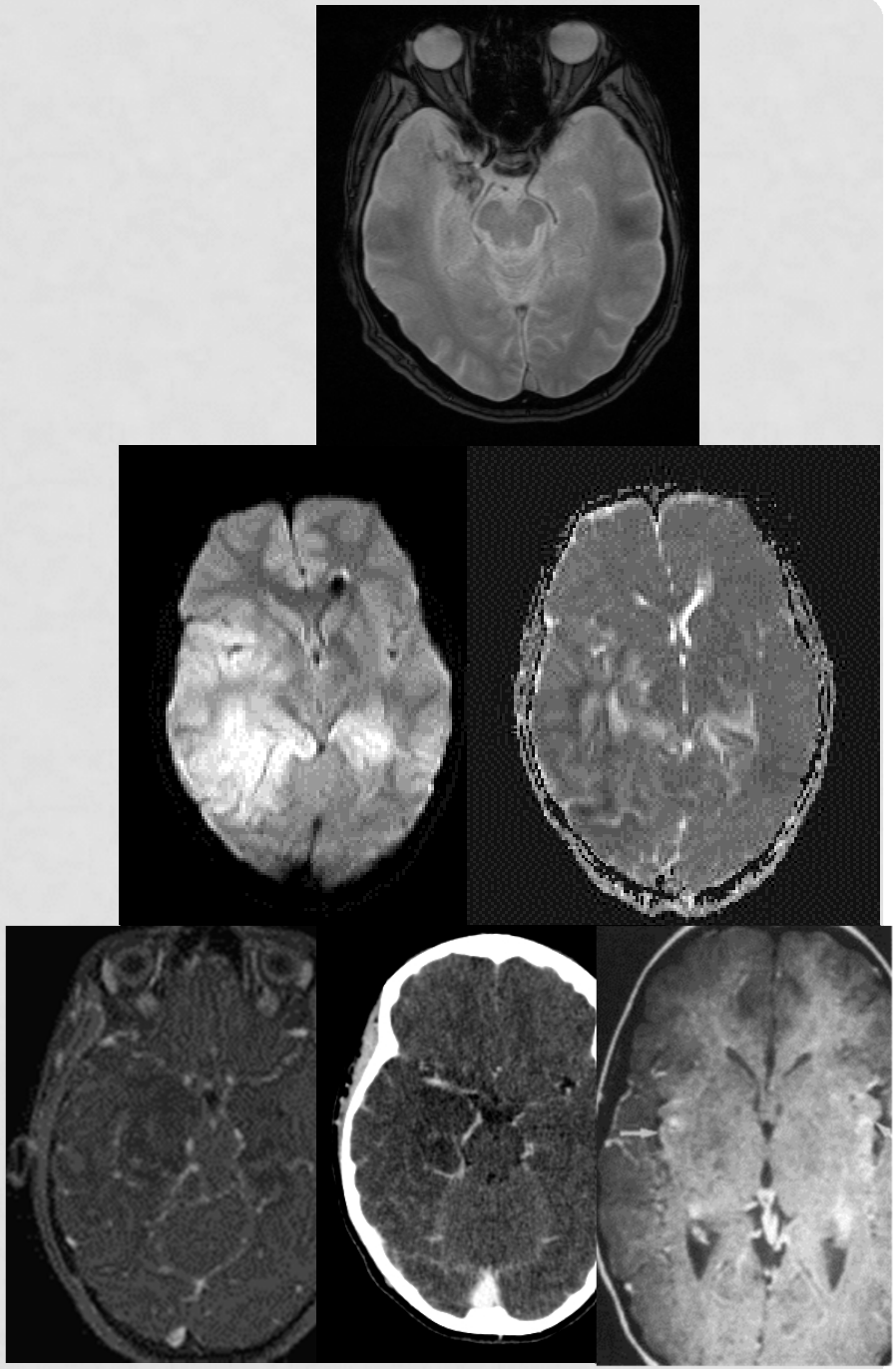
- Pathology: Necrotizing encephalitis
- 95 % Herpes Simplex virus type 1
- Mortality 50-70%
- Adults - Reactivation of latent infection - at age >50 years
- Children - Primary infection - at age 6 months to 3 years



- Lesions are typically located in the temporal lobe
  - medial before lateral
  - Extra-temporal regions: 40% of children (only 9-15% of adults)
- Extension to
  - insula
  - Orbital surface of frontal lobes
  - Cingulate gyrus
  - Thalamic lesions: children with parietal or opercular lesions (probably related to the existence of numerous thalamo-cortical connections)
- Asymmetric brain damage
  - Unilateral or
  - Bilateral
- GM first then WM

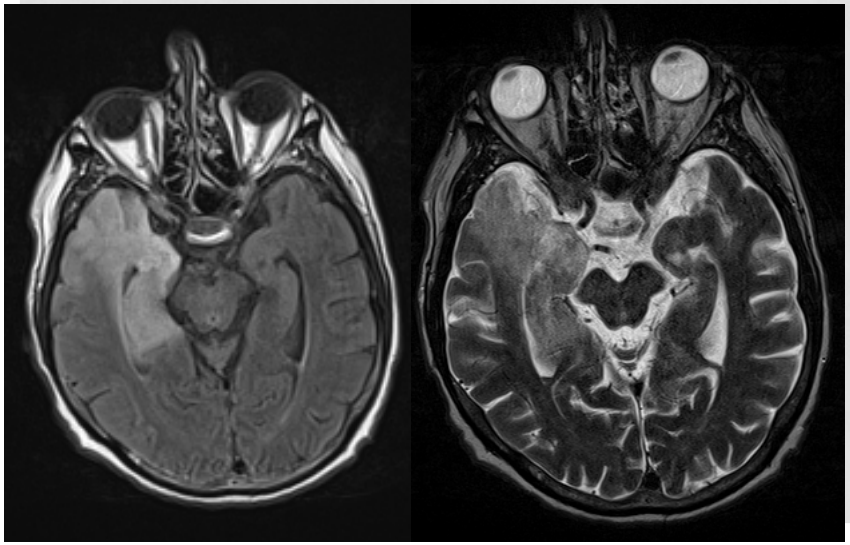


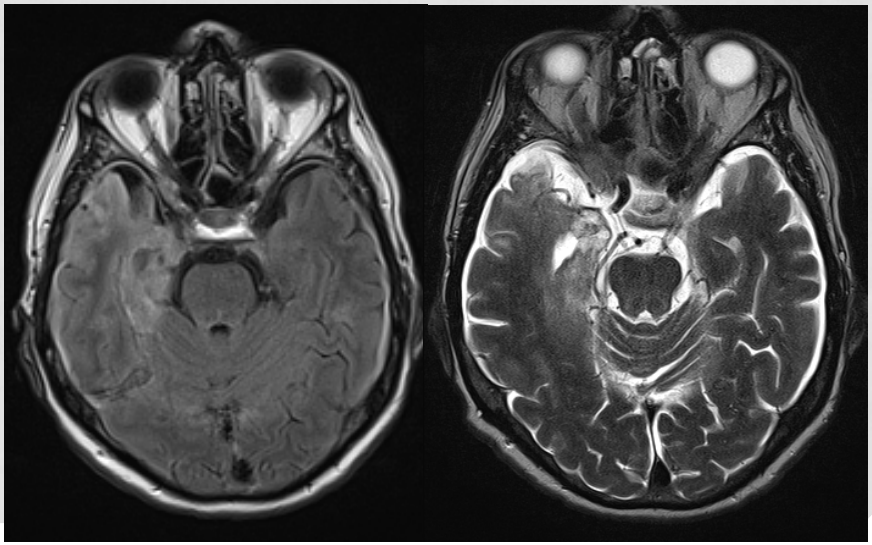
- Lesions tend to be hemorrhagic
- In the acute setting diffusion restriction can be seen
  - Low ADC in severe form
- Contrast enhancement
  - Variable
  - is not seen in the early stage of disease but occurs later
  - typical gyriform pattern of enhancement

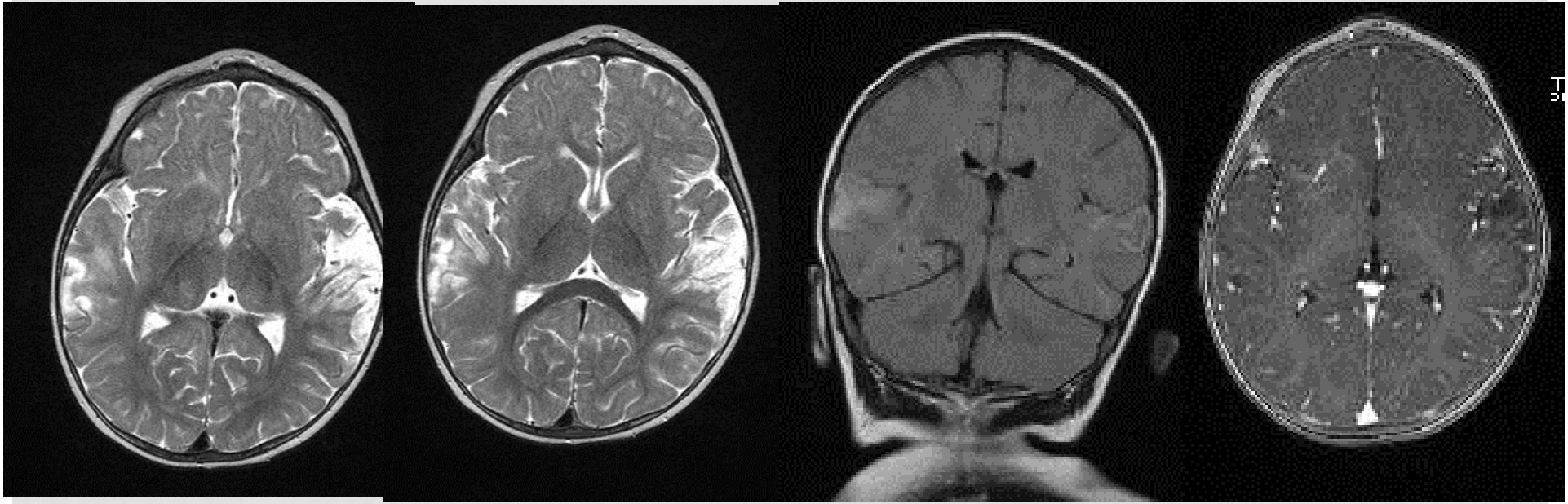


- MRI abnormalities may precede PCR detection of HSV
  - PCR sensitivity 96 %, specificity 99 %
    - In children specificity is lower 70 % to 75 %, virus absent in the CSF at the early phase
- **Normal imaging does not exclude DG**
- Rapid necrosis even with treatment

Follow up 15 days

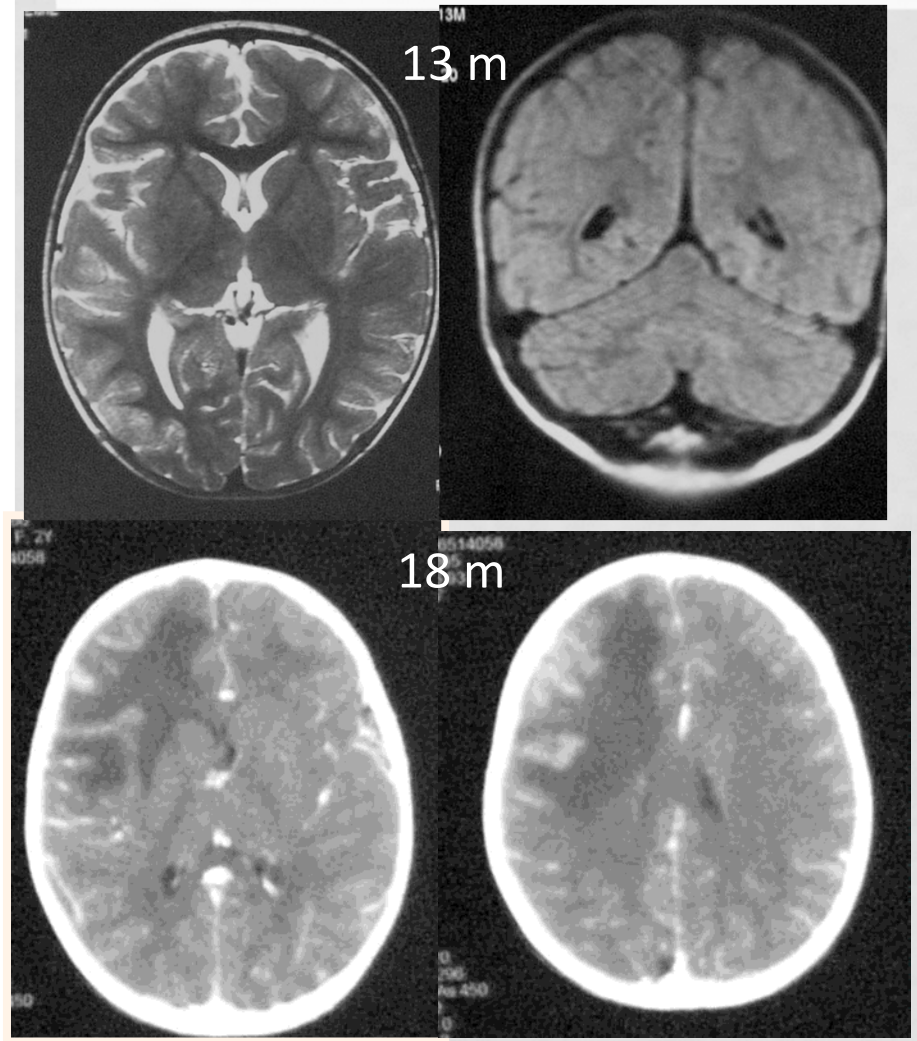






Sometimes the mesial temporal area is not involved

CASE 2



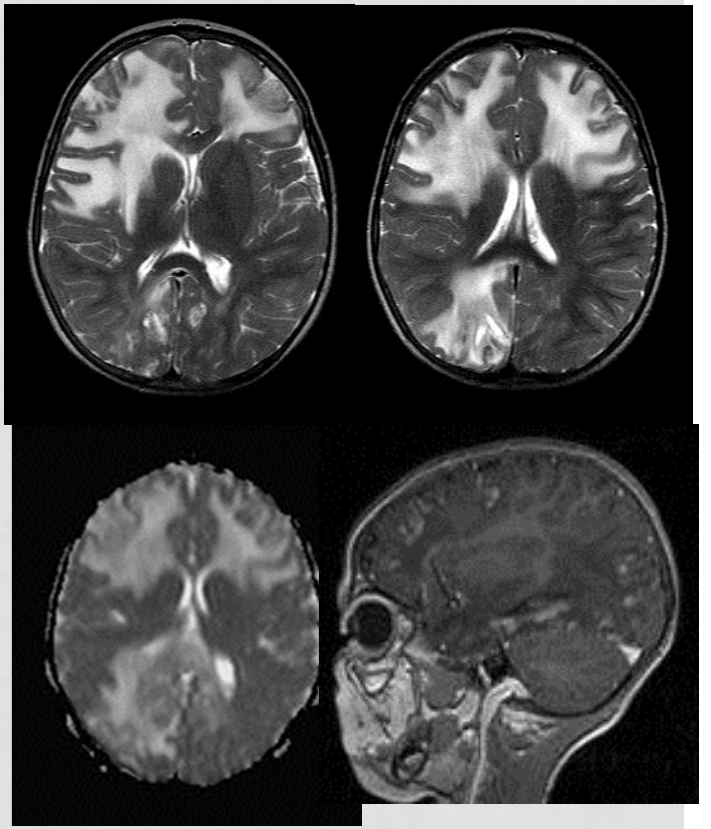
- Questions?

- 1. Gliomatosis
- 2. MS
- 3. Acute recurrence of HSV encephalitis
- 4. Stroke
- 5. toxic/metabolic

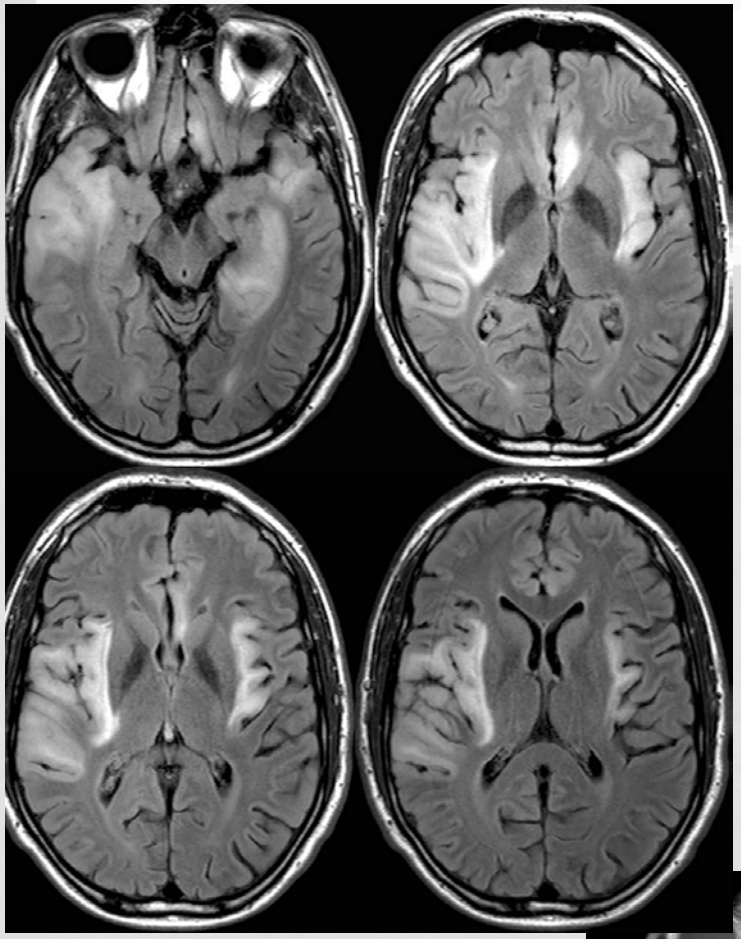
HSV ENCEPHALITIS
ACUTE RECURRENCE

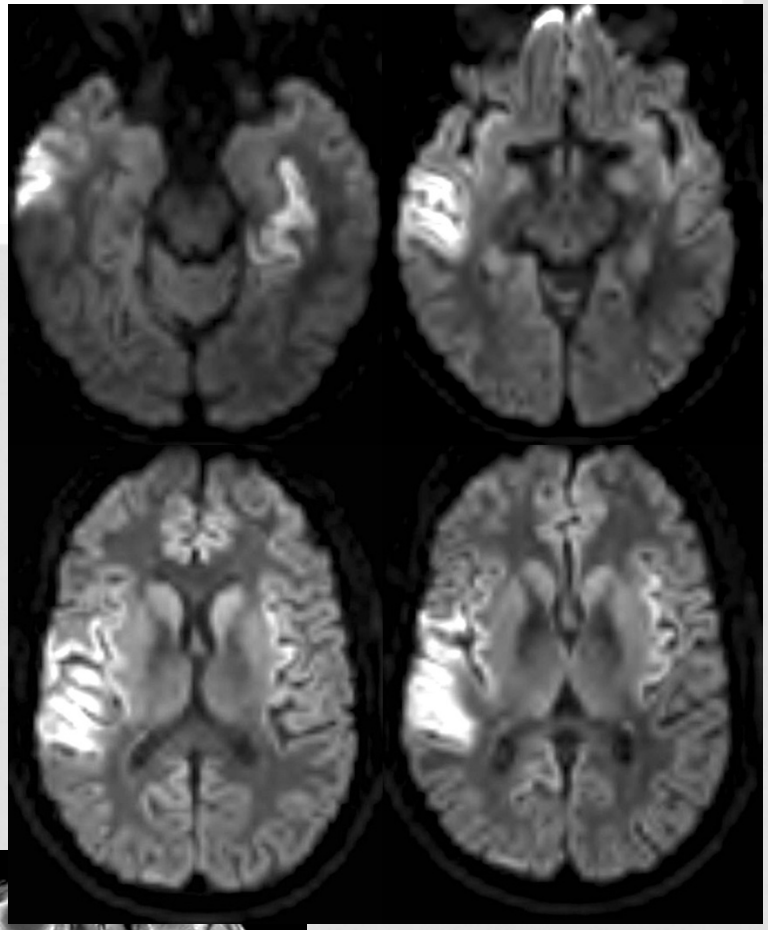
- 5 % to 30 %
- Viral replication or immune inflammatory response
- several days after end of treatment, months to years after initial episode
- New lesions at a distance from initial lesions: **Multifocal involvement of WM**
- **Infants and young children**
- **Genetic susceptibility for this secondary response**
- Three mutations have been identified in the immunological pathway of interferon-a production that do not modify the severity of the viral disease in general, but do alter the susceptibility to encephalitis

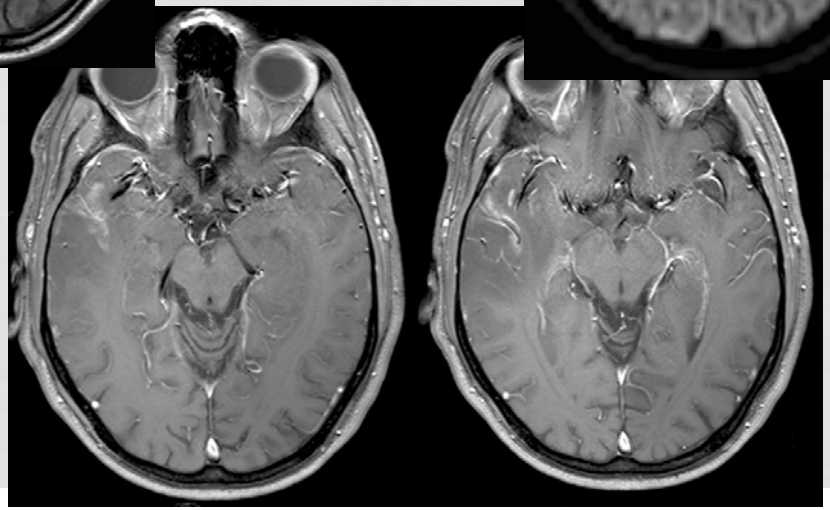
20 m



CASE 3







Courtesy Pr G Wilms
Leuven, Belgium

- Questions?

- 1. Herpes encephalitis
- 2. Gliomatosis cerebri
- 3. MELAS
- 4. ADEM
- 5. None of them

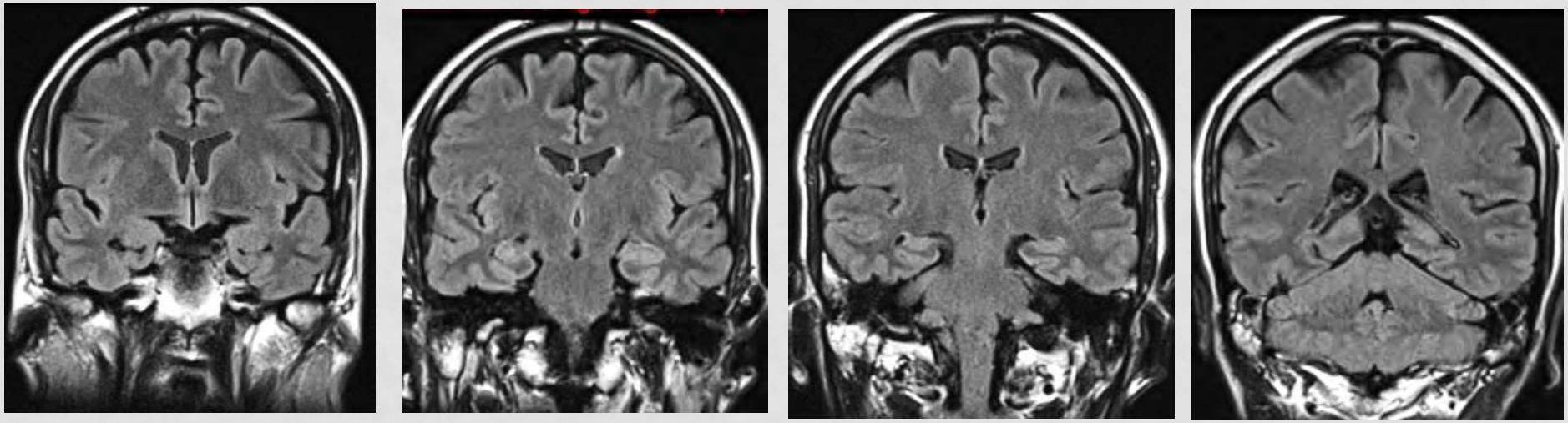
Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like episodes

- Is a mitochondrial cytopathy
- On MRI multiple cortical stroke-like lesions are seen, that can simulate herpes encephalitis

Sharfstein SR et al, Adult-onset MELAS presenting as herpes encephalitis. Arch Neurol. 1999

OTHER CAUSES OF SIGNAL ABNORMALITIES WITHIN THE TEMPORAL LOBE

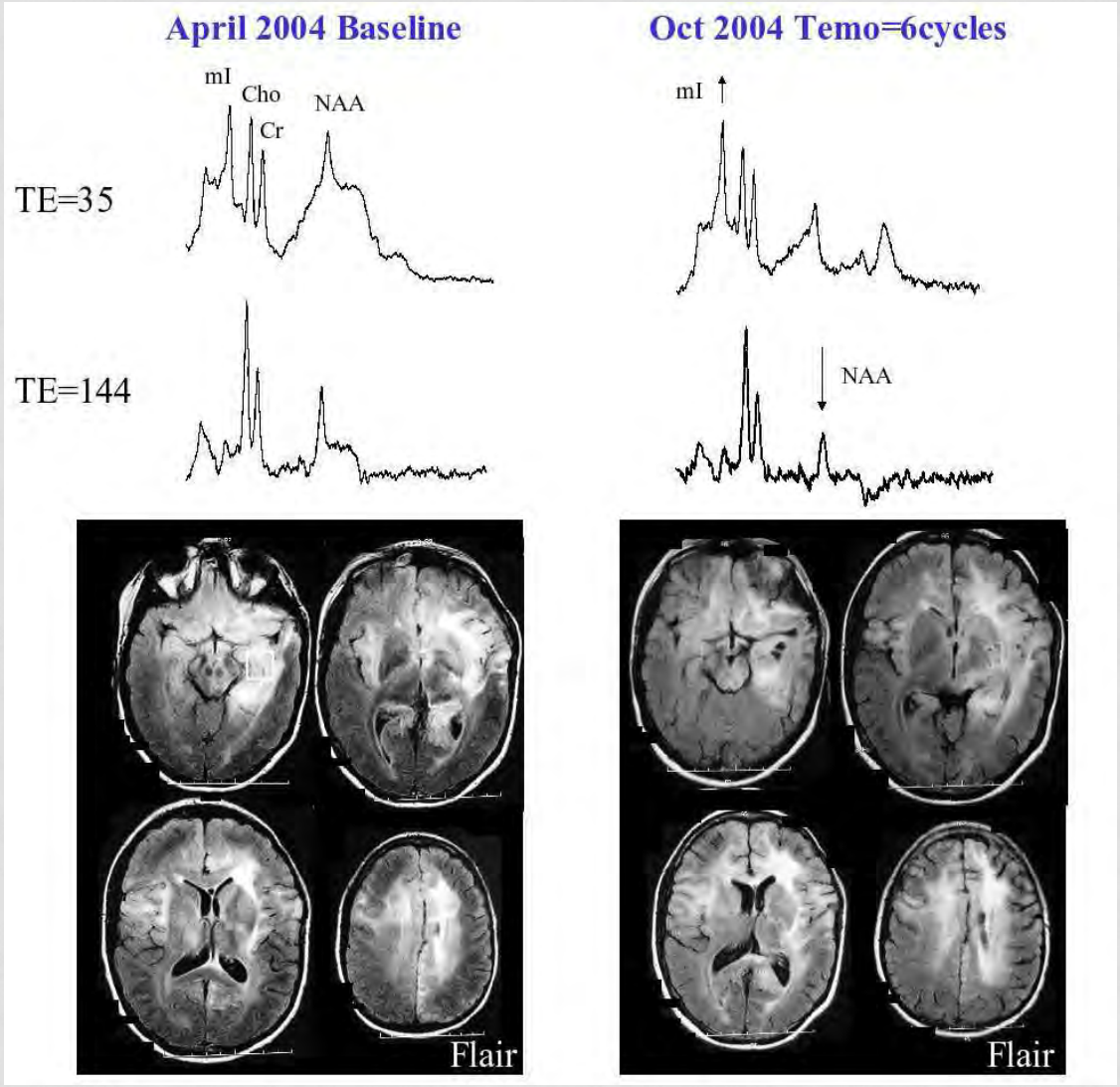
- Other infections: HHV6 (hematopoietic transplant), Whipple
- Tumor: DNET, gliomatosis
- Ischemia
- Limbic encephalitis
- Creutzfeld Jakob disease when involvement of the pulvinar is isolated



42 years male

- Whipple disease
- *Tropheryma whipplei*
- Digestive tract, heart & CNS

- Hippocampus
- Hypothalamus
- Thalamus
- Colliculi
- Periaqueductal GM
- Middle cerebellar peduncle

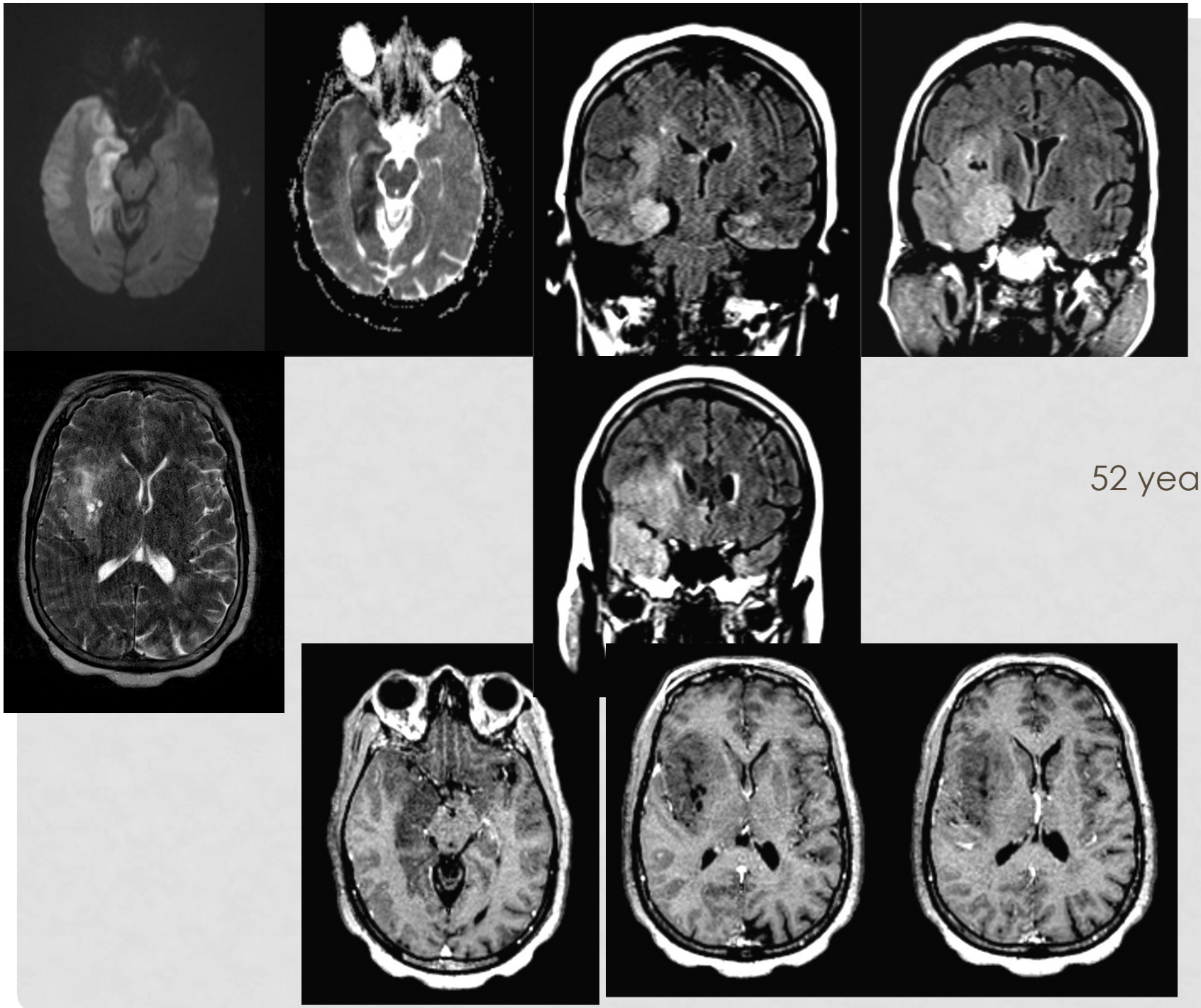


Gliomatosis

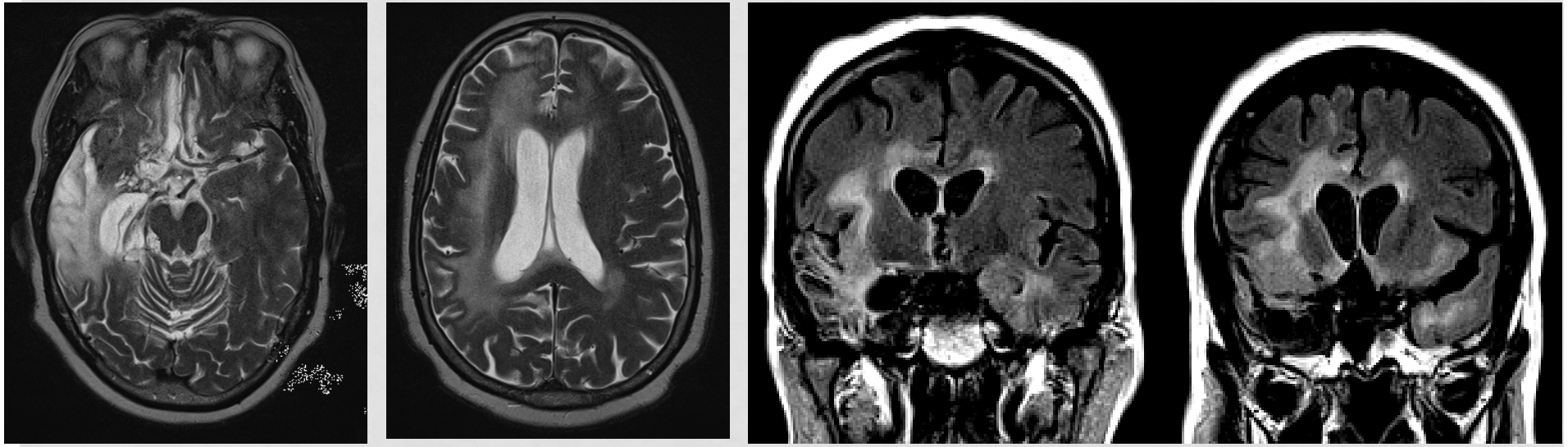
MRS HSV:
lactate ↑
no ↑ of Choline

- diffuse hyperI (FLAIR & T2 WI)
- involve at least 3 lobes
- considered to have a poor prognosis & poor response to treatments

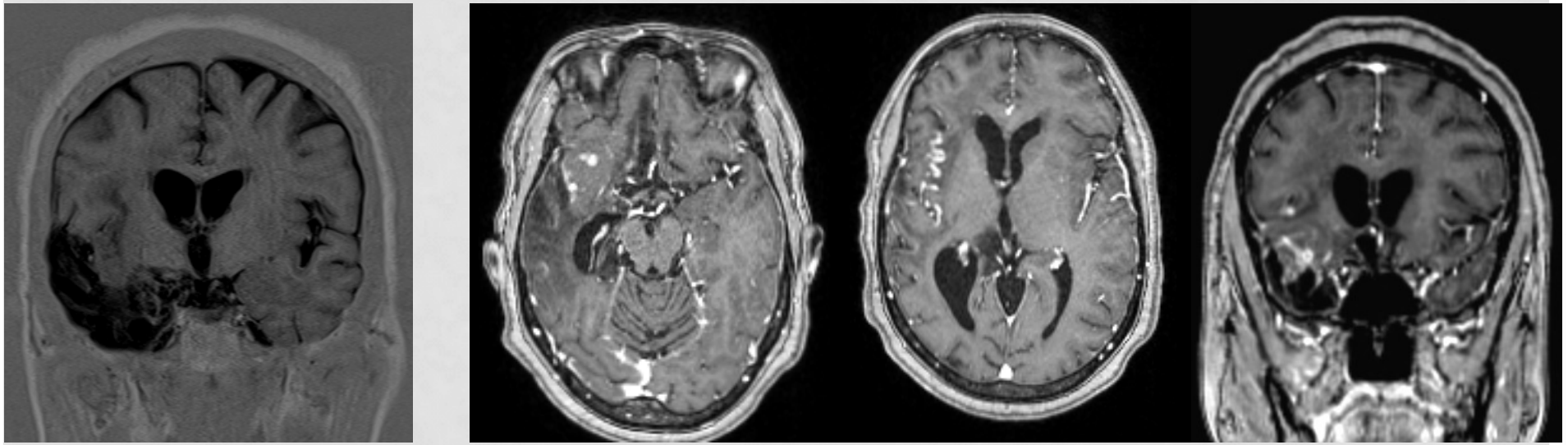
Courtesy Pr JM Constans
Amiens, France

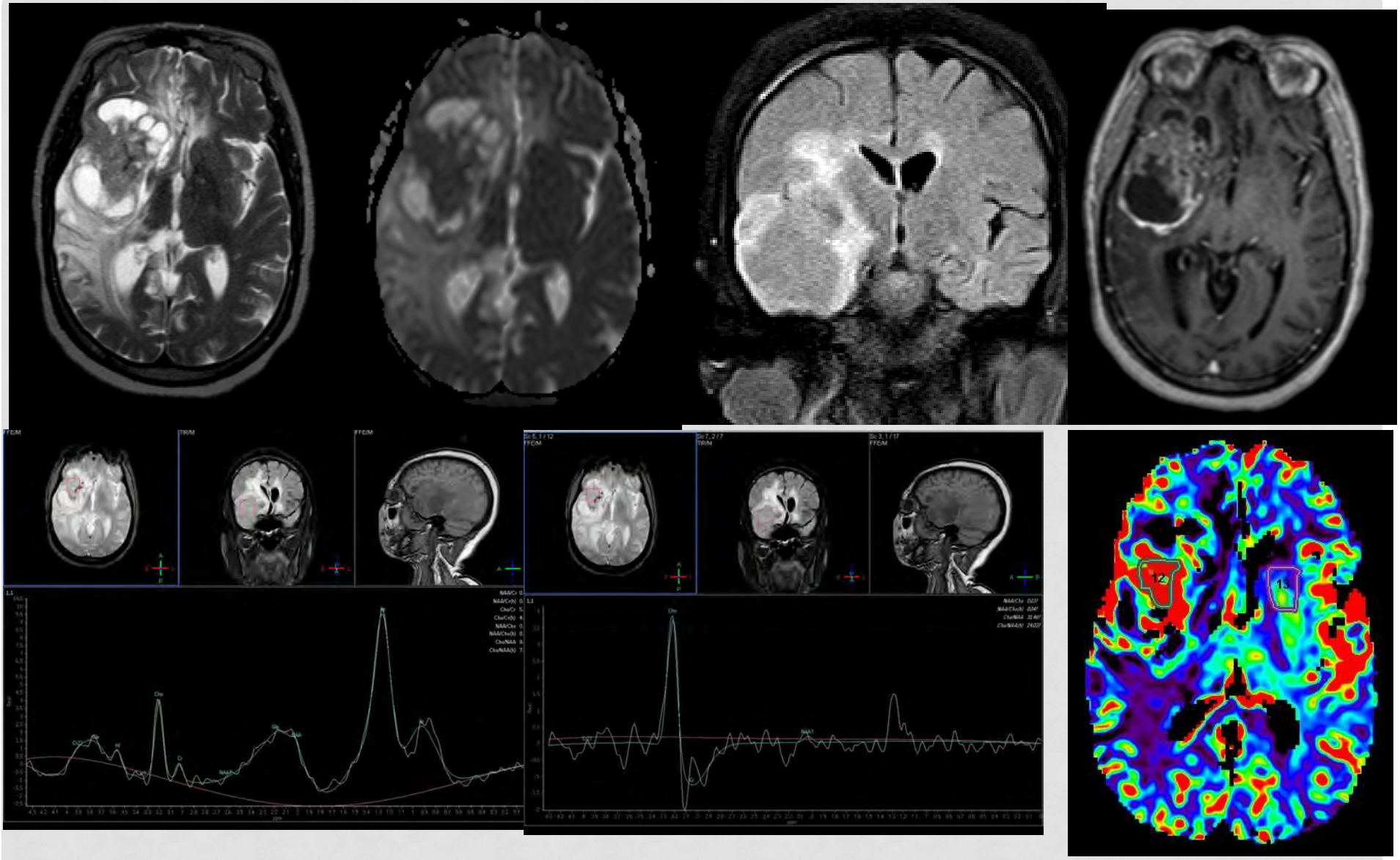


52 years

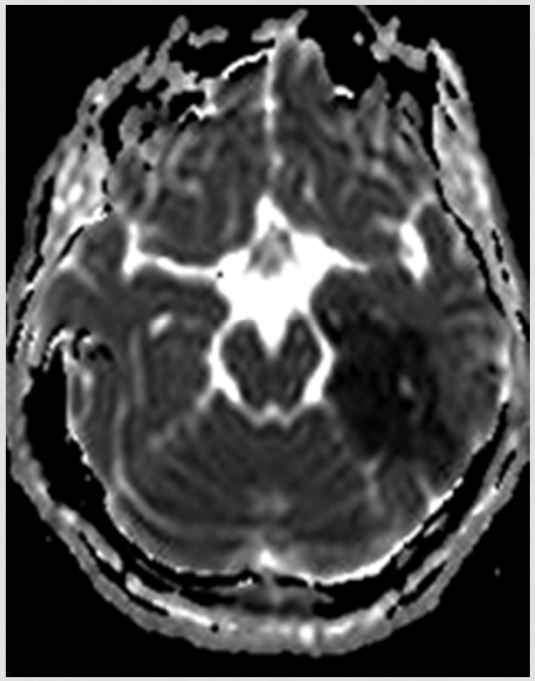


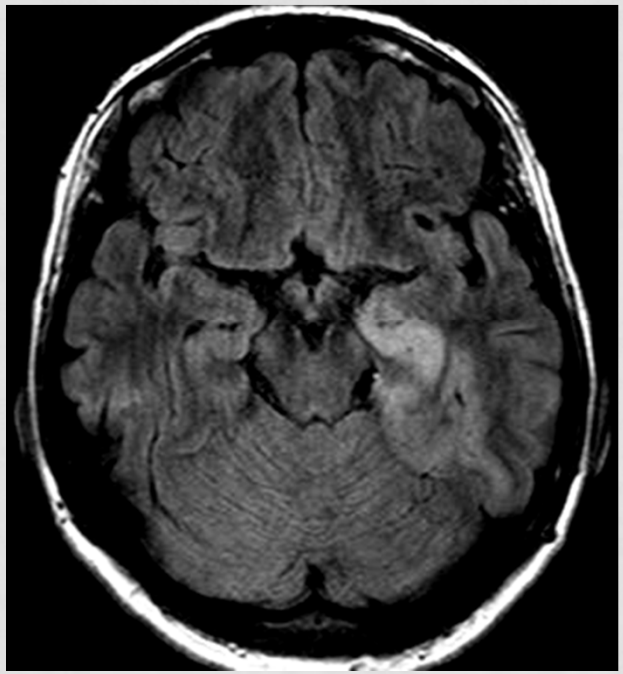
+ 9 months





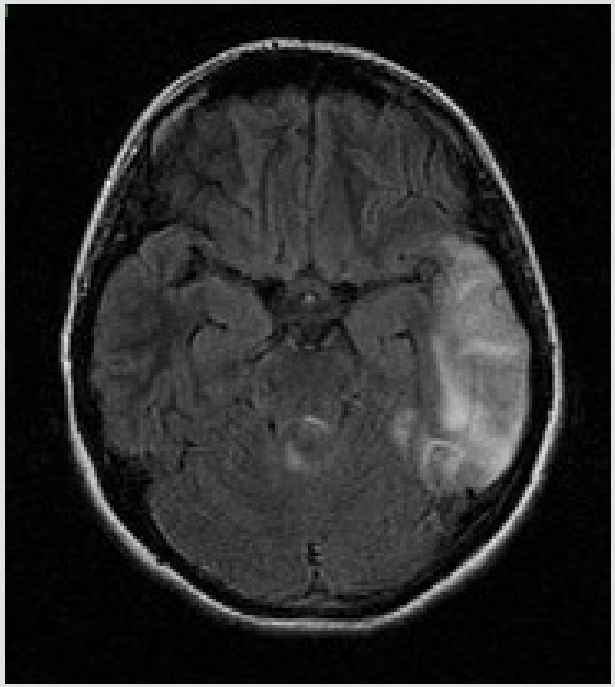
+ 2 years
Glioblastoma

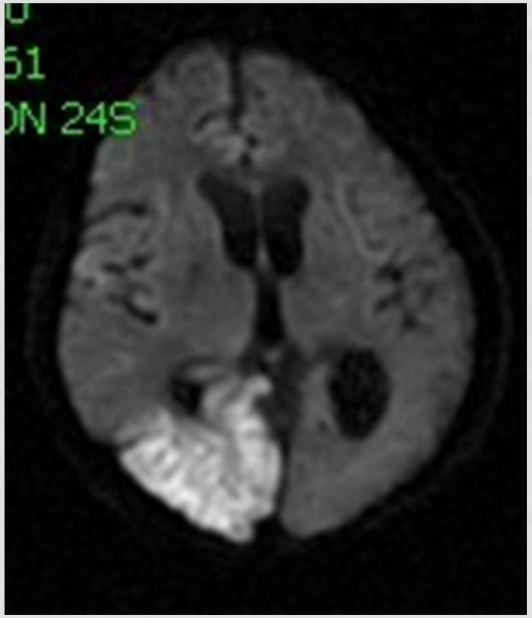




AChoA & PCA

MCA

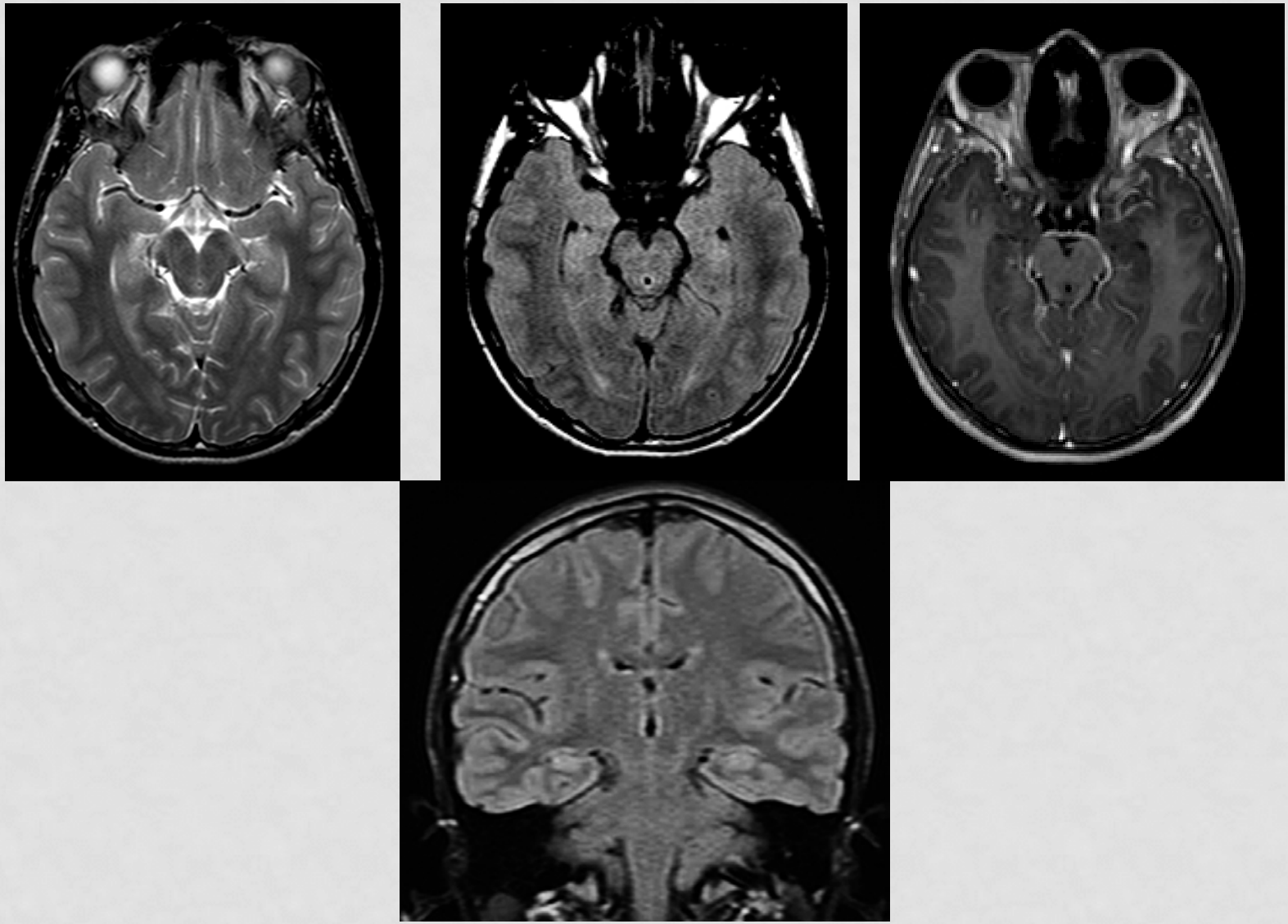




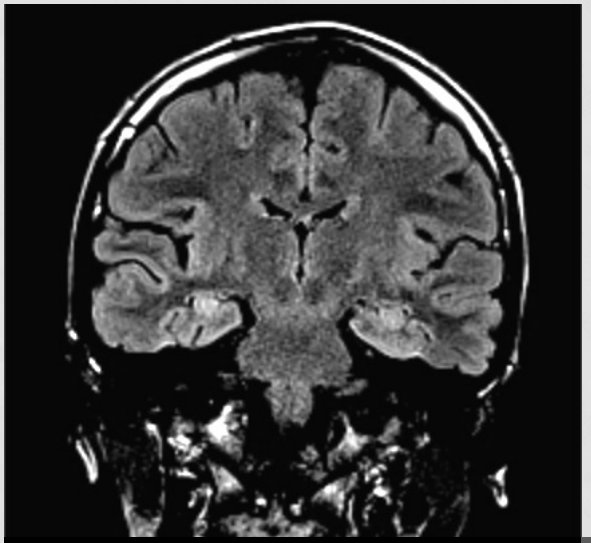
PCA

Ischemia

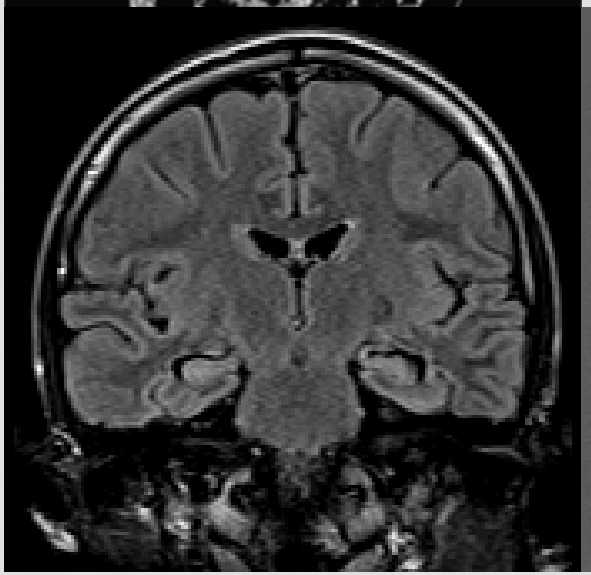
Courtesy Pr JM Constans
Amiens, France

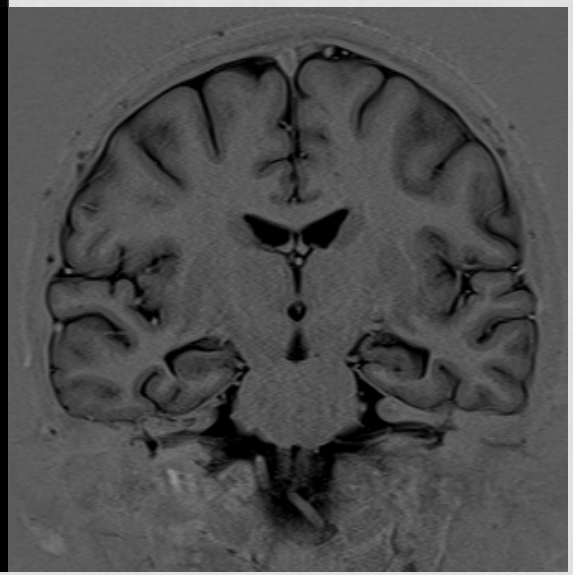


14 years nonneoplastic limbic encephalitis

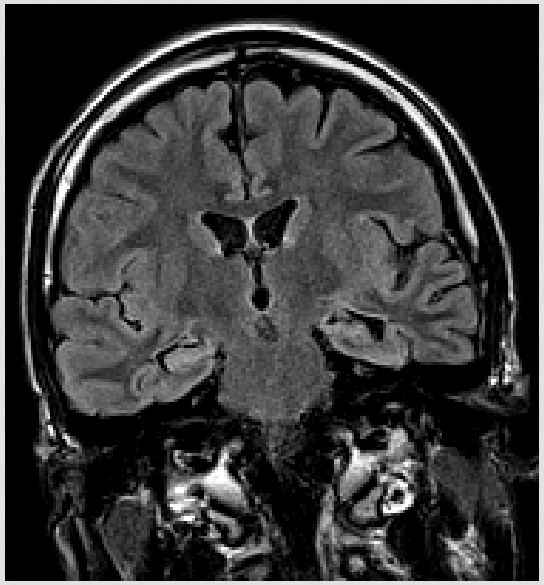


+ 7 days



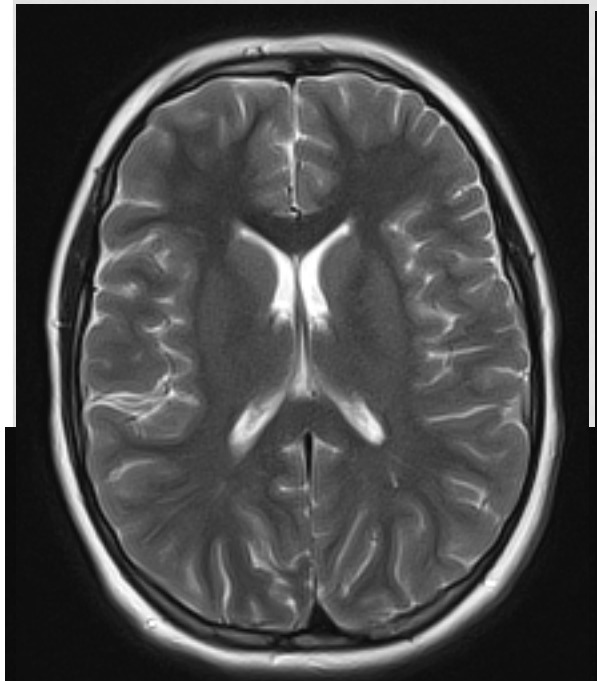
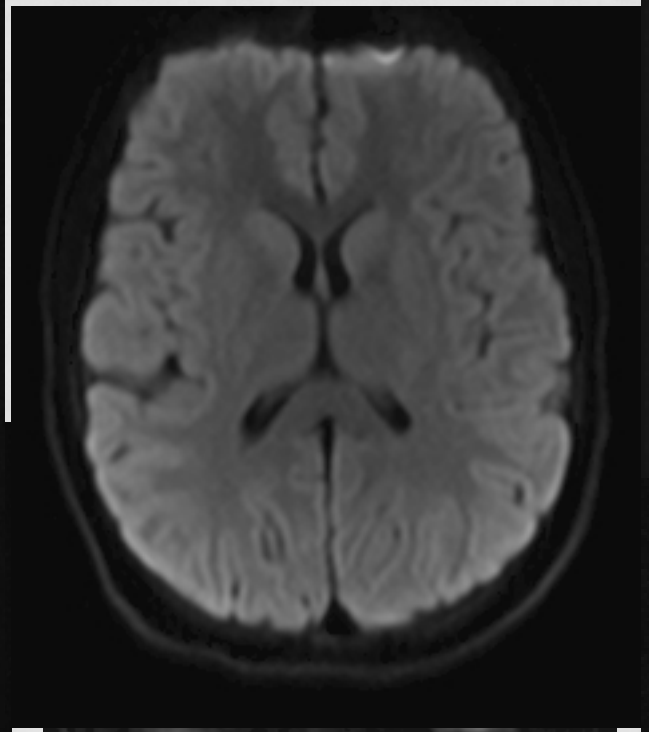
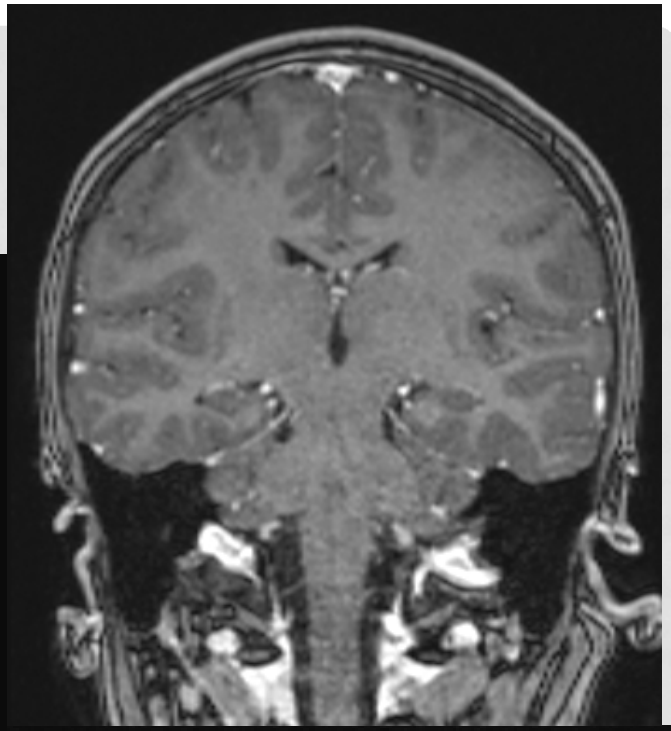
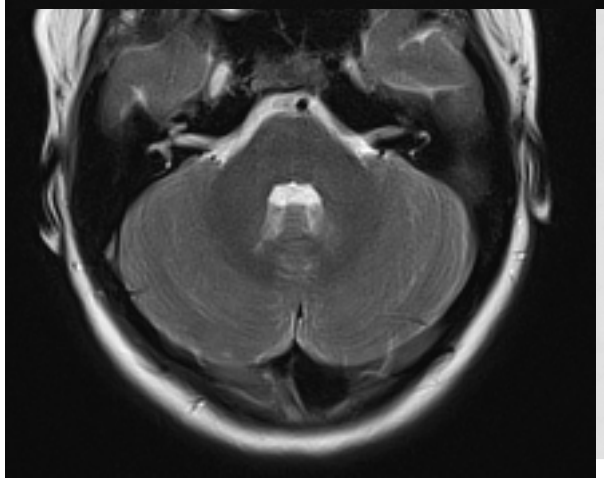
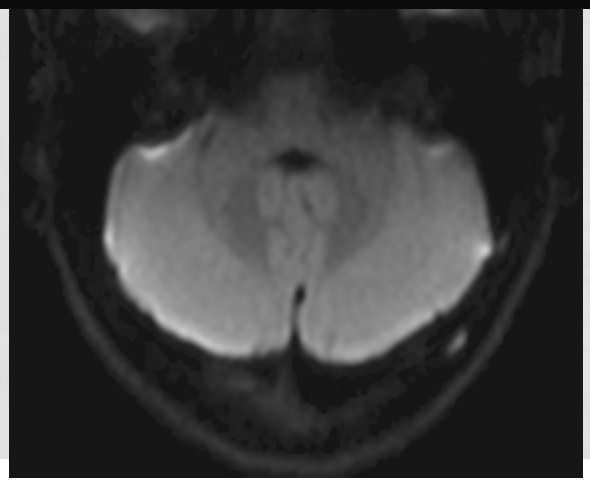
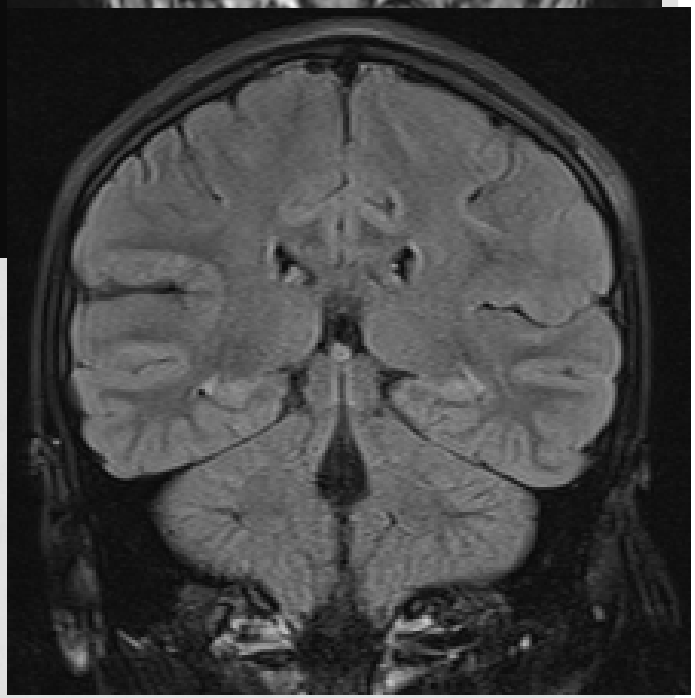


+ 1 month

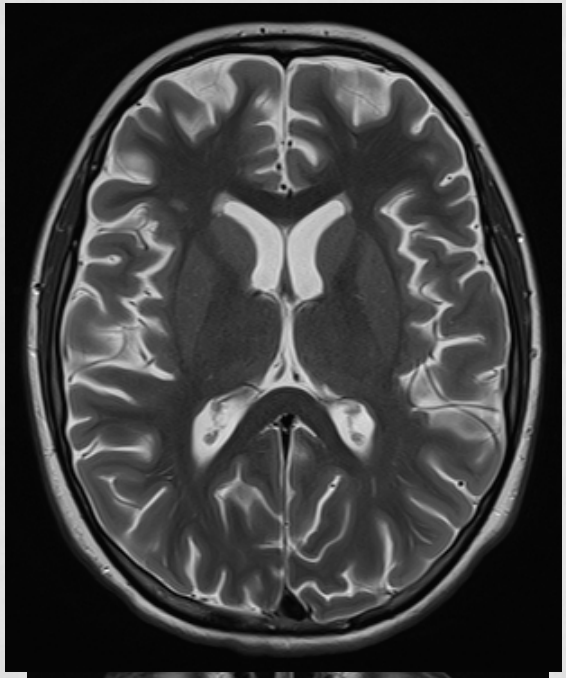


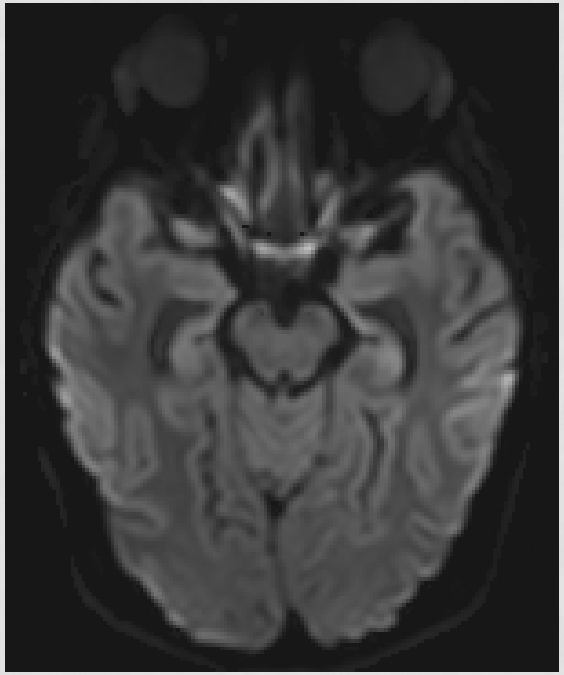
+ 1 year

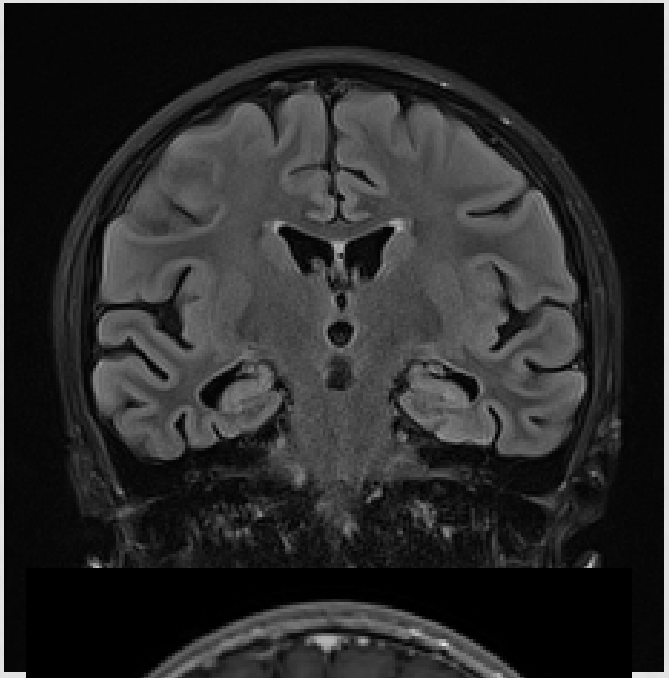
Anti NMDA-R
11y 5 months, Marta, acute ataxia, abnormal behaviour

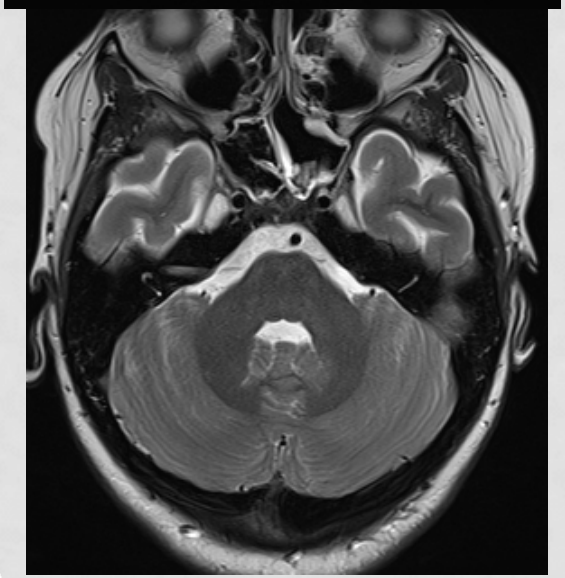







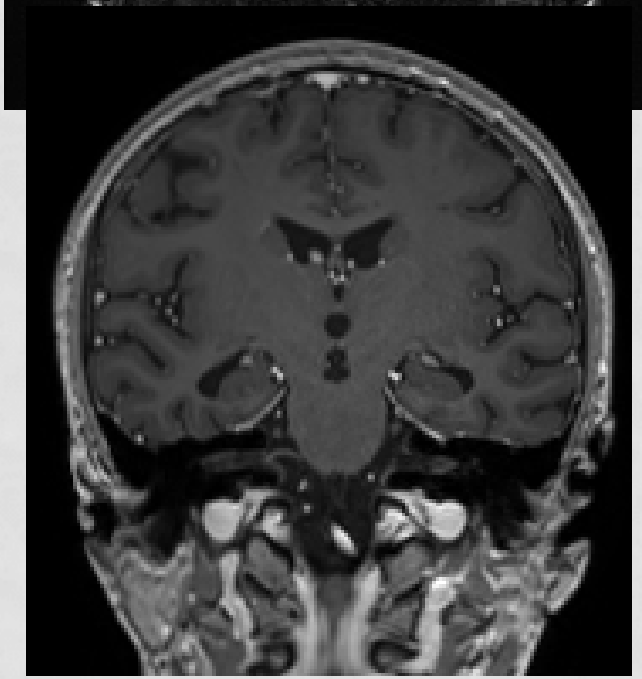
+ 2 months

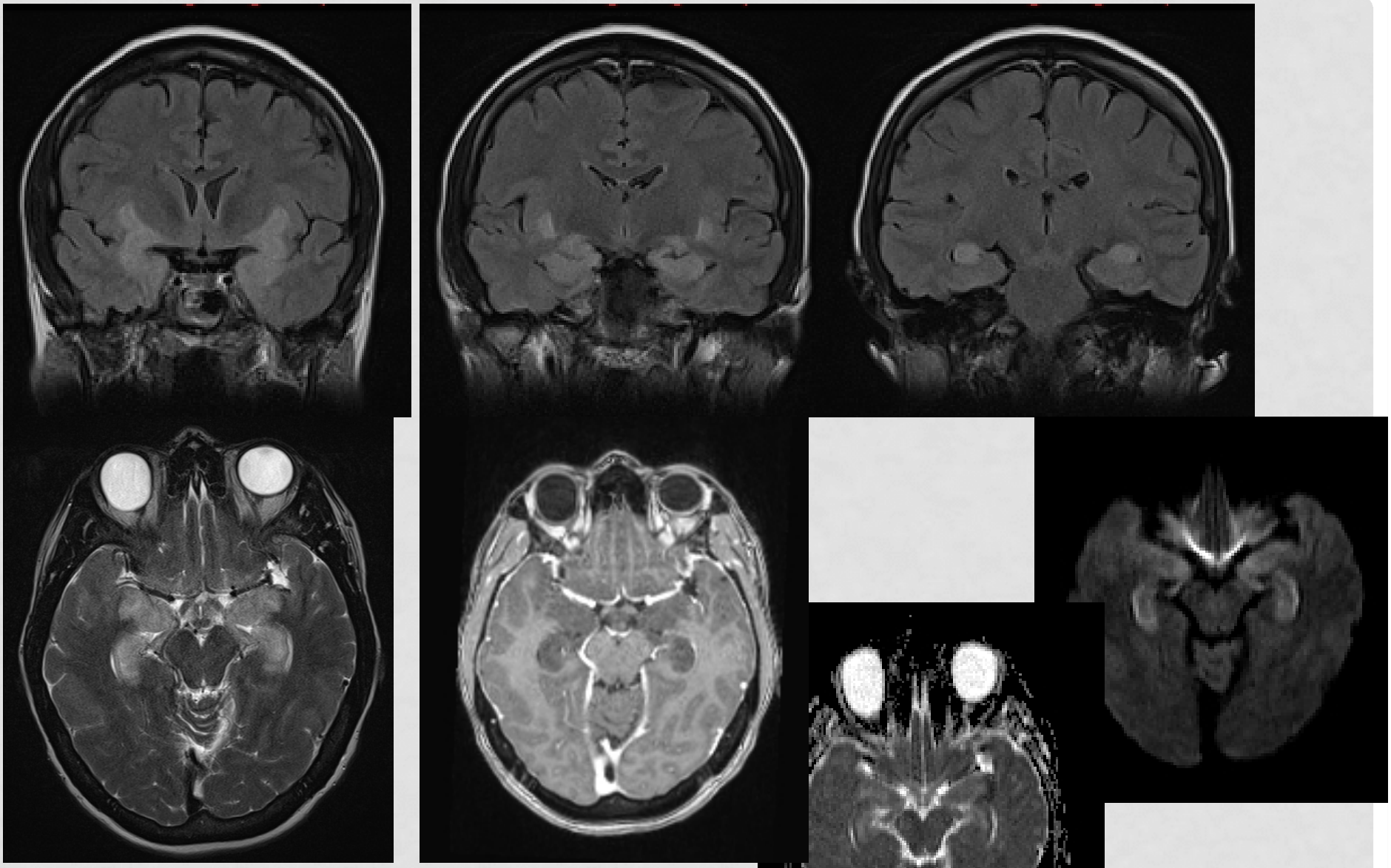






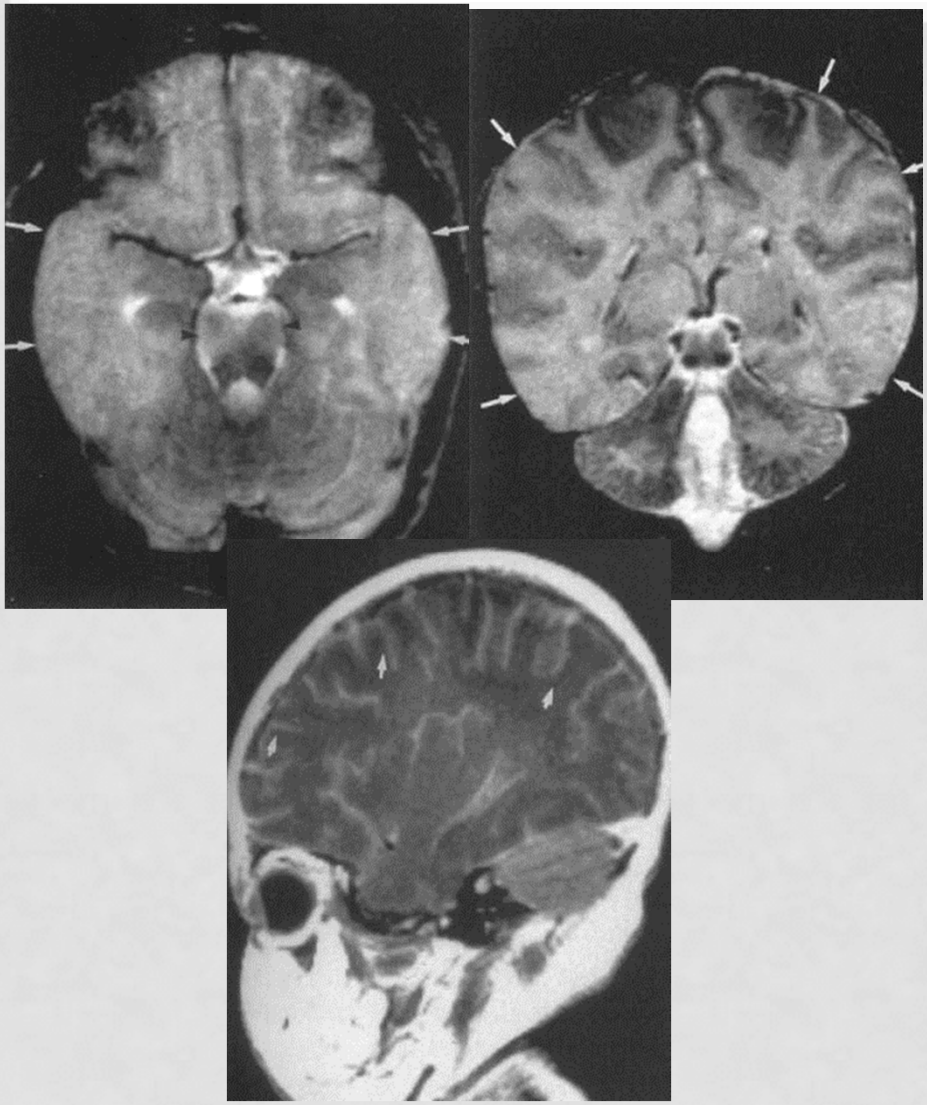






42 years, limbic E, headaches, memory loss, febrile convulsions
Lupus

CASE 4



- Questions?

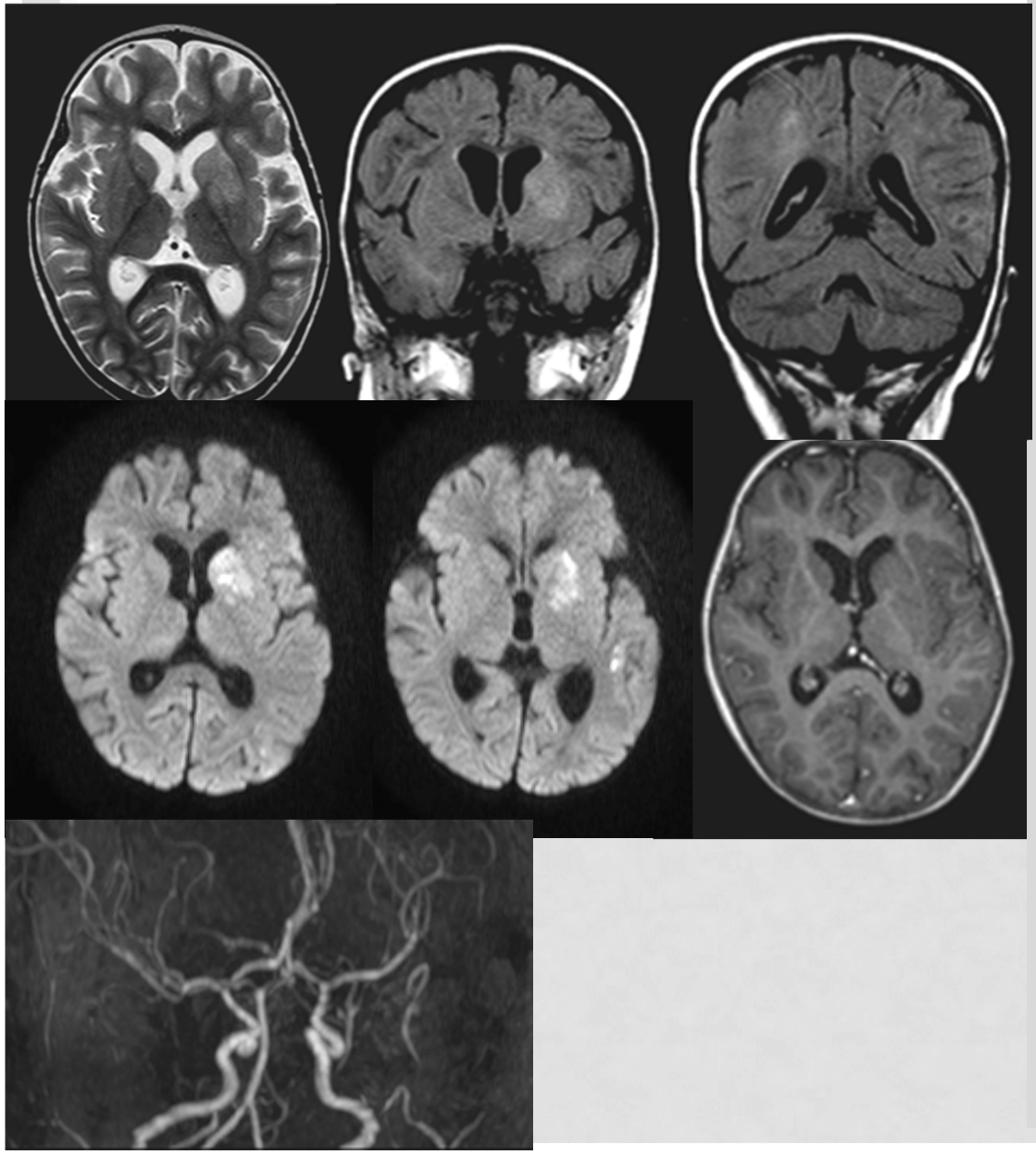
- 1. Birth aspyxia
- 2. Stroke
- 3. Tumor
- 4. Herpes encephalitis
- 5. Toxic-metabolic

HSV 2 ENCEPHALITIS

- Patchy, widespread areas of abnormal signal
- Primarily in the white matter
- Cortical gray matter
- Loss of brain substance occur rapidly , often as early as the second week

- Vaginal delivery of infected mother (type II herpetic lesions)
- Onset 1-2 weeks after birth
- Diffuse brain swelling
- Diffuse lesions of cerebral hemispheres

CASE 5



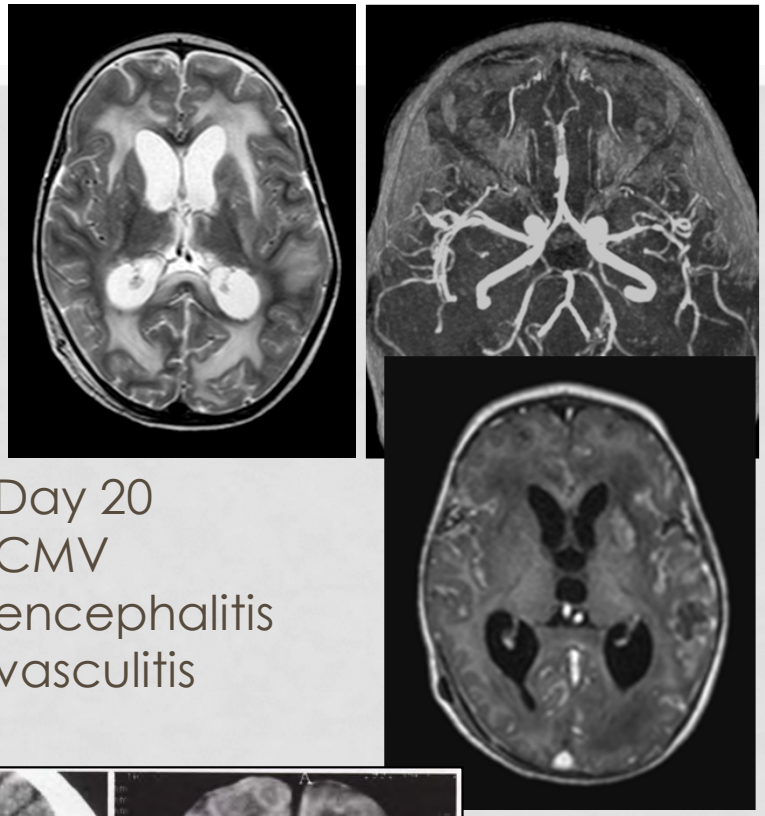
- 22 months old
- BMT
- Neurological deterioration
- No known pulmonary infection at day 1

- Questions?

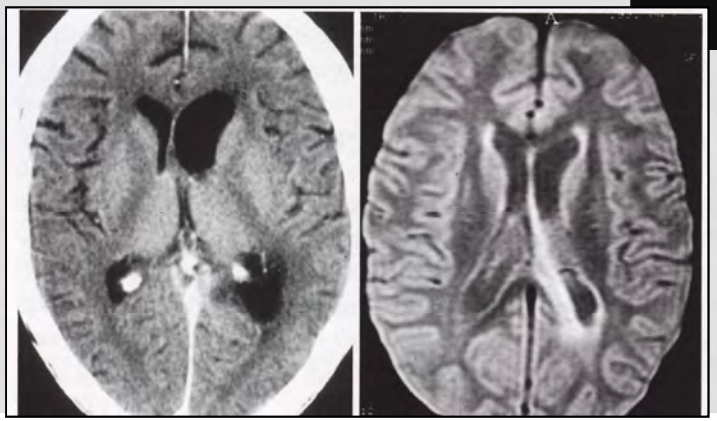
- 1. Stroke
- 2. Primary vasculitis
- 3. CMV encephalitis
- 4. ADEM
- 5. Metabolic

HERPES FAMILY: CMV ENCEPHALITIS

- CMV
  - GM & periventricular WM
  - Vasculitis
  - Immunocompromised
- Reactivation of latent infection
- Adults
  - Abnormal signal of ependyma (ventriculitis)



Day 20
CMV
encephalitis
vasculitis

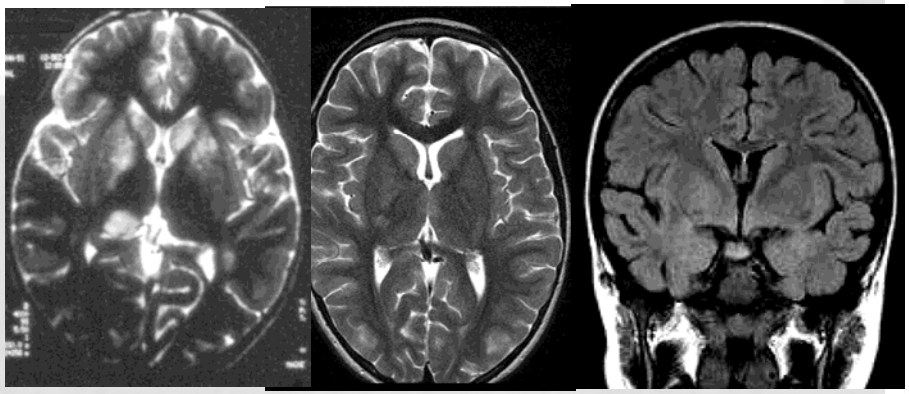


Pr JL
Dietemann
AIDS
CMV

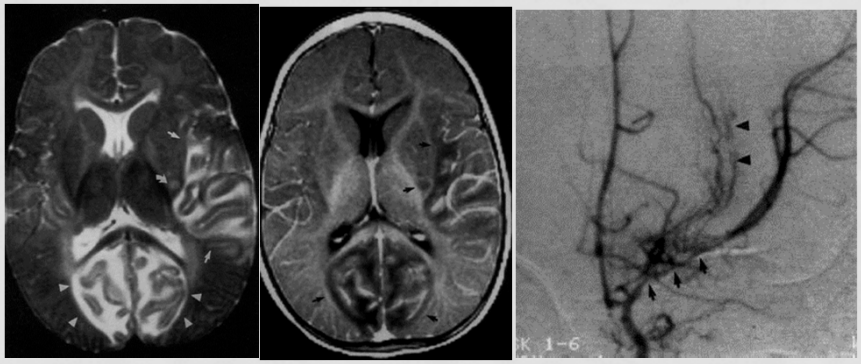
HERPES FAMILY ENCEPHALITIS

- Varicella zoster virus
  - WM & basal ganglia
  - Angiopathy: anterior circulation
  - Late stroke (months): basal ganglia
  - Acute ataxia

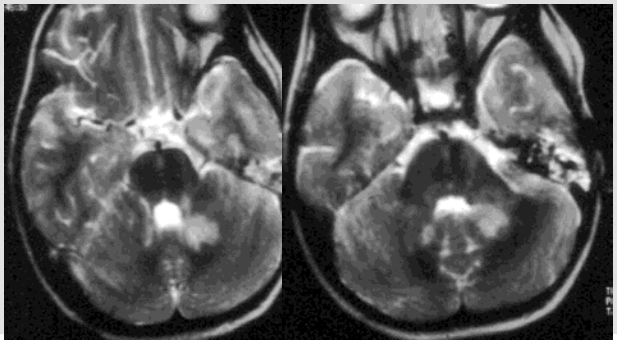
  - Adult immunocompromised patient
    - Multiple subcortical enhancing lesions



VZV



- EBV
  - GM & WM
  - Cerebral & cerebellum
  - myelitis
  - Immunocompromised



EBV

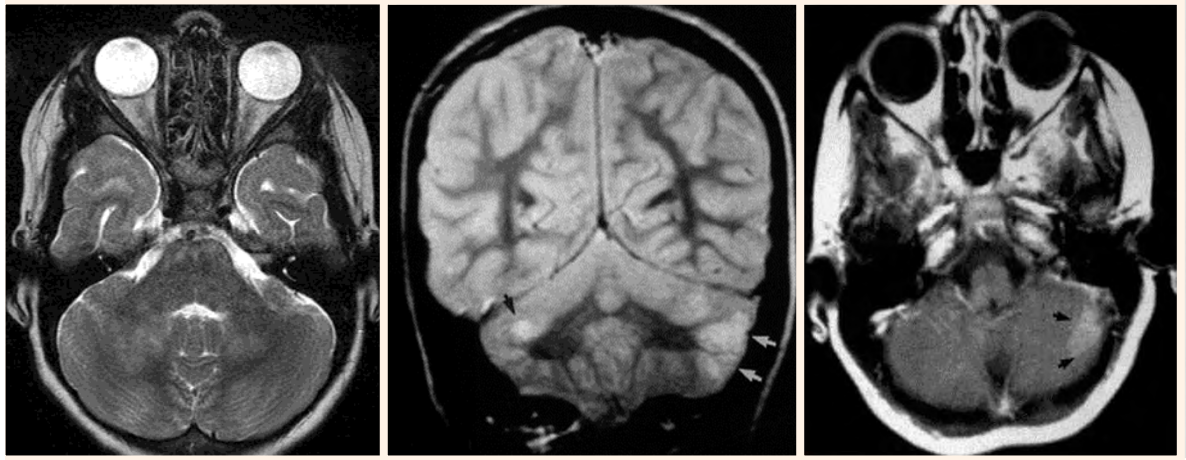
Acute cerebellitis

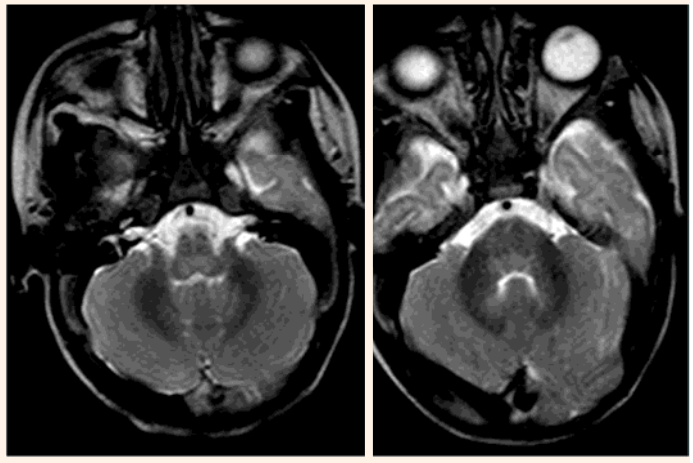
- Common cause: varicella
- Followed by: mumps, *mycoplasma*, EBV, influenza, rotavirus, enterovirus 71
- **Imaging not necessary**
  - Cerebellar swelling, Dentate nuclei, Hydrocephalus
  - Possible enhancement

Rhombencephalitis

- brainstem inflammatory disease that follows a viral illness
- Typical location: brainstem, with extension to thalamus & cerebellum in some cases
- Contrast enhancement is variable

enterovirus

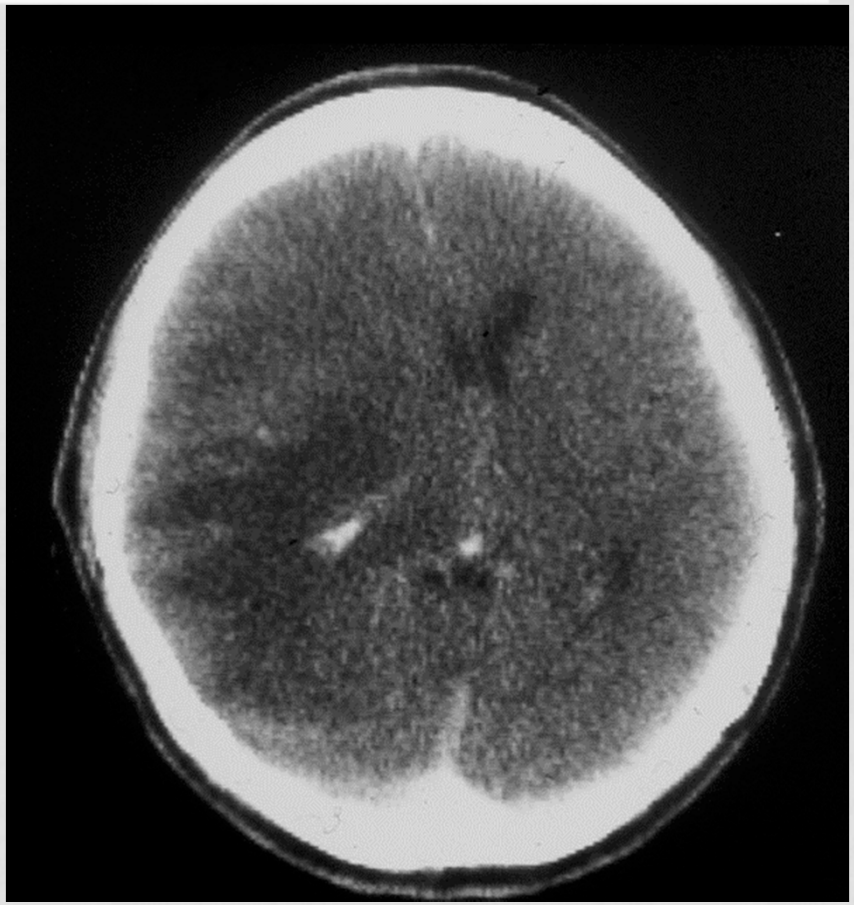




CASE 8

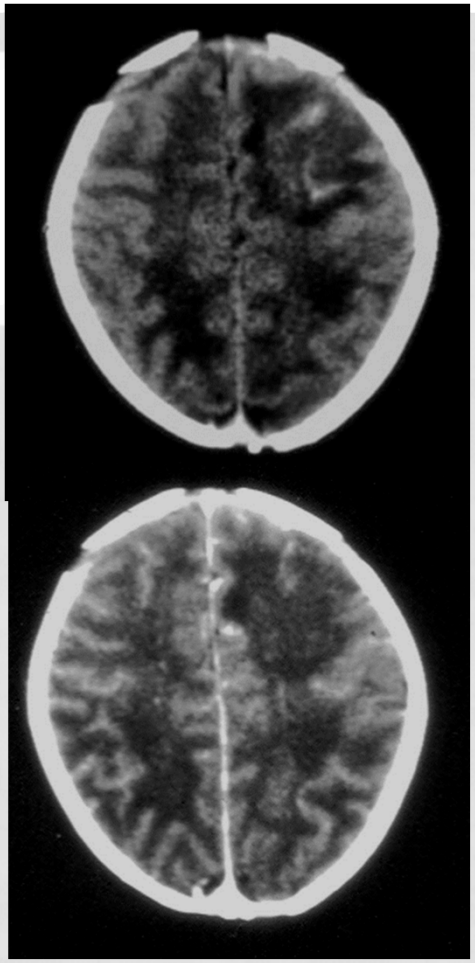
- Questions?

- 1. low grade glioma
- 2. cerebritis
- 3. venous thrombosis
- 4. stroke
- 5. None of them

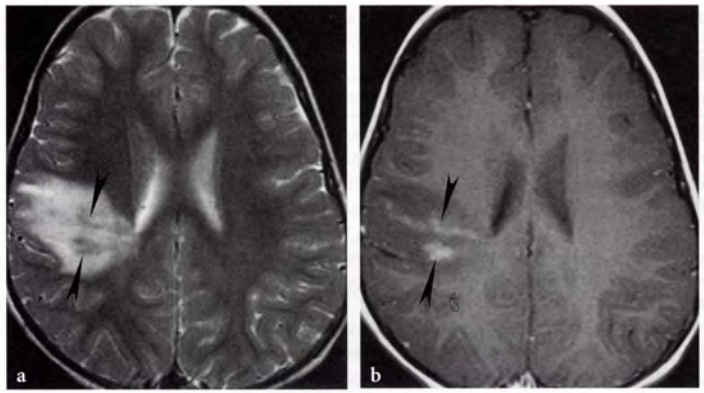


CEREBRITIS

- May be difficult to identify
- CT
  - Ill defined hypodense subcortical lesion
  - Mass effect
  - Foci of hemorrhage may be seen
- MR
  - Hyperintensity T2
  - Diffusion: restriction of diffusion
  - No enhancement
  - Patchy enhancement may be seen
  - Sulci & GM

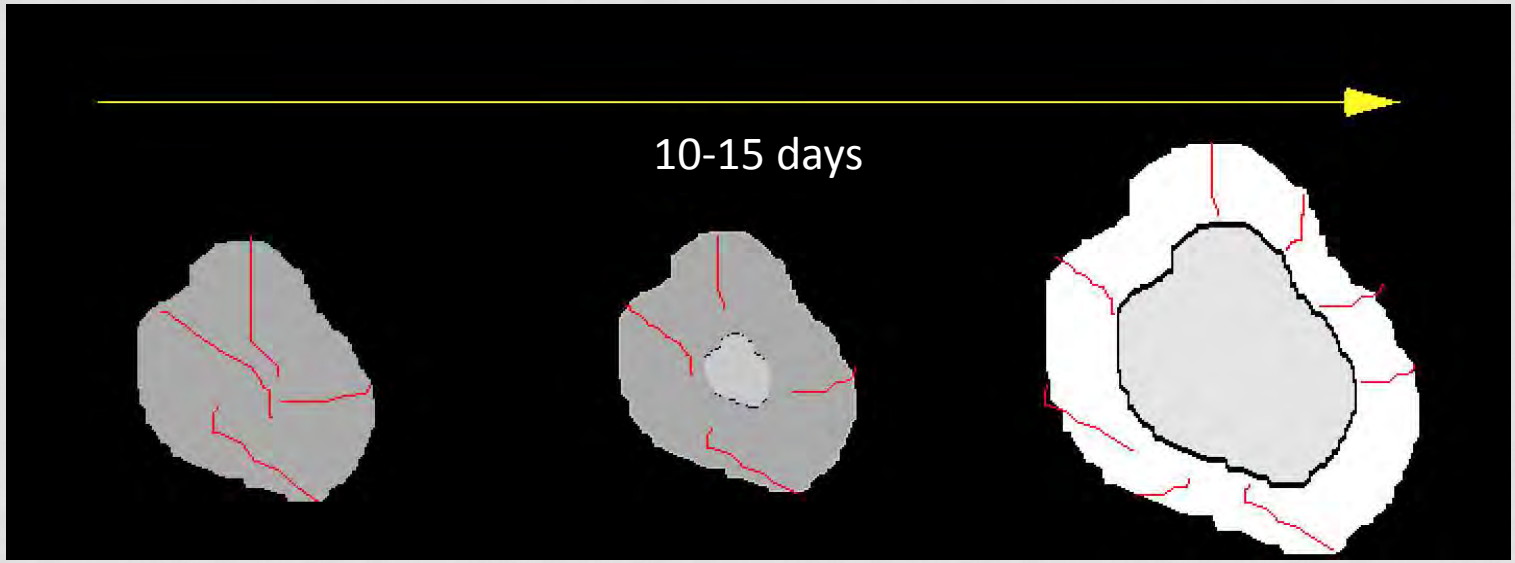


From P Tortori Donati



- Early stage of purulent infection

CEREBRITIS BEFORE 10-15 DAYS



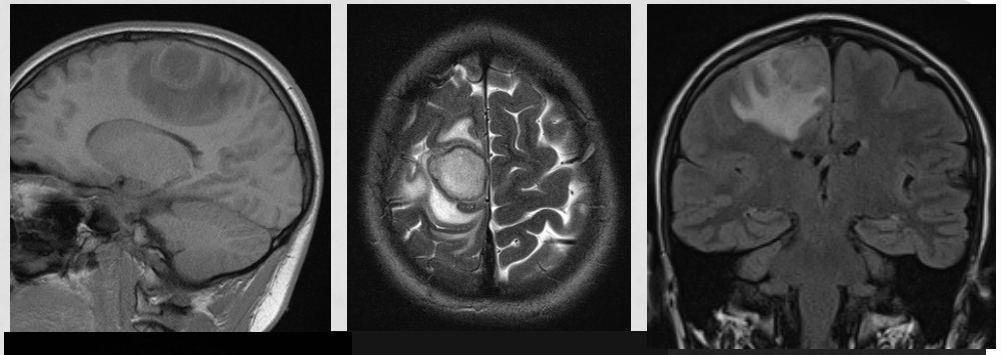
Early Cerebritis
3-5 days
Large edema

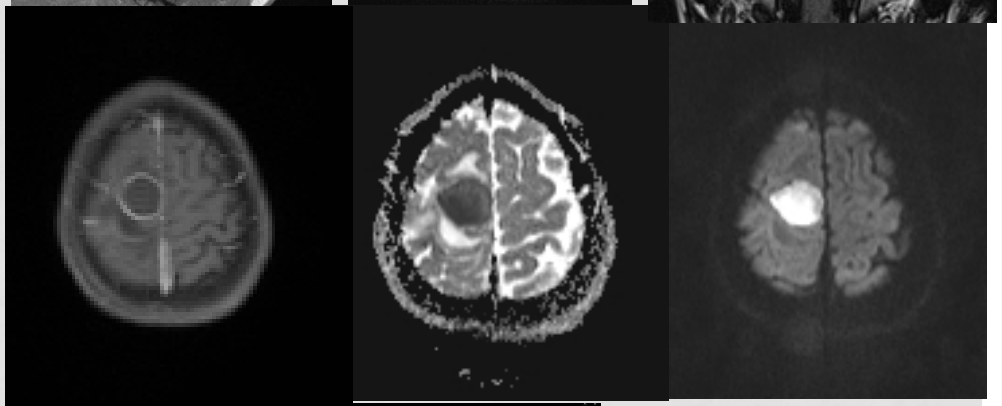
Late cerebritis-capsule formation
4-5 days to 2 wks
Progressive necrosis
Rim of inflammatory cells, macrophages,
granulation tissue, fibroblasts
Vascular proliferation

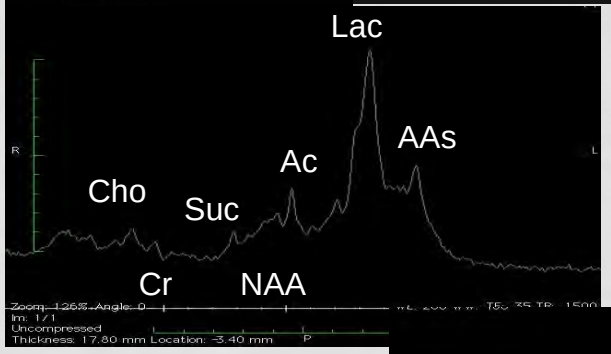
Abscess with capsule
Early capsule (begins 2 wks)
Collagenous capsule

Late capsule (wks to months)
Central cavity shrinks
Thick wall

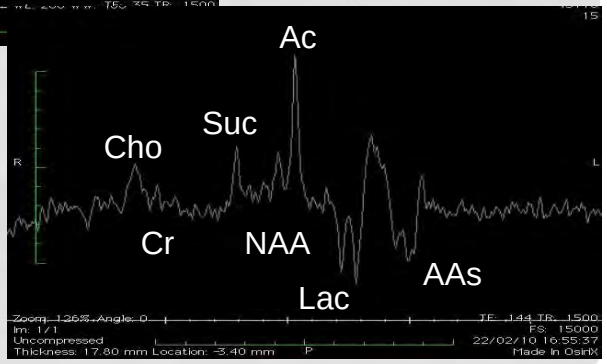
- Abscess
  - Central cavity
    - DWI: restriction of diffusion
  - MRS: lactate, lipids, amino acids (succinate and others)
  - PWI: low rCBV of capsule, increased permeability

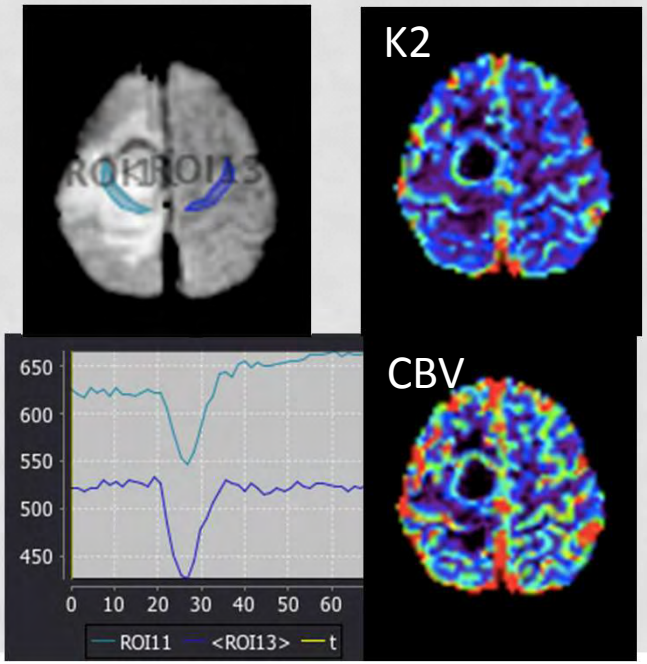




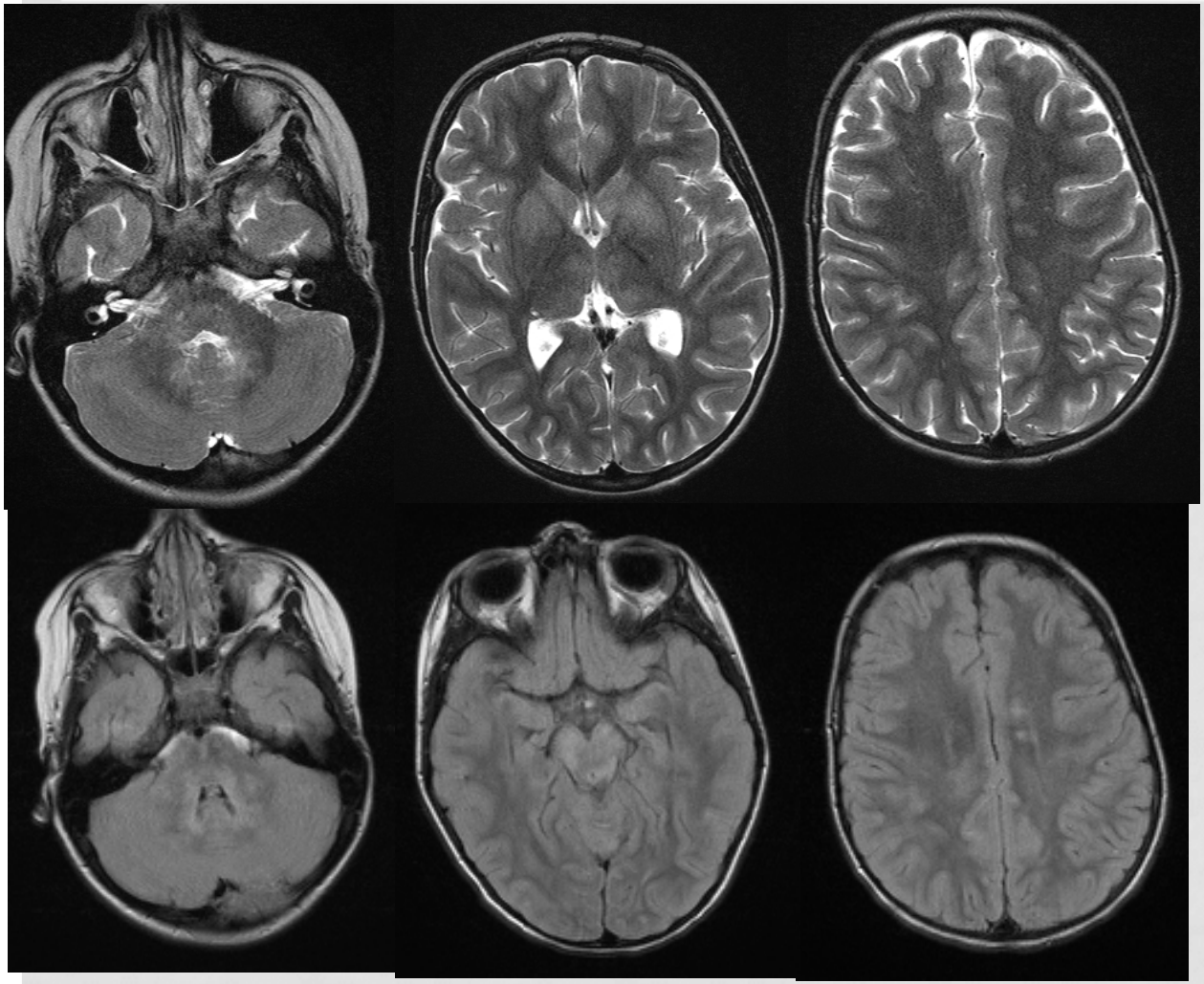


Pr JM Constans





CASE 9

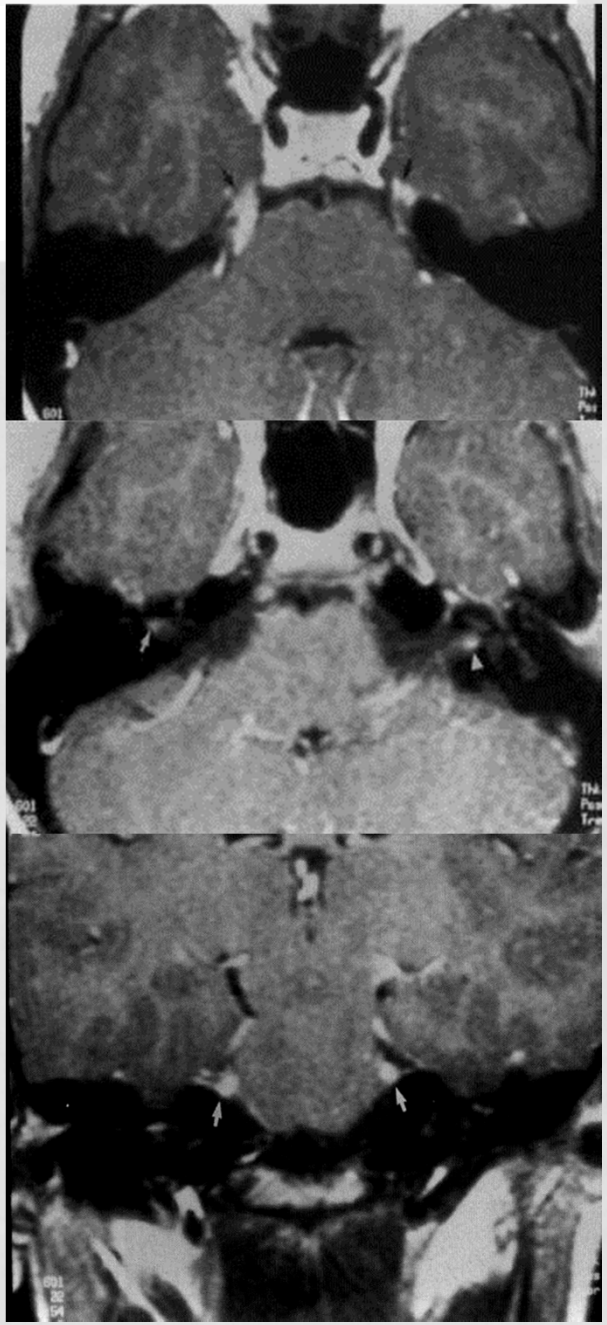


- Questions?

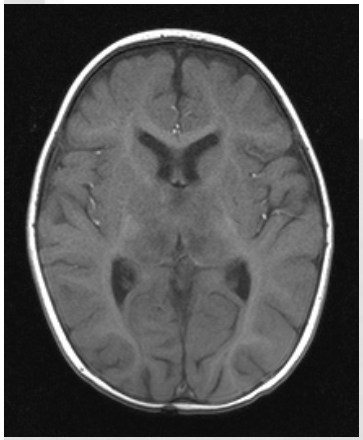
- 1. MS
- 2. ADEM
- 3. Lyme
- 4. vasculitis
- 5. toxic-metabolic

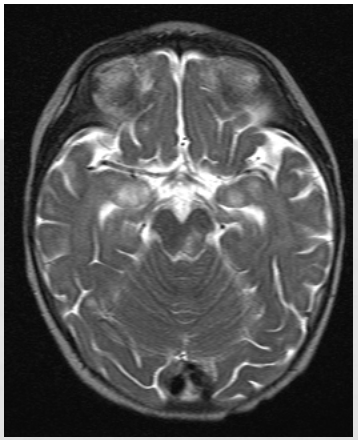
LYME (NEUROBORRELIOSIS)

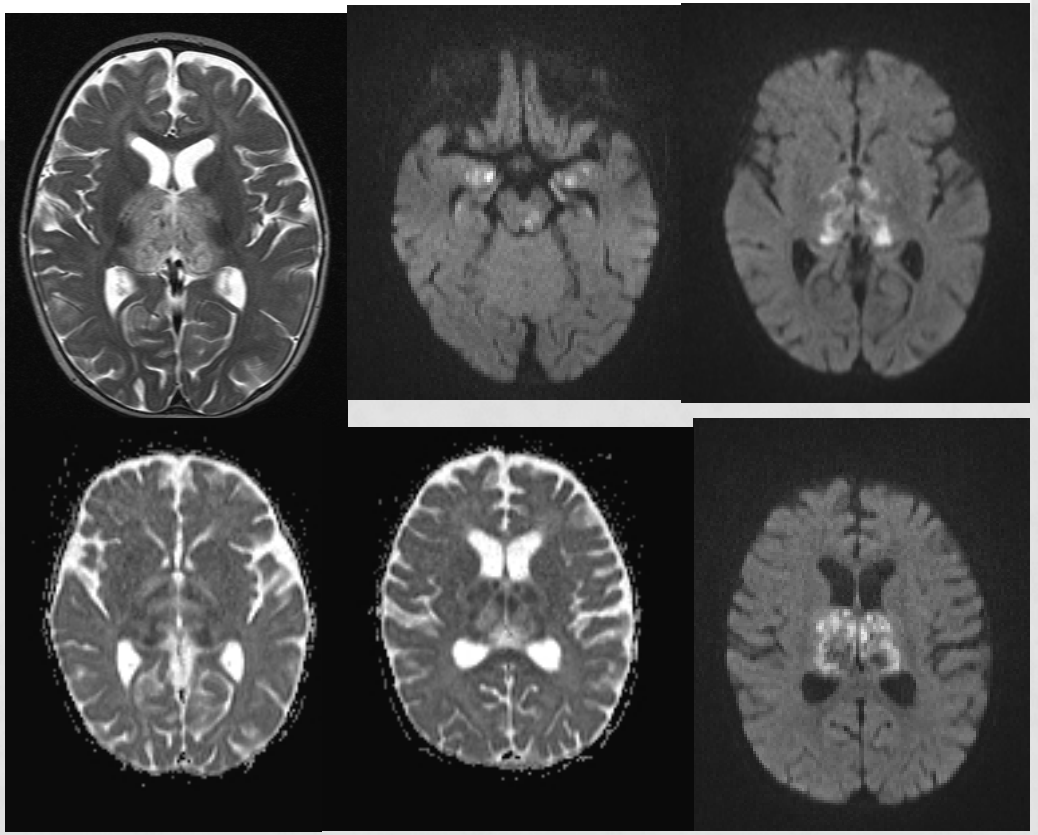
- Tick transmitted spirochetal disorder: Borrelia burgdorferi
- Neurological manifestations
  - meningitis, encephalitis, cranial neuritis, radiculoneuritis, myelitis
- Imaging
  - **May mimic MS, ADEM : WM**
  - Thalamus, basal ganglia
  - brainstem
  - Cranial neuropathy
    - thickened nerve and marked enhancement



CASE 10







- Questions?

1.Mitochondriopathy

2.Viral encephalitis

3.ANE (acute necrotizing encephalopathy)

4.Gayet Wernicke

5.None of them

8 months
High fever followed by coma
No known familial history
History of infection at 4 months of age: convulsions with fever
Lumbar puncture: non specific
No causative agent found

ACUTE NECROTIZING ENCEPHALOPATHY (ANE)

- Rapidly progressing encephalopathy
  - triggered by acute febrile diseases,
  - mostly viral infection,
  - influenza is the most common prodromal illness
- Poor prognosis with elevated mortality
  - but may regress completely with prompt steroid treatment
- Intracranial "cytokine storm"
  - causing blood-brain barrier damage: edema, petechial hemorrhages, necrosis
- No signs of direct viral invasion or parainfectious demyelination (in contrast to ADEM)
- Characteristic bilateral thalamus involvement
  - May extend to WM, brainstem
- ANE: isolated (sporadic)
- Familial, recurrent: ANE1 caused by mutation in the gene coding protein RANBP2 2q11-q13

3. SUBACUTE, PROGRESSIVE CHRONIC ENCEPHALITIS

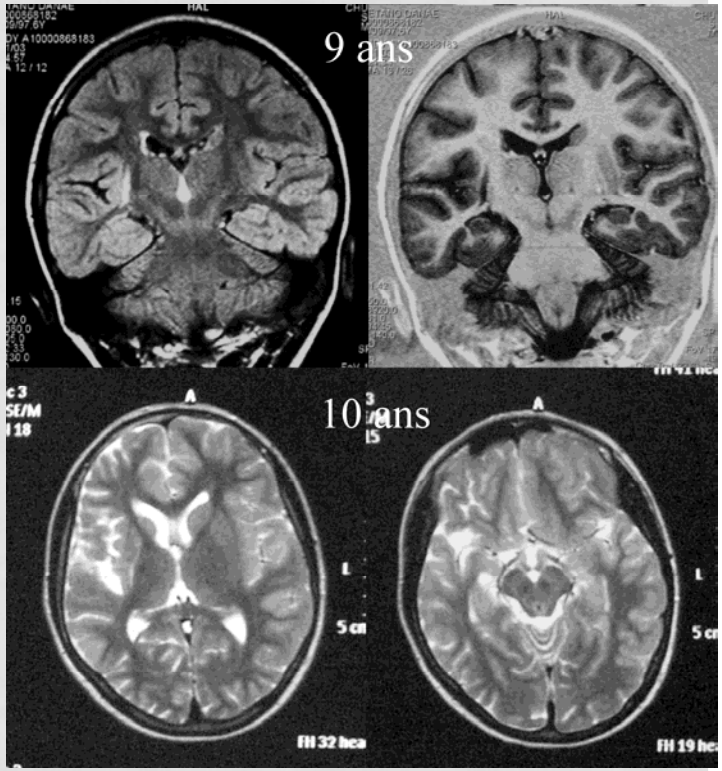
- Chronic and progressive encephalopathy of infectious origin
  - are not common in children
- They include
  - **the Rasmussen's encephalitis that is specific for the pediatric population**
  - the encephalitis/encephalopathy of acquired immunodeficiency syndrome (AIDS)
  - **the infection due to JC virus responsible for progressive multifocal leucoencephalopathy (PML)**
  - the complications of measles infections
  - and infections by prion agents: rare

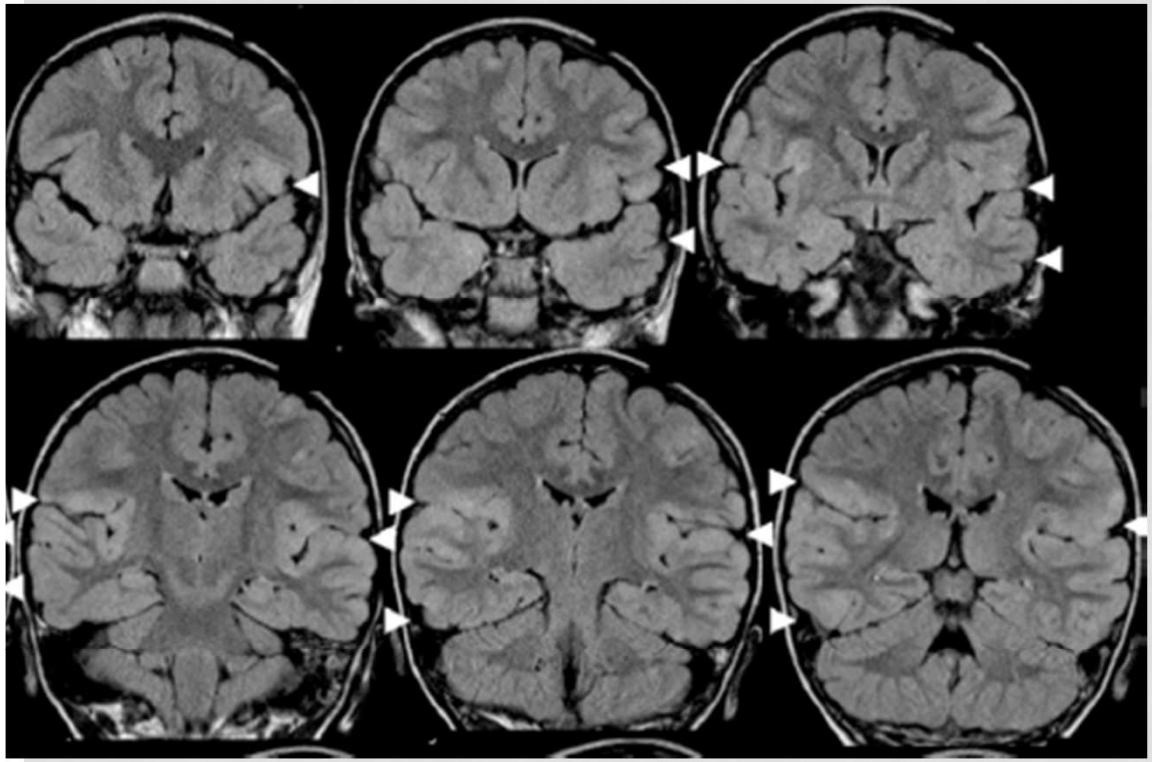
RASMUSSEN ENCEPHALITIS

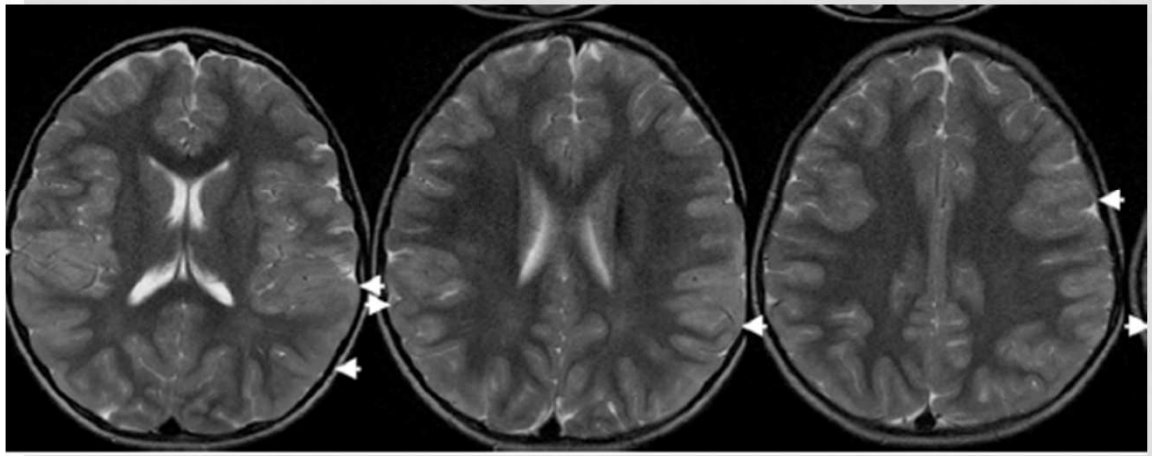
- Rare, but severe
- progressive disease of childhood or adolescence, median age of onset is 5 years
- severe epilepsy, progressive development of hemiparesis, and dementia
- Children otherwise normal
- Epilepsy
  - Most commonly partial onset,
  - involving the same side of the body
  - Epilepsia partialis continua is frequent (56 %)

- The etiology remains unknown: suggested mechanisms
  - chronic viral infection
  - Viral agents supposedly involved:
    - CMV, herpes simplex virus, Epstein-Barr virus, and slow virus
  - autoimmune theory postulates
    - the breach in the blood-brain barrier (caused by infection or another mechanism) allows the entry of autoantibodies to GluR3 into the brain, which causes activation of the glutamate receptors and subsequent seizures, and produces a vicious circle in which further breaches of the blood-brain barrier could be caused by the seizures

- Imaging: Normal at the onset
- Brain swelling : when seizures are frequent
- Unilateral enlargement of CSF compartments in the insular and peri-insular regions is seen after disease onset
- Atrophy, either focal or hemispheric, develops progressively
- increased signal intensity on T2 WI & FLAIR images is also seen in the affected cortex & underlying white matter: possible gliosis
- Involvement of the basal ganglia
- MRS: non specific; decreased NAA peak (related to neuronal loss); increased choline (due to increased cell membrane turnover associated with inflammation); *myo*-inositol (reflecting glial proliferation); lactate







5 years old
Recurrent focal seizures followed by status epilepticus

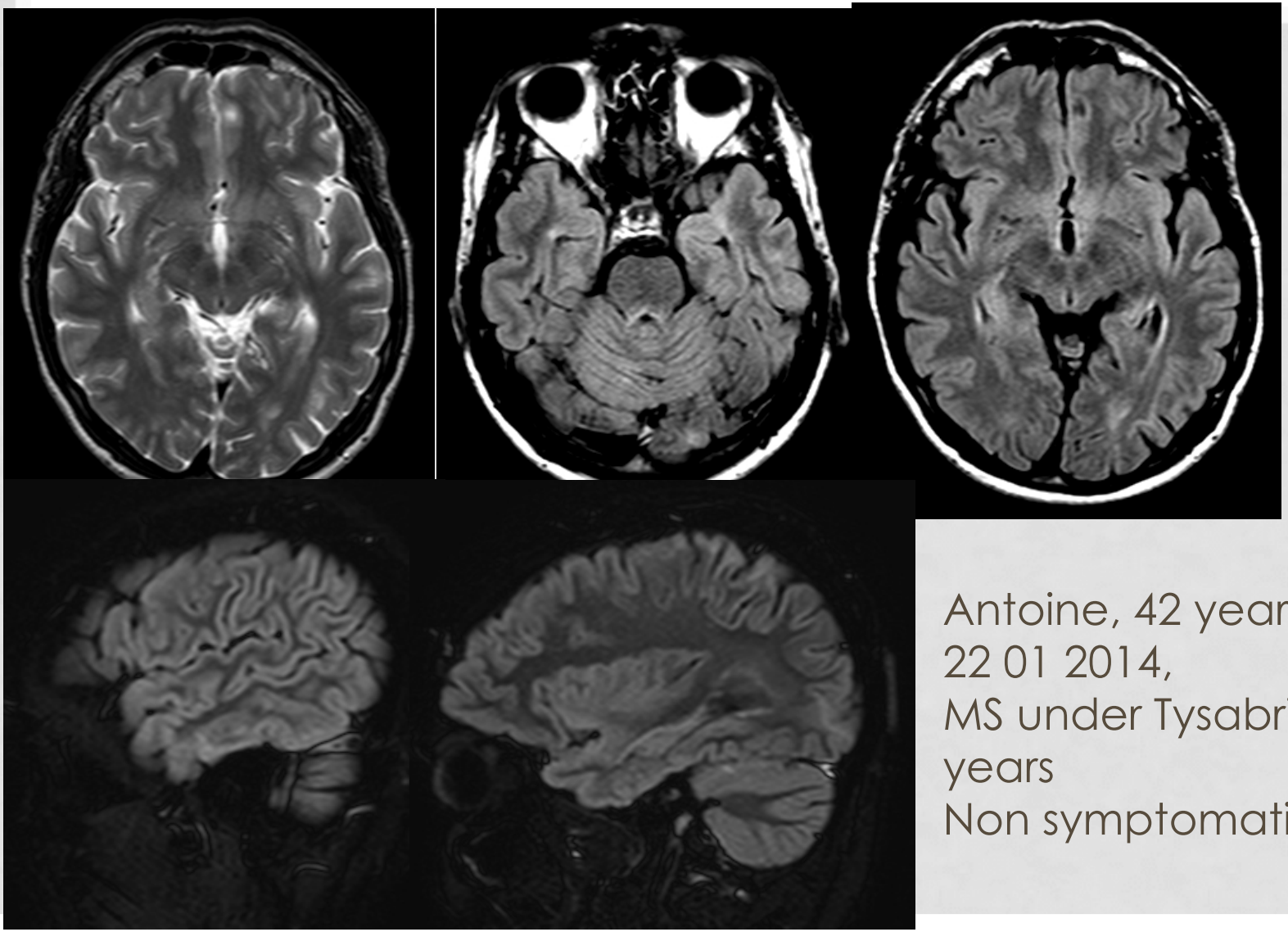
neuropil antibodies

Trt IVIg

Milh M et al, Brain Dev 2010

- FIRES: febrile infection-related epilepsy syndrome
- First epileptic seizure follows upper respiratory tract infection with fever
- Followed by recurrent partial or generalized seizures evolving to refractory status epilepticus lasting 1 to several weeks in a previously normal child

CASE 11



Antoine, 42 years
22 01 2014,
MS under Tysabri for 5 years
Non symptomatic

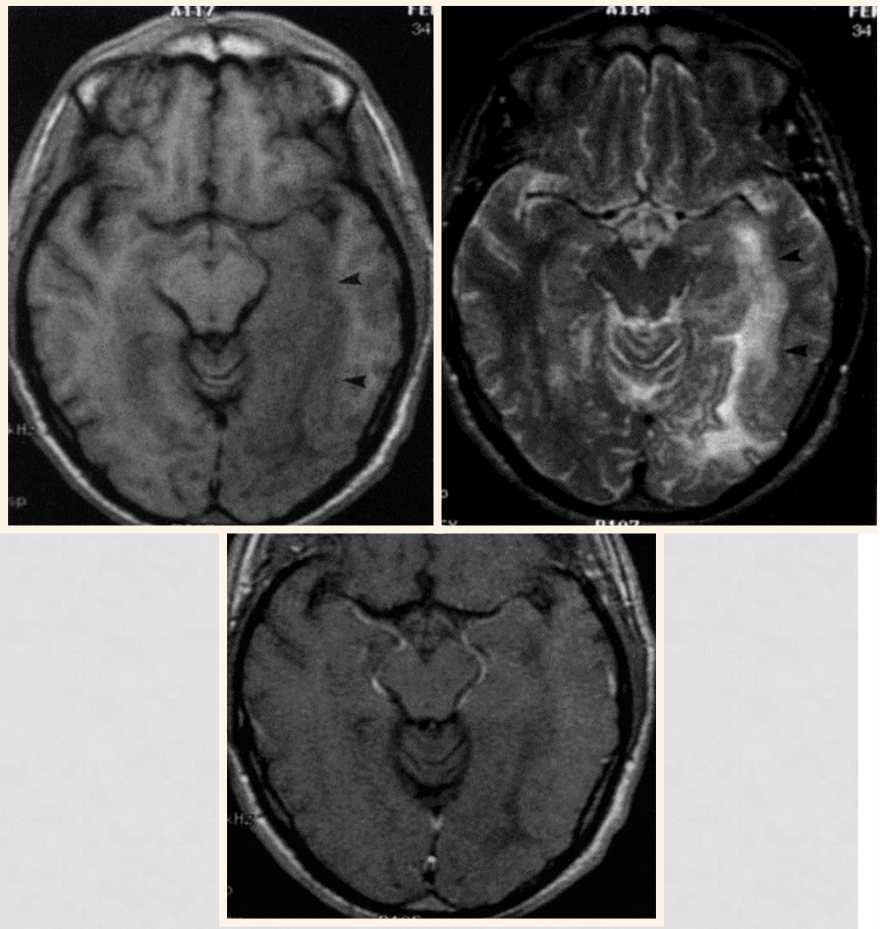
JC VIRUS : PROGRESSIVE MULTIFOCAL LEUCOENCEPHALOPATHY (PML)

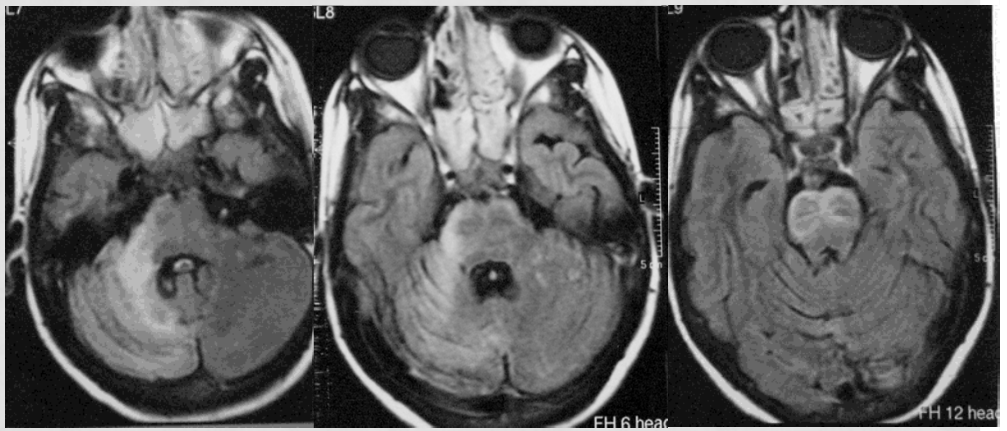
- Human polyomavirus
- Infects tonsils or GI-tract
- Virus remains latent in tonsils, GI-tract or tubular cells of the kidney

- Reactivation in immunodeficient patients (transplant, AIDS, MS..) because of immunosupressive medications
- Also seen in patients under natalizumab (Tysabri)
- Extremely rare in children

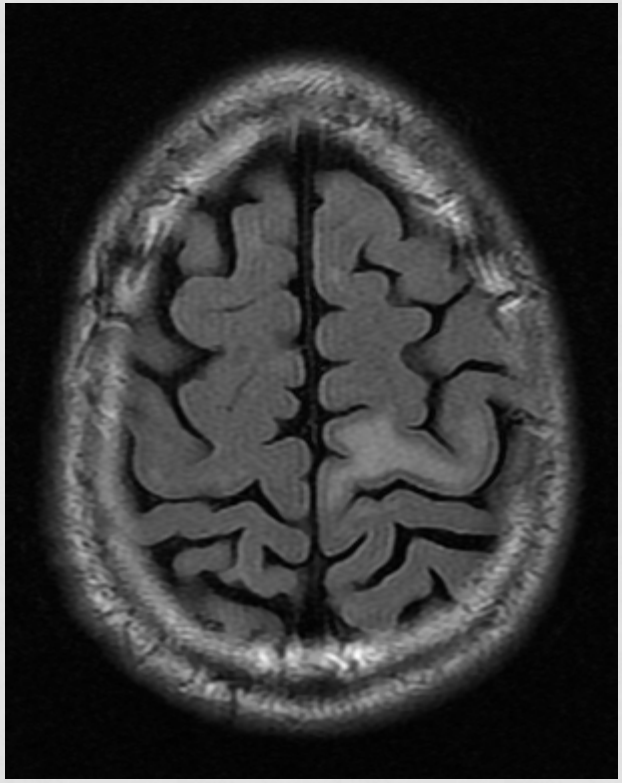
PML: IMAGING

- „*Scalloped*“ WM lesions: U fibers involved
- Multifocal, asymmetric
- Parieto-occipital, Basal Ganglia, PF
  - Cerebellar syndrome and multiple cranial nerve palsies related to involvement of the brainstem are most likely encountered in PML associated to AIDS
- Hypointense T1-WI/hyperintense T2-WI / FLAIR
- No enhancement or marginal contrast enhancement
- No/min mass effect





- Frontal predominance in MS patients treated by Tysabri
- Linear contrast enhancement is seen in up to 40% of cases

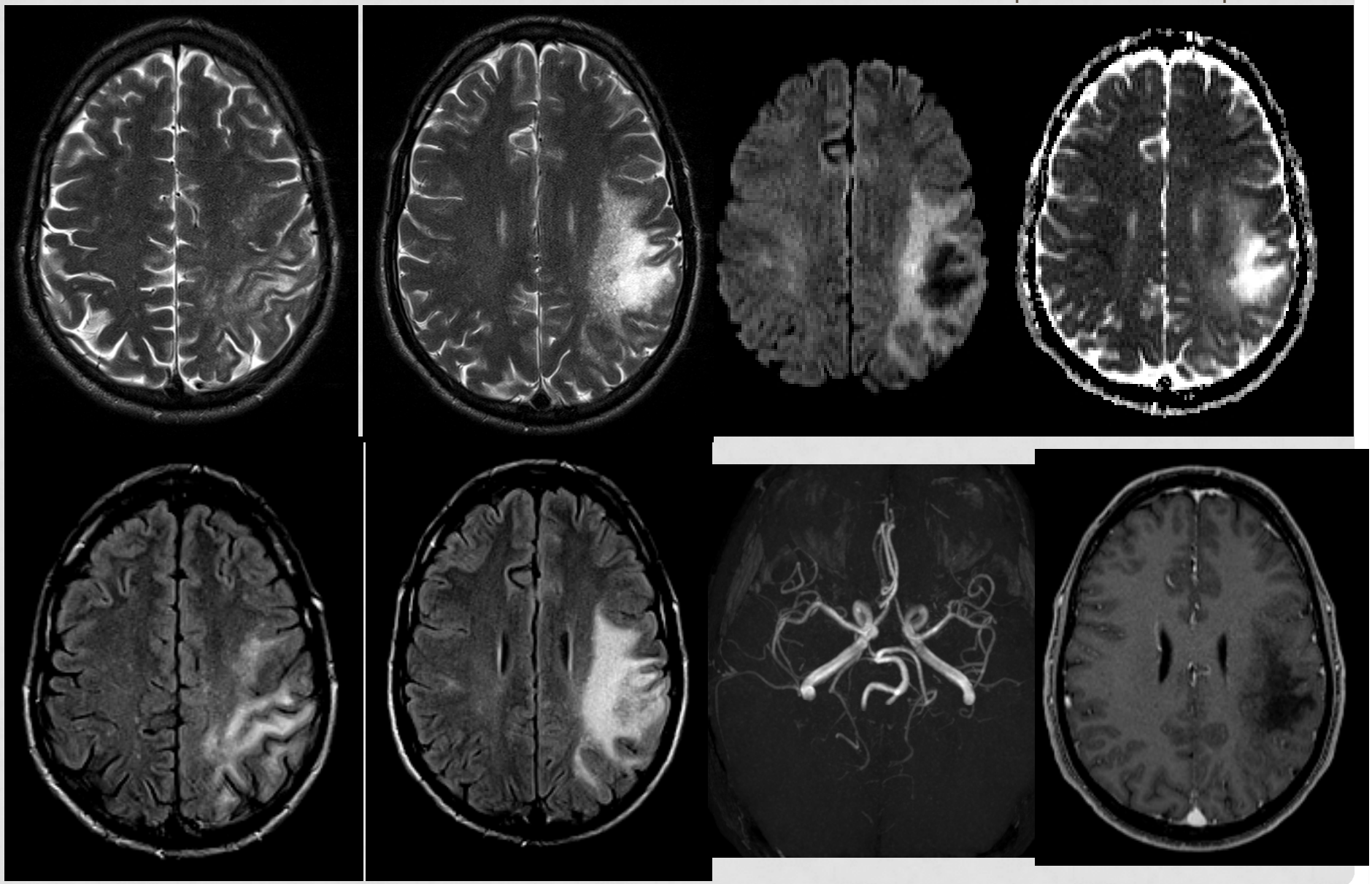


Pr JM Constans, Amiens

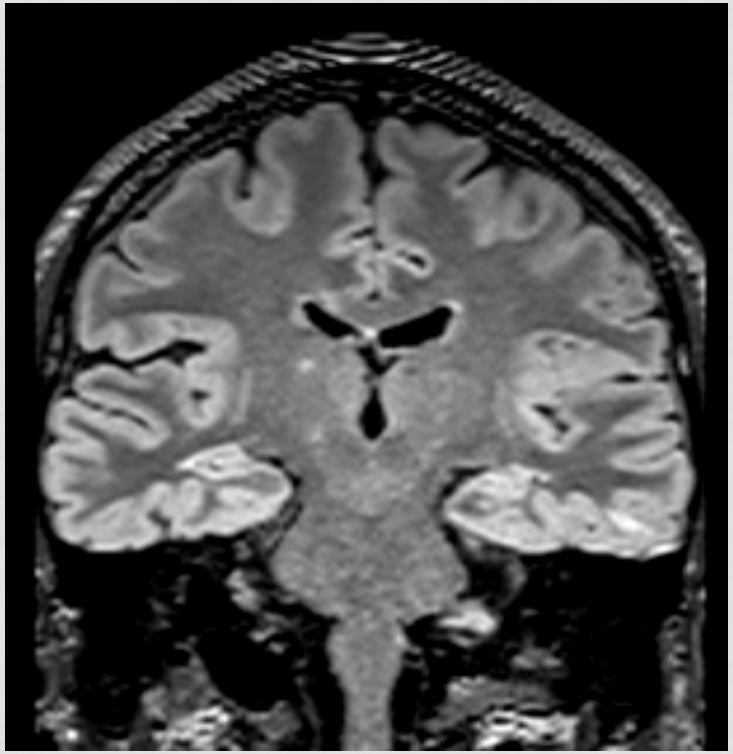
Flair NF et al Neurology 2012

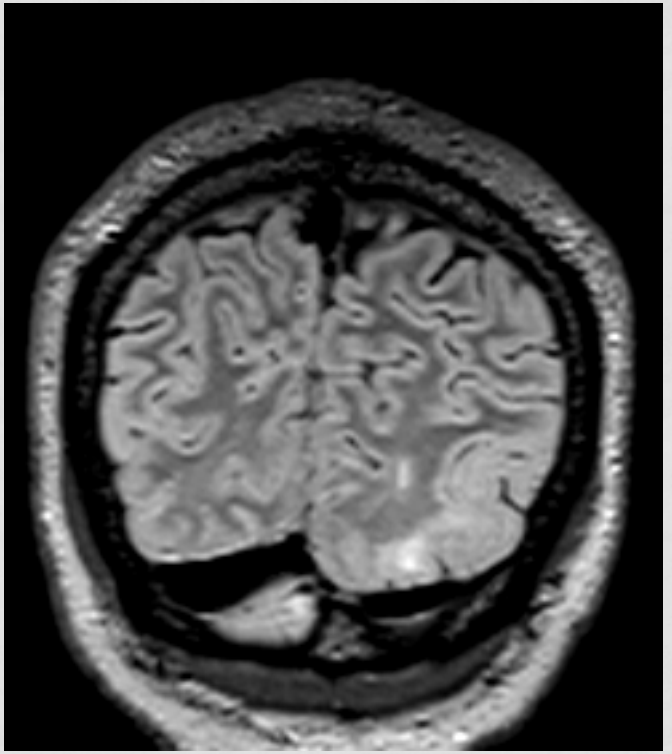
- May manifest as a stroke like episode

30 y
HIV & PML
Aphasia hemiparesis



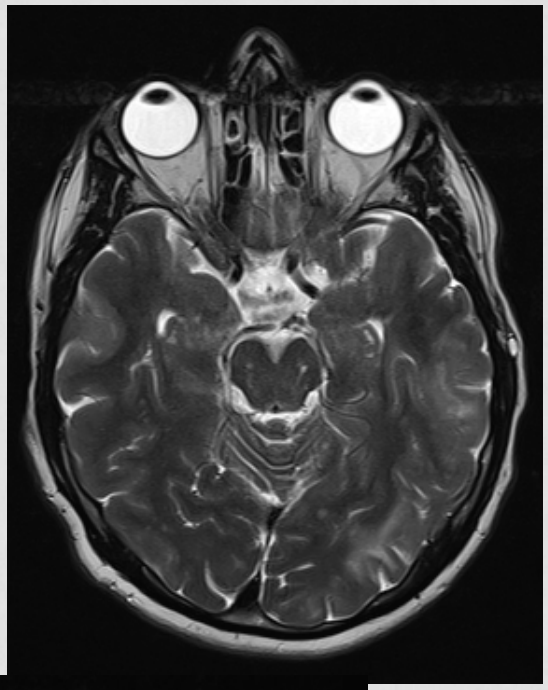
Case 11 2 months later 20 03 2014, ART
JC virus positive, symptomatic
Trt plasmatic exchange

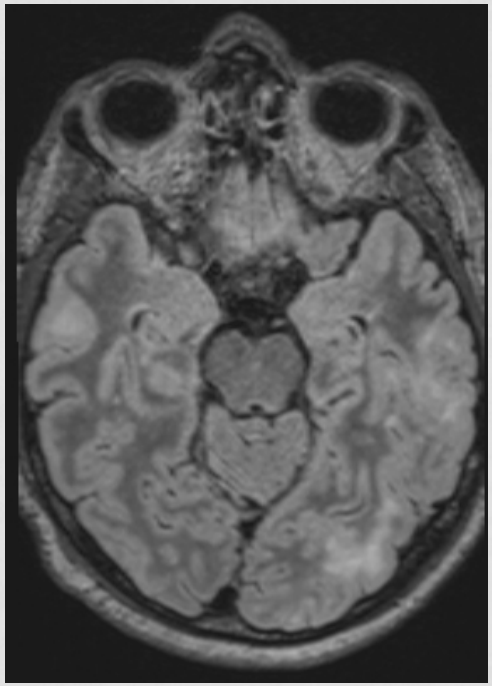


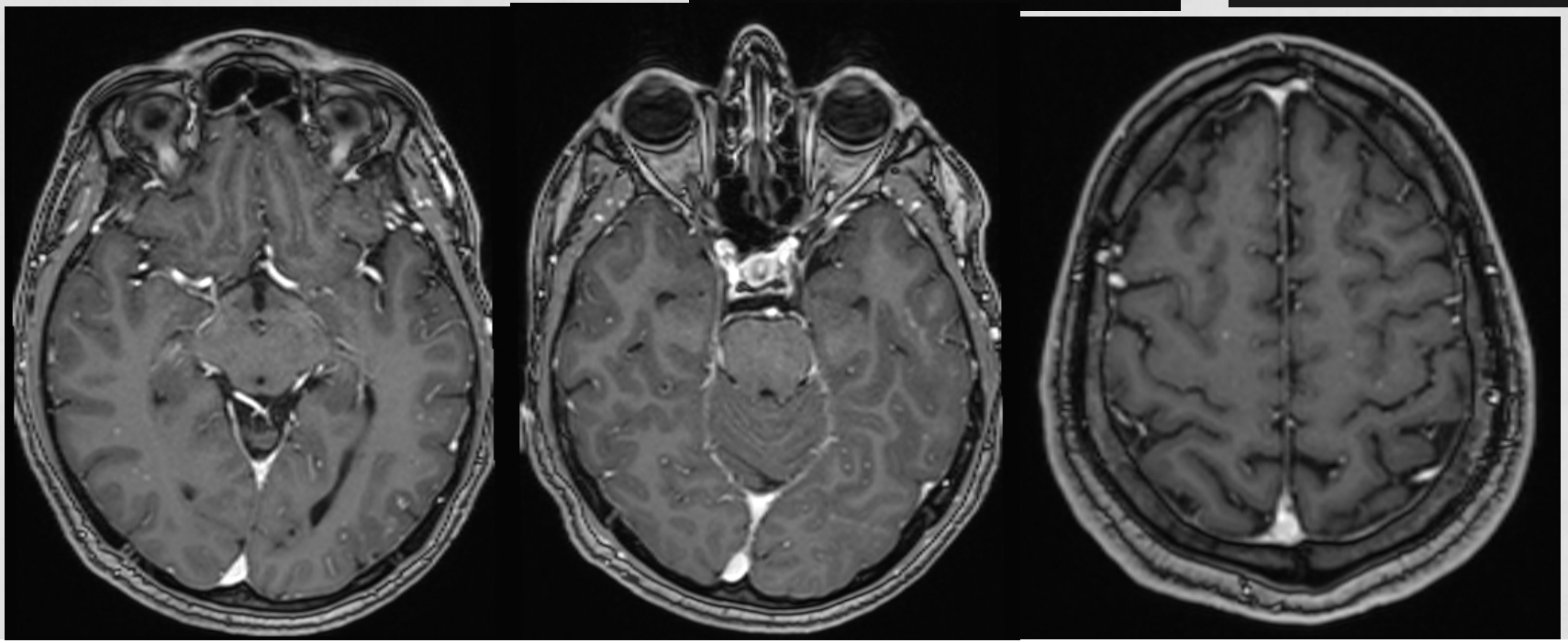


07 05 2014

Worsening of visual symptoms





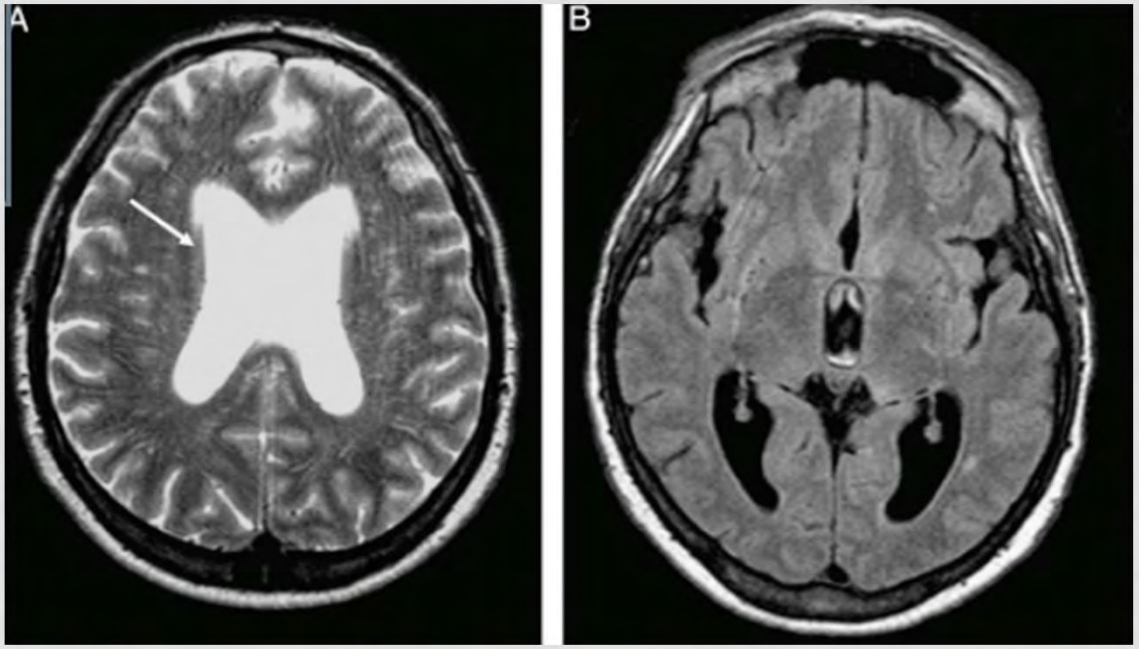


- Question?

- 1. Tumoral pathology (lymphoma)
- 2. Viral infectious pathology (HSV, CMV…)
- 3. Inflammatory pathology (IRIS)
- 4. Other infectious pathology (Toxoplasmosis, cryptococcosis, aspergillosis,..)

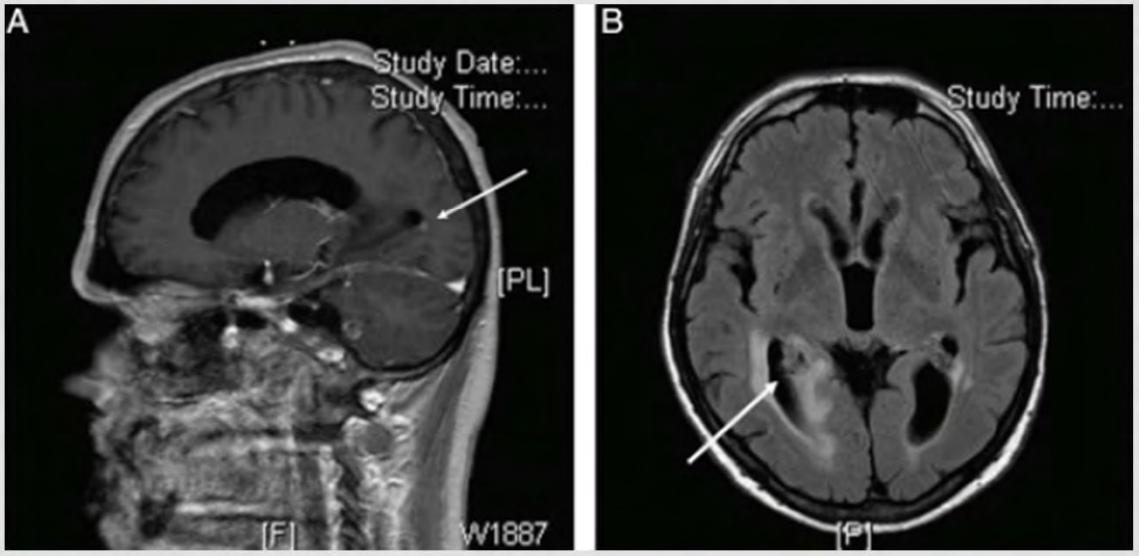
IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME

- Paradoxical clinical deterioration in HIV-patients under HAART (Highly Active Anti Retroviral Therapy)
- Exuberant granulomatous inflammation
- Patchy or punctiform enhancement
- Also described in patients with MS, Crohn disease treated with natalizumab (Tysabri-R), solid organ transplant recipients with immunosuppressive medications



Cryptococcus meningitis
Kydney transplant and immunossuppression trt

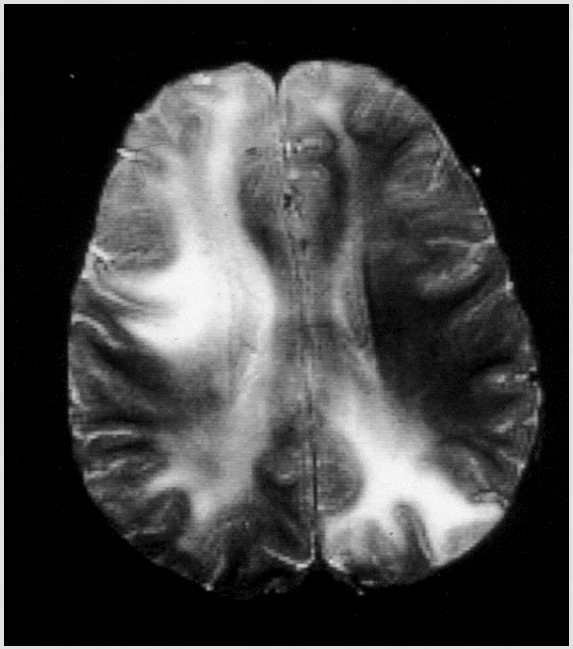
8 months later: unsteady gait, obnubilation, and anterograde amnesia

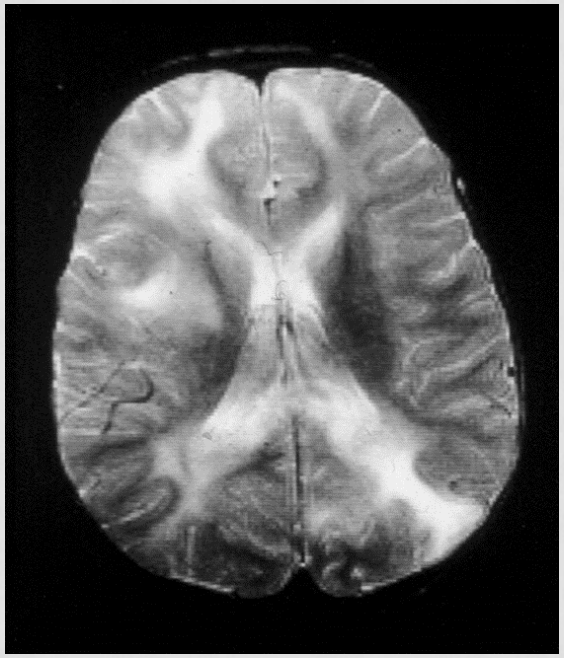


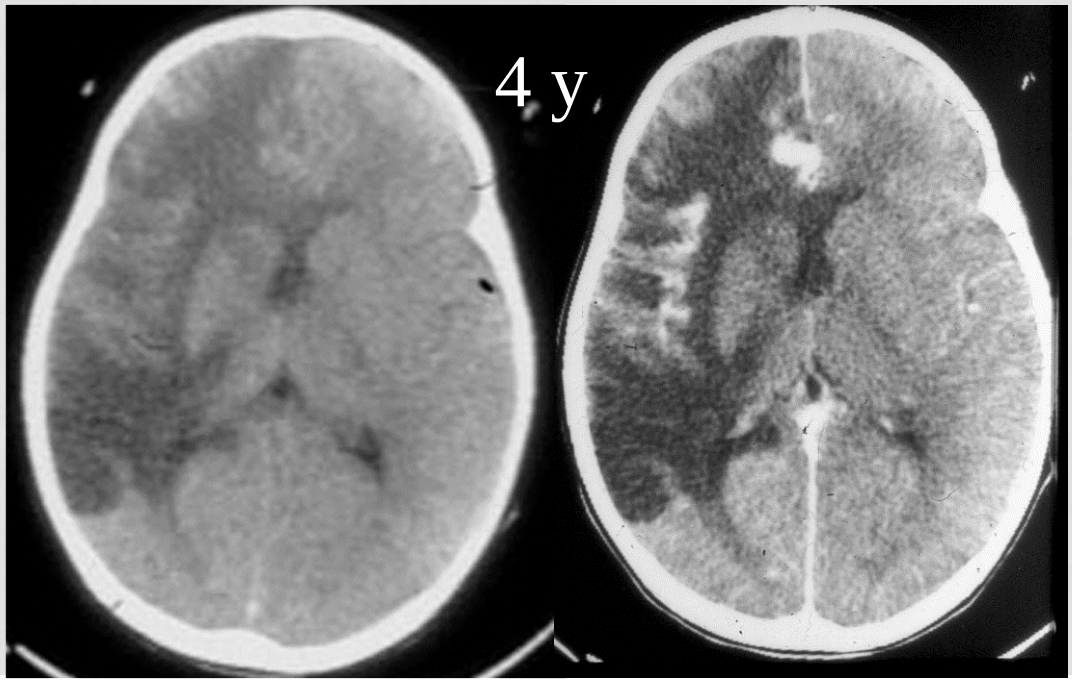
contrast enhanced ependymal nodule

MEASLES

- Causes "Subacute Sclerosing Panencephalitis" (SSPE)
- History of primary measles infection before age 2, followed by several asymptomatic years
- Also described after vaccination
- Gradual psychoneurological deterioration
- MR findings are aspecific
  - subcortical periventricular WM, bilateral and asymmetric distribution, encephalomalacia and atrophy eventually
  - Parietal temporal lobes
  - Mass effect enhancement may be seen
  - Basal ganglia, thalami, cerebellum, CC and spinal cord: less frequent
  - Brainstem: rare






4 y

CONCLUSIONS

- Neuroimaging has a central role in the early diagnosis of encephalitis
- Increased human susceptibility to infection (HIV, chemotherapy/BMT/steroid treatments, genetics)
- **The neuroimaging appearance of encephalitis and related disorders is non specific**
- and requires close clinical correlations
- and follow-up in order to establish a diagnosis