Seizure Protocol MRI
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American Epilepsy Society | Annual Meeting
<table>
<thead>
<tr>
<th>Name of Commercial Interest</th>
<th>Type of Financial Relationship</th>
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<tbody>
<tr>
<td>Sleep Medicine</td>
<td>Consultant</td>
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<tr>
<td>Best Doctors</td>
<td>Consultant</td>
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American Epilepsy Society  | 2013 Annual Meeting
Learning Objective

Understand the importance of having the appropriate seizure protocol MRI to enhance clinical diagnosis and management of seizures/epilepsy (because not all MRIs are created equal)
Understand the MRI Test

- Sensitivity/specificity depends on the use
- MRI data are gathered over time (~1 bit q5-10usec)
- Pts tolerate only certain amount time in scanner
- Time is costly so shorter sequences are more economical and more feasible
- MRI is a diagnostic tool that varies in quality, technique, and the interpretation
- MRI is normal in many seizure cases
- And, even if abnormal, the finding(s) may be unrelated to the cause of the seizure
Ideal or optimal MRI

- 3D acquisition of data to allow reformatting
- Thin slices with no gaps or skip
- High signal to noise ratio (SNR)
- High spatial resolution
- Good tissue contrast
- No artifacts
- Short duration
Different types of MRI for different seizure presentations

1. Acute seizure or probable seizure
2. Epilepsy in an adult
3. Epilepsy in a child/early onset

**Having a carefully defined MRI protocol substantially increases the sensitivity of finding a lesion**

1. Acute seizure or probable seizure
   Tumor, hemorrhage, stroke, infection, trauma
MRI features and Brain Tumors

- Location: Parenchymal vs Pial vs Dural
- Multiple vs. single
- Enhancing vs Non-enhancing
- Shape and Pattern of enhancement & edema
- Hypocellular vs Hypercellular
- High vs. Low Blood Volume
- Intra-tumoral calcium or hemorrhage
52 yo F w/ lung lesion and 3 GTCs found to have metastatic melanoma
64 yo M presenting with sz and progressive left arm weakness: GBM
21 yo M w/ hydrocephalous and daily seizures: callosal lipoma

SAG T1

T2 FLAIR
# MRI and Hemorrhage

<table>
<thead>
<tr>
<th>Phase</th>
<th>Time</th>
<th>Blood products</th>
<th>T1WI</th>
<th>T2WI</th>
<th>SWI/GRE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute</td>
<td>&lt;24 hrs</td>
<td>Oxyhemoglobin, Intracellular</td>
<td>Iso-</td>
<td>Hyper</td>
<td>Iso</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>1-3d</td>
<td>Deoxyhemoglobin, Intracellular</td>
<td>Hypo</td>
<td>Hypo</td>
<td>Hypo</td>
</tr>
<tr>
<td>Early Subacute</td>
<td>&gt; 3d</td>
<td>Methemoglobin, Intracellular</td>
<td>Hyper</td>
<td>Hypo</td>
<td>Iso</td>
</tr>
<tr>
<td>Late Subacute</td>
<td>&gt;7d</td>
<td>Methemoglobin, Extracellular</td>
<td>Hyper</td>
<td>Hyper</td>
<td>Iso</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt;14d</td>
<td>Ferritin/hemosiderin Extracellular</td>
<td>Hypo</td>
<td>Hypo</td>
<td>Hypo</td>
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</table>

Adapted from Atlas Sw, Thulborn KR. Intracranial Hemorrhage, 2002.
57 yo F with sz found to have a cavernoma
75 yo M w/ HTN presenting w/ confusion
## MRI and Ischemic Stroke

<table>
<thead>
<tr>
<th>Time</th>
<th>MRI APPEARANCE</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;6 hrs</td>
<td>DWI bright, ADC dark, nl FLAIR</td>
<td>Pure cytotoxic cell swelling</td>
</tr>
<tr>
<td>6hrs- 1 week</td>
<td>DWI bright, ADC dark, FLAIR bright, gyral swelling</td>
<td>Predominantly cytotoxic edema, increasing vasogenic edema</td>
</tr>
<tr>
<td>3d – months</td>
<td>Gadolinium enhancement</td>
<td>BBB breakdown</td>
</tr>
<tr>
<td>~1-2 wks</td>
<td>DWI bright, ADC normal or bright, FLAIR bright,</td>
<td>Decreasing cytotoxic edema</td>
</tr>
<tr>
<td></td>
<td>gyral swelling, variable enhancement</td>
<td>Predominantly vasogenic edema</td>
</tr>
<tr>
<td>&gt;2 wks</td>
<td>DWI normal, ADC bright, FLAIR bright, decreased</td>
<td>Resolving vasogenic edema</td>
</tr>
<tr>
<td></td>
<td>gyral swelling, variable enhancement</td>
<td>Increasing gliosis</td>
</tr>
<tr>
<td>Month(s)</td>
<td>DWI normal, ADC bright, FLAIR bright, volume loss,</td>
<td>Gliosis and encephalomalacia</td>
</tr>
<tr>
<td></td>
<td>no enhancement</td>
<td></td>
</tr>
</tbody>
</table>
28 yo F, 30 wks pregnant, presents w/ sz, mutism and R HP
MRI and Infections

MRI findings

- T1 post gado (BBB breakdown)
- DWI (cellular swelling)
- FLAIR (edema)
- SWI (hemorrhage, calcification)
- Pattern of appearance is important

Types of Infections

- Bacterial meningitis/cerebritis/abscess
- Fungal meningitis/cerebritis/abscess
- Viral meningitis/encephalitis
- Prions
58 yo presented rigors and confusion 5d after a camping trip: CSF 330 WBCs, Viral panel: EEE
69 yo man w/ 2 mo progressive mental status changes and szs
54 yo Haitian farmer presenting with sz

T2 FLAIR

Scolex of vesicular stage of Neurocysticercosis

Granular nodular
MRI and Trauma

Often normal in setting of mild trauma, but useful for axonal or vascular injury, contusions

<table>
<thead>
<tr>
<th>Technique</th>
<th>Acute</th>
<th>Chronic</th>
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<tbody>
<tr>
<td>DWI</td>
<td>Cell Swelling</td>
<td>Cell Loss</td>
</tr>
<tr>
<td>FLAIR</td>
<td>Edema, Mass Effect</td>
<td>Gliosis</td>
</tr>
<tr>
<td>GRE</td>
<td>Deoxyhemoglobin</td>
<td>Hemosiderin</td>
</tr>
<tr>
<td>T1</td>
<td>Mass effect</td>
<td>(Focal) Atrophy, Encephalomalacia</td>
</tr>
</tbody>
</table>
55F w/ sz and history of TBI 2 years ago

Right frontal encephalomalacia with tiny susceptibility artifact on GRE, focal atrophy on T1, representing prior contusion
MRI Protocol: Acute Seizure

• Sagittal T1: this study acts as a survey to look at entire brain (mass effect, herniation)
• AX T1: pre-contrast
• Axial T2 FLAIR: sensitive to brain edema, hemorrhage, or abnormal protein concentration
• DWI: infection, stroke
• Gradient echo (GRE) OR susceptibility weighted image (SWI): hemorrhage, brain mineralization
• T1 post-gadolinium (tumor, infection, altered BBB)
TIME= (40 min)
Normal MRI: Now what?

• Maybe it wasn’t a seizure
• Maybe it is non-lesional epilepsy
• Maybe the brain isn’t normal even though you have a “negative test” (false negative)
• Maybe the scan isn’t really normal
You have 5 seconds to find Waldo...
“Now what” algorithm

• Confirm whether images were optimal
• If not optimal, have an additional more optimal MRI protocol performed
• If you have optimal 1.5T imaging and still no dx, then consider a 3T MRI (discuss protocol directly with the subspecialist who will interpret it)
High Resolution MRI

3T identified new Lesions in 5% of cases, and changed Diagnosis in 26%.

Winston et al, Epil Research, 2013
2. Epilepsy, adult onset
Mesial Temporal Sclerosis (MTS)

- Accurate diagnosis critical as surgery is curative in 70-90%
- Atrophy of the hippocampus (T1)
- Loss of internal structure (T1 or T2)
- Increased signal intensity (T2 or FLAIR)
- Excellent pathological correlation
Coronal oblique

Sylvian fissure

Semin Ultrasound CT MRI 2008;29:2-14
24 yo w/ complex partial szs/weekly
Chronic Epilepsy Protocol: Adult onset

• Retrieve old scan for comparison (especially if it was normal)
• Thin slices, no gaps, COR oblique perpendicular to axis of hippocampi
  – T2 FLAIR med temp lobes
  – T1 3D SPGR med temp lobes or COR inversion recovery
• Selected whole brain sequences, if needed
• (40 min)
3. Epilepsy, early onset
Suspect Malformation of Cortical Development (MCD)

- History of early onset or childhood epilepsy, or in retrospect, spells that were likely to be seizures
- Seizures refractory to polytherapy
- “normal MRI”
Heterotopia

Schizencephaly

Polymicrogyria

PVNH

Double cortex: band

Lissencephaly

MCD
Developmental/Glioneuronal Tumors

Ganglioglioma  DNET (COR T1)  Hypothalamic Hamartoma
Chronic Epilepsy Protocol: Early onset

• Retrieve old scan, even if normal
• 3D Whole Brain high spatial resolution imaging of the gray white junction
  – Spoiled gradient recalled echo (SPGR) volumetric dataset, thin slices (≤1.2mm), no gaps
  – Or inversion prepared 3D T2
• AX 2D FLAIR T2 whole brain
• AX 2D proton density
• Selected whole brain sequences, if needed
• (40-60 min)
The Role of Repeating the MRI
Ticks and Fleas

2D T2 COR FLAIR
SEIZURE/EPILEPSY PROTOCOL

• AX T1 pre and post gado
• AX FLAIR
• AX DWI/ADC
• AX GRE or SWI
• COR oblique T2 FLAIR (thin cuts)
• COR T1 thin cuts (SPGR)

(40-60 minutes)
Advanced MRI: MR Spectroscopy (MRS)

- Noninvasive technique to measure metabolic act.
- No further hardware needed
- Focus on only one area in question
- Used for distinguishing tumors from radiation necrosis
- Most common use in epilepsy is to determine lateralization of MTS
Magnetic Resonance Spectroscopy

• N-acetyl-aspartate (NAA) is a neuronal marker
• NAA/Creatine ratio is an index of neuronal loss
• Reduced NAA/Cr occurs in the region of the epileptogenic zone
• Choline peak is increased where membrane turnover is found
MRS Confirms the Dysplastic Lesion

Lesion

Normal

Chol
Cr
NAA
Sz Protocol MRI: Key Lessons

- Imaging and review should be hypothesis-driven.
- Modalities inform each other: iterative process.
- Not all lesions seen on MRI are epileptogenic.
- May need to repeat the MRI (not all are equal).
- Communication with the neuroradiologist is key.
- Think about which protocol you need for the patient in front of you.