Neuroimaging in Sleep and Epilepsy

KATIE CARPENTER BAILEY MD
What we are going to do:
1. Review imaging modalities
2. Review important neuroanatomical areas of the sleep system
3. Review important neuroanatomical areas in epilepsy
4. Discuss imaging findings and common pathology of brain disorders
5. Briefly describe the state of the art in sleep and epilepsy imaging
BASICS....

- X-RAYS ARE ABSORBED TO DIFFERENT DEGREES BY DIFFERENT TISSUES

- Always describe CT findings as densities - isodense/hypodense/ hyperdense.
- Higher the density = whiter is the appearance
- Lower the density = darker the appearance
- Brain is the reference density
- Anything of the density as brain = isodense
- Higher density than brain = hyperdense (skull is the best example)
- Anything darker (lower density) than brain = hypodense (CSF and air are classical examples)
CT density measurement = Hounsfield unit

<table>
<thead>
<tr>
<th>Material</th>
<th>Hounsfield Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>air</td>
<td>--- 1000</td>
</tr>
<tr>
<td>fat</td>
<td>---70</td>
</tr>
<tr>
<td><strong>Pure water</strong></td>
<td><strong>0</strong></td>
</tr>
<tr>
<td>Csf</td>
<td>+8</td>
</tr>
<tr>
<td>White matter</td>
<td>+30</td>
</tr>
<tr>
<td>Gray matter</td>
<td>+45</td>
</tr>
<tr>
<td>blood</td>
<td>+70</td>
</tr>
<tr>
<td>Bone/calcification</td>
<td>+1000</td>
</tr>
</tbody>
</table>
## CT and Ionizing Radiation

<table>
<thead>
<tr>
<th>Diagnostic Procedure</th>
<th>Typical Effective Dose (mSv)</th>
<th>Number of Chest X rays (PA film) for Equivalent Effective Dose</th>
<th>Time Period for Equivalent Effective Dose from Natural Background Radiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest x ray (PA film)</td>
<td>0.02</td>
<td>1</td>
<td>2.4 days</td>
</tr>
<tr>
<td>Skull x ray</td>
<td>0.1</td>
<td>5</td>
<td>12 days</td>
</tr>
<tr>
<td>Lumbar spine</td>
<td>1.5</td>
<td>75</td>
<td>182 days</td>
</tr>
<tr>
<td>I.V. urogram</td>
<td>3</td>
<td>150</td>
<td>1.0 year</td>
</tr>
<tr>
<td>Upper G.I. exam</td>
<td>6</td>
<td>300</td>
<td>2.0 years</td>
</tr>
<tr>
<td>Barium enema</td>
<td>8</td>
<td>400</td>
<td>2.7 years</td>
</tr>
<tr>
<td>CT head</td>
<td>2</td>
<td>100</td>
<td>243 days</td>
</tr>
<tr>
<td>CT abdomen</td>
<td>8</td>
<td>400</td>
<td>2.7 years</td>
</tr>
</tbody>
</table>

2. Based on the assumption of an average "effective dose" from chest x ray (PA film) of 0.02 mSv.
3. Based on the assumption of an average "effective dose" from natural background radiation of 3 mSv per year in the United States.
**MRI**

- **Pros**
  - No ionizing radiation
  - Better detail of soft tissue structures
  - Can see old hemorrhage
  - Can see acute strokes

- **Cons**
  - Much longer than CT
  - Many contraindications- metal, pacers, spinal cord stimulators, etc.
    - (mrisafety.com)
  - More expensive
  - Claustrophobia
  - Loud noise
MRI Sequences

- “one if by land, two if by sea”
- T1- parenchyma is bright
- T2- fluid is bright
- FLAIR- “fluid attenuation inversion recovery”- edema is bright
- Gradient echo- iron and calcium are dark
- Post-contrast- T1 with gadolinium
- DWI- diffusion
- ADC map- another form of diffusion
All patients with epilepsy should undergo an MRI, except those with very typical forms of primary generalized epilepsy (e.g., juvenile myoclonic epilepsy, childhood absence) or benign focal epilepsies of childhood with characteristic clinical and EEG features (e.g., benign epilepsy with centrotemporal spikes, early-onset childhood epilepsy with occipital spikes (Panayiotopoulos type)) and adequate response to antiepileptic drugs (AEDs) (Commission on Neuroimaging of the International League Against Epilepsy, 1997; Berg et al., 2010; Gaillard et al., 2011).

There are two basic situations in which to perform an MRI in patients with seizures. The first applies to newly diagnosed patients and those with longstanding epilepsy who have not been properly investigated. The second applies to patients with refractory seizures and therefore candidates for surgery (Berg et al., 2010).
Not all MRI abnormalities cause seizures and not all seizures originate from identified structural cerebral abnormalities (Gaillard et al., 2011).
Epilepsy MRI Imaging

- For practical purposes, focal epilepsy can be divided into mesial temporal-lobe epilepsy (MTLE) and neocortical epilepsy.

- It is recommended that the MRI epilepsy protocols include a T1-weighted volumetric acquisition (3D) with isotropic voxel size of 1 mm or 1.5 mm in order to enable the reconstruction of images in any plane.

- Studies demonstrated that more sophisticated methods of image reconstruction from 3D acquisitions allow a better evaluation of patients with discrete structural lesions, in particular focal cortical dysplasia (FCD), where the main findings are cortical thickening, abnormal gyri, and poor delineation of the transition between white and gray matter.

- MRI investigation will detect most common lesions causing neocortical epilepsy, which are: low-grade tumors, malformations of cortical development, posttraumatic and post ischemic lesions, inflammatory infectious scars, cavernous malformations, and arteriovenous malformations.

Reticular Activating System

- Critical for the awake, conscious state
- Dorsal pathway:
  - Pons nuclei- Pedunculo-pontine tegmentum and Lateral dorsal tegmentum
  - Cholinergic projections go to:
    - thalamus (reticular thalamic nuclei)
    - medulla (sublateral dorsal nucleus)
- Ventral pathway: Mesopontine and hypothalamic nuclei
  - Monoaminergic projections go to thalamus, hypothalamus, basal forebrain, cerebral cortex
  - Areas involved:
    - Locus ceruleus- Norepinephrine
    - Ventral lateral medulla- Norepinephrine
    - Periaqueductal gray- Dopamine
    - Dorsal medial raphe- Serotonin
    - Hypothalamic tubulomammillary nuclei- Histamine
    - Lateral hypothalamus- Orexin or hypocretin
Sleep Pathways

- Sleep is generated in the anterior hypothalamus and basal forebrain.

- Thalamic areas involved in sleep:
  - Anterior and dorsomedial thalamic nuclei
  - Reticular thalamic nuclei
These Areas on MRI
Mammillary body

Thalamus

Hypothalamus
Pituitary gland

Pituitary stalk/infundibulum
Mesial Temporal Lobe

Hippocampus

From researchgate.net
MRA anatomy
Brain/ Brainstem Pathology on Imaging

- Most common - small vessel disease
- Infarct/ hemorrhage
- Neoplastic
  - Intra-axial
  - Pituitary/ Suprasellar/ Pineal regions
  - Extra-axial
- Inflammation/ Demyelinating Disease/ Autoimmune Disease
- Trauma
- Infection
- Congenital Anomalies
Small vessel disease/ chronic microvascular ischemia
# WMLs differential diagnosis

<table>
<thead>
<tr>
<th>Hypoxic/ischemic</th>
<th>Toxic/metabolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Atherosclerosis</td>
<td>- CO-intoxication, B12 deficiency</td>
</tr>
<tr>
<td>- Hyperhomocystinaemia</td>
<td>- Central pontine myelinolysis</td>
</tr>
<tr>
<td>- Amyloid angiopathy</td>
<td></td>
</tr>
<tr>
<td>- Diabetic microangiopathy,</td>
<td></td>
</tr>
<tr>
<td>- Hypertension</td>
<td></td>
</tr>
<tr>
<td>- Migraine</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Inflammation</th>
<th>Traumatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>- MS</td>
<td>- Radiotherapy</td>
</tr>
<tr>
<td>- Vasculitis: SLE, M. Behcet, Sjögren,</td>
<td>- Postcontusion</td>
</tr>
<tr>
<td>- Sarcoid,</td>
<td></td>
</tr>
<tr>
<td>- Inflammatory bowel disease</td>
<td></td>
</tr>
<tr>
<td>- (Crohn, colitis ulcerosa, coeliakie)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infectious</th>
<th>Hereditary</th>
</tr>
</thead>
<tbody>
<tr>
<td>- HIV, syphilis, Lyme (borreliose),</td>
<td>- Metabolic (symmetrical, dd: toxic)</td>
</tr>
<tr>
<td>- PML: progressive multifocal leukencephalopathy</td>
<td></td>
</tr>
<tr>
<td>- postinfectious: ADEM</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>- VR-spaces - Fazekas I</td>
</tr>
</tbody>
</table>
Infarcts and Hemorrhage
Acute medullary infarct

DWI (bright in acute stroke)

ADC (dark in acute stroke)

Axial T2 (brighter as stroke becomes more subacute and matches CSF in chronic stage)
Acute Pontine Infarcts

Occlusion of the intracranial vertebral arteries and proximal basilar artery with flow in the distal basilar artery.
Acute Thalamic Infarct

DWI

Axial T2
Bilateral Thalamic Infarcts
Acute Left Posterior Cerebral Artery Infarct

Bright on DWI

Dark on ADC

Abrupt cut off of the left PCA
Acute Infarct Left Hippocampus
Anoxic Injury

Diffuse signal abnormality throughout the cortex as well as the basal ganglia - most metabolically active brain areas.
Pontine Hemorrhage

CT

Axial gre
Intraparenchymal Hemorrhage

- Most common cause: trauma
- Non-traumatic Causes:
  - Hypertension
  - Coagulopathy
  - Underlying tumor
  - Underlying vascular malformation
  - Vasculitis
  - Drug abuse
  - Venous outflow obstruction
  - Stroke
  - Arteriopathy
Temporal Lobe Volume Loss
Intra-axial Tumors and Vascular Lesions
Brainstem gliomas account for ~25% of all posterior fossa tumors and are most common in children between 7 and 9 years of age. Brainstem gliomas are also seen in adults, although they are rare accounting for only 2% of adult brain tumors. They typically occur in younger adults (third and fourth decade) and tend to be of low grade.
Tectal Glioma
Hypothalamic Glioma
Hippocampal Glioma
Cortical Glioma

- Dysembryoplastic neuroepithelial tumors (DNET) are benign (WHO Grade I), slow growing, glioneuronal tumors.
- Usually found in cortical gray matter and are frequently associated with cortical dysplasia.
- They characteristically cause intractable partial seizures.
Hemangioblastoma

- Tumors of vascular origin and occur both sporadically and in patients with von Hippel Lindau syndrome.
- WHO Grade 1 tumor.
- Generally present on imaging as sharply demarcated homogeneous masses composed of a cyst with non-enhancing walls, a mural nodule which vividly enhances, often with prominent serpentine flow voids.
- Typically occur in young to middle-aged adults.
- Most frequently present with symptoms relating to:
  - Headaches: 70%
  - Hydrocephalus and symptoms of raised intracranial pressure: 50%
  - Cerebellar dysfunction: ~50-60%
  - Altered mental state: 10%
Metastatic Disease to Temporal Region- Intra-axial
Cavernoma

- Cavernous malformation/ slow flow venous malformation
- The majority of lesions remain asymptomatic throughout life and are found incidentally.
- Presentation due to hemorrhage may cause seizures or a focal neurological deficit depending on the location of the lesion.
- The risk of hemorrhage is 1% per year for familial cases and less for sporadic lesions.
- Composed of a cluster of dilated thin-walled capillaries, with surrounding hemosiderin. No normal brain between the interstices of these lesions.
- On MRI have a rim of signal loss due to hemosiderin deposition.
Arteriovenous malformation (AVM)