Continuing Education Seminar for Radiologic Technologists

Fundamental Clinical Brain MR Imaging – Applications and Protocols

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Objectives

- Review fundamental clinical cases that illustrate the wide array and utility of available brain MR imaging techniques/applications
- Gain insight into the rationale behind MR imaging protocols
- Develop a greater understanding of potential points of patient care impact

Outline

- Review fundamental clinical cases that illustrate the wide array and utility of available brain MR imaging techniques:
  - Gradient echo
  - Diffusion
  - FLAIR
  - IR/SPGR / MP-RAGE
  - MRA / MRV
  - Spectroscopy
  - Perfusion
- Review potential points of patient care impact:
  - Additional patient history?
  - Are the referring physician and/or neuroradiologist aware of the findings?
  - Would an additional study(s) or different protocol be better?
  - Would intravenous contrast be helpful?

Brain – Screen

- Indications
  - Screen: Altered mental status, Dementia, Psychiatric disorder, Headaches
- Sequences
  - 3 PL LOC
  - Sag T1 SE
  - Sag T2 FLAIR
  - Ax T2 FLAIR
  - Ax T1 SE
  - Ax T2 TSE FC
  - Ax DWI EPI
  - Cor T2 TSE
- Comments
  - Axial scans should be parallel to the AC-PC line

Potential Points of Impact

- Patient history?
  - Do we know more than “mental status change”?
  - Onset of symptoms?
  - Hypertension? Pregnancy? Steroids?
- Sequences to consider anticipating?
  - MR brain without contrast => evaluate for acute ischemia
  - Avoid contrast with pregnancy!
Findings

- **CT Head:**
  - Posterior > anterior cerebral hemispheric white matter hypodensity
  - Bilateral cerebellar hypodensity

- **MRI Brain:**
  - Abnormal T2 / FLAIR hyperintense signal within bilateral cerebral hemispheric white matter (parieto-occipital > frontal) and bilateral cerebellar hemispheres
  - Small amount of diffusion restriction within right cerebellar hemisphere

**Posterior Reversible Encephalopathy Syndrome (PRES)**

- Associated with a multitude of diverse clinical entities:
  - Acute glomerulonephritis, preeclampsia / eclampsia, SLE, thrombotic thrombocytopenic purpura, and hemolytic-uremic syndrome, as well as drug toxicity
  - Cytotoxic edema of cerebral ischemia demonstrates decreased water mobility
  - Vasogenic edema due to cerebrovascular autoregulatory dysfunction results in increased water mobility

- Most cases manifest with acute to subacute hypertension, and seizures are also frequent

- Two pathophysiologic mechanisms:
  - Cerebral vasospasm and resulting ischemia within the involved territories
  - Breakdown in cerebrovascular autoregulation with ensuing interstitial extravasation of fluid

**Middle-aged female with new onset parasthesias**
Potential Points of Impact

- Patient history?
  - Do we know more than “parasthesias”?  
  - Onset of symptoms? Prior history?
  - Neurologic deficits?

- Sequences to consider anticipating?
  - Sagittal FLAIR imaging (eg, multiple sclerosis)
  - Post-contrast images

Axial T1

Axial T2

Axial FLAIR

Sagittal T1

Sagittal FLAIR
Findings

• MRI Brain:
  – Hyperintense FLAIR signal scattered throughout the frontal-parietal white matter
  – Involves corpus callosum, immediate pericallosal white matter, and callosal-septal junction on the sagittal FLAIR sequence

Multiple Sclerosis

• Inflammatory demyelinating condition of the central nervous system (CNS) that is generally considered to be autoimmune in nature
• White matter tracts are affected, including those of the cerebral hemispheres, infratentorium, and spinal cord
• Clinical diagnosis supported by radiologic findings

Potential Points of Impact

• Patient history?
  – Do we know more than “obtundation”?
  – Onset of symptoms?
  – History of cardiopulmonary arrest?
• Are the referring physician and/or neuroradiologist aware?
• Other studies to consider anticipating?
  – MRI => confirm suspected acute ischemia

3 month-old male with obtundation
Findings

- Diffuse reversal of gray/white matter densities
  - Decreased density of cerebral cortical gray matter
  - Relatively increased density of thalami, brainstem, and cerebellum
- Relatively decompressed ventricles, with diffuse loss of sulcation and effaced suprasellar cistern
  - Indicative of diffuse cerebral edema and early transtentorial herniation

Diffuse hypoxic-ischemic cerebral injury

- Major cause of morbidity in children
  - Clinical discrepancies should raise possibility of nonaccidental trauma
- Several possible reasons for anoxic injury:
  - Anoxic anoxia - not enough oxygen - uncommon
  - Anemic anoxia - not enough blood or hemoglobin
  - Acute hemorrhage
  - Chronic anemia
  - Carbon monoxide poisoning
  - Stagnant (ischemic) anoxia (hypoxic-ischemic injury) - not enough blood flow - most common
    - Localized (such as ischemic strokes)
    - Generalized (circulatory collapse / arrhythmias / cardiac arrest)
- CT reversal sign (reversal of gray/white matter densities)

Headache

Potential Points of Impact

- Patient history?
  - Do we know more than "headache"?
  - Onset of symptoms? Recent trauma?
  - Neurologic deficits?
- Are the referring physician and/or neuroradiologist aware?
Findings

- MRI Brain:
  - Hyperintense FLAIR signal within scattered cerebral sulci
  - Unremarkable on other sequences
  - Patent basilar cisterns

Subarachnoid hemorrhage

- Differential diagnosis of hyperintense FLAIR signal within subarachnoid space:
  - Subarachnoid hemorrhage
  - Meningitis / pus
  - Carcinomatosis
  - Supplemental oxygen
    - Caution: Most sedated / general anesthesia patients will have this finding—be careful not to overcall in this setting!

Brain – Tumor or Infection

- Indications:
  - Tumor, Infection, Meningitis

- Sequences:
  - 3PL LOC
  - SAG T1 SE
  - SAG T2 FLAIR
  - AX T1 SE
  - AX T2 TSE FS
  - AX DW EPI
  - SAG T1 IRSPGR 3D +C
  - AX T1 IRSPGR 3D +C MPR
  - COR T1 SE FS +C OPT
  - SAG T1 SE +C OPT
  - AX T1 SE +C OPT

  - Optional
    - SPECT – Single Voxel
    - SPECT – Multi Voxel

Potential Points of Impact

- Mental status change

- Patient history?
  - Do we know more than “mental status change”? Onset of symptoms? Neurologic deficits? Fever? Elevated WBC?

- Other studies to consider anticipating?
  - MRI brain => include post-contrast images
  - Include ADC map to assess diffusion restriction
Findings

- **MRI Brain:**
  - Left frontal lobe intra-axial lesion with fluid-like signal characteristics
  - Bright T2, low FLAIR, low T1
  - Partial low T2 rim, peripheral enhancement
  - Large area of central restricted diffusion
  - Marked surrounding vasogenic edema

Cerebral abscess

- **Key characteristics:** central diffusion restriction; rim enhancement; vasogenic edema
- **Differential diagnosis:**
  - Non-Hodgkin's lymphoma
  - Glioblastoma (GBM)
  - Tumefactive MS
  - Metastasis
  - Resolving hematoma
  - Infarct

Potential Points of Impact

- **Mental status change**
- **Patient history?**
  - Do we know more than “mental status change”?
  - Onset of symptoms?
  - Neurologic deficits? Fever? Elevated WBC?
- **Other studies to consider anticipating?**
  - MRI brain => include post-contrast images, particularly if infectious process is suspected
Findings

- **CT Head:**
  - Layering debris within dilated ventricles
  - Gross pus on ventriculostomy

- **MR Brain:**
  - Extensive leptomeningeal enhancement (including brainstem, cord) and ependymal enhancement
  - Parenchymal enhancement along lenticulostriate distribution
  - Scattered small infarcts
  - Diffusion restriction within layering intraventricular pus
  - Hydrocephalus
Meningitis / Ventriculitis

- Debris in ventricles with ependymal enhancement → ventriculitis
- Can have ependymal enhancement with NHL, spread of GBM, etc. but usually more nodular/focal and w/o debris
- Often associated with abscess rupture or indwelling shunt catheter
- 40-80% mortality but often indolent

Potential Points of Impact

- Patient history?
  - Do we know more than "severe headache"?
  - Any neurologic symptoms?
  - Recent surgery?
- Other studies to consider anticipating?
  - MRI brain WITH contrast – concern for meningitis
Findings

MR: Brain without and with contrast:
- Diffuse thin pachymeningeal / dural enhancement
- Depressed appearance of brain and brainstem
- Small bilateral extra-axial fluid collections which are T1 isointense and T2 hyperintense (3 mm maximum diameter)

Intracranial Hypotension

- Result of low CSF volume caused by:
  - Head trauma
  - Tear in spinal nerve root sheath, perineural cyst, or spinal arachnoid diverticulum
  - Iatrogenic causes
    - Lumbar puncture
    - Overdraining ventricular or spinal shunts
  - Spontaneous
    - Results from rupture of spinal arachnoid membrane, which allows CSF passage into subdural or epidural space
- MR imaging:
  - Diffuse, smooth dural / pachymeningeal enhancement
  - Contrast material accumulates in engorged dural veins and in interstitium of dura
  - Subdural fluid collections
  - Mostly bilateral and without significant mass effect
  - Brain descent
    - Inferior displacement relative to incisural line

Brain – Trauma / Hemorrhage

- Indications
  - Trauma, Hemorrhage
- Sequences
  - 3 PL LOC
  - Sag T1 SE
  - Sag T2 FLAIR
  - Ax T2 FLAIR
  - Ax T1 SE
  - Ax T2 TSE FS
  - Ax DWI EPI
  - Cor T2 TSE
  - Ax GRE
- Comments
  - Axial GRE should have TE>25

7-month old male with scalp swelling
Potential Points of Impact

• Patient history?
  – Do we know more than “scalp swelling”?
  – History of trauma or fall?
• Are the referring physician and/or neuroradiologist aware?
  – Must exclude non-accidental trauma
• Other studies to consider anticipating?
  – CT => characterize suspected fracture and evaluate for potential associated intracranial hemorrhage
  – Soft tissue and bone algorithms
  – MRI => assess for additional parenchymal injuries and potentially assist with injury / hemorrhage dating
Findings

- Skull radiographs:
  - Suspected oblique right parietal skull fracture

- CT head without contrast:
  - Asymmetric mid right frontoparietal scalp soft tissue swelling, with suspected nondisplaced oblique right parietal skull fracture
  - Heterogeneously hyperdense lenticular right parietal extra-axial fluid collection (2 cm maximum thickness), with relative effacement of adjacent sulci and right lateral ventricle

- MRI Brain without contrast:
  - Heterogeneously lenticular right parietal extra-axial fluid collection (T1 hyperintense; T2/FLAIR mildly hyperintense), with relative effacement of adjacent sulci and right lateral ventricle

Non-accidental trauma

- CT – recommended initial evaluation of non-accidental trauma
  - High sensitivity in detecting acute intracranial blood, fractures, cerebral edema and hypoxic ischemic injury
  - Attenuation of subdural / epidural hematoma varies by chronicity:
    - Acute – hyperdense
    - Subacute – isodense
    - Chronic – hypodense
  - Various factors such as low hematocrit and active hemorrhage may affect overall density

- MRI – essential second investigation
  - Best performed 5-10 days after insult
  - Can reliably differentiate between acute and chronic subdural hematoma
  - Most sensitive modality for detecting early ischemic changes
  - Clearly delineates anatomical locations that are difficult to image with CT
  - Posterior fossa, anterior part of middle cranial fossa, close to inner table of skull

80 year-old male with dementia that has progressed over the past 4 years

Potential Points of Impact

- Patient history?
  - Do we know more than “dementia”?
  - Previous CVA symptoms? Risk factors?
  - Neurologic deficits?

- Other studies to consider anticipating?
  - GRE imaging – evaluate for previous hemorrhage associated with infarcts
Findings

- MRI Brain:
  - Multiple foci of signal loss / “blooming” in cortical-subcortical locations
  - Consistent with chronic microhemorrhages

Cerebral Amyloid Angiopathy

- Cerebrovascular disorder characterized by deposition of β-amyloid protein in the media and adventitia of small and medium-sized vessels
- Both sporadic and hereditary forms may occur
- Manifests radiologically as part or all of a constellation of findings including acute or chronic ICHs in a distinctive cortical-subcortical distribution, leukencephalopathy, and atrophy

Brain – Seizure / Dysplasia

- Indications
  - Seizure, Dysplasia, Mesial temporal sclerosis
- Sequences
  - 3 PL LOC
  - Sag T1 SE
  - Ax T2 FLAIR
  - Ax T1 SE
  - Ax T2 TSE FS
  - Ax DWI EPI
  - Cor T2 TSE FS (angled perpendicular to temporal lobes)
  - Cor FLAIR (angled perpendicular to temporal lobes)
  - Ax IR SPGR / MP-RAGE or Cor IR-SPGR / MF-RAGE
- Comments
  - Coronal sequences should be thin section perpendicular to the long axis of the hippocampus
2 year-old female with seizures

Potential Points of Impact

• Patient history?
  – Do we know more than "seizures"?
  – Onset of symptoms?
  – Prior studies / previous surgery / trauma?
• Pulse sequences to consider anticipating?
  – Axial IR-SPGR / MP-RAGE
  – Coronal T2 and FLAIR through hippocampi

Findings

• MRI Brain:
  – In a normal brain, white matter is in the interior, and gray matter is mostly on the surface

  – In patients with periventricular nodular heterotopia, clumps of gray matter, called nodules, appear deep within the brain, instead of on the surface

  – Image courtesy of Bernard Chang, MD, Beth Israel Deaconess Medical Center (via Internet for teaching)

Periventricular Nodular Heterotopia

• In a normal brain, much of the gray matter (consisting mostly of nerve cells) appears on the brain's surface, while white matter (consisting mostly of nerve fibers, or "wiring" interconnecting areas of gray matter) runs deeper in the brain

• In PNH, a migrational abnormality occurs during development – portions of gray matter sit deep in the brain's core, in the white matter, having failed to migrate out to the surface
  – May serve as elliptogenic foci
Brain – Advanced Protocols

• Dural venous sinus thrombosis
  – Cor 2D TOF SPGR
  – Sag 2D TOF SPGR (slight oblique angle)

• Stroke, TIA, Vertebrobasilar infarct, Aneurysm
  – Ax 3D TOF SPGR
  – Ax Perfusion

• Tumor, Metabolic abnormality
  – Single voxel spectroscopy (short and long echo; eg. TE 35 and 144) on all new mass lesions
  – Multi voxel spectroscopy - suspected gliomas
  – Perfusion
    • Gd – 20 ml @ 3-5 ml/s

Potential Points of Impact

• Patient history?
  – Do we know more than “mental status changes”?
  – Any neurologic symptoms?
  – Recent surgery? Dehydration?

• Are the referring physician and/or neuroradiologist aware?

• Other studies to consider anticipating?
  – CTV head + contrast; reconstructions / MIP images
  – MRI brain – evaluation for ischemia / hemorrhage

30 year-old female with mental status changes
Findings

- CT Head without contrast:
  - Tubular hyperdensity within superior sagittal sinus, torcula, and left frontal cortical veins

- CT Venography with contrast:
  - Extensive irregular filling defects within superior sagittal sinus extending to torcula, left transverse sinus, and left sigmoid sinus
  - Irregular filling defects within left frontal cortical veins

- MRI Brain without contrast:
  - Tubular T1 hyperintense signal and loss of normal flow void within superior sagittal sinus, torcula, left transverse sinus, and left frontal cortical veins
Dural Venous Thrombosis

• "empty delta sign"
  - With CT venography, the thrombus does not enhance but dura enhances
  - Triangular defect can be demonstrated within superior sagittal and transverse sinuses
  - Seen in 25-75% of cases
  - Because of volume averaging, may fail to demonstrate a hyperattenuating sinus or an empty delta sign in horizontal segment of superior sagittal sinus or transverse sinus
  - Can disappear in chronic stages with enhancement of organized clot

Dural Venous Thrombosis

• "dense triangle sign"
  - Hyperdense triangular shaped structure along posterior aspect of superior sagittal sinus

• "cord sign"
  - Hyperdense tubular superficial cortical vein or dural venous sinus

• Represent intravascular acute blood clots
  - Takes approximately 1–2 weeks to disappear

Caution
  - Similarly increased attenuation of cerebral venous sinuses may also represent polycythemia
  - Nonmyelinated brain in neonates makes sinuses appear hyperattenuating

Dural Venous Thrombosis

• MRI
  - Main sign is lack of expected signal flow void on standard T1- and T2-weighted images
  - Challenging diagnosis in acute stage
    - Hypointense signal of acute thrombus mimics normal flow void on T2-weighted images
  - Absence of flow void on T1-weighted images must be carefully sought because thrombus may be isointense / mildly hyperintense to brain tissue

Potential Points of Impact

• Patient history?
  - Do we know more than "headaches"?
  - Any neurologic symptoms?

• Concern for cerebral aneurysm?

• Other studies to consider anticipating?
  - CTA head - reconstructions / MIP images
  - Cerebral angiogram – if warranted
Findings

- **CT head angiography:**
  - Large aneurysm projects superiorly from basilar tip
  - 20 mm CC, 13 mm transverse, and 23 mm AP
  - Origins of both posterior cerebral arteries and superior cerebellar arteries appear incorporated into aneurysm neck
  - Aneurysm projects superiorly into third ventricle and compresses foramen of Monro

- **Cerebral angiography:**
  - Large, lobulated aneurysm at basilar artery tip
  - Left P1 segment arises from base of aneurysm
  - Right P1 segment appears clear

Basilar Tip Aneurysm

- Occurs at distal bifurcation of basilar artery, between origin of two posterior cerebral arteries

- Intracranial aneurysm distribution:
  - 30-35% => anterior communicating artery
  - 30-35% => posterior communicating artery origin
  - 20% => middle cerebral artery bifurcation
  - 5% => basilar artery bifurcation or tip
  - 5% => other posterior fossa vessels

- Conventional angiography – remains gold standard for detection and characterization of cerebral aneurysms

- CTA can detect more than 95% of aneurysms identified on conventional angiography

Potential Points of Impact

- **Patient history?**
  - Do we know more than “tumor”?
  - Previous surgery?
  - Neurologic deficits?
  - Outside images available for radiologist review?

- **Other studies to consider anticipating?**
  - MR spectroscopy
  - MR perfusion
  - Post-contrast IR-SPGR (for radiation therapy)
Findings
- MR Spectroscopy:
  - Increased choline
  - Decreased NAA
  - Decreased creatine

MR Spectroscopy
- Useful in tumor evaluation and surgical / biopsy planning
- Although water and fat contribute virtually all of the signal in proton MR imaging, it is possible to suppress these signals and assess the signal from other metabolites including choline, creatine, and NAA
  - Altered in concentration in various disease processes, particularly tumors
- Metabolic mapping of spectra allows rapid assessment of spectral peaks and choline map also demonstrates the most malignant site to biopsy
- Elevated choline probably represents the cell membrane breakdown secondary to the tumor, while NAA is a metabolite of normal neuronal tissue

Potential Points of Impact
- Patient history?
  - Do we know more than "tumor"?
  - Previous surgery and/or biopsy results?
  - Neurologic deficits?
  - Outside images available for radiologist review?
- Other studies to consider anticipating?
  - MR spectroscopy
  - MR perfusion
  - Post-contrast IR-SPGR (for radiation therapy)
Findings

- MR Brain:
  - Large avidly enhancing mass centered within the left insula

- MR Perfusion:
  - Significant patchy hyperperfusion – increased CBF compared with surrounding cortex / juxtacortical white matter

MR Perfusion

- Useful in brain tumor evaluation and surgical / biopsy planning
- Uses contrast which has slightly different magnetic characteristics from blood – causes a disturbance in the localized magnetic field
- Signals are analyzed mathematically and expressed as an image (e.g. CBF, CBV, MTT maps). By offsetting the changes in shape in the flow of the contrast bolus against time, it is possible to calculate how much blood is reaching the area of concern within the brain.

THANK YOU!

HAVE A GREAT DAY!