Evaluating Pulsatile Tinnitus

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Echo mammaire : un luxe ?

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MR imaging in patients with tinnitus

Tinnitus

T2/T1 of the brain

T2-W GE/TSE of the temporal bone
Un-enhanced MR-Angiography (TOF)
Gd-enhanced MR-Angiography
T1+Gd of the temporal bone

severe/un-explained fMRI

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Disclosures

- Philips Medical Systems (The Netherlands)
  - Clinical Training
  - Lectures
  - Customers site visits
- NewTom (Italy)
  - Hardware & software provide for testing

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Temporal bone: head-coil

Flex-S coils needed at 1.5 T, the same quality is possible
the head-coil at 3.0 T less manipulations for nurses!

Frontal
Meningomas
Occipital

ASNR 53rd Annual Meeting
April 25-30, 2015, Chicago, USA
Neuro-vascular conflicts a cause of tinnitus?
- Localisation of the conflict at the REZ
- Conflict in the IAC, bone conduction?
- Point of contact (clock), volume CPA
- Dynamic visualisation of conflict

Conflict in the CPA, no tinnitus, no bone conduction

Findings supporting the diagnosis (experience with nerve V-VII)
- REZ and/or CNS-segment
- Artery (> than vein)
- Vessel crosses nerve in perpendicular way
- Displacement of nerve

Score x/4

Pulsatile tinnitus
- T2/T1 of the brain
- T2 GE/TSE Temporal bone
- un-enhanced MRA (TOF)
- Gd-enhanced MRA (TOF)
- 3D(FFE) T1 + Gd TBone
- severe/un-explained
- conventional angiography

Non-pulsatile tinnitus
- T2/T1 of the brain
- T2 GE/TSE Temporal bone
- un-enhanced MRA (TOF)
- Gd-enhanced MRA (TOF)
- 3D(FFE) T1 + Gd TBone
- severe/unexplained
- or bilateral fMRI

Pulsatile tinnitus on the right
- AICA
However, whatever we look at, the predictive value of imaging is poor.

**MR imaging in patients with tinnitus**

**Vascular Loops at the Cerebellopontine Angle: Is There a Correlation with Tinnitus?**

G. Katibilan, K. Colha, G. Aydogdu, T. Oner, F. Capanlar, and M. Telikci

**Summary**

The advantage of phased array coils is that superficial vessels become visible

**TE = 2.3**

**Bandwidth = 1,000 → 3,000 pixels**

**TE = 6.9**

**Thickness = 0.7**

**Slices: 125**

**TE = 6.9**

**Thickness = 0.3**

**Slices: 324 (SENSE needed)**
MR imaging in patients with tinnitus

- Dural fistulas & glomus tumors, best when MR-technique is used
  - TOF un-enhanced sequence
  - Adapted for more peripheral vessels (TE)
  - Covers the complete region at risk (+ upper neck)
  - Isotropic submillimetric

Glomus Tumor

Glomus jugulotympanicum
Left pulsatile tinnitus

Dural fistula

TOF angio-MR, without Gd in a patient with pulsatile

Precocious filling of the sigmoid sinus
<table>
<thead>
<tr>
<th>Aplasia of the ICA (3-year-old child)</th>
<th>ICA aneurysm</th>
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MR imaging in patients with tinnitus:

- **Pulsatile tinnitus**
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  - (un-enhanced MRA (TOF))
  - (Gd-enhanced MRA (TOF))
  - 3D(FFE) T1 + Gd TBone

- severe/unexplained
- conventional angiography

- **The value of Gd-enhanced T1-weighted images**
  - Detection of schwannomas
  - Detection of meningiomas
  - Idiopathic Intracranial Hypotension
  - Detection of other causes...
Meningioma en plaque

Otosclerosis

Spontaneous intracranial hypotension

20% have Ménière-like cochleovestibular symptoms
- Tinnitus
- Vertigo
- Low frequency SNHL

T2/T1 of the brain                    T2/T1 of the brain
Pulsatile tinnitus                 Non-pulsatile tinnitus
T2 GE/TSE Temporal bone             T2 GE/TSE Temporal bone
un-enhanced MRA (TOF)                un-enhanced MRA (TOF)
Gd-enhanced MRA (TOF)                Gd-enhanced MRA (TOF)
3D(FFE) T1 + Gd TBone                3D(FFE) T1 + Gd TBone

severe/unexplained or bilateral fMRI

Why is CT still needed
In case of severe tinnitus and negative MR study CT is performed to exclude:
- Jugular diverticulum or ear
- Protruding and/or dehiscent jugular bulb
- Paget's disease
- Otosclerosis
- Stapedial artery etc.

MR imaging in patients with tinnitus

MR imaging in patients with tinnitus
Protruding and dehiscent jugular bulb

Paget’s disease: cotton wool pattern

**Conclusion**

- The most frequent causes of pulsatile tinnitus must be excluded:
  - Dural fistulas: MRA – (MRA +)
  - Glomus tumours: MRA -
  - VIIIth nerve schwannomas: submillimetric 3D T1+Gd & 3D TSE/GE T2
  - Meningiomas: 3D T1+Gd
  - More rare pathology detected by same sequences
- Non-pulsatile tinnitus
  - Auditory fMRI

**MR imaging in patients with tinnitus**
ACUTE HEARING LOSS AND NON-PULSATILE TINNITUS

JOEL D. SWARTZ, M.D.

NOTHING TO DISCLOSE

ACUTE HEARING LOSS

• CONGENITAL
• INFLAMMATORY
• NEOPLASTIC
• ISCHEMIC
• TRAUMATIC

ACUTE HEARING LOSS

• CONGENITAL/DEVELOPMENTAL
• INFLAMMATORY
• NEOPLASTIC
• VASCULAR
• TRAUMATIC

LARGE VESTIBULAR AQUEDUCT (LARGE ENDOLYMPHATIC DUCT AND SAC)

MOST COMMON DEMONSTRABLE FINDING RELATED TO CONGENITAL SENSORINEURAL HEARING DEFICIT

ACUTE HEARING LOSS ASSOCIATED WITH A RELATIVELY MINOR PRECIPITATING EVENT IS A CLASSIC CLINICAL MANIFESTATION OF THIS ENTITY

TRAUMA
UPPER RESPIRATORY INFECTION
BAROTRAUMA

MONDINI DEFORMITY: Incomplete Partition II
ACUTE HEARING LOSS

• CONGENITAL
• INFLAMMATORY
• NEOPLASTIC
• ISCHEMIC
• TRAUMATIC

LABYRINTHITIS

• INVASION OF PERILYMPHATIC SPACES OF THE INNER EAR
• SECONDARY CHANGES IN THE ENDOLYMPHATIC SPACES (MEMBRANOUS LABYRINTH)
• SNHL AND VERTIGO - OFTEN RECURRENT OR DEBILITATING

LABYRINTHITIS CLASSIFICATION

ROUTE OF SPREAD

• TYMPANOGENIC
• MENINGOGENIC
• HEMATOGENIC
• POSTTRAUMATIC

AGENT

• VIRAL
• BACTERIAL
• AUTOIMMUNE
• LUETIC

TYMPANOGENIC LABYRINTHITIS

► MIDDLE EAR DISEASE, UNILATERAL
► PROPAGATION OF DEBRIS INTO LABYRINTH VIA THE OW, RW OR LABYRINTHINE FISTULA
► IATROGENIC - PROSTHETIC STAPEDECTOMY

MENINGOGENIC LABYRINTHITIS

► MENINGITIS, USUALLY BILATERAL
► VIA IAC TO VESTIBULE OR COCHLEAR APEX
► COCHLEAR AQUEDUCT LESS COMMON
► MOST COMMON CAUSE OF ACQUIRED CHILDHOOD DEAFNESS

ACUTE/SUBACUTE LABYRINTHITIS

► CT IS NORMAL
► SOLE IMAGING FINDING IS ENHANCEMENT OF THE NORMALLY NON-ENHANCING FLUID-FILLED SPACES OF THE LABYRINTH ON ENHANCED T1WI
► MAJORITY OF PATIENTS WITH ACUTE/SUBACUTE LABYRINTHITIS WILL NOT HAVE LABYRINTHINE ENHANCEMENT (OR ANY OTHER IMAGING FINDING)
**ACUTE/SUBACUTE LABYRINTHITIS**

- Rare, immune complexes damage membranous labyrinth
- [+] Lymphocyte transformation test (93% specific, 50-80% sensitive)
- Cogan’s syndrome: prototypical autoimmune inner ear disorder: nonsyphilitic interstitial keratitis and auditoryvestibular dysfunction, usually with preceding URI
- Imaging findings reminiscent of labyrinthitis, hearing loss may by of sudden onset

**AUTOIMMUNE LABYRINTHITIS**

**ACUTE HEARING LOSS**

- Congenital
- Inflammatory
- Neoplastic
- Ischemic
- Traumatic

**SCHWANNOMA OF THE 8TH CRANIAL NERVE**

- Acoustic neuroma

- 10% of patients with acoustic tumors present with acute hearing loss
- 1% of patients presenting with acute hearing loss will have acoustic tumors.

**ACUTE HEARING LOSS**

- 10% of patients with acoustic tumors present with acute hearing loss
- 1% of patients presenting with acute hearing loss will have acoustic tumors.

**Cogan Syndrome**

(Case courtesy Bernadette Koch, M.D.)

**CHILD WITH ORBITAL PSEUDOTUMOR, UVEITIS AND SNHL**

**SCHWANNOMA WITH HEMORRHAGE**

(Courtesy R. Wiggins)
ACOUSTIC NEUROMA
LARGER LESIONS MAY HAVE CYSTIC OR NECROTIC COMPONENTS

COURTESY R. WIGGINS

MENINGEAL METASTASES

IDENTICAL APPEARANCE
- MENINGITIS (USUALLY GRANULOMATOUS)
- SARCOIDOSIS
- LEUKEMIA/LYMPHOMA

T1C- AXIAL
T1C+ CORONAL

T1C+ CORONAL

ACUTE HEARING LOSS
• CONGENITAL
• INFLAMMATORY
• NEOPLASTIC
• ISCHEMIC
• TRAUMATIC

*ANTERIOR INFERIOR CEREBELLAR ARTERY (INTERNAL AUDITORY ARTERY ARISES IN >90%)

POSTERIOR INFERIOR CEREBELLAR ARTERY
SUPERIOR CEREBELLAR ARTERY

ARTERIAL SUPPLY TO THE COCHLEA:
❖ BASILAR TURN (HIGHER FREQUENCIES) IS FED FIRST BY THE MAIN COCHLEAR ARTERY
❖ COCHLEAR APEX (LOWER FREQUENCIES) FED LAST
❖ PREDOMINANCE OF LOWER FREQUENCY AUDITORY DISTURBANCE

ARTERIAL SUPPLY TO THE COCHLEA:

AUDITORY PATHWAY
• COCHLEA - COCHLEAR NERVE
• INTERNAL AUDITORY CANAL
• CEREBELLOPONTINE ANGLE
• MEDULLA (COCHLEAR NUCLEI)
• TRAPEZOID BODY (CROSSED FIBERS)
• LATERAL LEMNISCUS
• MIDBRAIN (INFERIOR COLLICULI)
• THALAMUS (MEDIAL GENICULATE BODY)
• SUPERIOR TEMPORAL GYRUS

INTRA-AXIAL AUDITORY PATHWAY
• ISCHEMIC
• NEOPLASTIC
• TRAUMATIC
• DEMYELINATING
INTRA-AXIAL AUDITORY PATHWAY

UNILATERAL - TRUE UNILATERAL RETROCOCHLEAR LOSS CAN RESULT ONLY FROM AN INVOLVEMENT OF THE COCHLEAR NERVE OR COCHLEAR NUCLEI. THOSE LESIONS IN MORE PROXIMAL AUDITORY PATHWAY RESULT IN BILATERAL SNHL MORE NOTICEABLE ON CONTRALATERAL SIDE. CORTICAL (TEMPORAL) INSULTS RESULT IN AUDITORY AGNOSIA WHICH IS IMPAIRED INTERPRETATION OF SOUND.

COCHLEAR NUCLEI UPPER MEDULLA

LESIONS OF THE COCHLEAR NUCLEI LOCATED WITHIN THE POSTEROLATERAL ASPECT OF THE UPPER MEDULLA MAY CAUSE UNILATERAL RETROCOCHLEAR LOSS CLINICALLY INDISTINGUISHABLE FROM THAT CAUSED BY INTRACANALICULAR LESIONS.

ACUTE HEARING LOSS

- CONGENITAL
- INFLAMMATORY
- NEOPLASTIC
- ISCHEMIC
- TRAUMATIC

ACUTE POST-TRAUMATIC HEARING LOSS SENSORYNEURAL

- ‘COCHLEAR CONCUSSION’
- FRACTURE / PERILYMPHATIC FISTULA
- INTRALABYRINTHINE HEMORRHAGE
- AUDITORY PATHWAY

TRANSVERSE FRACTURE (FRACTURES WITH A TRANSVERSE COMPONENT)

- PERPENDICULAR TO LONG AXIS OF PETROUS PYRAMID
- BLUNT OCCIPITAL BLOW
- TEMPORAL BONE ENTRY POINT OFTEN NEAR VESTIBULAR AQUEDUCT
- MEDIAL/LATERAL SUBTYPES
**Transverse Fracture**

**Medial Subtype**
- Travesses fundus of IAC
- SNHL secondary to cochlear nerve transection
- Complete and permanent

**Lateral Subtype**
- Travesses bony labyrinth resulting in SNHL often with perilymphatic fistula (PLF)
- Perilymphatic fistula - communication between the middle ear and the inner ear

**Intralabyrinthine Hemorrhage**
- Non-contrast T1 weighted image
  - Trauma
  - Labyrinthitis
  - Coagulopathy
  - Tumor

Non-contrast T1 weighted images are crucial in this context so as not to confuse enhancement with hemorrhage.

**Acute Post-Traumatic Hearing Loss - Conductive**

Very common after injury.

Causes:
- Hematotympanum
- Tympanic membrane damage
- Ossicular discontinuity

Chl which persists after blood is resorbed and the TM is healed/repaird is presumably due to ossicular damage.

**Conductive Hearing Loss - Ossicular Discontinuity**

- Ossicular support:
  - Malleus: anterior/lateral/superior malleal ligaments, tympanic membrane (short process and manubrium)
  - Stapes: stapediovestibular articulation, stapedius tendon/incudostapedial articulation
  - Incus: relatively heavy (25G), minor ligamentous support
  - *Incus* - most vulnerable ossicle

*Presumed caused is tetanic contraction of tensor tympani and stapedius tendons

In order to diagnose, a sound knowledge of the normal anatomy is needed, best appreciated on axial images.
INCUDOSTAPEDIAL SUBLUXATION

- NORMAL INCUDOSTAPEDIAL ARTICULATION

CONDUCTIVE HEARING LOSS OSSICULAR DISCONTINUITY

- INCUDOSTAPEDIAL SUBLUXATION
- MALLEOINDUDAL SUBLUXATION
- INCUS DISLOCATION
- STAPES FRACTURE/DISLOCATION
- MALLEUS FRACTURE
- SIMILAR PATHOPHYSIOLOGY TO ISJS
- BEST SEEN ON AXIAL IMAGES AS SEPARATION OF ‘ICE CREAM’ FROM ‘CONE’

MALLEOINDUDAL SUBLUXATION

CONDUCTIVE HEARING LOSS OSSICULAR DISCONTINUITY

- INCUDOSTAPEDIAL SUBLUXATION
- MALLEOINDUDAL SUBLUXATION
- INCUS DISLOCATION
- STAPES FRACTURE/DISLOCATION
- MALLEUS FRACTURE
- SEPARATION FROM MALLEOINCUDAL AND INCUDOSTAPEDIAL ATTACHMENTS
- PARTIAL OR COMPLETE, IF COMPLETE- THE INCUS MAY RESIDE IN ATTIC, MIDDLE EAR, EAC, OR BE COMPLETELY ABSENT (RESORBED OVER TIME)

INCUS DISLOCATION

- Y-SHAPE
INCUS DISLOCATION

CONDUCTIVE HEARING LOSS
OSSICULAR DISCONTINUITY

• INCUS DISLOCATION
• STAPES FRACTURE/DISLOCATION
• MALLEUS FRACTURE

• TWISTING TORSION OF INCUS IS PRESUMED PATHOPHYSIOLOGY
• DIAGNOSIS MORE DIFFICULT BUT SHOULD BE SUSPECTED IF THE NORMAL STAPES IS NOT VISUALIZED IN THE AXIAL PLANE IN THE APPROPRIATE CLINICAL CONTEXT
• ASSOCIATION WITH PERILYMPHATIC FISTULA

STAPES FRACTURE/DISLOCATION

CONDUCTIVE HEARING LOSS
OSSICULAR DISCONTINUITY

INCUDOSTAPEDIAL SUBLUXATION
MALLEOINDUDAL SUBLUXATION
INCUS DISLOCATION
STAPES FRACTURE/DISLOCATION
MALLEUS FRACTURE

NORMAL COURTESY F. VEILLON
Evaluation & Endovascular Management of Vascular Skull Base Lesions

Robert W. Hurst MD
University of Pennsylvania

Outline

- Basics of Skull Base Endovascular Rx
  - Embolic Agents & Devices
  - Anatomic Considerations-Dangerous Anastomoses
  - Vascular Anomalies: Aberrant ICA
- Lesions:
  - Extradural carotid aneurysms
  - Tumors
    - Paraganglioma
    - Dural Arteriovenous Fistula

Embolization Agents

- Coils
- Particles: PVA
- Liquid Agents
  - nBCA
  - Onyx

“Dangerous Anastomoses”

- Arterial Anastomoses between ECA → ICA / VA
- Remnants of embryologic neural crest or matameric arterial systems
- Critical to maintain CNS supply in occlusive vascular Dz - Common
- Allow Emboli → CNS; Limit locations / agents for safe embolization
- Emphasize anatomy

Skull Base Vascular Lesions & Anomalies

- Aberrant ICA
- Aneurysm – Extradural
- Paraganglioma
- Dural AV Fistula
- Meningioma
- Cholesteatoma
- Nerve sheath tumor
- Chondroid tumor/chordoma
- Metastasis

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Aberrant Internal Carotid Artery

- Described: Lepayower - 1971
- ICA → Middle Ear
- Rare
- F >> M;
- R > L; 15% bilateral
- Hearing loss 55%
- Clinical: Pulsatile tinnitus; serous otitis media; Otolgia
- Reddish-blue tympanic mass
- Imaging: Enhancing mid. Ear mass; absent lat wall/vertical carotid canal

2004 Arch Otol. V.130; p112

Aberrant ICA: Embryology

- Failure of development of cervical ICA
- Hypertrophy of inferior tympanic & caroticotympanic aa.


Aberrant Internal Carotid Artery: Imaging

- Enlargement of the inferior tympanic canal
- Enhancing mass in the hypotympanum
- Absent bony posterior wall of the carotid canal

35 y.o. Female

- Right Sided Pulsatile Tinnitus
- Red mass seen on TM exam
- Dx: Glomus tympanicum
- Rx: Surgical resection

Aneurysms of the Extradural ICA

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<tr>
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<th>Freq.</th>
<th>Etiology</th>
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<td>15%</td>
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Cavernous ICA Aneurysms

- #1 Extradural ICA location
- 2.9% intracranial aneurysms
- F:M=10:1; 20% bilateral; frequently giant
- Px: Cav Sinus Mass:
  - Diplopia (CN 6,3,4)
  - Pain (CNS)
  - Rupture-CC fistula;
  - Hemorrhage (Mild)
- Etiology:
  - Congenital/Developmental
  - Most common
  - Traumatic
  - Mycotic (Rare)
Rx of Cavernous ICA Aneurysms

- **Extradural** → Small Cavernous Aneurysms
  - Rarely cause Sx; Often require No Rx

  - **Asymptomatic**
    - > 12 mm
    - Extends into SA Space
    - Extends into Sphenoid Sinus
    - Enlarging

- **Symptomatic (#1 = Cav Sinus Sx)**
  - SAH/ Epistaxis
  - CCF
  - Pain
  - Ophthalmoplegia, Visual loss

Rx of Cavernous Carotid Aneurysms

- **Flow Diverter Devices**
- Coiling ± Stent
- Carotid Occlusion
- Not Amenable to Surgical Rx

Pipeline® Embolization Device

- Braided cylindrical mesh device
- 30-35% surface coverage
- Implanted across the aneurysm neck and re-lines the diseased vessel
- Provides treatment option for complex unruptured aneurysms:
  - Large, Giant, Wide Neck, Fusiform
- 7-10% surface coverage

Flow Diversion

- Low-porosity stent in parent artery - reduces blood flow in aneurysm → stagnation and thrombosis
• 59 yo: L CN 6 palsy

• 9 mos later: Complete Aneurysm Occlusion

• 227 cavernous carotid aneurysms Rx'd w Pipeline: M/M = 0.4% / 3.1%

• Tanweer, AJNR 2014 35: 2734-2740

• 62-year-old: Iatrogenic right ICA pseudoaneurysm sustained during pituitary surgery
Coiling 31 CCAn: M/M = 0% (95% CI, 0 to 13.1%)

Van Rooij. AJNR 2012;33:323–26

68 Female

- Left CN 3, CN 6
- Excruciating pain and paresthesia in L V1

50 patients w ICA occlusion after occlusion test: M/M: 0 / 2% (95% CI, 0.01 to 11.5%; mortality, 95% CI, 0 to 8.5% morbidity) Van Rooij. AJNR 2012;33:323–26

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Petrous ICA Aneurysms

- Uncommon
- Sx:
  - Hearing loss (CHL or SNHL) often 1st Sx
  - HA, facial pain, tinnitus
  - Rupture w/o otorrhagia or epistaxis - 25%
- Triad of otorrhagia, epistaxis, & neuro deficit → unique
58 yo massive Epistaxis; Otorrhagia; Hypotension

Rx: Emergent Carotid Occlusion

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Angio: Cervical ICA Dissection

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<th>Author/ N</th>
<th>Stenosis</th>
<th>Aneurysm</th>
<th>Intimal Flap</th>
<th>ICA Occlusion</th>
<th>Branch Occlusion</th>
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<td>Mokri /65</td>
<td>75%</td>
<td>40%</td>
<td>30%</td>
<td>20%</td>
<td>10%</td>
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<td>Dżiewas /78</td>
<td>5%</td>
<td>40%</td>
<td></td>
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<td>40%</td>
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<td>Baumgartner /200</td>
<td>15%</td>
<td>15%</td>
<td>10%</td>
<td>1%</td>
<td>55%</td>
</tr>
<tr>
<td>Pelkonen /76</td>
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Angio: Cervical ICA Dissection

Angiogram: Cervical ICA Dissection

Cervical ICA Dissecting Aneurysms: Endovascular Rx

- Most require no specific Rx:
  - (71 pt.; 49.3% had total of 42 aneurysms)
    - F/U: 3 yr, none had Sx
    - 46% unchanged, 36% disappeared, 18% decreased in size [Frontal Strok, 2013, 322(3): p. 488-23]
- Rx if:
  - Symptomatic despite appropriate medical management → Embolization or Hypoperfusion
  - Sx from enlargement / mass effect / compression
  - Risk of hemorrhage into adjacent sinus or skull base cavity
34 yo female
- MVA
- Severe neck pain
- Initially normal neurologically
- MRI: Dissection
- Rx’d w ASA
- 5 days: L hemiparesis

Paraganglioma
- #2 skull base tumor (#1= acoustic neuroma)
- Neural crest origin → paraganglia (glomus bodies)
- Spread by local invasion (mets 5%)
- Catecholamine secretion in 1%

Paraganglioma

- 4 typical locations in carotid sheath/skull base
  - Carotid bifurcation - Carotid body tumors (#1 HN location)
  - CNX perineurium - Vagale
  - CN IX tympanic br - Tympanicum
  - Jugular bulb adventitia - Jugulare
- Larger paragangliomas (>1 cm) involve both: Tym & Jug - Jugulotympanic.


MR

- Low T1
- Hi T2
- “salt and pepper”
- intense homogeneous enhancement

Paraganglioma:Angio

- Enlarged aa.
- Tumor Stain
- Rapid venous drainage
- Jug. Vein compromise ➔ collaterals

- VanLennep, INR, 8: 127-134, 2002

Paraganglioma:Angio

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- VanLennep, INR, 8: 127-134, 2002
Angio Embolization

Preop embol reduces intraoperative blood loss significantly - esp. vagal, jug-tympanic paragangliomas


Carotid Body - Vagale Tumor

Dangerous Anastomosis: AsPha → VA

Pre-Embo
Dural Arteriovenous Fistula

- Disorder of Dura ➔ Abnormal AV shunts within dura-usually within/near dural sinus walls
- 0.16/100k; 10-15% of intracranial AVM
- M:F=1:3
- Etiology: Acquired opening of AV shunts between dural aa- vv
- Sinus thrombosis/trauma /infection/ surgery

Primary Rx:
Embo

DAVF Locations

- TS, SS: 20-60%
- Cav sinus: 20-40%
- Less Common Locations
- Propensity for drainage depends on Location ➔ Clin Course

Onyx in DAVF

- 50 patients; 63 cranial DAVFs
- Rx w Onyx ± other agents
- Complete angiogram @ 5 mo:
  - Onyx alone: 87%
  - Onyx + other agent: 79%
- Permanent complict.: 2%
Summary

- Basics of Skull Base Endovascular Rx
  - Embolic Agents & Devices
  - Anatomic Considerations-Dangerous Anastomoses
  - Vascular Anomalies: Aberrant ICA
- Lesions:
  - Extradural carotid aneurysms
  - Tumors
    - Paraganglioma
  - Dural Arteriovenous Fistula
ASNR 2015

High Resolution 3D MRI of the Skull Base

Ari M. Blitz, MD
Director, Skull Base Imaging
Assistant Professor, Neuroradiology
Johns Hopkins Hospital

Disclosures

- Honorarium, Siemens
- Study Reader, Bayer Pharmaceuticals
- Lead radiologist, Aesculab hydrocephalus study

- The content of this lecture does not constitute an endorsement of any product by the speaker or by Johns Hopkins Medical Institutions.

Objectives

- The second (companion) portion of this presentation will be delivered by Dr. Aygun
- This presentation will use visualization of the cranial nerves as a model for the different compartments of the skull base
- The participant will be able to list the cranial nerve segments.
- The participant will be able to name 3D MRI sequences that allow for visualization of the various cranial nerve segments.

Outline

Part I: (Blitz)
- Introduction
- Technique
- Cranial nerves
  - Segmental anatomy
  - Pathologic cases

Part II: (Aygun)
- Masses
  - Extent
  - Relation to critical structures
- Operative planning
- Summary

3D Isotropic Imaging

3D Skull Base Protocol

- Pre-contrast
  - VIBE
  - CISS
  - STIR
  - SPACE
- Post-contrast
  - VIBE w/ FS
  - CISS
**Skull Base Protocol (as hung for interpretation)**

Pre-contrast

Post-contrast

**Skull Base Protocol Parameters**

STIR SPACE VIBE CISS

Pre-contrast

Post-contrast

CISS VIBE FAT

Localizer performed 1st
Also often included:
- Sag T1 head
- Axial FLAIR head
- DWI head
- Axial T1 post contrast head

**Modifications to protocol: CN IV**

- CN IV.c is the only CN to arise along the dorsal aspect of the brainstem
- CN IV is smaller than the other oculomotor CN’s and requires smaller voxels
- CN’s are smaller in pediatric patients and adjustments should be made PRN

**Pediatric Disclaimer:** MR scanning has not been established as safe for imaging fetuses and infants less than two years of age. The responsible physician must evaluate the benefits of the MR examination compared to those of other imaging procedures.

**Cranial Nerve Anatomy**

**Cranial Nerve Segments An Imaging Classification**

- a. nuclear
- b. parenchymal fascicular
- c. cisternal
- d. dural cave
- e. interdural
- f. foraminal
- g. extra-foraminal

(can be referred to in short hand as CN #.x where x is the segment)
Imaging Nuclear (a) and Parenchymal fascicular (b) Segments

- Surrounded by brainstem parenchyma
- Not directly visualized
- The location of the CN.a and CN.b segments is deduced with respect to known anatomic landmarks
- Imaged with standard head MRI (and/or DTI)

Imaging Cisternal (c) and Dural Cave (d) Segments

- Surrounded by cerebral spinal fluid (CSF)
- Well visualized on thin section T2-weighted images
- 3D SSFP or T2 SPACE

CN III.c

CN III.d

CN V.c and CN V.d: Trigeminal
Imaging the Interdural (e) Segment

- Surrounded by venous blood
- Not well visualized on traditional T2-weighted images
- Use contrast enhanced images
- Contrast enhanced SSFP images are ideal

Cavernous sinus

- CN III.e
- CN IV.e
- CN VI.e
- CN V.1.e
- CN V.2.e

(CISS with contrast)

Imaging the Foraminal (f) Segment

- Surrounded by venous blood and bone
- Not well visualized on traditional T2-weighted images
- Again, use contrast enhanced images
- Contrast enhanced SSFP images are ideal

CN III.f

Imaging the Extra-foraminal (g) Segment

- Surrounded by muscle, fat, etc...

V.2.f
Imaging Technique Varies by Segment!

Case 1
CN II.c Pathology

Case 2
CN III.c-e Pathology
Key Points

• The environment of the cranial nerves changes as they extend from the brainstem into the extra-cranial space
• We divide the cranial nerves into segments based on their environment and each segment has different imaging strategies
• Our high resolution 3D skull base protocol with contrast allows for visualization of each segment
• The exam can be tailored by the technologist and takes ~25 minutes

Thank you!

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Companion talk by my colleague Dr. Aygun to follow…

(Contents of this talk are copyright Ari Blitz, MD 2014 unless otherwise noted)
3D High Resolution MRI for Clinical Problem Solving:

Nafi Aygun, MD
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Johns Hopkins University
naygun1@jhmi.edu

3D High Resolution MRI

- Rationale
- Protocol
- Cases
- Pros and Cons

Rationale

- Standard protocols leave many clinically relevant questions unanswered
- Functional techniques inc. PET fall short
- Anatomic imaging needs improvement

Protocol

- Pre contrast VIBE (T1W GRE)
- Post contrast VIBE (fat suppressed)
  - Isotropic voxel size 0.8-1 mm
- Pre and post contrast CISS
  - Isotropic voxel size 0.6 mm
- Axial STIR SPACE
  - Sub milimetric
- Coverage?
- 3T Siemens
Post-contrast CISS

- T1- weighting
- Gadolinium enhancement of tissues and vascular structures
- A different kind of contrast!

CPA MENINGIOMA

Pre
Post

Normal Cavernous Sinus

3rd CN, Superior and inferior Orbital fissures
Soft Tissues of the Neck

Facial Nerve

Cases

SCCa of the Skin
Schwannoma vs. Pleomorphic Adenoma

Pleomorphic Adenoma

Schwannoma (Presumed)

Pleomorphic Adenoma
<table>
<thead>
<tr>
<th>Sinonasal Cancer; Dural Invasion</th>
<th>Sinonasal Cancer; Periorbital Invasion</th>
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<tbody>
<tr>
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<th>Sinonasal Cancer: Posterior Extent</th>
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</tbody>
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Sinonasal Cancer: Skull base, periorbital extent?

Esthesioneuroblastoma

Isolated Right CN III palsy
Isolated Right CN VI palsy

Planum Meningioma

Planum Meningioma: Optic nerve

Optic Glioma vs. Schwannoma
Orbital Nerves

Pituitary adenoma; Optic nerves

Sympathetic ganglion
Schwannoma
Chondrosarcoma
Intradural Extension of Tumor

Chondrosarcoma: Carotid encasement?

Chordoma: VI th. CN and Intradural extension

Pulsatile tinnitus and EAC bleeding
Cellular Myofibroblastic Tumor
Focal Facial nerve invasion

Lesion Vascularity