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Brain Tumor Syllabus

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ASTROCYTOMA

Terminology
- Primary brain tumor of astrocytic origin with intrinsic tendency for malignant progression, degeneration into anaplastic astrocytoma (AA).

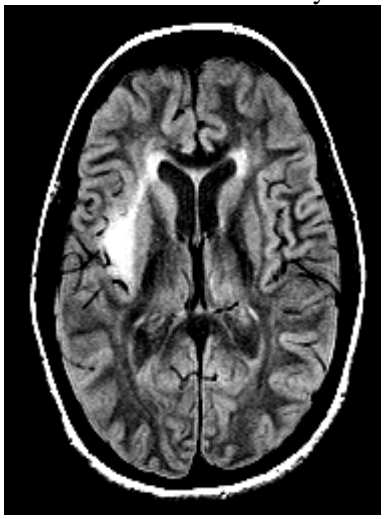
Imaging Findings
- Best diagnostic clue: Focal or diffuse nonenhancing white matter (WM) mass
- Cerebral hemispheres, supratentorial 2/3
- May appear circumscribed on imaging but isn't; tumor cells typically found beyond imaged signal abnormality!

Top Differential Diagnoses
- Anaplastic astrocytoma (AA)
- Ischemia
- Cerebritis

- Oligodendroglioma
- Herpes encephalitis
- Status epilepticus

Pathology
- Represents 25-30% of gliomas in adults
- 10-15% of all astrocytomas
- Diffusely infiltrating mass with blurring of GM/WM interface
- WHO grade II

Clinical Issues
- Majority occur between ages of 20-45 years
- Inherent tendency for malignant progression of AA = major cause of mortality
- Median survival 6-10 years.



PEDIATRIC BRAINSTEM GLIOMA

Terminology
- Tectal glioma (tectal)

- Focal tegmental mesencephalic (FTM)
- Diffuse (intrinsic) pontine glioma (DPG)
- Heterogeneous group of focal or diffuse gliomas involving mesencephalon, pons, or medulla

Imaging Findings

- Classic imaging appearance varies with tumor type and location
- Tectal: Pilocytic, focal, variable enhancement/Ca ++
- FTM: Pilocytic, cyst plus nodule
- DPG: Fibrillary, diffuse, nonenhancing
- All BSG are not equal! Geography predict prognosis
- Borders/margins predictive of prognosis

Top Differential Diagnoses

- Congenital aqueductal stenosis vs tectal glioma
- Alexander disease vs tectal glioma
- Neurofibromatosis type 1 vs DPG
- Other brainstem gliomas

Pathology

- General path comments: No metastases outside CNS
- Epidemiology: 10-20% pediatric brain tumors

Diagnostic Checklist

- Not all expansile brainstem lesions are neoplasms
- Geography predicts prognosis
-

PEARLS IN THE DIAGNOSIS OF BRAIN STEM GLIOMAS

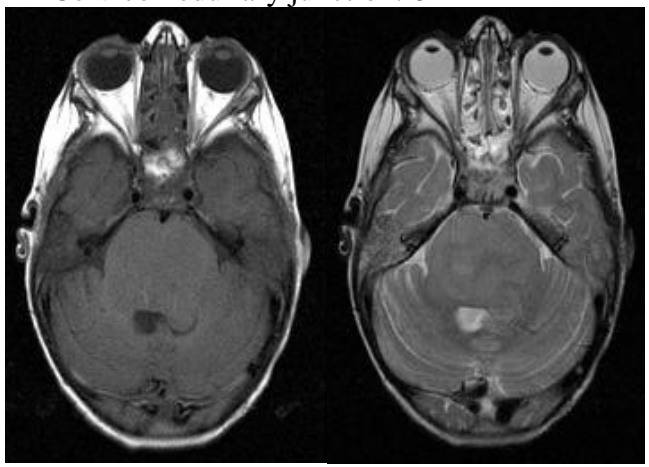
- Peak age: 6-8 years
- Signal: Hyperintense on T2 (95%), exophytic tumor produces brighter signal
- Growth patterns
    a. Cystic tectum (20%)

b. Infiltrative, pons (70%)

- Dedifferentiation of glioblastoma multiforme 5-7% of pediatric cases
- Location
    a. Pontomedullary (80%)
    b. Midbrain (60%)
    c. Cerebellum (40%)
    d. Cervical cord (35%)
    e. Posterior thalamus (30%)
- Enhancement: Variable and may not be present
- Calcification: Less than 4%
- Differential diagnosis
    a. Vascular malformation (flow void or hemorrhage signal, non-expansile)
    b. Metastases (adults with history of primary neoplasm, contrast enhancement)

RANGE OF NORMAL ANTEROPOSTERIOR BRAIN STEM DIAMETERS

- Midbrain tegmentum: 11-15 mm
- Pons: 24-29 mm
- Pontomedullary junction: 14-17 mm
- Cervicomedullary junction: 8-11 mm



ANAPLASTIC ASTROCYTOMA

Terminology
- Diffusely infiltrating astrocytoma with focal or diffuse anaplasia and a marked proliferative potential

Imaging Findings
- Infiltrating mass that predominately involves white matter (WM)
- Variable enhancement, typically none; may be focal or patchy
- Hemispheric WM, frontal & temporal lobes common
- Neoplastic cells almost always found beyond areas of abnormal signal intensity

Top Differential Diagnoses
- Low grade glioma
- Glioblastoma multiforme (GBM)
- Cerebritis
- Ischemia
- Oligodendroglioma
- Status epilepticus
- Herpes encephalitis

Pathology
- AA have histologic and imaging characteristics among spectrum between low grade astrocytoma and GMB
- 1/3 of astrocytomas
- May appear discrete but tumor always infiltrates adjacent brain
- WHO grade III

Clinical Issues
- Median survival 2-3 years
- Commonly arise as recurrence after resection of a grade II tumor

GLIOBLASTOMA MULTIFORME

Terminology
- Rapidly enlarging malignant astrocytic tumor characterized by necrosis and neovascularity
- Most common of all primary intracranial neoplasms

Imaging Findings
- Best diagnostic clue: Thick, irregular-enhancing rind of neoplastic tissue surrounding necrotic core

Top Differential Diagnoses
- Abscess
- Metastasis
- Primary CNS lymphoma
- Anaplastic astrocytoma (AA
- "Tumefactive" demyelination
- Subacute ischemia
- Status epilepticus

Pathology
- Two types, primary (de novo) and secondary (degeneration from lower grade astrocytoma)
- 50-60% of astrocytomas
- WHO grade IV

Clinical Issues(Filippi et al., 1996)
- Age: Peak 45-70 years but may occur at any age
- Relentless progression
- Prognosis is dismal (death in 9-12 months)

Diagnostic Checklist
- Viable tumor extends far beyond signal abnormalities!

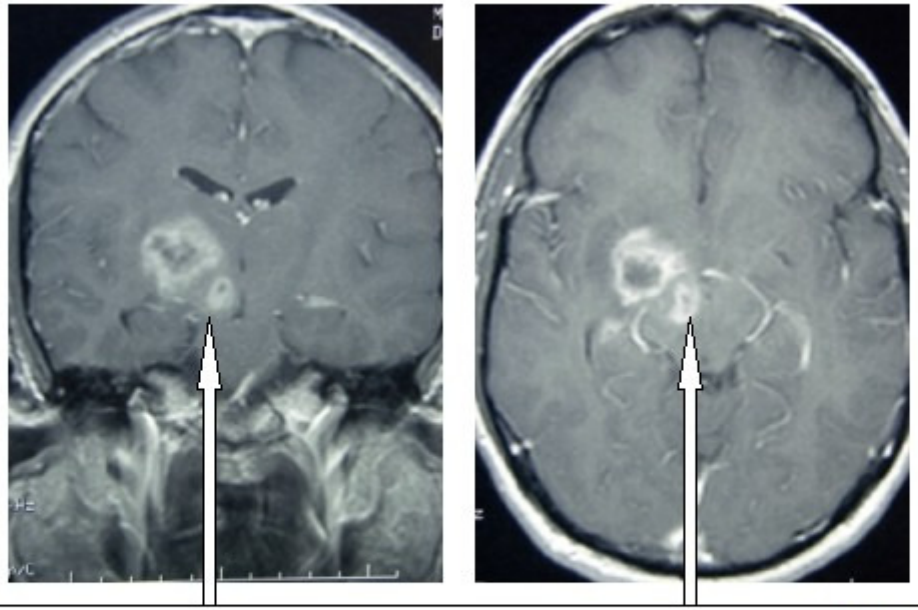


Figure 1A (Left): MRI Scan (Coronal View-Gadolinium enhanced). 45 year old male with a deep seated Right Thalamic region Glioblastoma with direct infiltration of the Brainstem (Arrow).

Figure 1B (Right): MRI Scan (Transaxial View-Gadolinium enhanced) in the same patient as Figures 1A & 2.) Partially Cystic Right Thalamic Region Glioblastoma infiltrates the Brainstem along fibre tracts (Arrow).

GLIOSARCOMA

Terminology

- Rare malignant neoplasm with both glial, mesenchymal elements

Imaging Findings

- Heterogeneously enhancing mass with dural invasion, +/- skull involvement

May be indistinguishable from GBM

Top Differential Diagnoses
- Glioblastoma multiforme (GBM)
- Metastasis
- Abscess
- Hemangiopericytoma
- Malignant meningioma

Clinical Issues
- Poor prognosis, median survival of 6-12 months

GLIOMATOSIS CEREBRI

Terminology
- Diffusely infiltrating glial tumor involving two or more lobes, frequently bilateral
- Infiltrative extent of tumor is out of proportion to histologic and clinical features

Imaging Findings
- Best diagnostic clue: T2 hyperintense infiltrating mass with enlargement of involved structures
- Typically hemispheric white matter involvement, may also involve cortex (19%)
- May cross corpus callosum or massa intermedia
- Morphology: Infiltrates, enlarges yet preserves underlying brain architecture
- Typically no or minimal enhancement
- Marked elevation of myo-inositol (mI)

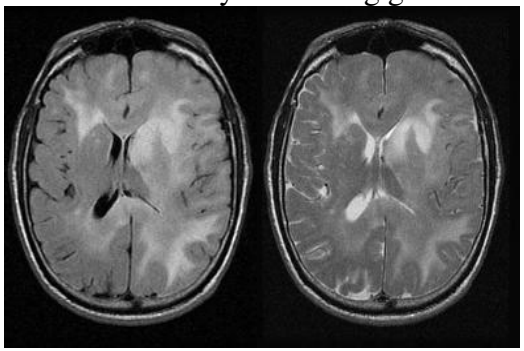
Top Differential Diagnoses
- Arteriolosclerosis
- Anaplastic astrocytoma (AA)
- Viral encephalitis

- Lymphoma

Pathology
- Underlying brain architecture preserved
- Usually WHO grade III

Clinical Issues
- Peak incidence between 40-50 years
- Poor prognosis

Diagnostic Checklist
- Rare diffusely infiltrating glial tumor that can be mistaken for nonneoplastic WM disease



PILOCYTIC ASTROCYTOMA

Terminology
- Pilocytic astrocytoma (PA), juvenile pilocytic astrocytoma (JPA)

Imaging Findings
- Cystic cerebella mass with enhancing mural nodule
- Enlarged optic nerve/chiasm/tract with variable enhancement
- Paradoxical findings: MRS does not accurately reflect historical behavior of tumor
- Multiplanar or 3D volume post contrast imaging key to showing point to origin and degree of extension

Top differential Diagnoses

- Medulloblastoma (PNET-MB)
- Pilomyxoid astrocytoma

Pathology

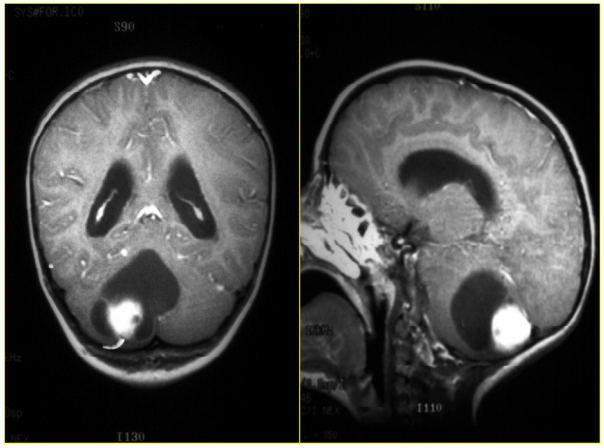
- 15% of NF1 patients develop PAs, most commonly in optic pathway
- Up to 1/3 of patients with optic pathway PAs have NF1
- WHO grade 1

Clinical Issues

- Peak incidence: 5-15 years of age
- Older than children with medulloblastoma

Diagnostic Checklist

- Generally not a reasonable diagnostic consideration in adults
- An enhancing intra-axial tumor with cystic change in a “middle-age” child is more likely to be PA than anything else



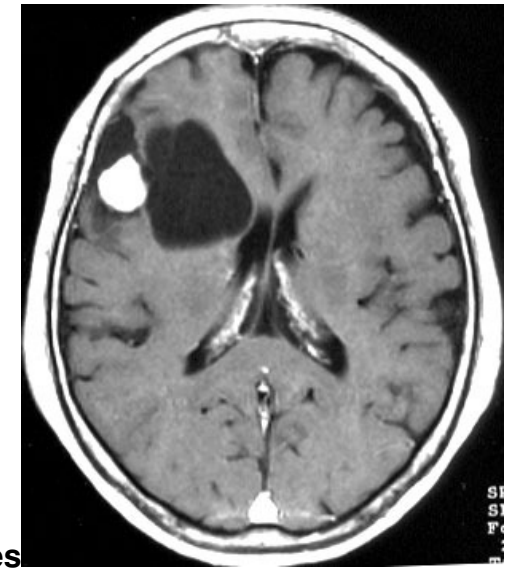
PLEOMORPHIC XANTHOASTROCYTOMA

Terminology

- Distinct type of (usually) benign supratentorial astrocytoma found almost exclusively in young adults

Imaging Findings

- Supratentorial cortical mass with adjacent enhancing dural "tail"
- Temporal lobe most common



Top Differential Diagnoses

- Ganglioglioma
- Pilocytic astrocytoma
- Dysembryoplastic neuroepithelial tumor (DNET)

- Oligodendroglioma
- Meningioma
- Low grade astrocytoma (Grade II)

Pathology
- Superficial, circumscribed astrocytic tumor noted for cellular pleomorphism and xanthomatous change
- < 1% of all astrocytomas
- Cystic mass with mural nodule abutting meninges
- Deep margin may show infiltration of parenchyma
- WHO grade II

Clinical Issues
- Majority with long-standing epilepsy, often partial complex seizures (temporal lobe)
- Tumor of children/young adults

Diagnostic Checklist
- Cortical mass & meningeal thickening in a young adult with long seizure history? Think PXA!

SUBEPENDYMAL GIANT CELL ASTROCYTOMA

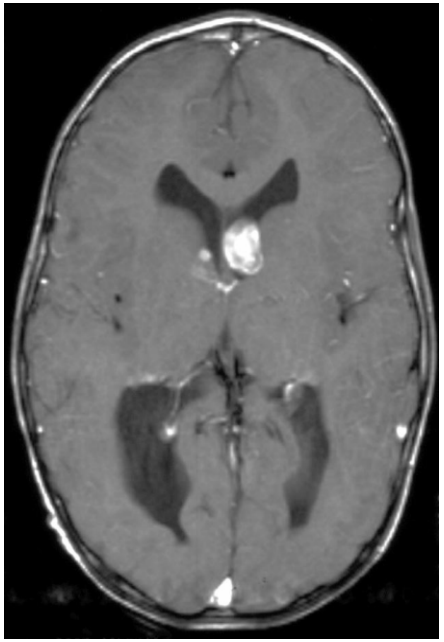
Imaging Findings
- Enlarging, enhancing intraventricular mass in patient with tuberous sclerosis complex (TSC)
- Location: Almost always near foramen of Monro
- Well marginated, often lobulated
- Heterogeneous, strong enhancement
- Presence of interval growth suggests SGCT
- Enhancement alone does not allow discrimination from hamartoma
- FLAIR MR to detect subtle CNS features of TSC
- Recommend brain MR with contrast every 1-2 years for SGCT follow-up

Top Differential Diagnoses
- Choroid plexus tumors
- Astrocytoma
- Germinoma
- Subependymoma

Pathology
- Most common CNS neoplasm in TSC
- Does not seed CSF pathways
- WHO grade I

Clinical Issues
- Increased ICP secondary to tumor obstructing foramen of Monro

Diagnostic Checklist
- SGCA in tuberous sclerosis patient with worsening seizures and/or symptoms of ventricular obstruction



OLIGODENDROGLIOMA

Terminology

- Well-differentiated, slowly growing but diffusely infiltrating cortical/subcortical tumor

Imaging Findings

- Best diagnostic clue: Partially Ca++ subcortical/cortical mass in middle-aged adult
- Typically involves subcortical white matter (WM) and cortex
- Majority supratentorial (85%), hemispheric WM
- Most common site is frontal lobe
- May involve temporal, parietal or occipital lobes
- Morphology: Infiltrative mass that appears well demarcated
- Majority calcify, nodular or clumped Ca++ (70-90%)

Top Differential Diagnoses

- Anaplastic oligodendroglioma (AO)
- Astrocytoma
- Ganglioglioma
- Dysembryoplastic neuroepithelial tumor (DNET)
- Pleomorphic xanthoastrocytoma (PXA)
- Cerebritis
- Ischemia

Pathology

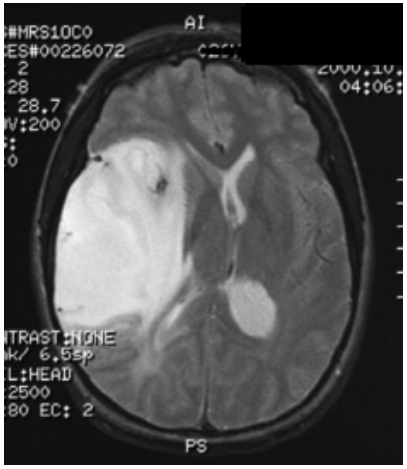
- WHO grade II

Clinical Issues

- Seizures, headaches
- Peak incidence 4^{th} and 5^{th} decades
- Surgical resection in primary treatment

Pearls in the Diagnosis of Oligodendroglioma

- Peak age: 30-50 years
- Calcification: 80%
- Cyst formation: 10%
- Edema: 50%
- Location: frontotemporal centrum
- Hemorrhage: 80-90%
- Margins: poorly defined; therefore, all areas of T2 relaxation prolongation should be radiated
- Enhancement: moderate and inhomogeneous
- Differential Diagnosis:
    - Meningioma (pseudocapsule, extra-axial)
    - Ganglioglioma (males, temporal love and anterior third ventricle predilection)
    - Calcified glioma (less infiltrative, more mass effect, less calcification)
    - Arteriovenous malformation (hypointense flow void signal, T2 dependent hemosiderin)



ANAPLASTIC OLIGODENDROGLIOMA

Terminology

- Oligodendroglioma with focal or diffuse histologic features of malignancy

Imaging Findings

- Best diagnostic clue: Calcified frontal lob mass involving cortex/subcortical white matter (WM)
- May appear discrete, but always infiltrative
- Neoplastic cells almost always found beyond areas of abnormal signal intensity

Top Differential Diagnoses

- Oligodendroglioma
- Anaplastic multiforme (GBM)
- Cerebritis
- Ischemia

Pathology
- Well-differentiated (grade II) and anaplastic (grade III) types of oligodendroglioma
- Oligoastrocytoma (mixed tumor with 2 distinct neoplastic cell types) are common (50%)
- Oligos have better prognosis that astrocytomas of same grade
- Average number of chromosomes involved is higher in grade III than grade II oligos
- 20-50% of oligodendrogliomas are anaplastic
- WHO grade III

Clinical Issues
- Peak incidence fourth through sixth decade
- Median survival 4 years
- Local tumor recurrence common

EPENDYMOMA

Terminology
- Sloe-growing tumor of ependymal cells

Imaging Findings
- Soft or "plastic" tumor: Squeezes out through 4^{th} ventricle foramina into cisterns
- $2/3^{rd}$ infratentorial, 4^{th} ventricle
- $1/3^{rd}$ supratentorial, majority periventricular WM
- Ca++ common (50%); +/- cysts, hemorrhage
- MR spectoroscopy alone does not reliably differentiate ependymoma from astrocytoma or PNET-MB
- High-quality sagittal imaging can distinguish point of origin as floor vs roof of 4^{th} ventricle

Top Differential Diagnoses
- Medulloblastoma (PNET-MB)

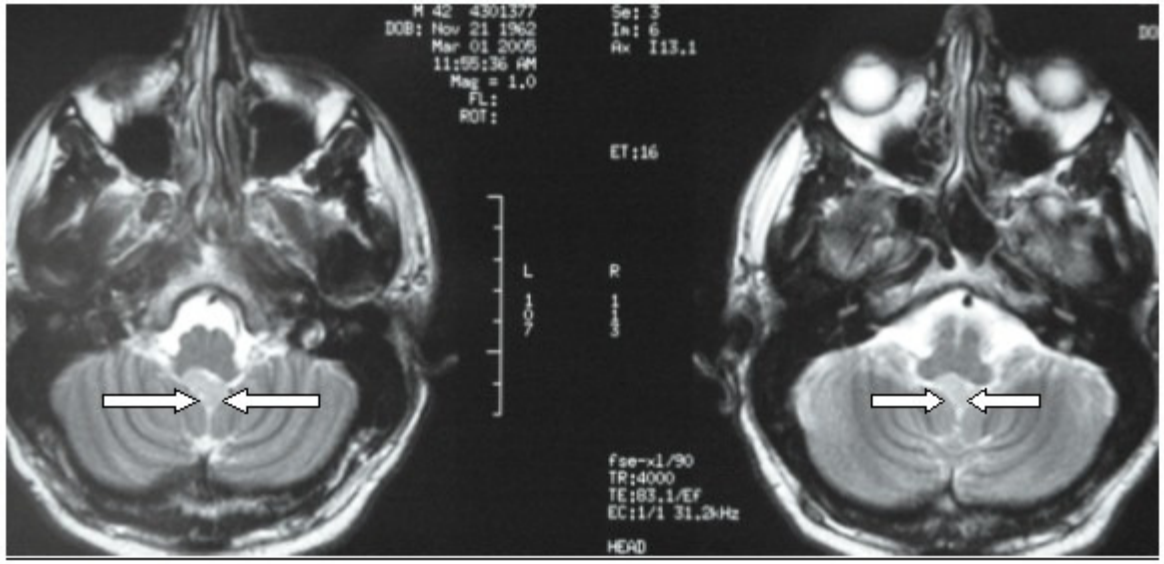
Pathology
- Arise from ependymal cells or ependymal rests
- Third most common posterior fossa tumor in children (after PA and PNET-MB)
- WHO grade II (low grade, well-differentiated)
- WHO grade III (high grade, anaplastic)

Clinical Issues
- Clinical profile: 1-5 yo with headache, vomiting
- Gross total resection + XRT correlates with improved survival

Diagnostic Checklist
- Indistinct interface with floor of 4^{th} ventricle = ependymoma

Pearls in the Diagnosis of Ependymoma
- Tendency to involve the filum
- Hemorrhagic in a significant percentage of the cases, especially the myxopaillary form of the filum
- Tendency to form cysts
    a. Marginal cysts located at the tumor edge and easily amenable to syringotomy decompression
    b. Central cysts which are located within the lesion and must be excised if they are to be treated
- Ependymomas tend to have hypointense T1 and hyperintense T2 signal and either show cyst formation or signal inhomogeneity
- Filum location is a strong tip-off to the diagnosis (these lesions tend to seed the CSF and may be a result of drop metastases)
- Enhancement is moderate to marked

- Indistinct interface with roof of 4^{th} ventricle = PNET-MB



MRI Scan (Transaxial View-same patient as Figures 1, 4 & 5) 4th Ventricular Ependymoma. The tumor (centrally located lighter "grey" mass) fills the 4th Ventricle in these images (Arrows)

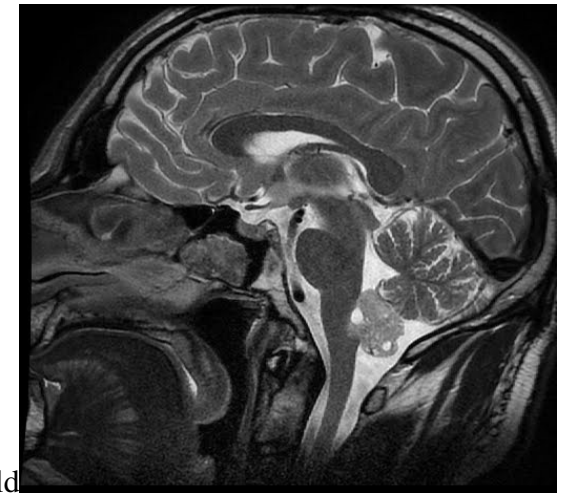
SUBEPENDYMOMA

Terminology

- Rare, benign well-differentiated intraventricular ependymal tumor

Imaging Findings

- Best diagnostic clue: T2 hyperintense lobular, nonenhancing intraventricular mass
- Intraventricular, inferior 4th ventricle typical (60%)
- Other: Lateral > 3rd ventricle > spinal cord
- Well-defined solid lobular mass
- When large, may see cysts, hemorrhage, Ca++



- Variable enhancement, typically none to mild

Top Differential Diagnoses

- Ependymoma
- Central neurocytoma
- Subependymal giant cell astrocytoma
- Choroid plexus papilloma (CPP)
- Hemangioblastoma
- Metastases

Pathology

- WHO grade I

Clinical Issues

- Most asymptomatic
- Other signs/symptoms: Related to increased intracranial pressure, hydrocephalus
- Middle-aged/elderly adult, (typically 5^{th}-6^{th} decades)
- Surgical resection is curative in most cases

Diagnostic Checklist

- 4th or lateral venricular hyperintense mass in an elderly male? Think sybependymoma!

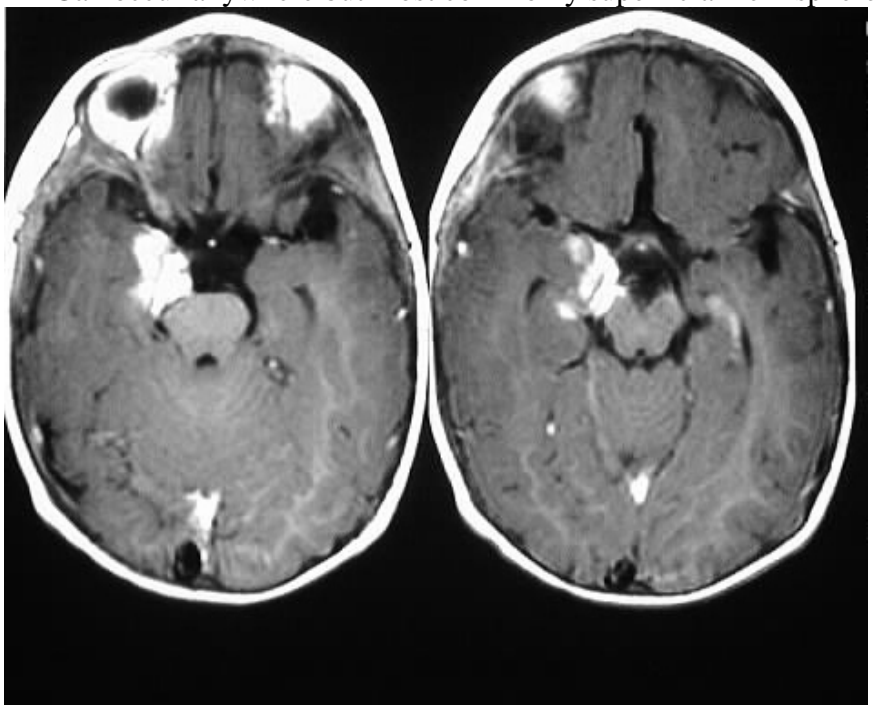
GANGLIOGLIOMA

Terminology

- Well differentiated, slowly growing neuroepithelial tumor composed of neoplastic ganglion cells and neoplastic glial cells

Imaging Findings

- Best diagnostic clue: Partially cystic, enhancing, cortically-based mass in child/young adult with TLE
- Can occur anywhere but most commonly superficial hemispheres, temporal lobe



Top Differential Diagnoses

- Pleomorphic xanthoastrocytoma (PXA)

- Dysembryoplastic neuroepithelial tumor (DNET)
- Pilocytic astrocytoma
- Low grade astrocytome (grade II)
- Oligodendroglioma

Pathology
- Cortical dysplasia is commonly associated
- Most common mixed neuronal-glial tumor
- WHO grade I and II

Clinical Issues
- Clinical profile: Most common neoplasm causing chronic temporal lobe epilepsy
- Excellent prognosis if surgical resection complete
- Malignant degeneration is rare, approximately 5-10% (glial component)

Diagnostic Checklist
- In young patient with history of temporal lobe epilepsy, think ganglioglioma
- Cyst with an enhancing mural nodule is classic, but nonspecific for ganglioglioma

Pearls in the Diagnosis of Ganglioglioma
- Age: 15-30 years
- Gender: slight male predominance
- Location: temporal lobe or anterosuperior third ventricle
- Signal: nonspecific hypointense T1 and hyperintense T2
- Enhancement: variable and nominal to moderate
- Cyst formation: 10%
- Hemorrhage: rare
- Calcification: 10-20%
- Insidious seizure history and lesion in the correct location are tipoffs to diagnosis
- Differential diagnosis:
    - Oligodendroglioma: more edema

- Vascular malformation: less mass effect, no edema, flow void, hemosiderin
- Colloid cyst: smaller size, hyperintense T1

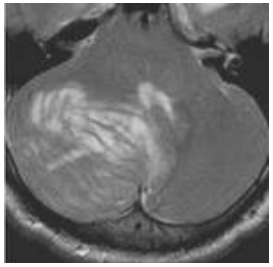
DYSPLASTIC CEREBELLAR GANGLIOCYTOMA

Terminology

- Best known as Lhermitte-Duclos disease (LDD)

Imaging Findings

- Best diagnostic clue: Widened cerebellar folia with striated appearance on MR
- Characteristics "layered" or striated" pattern of alternating isointense and hyperintense signal
- Also called " laminated", "corduroy", "lamellar", "folial"
- No diffusion disturbance on ADC maps
- T1 C+: Rare lesions enhance
- PET: Elevated 18-FDG uptake
- Elevated 201-thallium uptake on delayed imaging



Top Differential Diagnoses

- Cerebellar infarction
- Acute cerebellitis
- Leptomeningeal

Pathology

- Associated with Cowden syndrome
- Characterized by development of multiple hamartomas

- Increased risk of thyroid and breast carcinoma
- Some evidence supports that all cases of LDD have Cowden syndrome
- Replacement and expansion of granular layer by large neurons

Diagnostic Checklist
- Striated cerebellar hemisphere is "Aunt Minnie" for LDD

DESMOPLASTIC INFANTILE GANGLIOGLIOMA

Terminology
- Desmoplastic infantile (DIG, DIGG) or desmoplastic infantile astrocytoma (DIA)

Imaging Findings
- Large cyst + cortical-based enhancing tumor nodule
- Location: frontal > parietal > temporal
- Solid tumor module(s) enhance markedly
- Enhancement of leptomeninges, dura adjacent to solid tumor is typical

Top Differential Diagnosis
- Primitive neuroectodermal tumor (PNET)
- Supratentorial ependymoma
- Pleomorphic xanthoastrocytoma (PXA)
- Hemangioblastoma
- Ganglioglioma
- Pilocytic astrocytoma

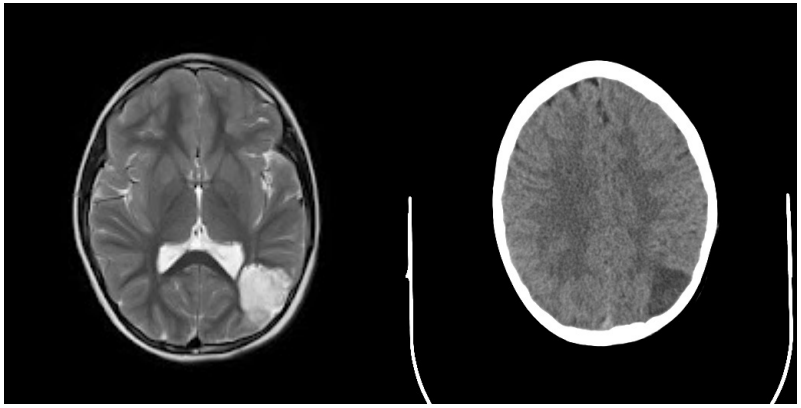
DNET

Terminology

- Dysembryoplastic neuroepithelial tumor (DNET)
- Benign, focal, intracortical mass superimposed on background of cortical dysplasia

Imaging Findings

- Best diagnostic clue: Well-demarcated, wedge-shaped "bubbly" intracortical mass in young patient with longstanding partial seizures
- Temporal lobe (often amygdale/hippocampus) most common site
- Intracortical mass scallops inner table of skull and "points" towards ventricle
- Minimal or no mass effect



Pathology

- Approximately 1-2% of primary brain tumors in patients < 20 years
- Reported in 5-80% of epilepsy specimens

Clinical Issues

- Clinical profile: Longstanding (difficult to control) partial complex seizures in child or young adult
- NO or very slow increase in size over time
- Rare recurrence
- Beware of atypical features (enhancement) on pre-op imaging

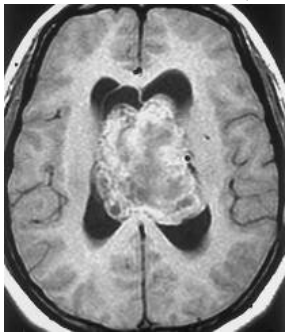
CENTRAL NEUROCYTOMA

Terminology

- Intraventricular neuroepithelial tumor with neuronal differentiation

Imaging Findings

- Best diagnostic clue: "Bubbly" mass in frontal horn or body of lateral ventricle
- Intraventicular mass attached to septum pellucidum
- Circumscribed, lobulated mass with intratumoral "cysts"
- Ca++ common, 50-70%



Top Differential Diagnosis

- Subependymoma
- Subependymal giant cell astrocytoma (SGCA)
- Metastasis
- Ependymoma
- Choroid plexus papilloma (CPP)
- Meningioma
- Cavernous malformation

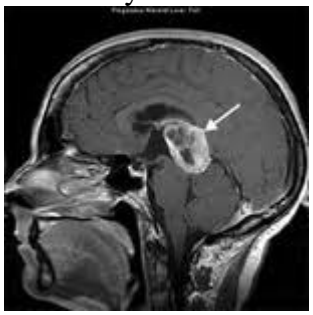
Pathology
- < 1% of all primary intracranial neoplasms
- Represents 50% of intraventricular tumors on patients 20-40 years
- Resembles oligodendroglioma
- WHO grade II

Clinical Issues
- Most common signs/symptoms: Headaches, increased intracranial pressure, mental status changes, seizure
- Hydrocephalus secondary to forearm of Monro obstruction
- Complete surgical resection is treatment of choice

PINEOBLASTOMA

Terminology
- Highly malignant, primitive embryonal tumor of pineal gland

Imaging Findings
- Large, heterogeneous pineal mass with "exploded", peripheral Ca++
- Nearly 100% with obstructive hydrocephalus



Top Differential Diagnoses
- Germ cell tumors (GCTs)
- Meningioma
- Pineocytoma (PC)
- Metastases

Pathology
- PBs exhibit little to no differentiation, similar to other PNETs

Clinical Issues
- Elevated ICP (hydrocephalus): Headache, nausea, vomiting, lethargy, papilledema, abducens nerve palsy

Diagnostic Checklist
- Both PBs and germinomas frequently hyperdense on CT (hypointense T2WI) and prone to CSF dissemination
- Peripheral "exploded" Ca++ in PB and central "engulfed" Ca++ in germinoma classic but not always identified

PINEOCYTOMAS

Terminology
- Slow-growing pineal parenchymal tumor of young adults composed of small, uniform mature cells resembling pineocytes

Imaging Findings
- Enhancing, circumscribed pineal mass which "explodes" pineal Ca++
- May mimic pineal cyst or pineoblastoma

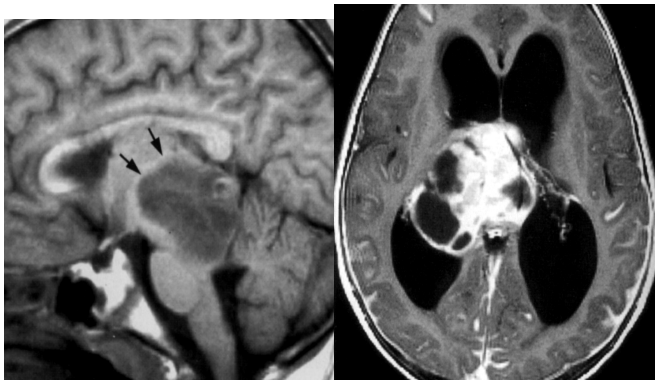
Top Differential Diagnosis
- Pineoblastoma
- Nonneoplastic pineal cyst
- Astrocytoma
- Other germ cell tumors (GCT)

- Meningioma

Pathology
- Pineocytoma and pineoblastomas account for 15% of pineal region neoplasms
- Pineocytomas represents approximately 45% of pineal parenchymal tumors
- Pineal parenchymal tumors << germinoma
- Cysts and small areas of hemorrhage may be seen
- May compress but do not invade adjacent structures
- WHO grade II

Clinical Issues
- Overall 5 year survival 86%

Diagnostic Checklist
- PCs “explode” gland Ca++ while germinomas “engulf” gland Ca++
- Imaging of pineocytoma may be nonspecific



MEDULLOBLASTOMA (PNET-MB)

Terminology
- Medulloblastoma (MB), posterior fossa PNET, PNET-MB
- Malignant, invasive, highly cellular embryonal tumor

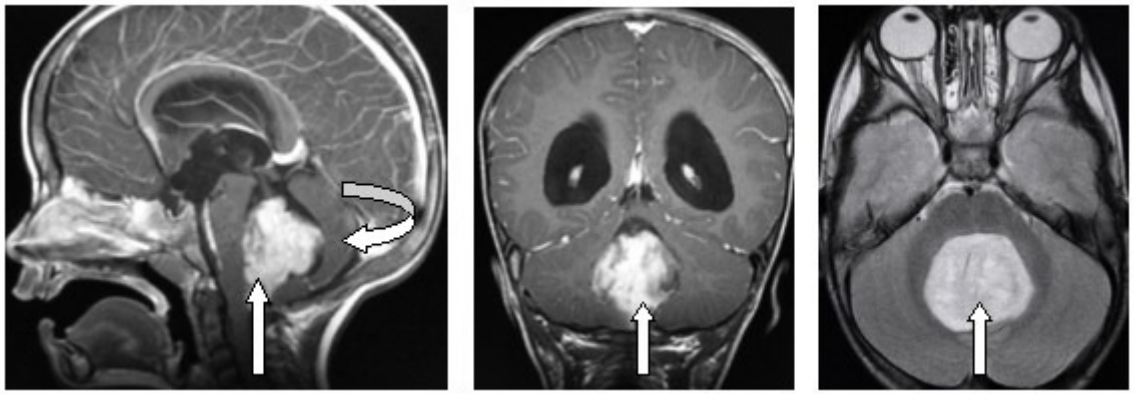
Imaging Findings
- Solid mass in 4^{th} ventricle
- Hydrocephalus common (95%)
- > 90% enhance
- Contrast essential to detect CSF dissemination
- Contrast-enhanced MR of spine (entire neuraxis)

Top Differential Findings
- Cerebellar pilocytic astrocytoma (PA)
- Ependymoma
- Choroid plexus papilloma (CPP)
- Atypical teratoid/rhabdoid tumor (AT/RhT)

Pathology
- 15-20% of all pediatric brain tumors
- WHO grade IV

Clinical Issues
- Ataxia, signs of increased intracranial pressure
- Relatively short (< 1 month) of symptoms
- Rapid growth with early subarachnoid spread
- "Standard risk" clinical profile
- "High risk" clinical profile

Diagnostic Checklist
- Remember AT/RhT in patients under 3 years

- 4th V tumor arising from roof = PNET-MB
- 4th V tumor arising from floor = ependymoma
- 

(Left): MRI Scan (Sagittal View-Gadolinium Enhanced) of a Young child with a large 4th Ventricular Medulloblastoma (Vertical Arrow) with compression of the Cerebellum (Curved Arrow).

Figure 1B (Center): MRI Scan (Coronal View-same patient). The tumor has considerable "enhancement" indicating a substantial blood supply.

Figure 1C (Right): MRI Scan (Transaxial View-same patient). The tumor (Arrow) fills the 4th Ventricle demonstrates extensive "enhancement".

Distinctions among medulloblastoma, ependymoma, and astrocytoma in posterior fossa

Feature	*Medulloblastoma*	*Ependymoma*	*Astrocytoma*

Unenhanced CT	Hyperdense	Isodense	Hypodense
Enhancement	Moderate	Minimal	Nodule enhances, cyst does not
Calcification	Uncommon (10%-21%)	Common (40%-50%)	Uncommon (< 10%)
Origin	Vermis	4th ventricle ependyma	Hemispheric
T2WI	Intermediate	Intermediate	Bright
Site	Midline	Midline	Eccentric
Subarachnoid seeding	15%-50%	Uncommon	Rare
Age (yr)	5-12	2-10	10-20
Cyst formation	10-20%	15%	60-80%
Foraminal spread	No	Yes (Luschka, Magendie)	No
Hemorrhage	Rare	10%	Rare
MRS			
Metabolite			
NAA	Low	Intermediate	Intermediate
Lactate	Absent	Often present	Often present
Choline	High	Less elevated	High

SUPRATENTORIAL PNET

Terminology

- Supratentorial primitive neuroectodermal tumor (S-PNET)
- Cerebral embryonal tumor composed of undifferentiated neuroepithelial cells

Imaging Findings

- Best diagnostic clue: Large, complex hemispheric mass with minimal peritumoral edema
- Isoattenuating to hyperattenuating
- Calcification (50-70%)
- Hemorrhage and necrosis common
- Heterogeneous enhancement

- DWI: Restricted diffusion common
- Best imaging tool: Enhanced MR of brain and spine
- Adding post-enhanced FLAIR aids in detecting leptomeningeal metastases

Top Differential Diagnosis

- Astrocytoma
- Ependymoma
- Oligodendroglioma
- Atypical teratoid/rhabdoid tumor

Top Differential Diagnosis

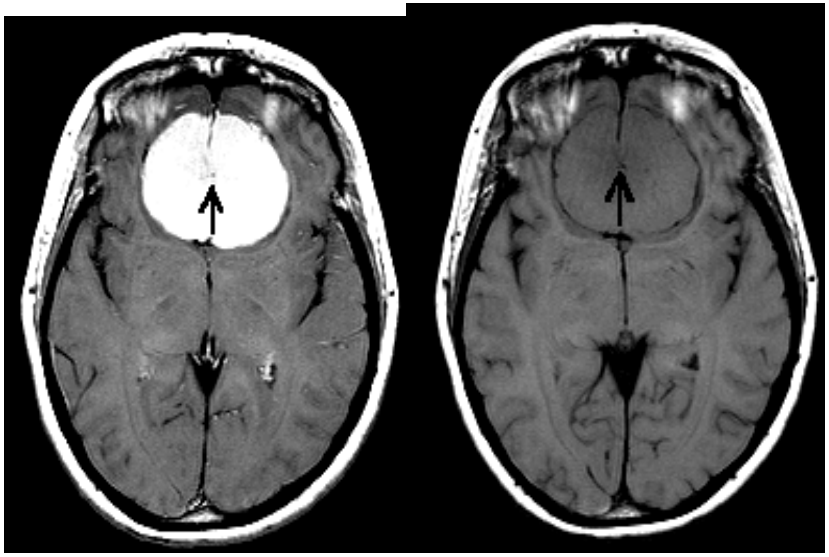
- Astrocytoma
- Ependymoma
- Oligodendroglioma
- Atypical teratoid/rhabdoid tumor

Pathology

- WHO grade IV

Clinical Issues

- Vary with site of origin and size of tumor
- Hemispheric – seizures, disturbed consciousness, motor deficit, elevated ICP
- Suprasellar – visual disturbance, endocrine problems
- Clinical profile: Infant presenting with microcephaly, seizures and large hemispheric mass
- S-PNET → 30-35% 5 year survival



MENINGIOMA

Imaging Findings

- Best diagnostic clue: Dural-based enhancing mass w/cortical buckling & trapped CSF clefts/cortical vessels
- Hyperostosis, irregular cortex, tumoral calcifications, increased vascular markings
- Brain cysts & trapped pools of CSF common
- Peritumoral hypodense vasogenic edema (60%)
- > 95% enhance homogeneously & intensely
- Elevated levels of Alanine at short TE
- DSA: "Mother-in-law" sign → comes early, stays late
- Best imaging tool: MRI + contrast

Top Differential Diagnoses

- Dural metastasis

- Granuloma (sarcoid, TB)
- Idiopathic hypertrophic pachymeningitis
- Extramedullary hematopoiesis

Pathology

- Loss of one copy of chromosome 22 is most prevalent chromosomal change in meningioma
- Arise from arachnoid meningothelial ("cap") cells
- Most common adult intracranial tumor (13-20%)

Clinical Issues

- < 10% of all meningiomas ever cause symptoms
- Age: Middle decade of life
- Gender: M:F ranges 1: 1.5 to 1:3
- Ethnicity: More common in African-Americans
- Generally grow slowly, compress adjacent structures
- Asymptomatic followed with serial imaging

Diagnostic Checklist

- Preoperatively define ENTIRE tumor extent

Signs of Meningioma

- Iso- or Hypointensity on T2 images
- Inward white matter buckling
- Speckled T2 signal hyperintensity (microcyst formation)
- Focal T2 hypointensity (macrocalcifications)
- Prominent circumferential flow void (hypervascularity)
- Cisternal widening
- Calvarial hypointense T1 and T2 (sclerosis) or hyperintense T2 (edema or hyperemia)
- Pseudocapsule formation due to desmoplasia or fibrosis, displaced dura, CSF, and vessels
- Variable edema (edema can be pronounced)
- En plaque variant, ridge
  b. Wraps around structures especially basilar skull vessels

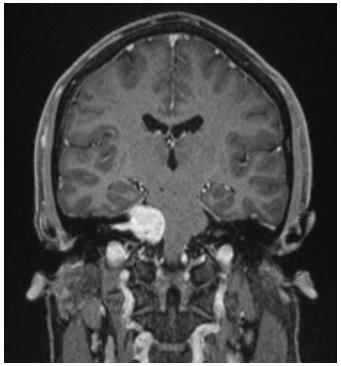
c. Aggressive
d. Propensity for sphenoidal ridge
e. Fibrovascular histology more common

SCHWANNOMA

Terminology

- Benign encapsulated nerve sheath tumor composed of differentiated neoplastic schwann cells

Imaging Findings

- Best diagnostic clue: VS looks like “ice cream on cone”; parenchymal looks like cyst with nodule
- All cranial nerves (exceptions: Olfactory, optic nerves) have myelinated schwann cell sheaths and are sites for intracranial schwannomas
- 1-2% intracerebral
- 

Top Differential Prognosis

- Pleomorphic xanthoastrocytoma (PXA)
- Pilocytic astrocytoma
- Ganglioglioma
- Metastasis
- Hemangioblastoma

Pathology

- Schwannomas = 5=8% of all intracranial neoplasms
- Two types of tissue (Antoni A, B)

Clinical Issues

- Age: 70% of parenchymal schwannomas present before age of 30
- Slowly growing: recurrence after surgery < 10%
- Malignant degeneration exceptionally rare

Diagnostic Checklist

- Cystic, calcified, enhancing hemispheric parenchymal mass in a young patient isn't necessarily a glioma!

Differential diagnosis of meningioma versus schwannoma

Feature	***Meningioma***	***Schwannoma***
Dural tail	Frequent	Extremely rare
Boney reaction	Osteolysis or hyperostosis	Rare
Angle made wit dura	Obtuse	Acute
Calcification	20%	Extremely rare
Cyst/necrosis	Rare	Up to 10%
Enhancement	Uniform	Inhomogeneous in 32%
Extension into the internal auditory canal	Rare	80%
MRS	Alanine	Taurine, GABA
Precontrast CT attenuation	Hyperdense	Isodense
Hemorrhage	Rar	Somewhat more common

NEUROFIBROMA

Terminology
- Plexiform NF (PNF) = infiltrative extrapeural tumor typically associated with neurofibromatosis 1 (NF1)

Imaging Findings
- Best diagnostic clue: "Worm-like" soft tissue mass infiltrative scalp, orbit or parotid in patient with NF1

Top Differential Diagnoses
- Schwannoma
- Malignant peripheral nerve sheath tumor (MPNST)
- Vascular malformation of scalp
- Sarcoma or lymphoma of skull/scalp
- Metastasis
- Chronic interstitial demyelinating polyneuropathy (CIDP)

Diagnostic Checklist
- Look for other stigmata of NF1 (café-au-lait spots, axillary freckling, Lisch nodules, etc)

PEARLS IN THE DIAGNOSIS OF GLOMUS TUMOR
- Age: 30-50 years
- Signal
    a. Intermediate T1, hyperintense T2
    b. Punctuate speckled salt and pepper areas pinpoint flow void due to hypervascularity
- Location
    a. Jugular foramen (50-60%)= glomus jugulare
    b. Tympanic branch or Jacobson's nerve, glomus tympanicum (20-30%)
    c. Auricular branch or Arnold's nerve, glomus vagale (10%)
    d. Periganglia cells of the carotid bifurcation= glomus caroticum (less than 10%)Female predilection

- Multiplicity (10%) particularly with multiple endocrine adenomatosis (3%)
- Eight percent harbor other neoplasms
- Most common primary middle ear neoplasm
- Second most common temporal bone neoplasm
- Enhancement: pronounced, early, mottled
- Associated thrombosis or flow phenomenon in the jugular vein
- Shape: Triangular shape in the coronal projection
- Differential diagnosis
    a. Neuroma: No punctate flow void, more rounded shape
    b. Meningioma: Flat dural margin, homogeneous enhancement, wraparound vessels with infiltrative growth pattern
    c. Normal slow flow in jugular vein

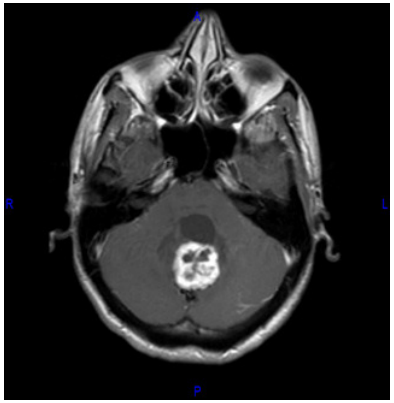
HEMANGIOBLASTOMA

Terminology

- HGBL currently classified as meningeal tumor of uncertain histogenesis (WHO, 2000)

Imaging Findings

- Best diagnostic clue: Adult with intra-axial posterior fossa mass with cyst, enhancing mural nodule abutting pia
- 90-95% posterior fossa
- Size: Size varies from tiny to several cms
- 60% cyst + "mural" nodule
- 40% solid

- 

Top Differential Diagnoses

- von Hippel-Lindau syndrome (VHL)
- Metastasis
- Astrocytoma
- Vascular neurocutaneous syndrome
- Cavernous malformation (CM)
- Clear cell ependymoma

Pathology

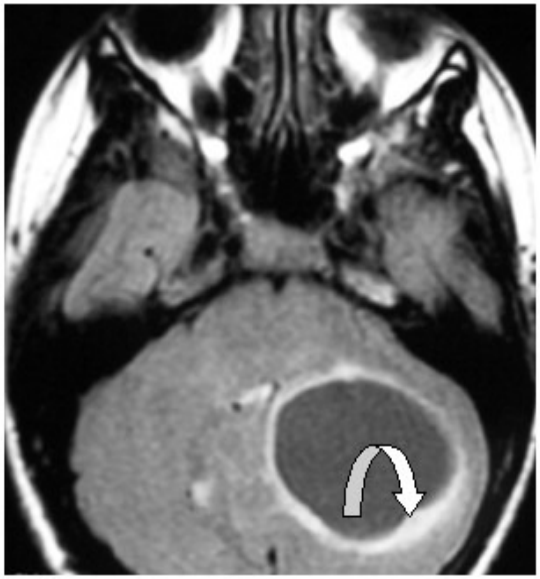
- VHL phenotypes (based on presence, absence of pheochromocytoma and renal cell carcinoma)
- 1-2% of primary intracranial tumors
- 7-100% of posterior fossa tumors
- Secondary polycythemia (may elaborate erythropoietin)
- WHO grade I

Diagnostic Checklist

- Screen entire neuraxis for other HGBLs
- Most common posterior fossa intra-axial mass in middle-aged/older adult = metastasis, not HGBL!

Pearls in the Diagnosis of Hemangioblastoma

- Sixty percent are intramedullary; 40% are extramedullary but intradural
- Thirty percent have Hippel-Lindau disease
- Thoracic location in 50% and cervical location in 40%
- Lesions are single in 80% and multiple in 20%
- Cyst formation in 40%
- MR findings
    a. Intermediate signal intensity nodule which enhances
    b. Speckled foci or flow void signal in and about the nodule
    c. Areas of marginal cyst formation above and below the lesion
    d. Round or ovoid shape



MRI Scan (Gadolinium Enhanced Axial View) Left Cerebellar Hemisphere Cystic Tumor.

The "Mural Nodule" (Curved Arrow) "enhances" due to its considerable blood supply and is responsible for the formation of the fluid of the cyst. The "Nodule" must be entirely removed in order to "cure" this lesion, particularly in "Sporadically" occurring cases.

Cysts of this size cause considerable pressure on the Brain Stem which can threaten the patient's life if left untreated.

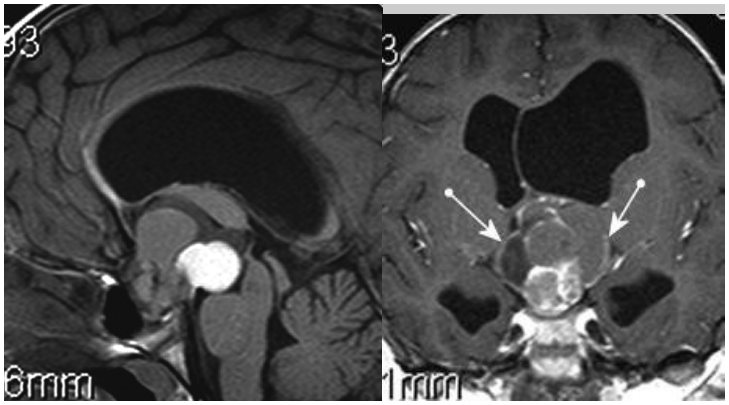
HEMANGIOPERICYTOMA

Terminology

- Sarcoma related to neoplastic transformation of pericytes, contractile cells about capillaries

Imaging Findings

- Lobular enhancing extra-axial mass with dural attachment, +/- skull erosion
- May mimic meningioma, but without Ca++ or hyperostosis
- Typically involve falx, tentorium, or dural sinuses
- Marked enhancement, often heterogeneous



Top Differential Diagnoses

- Meningioma
- Dural metastases
- Lymphoma
- Neurosarcoidosis
- Gliosarcoma
- Solitary fibrous tumor

Pathology
- HPC is a distinctive mesenchymal neoplasm unrelated to meningioma
- Represents < 1% of primary CNS tumors
- Represents 2-4% of all meningeal tumors
- WHO grade II or III (anaplastic)

Clinical Issues
- Most common 4^{th}-6^{th} decade, mean age 43 years
- Extracranial metastases common, up to 30%

Diagnostic Checklist
- When a "meningioma" has atypical features (frank bone erosion, multiple flow voids) think HPC!

PEARLS IN THE DIAGNOSIS OF CRANIOPHARYNGIOMA

- Peak age: Biphasic, 3-5 years and 50-60 years
- Location
    a. Suprasellar (80%)
    b. Sellar/suprasellar (15%)
    c. Sellar (4%)
    d. Third ventricle (0.5%)
    e. Nasopharynx (0.5%)

- Signal
    a. Hyperintense T1 & T2: Hydrolyzed cholesterol and/or blood (65%)
    b. Intermediate T1, hyperintense T2: Keratin (20%)
    c. Hypointense T2: Calcification (75%)
    d. Hypointense T1, hyperintense T2: Cyst formation (40%)
    e. Intermediate T1 and T2: Solid tumor (15%)
- Calcification (75%): Twice as frequent in children as adults (Rathke's cyst do not calcify)

- Origin: Rathke's cells or pouch having multilayered complex epithelium (Rathke's cysts have a simple single-layereed epithelium
- Enhancement: Variable, none to mild
- Hemorrhage: (10%)
- Differential diagnosis: Meningioma (homogenous, marked enhancement); aneurysm (flow void or clot); pituitary adenoma (more homogeneous signal, noncalcified, usually no hyperintense T1 signal)

PRIMARY CNS LYMPHOMA

Terminology
- Malignant primary CNS neoplasm composed of B lymphocytes

Imaging Findings
- Best diagnostic clue: Enhancing lesion(s) within basal ganglia, periventricular WM
- 90% supratentorial
- Deep gray nuclei commonly affected
- Often involve, cross corpus callosum
- Frequently abut, extend along epenymal surfaces

Top Differential Diagnoses
- Toxoplasmosis
- Glioblastoma multiforme (GBM)
- Abscess
- Progressive multifocal leukoencephalopathy (PML)

Pathology
- 98% B cell, non-Hodgkin lymphoma (NHL)
- Incidence increasing in immunocompetent, immunocompromised
- 1-7% of primary brain tumors, incidence rising
- Represents approximately 1% of lymphomas

Clinical Issues
- Median survival 17-45 months

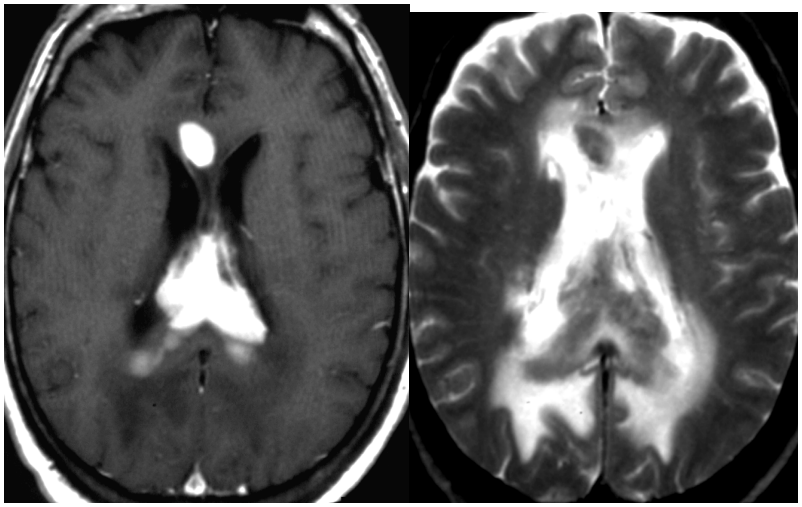
- AIDS median survival 2-6 months
- Stereotactic biopsy, followed by radiation therapy and chemotherapy

Diagnostic Checklist

- Imaging and prognosis varies with immune status
- Periventricular location and Subependymal involvement is characteristic of PCNSL
-

PEARLS IN THE DIAGNOSIS OF PRIMARY & SECONDARY BRAIN LYMPHOMA

- Location
    a. Deep white gray matter (70%)
    b. Leptomeningeal signal: Intermediate T1 and isointense to hyperintense T2 (30%)
- Enhancement: Marked
- Margin: Sharply marginated intra-axial lesions which may be large, and round or oval in shape
- Edema: Mild to moderate
- Cyst formation: Extremely rare
- Hemorrhage
- Calcification: Rare
- Multiplicity: Extremely common
- Tipoff: Clinical history of immunosuppression or AIDS



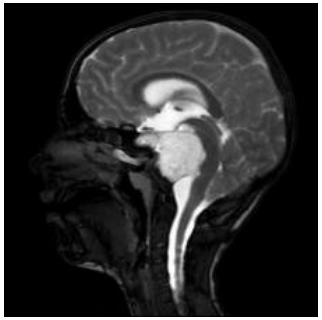
CHORDOMA

Terminology

- Malignant tumor arising from notochord remnants

Imaging Findings

- Mass is hyperintense to discs on T2WI, with multiple septa
- Histologic identification of physaliphorous cell confirms diagnosis
- Location: Sacrococcygeal > spheno-occipital >> vertebral body
- Size: Several cm at presentation
- Morphology: Midline lobular soft tissue mass with osseous destruction
- May extend into disc, involve 2 or more adjacent vertebrae
- May extend into epidural/perivertebral space, compress cord
- May extend along nerve roots, enlarge neural foramina
- Amorphous intratumoral Ca++



Pathology

- 3 types described
- Typical: Lobules, sheets, and cords of clear cells with intracytoplasmic vacuoles (physaliphorous cells); abundant mucin
- Chondroid: Hyaline cartilage (usually spheno-occipital region)
- Dedifferentiated: Sarcomatous elements (rare, highly malignant)

Pearls in the Diagnosis of Chordoma

- Age: 30-40 years
- Location:
    - Sacrum 50%
    - Clivus 30%
    - Cervical (C2) 20%
- Intermediate T1 signal, mottles hyperintense T2 signal
- Calcification 30-50%
- Enhancement: none to mild
- Shape: cauliflower-like shape in the C2 and clival regions
- Tipoffs: cauliflower-shaped or exophytic mass involving intervertebral disc (sacrum) or straddling the clivus or C2 anteriorly and posteriorly in the sagittal projection

PARENCHYMAL METASTASES

Terminology
- Parenchymal tumors that originate from, but are discontinuous with, other CNS primary or extracranial systemic neoplasms

Imaging Findings
- Best diagnostic clue: Discrete parenchymal mass(es) at gray-white interface
- Best imaging tool: Contrast-enhanced MRI >> CECT
- Protocol advice: Double or triple-contrast dose increases sensitivity but questionable value on routine basis

Top Differential Diagnoses
- Abscess
- Malignant glioma
- Thromboembolic stroke(s)
- Demyelinating disease

Pathology
- Prevalence of metastases vs primary CNS neoplasms increasing
- Now account for up to 50% of all brain tumors
- Seen in 25% of cancer patients at autopsy
- Metastases usually displace rather than infiltrate tissue

Clinical Issues
- Median survival with whole brain XRT = 3-6 months

Diagnostic Checklist
- White matter disease ("UBOs") in elderly patient can be caused by multifocal metastases
- Use contrast-enhanced scans

PEARLS IN THE DIAGNOSIS OF METASTATIC DISEASE

Hemorrhagic Metastases
- Choriocarcinoma: 90-95%
- Melanoma: 85%

- Hypernephroma: 65%
- Thyroid carcinoma: 55%
- Lung carcinoma: 15%
- Breast carcinoma: 10%
- Alimentary tract carcinoma: 5-10%
- Other: Less than 5%

Subtypes with a Lower Propensity toward Brain Metastases

- Squamous cell carcinoma
- Sarcoma

Primary Brain Tumors Which May Metastasize Peripherally

- Medulloblastoma
- Cerebellar sarcoma
- Glioblastoma multiforme

Subependymal or Intraventricular Tumor Spread

- Melanoma
- Lymphoma
- Breast carcinoma
- Lung carcinoma

Leptomeningeal or Dural Carcinomatosis

- Breast carcinoma
- Lung carcinoma (adenocarcinoma)
- Melanoma
- Lymphoma

Brain Metastases without Edema

- Squamous cell carcinoma

Cystic Metastases
- Lung carcinoma (oat cell)
- Radiated metastases

Isointense Metastases
- Metastatic colon carcinoma (70%)
- Prostate carcinoma (60%)
- Osteogenic sarcoma
- Melanoma (30%)

Pure Cortical Metastases
- Melanoma
- Chroiocarcinoma
- Lung carcinoma

Intraventricular Choroidal Metastases
- Lung carcinoma
- Colon carcinoma
- Breast carcinoma

INTRACRANIAL CYSTS ON MR

- Arachnoid cyst: Extra-axial, no edema, non-enhancing
- Cystic neoplasm (low tumor density): Edema, enhances
- Chronic subdural hematomoa or hygroma: Extra-axial
- Suprasellar cyst from dilated third ventricle
- Interhemispheric cyst from porencephaly
- Posterior fossa cyst form of Dandy-Walker malformation
- Enlarged cisterna magna
- Post-infarct cystic encephalomalacia

- Cysts associated with isodense tumors
    a. Ganglioma: May calcify
    b. Cerebellar hemangioblastoma
    c. Cystic astrocytoma: Enhancing nodule
- Cysticerosis: Calcification

ARACHNOID CYST

Terminology

- Arachnoid cyst (AC), subarachnoid cyst
- Intra-arachnoid CSF-filled sac that does not communicate with ventricular system

Imaging Findings

- Best diagnostic rule: Sharply demarcated round/ovoid extra-axial cyst that follows CSF attenuation/signal
- 50-60% middle cranial fossa (MCF)
- Sharply-marginated extra-axial fluid collection isointense with CSF
- FLAIR: Suppresses completely with FLAIR
- DWI: No restriction

Top Differential Diagnoses

- Epidermoid cyst
- Chronic subdural hematoma
- Subdural hygroma
- Other nonneoplastic cysts

Pathology

- 1% of all intracranial masses
- If in middle fossa, temporal lobe may appear (or be) hypoplastic
- Subdural hematoma (increased prevalence, especially MCF)
- Acs displace but don’t engulf vessels, cranial nerves

Clinical Issues

- Often asymptomatic, found incidentally

Diagnostic Checklist

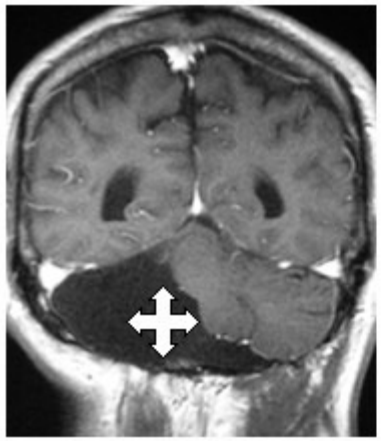
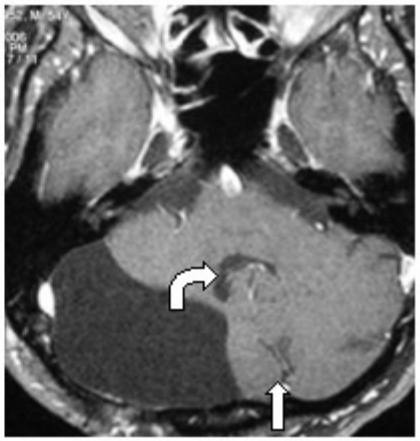
FLAIR, DWI best sequences for distinguishing etiology of cystic-appearing intracranial Pearls in the Diagnosis of Arachnoid Cyst

- Location: extra-axial
    a. Middle cranial fossa, peritemporal
    b. Cerebral convexity
    c. Posterior fossa, retrocerebellar
    d. Suprasellar quadrigeminal plate cisterm
- Homogeneous water-like signal, hypointense on T1 and very hyperintense on T2 (Remember that pulsation in the cyst may create the false impression of a nodule inside)
- There should be no evidence of intra-axial edema
- The margins of the lesion are smooth, sharp and straight, particularly in the middle fossa cysts along the posterior margin
- Suprasellar cysts are oval or square in shape, splay the cerebral peduncels and carotid termini, and push the mamillary body upward and posterior

Differential Diagnosis

- Epidermoid: Hypintense T1, mixed hyperintense T2, does not match CSF fluid
- Ependymal- or nonependymal-lined cyst: Intra-axial
- Craniopharyngioma: Mixed T1 and T2 signal, variable hyperintense fat
- Dermoid tumor: Hyperintense T1 & T2
- Cystic glioma: Hypointense T1, hyperintense T2, intra-axial with surrounding edema

- Masses

This is a series of MRI Scans of a 54 year old Male who complained about recent hearing impairment and ringing ("Tinnitus") in his Right ear. The scans show a large Arachnoid Cyst in the Right Cerebellar Pontine Angle of the Posterior Cranial Fossa.

DERMOID CYST

Imaging Findings

- Best diagnostic clue: Fat appearance + droplets in cisterns, sulci, ventricles if ruptured
- T1 C+: With rupture: Extensive MR enhancement possible from chemical meningitis
- Use fat-suppression sequence to confirm diagnosis

Top Differential Diagnoses

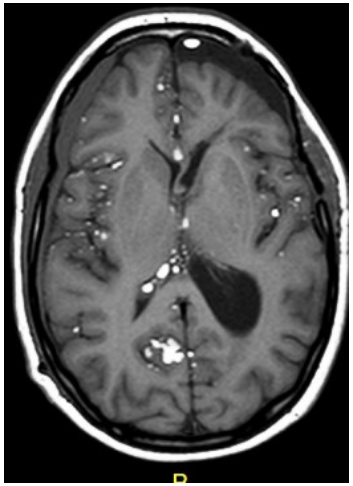
- Epidermoid cyst
- Craniopharyngioma
- Teratoma

Pathology

- Rare: < 0.5% of primary intracranial tumors
- Unilocular cyst with thick wall of connective tissue

Clinical Issues
- Uncomplicated dermoid: Headache (32%), seizure (30%) are most common symptoms
- 30-50 y
- Gender: Slight male predilection
- Larger lesions associated with higher rupture rate
- Rupture can cause significant morbidity/mortality
- Rare malignant transformation into SCCa
- Treatment: Complete microsurgical excision

Diagnostic Checklist
- Follows fat characteristics on NECT and T1WI fat-suppressed MRI

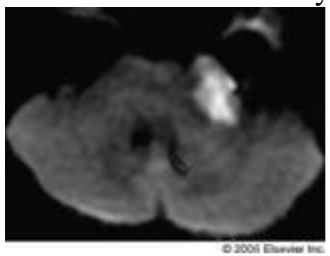


EPIDERMOID CYST

Terminology
- Intracranial epidermoids are congenital inclusion cysts

Imaging Findings
- Best diagnostic clue: CSF-like mass insinuates cisterns, encases nerves/vessels

- Morphology: Lobulated, irregular, “cauliflower-like” mass with “fronds”
- FLAIR: Usually doesn’t completely null
- Restricted diffusion yields high signal



Top Differential Diagnoses

- Arachnoid cyst
- Inflammatory cyst (i.e., neurocysticercosis)
- Cystic neoplasm
- Dermoid cyst

Pathology

- 0.2-1.8% of all primary intracranial tumors

Clinical Issues

- Most common symptom: Headache
- Cranial nerve 5,7,8 neuropathy common
- Age: Presents at 20-60 y with peak at 40
- Grows slowly: Epithelial component growth rate commensurate to that of normal epithelium
- Rare malignant degeneration into squamous cell carcinoma (SCCa) reported
- Treatment: Microsurgical resection

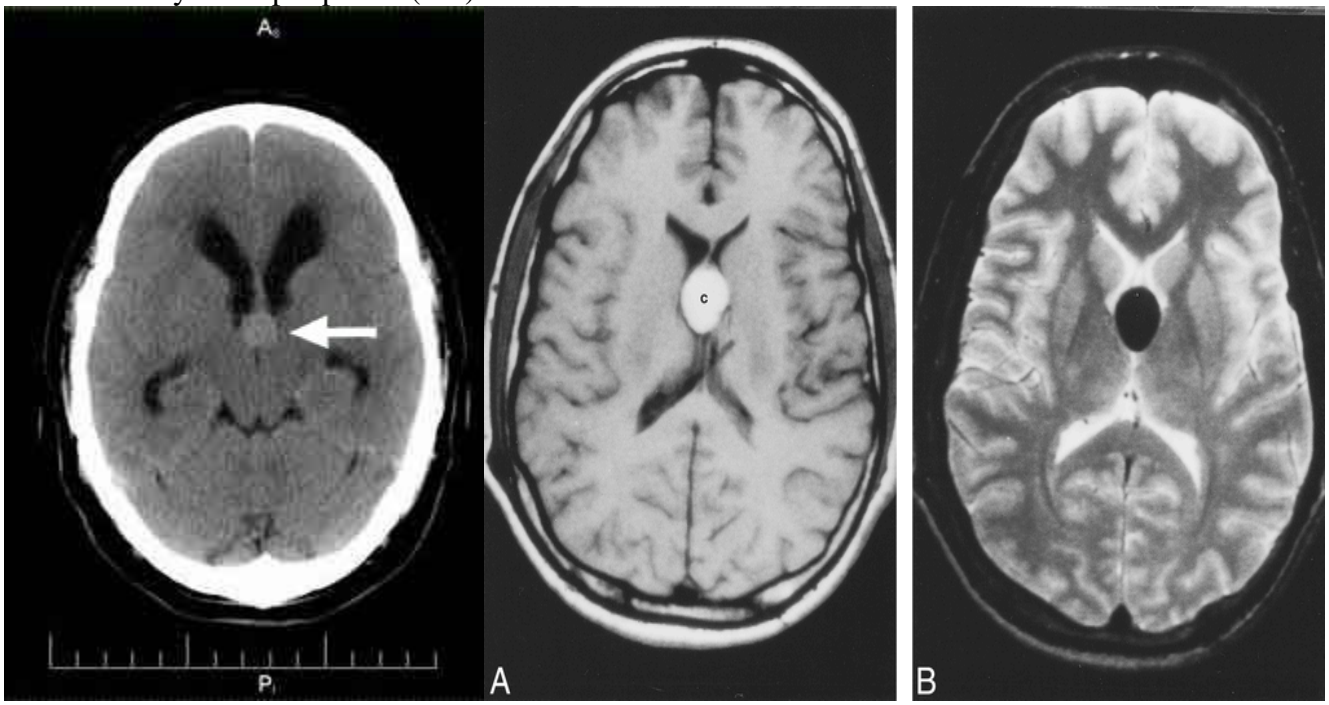
Diagnostic Checklist

- Resembles CSF on imaging studies, except usually incomplete nulling on FLAIR
-

COLLOID CYST

Imaging Findings

- Best diagnostic clue: Hyperdense foramen of Monro mass on NECT
- Most anterosuperior ventricle location
- Size < 1.5 centimeters
- No or nominal contrast enhancement
- Density correlates inversely with hydration state
- 2/3 hyperintense on T1WI
- Majority isointense to brain on T2WI (small cysts may be difficult to see!)
- 25% mixed hypo/hyper ("black hole" effect)
- Rare: May show peripheral (rim) enhancement



Indirect Signs

- Intermittent hydrocephalus
- Paramagnetic

Appearance of Colloid Cysts by Signal Intensity

- Paramagnetic type, most common (60%): Hyperintense T1, hypointense T2, peripheral hyperintense T2 rim*
- Cystic type, uncommon (20%): Hypointense T1, hyperintense T2
- Isointense type, rare (10%): Isointense T1 and isointense or minimally hyperintense T2
- Mixed type, rare (less than 105): Hyperintense T1, isointense T2

*Fluid levels with T2 hypointensity layering dependently are seen in 30-40% of cases.

Top Differential Diagnoses

- Neurocysticercosis
- CSF flow artifact (MR "pseudocyst")
- Neoplasm
- Choroid plexus mass

Pathology

- From embryonic endoderm, not neuroectoderm!
- 0.5 – 1.0% primary brain tumors
- 15-20% intraventricular masses

Clinical Issues

- Headache (50-60%)
- 3^{rd}-4^{th} decade
- 90% stable or stop enlarging
- 10 % enlarge
- May enlarge rapidly, cause coma/death!

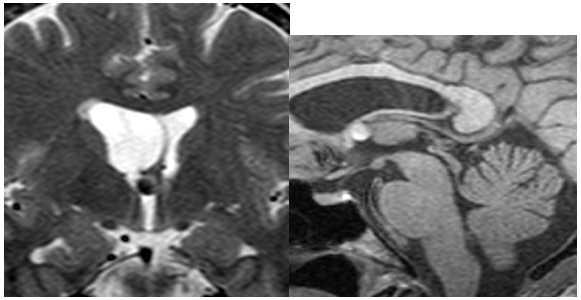
Diagnostic Checklist

- Notify referring MD immediately if CC identified (especially if hydrocephalus is present)

Pearls in the Diagnosis of Colloid Cysts

- Signal intensity similar to hemorrhagic cysts or cystic craniopharyngioma
- Paramagnetic type, most frequent

- Most anterosuperior third ventricle location
- Intermittent hydrocephalus
- Minimal enhancement on CEMR
- Internal constituents include calcium, magnesium, copper, iron, manganese, and sodium
- Rare in the pediatric population
- May simulate melanoma metastases



RADIATION NECROSIS & RECURRENT NEOPLASM

Radiation Necrosis

- Hypointense T1, iso- to hyperintense T2, variable central HypointensityEnhancement is corrugalated, wavy & irregular (wrinkled configuration)

Recurrent Neoplasm

- Variable T1 and T2
- Masslike & Nodular
- Edema has no respect for the forceps major or corpus callosum
- Metabolism on Pet usually equals or exceeds that of white matter
- May recur early or late

SIGNS OF INTRACRANIAL NEOPLASTIC HEMORRHAGE

- Intermediate signal intensity on T1 and T2
- Signal alteration which does not correspond to any known pattern of blood evolution over an appropriate period of time
- Delayed temporal resolution of hemorrhage
- Bizarre or complex signal intensities
- Irregular, absent or complex hemosiderin rings
- Persistent or exaggerated edema
- Lesion multiplicity
- Absence of hypointense calcification
- Disproportionate mass effect for lesion size
- Nodular enhancement

FREQUENCY OF HEMORRHAGE IN INTRACRANIAL NEOPLASMS

- Pineal choriocarcinoma or teratocarcinoma (90%)
- Primary intracranial neuroblastoma (85%)
- Oligodendroglioma (80%)

- Melanoma (70%)
- Ependymoma (55%)
- Glioblastoma multiforme or high-grade astrocytic neoplasm (35%)
- Metastasis (especially renal cell, thyroid, bronchogenic, melanoma and choriocarcinoma)
- The frequency of intracranial neoplastic hemorrhage increases dramatically after cranial irradiation

CYSTIC PITUITARY MASSES

Empty Sella

- Homogeneous T1 hypointensity, T2 hyperintensity: Pure water signal
- Midline pituitary stalk
- Paucity of solid pituitary tissue
- No evidence of chiasmatic or suprasellar mass effect
- Enhancement of normal pituitary tissue only

Arachnoid Cyst

- Homogenous T1 hypointensity, T2 hyperintensity: Pure water signal
- Sellar, suprasellar, retrosellar, and perisellar mass effect are common
- Effaced and displaced pituitary stalk, multidirectional
- Enhancement of normal pituitary tissue

Cystic Craniopharyngioma

- May have foci of T1 hyperintensity
- May have hypointense T2 calcification
- Suprasellar +/- intrasellar extension
- Mixed enhancement

Cystic Pituitary Adenoma or Neuroectodermal Tumor

- Moderate T1 hypointensity, T2 hyperintensity: Proteinaceous water signal (therefore no exact CSF match)
- Displaced pituitary stalk
- Hormonal abnormalities frequently present
- Intra- and suprasellar mass effect and/or extension

- Foci of nodular enhancement from solid components

Dermoid
- Hyperintense T1, hyperintense T2
- Predominantly suprasellar
- Nonenhancing

Epidermoid
- Hypointense T1, hyperintense T2
- Inhomogeneous
- Minimal peripheral enhancement
- Irregular shape

Cystic Optic Chiasmatic/Hypothalamic Glioma
- Neurofibromatosis frequently associated
- Origin in, or involvement of, the optic chiasm or hypothalamus
- Mild enhancement

CRITERIA FOR SELLAR MICROADENOMA

Direct Criteria
- T1 hypointensity < 1 cm in size
- Hypointense on CEMR < 8 minutes, can be isointense 8-40 minutes and hyperintense on CEMR > 40 minutes

Indirect Criteria
- Stalk deviation
- Abnormal gland height (greater than)
    a. 6-7 mm male or prepubescent
    b. b. 7-11 mm gravid female postpuberty
    c. c. 10-13 mm female, peripartum
- Floor deviation
- Gland size asymmetry

PEARLS IN THE DIAGNOSIS OF PITUITARY MACROADENOMA

- Greater than 1 cm
- Isointense on T1
- Iso- or hyperintense on T2 with mild to moderate enhancement
- Diaphragma sellar notching helps differentiate extrasellar mass growing down from pituitary mass growing up
- Complications of macroadenoma
    a. Chiasmatic compression
    b. Cavernous sinus invasion
    c. Prolactin levels > 2,000 picograms/dl implies cavernous invasion
- Cont. of Complications of macroadenoma
    ii. Abuts the lateral dural sinus walls
    iii. Loss of speckled signal in the cavernous sinus distribution
        c. Pituitary hemorrhage or apoplexy
        d. Pituitary adenoma recurrence
        e. Differential diagnosis of macroadenoma
    i. Craniopharyngioma
    ii. Meningioma (dark-isointense)
    iii. Aneurysm (lamellated)
    iv. Metastases (isointense-bright)

INTRASELLAR PITUITARY SIGNAL VOIC OR HYPOINTENSITY

Common

- Volume averaging of the parasellar carotid artery dura and CSF

Uncommon
- Aneurysm of the paracavernous carotid artery

Rare
- Vascular malformation of the paracavernous carotid artery
- Primitive trigeminal artery aneurysm
- Pituilith
- Intrasellar calcified meningioma
- Pituitary gas associated with abscess formation or prior surgery
- Intrasellar calcified craniopharyngioma
- Postsurgical intrasellar susceptibility effect (clip, metal, etc.)

INTRACRANIAL SIGNAL: HYPERINTENSE T1, HYPOINTENSE T2

Common
- Subacute hematoma or clot (mid or high field)
- Flow (first echo slice entry phenomenon)
- Melanoma metastases
- Hemorrhagic metastases (choriocarcinoma, neuroblastoma, embryonal cell carcinoma, thyroid carcinoma, renal cell carcinoma, malignant melanoma)
- Lipoma
- Pantopaque

UncommonColloid cyst
- Calcified xanthogranuloma
- Aneurysm (with clot or slice entry)

- Craniopharyngioma

INTRACRANIAL SIGNAL: HYPO-TO ISOINTENSE T1, HYPOINTENSE T2 (1.5T)

Common

- Acute hematoma
- Flow (first echo flow void, second echo rephasing)
- Aneurysm with flow phenomenon or acute clot
- Calcification (nontraumatic, nonhemorrhagic)
- Brain iron
- Neoplasm with acute hemorrhage or extensive calcification
- Meningioma
- Metastases (colon, prostate, osteogenic sarcoma, breast
-

Uncommon

- Colloid cyst
- Melanoma

Rare

- Chloroma

INTRACRANIAL SIGNAL: ISOINTENSE-T1 & T2

Common

- Hyperacute hematoma (transition to acute hematoma [1.5 T])
- Acute hematoma (mid field [0.5 T])
- Subacute hematoma (high field)

- Flow (combinations of flow void adding to flow-related hyperintensity)
- Aneurysm with flow phenomenon
- Meningioma
- Brain iron (low field)
- Isointense metastases (colon, prostate, osteogenic, sarcoma, breast)
- Hamartoma

Uncommon

- Colloid cyst

Rare

- Medulloblastoma or adult cerebellar sarcoma
- Lymphoma
- Tuberculoma
- Chloroma

BLACK SIGNAL

- Flow
- Air or gas
- Hemosiderin (T2 dependent)
- Iron, copper or metal
- Bone
- Calcium
- Superparamagnetic contrast agents
- Ligaments, tendons, fascia
- Magnetic susceptibility
-

HYPOINTENSE RINGS

- Hemosiderin ring: Chronic hematoma
- Susceptibility rim artifact: Glial or astrocytic neoplasm
- Pseudocapsule of dura, cerebrospinal fluid, cleft, desmoplasia, vessels: Meningioma
- Fibrous rim: Abscess, neurocysticerosis, meningioma

INTRAVENTRICULAR MASSES

Hypointense T1, Hyperintense T2, Nonenhancing

- Arachnoid cyst
- Cysticerosis
- Colloid cyst (3rd ventricle)
- Cystic craniopharyngioma
- Cystic meningioma
- Dandy-Walker cyst (4th ventricle)
- Epidermoid (4th ventricle)
- Neuroepithelial cyst, intraventricular type
- Neuroepithelial cyst (xanthogranuloma of the choroid plexus)

CSF Signal Mass

- Arachnoid cyst
- Cysticerosis
- Dandy-Walker cyst or variant (4th ventricle)
- Mega cisterna magna (pseudocyst)
- Trapped ventricle (pseudomass)Hypointense T1 & T2, Enhancing
- Acute hematoma
- Calcified giant cell astrocytoma
- Calcified flomus of choroid plexus

- Dense or calcified metastases (prostate, colon, osteogenic sarcoma)
- Heavily calcified meningioma
- Hemorrhagic ventricular metastases

Hyperintense T1, Hypointense T2

- Colloid cyst
- CSF flow
- Dermoid
- Early subacute hemorrhage
- Intraventricular craniopharyngioma (heavily calcified)
- Lipoma
- Pantopaque
- Xanthogranuloma of the choroid plexus

Hyperintense T1 & T2

- Dermoid
- Flow
- Intraventricular craniopharyngioma (3rd ventricle)
- Late subacute hemorrhage

Intraventricular Masses by Site

	Lateral	*Third*	*Fourth*
NEOPLASMS			
Choroid plexus pspilloma/ca	Common, pediatric	Common from	Common, adult

Craniopharngioma Ependymoma Medulloblastoma		suprasellar growth	
Meningioma	Common, glomus, atrium		Along choroid plexus
Metastases	Yes	Yes	Yes
Choroid	Rare	Typical	Not reported

Differential of temporal lobe lesions

Tumor	***Age***	***Demarcation***	***Edema?***	***Percent of Tumors Causing Temporal Lobe Seizures***	***Hemorrhage***	***Cyst Formation***	***Enhancement***	***Cortical Involvement***	***Calcification***
Ganglioglioma	0-30	Well	Very little	40%	Rare	Common	Uncommon	Common	Variable
Low grade astrocytoma	0-30	Well	Yes	26%	Rare	Common	Uncommon	Uncommon	Variable to uncommon
DNET	10-20	Well	None	18%	Common	Common, dominant multiple	Uncommon to variable	Always	Common
Oligodendrogli-oma	30-60	Less well	Yes	6%	Variable	Variable	Common	Variable	Common
PXA	10-35	Well, but malignant change in 20%	Uncommon	4%	Rare	Common	Common in mural nodule	Common, meningeal attachment	Rare
Desmoplastic infantile ganglioglioma	0-1	Well	Occasiona-lly	< 3%	No	Common	Common in nodule demoplasia	Dural attachment	None

Scar versus residual tumor

Feature	***Scar***	***Tumor***
Enhancement within 1-2 days	No	Yes
Enhancement after 3-4 days	Yes	Yes
Change in size with time	Decreases	Increases
Type of enhancement	Linear, outside preoperative tumor bed	Nodular, solid
Mass effect edema	Decreases	Increases

Neuroimaging in Neuro-Oncology
2015

An estimated 25,000 primary malignant and 53,000 non-malignant tumors are expected to be diagnosed in the United States in 2016. The prevalance of primary malignant CNS tumors is 61.9 per 100,000, compared to a non-malignant prevalence rate of 177.3 per 100,000. In 2016, 16,000 deaths will result from tumors of the CNS. The most commonly occuring malignant CNS tumor is glioblastoma, and the most common non-malignant primary tumor is megingioma.[1] (KP 1) One half of all benign primary brain tumors are meningiomas (KP 2).

Intra-axial lesions are those that arise from cells in the brain, in contrast to extra-axial lesions that arise from the nerves, meninges, and other structures outside of the brain. CNS neoplasms present with various symptoms. The location of the tumor, of course, determines which symptoms are most likely to be manifested. Generally, symptomatology arises over a sub-acute to chronic time period. However, if a patient presents with sudden onset of focal deficits from an underlying neoplasm, then one should suspect that there has been an acute complication such as hemorrhage into the core of the tumor.[2] Metastatic disease must be suspected in patients with CNS neoplasm presenting with systemic signs such as fever and weight loss.[3]

When a tumor is suspected, neuroimaging is crucial for diagnosis and preoperative planning as well as for post-treatment evaluation and follow-up. Characterization of brain tumors on MRI involves an initial determination of whether the mass is actually a neoplasm. Despite the high resolution and tissue contrast provided by MRI, one has to be cognizant that neuroimaging is not yet a substitute for tissue diagnosis and therefore caution must always be taken when interpreting

imaging findings. MRI with and without gadolinium is the imaging test of choice, however CT remains useful due to its wide availability, relative low cost (KP 3). MRI contrast enhancement on T_1- weighted images represent breakdown of the blood-brain barrier (BBB) where gadolinium has leaked out (KP 4). However, it is not synonymous with a malignant brain tumor. Vasogenic edema can be subtle on contrast-enhanced T_1-weighted images, but is easily noted on T_2-weighted images because of the high sensitivity to changes in water content of the brain. Fluid-attenuated inversion recovery (FLAIR) imaging is a type of T_2-weighted imaging sequence where CSF signal is suppressed in order to better evaluate regions adjacent to the ventricles or sulci.[4]

Table 1. MRI Signal Characteristics in Pathologic Brian Tissues

	T1	**T2**	**CT**	**Enhancement Pattern**
Infarct	Dark	Bright	Dark	Subacute
Hemorrhage	Bright unless superacute or chronic	Bright unless superacute or chronic	Bright	No
Tumor	Dark	Bright	Dark unless calcified	YES

T1- and T2-weighted sequences

In general brain tumors are hypointense on T1 weighted images and hyperintesnse on T2 weighted images, reflecting increased water content of the neoplasm as well as vasogenic edema. But there are important exceptions to this generalization. Tumors that are hypointense on T2 include neoplasms that have paramagnetic effects such as iron, hemosiderin, deoxyhemoglobin, and melanin. (KP 5) In addition, hypointensities on T2 can be due to calcification, high

nucleus/cytoplasm ratio (i.e., PNET, lymphoma), dense cellularity, fibrocollagenous stroma, very high protein concentration, and signal flow voids from rapid flow. Hyperintensity on T1 weighted images within brain tumors can be due to methemoglobin, melanin, some forms of calcification, natural occurring ions associated with cellular necrosis such as manganese, iron, and copper, high protein, fat, and flow related enhancement in tumor vessels. (KP 6)

Another important sequence is susceptibility-weighted imaging (SWI). SWI uses post-processing to accentuate differences in susceptibility effect between tissues. SWI is extremely sensitive for detecting iron and blood products and is particularly useful for detecting microhemorrhages that are below the resolution of T1 and T2 weighted images. SWI is more sensitive compared to conventional imaging techniques for showing small vessels, calcification, and microhemorrhage in brain tumors. The use of SWI is especially useful in the evaluation of post-radiation microhemorrhage.[5]

Table 2. Short T2 and Short T1 in Brain Tumors

Short T2 (Hypointensity)	Short T1 (Hyperintensity)
Iron with necrosis (GBM)	Methemoglobin (subacute blood)
Hemosiderin (chronic bleed)	Melanin (melanoma)
Deoxyhemoglobin (acute bleed)	Manganese
Melanin (melanoma)	Calcium Iron
Ferritin	Coppper
Calcification (oligodendroglioma)	High protein (colloid cyst, cranio)
High nucleus:cytoplasm ratio (PNET, lymphoma)	Fat (lipoma, dermoid)
Dense cellularity	Cholesterol
Macromolecule content	Paramagnetic agent (gadolinium)
Fibrocollagenous stroma (meningioma)	Flow-related enhancement in tumor vessel
Mucin (colon carcinoma)	Calcium
High protein content (Craniopharyngeoma)	

Flow void (Hemangioblastoma, GBM) Air	

Diffusion weighted imaging (DWI)

Molecular mobility is the essential contrast mechanism exploited in DWI, reflected in a measurement of the apparent diffusion coefficient (ADC). Restricted diffusion represents non-isotropic (anisotropic) molecular movement. The greater the density of structures impeding water mobility, the lower the ADC; therefore ADC is considered a noninvasive indicator of cellularity or cell density. DWI is an echo planar imaging-based sequence that can acquire a sufficient number of images in milliseconds.[6] Increased ADC implies unrestricted water motion and decreased ADC indicates restricted diffusional motion.[7] Therefore, DWI/ADC is extremely sensitive in detecting pathologies such as acute ischemia, abscess, hypercellularity, and postoperative brain injury (KP 7). Recent findings indicate that in gliomas, the higher the WHO tumor grade the lower the ADC.[7]

DWI sequences have been adapted to perform DTI by acquiring data in six or more directions. DTI allows for visualization of the location, orientation, and anisotropy in the white matter tracts of the brain and spinal cord. DTI can be used to provide maps of white matter fiber tracts (tractography) in tissues adjacent to tumors [9,10.] By elucidating the anatomical relationship of motor and sensory pathways to tumor tissue, DTI can assist in surgical planning to limit surgical morbidity. [10]

Table 3. Comparison of tumor imaging with different diagnostic modalities.

Imaging Technique	Utility in Brain Tumor Imaging
CT	Mass effect, herniation, hydrocephalus, hemorrhage, calcifications
Pre and post-contrast T1	Enhancement characteristics, necrosis, extent of the enhancing portion of the tumor
T2/T2 FLAIR	Peri-tumoral edema (vasogenic and infiltrative), non-enhancing tumor
T2* susceptibility sequence (SWI)	Blood products, calcifications, radiation induced chronic micro-hemorrhages
DWI/ADC	Reduced ADC in highly cellular portions of tumor, post-operative injury
DTI	Tractography for surgical planning/navigation
Perfusion weighted Imaging	Tumor/tissue vascularity
MR spectroscopy	Metabolic profile
fMRI	Pre-operative functional mapping, research into treatment effects

Perfusion imaging

Perfusion imaging proposes to measure the degree of tumor angiogenesis and capillary permeability, both of which are important biological markers of malignancy, grading, and prognosis, particularly in gliomas. The most robust quantitative variable derived is relative cerebral blood volume (rCBV), which can be correlated to tumor angiogenesis. Cellular studies have demonstrated a strong positive correlation between tumor, rCBV, and astrocytoma grading. Low grade astrocytomas have significantly lower average rCBV than anaplastic astrocytoma or glioblastoma. rCBV maps may be used to select biopsy sites for enhancing and non-enhancing tumors by highlighting a “hot” (i.e., hypervascular) area. [11] Perfusion imaging has been used to characterize glioma WHO grade, tumor genotype, guide biopsy, and provide prognostic

information.[12] Thus, perfusion imaging together with conventional MRI should be considered in the diagnosis and monitoring of brain tumors before, during, and after therapy.

Magnetic Resonance Spectroscopy (MRS)

MRS is a powerful technique that offers unique metabolic information regarding brain tumor biology that is not available from anatomic imaging. Potential uses of MRS include refinement of preoperative differential diagnoses, biopsy site selection, monitoring of response to treatment, and distinction of progressive tumors from treatment effects. There is a characteristic spectrographic appearance of a tumor, which includes an increase in the choline (Cho) peak, a decrease in the N-acetyl aspartate (NAA) peak, an increased Cho/creatine ratio, and in some cases the presence of a lactate peak. (KP 8)[13,14] Spectroscopic imaging improves the specificity of brain tumor imaging, because it not only enables biochemical assessment of tumor dynamics, but also can show residual or recurrent tumor beyond the margins of the tumor as seen on structural images.[14]

Functional MRI

Functional magnetic resonance imaging (fMRI) is a neuroimaging technique that can depict dynamic blood oxygenation changes in the vessels of the brain and thereby allows for an estimation of brain activity in a non-invasive manner. The increased blood flow is not matched by an increase in oxygen extraction, so the concentration of deoxyhemoglobin is reduced. Since deoxyhemoglobin is paramagnetic, there is a change in the T2* signal. This change, referred to as blood-oxygen level dependent contrast (BOLD), is detected by the MRI sequence. [15]

Functional MRI is used primarily for preoperative localization of eloquent brain regions prior to tumor resection in attempt to minimize intraoperative morbidity. Functional MRI reliably images the primary motor and sensory areas of the cortex. The ability to identify speech areas may make the WADA test for speech and memory laterality someday obsolete [16]

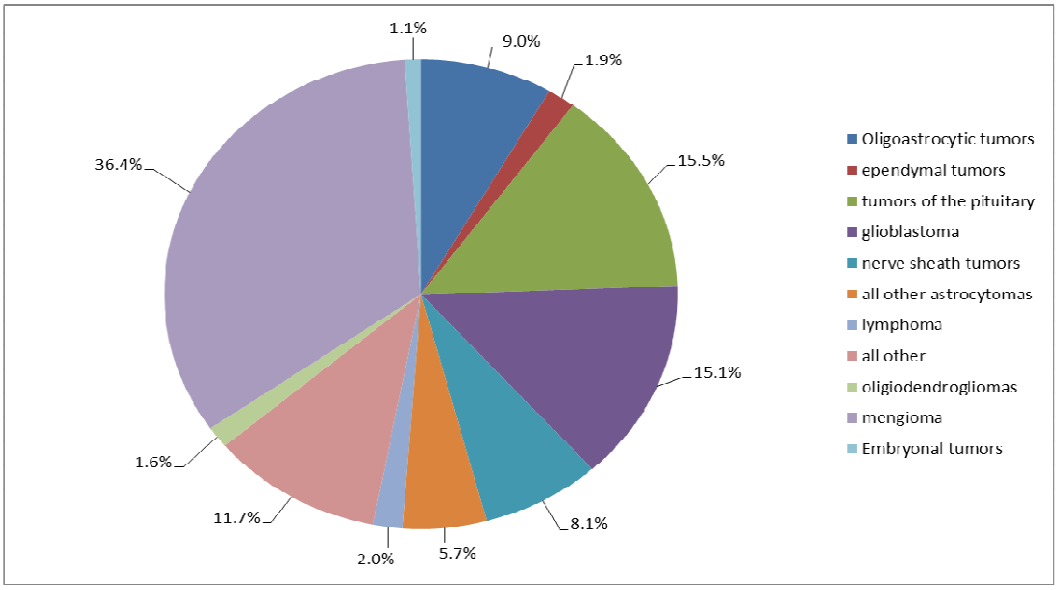


Figure 9a. Distribution of All Primary Brain and CNS Tumors by Histology Groupings, Central Brain Tumor Registryof the United States Statistical Report. CBTRUS Statistical Report: NPCR and SEER, 2008-2012. (Reference 1)

Gliomas

Glioma is a tumor arising from glial cells including astrocytes, oligodendrocytes, ependymal cells, and cells of the choroid plexus. Estimated 50% of all primary brain tumors are astrocytomas.[4] The histological features in grading of gliomas include necrosis, vascular proliferation, mitotic index, and nuclear atypia.[3] Although grading of gliomas is ideally based on histological evaluation, there is a significant correlation between rCBV and glioma grade, allowing for a noninvasive method for estimating tumor grade.[17] Low-grade astrocytomas have lower mean rCBVvalues than do anaplastic astrocytomas or glioblastomas (KP 9). This

relationship between rCBV and tumor grade is consistent with other studies demonstrating that microvascular density in low grade astrocytomas is lower than in anaplastic astrocytomas or glioblastomas.

Glioblastoma is the most highly vascularized glioma and comprises 66% of all gliomas. Histologically, glioblastomas have necrosis, vascular proliferation, or pleomorphic cells, but a single tumor may exhibit different histological features at different sites. Imaging features in addition to immunohistochemical staining may provide clues to help correlate overall tumor prognosis.[4] Studies have shown that rCBV measurements of a tumor can predict the time to progression or survival in patients with glioma. Patients with high pre-treatment rCBV have lower survival rates when compared to patients with lower pre-treatment rCBV; longer survival in the latter case is >15 months.[17] This data is helpful in designing individualized treatment plans.

Grade I Astrocytoma

Astrocytomas are common intracranial neoplasms in children and young adults. About 75% are juvenile pilocytic astrocytomas (JPAs), which are WHO grade I tumors.[4] They typically occur in the cerebellar hemispheres and produce symptoms of elevated intracranial pressure, headache and ataxia.[3] They may be associated with neurofibromatosis type I (NF1), especially if the optic pathways are involved. These tumors are usually amenable to surgical resection.

Optic nerve gliomas are JPAs arising from the optic nerve, chiasm, or optic tract and almost exclusively affect children and teenagers. Abnormal signals are observed throughout the visual

pathway, including the optic tracts, lateral geniculate body, and optic radiations. They are often silent early on and usually present with progressive visual loss. On imaging, JPAs are well circumscribed with a large cystic component and an enhancing mural nodule. The cyst is often isointense to CSF on all sequences, though cysts with high protein content may be hyperintense to CSF on certain sequences.[3] The cystic component is hypointense on T1 and hyperintense on T2, and the solid component is isointense on T1 and hyperintense on T2. After gadolinium, the wall of the cyst shows heterogeneous enhancement.3

Table 4. Cortical Based Masses with a cyst and nodule

Common	**Less Common**	**Rare**
Ganglioglioma	Pilocytic Astrocytoma	Hemangioblastoma
Metastasis	Pleomorphic xanthoastrocytoma	Papillary glioneural tumor
	Glioblastoma multiforme	Ganglioglioma
		Schwannoma

Subependymal giant cell astrocytomas (SEGAs) are benign intraventricular tumors without anaplastic potential comprised of variably sized astrocytic-appearing cells with variable signal intensity on both CT and MRI, depending on the degree of calcification. They are seen in 6-16% of patients with tuberous sclerosis complex (TSC) [18] They arise from subependymal cells and project into the lateral ventricle adjacent to the septum pellucidum. They may be indistinguishable from subependymal nodules (SENs) histopathologically. On MRI they appear

larger in size and have progressive increase in size during serial imaging, unlike SEN. They may require surgical resection if they show rapid growth and cause obstructive hydrocephalus.[10]

Grades II and III Astrocytomas

Grade II astrocytomas, known as diffuse astrocytomas, have an average survival time of five to six years, while grade III astrocytomas, known as anaplastic astrocytomas, have an average survival time of two and a half years. Histologically, these tumors lack pleomorphic cells, necrosis, or vascular proliferation, though mitoses may be seen in anaplastic astrocytomas.[3] For diffuse astrocytoma, any focus of elevated rCBV is concerning for malignant transformation. A non-enhancing anaplastic astrocytoma behaves like a low grade tumor, whereas presence of enhancement is associated with risk of recurrence and shortened survival, behaving similar to glioblastoma (Grade IV).[19]

Grade II gliomas appear T1 hypointense and T2 hyperintense. Diffusion is not restricted and no enhancement is seen after contrast administration. There is also swelling and thckening of both white and grey matter. In grade II tumors, MRS shows mild NAA reduction and mildly increased choline, and perfusion MRI does not show elevated rCBV. In contrast, for grade III tumors perfusion MRI will show increased rCBV values and spectroscopy will show a higher choline peak and a lower NAA peak.

Grade IV Astrocytomas (Glioblastoma)

Grade IV astrocytomas, commonly known as glioblastomas, are the most common and the most

lethal of astrocytomas, with median survival of 15 months. They are slightly more common in men over the age of 50 and almost always occur in the cerebral hemispheres. On imaging studies, the tumor may appear as a discrete mass, with neoplastic cells spread along white matter pathways throughout the brain by the time of diagnosis.[4] It is believed that 60% of glioblastomas arise *de novo* in adults older than the age of 50. Secondary GBM typically develops in younger patients under the age of 45 through malignant progression from a low-grade astrocytoma or anaplastic astrocytoma.

Glioblastomas have heterogeneous enhancing patterns with central non-enhancing areas representing necrotic tissue. T2-FLAIR MRI shows extensive vasogenic edema within the white matter. Glioblastomas may spread from one hemisphere to the other via the corpus callosum producing a butterfly appearance.[20]

Gliomatosis cerebri is a type of malignant astrocytoma, generally WHO Grade III with extensive tumor infiltration without a discrete mass or necrosis. T2-weighted sequences show a hyperintense infiltrating mass with small ventricles and effaced cortical sulci. With gadolinium, there may be small areas of patchy enhancement.[21] Gliomatosis cerebri commonly presents in people younger than 40 years of age. With radiation and chemotherapy, survival may be prolonged to nearly 3 years.[4]

In some patients with high grade glioma, shortly after concurrent chemotherapy and radiation therapy, follow-up imaging demonstrates an increase in contrast-enhancing lesion size with subsequent improvement or stabilization. This is termed “pseudoprogression.” It is a treatment-

related phenomenon and is most likely due to reactive inflammation, edema, and abnormal vessel permeability. Some studies note improved survival in the setting of pseudoprogression and it may correlate with the extent of the patient's innate immune response to the tumor.[22] Most pseudoprogression occurs within three months of the end of radiation therapy. However, pseudoprogression occurring after this three month period but within the first year is not uncommon, particularly in tumors with MGMT promoter methylation. [23] No single imaging technique has been validated to recognize and adequately establish a diagnosis of pseudoprogression from true progression and therefore at present the diagnosis is made based on suspicion and comparison to follow-up studies.[24]

Another treatment related imaging phenomenen is "pseudoresponse" which is due to the use of antiangiogenic agents such as bevacizumab. Pseudoresponse is characterized by a marked decreased in the enhancing portion of the lesion as early as 1 to 2 days after initiation of therapy, and is associated with radiologic response rates of 25% to 60%.[25] However enlargement of the nonenhancing portion of the lesion on T2-weighted sequences was observed on follow-up scans. This phenomenon may explain why overall survival increases are modest at best, despite the percepetion (by imaging) of an excellent initial response to treatment.

Not surprisingly, both phenomena (pseudoprogression and pseudoresponse) have to potential to confound the imaging assessment of CNS neoplasms.[26] In 2010, the Response Assessment in Neurooncology (RANO) criteria were published, updating the Macdonald criteria in several important ways, while maintaining a reliance upon the product of perpendicular diameters of contrast-enhancing lesions as an indicator of tumor size and status. [27]

Table 5. Response Assessment in Neuro-Onocology (RANO) Criteria

	Complete response	Partial response	Stable disease	Progressive disease
T1-Gd +	None	≥ 50% decrease	< 50% decrease to < 25% increase	≥ 25% increase
T2/FLAIR	Stable or decrease	Stable or decrease	Stable or decrease	Stable or increase *
New Lesion	None	None	None	Present *
Corticosteroids	None	Stable or decrease	Stable or decrease	NA
Clinical status	Stable or increase	Stable or increase	Stable or increase	Decrease*
Requirement for response	All x4weeks	All x 4 weeks	All	Any *

* Progression occurs when any of the criteria is/are present

Treatment follow-up: radiation necrosis versus recurrent or residual tumor

Distuingishing radiation necrosis from recurrent tumor has significant therapeutic implications. Patients with recurrent tumors may benefit from further surgical management with or without adjuvant chemotherapy, as opposed to patients with radiation necrosis, who may be treated conservatively with steroids or antiangiogenic agents. [28]

Delayed radiation necrosis and recurrent tumor can both appear as a new or enlarging lesion with surrounding edema, and conventional contrast-enhanced CT and MRI are not reliable in discriminating the two. Delayed radiation necrosis pathologically shows features of extensive endothelial injury and fibrinoid necrosis whereas recurrent tumor is characterized by vascular proliferation. MRI based rCBV mapping can reveal differences in vascularity and may help

differentiate one process from the other.[21] (KP 10) Radiation necrosis appears as a lesion heterogeneously hypointense on T1 and hyperintense on T2 with inhomogeneous peripheral enhancement after contrast administration. Hypointensity on T2* sequences such as GRE or SWI may reflect the presence of hemorrhage or blood breakdown products. rCBV values are generally lower in radiation necrosis compared to recurrence of neoplastic disease. A lesion due to radiation necrosis may develop distant from the original tumor site while this is less common in tumor recurrence.

Oligodendrogliomas

Oligodendrogliomas arise from oligodendrocytes and represent about 5-10% of primary CNS tumors in adults. They generally show some calcification and are less avidly contrast enhancing than gliomas. There genetic signature is co-deletion of chromosomal arms 1p and 19q. Those without this co-deletion have a poorer prognosis and are less responsive to chemotherapy.[4]

The tumor is most often T1 hypointense, heterogeneously T2 hyperintense, and calcifications, if present, are hyperdense on CT and hypointense on T2* images. With contrast, the lesion shows heterogeneous enhancement. MRS may show elevated choline. rCBV is less reliable to assess the grade of oligodendrogliomas, since rCBV may be elevated even in low-grade forms.[1]

Astrocytomas and oligodendrogliomas cannot be differentiated from one another based on imaging alone, although there are some characteristics that favor oligodendrogliomas, such as cortical involvement, presence of calcification, heterogeneous signal, including in some cases the presence of an intratumoral cyst, and subtle patchy enhancement. For low grade tumors including

astrocytomas and oligodendrogliomas, every new MRI should be compared with previous studies over as long period of time as possible to detect the presence of gradual interval growth.

Ependymomas

Ependymomas arise from ependymal cells lining the ventricles. Since they tend to fill the fourth ventricle and expand through the foramen of Luschka, Magendie, and magnum, they present primarily with signs of increased intracranial pressure and hydrocephalus.[4] These tumors often cause "dropped metastasis", spreading from the fourth ventricle to the spinal canal via CSF and producing distant spinal metastatic foci along the CSF spaces without intervening lesions. Therefore imaging of the entire neuraxis with MRI is essential. Calcification and microhemorrhage is observed in about half of ependymomas.[3]

Ependymomas appear heterogeneous due to the concurrent presence of necrosis, calcification, methemoglobin, hemosiderin, and hypervascularity. They most often appear T1 iso- to hypo-intense and T2 iso- to hyper-intense. Cystic and hemorrhagic components are rare. Enhancement with gadolinium is usually heterogeneous.[4]

Table 6. Intraventricular Masses

Ependymoma
Subependymoma
Giant Cell Astrocytoma
Central Neurocytoma
Choroid Plexus Papilloma
Meningioma
Ectopic Pinealoma
Epidermoid Tumor
Neuroepithelial Cysts

Primary CNS Lymphoma

Primary CNS lymphomas (PCNSL) comprises 3% of all intracranial neoplasms, and is the most common intracranial neoplasm in acquired immune deficiency syndrome (AIDS) patients. In such patients, it is linked with Epstein-Barr virus (EBV) infection. There is no surgical option in such cases, other than diagnostic biopsy. Chemotherapy and radiation may benefit immunocompetent patients, whereas with human immunodeficiency virus, the prognosis is poor.[4] The tumor behavior, and hence the imaging appearance, of CNS lymphoma is different in immunocompetent and immuncompromised patients.

In immunocompetent patients, lymphoma is mostly unifocal, with defined or lobulated margins. In more than 80% of cases, the lesion is close to the ventricular ependyma or to the meningeal surface. In 20-40% of cases there are multiple lesions. Involvement of basal ganglia, thalamus and corpus callosum is typical. Peri-lesional edema is usually present but less prominent than that in malignant gliomas or metastaes. There is no calcification or hemorrhage, and necrosis is rare. [4]

In patients with AIDS, lymphoma is most frequently multifocal. The lesions tend to be smaller, each with more irregular margins, and not necessarily in contact with the ependyma or meningeal surfaces. There is often more extensive perilesional edema, as well as possible hemorrhage and necrosis. With gadolinium, there is often peripheral rim enhancement.[3]

Lymphomas are hypercellular tumors with high nuclear-cytoplasmic ratio; therefore, PCNSL frequently show reduced diffusivity on DWI. (KP 11) Because intratumoral hemorrages are much more frequently seen in high grade gliomas, GRE and SWI sequences can help distinguish

PCNSL from high-grade gliomas.[29]

Hemangioblastoma

Hemangioblastoma is a benign tumor comprised of an overgrowth of capillaries, most commonly occurring in the posterior fossa. It may produce symptoms of increased intracranial pressure and cerebellar dysfunction. These tumors are WHO grade 1. They may be associated with von Hippel-Lindau (VHL) syndrome, in which case they occur throughout the CNS as well as the retina.[3] The cystic component of the tumor is T1 hypointense and T2 hyperintense; the solid, pial component is T1 isointense and slightly T2 hyperintense. Inside the solid component, there may be serpiginous flow-voids. Following contrast administration, there is a marked enhancement of the subpial nodule without enhancement of the cyst wall. In patients with known or suspected VHL syndrome, imaging of the entire spine as well as examination of the retina is necessary to assess for multiple lesions.[3]

Primitive neuroectodermal tumors (Medulloblastoma)

Primitive neuroectodermal tumors (PNETs), which were formerly called medulloblastomas, occur in children and young adults and arise from the primitive neuroectoderm of the cerebellar vermis in the fourth ventricle. They present with signs of increased intracranial pressure or cerebellar dysfunction. Imaging of the entire neuroaxis is important as these tumors can metastasize throughout the CNS.[4] This neoplasm is T1 iso- to hypo-intense and T2 isointense. The tumor shows avid enhancement with gadolinium in about 90% of cases. Due to high cellularity there may be restricted diffusion. MRS shows elevated choline and reduced NAA. Due to possible leptomeningeal metastasis, which occurrs in about 33% of patients at diagnosis,

the MR study should include brain and entire spine.[3]

Ganglioglioma

Ganglioglioma are slow growing tumors that occur in children and young adults, and that may undergo malignant transformation.[4] They are generally cystic with or without a solid component with variable degree of enhancement and calcifications.[1] Gangliogliomas are commonly found in patients with medically refractory seizures. They are usually cystic masses, rarely solid, with well-defined margins, T1 iso- to hypo-intense and T2 hyperintense. Calcifications appear as areas of hypointensity in T2 and T2*. With contrast, the enhancement pattern is heterogeneous, with various intensity, and nodular or multinodular morphology. The lesion is usually surrounded by mild vasogenic edema.[3]

Central Neurocytoma

Central neurocytomas are benign tumors arising within the lateral ventricles, with variable degrees of enhancement and with a cystic component. The solid component of the tumor is isointense to gray matter on T2 weighted images, and internal flow-voids may be seen. CT frequently demonstrates calcification adjacent to the region of the foramen of Monro. [4]

The solid components of the tumor are T1 isointense while the cystic components are hypointense; T2 signal is heterogeneous, predominantly hyperintense with a bubble-like appearance. Calcifications are hypointense on T2, and there may be flow voids from pathological vascularization. There is variable enhancement with gadolinium. There is a strong choline peak on MRS.[3]

Meningioma

Meningiomas are highly vascular, extra-axial tumors that are generally benign. They account for 20% of primary CNS neoplasms. They derive their blood supply primarily from the meningeal arteries with tumor capillaries that lack a blood brain barrier. Roughly 95% of meningiomas are supratentorial, but they can be found anywhere along the dura. Meningiomas are hypervascular on perfusion MRI with highly permeable capillaries.[3] (KP 12) Meningiomas can occur as a result of a mutation in chromosome 22, or as part of neurofibromatosis type II (NF2).(KP13) Symptoms are produced by compression of adjacent tissues by the tumor.[4] Seizures may occur due to its contact with the gray matter..

On MRI, most meningiomas uniformly and avidly enhance with contrast and may produce significant surounding edema. A heterogeneous pattern of enhancement in a meningioma and significant peritumoral edema are clues that the tumor is more aggressive. A dural tail may be observed and refers to enhancement of the meninges flanking the tumor. Meningiomas may also be accompanied by hyperostosis of overlying bone, or with cyst formation.[3]

Meningiomas are iso- or slightly hypo-intense to the cortex in T1 sequences, with variable signal on T2. At the periphery of the tumor, a dilatation of the subarachnoid spaces and invagination of the cortex can be seen due to the compressive and displacing effect of meningiomas.[7]
After contrast injection there is early enhancement, diffuse and marked with the exclusion of cystic or calcified components. Spectroscopy shows an alanine peak without NAA peak.[1]
MRI angiography documents the relationship between the meningioma and venous and arterial vascular structures. For surgical planning, it is important to identify infiltration or occlusion of

and adjacent venous sinus.

Hemangiopericytomas

Hemangiopericytoma is an aggressive extra-axial tumor arising from the pericytes of the meninges and accounting for less than one percent of all intracranial neoplasms. These tumors adhere to the dura, and so a dural tail may be present in addition to homogeneous enhancement, but no calcification is demonstrated on CT.[3] The mass is T1 isointense and heterogeneous, and T2 iso- to hyper-intense. Inside the tumor there may be tubular and serpiginous structures, which appear as flow-voids and result from extensive neo-vascularization. With contrast, there is marked and heterogeneous enhancement, due to the hypervacularity as well as possible areas of necrosis.[1]

CNS Metastatic Disease

Brain metastases outnumber primary neoplasms by at least 10 to 1, and occur in 20 to 40% of cancer patients. Subsequent median survival is less than 6 months.[30] It is estimated that up to 170,000 new cases of brain metastases occur in the United States each year .[31] The tumor cells spread via the bloodstream, therefore these lesions are more likely to be deposited at end-arterial territories, such as the cortical gray-white junction. Lung cancer accounts for 50% of all brain metastases, followed by breast cancer, melanoma, and renal cell carcinoma. Approximately one-third of patients with a metastatic brain lesion do not have a known primary malignancy.[4]

Metastases are variasble in appearance but are typically T1 iso- to hypo-intense, except if they are mucinous in which case they will be T1 hyperintense. They most often demonstrate heterogeneous T2hyperintensity. In some metastatic lung muco-epidermoid metastases, there is a

marked hypointense signal on T2. Generally metastatic lesions show no restricted diffusion. Peritumoral edema is shown as an area of hypointense signal on T1 and hyperintense on T2. After contrast injection, enhancement is variable in morphology and frequently ring-like, due to the presence of central necrosis. It is important to use a sufficient dose of gadolinium, late acquisition of images (at least 10 minutes) and to acquire thin slices (< 3mm) and/or volumetric acquisitions. One must pay close attention to subcortical micrometastases, which are sometimes difficult to differentiate from small pial vessels. There is a increased choline peak on spectroscopy with high values of rCBV at perfusion with increased permeability due to the presence of pathological neovascularization.

Metastatic lesions tend to be multiple, however solitary lesions do occur and can present diagnostic challenge. In such cases, perfusion MRI may be useful to detect peritumoral relative CBV. Specificaly, the T2-hyperintense region surrounding enhancing brain metastses represents pure vasogenic edema, while in glial tumors, this often represents a combination of vasogenic edema and infiltrative neoplastic cells. Perfusion MRI with rCBV map may help to differentiate pure vasogenic edema from infiltrative edema.[21]

Table 7. MRI Characteristics of Brain Tumors

Grade I Astrocytoma	→Cystic component is hypo intense on T1 and hyper intense on T2 →Solid component is isointense on T1 and hyper intense on T2 →Heterogeneous enhancement of solid component and cystic component's wall.
Grade II Astrocytomas	→Hypointense on T1 and hyperintense on T2 →Diffusion is not restricted →No enhancement (if present, relate to oligodendroglioma) →Bulging of gray and white matter →Spectroscopy shows mild NAA reduction and increased choline →Perfusion MRI shows normal or reduced CBV values
Grade III Astrocytomas	→Usually hypointense on T1 and heterogeneously hyper on T2 →Diffusion is not restricted →Enhancement in-homogeneously and can be absent as well →Perfusion MRI can be helpful to identify more aggressive lesions →Spectroscopy will show high choline peak with reduced NAA
Gliomatatosis cerebri (WHO Grade III)	→Hypointense on T1 and hyperintense on T2 →No restricted diffusion on DWI

	→Absent or minimal enhancement → Perfusion MRI shows low values of CBV →Spectroscopy marked increase in myo-inositol and reduced NAA
Grade IV Astrocytomas	→Hypointense on T1, hyperintense on T2, inhomogeneous due to necrotic areas →Marked perilesional vasogenic edema →No restricted diffusion on DWI →Enhancement is predominantly peripheral with ring appearance →Lesion extends far from the areas of enhancement →Newly formed pathological vessels seen as signal voids in T2 →Perfusion MRI shows marked increase of CBV inside solid component →Spectroscopy with a considerable increase of choline peak, marked reduction of NAA and the appearance of lactate and lipids, due to the presence of the necrotic areas
Oligodendrogliomas	→Hypointense on T1, heterogeneously hyperintense on T2 →Heterogeneous enhancement →Spectroscopy peaks may show high choline →Values of CBV perfusion cannot be used to assess the real grading, since they are high even in low-grade forms ("confounding factor")
Ependymomas	→Heterogeneous signal (necrosis, calcification, methemoglobin, hemosiderin, vascularity) →Iso-hypo intense in T1 and iso-hyper intense in T2 →Enhancement is usually dishomogeneous
CNS Lymphoma	→In immunocompetent patients →Iso-hypo intense compared to gray matter on T1 and T2 →Homogeneous enhancement →Due to high cellularity, diffusion is restricted →Spectroscopy shows increased choline and lipid peaks →Perfusion shows CBV values lower than expected in high-grade glial gliomas →In immunocompromised patients, signal may be heterogeneous because of hemorrhage and necrosis →Iso-hypo intense compared to gray matter on T1 and T2 →Mild perilesional edema →Predominantly peripheral enhancement with a necrotic core
Central Neurocytoma	→Solid components isointense on T1 while the cystic ones are hypointense →T2 is heterogeneous, predominantly hyperintense with bubble-like appearance →Variable enhancement with gadolinium →Strong choline peak on spectroscopy
Meningioma	→Iso-hypo intense to the cortex on T1, isointense to gray matter, but with variable signal on T2 →At periphery dilatation of the subarachnoid spaces and invagination of the cortex from compressive effect →After contrast injection there is early enhancement, diffuse and marked with the exclusion of cystic or calcified components →Spectroscopy shows alanine peak without NAA peak
CNS Metastasis	→Iso-hypo intense on T1 (except if mucinous) →Heterogeneously hyper intense on T2 (except muco-epidermoid type when marked hypointense) →Peritumoral edema seen as hypo intense signal on T1 and hyper intense on T2 →Enhancement is variable, more frequently ring-like →Peak of choline on spectroscopy → High values of CBV on perfusion with increased permeability due to the presence of pathological neovascularization

Conclusion:

MRI with gadolinium is the imaging study of choice in patients with a suspected CNS neoplasm. The contrast enhanced T_1- weighted images show breakdown of the blood-brain barrier. This is useful in characterizing a myriad of CNS pathologies and does not necessarily imply that the mass is malignant.[3] Stroke, abscesses, or demylination in addition to neoplasm will show contrast enhancement with gadolinium. Diffusion-weighted imaging is used to assess for

ischemia and hypercellularity within a lesion, and in particular can help differentiate abscesses from malignant gliomas.[2] Perfusion imaging provides quantitative estimates of cerebral blood volume (CBV), which reflects the microvasculature and angiogenesis associated with the mass. Higher rCBV tends to correlate well with the grade of neoplasm; lower values for low-grade astrocytomas versus higher values for anaplastic astrocytomas or glioblastomas. rCBV mapping can reveal differences in vascularity and may help differentiate radiation necrosis from recurrent tumor. Lastly, rCBV mapping, as well as MRS, can help to differentiate a solitary metastatic focus from a high grade glioma.[21]

The incorporation of biological and functional data into structural imaging will surely aid the clinician in both diagnostic and therapeutic aspects of neuro-oncologic care. It is likely that ongoing developments in imaging will enable personalized treatment protocols and ultimately, improved prognoses and survival.[28 32]

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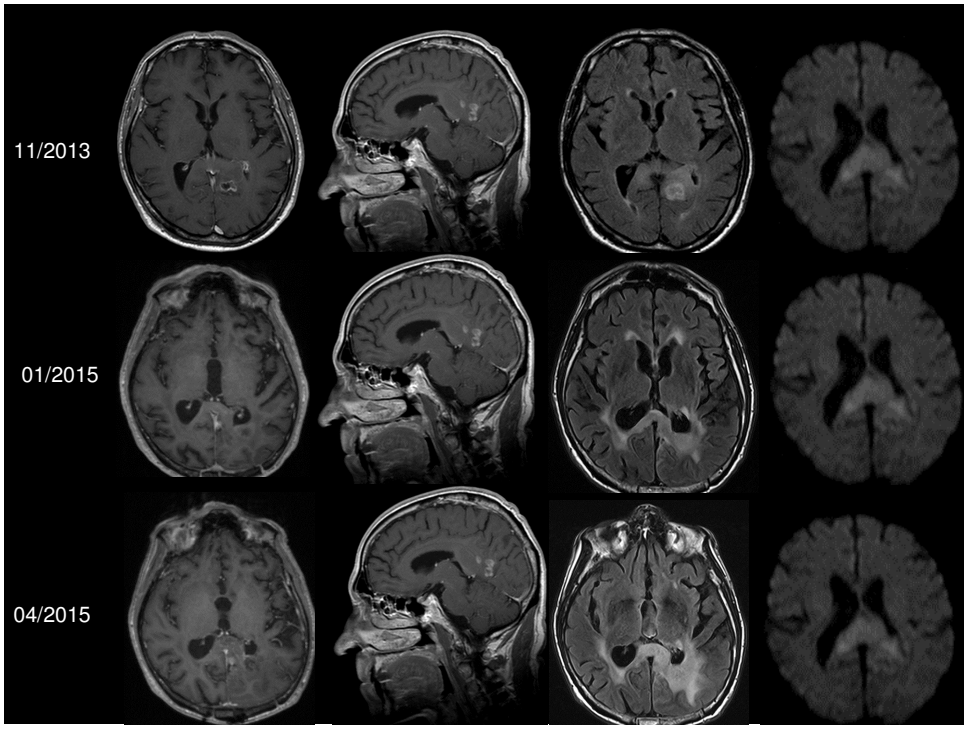
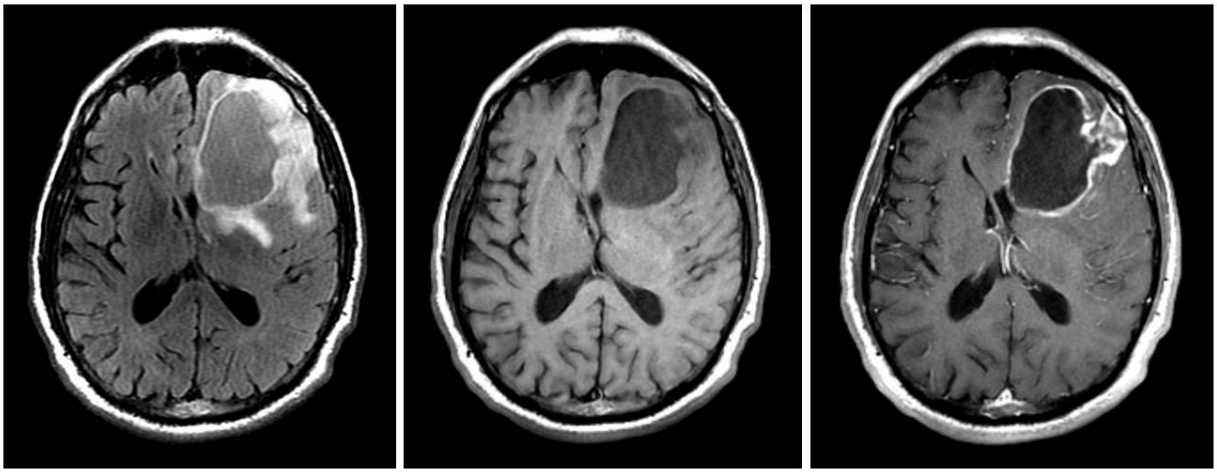


Anaplastic Astrocytoma

Left to right: T1W Post-contrast, Sagittal Post-contrast, Axial FLAIR, DWI

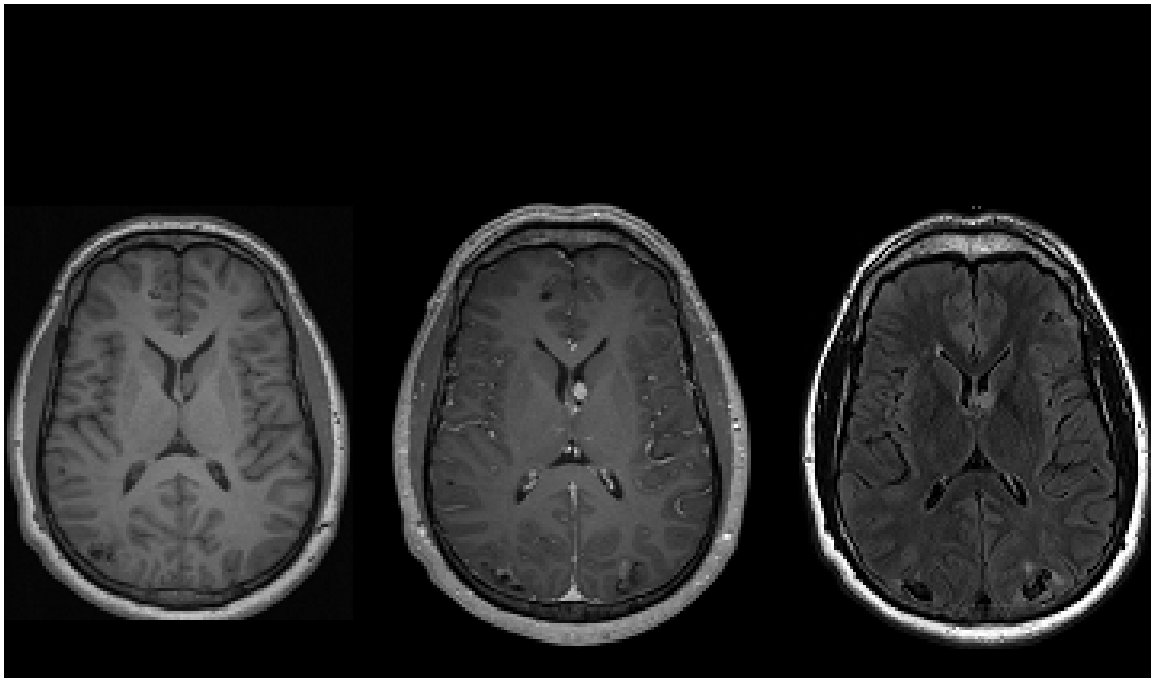
75-year-old right-handed male who presented with seven months of progressive cognitive issues with word finding and memory difficulties that worsened over four to five weeks. MRI of the brain with and without contrast demonstrated a mass lesion in the left occipital lobe extending to the splenium of the corpus callosum with prominent restrictive diffusion in areas of ring enhancement and hemorrhagic products. Biopsy confirmed anaplastic astrocytoma (WHO grade III). The patient underwent surgical resection in November, 2013 and completed treatment with Temodar and concurrent radiation therapy. The patient then started treatment with adjuvant Temodar. His condition continued to worsen and Avastin was added but discontinued due to side effects. A repeat MRI was performed in January, March, and April, 2015 demonstrating enlargement of the splenium of corpus callosum and increased prominence of the white matter changes, suggesting progression of disease. In March, continued treatment versus hospice was discussed and family opted for ongoing treatment. Avastin was administered IV every two weeks and metronomic dosing of Temozolomide was initiated. In May 2015, the patient opted for hospice care and passed away shortly thereafter.

(A).Prominent areas of T1 hypointensity and T2-FLAIR hyperintensity are seen that have some heterogeneity of signal. (B) Following contrast administration, a mixed pattern is seen with subtle linear and curvilinear areas of enhancement adjacent to the posterior horn of the left lateral ventricle extending into posterior temporoparietal white matter and extending into the splenium, which is increased in size. (C) Prominent areas T2-FLAIR hyperintenities noted throughout the white matter in both cerebral hemispheres. (D) DWI demonstrates restricted diffusion in these regions indicative of hypercellularity.



Glioblastoma

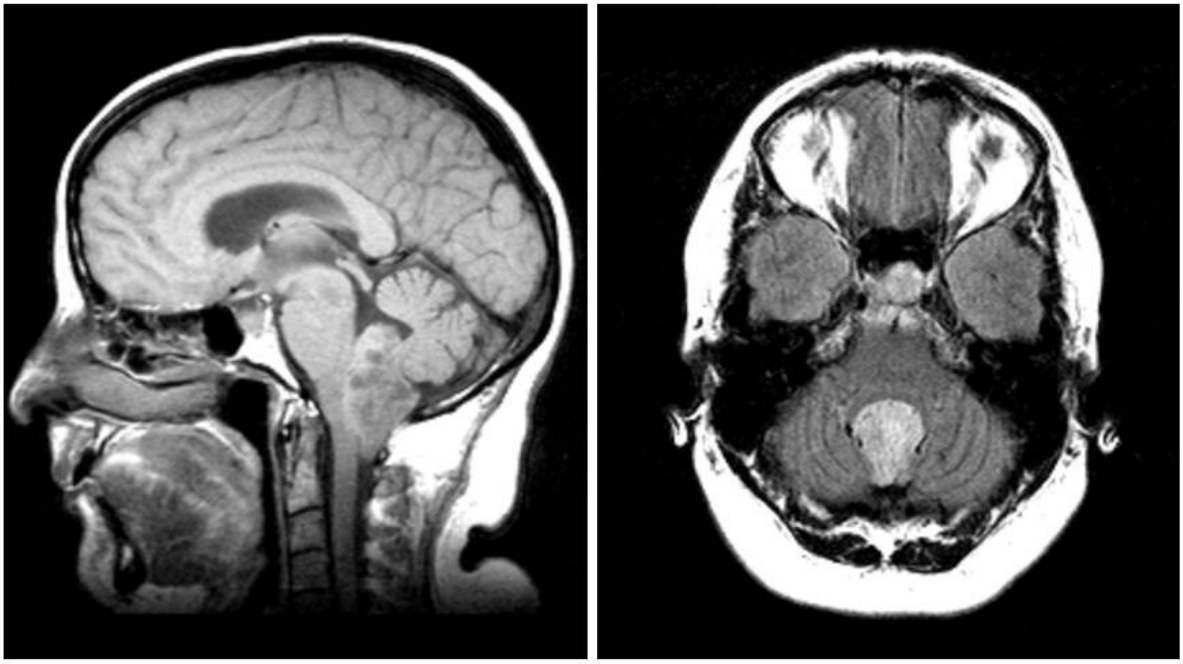
60 year old male with headaches and recent personality changes presented with altered mental status. A. Axial FLAIR image of the brain showing a large left frontal nodular and cystic mass with surrounding edema. The overlying cortical sulci and underlying frontal horn of the left lateral ventricle are effaced and there is rightward shift of midline structures with subfalcine herniation of the genu of the corpus callosum (not shown). Axial T1 pre- (B) and post contrast (C) images show an irregular rim of enhancement. The internal contents of the cyst contain necrotic debris and are non-enhancing.



Subependymal giant cell astrocytoma (SEGA)

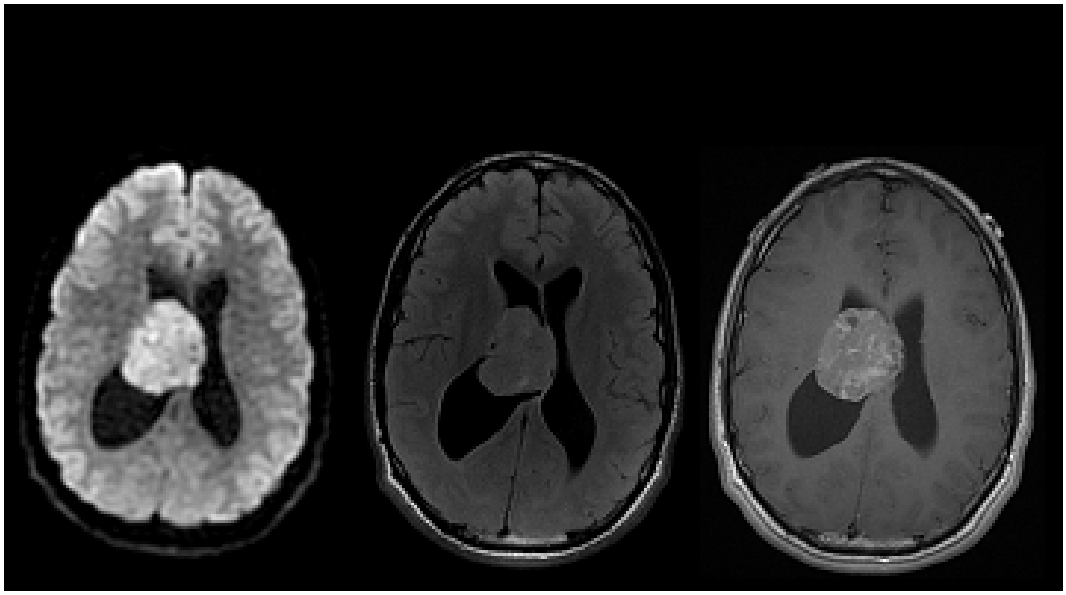
A 30 year old male with a history of epilepsy, tuberous sclerosis, obsessive-compulsive disorder, and neurofibromatosis.

MRI of the brain demonstrated numerous cortical-based T2 hyperintense lesions supratentorially bilaterally, many of which also exhibited prominent calcification. These are felt to be representative cortical tubers/hamartomas consistent with the history of tuberous sclerosis. The mass lesion, seen above, within the left lateral ventricle anteriorly at the level of the foramen of Monro, exhibit intense contrast enhancement, but no obstruction of CSF flow. In view of the patient's history this likely represents subependymal giant cell astrocytoma (SEGA).



Ependymoma

A. Sagittal T1 image of the brain shows a somewhat heterogeneously hypointense midline expansile mass extending inferiorly from the fourth ventricle into the foramen magnum. B. Axial T2-FLAIR image shows the hyperintense mass within the fourth ventricle.

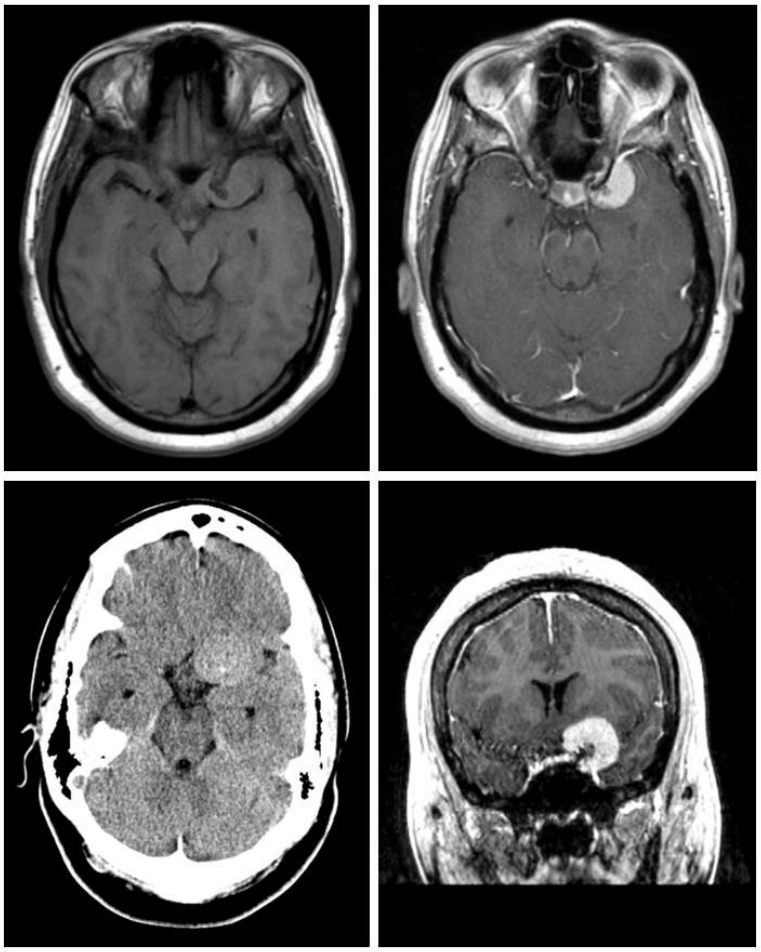


Central Neurocytoma

17-year-old young man, who was complaining of headaches for a period of 6 weeks. There was no nausea, vomiting; the headaches were continuous and made worse by changes in position, coughing, and sneezing.

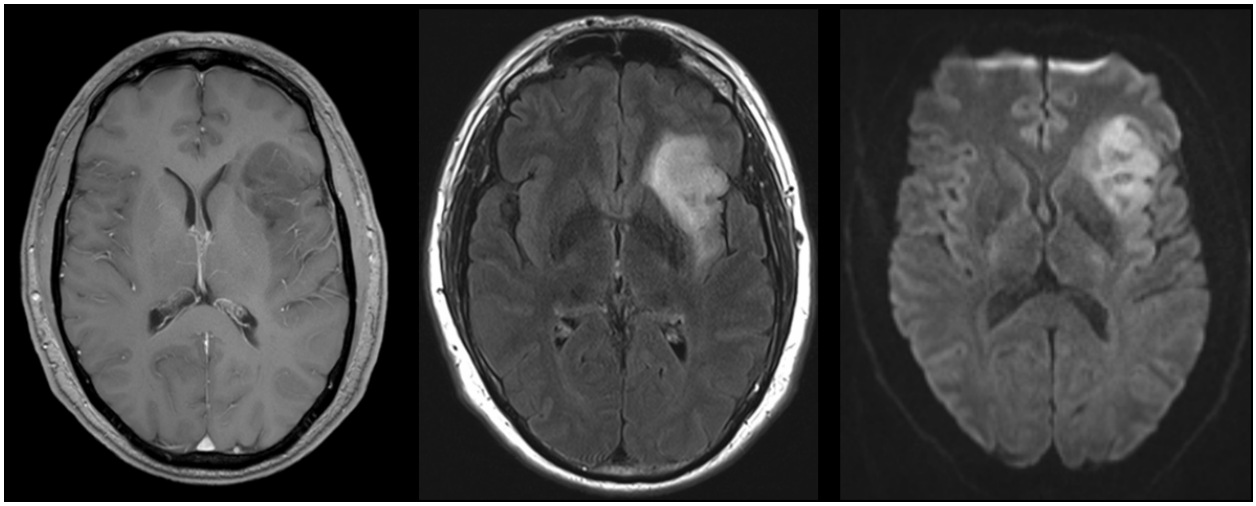
On neuroimaging was found to have an intraventricular mass on the right and underwent resection on April, 2012 after which he became asymptomatic.

Tumor was an atypical neurocytoma. The microscopic evaluation of the tumor showed delicate vascular subdivisions, granular. It also had philic cytoplasm, Homer-Wright-type rosettes, perivascular pseudorosettes. No calcifications were noted. It was synaptophysin positive. Tumor cells were negative for EMA, P53. MIB fraction index was 4%. There was no necrosis.



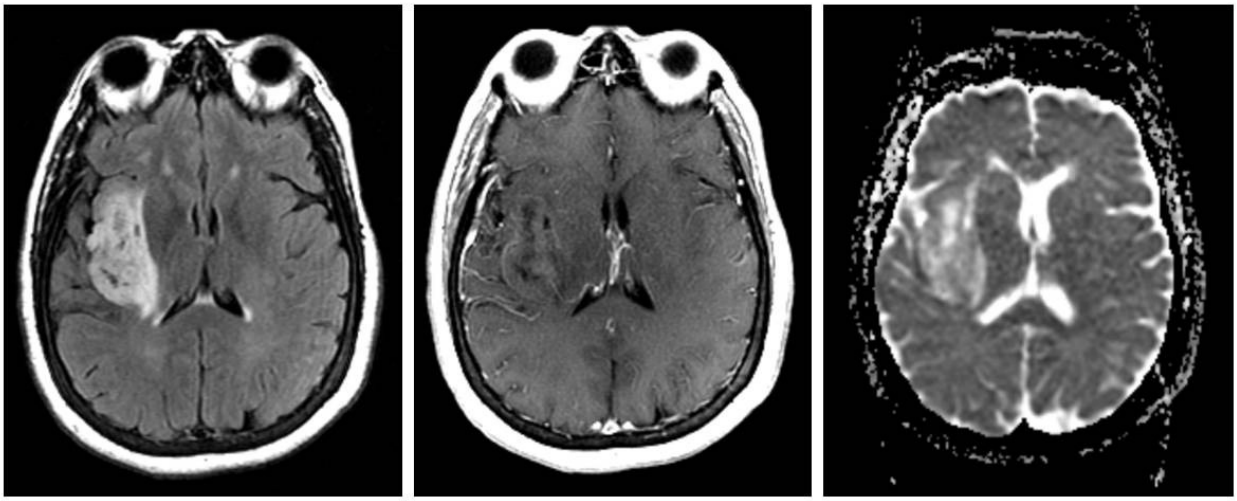
Meningioma

50 year old male with seizures found to have this extraaxial mass on MRI with a dural tail. Axial T1 (A) and T1-postcontrast (B) images of the brain showing an isointense and avidly enhancing extraaxial mass arising from the dura overlying the left cavernous sinus. C. Axial CT shows mild hyperattenuation (hyperdensity) of the lesion relative to the surrounding brain. D. Coronal T1-post contrast image further demonstrates the contiguity of the mass with the dura overlying the left cavernous sinus.



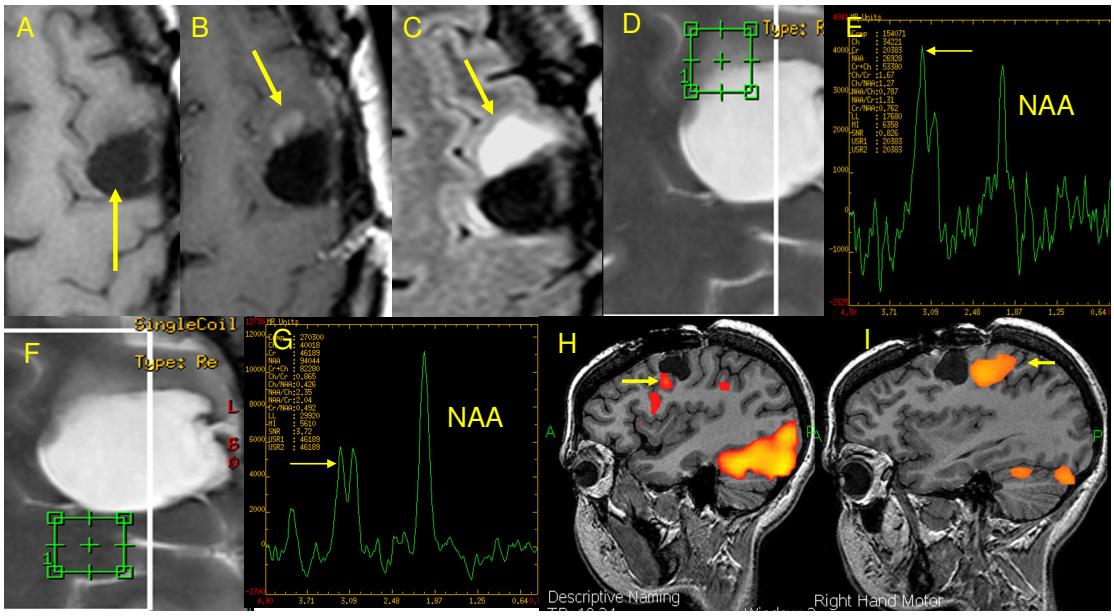
Oligodendroglioma

56-year-old male with an intermittent numbness and shaking sensation of the right hand with intermittent episodes of unresponsiveness for two months.
Biopsy confirmed a diagnosis, with WHO grade 2 oligodendroglioma in the left insular area. Due to the high MIB-index, which was 11% focally, the patient underwent concurrent chemoradiation followed by temozolomide treatment.



Oligoastrocytoma.

Axial T2-FLAIR image of the brain shows a region of abnormal hyperintensity centered within the right insula. There is minimal mass effect on surrounding structures. B. Axial T1-postcontrast image showing minimal enhancement of the lesion. C. apparent diffusion coefficient (ADC) map shows no hypointensity and therefore no evidence of hypercellularity.



A 31-year-old female operated one year ago for an oligoastrocytoma located in the superior aspect of the left middle frontal gyrus. On a 3.0 Tesla magnet a routine follow-up T1WI image (A) confirms a postoperative cavity (arrow), although contrast T1WI (B) shows a new area of enhancement just anterior to the cavity (arrow). FLAIR (C) shows a enlarging area of hyperintensity or long T2(arrow). Pre-operative MRS (D,E) shows decrease NAA and increase in choline peak (arrow) in the area of progression, consistent with tumor recurrence. When the voxel is placed posterior to the cavity, the MRS is normal (F,G). Functional MRI in the sagittal plane using descriptive naming (H) indicates activation in the left premotor cortex just below the enhancing lesion (arrow). For right hand activation (I) was found adjacent to the posterior margin of the post-op cyst that was normal on MRS. These test results aided the neurosurgeon in the resection of the enhancing lesion and adjacent T2W hyperintensities without post-operative residual neurologic deficits. Pathology was consistent with an anaplastic astrocytoma.