Dermoid, Epidermoid, Cysts, Lipoma

Last updated: September 13, 2019

Dermoid, Epidermoid, Cysts, Lipoma

- Benign dysraphic malformations (dysgenesis) at vestigial stage of development. *Traditionally, such disorders have been described as primary failure of neurulation (later stage in embryogenesis than gastulation).
- Primary disruption of tissues derived from one or more of three germ cell layers: 
  - DERMID AND EPIDERMIOS CYSTS, DERMAL SINUS TRACTS – surface ectoderm malformation.
  - NEURENTERIC CYSTS, RATIHE CEYSTS, COLLOID CYSTOS – all the same just different location – endodermal malformation.
- Frequently associated with mesodermal malformations (esp. vertebrae - hemivertebrae, absent or fused vertebrae, butterfly vertebrae, midline bony spurs).
- Can manifest in patients of any age.
- Clinically onset is usually insidious; occasionally cyst rupture and spillage of irritative contents into subarachnoid space – acute meningeal symptoms or increased ICP.
- Treatment of each of these entities is primarily surgical; radiotherapy and chemotherapy have little to offer.

Neureneretic cyst

- Normal embryologic transitory communication between Neural tube (neurenteric canal), notochordal canal, and gut endoderm. Canal persistence – paravertebral neurenteric cyst(s) or fistula(s).

Pathology

- Benign slow-growing cysts
- Location (intra and extra axial): 
  A. Most commonly – spinal canal (particularly in low cervical or high thoracic region).
  B. Intracranial (most often posterior fossa – pontomedullary region, cerebellopontine angle, parasellar area, and craniocervical junction)
- Cyst wall is composed of well-differentiated cuboidal, columnar, or ciliated epithelium, with or without goblet cells or microvilli. 
- Cyst fluid: clear and colorless, milky, yellowish, gelatinous, xanthochromic, and blackish and viscous.
- Epithelium may resemble intestinal or respiratory type, and there may be smooth muscle in underlying fibrovascular tissue.
- Cyst fluid: often parallel signal intensity of CSF, may show increased intensity in cyst and some parenchymal hyperintensity around tumor, consistent with edema.
- Intracranial cyst rupture and spillage of irritative contents into subarachnoid space – acute meningeal symptoms or increased ICP.
- Treatment of each of these entities is primarily surgical; radiotherapy and chemotherapy have little to offer.

Clinical findings

- Slow and insidious compression of adjacent spinal cord
- Persistent fistula (between enteric structures and these cysts within CNS) – recurrent aseptic meningitis.

Imaging

Differential – from simple arachnoid cysts to complex teratomas.

MRI: 
- Variable and heterogeneous cyst signal (often parallel signal intensity of CSF), no enhancement (some variable wall enhancement may be present)
- Reports of TI signal changes in these tumors, resulting from changes in protein concentration or hemorrhage.
- Cysts have tendency to insinuate between surrounding structures.

A. T1-BRE - hypointense suprasellar mass.
B. Hypointense midline suprasellar lesion.
C. T1-MRI – cystic lesion
D. Contrast-enhanced T1-MRI – rim of cyst wall enhancement and region of intracyst enhancement.
E. T2-MRI – hyperintense signal of cyst contents that follows signal change in cerebrospinal fluid.
F. FLAIR MRI – increased intensity in cyst and some parenchymal hyperintensity around tumor, consistent with edema.
G. Protein density axial MRI – cyst contents somewhat heterogeneous.
H. Gradient echo axial MRI – cyst heterogeneity.
TREATMENT

- primarily surgical - complete resection (not always feasible given difficulty of completely removing cyst wall at locations where it adheres to adjacent structures).
  - simple cyst aspiration, cyst wall marsupialization, or creation of cyst-subarachnoid shunt, are considered in difficult cases.
  - aseptic meningitis has not been a problem one might expect.

- recurrence is possible even after gross total resection → reoperation.
- radiotherapy and chemotherapy have little to offer; whether there may be some role for radiosurgery in treatment of residual neuroenteric cysts remains to be seen.

DERMOID tumor, EPIDERMOID tumor - benign inclusion cysts (not true neoplasms!) composed of ectodermal elements.

dif. CHOLESTEATOMA → see p. Ear38

ETIOLOGY - congenital (embryonic remnants) - inclusion of ectodermal epithelial elements:

a) during 3-5th embryonic weeks when neural tube closes at midline → midline tumors (esp. DERMODS)

b) during formation of secondary cerebral vesicles → lateral tumors (esp. EPIDERMODS).

N.B. EPIDERMODS also may be acquired – due to trauma, frequently from lumbar puncture (epithelial cells deposited within spinal canal, mostly historical when spinal needles didn’t have stylet).

PATHTHOLGY - similar appearances and developmental origins;
- both contain stratified squamous epithelium found in skin.
- centrally, both contain desquamated epithelial keratin and some lipid material (cyst fluid may contain cholesterol crystals).
- external surface is smooth, lobulated; EPIDERMOID has pearly appearance (“pearly tumors” or “keratin pearls” in wall) due to capsule of stratified squamous epithelium.

- histo - cell nuclei among keratin (vs. pure keratin s. “wet keratin” in craniopharyngioma)

- EPIERMODS have outer connective tissue capsule and are lined with stratified squamous epithelium (i.e. composed of ectodermal remnants).

- DERMODS have outer connective tissue capsule and are lined with stratified squamous epithelium, which also contains hair follicles, sebaceous glands, and sweat glands (i.e. composed of ectodermal and mesodermal remnants).

- expand slowly over many years due to central accumulation of epithelial debris and glandular secretions – predictable linear growth due to accumulation of keratin (vs. tumors – grow exponentially due to cell multiplication).
- recurrent usually simply; EPIERMODS calcify uncommonly (but when it occurs, it is feature that helps in distinguishing from arachnoid cysts).
- malignant transformation is rare.

LOCATION - sites of epithelial deposition can occur anywhere between neural tube and overlying skin surface (depending on stage of intratireine development at which they arise, they can lie within ventricular system, brain parenchyma, subarachnoid space, bones of skull, or even extracranially).
**DERMOID**

- **DERMOID (INTRACRANIAL)** - most commonly intradural, midline; 2/3 in posterior fossa (extradural, vermian, or within 4th ventricle); also suprasellar region, subfrontal areas, other sites – scalp (commonest location in childhood), skull, orbit, nasal, oral cavity, neck.

- **DERMOID (SPINAL)** - most commonly near thoracolumbar junction, tends to involve conus medullaris and cauda equina.

**DIAGNOSIS**

- Absence of edema is characteristic!

**Plain radiographs** – local bone expansion or erosion, lytic lesions with thin sclerotic margin.

**CT** – well-circumscribed, unilocular cystic mass; calcifications in tumor wall.

- Contrast enhancement is uncommon! (**EPIDERMOID** wall may sometimes enhance).

**DERMOID** generally do not produce clinical symptoms until 2nd to 3rd decade of life (i.e. > 20 yrs) (rarely more slowly than dermoid) – 40-50 yrs.

**Symptoms & signs** are associated with slowly progressing mass/pressure effect (seizures, diabetes insipidus, hypopituitarism, etc.).

- blockage of CSF flow occurs only rarely!

- cyst rupture → giant granulomatous chemical meningitis (rarely results in infarction from vasospasm).

- associated dermal sinus tracts / dimples are common: Any infant with dermal sinus tract → neuroradiological evaluation!

- congenital lumbar dermal sinus may terminate in **EPIDERMOID** (less frequently **DERMOID**) within or near conus medullaris or cauda equina; often associated with spinal dysraphism and vertebral abnormalities.

- congenital nasal dermal sinus may be associated with **DERMOID** or **EPIDERMOID**.

- if associated dermal sinus tract becomes infected → recurrent bacterial meningitis.

**Angiography** – avascular mass.

Prenatal diagnosis with **ultrasound** (and resection shortly after birth) are now possible.

**Supratentorial dermoid**

A) contrast T1 – high signal intensity suprasellar mass extending along planum sphenoidale.

B) contrast T1 with fat saturation – small amount of enhancement along peripheral as well as central portion of lesion (may be slightly heterogeneous due to additional cystic components).

**Lesion characteristics** similar to fat (glandular secretions) – midline mass hyperintense on both T1 and T2 – unique tumor!

- chemical-shift artifact is often present on T2 images as markedly hypointense band posterior to fat-fluid interface.

**EPIDERMOID** – characteristics similar to CSF – variably hypointense on T1 and variably hyperintense on T2 – mimics arachnoid cyst (H: DWI – epidermoid has diffusion restriction).

**MRI**

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**DERMOID, EPIDERMOID, CYSTS, LIPOMA**

**Dermoid (nonenhanced CT)** - large, well-circumscribed low-attenuating cystic lesion in right temporal lobe lateral to cranial midline; peripheral marginal calcification; no erosion in adjacent bone of sella.

**Same dermoid (contrast CT)** - partial marginal enhancement; attenuation degree in center of lesion consistent with fat.

**Same dermoid (T1)** - hyperintense signal in lesion; multiple small hyperintense foci along sulci of right temporal lobe (represent fat droplets in subarachnoid space from focal dermoid rupture).

**Same dermoid (T1 with contrast)** - hyperintense lesion (hyperintensity is due to short T1 of fat); multiple hyperintense foci (fat droplets) in subarachnoid spaces; mild midline septal shift to left; chemical-shift artifact at superior marginal surface of lesion.

**Epidermoid (T1 with contrast)** - suprasellar, prepontine, and interpeduncular location of nonenhancing tumor (signal intensity similar to CSF).

**Epidermoid (A: T2-weighted, B: T1-weighted MRI)** - Sylvian fissure is filled by mass which extends into chiasmatic cistern and encases left internal carotid artery termination (arrowhead); signal is similar to CSF on T2, but slightly higher than CSF on T1 (white arrow).

**Epidermoid (A-T2 MRI – large homogeneous mass, which is slightly higher in signal than CSF, fills right cerebellopontine angle)**

**Epidermoid (B-T1 MRI – lesion is again noted to be hyperintense to cerebrospinal fluid).**

**TREATMENT**

- Complete surgical excision is curative.
  - Epidermoids do not invade but interdigitate around vital structures - compounding surgical removal.
  - Avoid spilling of contents → chemical meningitis (15%); prophylaxis - irrigation with dexamethasone solution intraop + 2 weeks of oral dexamethasone (↑↑↑risk of infection).
  - Associated dermal sinuses should be removed completely.

Chemotherapy and radiotherapy are not useful.

**EPIDERMOID CYSTS (from WHO manual)**

- Benign ectopic inclusions of epithelial cells during gastrulation (i.e. malformations of surface ectoderm) → secondary disruption of neural tube closure (i.e. dysraphism is secondary).

**PATHOLOGY**

- Location – tend to be off midline.
  - **SPINE** - intradural extramedullary (rarely intramedullary)
  - **CRANIAL** (0.2% to 1.8% of all intracranial tumors):
    - **intradural** (usually extra-axial) - cerebellopontine angle (may extend into Meckel's cave) or paravertebral cisterns
    - **extradural** (usually arising in diploic space of calvaria)
- Thin capsule of stratified, keratinized squamous epithelium.
- Cyst contains accumulation of desquamated epithelial cells, keratin, and cholesterol (characteristic pearly flakes).
- Malignant transformation is rare, but squamous cell carcinoma has been reported (15 cases with leptomeningeal dissemination of squamous cell carcinoma); malignant melanoma has also been reported in temporal lobe epidermoid.
- Immunohistochemical staining positive for carbohydrate antigen CA19-9* (also detected in serum - can be used to evaluate for tumor recurrence or progression).
**CLINICAL FINDINGS**

- reported from infancy to adulthood + incidental findings at autopsy.
- average age at detection is 35-40 years.
- female predominance
- grow linearly (similar to normal skin) → insidious onset (median duration of symptoms 4-14 years).
- picture of acute meningitis may indicate epidermoid cyst rupture.
- spinal epidemoid tumors are frequently associated with vertebral anomalies.

**EXTRADURAL LESIONS** - manifest as local mass.

**INTRADURAL TUMORS** - headache (because of common parasellar location), visual disturbance, and to lesser extent, hypothalamic alterations.

- tumors in middle fossa grow quite insidiously and are often asymptomatic.
- tumors in cerebellopontine angle may cause ataxia, dizziness, or cranial nerve deficits.

**IMAGING**

**CRANIAL XR** - for intradiploic tumors - typically in cranial vault (may also occur in orbital region)
- lytic erosion of skull, sharply delineated sclerotic edge and scalloped margins on plain radiographs.
- inner table of skull is usually destroyed and outer table thinned.

**CT** - homogenous nonenhancing hypodense lesion in subarachnoid space without surrounding edema.
- occasionally, high density masses (“white epidermoid”) - difficult diagnosis.

**Differential diagnosis**
- arachnoid cyst, Rathke's cleft cyst, craniopharyngioma.
- differentiation from ARACHNOID CYST:
  1) diffusion restriction on DWI (vs. arachnoid cysts – no diffusion restriction)
  2) more fat density than CSF density
  3) extend into subarachnoid space and enlarge it (vs. arachnoid cysts cause more focal mass effect).

**MRI** - hypointense to hypointense, heterogeneous, multiloculated.
- T1 hypointensity + T2 hyperintensity
- some tumors show rim enhancement
- no surrounding edema
- hydrocephalus is rare
- DW imaging is superior to other types of MRI sequencing in delineating borders of epidermoid cysts.

A. Contrast-enhanced, T1-MRI - hypointense mass without enhancing components in midline posterior fossa. The mass is compressing brainstem and obstructing egress of cerebrospinal fluid from fourth ventricle, thereby causing hydrocephalus.

B. More lateral extent of this tumor.

C. Mass insinuating between normal posterior fossa structures and extending to involve both right cerebellopontine angle and midline structures.

D. Contrast-enhanced, FLAIR MRI - marked heterogeneity within cyst and discontinuous rim of cyst wall enhancement.

**TREATMENT**

- symptomatic patients benefit from surgery (technically difficult because of tumor adhesions)
- tumor is well demarcated, with smooth, hypovascular capsule
- primary intracapsular debulking (CUSA is extremely useful) → removal of capsule (fragments of capsule adherent to important structures are left when necessary to avoid neural or vascular injury).
  - although subtotal resection increases risk for recurrence, slow growth of epidermoid cysts makes this less problematic.
  - capsule should not be removed from intramedullary cysts because of risk of causing neurological deficits or preoperative cyst rupture (radiographic finding of small fat globules in subarachnoid and intraventricular spaces)
- Meningitis → steroids.
- no established role for radiotherapy or chemotherapy for residual / recurrent epidermoid cysts.

**DERMIEID CYSTS (from WHO manual)**

- manifest at earlier age than epidermoids and have shorter duration of symptoms (duration of symptoms average 8.5 years, vs. 16 in epidermoids).
- female predominance
- denser tumors than epidermoids → more focal mass effect - patients are initially seen at younger age (average age at diagnosis - 15 years, vs. 35-40 in epidermoids).
- tend to occur at MIDEline (when extradural, may arise at anterior fontanelle).
- reported in association with Klippel-Feil syndrome.

**PATHOLOGY**

- positive for CA19-9* in many cases (serum CA19-9 levels tend to be higher than in epidermoid cysts - means of follow-up for residual or recurrent disease). *tumor marker for pancreatic cancer
- dermoids contain elements of dermis (hair and hair follicles + apocrine, sebaceous, or sweat glands)
- epithelial cell lining may be less differentiated (than in epidermoids)

Squamous epithelial cyst with adherent keratin debris, consistent with dermoid cyst. H & E, magnification x40.

**CLINICAL FINDINGS**

- local neural deficits, headache, or meningitis.
- dermoid cyst rupture - previously thought to be uniformly fatal.

**IMAGING**

- CT - hypodense and avascular, no contrast enhancement.
- extradural cranial lesion shows typical bony erosive changes seen with epidermoids.
- MRI - more signal heterogeneity than epidermoids; high fat content, high signal on both T1- and T2- weighted images – unique tumor!
- nonenhancing (rare cases of capsule contrast enhancement and even enhancing mural nodule should raise suspicion for neoplasm!).
- more solid (than epidermoid tumors) - less likely to grow between neurovascular structures and tend to demonstrate more of local mass effect.
- edema is lacking.
- dermoid cyst rupture → fat droplets may be seen throughout subarachnoid or intraventricular space, localized dissemination in sulci causing widening, perhaps contained by pia or inflammatory tissue; hydrocephalus secondary to CSF obstruction by fat droplets.

**TREATMENT**

- surgical extirpation is treatment of choice - less problematic (than epidermoid tumors) because of firmer consistency, adherence of tumor capsule to vascular and neural structures → more conservative surgical approach.
- complete excision decreases risk for both postoperative chemical meningitis and tumor recurrence.
- tumor recurrence has been reported → reoperate.
- no current role for radiotherapy or chemotherapy.

**COLLOID CYSTS**

- congenital benign tumor that can cause sudden death because of their location (almost always found in 3rd ventricle → obstructive hydrocephalus).
- 0.5-1% of all primary brain tumors (15-20% of all intraventricular masses).

**ETIOLOGY**

- possible sources:
  - a) in 1930, Sjovall hypothesized that colloid cysts are remnants of PARAPHYSIS (embryonic midline structure within diencephalic roof immediately rostral to telencephalic border, in posterior lip of foramen of Monro); - cells of paraphysis are similar to those found in colloid cysts (i.e. low columnar epithelial cells without cilia or blepharoplasts) - colloid cysts were called paraphysial cysts for 50 years.
  - b) diencephalic ependyma
c  - c) invagination of neuroepithelium of ventricle
d  - respiratory epithelium of endodermal origin.

**PATHOLOGY**

- gray in anterior superior portion of 3rd ventricle between fornices, immediately dorsal to Mono's foramen;
- also have been reported to arise in septum pellucidum, 4th ventricle, sella turcica.
- attached to roof of 3rd ventricle (and frequently to choroid plexus).
- gross appearance of small white bull
- lined with simple or pseudostratified cuboidal or low columnar ciliated epithelial cells (PAS-positive; stain positively for S100 and negatively for glial fibrillary acidic protein, vimentin, and neurofilament).
- epithelial lining secretes mucinous fluid (greenish, of variable viscosity and can be rubbery) → cyst enlargement.

**CLINICAL FEATURES**

- classic intermittent obstructive hydrocephalus with paroxysmal headache associated with changing head position (large cyst obstructing Monro foramen).
- Positronial headache:
  - usually present in age 20-50 yrs (youngest reported case - 2-month-old infant).
  - other reported symptoms (sometimes related to changes in posture).

**TREATMENT**

- complete excision decreases risk for both p ...
The transcortical approach carries increased incidence of epilepsy. If it is attached to either thalamostriate vein, it must be decompressed, completely remove it in order to prevent recurrence. Leaving small portion of cyst behind may be necessary if it will be impossible to obtain a clear view of fornix. Avoid excessive retraction of walls of lateral ventricle because genu of internal capsule is in subependyma. If cyst contents are aspirated, internally decompressing walls of cyst. The cyst should be reaspirated after cyst protein density visible on T1 and hypointense on T2. N.B. prevention of sudden death (incidence appears to be low) has been reported; may not correlate to tumor size, degree of ventricular dilatation, or duration of symptoms; suggested mechanisms: a) acute hydrocephalus b) hypothalamic dysfunction

**TREATMENT - STRATEGY**

Immediate attention to hydrocephalus!

**Strategy**

a) large cyst & hydrocephalus → surgery.
   - if patient is too ill → bilateral CSF diversion or unilaterally septostomy
b) small cyst, large ventricles, few or no symptoms → observation with serial MRIs.
c) small cysts and normal-sized ventricles → observation.

N.B. prevention of sudden death is not indication for surgery in asymptomatic patients with small cysts and no hydrocephalus!

- explain to patient that cyst stretches fornix - look for preoperative short memory deficits – those may worsen postop if fornix is further violated surgically.

**TREATMENT - SURGERY**

a) endoscopic-transventricular approach – only if hydrocephalus is present
b) transcortical-transventricular approach – only if hydrocephalus is present
c) transcallosal approach – see p. Op340

Surgical approaches.

- **Transcortical approach**
  
  The transcortical approach involves making corticectomy over middle frontal gyri and proceeding to frontal horn of lateral ventricle. Intraoperative ultrasonography may aid in approach to ventricle. The Monro foramen is visualized at convergence of septal veins, thalamostriate vein, and choroid plexus. The fornix arches over supracallosal anterior margins of foramen. Avoiding fornix is important because unilateral fornix damage has been associated with amnesia. The cyst should be readily visualized through foramen. The cyst is punctured and contents are aspirated, internally decompressing walls of cyst. Avoid excessive retraction of walls of lateral ventricle because genu of internal capsule is in subependyma. Other concerns include damaging thalamostriate veins, which can result in basal ganglia damage. After cyst has been associated with amnesia. The cyst should be reaspirated after cyst protein density visible on T1 and hypointense on T2.

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Intraoperative photograph showing removal of cyst, leaving dilated Monro foramen. The third ventricle can be seen through opening.

Endoscopic approach
The endoscopic approach is same as transcortical approach, with exception that former is accomplished through burr hole. The cyst is punctured and aspirated through working channels of endoscope. This complication may be secondary to spillage of cyst contents or to bleeding during surgery. A ventricular catheter may be placed intraoperatively to safeguard against ventricular dilatation.

Postoperative Follow-up
Hydrocephalus may develop despite cyst removal; H: periodic CT.
Cyst may recur if partially excised!

ARACHNOID CYSTS

References
Mark S. Greenberg “Handbook of Neurosurgery” 8th ed. (2016), ch. 15.1

Features
• congenital - splitting of arachnoid membrane (thus they are technically intra-arachnoid cysts), contain fluid identical to CSF
• arise anywhere on brain surface or in spinal canal.
• almost all occur in relation to an arachnoid cistern (exception: intrasellar – only one that is extradural).
• incidence: 5 per 1000 in autopsy series (~ 1% of intracranial masses).
• male:female ratio = 4:1.
• bilateral arachnoid cysts may occur in Hurler syndrome (a mucopolysaccharidosis).
• lined with meningothelial cells positive for epithelial membrane antigen (EMA) and negative for carcinoembryonic antigen (CEA) - two types:
  1) “simple arachnoid cysts”: arachnoid lining with cells that appear to be capable of active CSF secretion (middle fossa cysts seem to be exclusively of this type).
  2) cysts with more complex lining which may also contain neuroglia, ependyma, and other tissue types
• do not communicate with the ventricles or subarachnoid space.
• incised or have septations.
• incidence of growth – about 2% (some grow to remarkable size).
• smooth surface (vs. EPIDERMOIDS – cauliflower-like deep clefts).
• may hemorrhage (into cyst or subdural compartment): middle fossa cysts are notorious for hemorrhage due to tearing of bridging veins – some sports organizations do not allow participation in contact sports for these patients.
• obsolete term - “temporal lobe agenesis syndrome” - findings with middle cranial fossa ACs; however, the brain volumes on each side are actually the same (bone expansion and brain matter account for the parenchyma that appears to be replaced by the AC).
• clinically: majority are incidental findings; most commonly symptomatic location – suprasellar:
  a) Hydrocephalus (probably due to compression of the third ventricle)
  b) endocrine symptoms: occurs in up to 60% (incl. precocious puberty)
  c) head bobbing (the so-called “bobble-head doll syndrome” - considered suggestive of suprasellar cysts, but occurs in as few as 10%).
  d) visual impairment
• imaging – menancing, extraparenchymal cyst with characteristics exactly mimicking CSF:
  – remodeling of bone (chronic nature).
  – no diffusion restriction on DW-MRI (vs. EPIDERMOID - diffusion restriction).
  – evaluation with CSF contrast or flow studies (cisternograms, ventriculograms) are only occasionally necessary for midline suprasellar and posterior fossa cysts.
  – never calcify!
CT: Visualization of sphenoid fissure arachnoidal cysts.

Type I - small, biacous, located in sphenoid sinus or trigone.

Type II - large, fluid-filled structure expands left cerebellopontine angle cistern (arrowheads); note elongation and thinning of cranial nerves VII and VIII (white arrow).

**TREATMENT**

- asymptomatic: best left alone regardless of size or location (in adults, a single follow-up imaging in 6–8 months is adequate to rule out any increase in size; children need to be followed until adulthood).

- indications for surgical treatment:
  1. Accelerated head growth in younger patients.
  2. Significant midline shift.
  3. Enlarging over time.
  4. Obstructive hydrocephalus.

- surgery in symptomatic cases yields good results:
  a) Cyst aspiration – high rate of recurrence.
  b) Cyst fenestration – best treatment!
  c) Cyst shunting – second best treatment (due to risk of shunt dependence); for shunting into peritoneum, use a low pressure valve; if there is concurrent ventriculomegaly, may also place a ventricular shunt (e.g. through a “Y” connector).
  d) Cyst excision – significant morbidity and mortality (may be due to abrupt decompression), subsequent scarring may block fenestration allowing reaccumulation of cyst, flow through subarachnoid space may be deficient

**LIPOMA**

- derived from mesoderm.
- occur chiefly in midline (esp. over corpus callosum*, vermis, quadrigeminal cistern, spinal dural sac).
- characteristic appearance on both CT and MRI - fat density; calcification is frequent in peripheral.

**MUCOCELE**

Frontal mucocele (contrast MRI) - large mucocele compressing frontal lobe, with chronic inflammation of nasal mucosa obstructing nasal sinuses.
DERMOID, EPIDERMID, CYSTS, LIPOMA

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