Intracranial Cysts: Radiologic-Pathologic Correlation and Imaging Approach

Cysts and cystic-appearing intracranial masses have a broad imaging and pathologic spectra. The authors review the pathologic findings, origin, radiologic appearance, and differential diagnosis of many different intracranial cysts. A diagnostic algorithm based on most common anatomic locations is presented that helps narrow the differential diagnosis.

© RSNA, 2006
Cysts are common findings at magnetic resonance (MR) and computed tomographic (CT) brain imaging. Their histopathologic spectrum is broad, and differentiation of these cysts on the basis of imaging findings alone can be problematic. In this article, we will first review the pathologic and imaging spectra of nonneoplastic and tumor-associated nonneoplastic cysts (Table 1). We will discuss the major differential diagnoses for each cyst. We will then present an algorithmic location-based diagnostic approach for these cysts. Cystic and necrotic neoplasms, as well as brain abscesses, are excluded from the discussion.

**Choroid Plexus Cysts**

**Pathologic Findings**

Choroid plexus cysts (CPCs) are nonneoplastic epithelial-lined cysts of the choroid plexus (1,2) (Fig 1). They are the most common of all intracranial neuroepithelial cysts, occurring in up to 50% of autopsy cases. Most are bilateral and located in the lateral ventricular atria. The third ventricle is a rare but reported location (1). Most CPCs are asymptomatic and are found incidentally, typically in neonates and older adults. Symptomatic lesions are rare since the atria typically enlarge to accommodate the cyst (2,3).

CPCs occur when lipid accumulates in the choroid plexus from degenerating and/or desquamating choroid epithelium (1). CPCs can be almost entirely cystic, nodular, or partially cystic. They appear as nodular, yellowish gray masses within the glomus of the choroid plexus. Most are small, measuring 2–8 mm in diameter. Cysts greater than 2 cm are rare.

Microscopic analysis of CPCs reveals neuroepithelial microcysts containing nests of foamy lipid-laden histiocytes. Chronic inflammatory lymphocytic and plasma cell infiltrates, cholesterol clefts, hemosiderin, and peripheral psammomatous calcium are part of the CPC spectrum (1).

**Imaging**

CPCs are iso- to slightly hyperattenuated on nonenhanced CT scans compared with CSF. Peripheral calcification is common. The cysts show enhancement that varies from none to striking. Signal intensity on MR images is variable. Most are iso- or hyperintense on precontrast T1-weighted MR images compared with CSF and show rim or nodular contrast enhancement. CPCs are usually hyperintense to CSF on T2-weighted images, especially with long repetition/short echo time sequences. The majority do not become completely hypointense (suppress) on fluid-attenuated inversion-recovery (FLAIR) images and remain slightly or moderately hyperintense to CSF. Two-thirds show restriction (high signal intensity) on diffusion-weighted images (1,2,4). Real-time prenatal ultrasonographic (US) findings demonstrate a cyst greater than 2 mm surrounded by echogenic choroid.

**Differential Diagnosis**

The major differential diagnosis is ependymal cyst and villous hyperplasia of the choroid plexus. Ependymal cysts do not enhance. Villous hyperplasia is very rare and, when present, enhances strongly and relatively uniformly. Disturbed CSF flow and pseudolesions can also be seen on US images but are most striking around the foramen of Monro (interventricular foramen) and within the ventricular body, not the atria. Colloid cysts should not be mistaken for CPCs since they typically occur only at the foramen of Monro (see below).

**Enlarged PVSs**

**Pathologic Findings**

Enlarged PVSs, also known as Virchow–Robin spaces, are pial-lined interstitial fluid-filled structures that accompany penetrating arteries and veins (Fig 2). They do not communicate directly with the subarachnoid space (5,6). They are common, incidental, “leave me alone” lesions that should not be mistaken for more ominous disease (5). They frequently appear in the inferior basal ganglia, clustering around the anterior commissure and surrounding the lenticulostriate arteries as they superiorly course through the anterior perforated substance. Other common locations include the midbrain, deep white matter, and subinsular cortex. They can also be found in the region of the thalami, dentate nuclei, corpus callosum, and cingulate gyrus (5,6).

Microscopically, PVSs consist of a single or double layer of invaginated pia. They are typically very small or inapparent as they pass through the cortex, enlarging in the subcortical white matter. They are typically not associated with gliosis in the surrounding parenchyma (5).

**Imaging**

Prominent PVSs are considered a normal variant. Most appear as smoothly demarcated fluid-filled cysts, typically less than 5 mm in diameter, and often occur in clusters in the basal ganglia or midbrain. They are isointense to CSF at
all sequences, including FLAIR. Most show normal signal intensity in the adjacent brain; 25% may have a small rim of slightly increased signal intensity. They do not enhance, cause focal mass effect, or restrict on diffusion-weighted images. In older patients, basal ganglia PVSs sometimes become prominent and sievelike, a condition known as état criblé, or cribriform state.

Occasionally PVSs may become very large and appear bizarre. They are probably caused by the accumulation of interstitial fluid between the penetrating vessels and the pia. If interstitial fluid egress is blocked, fluid accumulates and the PVSs dilate (5). These lesions cause focal mass effect and occasionally even hydrocephalus. Rarely, so-called giant or tumefactive PVSs may be mistaken for more ominous disease (7).

**Differential Diagnosis**
Enlarged PVSs are often mistaken for multiple lacunar infarcts, cystic neoplasms, and infectious cysts. Lacunar infarcts can usually be distinguished from PVSs since many exhibit adjacent

---

**Table 1**

<p>| Classification of Intracranial Cysts according to Origin or Pathogenesis |</p>
<table>
<thead>
<tr>
<th>Origin or Pathogenesis</th>
<th>Cyst</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal and/or variant</td>
<td>Choroid plexus (xanthogranuloma), enlarged perivascular spaces (PVSs), ependymal, neuroglial, pineal</td>
</tr>
<tr>
<td>Congenital</td>
<td>Arachnoid, colloid, epidermoid, dermoid, neurenteric, Rathke cleft</td>
</tr>
<tr>
<td>Traumatic and/or vascular infectious</td>
<td>Porencephalic, neurocysticercosis, hydatid, other parasitic cysts</td>
</tr>
<tr>
<td>Tumor-associated nonneoplastic</td>
<td>Meningioma (with trapped cerebrospinal fluid [CSF]), schwannoma (with arachnoid cyst), pituitary adenoma (with enlarged PVSs), craniopharyngioma (with enlarged PVSs)</td>
</tr>
</tbody>
</table>

---

**Figure 1**

**a.** Transverse graphic representation shows multiple cystic masses in the choroid plexus glomi (arrows). Most CPCs are actually degenerative xanthogranulomas. (Image courtesy of Amirsys, Salt Lake City, Utah.)

**b.** Transverse contrast-enhanced T1-weighted MR image in a healthy 52-year-old man shows bilateral CPCs with peripheral and nodular enhancement (arrows).

**Figure 2**

**a.** Coronal gross slice of autopsied brain with postmortem gas in bilateral enlarged PVSs. (Image courtesy of E. T. Hedley-Whyte, MD, Massachusetts General Hospital, Boston, Mass.)

**b.** Transverse contrast-enhanced T1-weighted MR image shows typical nonenhancing enlarged PVSs in right basal ganglia. The cluster of variably sized cysts is a common appearance.

**c.** Transverse T2-weighted MR image shows multiple bizarre-appearing cysts (arrows) in centrum semiovale and subcortical white matter of both hemispheres. The cysts vary in size and focally expand but otherwise spare the overlying cortex.
parenchymal hyperintensity (so-called état lacunaire). Cystic neoplasms rarely exhibit signal intensity exactly like the CSF. Neurocysticercosis cysts may have a scolex (parasite head), and the cyst walls often enhance. Neurocysticercosis cysts may be multiple but do not typically occur in clusters within the brain parenchyma.

**Ependymal Cysts**

**Pathologic Findings**

Ependymal cysts are rare, benign, ependymal-lined cysts of the lateral ventricle or juxtaventricular region of the temporoparietal region and frontal lobe (Fig 3). They have been infrequently identified in the subarachnoid spaces, brainstem, and cerebellum (3,8,9). Most are incidental, but symptomatic cysts may manifest with headache, seizure, and/or obstructive hydrocephalus. Fewer than 25 symptomatic cysts have been reported in the literature (8).

Ependymal cysts are thought to arise from sequestration of developing neuroectoderm during embryogenesis. They are thin walled and filled with clear serous fluid secreted from ependymal cells. Columnar cells, with or without cilia, line ependymal cysts. They have vesicular nuclei and eosinophilic cytoplasm (10).

**Imaging**

The best diagnostic clue is a nonenhancing thin-walled CSF-containing cyst of the lateral ventricle (8).

**Differential Diagnosis**

The differential diagnosis for an ependymal cyst includes CPC, arachnoid cyst, neurocysticercosis, and asymmetric ventricles (8,10). Part or all of a ventricle (most often the temporal horn, atria of lateral ventricles, or fourth ventricle) may also enlarge if it is “trapped” by neoplasm or infection. CPCs are usually not identical to CSF at all imaging sequences, are typically bilateral, and often enhance. Arachnoid cysts occur in the subarachnoid spaces. Intraventricular neurocysticercosis cysts have a hyperintense rim and scolex on FLAIR images. Large CSF-appearing cysts may occur along the choroid fissure and can be either ependymal or lined with arachnoid (Fig 4b).

**Neuroglial Cysts**

**Pathologic Findings**

Neuroglial (also called glioeependymal) cysts are benign epithelial-lined lesions that occur anywhere in the neuraxis (Fig 4). They are uncommon, representing fewer than 1% of intracranial cysts (11). While they may occur in myriad locations, the frontal lobe is the most typical location (11). Also, intraparenchymal neuroglial cysts are more common than extraparenchymal cysts.

Intraparenchymal neuroglial cysts are congenital lesions, arising from embryonic neural tube elements that become sequestered within the developing white matter. They are rounded, smooth, and unicellular and contain clear fluid that resembles CSF. They are lined by ependymal (columnar epithelium) or choroid plexus cells (low cuboidal epithelium) (11).

**Imaging**

The best diagnostic clue to a neuroglial cyst is a nonenhancing CSF-like parenchymal cyst with minimal to no surrounding signal intensity abnormality. The cysts are benign-appearing lesions with smooth, rounded borders (11). Size is variable.

**Differential Diagnosis**

Other lesions that may be mistaken for a neuroglial cyst include an enlarged PVS, infectious cyst, porencephalic cyst, and arachnoid cyst. Enlarged PVSs are typically multiple and...
cluster around the basal ganglia. Infectious cysts, such as neurocysticercosis, are typically smaller than 1 cm and can partially enhance. Porencephalic cysts communicate with the lateral ventricle and show surrounding gliosis. Arachnoid cysts are typically extraaxial (11).

**Pineal Cysts**

**Pathologic Findings**

Pineal cysts and cystic degeneration of the pineal gland with some residual pineal parenchyma are common; they are seen in up to 10% of cases at routine imaging and in 20%--40% of cases at autopsy (12–14) (Fig 5). Microscopically, benign pineal cysts exhibit three distinct layers. The outer layer consists of a delicate layer of fibrous connective tissue. The middle layer is composed of pineal parenchyma with or without calcium. The inner layer is composed of finely fibrillar glial tissue that often contains hemosiderin deposits (12,15,16).

Theories regarding the origin of pineal cysts and/or cystic degeneration of the pineal gland include ischemic glial degeneration with or without hemorrhagic expansion, preexisting cysts that enlarge under hormonal influence, and enlargement of the embryonic pineal cavity (2,12). At surgery, they are smooth unilocular cysts with a soft tan to yellow wall. Contents vary from clear to yellow (most common) to hemorrhagic. Eighty percent are smaller than 1 cm in diameter (12). Cysts larger than 1.5 cm may result in hydrocephalus by causing compression of the tectum and aqueduct (14,15).

**Imaging**

The best diagnostic clue is unilocular fluid-filled mass within the pineal gland. Attenuation or signal intensity varies with cyst content. One-fourth have rim or nodular calcium in the cyst wall on nonenhanced CT scans. Rim or nodular enhancement is also common. On T1-weighted MR images, 55%–60% are slightly hyperintense to CSF. Most do not appear hypointense on FLAIR images, and 60% enhance with use of contrast material (12).

**Differential Diagnosis**

Pineal cysts are most often mistaken for—and may be indistinguishable from—a benign pineal parenchymal neoplasm called a pineocytoma. Pineocytomas are more likely to have solid components, but it may be impossible to distinguish the two with imaging studies alone. Both benign nonneoplastic pineal cysts and the typical pineocytoma grow extremely slowly, so follow-up scans are often not helpful. CT- or MR-guided stereotactic biopsy may be needed for the evaluation and management of symptomatic cases. Other cysts in the quadrigeminal cistern that mimic pineal cysts include arachnoid cysts (no calcium) and, rarely, epidermoid cysts (2,12).

**Arachnoid Cysts**

**Pathologic Findings**

Arachnoid cysts are benign, congenital, intraventricular space-occupying lesions that are filled with clear CSF (Fig 6). They do not communicate with the ventricular system (17,18). The cysts tend to be unilocular, smoothly marginated expansile lesions that are molded by the surrounding structures. They are common, representing 1% of all intracranial masses. The incidence is somewhat higher in men (15).

Most arachnoid cysts are supratentorial. Fifty to 60% are found in the middle cranial fossa, anterior to the temporal lobes. Other locations include the suprasellar cistern and posterior fossa (10%), where they occur most commonly in the cerebellopontine angle cistern. Less common locations are within the interhemispheric fissure; over the cerebral convexity; or in the choroideal fissure, cisterna magna, quadrigeminal cistern, and the vermis fissures (15–18).

The precise mechanism for the formation of arachnoid cysts is not known (15,18). It is possible that they are secondary to “splitting” or a diverticulum of the developing arachnoid. A newer concept for the middle fossa arachnoid cyst is the failure of temporal embryonic meninges to merge as the sylvian fissure forms. These two layers remain separate, forming duplicate arachnoid. Other mechanisms might include active fluid secretion by the cyst wall, slow distention by CSF pulsations, or one-way ball-valve flow of CSF (17). The cause has also been attributed to trauma, mastoiditis, meningitis, and subarachnoid hemorrhage (19). Arachnoid cysts are generally stable over time, although cases of sudden or progressive enlargement, as well as spontaneous resolution, have been reported (20,21).

Arachnoid cysts collapse on incision. Therefore, surgical specimens are usually limited to a portion of the outer wall. The transparent cyst wall is separate from the inner dural layer and the underlying pia-arachnoid. Most are filled with clear colorless fluid. The size varies, from small and incidental to a large space-
occupying lesion with extensive compression of the underlying brain (15,17,18).

Microscopically, the cyst wall is made of a vascular collagenous membrane lined by flattened arachnoid cells (3,18). Arachnoid cysts lack a glial-limiting membrane or an epithelial lining (2).

Imaging
The best diagnostic clue is a sharply demarcated extraaxial cyst that can displace or deform adjacent brain. Scallop ing of the adjacent calvarium is often seen. The classic arachnoid cyst has no identifiable internal architecture and does not enhance. The cyst typically has the same signal intensity as CSF at all sequences. Occasionally, however, hemorrhage, high protein content, or lack of flow within the cyst may complicate the MR appearance (15,17,18). Arachnoid cysts have an increased prevalence of coexisting subdural hematomas, especially when they occur in the middle cranial fossa.

Differential Diagnosis
The most difficult lesion to distinguish from the arachnoid cyst is an epidermoid cyst. Epidermoid cysts can appear nearly identical to CSF on CT scans. On MR images, epidermoid cysts appear isointense to CSF, although close inspection often shows they are not precisely identical in signal intensity to CSF. Arachnoid cysts typically suppress completely on FLAIR images and do not restrict on diffusion-weighted images. Occasionally an arachnoid cyst can be slightly hyperintense on images obtained with a long repetition time and a short echo time. Arachnoid cysts displace adjacent arteries and cranial nerves rather than engulf them, as epidermoid cysts often do (17–19).

Chronic subdural hematoma and porencephalic cyst can also be confused for an arachnoid cyst. Chronic subdural hematomas do not typically show CSF signal intensity on MR images and often have an enhancing membrane. Porencephalic cysts often follow a history of trauma or stroke. The cysts are normally surrounded by gliotic brain (17).

Colloid Cysts
Pathologic Findings
Approximately three people per million per year receive a diagnosis of a colloid cyst (22) (Fig 7). Colloid cysts are benign mucin-containing cysts and account for 0.5%–1% of primary brain tumors and 15%–20% of intraventricular masses (16,22,23). More than 99% are found wedged in the foramen of Monro. The cysts are typically attached to the anterosuperior portion of the third ventricular roof. The pillars of the fornix straddle the cyst. The posterior aspect of the frontal horns is often splayed laterally. Rarely, cysts are found at other sites, including the lateral ventricles, cerebellar parenchyma, and various extraaxial locations (22,23). Even relatively small colloid cysts may produce sudden acute hydrocephalus. Occasionally brain herniation with rapid clinical deterioration and even death ensue (22,23).

Like neurenteric and Rathke cleft cysts, colloid cysts are derived from embryonic endoderm (not neuroectoderm) (15). Colloid cysts originate when ectopic endodermal elements migrate into the velum interpositum during embryogenesis (23,24). Contents slowly accumulate from epithelial secretory and breakdown products (23). The cysts are smooth and spherical, varying in size from 0.3 cm to more than 4 cm in diameter (16,22). The mean size is 1.5 cm. The cysts are filled with viscous gelatinous material that consists of mucin, blood degradation products,

Figure 6
(a) Submentovertex view of autopsied brain with large middle fossa arachnoid cyst, which is contained within split layers of arachnoid. (Image courtesy of J. Townsend, MD, University of Utah School of Medicine.) (b) Transverse T2-weighted MR image shows extraaxial CSF-like arachnoid cyst in anterior middle cranial fossa (straight arrow). The temporal lobe is hypoplastic with posteriorly displaced temporal horn (curved arrow). (c) Transverse diffusion-weighted MR image shows no restriction (an epidermoid cyst would not suppress completely on FLAIR image and would restrict on diffusion-weighted image) and a classic arachnoid cyst (arrows).
foamy cells, and cholesterol crystals (18,23).

Colloid cysts are characterized by a simple to pseudostratified epithelial lining with interspersed mucous goblet cells and occasional scattered cilia (22). The epithelial layer rests on a delicate layer of collagen and fibroblasts (18).

Imaging
The best diagnostic clue to a colloid cyst is its location at the foramen of Monro. The classic colloid cyst appears as a well-delineated hyperattenuated mass on nonenhanced CT scans. Attenuation correlates inversely with hydration state. On T1-weighted MR images, two-thirds of colloid cysts are hyperintense. The majority are isointense to brain on T2-weighted images. Some demonstrate peripheral rim enhancement (22,23). Occasionally, colloid cysts expand rapidly. These colloid cysts typically have a higher water content, which reflects ongoing cyst expansion. Thus, it is hypothesized that potentially the most “dangerous” lesions are hypointense on T1- and hyperintense on T2-weighted images (24).

Differential Diagnosis
The imaging appearance of a colloid cyst is almost pathognomonic. The most common “lesion” mistaken for a colloid cyst is CSF flow artifact (MR pseudocyst) caused by pulsatile turbulent CSF flow around the foramen of Monro. Occasionally, a neurocysticercus cyst may occur at the foramen of Monro. Neoplasms such as subependymoma or choroid plexus papilloma that may occur at the foramen of Monro are much less common and typically enhance (23).

Epidermoid Cysts

Pathologic Findings
Intracranial epidermoid cysts are congenital inclusion cysts (Fig 8). Epidermoid cysts comprise 0.2%–1.8% of primary intracranial tumors and are four to nine times as common as dermoid cysts (18,25). The most common location for epidermoid cysts is the cerebellopontine angle cistern (40%–50%), where they are the third most common overall cerebellopontine angle cistern–internal auditory canal mass (after acoustic schwannoma and meningioma). Epidermoid cysts also occur in
the fourth ventricle (17%) and the sellar and/or parasellar regions (10%–15%). Less common locations include the cerebral hemispheres or brainstem. Ten percent of epidermoid cysts are extradural, located in the skull or spine. All are located off the midline (25,26). Most are asymptomatic but may occasionally result in mass effect, cranial neuropathy, or seizure (26). Occasionally, epidermoid cysts rupture and may excite a granulomatous meningitis (16,27).

Epidermoid cysts arise from ectodermal inclusion during neural tube closure in the 3rd–5th week of embryogenesis. Epithelial cell rests may be transplanted to regions such as the cerebellopontine angle by the laterally migrating otic capsule or developing neurovasculature (19). Acquired epidermoid cysts may develop as a result of trauma but are uncommon in the brain (25).

To the surgeon or pathologist, the irregular lobulated surface of the epidermoid glistens with the sheen of mother-of-pearl. This so-called “beautiful tumor” has an irregular cauliflower-like outer surface that grows to a pleasurable soft, waxy, or flaky keratohyalin material that results from the progressive desquamation of the cyst wall (18).

The microscopic cyst lining consists of stratified squamous epithelium supported by an outer layer of collagenous connective tissue. Cystic contents usually include debris, keratin, water, and cholesterol laid down in a lamellar fashion. Epidermoid cysts do not contain dermal appendages.

**Pathologic Findings**

Like epidermoid cysts, dermoid cysts are congenital ectodermal inclusion cysts (28) (Fig 9). They are extremely rare, constituting fewer than 0.5% of primary intracranial neoplasms and are four to nine times less common than epidermoid cysts (18,25). They tend to occur in the midline, parietal, parasellar, or frontonasal regions (18,28). Other dermoid cysts are midline in the posterior fossa, where they occur either as vermian lesions or within the fourth ventricle (15,18,28). These cysts increase in size by means of glandular secretion and epithelial desquamation. Growth can lead to rupture of the cyst contents, causing a chemical meningitis that may lead to vasospasm, infarction, and even death (29). Malignant transformation into squamous cell carcinoma has also been described (28).

A common misconception is that dermoid cysts arise from both ectodermal and mesodermal elements. They do not: Their origin is strictly ectodermal (18). Dermoid cysts arise from the inclusion of ectodermally committed cells at the time of neural tube closure (3rd–5th week of embryogenesis [16,28,29]). The capsule of dermoid cysts consists of simple epithelium supported by collagen. In thicker parts, the lining is supplemented with dermis containing hair follicles, sebaceous glands, and apocrine glands (16,28). The active production of hair and oils by the dermal appendages has been implicated in the earlier rupture when compared with keratin-producing epidermoid cysts (16).

The dermoid cyst is a well-defined, lobulated, “pearly” mass of variable size. The capsule is thicker than that of the epidermoid cyst and often contains plaques of calcification. Characteristically, the cyst contains thick, disagreeable, foul-smelling, yellow material due to the secretion of sebaceous glands and desquamated epithelium. The cysts may also contain hair and/or teeth (15,16,18,28).
Neurenteric Cysts

Pathologic Findings

Neurenteric cysts are congenital, benign, malformed endodermal lesions in the central nervous system (Fig 10). They are approximately three times as common in the spine, compared with the brain (30). Most intracranial neurenteric cysts are found in the posterior fossa. They are typically in the midline, anterior to the brainstem. They can also be found in the cerebellopontine angle or clivus. Supratentorial cysts have rarely been reported (30).

While the precise origin is unknown, neurenteric cysts probably arise at the time of notochordal development during the transitory existence of the neurenteric canal. The notochord and foregut fail to separate, causing primitive endodermal cells to be incorporated into the notochord. These displaced alimentary cells ultimately become the cyst (18,30,31).

The size of the cysts is variable, usually measuring less than 2 cm. Gross cysts are smooth, thin-walled, and transparent. The contents vary from clear to mucoid or xanthochromic (30).

Microscopic examination of the cyst wall demonstrates endothelium-lined structures of cuboidal to columnar cells (partially ciliated). The epithelium may be pseudostratified in places and typically has ciliated and goblet cells (31,32). The cysts contain only endodermal elements and closely resemble gastrointestinal tract mucosa (31).

Imaging

The best diagnostic clue for a neurenteric cyst is a round and/or lobulated, nonenhancing, slightly hyperintense mass in front of the medulla. The signal intensity characteristics vary depending on the protein content of the cysts. Most are proteinaceous with a T1-weighted imaging appearance that is iso- to slightly hyperintense compared with the CSF and a T2-weighted imaging appearance that is very hyperintense (30,33–35). Neurenteric cysts are hyperintense on FLAIR images and may show mild restriction on diffusion-weighted images. They very rarely show rim enhancement.

Differential Diagnosis

Neurenteric cysts may be confused with an epidermoid, craniopharyngioma, teratoma, or lipoma. Epidermoid cysts typically resemble CSF (not fat), lack dermal appendages, and are usually located off midline. Like dermoid cysts, craniopharyngiomas are suprasellar, with a midline location, and demonstrate nodular calcification. However, most craniopharyngiomas are strikingly hyperintense on T2-weighted images and enhance strongly. Teratomas may also have a similar location but usually occur in the pineal region. Lipomas demonstrate homogeneous fat attenuation and/or signal intensity and show a chemical shift artifact, which typically does not occur with dermoid cysts.

Rathke Cleft Cysts

Pathologic Findings

Rathke cleft cysts are congenital non-neoplastic cysts arising from remnants of the embryonic Rathke cleft (36) (Fig 11). They are common incidental intracranial cysts and are found in 13%–33% of routine autopsy series (36,37). Forty percent are completely intrasellar, while 60% have some suprasellar extension (36). Completely suprasellar cysts are rare (18). Symptoms occur from compression of the optic chiasm, hypothalamus, or pituitary gland (15).

Rathke cleft cysts probably arise from the failure of obliteration of the Rathke pouch, which develops as a rostral outpouching of the primitive oral cavity during the 3rd or 4th week of embryogenesis (37). This rostral elongation of the pouch forms the craniopharyngeal duct. The proximal portion of the duct closes early during development, but a remnant may persist between the pars distalis and pars intermedia. Occasionally, this remnant gives rise to the macroscopic Rathke cleft cyst (18).

Rathke cleft cysts are smoothly margined cysts that vary in size from a few millimeters to 1–2 cm. The contents vary
from clear CSF-like fluid to thick mucoid material (36). Microscopically they are similar to other endodermal cysts (neurenteric and colloid). They are lined by pseudostratified or single-layered columnar or cuboidal epithelium. Cilia and scattered mucin-secreting goblet cells are common. Many cysts have squamous differentiation, and cornified squamous pearls are occasionally identified (18). The intracystic nodule consists of mucinous material at histologic examination. Biochemical analysis of this material is consistent with cholesterol and protein (37).

Differential Diagnosis

The differential diagnosis for Rathke cleft cysts includes craniopharyngioma, cystic pituitary adenoma, or other nonneoplastic cysts (36). Unlike Rathke cleft cysts, craniopharyngiomas typically demonstrate calcification and approximately 90% have nodular, globular, or rim enhancement. The presence of solid enhancing nodules in the cyst wall also favors the diagnosis of craniopharyngioma (37,38). The rare noncalcified cystic nonenhancing craniopharyngioma, a finding more common in adults than in children, may be impossible to distinguish from Rathke cleft cyst with imaging findings alone.

Porencephalic Cysts

Pathologic Findings

Porencephalic cysts are congenital or acquired cavities within the cerebral hemisphere that usually—although not invariably—communicate directly with the ventricular system (1). They can be cortical or subcortical, unilateral or bilateral (39). The location often corresponds to territories supplied by the cerebral arteries.

Congenital porencephalic cysts originate from a fetal or perinatal encephaloclastic process that results from intrauterine vascular or infectious injury. Acquired cysts are secondary to injury later in life and are usually secondary to trauma, surgery, ischemia, or infection (Fig 12a).

Porencephalic cysts vary greatly in size. They are typically CSF-filled cavities with a smooth wall and are lined with gliotic or spongiotic white matter. The adjacent skull may demonstrate remodeling due to chronic CSF pulsations (39).

Imaging

The typical porencephalic cyst is a cystic space in the brain parenchyma that communicates with an enlarged adjacent ventricle. The cysts have the same appearance as CSF at all MR sequences (39). Adjacent white matter typically shows hyperintensity on T2-weighted and FLAIR images.

Figure 11: Sagittal postcontrast T1-weighted MR image shows rounded mass (solid arrow) separate from and just above the pituitary gland (open arrow). This cyst has moderately high protein content and is isointense with brain, not CSF. Location is typical for a Rathke cleft cyst, confirmed at surgery. (Image courtesy of J. Rees, MD, MedTell International, McLean, Va.)

Figure 12: (a) Close-up of autopsy brain specimen shows large acquired porencephalic cyst in the temporal lobe that communicates with temporal horn of the lateral ventricle (arrows). (Image courtesy of E. Ross, MD, Loyola University School of Medicine, Chicago, Ill.) (b) Coronal T1-weighted MR image in another case shows enlarged left temporal horn (black arrow) that communicates with peripherally located porencephalic cyst (white arrows). Cyst extends to the brain surface.
Differential Diagnosis
The differential diagnosis for the porencephalic cyst includes arachnoid cyst, schizencephaly, ependymal cyst, encephalomalacia, and hydranencephaly. Arachnoid cysts are extraaxial and displace the brain cortex away from the adjacent skull. Schizencephaly is a CSF-filled cavity that is lined with heterotopic gray matter and extends all the way from the ventricle to the brain surface. Ependymal cysts are typically intraventricular with normal surrounding brain tissue (39).

Neurocysticercosis
Pathologic Findings
Cysticercosis is the most common, most widely disseminated parasitic infection in the world (40,41) (Fig 13). Neurocysticercosis occurs in 60%–90% of all cases of systemic cysticercosis. Most neurocysticercosis cysts are found in the subarachnoid spaces, typically the basal cisterns and deep within the sulci. Inflammatory reaction provoked by the cyst may cause adhesion of the adjacent gyral surfaces, giving the mistaken impression that the cyst is intraparenchymal. Other common locations include the hemispheric parenchyma at the gray matter–white matter interface and in the ventricles (fourth ventricle is most common) (40). Rarely, cysts are found in the sella, orbit, or spinal cord (41). Seizures are the common manifestation of neurocysticercosis (40,42).

Imaging
Imaging findings in neurocysticercosis vary with the stage of cyst development. The early vesicular stage is typified by a smooth thin-walled cyst that is CSF-like on CT and MR images. Edema and contrast enhancement are rare. A mural nodule is often present that represents the viable larval scolex, the “cyst with a dot” appearance (40,41). When cyst degeneration begins (colloidal-vesicular stage) and host inflammatory response ensues, pericystic edema and cyst wall enhancement are present. Cyst fluid is hyperintense to CSF on MR images during this stage (41).

In the healing, or granular nodular, stage, nonenhanced CT scans show an isodense cyst with a hyperattenuated calcified scolex. Surrounding edema is still present, and enhancement following contrast material administration persists. The residual cyst is isointense to the brain on T1-weighted images and it is isointense to hypointense to T2-weighted images. Nodular or micro-ring enhancement is common at this stage, suggesting granuloma. Occasionally, a “target” or “bull’s eye” appearance is seen with the calcified scolex in the center of the mass (41).

In the quiescent or residual stage, small calcified nodules without mass effect and usually without enhancement are seen (40,41). Multifocal lesions and lesions in different stages of development are common.

Differential Diagnosis
The differential diagnosis for neurocysticercosis includes abscess, tuberculosis, neoplasm (primary or metastatic), enlarged PVSs, and other parasitic infections. Abscesses have a T2-hypointense rim, whereas neurocysticercosis cysts are typically isointense except when they are in the ventricles where the rim is hyperintense on FLAIR images. Tuberculomas often occur with meningitis, are rarely cystic, and are often profoundly hypointense on T2-weighted images. Enlarged PVSs have the same appearance as CSF at all MR sequences and do not enhance. None of these cystic lesions has the characteristic “cyst with dot” appearance (40).
Hydatid Cysts

Pathologic Findings
Intracranial hydatid cysts are parasitic infections caused by the larval stage of *Echinococcus granulosus* (41) (Fig 14). The cysts preferentially affect the liver but may also involve the lungs, bone, and brain. Cerebral hydatid cysts are rare, seen in only 2% of cases (41,43). The most common location for intracranial hydatid cysts is the hemispheric parenchyma, particularly in the perfusion territory of the middle cerebral artery (41,44). The subarachnoid spaces are another common site of involvement.

Hydatid cysts are usually spherical, solitary, and unilocular. They grow slowly and are typically large, averaging 4–10 cm in diameter (45). The cysts contain translucent fluid and may also contain daughter cysts with an appearance resembling small white grapes. Protoscolices within cysts form a granular deposit known as hydatid sand (44).

Imaging
The best diagnostic clue of a hydatid cyst is a single, large, thin-walled, spherical, nonenhancing CSF-attenuation cyst in the parietal region of the brain. Perilesional edema is usually absent. The two visible imaging components are the cyst and the pericyst. The pericyst is a peripheral capsule of the cyst. While MR imaging is more sensitive in demonstrating the pericyst, CT is more sensitive in depicting cyst calcification (43). Multilocular or multiple lesions occur but are rare (43).

Differential Diagnosis
The differential diagnosis for hydatid cysts includes arachnoid cyst, epidermoid cyst, and neurocysticercosis (41).

Other Parasitic Cysts

Pathologic Findings
A number of parasites may occasionally infect the central nervous system and appear at least partially cystic (Fig 15a). Amebiasis, paragonimiasis, schistosomiasis, and sparganosis can cause both unilocular and complex intraparenchymal cysts with or without accompanying meningoencephalitis. Perilesional edema and petechial hemorrhage are common.

Imaging
Complex conglomerate cysts with thick enhancing rims and striking adjacent edema are common.
Differential Diagnosis

Complex conglomerated parasitic cysts of any origin may mimic primary or metastatic brain tumor. The patient’s personal and travel history, as well as serologic findings, are key to the diagnosis.

Neoplasm-associated Benign Cysts

Pathologic Findings

Extraaxial tumors such as meningioma, schwannoma, craniopharyngioma, and pituitary macroadenoma may be associated with large nonneoplastic cysts (Fig 16). These peritumoral cysts appear to contain CSF. Some, such as cysts that occur adjacent to vestibular schwannoma, are true arachnoid cysts. Others, such as meningioma, trap CSF within the cleft between the expanding tumor and the adjacent brain. Craniopharyngiomas and pituitary macroadenomas with suprasellar extension may obstruct

Table 2

<table>
<thead>
<tr>
<th>Classification of Intracranial Cysts according to Most Common Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyst</td>
</tr>
<tr>
<td>Arachnoid</td>
</tr>
<tr>
<td>Choroid plexus</td>
</tr>
<tr>
<td>Colloid</td>
</tr>
<tr>
<td>Craniopharyngioma (with enlarged PVs)</td>
</tr>
<tr>
<td>Dermoid</td>
</tr>
<tr>
<td>Enlarged PVs</td>
</tr>
<tr>
<td>Epidermoid</td>
</tr>
<tr>
<td>Ependymal</td>
</tr>
<tr>
<td>Hydatid</td>
</tr>
<tr>
<td>Meningioma (with trapped CSF)</td>
</tr>
<tr>
<td>Neurenteric</td>
</tr>
<tr>
<td>Neurocysticercosis</td>
</tr>
<tr>
<td>Neuroglial</td>
</tr>
<tr>
<td>Pineal</td>
</tr>
<tr>
<td>Pituitary adenoma (with enlarged PVs)</td>
</tr>
<tr>
<td>Porencephalic</td>
</tr>
<tr>
<td>Rathke cleft</td>
</tr>
<tr>
<td>Schwannoma (with arachnoid cyst)</td>
</tr>
</tbody>
</table>
and enlarge adjacent PVSs. Interstitial fluid is retained within the enlarged PVSs and may result in edema along the optic tracts. Occasionally, large peritumoral cysts can be identified.

Imaging

Tumor-associated arachnoid cysts and cystic PVSs do not enhance. Most parallel CSF in signal intensity. If protein content is elevated within the trapped pools of CSF, the arachnoid cysts may appear slightly hyperintense to normal CSF on MR images.

Differential Diagnosis

True arachnoid cyst associated with neoplasm may be impossible to distinguish from enlarged PVSs with trapped interstitial fluid unless biopsy is performed.

Location-based Diagnostic Approach

A location-based approach to intracranial cysts is helpful in establishing an appropriate differential diagnosis. Many intracranial cysts occur in characteristic locations (Table 2). Some locations are virtually pathognomonic for certain lesions (eg, colloid cyst in anterosuperior aspect of the third ventricle), while others are suggestive of—but not specific for—a particular diagnosis (eg, middle cranial fossa and arachnoid cyst).

A series of imaging questions, posed in the following order, results in a useful diagnostic algorithm (Fig 17): (a) Is the cyst intra- or extraaxial? (b) Is it supra- or infratentorial? (c) If extraaxial, is it midline or off midline? (d) If intraaxial, is it intraparenchymal or intraventricular? (e) If intraventricular, what is the specific location? (f) Is the cyst CSF-like on CT and/or MR images or is it different? (g) Are there any special distinguishing imaging features (eg, calcification, enhancement, diffusion restriction)?

Conclusion

A broad spectrum of diseases can cause intracranial cysts. The combination of a location-based algorithm with specific imaging findings, such as presence or absence of calcification, enhancement, MR signal intensity at different sequences, and presence or absence of restriction on diffusion-weighted images, permits a narrowed differential diagnosis when an intracranial cyst is identified on imaging studies.

References

23. Pollock BE, Schreiner SA, Huston J 3rd. A series of imaging questions, posed in the following order, results in a useful diagnostic algorithm (Fig 17): (a) Is the cyst intra- or extraaxial? (b) Is it supra- or infratentorial? (c) If extraaxial, is it midline or off midline? (d) If intraaxial, is it intraparenchymal or intraventricular? (e) If intraventricular, what is the specific location? (f) Is the cyst CSF-like on CT and/or MR images or is it different? (g) Are there any special distinguishing imaging features (eg, calcification, enhancement, diffusion restriction)?


