



Histological Findings of Arachnoid Cysts

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1 Introduction

Arachnoid cysts (ACs) represent benign fluid-filled cystic lesions that may arise at essentially any site within the central nervous system (CNS) in which arachnoid cap cells reside. Although they frequent the temporal operculum/Sylvian fissure and temporal pole, alternate locations include various posterior fossa sites (cerebello-pontine angle, quadrigeminal region [2, 65], cisterna magna), sella/suprasellar regions [36], or along the optic nerves or spinal cord; few intraventricular [23] examples have been described [47, 51, 54, 67]. ACs occur throughout all age groups and may be congenital or acquired secondary to a variety of pathologies that result in inflammation/irritation or hemorrhage involving the leptomeninges.

The majority of ACs are found incidentally on neuroimaging studies, necessitating only conservative management. On both computed tomography (CT) and magnetic resonance imaging (MRI) scan techniques, ACs present a thin wall that lacks enhancement after contrast administration and fluid contents with signal density/intensity typical of cerebrospinal fluid (CSF). On occasion, sizeable and/or strategically situ-

ated ACs may become symptomatic, resulting in headaches, dizziness, seizures, weakness, cognitive dysfunction, or focal deficits [67]. Potential surgical options include cystoperitoneal shunting, surgical resection, aspiration, or fenestration. The reader is directed to the respective chapters in this book for a more detailed discussion of the clinical presentation, localization, neuroimaging features, and treatment options related to ACs.

The focus of the current chapter is to outline the spectrum of histological, immunohistochemical, and ultrastructural features of ACs. Comparisons will be drawn with other cystic lesions of the CNS to facilitate a tissue-driven differentiation of ACs from the multitude of potential clinicoradiographic mimics. A brief review of unusual presentations of histologically confirmed ACs is also provided, to include co-occurrence of ACs with a variety of other neoplastic and nonneoplastic pathologies.

2 Microscopic and Immunohistochemical Features of Arachnoid Cysts

Macroscopically, ACs present as fluid-filled cysts with generally delicate walls ranging from thin and translucent to thicker and opaquer. Though fluid contents are often clear and colorless, prior hemorrhage may result in more xanthochromic discoloration.

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2.1 Spectrum of Findings by Light Microscopy

The AC lining is composed of two layers. Meningothelial cells, often flattened and/or focally denuded, form the inner layer; the outer layer is collagenous and variable in thickness [9, 17, 25, 31, 33, 39, 70] (Figs. 1 and 2). More opaque-appearing cyst walls tend to be associated with thicker hyalinized collagen components. Foci of meningothelial hyperplasia may be encountered, as can be the occasional sparse inflammatory infiltrate. Ciliated or squamous epithelial cells are absent. The adjacent CNS parenchyma, if sampled, may show reactive astrocytosis.

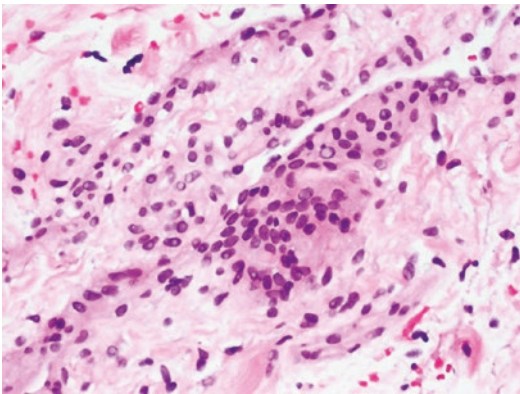


Fig. 1 Microscopic appearance of a typical AC showing bland meningothelial cells upon fibrous tissue (hematoxylin and eosin, ×200)

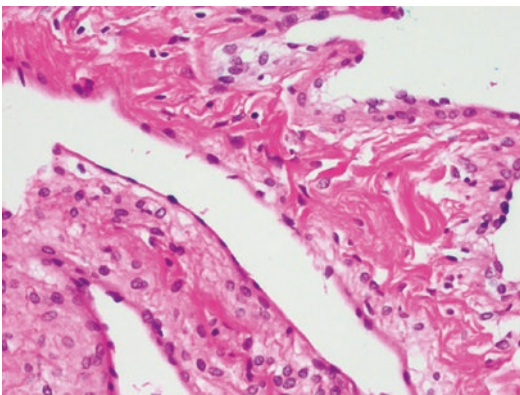


Fig. 2 Some ACs have a more attenuated lining (hematoxylin and eosin, ×200)

On occasion, other histological elements may be encountered within the walls of otherwise typical ACs. For instance, hemosiderin-containing macrophages trapped within AC walls may be seen and imply a prior site of hemorrhage [61]. Morioka et al. described finding nevus cells within a posterior intradural extramedullary thoracic spinal AC that arose in a patient with a large congenital melanocytic nevus involving the skin of his back [41]. ACs with heterotopic neuroglial tissue have been described in multiple instances, mainly in ACs arising in posterior fossa sites [5, 7]. Few ACs containing ectopic choroid plexus have been documented, all within the posterior fossa/cerebellopontine angle region, both in children [24, 55] and adults [59]. Candy et al. report a unique “dual” cyst arising from the cauda equina in a 68-year-old man; this cyst was lined by areas of pseudostratified ciliated columnar epithelium (bronchogenic cyst lining) as well as regions with “pleated fibrovascular tissue lined by monomorphic arachnoid cells” (AC lining). The AC was successfully treated by marsupialization [12].

2.2 Common Patterns of Immunohistochemical Labeling

The meningothelial lining cells of ACs will stain positive for immunohistochemical stains targeting epithelial membrane antigen (EMA) and vimentin [29, 40] (Fig. 3). Unlike a variety of other cystic lesions that may be encountered within the CNS, ACs do not express cytokeratins, glial fibrillary acidic protein (GFAP), S100 protein, transthyretin (prealbumin), or carcinoembryonic antigen (CEA) [40]. That said, immunohistochemistry could be quite helpful in discriminating an AC from other potential diagnostic mimics, as described below.

Several studies for determination of sex hormone status in the lining cells of ACs have been published. Similar to meningiomas, a substantial proportion of ACs will possess progesterone receptor positivity by immunohistochemistry [20, 32, 66]. In contradistinction, ACs have been

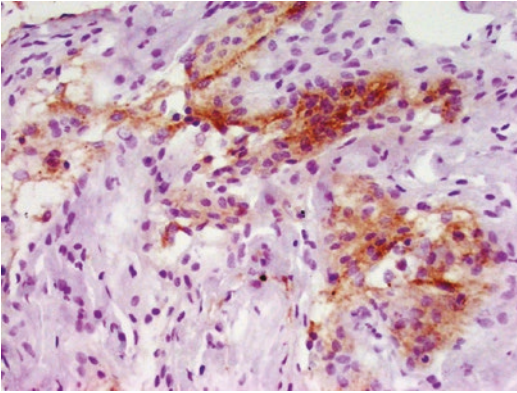


Fig. 3 Meningeothelial cells of AC are immunopositive for epithelial membrane antigen (EMA) stain (anti-EMA, $\times 200$)

found to lack both estrogen and androgen receptors (which may be detected in a minor subpopulation of meningiomas), and likewise their lining consistently expresses aromatase, a feature not typical of meningiomas [32]. The clinical relevance of these findings remains unclear.

3 Ultrastructural Features of Arachnoid Cysts

Scanning electron microscope (SEM) examinations of ACs have documented cyst linings rich in microvilli of varying densities. Scattered larger cell surface projections with a filiform or club-like morphology have been encountered [20, 22].

Ultrastructural features observed by transmission electron microscopy (TEM) are those of a single layer of lining cells (meningeothelial) that are separated from the surrounding collagen-rich connective tissue by a distinct (though variably discontinuous) basement membrane [20, 22, 38, 42]. Some authors have identified ACs composed of only fibrous tissue walls devoid of a meningeothelial cell lining, though in other cases the meningeothelial lining was multiple cell layers thick [20, 38]. Splitting of the arachnoid membrane at the cyst margin has been observed [54]. Closer inspection of the lining cells finds surface microvilli often containing vacuoles and

sometimes with elongated bulbous formations, but no true cilia [20, 22, 38]. These cells contain irregular-shaped, lobulated nuclei with nucleoli, rough and smooth endoplasmic reticulum, and numerous mitochondria. Multivesicular bodies with coated granules have been demonstrated, as have numerous small intracellular vesicles and pinocytotic pits [20–22]. Where lining cells adjoin, there are often desmosome-like junctional complexes or intercellular interdigitations with sinusoidal dilations containing complex and often tortuous processes [20–22, 38, 42]. Intracytoplasmic tonofilaments have been described, interacting with desmosome-like complexes [21, 22].

More recently, Rabiei et al. conducted a comparative ultrastructural analysis of symptomatic ACs from 24 patients [52]. The diversity of ultrastructural features that they demonstrated led the authors to separate the cysts in their cohort into three groups: (a) cysts lined with arachnoid tissue, (b) fibrous walled cysts, and (c) “cysts with aberrant structure.” The first two categories correlate with findings previously described by others above, whereas the “aberrant structure” group had more unusual findings. Although they too were lined by cells with surface microvilli, some also had demonstrable cilia and/or apical secretory-type granules. Subepithelial neuropil and glial components were also seen in this group [52]. From these ultrastructural findings, the authors postulate that these varying cyst wall components provide “different barrier properties and fluid turnover characteristics” [52].

4 Pathologic Differentiation of Arachnoid Cyst from Other Cystic CNS Lesions

ACs need to be differentiated from a wide variety of other lesions that may arise within the CNS. These include neoplastic, infectious, and other nonneoplastic processes that may form (or be associated with) cysts.

4.1 CNS Infections Presenting as Cysts

Several infectious processes may present as cystic lesions within the CNS. Bacterial, fungal, or toxoplasma abscesses all may at least superficially appear “cystic.” Their neuroimaging characteristics are typically quite dissimilar from those noted elsewhere for AC in that infectious abscesses contain necrosis and/or fibrinopurulent debris within their central cores and manifest with ring enhancement on post-contrast images. Histological assessment will easily discriminate these necroinflammatory lesions from AC or other CNS cysts.

In contradistinction, parasitic cysts tend to be more truly cystic in architecture, depending on their life stage and region of CNS involvement. Neurocysticercosis, resulting from CNS infection by the pork tapeworm *Taenia solium*, may manifest as intraparenchymal, intraventricular, or subarachnoid/leptomeningeal lesions. Parenchymal cystic lesions are most common and tend to be small and multiple, whereas intraventricular cysts tend to be solitary. Infectious cysts within the subarachnoid space may grow quite large, and some may take on a grapelike “racemose” appearance [60]. It is these subarachnoid and intraventricular infectious cysts that may closely mimic ACs, given that they tend to lack identifiable scolex structures (more typical of the intraparenchymal lesions) and the cyst contents have similar signal characteristic to CSF on MR images. With the eventual death of the encysted parasites, the imaging findings change as well, with surrounding edema and thickening and enhancement of the cyst wall eventually giving way to cyst collapse, retraction, and calcification [64]. Histological examination of the larva cyst wall shows three layers of nonhuman tissue: an outer cuticular, middle cellular, and inner fibrillary layer. A scolex may or may not be present [10, 15].

Neurohydatidosis, resulting from infection with the parasite *Echinococcus granulosus* (or rarely other *Echinococcus* species), is much rarer than parasitic involvement of the liver and lungs.

The majority of cerebral hydatid cysts involve cerebral parenchyma supplied by the middle cerebral artery. They may be solitary or multiple. Neuroimaging studies reveal circumscribed non-enhancing intraparenchymal cysts containing fluid isointense with CSF on all MR imaging sequences [11, 49, 50]. Therefore, apart from their typically intraparenchymal location, they show significant neuroimaging overlap with AC. Histological examination of the cyst wall would confirm the diagnosis, as, similar to neurocysticercosis, hydatid cysts have multilayer walls of nonhuman tissue, often with associated brood capsules [10, 45, 63].

4.2 Neuroenteric (Enterogenous/Endodermal) Cysts

Neuroenteric cysts, as their name implies, are benign cysts lined by epithelium that may resemble that typical of the gastrointestinal or respiratory tract. They arise from congenitally displaced elements of the alimentary tract. These cysts most frequently arise in the subarachnoid space anterior to the spinal cord, though they have been encountered in intracranial sites as well, especially within the posterior fossa. Spinal dysraphism may accompany those arising at lumbosacral levels. They typically come to clinical attention due to local mass effect on adjacent structures. By neuroimaging studies, they may resemble a wide variety of CNS cystic lesions, including AC, in that the MRI signal characteristics of their contents are similar to that of CSF. They may closely resemble colloid or Rathke’s cleft cysts in neuroimaging characteristics, though location typically gives a strong clue for differentiating between these entities [19]. At surgery, they are mostly small in size, thin-walled, and containing mucinous material. Similar to AC, they do have a thin fibrous wall; however, the lining is that simply of pseudostratified columnar epithelium, variably with cilia, and frequently containing goblet cells [62]. Squamous metaplasia is not uncommon and may sometimes be associated with xanthogranulomatous inflam-

mation [68]. Occasionally, the lining epithelium is more complex and closely resemble gastrointestinal or respiratory-type epithelium; seromucinous glands, ganglia, and lymphoid elements may be present [19]. Given the presence of goblet/mucin-producing cells, the epithelium is typically positive for mucin stains, and the immunohistochemical profile includes positivity for cytokeratin, EMA, CEA, and CA19-9 [1, 19, 40]. S100 may be positive on occasion, but stains targeting GFAP, NSE, and vimentin should be negative [19, 29]. Collagen IV stain decorated basement membrane beneath the epithelial surface.

4.3 Colloid Cyst

Colloid cysts represent mucin-filled benign cysts that arise within the rostral regions of the third ventricle, often near the foramen of Monro. The vast majority occur within young to middle-age adults, presenting with complaints of headache. These cysts may cause intermittent obstruction of CSF flow, and the so-called drop attacks of transient leg paralysis and sudden death may result. Ventriculitis may develop from cyst rupture. Neuroimaging studies typically show a round discrete third ventricular lesion that is bright on T2 and FLAIR images, and does not enhance post-contrast (though there may be a thin rim of enhancement of the capsule itself). Microscopically, though they like AC have a thin fibrous wall, colloid cysts are lined by a ciliated cuboidal to columnar pseudostratified epithelium with variable numbers of interspersed goblet cells. Lining epithelium may also be flattened/attenuated or pseudostratified. Cyst contents are amorphous eosinophilic material, similar to that seen within Rathke's cleft cysts [62]. The epithelial lining cells are immunopositive for both EMA and cytokeratin, the later stain easily differentiating them from AC. The lining is also variably positive for CEA and is negative for GFAP, transthyretin, and S100 [16]. Collagen IV stain decorated subepithelial basement membrane. Periodic acid-Schiff (PAS) stain may dec-

orate cells with mucin production in some cases. Unlike some other CNS cysts (Rathke's cleft or enterogenous cysts), colloid cysts do not develop squamous metaplasia. Long-standing cysts not infrequently incite a surrounding xanthogranulomatous inflammatory reaction.

4.4 Rathke's Cleft Cyst

Similar to neuroenteric cysts, Rathke's cleft cysts are also mucus-containing cysts, in this case arising from Rathke's cleft remnants within the sellar/suprasellar region. Though larger cysts may become symptomatic, oftentimes, these benign cysts appear as incidental findings on neuroimaging studies or at autopsy. Histologically, they are lined by pseudostratified columnar ciliated epithelium with goblet cells [39, 62] (Fig. 4). Squamous metaplasia may be present, as may a xanthogranulomatous reaction in the surrounding tissue. Cyst content itself, when submitted for histological review, is generally an amorphous pink/eosinophilic material. The lining epithelium is consistently immunopositive for both cytokeratin and EMA [16], as well as mucin stains [40]. CEA and S100 expression is more variable [16, 29]. Collagen IV stain highlights basement membrane beneath the epithelium.

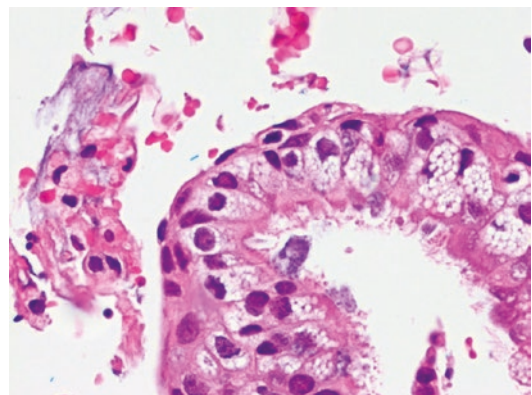


Fig. 4 Rathke's cleft cyst contains a lining of pseudostratified ciliated columnar epithelium with scattered mucin-containing goblet cells (hematoxylin and eosin, $\times 200$)

4.5 Epidermoid and Dermoid Cysts

Epidermoid cysts may arise throughout the neuroaxis, though the cerebellopontine angle is their most favored site. The floor of the fourth ventricle, suprasellar region, and along the spinal cord are other common sites, and they may involve intradiploic calvarium. On neuroimaging studies (both CT and MRI), they may closely mimic AC in that their cyst contents of cellular debris and high cholesterol content can have similar density to CSF. They typically do not enhance after contrast administration, though they may infrequently show focal wall enhancement. Diffusion-weighted imaging (DWI) sequences may help in differentiating epidermoid from ACs, epidermoid cysts being very bright on DWI, which is not a feature of ACs. Often appearing pearly white on gross inspection, epidermoid cysts have fibrous walls lined by mature keratinizing squamous epithelium (Fig. 5). These cysts are filled with anucleated sloughed squames. Lining cells are immunopositive for both cytokeratin and EMA [62].

In contradistinction, dermoid cysts are much less common than epidermoid cysts within the CNS. Dermoid cysts most frequently affect children, and they tend to arise along midline structures, including in association with fontanelles and midline cerebellum/fourth ventricular, or

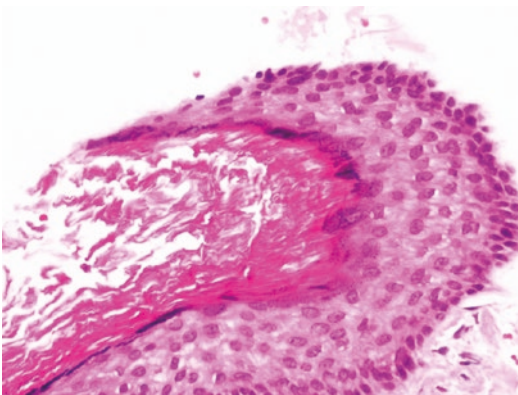


Fig. 5 Epidermoid cyst lining is that of a stratified squamous epithelium (hematoxylin and eosin, $\times 200$)

along the spinal canal. Similar to epidermoid cysts, they have fibrous walls and lining of mature keratinizing squamous epithelium; however, they also contain skin adnexa, and their cyst contents contain hair and sebum in addition to anucleated squames [62]. Also, similar to epidermoids, the lining epithelium is immunopositive for cytokeratin and EMA. Cyst rupture may result in a chemical meningitis with foreign body granulomatous inflammatory reaction.

4.6 Choroid and Ependymal Cysts

Choroid plexus cysts are one of the most common incidental findings detected at brain autopsy. They may be multiple and are generally asymptomatic. On occasion, large choroid plexus cysts might come to clinical attention due to symptoms related to intermittent hydrocephalus due to ball-valve-like mechanical obstruction. Choroid plexus cyst lining, histologically similar to normal choroid plexus epithelium, is immunopositive for cytokeratin, transthyretin, and S100 [16, 29].

Ependymal cysts are uncommon benign cystic lesions that may arise throughout the brain and spinal cord. They are often paraventricular or intraventricular in location and are frequently large with internal septations. Neuroimaging studies show their contents to have signal characteristics typical of CSF, being hypointense on T1-weighted, hyperintense on T2-weighted, and without diffusion restriction on the respective MRI sequences. At surgery, these may closely resemble ACs in gross appearance; however, histologically, they are quite dissimilar. Unlike ACs, ependymal cysts do not have fibrous walls; instead, their simple cuboidal to columnar (and often ciliated) lining epithelium rests upon a surrounding neuroglial wall [62]. Some ependymal cysts alternately have quite gliotic walls, often bearing abundant Rosenthal fibers. Ependymal cysts are immunopositive for S100 and GFAP [16], a testament to their glial lineage. Cytokeratin positivity is lacking, and there is likewise no collagen IV basement membrane staining.

4.7 CNS Neoplasms with Cystic Components

Multiple CNS tumors characteristically have prominent cystic components that might potentially mimic AC. For instance, ACs that arise within the sella/suprasellar region may mimic not only Rathke's cleft cyst as noted above but also craniopharyngioma or cystic teratoma. Both craniopharyngioma and teratoma may present as purely cystic lesions on neuroimaging studies, though they much more frequently exhibit a mixed solid and cystic or purely solid appearance. Calcification (best seen on CT imaging) may also be present, particularly in craniopharyngioma; this would be unexpected for AC [58]. Additional CNS neoplasms that often have a cystic component (which may predominate) include pilocytic astrocytoma, pleomorphic xanthoastrocytoma, ganglion cell tumors (ganglioglioma, gangliocytoma, and desmoplastic infantile ganglioglioma), and hemangioblastoma. Confirmatory histology examination easily discriminates between these lesions and is crucial in guiding patient management and follow-up.

4.8 Miscellaneous Cystic CNS Lesions

Pineal cysts are benign intrapineal cystic lesions that are detectable in approximately 2% of adults, usually encountered as incidental radiographic or autopsy findings. Rarely do they enlarge enough to result in aqueductal compression and resultant clinical symptoms. Although they are well-circumscribed and have CSF-like cyst contents on neuroimaging studies, the similarity with ACs ends there. Pineal cysts characteristically have three detectable layers on histological inspection. The outer layer is leptomeningeal, surrounding a layer of compressed pineal parenchyma, which may show calcification. The innermost layer is essentially a glial scar, often with many Rosenthal fibers and sometimes hemosiderin [39].

Cystic CNS lesions may result from surgical resection of some other process or alternately from traumatic damage, hemorrhage, or brain

infarct. All of these give rise to encephalomalacia, regions of parenchymal loss. Though their contents have neuroimaging qualities akin to CSF (similar to ACs), their walls are made up only of the surrounding gliotic brain parenchyma. Similarly, porencephalic cysts result from encephaloclastic insults during early development, producing cystic lesions that communicated with the ventricle and/or subarachnoid space. These pores have smooth walls lacking gliosis, and they contain CSF; however, unlike ACs, they are intraparenchymal and not purely leptomeningeal [28].

Posterior fossa cysts include such developmental abnormalities as Blake's pouch and cysts associated with Dandy-Walker malformation (DWM). Both of these cysts may mimic AC in terms of having a smooth wall and containing CSF; however, the finding of additional anatomic clues on neuroimaging studies will help discriminate between these entities [43]. Blake's pouch and DWM will have an enlarged retrocerebellar space, containing a membranous cyst that is in continuity with the fourth ventricle, and DWM will additionally have evidence of cerebellar vermician hypoplasia or absence [3, 8]. In contradistinction, posterior fossa ACs are distinctly separated from the fourth ventricle, and their fibrous walls are lined only by meningotheelial elements; ACs lack ependyma, glia, or neuronal elements that are commonly present in the walls of Blake's pouch or Dandy-Walker cyst [3, 26, 37].

4.9 Unusual Clinicoradiographic Presentations of Arachnoid Cysts for which Histological Confirmation Is Prudent

Although the vast majority of ACs are encountered in locations in which arachnoid cells typically reside (along the surfaces of the brain and spinal cord), on occasion, they may present at unexpected locations. For instance, extradural [4, 14, 27, 34] and intradiploic ACs [6, 30, 69] have been described. Fleck et al. encountered an AC confined to the internal auditory canal [18]. ACs may also rarely present as entirely intraparenchy-

mal cystic lesions, either within the brain [48] or intramedullary spinal cord [46, 57]. In these circumstances, histological examination will be crucial in confirming the definite diagnosis, differentiating ACs from the various entities that would enter into the differential diagnosis as noted above.

ACs may also present concurrently with other unrelated lesions. For example, Rao et al. describe an intrathoracic meningocele pouch found incidentally during evaluation for a dorsal intradural extramedullary AC extending from T1 to T5 spinal levels [53]. Nishio and colleagues describe the co-occurrence of hypothalamic hamartoma with an AC in a pediatric patient [44]. Co-occurrence of intracranial aneurysm and AC has been encountered on multiple occasions. In one case series of nine patients, ACs were mostly within the middle cranial fossa and intracranial aneurysms involving the anterior circulation; these patients present with a mix of signs and symptoms related to aneurysm rupture or mass effect/seizures related to the ACs [13]. Meningioma and AC may also co-occur, sometimes as physically separate lesions [71] or alternately as meningioma arising from the wall of an AC. Of interest, such neoplastic transformations have included examples of higher-grade meningiomas [World Health Organization (WHO) grades II and III] [35, 56]. Histological assessment is clearly crucial in these circumstances for prognostication and appropriate treatment planning, as these higher-grade meningiomas are significantly more biologically aggressive than their WHO grade I counterparts.

5 Conclusion

ACs are benign cystic mass lesions that arise throughout the CNS in areas typically populated by arachnoid cap cells. They occur throughout all age groups and may be congenital or acquired secondary to a variety of pathologies that result in inflammation/irritation or hemorrhage involving leptomeninges. In this chapter, we outlined the typical histological, immunohistochemical,

and ultrastructural features of ACs. Some uncommon unusual histological findings of AC were also highlighted. Comparisons were drawn with a wide variety of other cystic lesions of the CNS, highlighting pertinent features to facilitate differentiation of ACs from the multitude of potential clinicoradiographic mimics. Lastly, ACs may be encountered at unusual locations or alongside other unrelated lesions; in these circumstances, histological examination is crucial to determine the ultimate diagnosis.

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