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Arachnoid cysts (ACs) are benign developmental CSF-like collections encased within an arachnoid membrane. They are randomly encountered at the surface of the brain, with a predilection for the middle cranial fossa or the cerebellopontine angle. ACs located in the sellar area represent slightly less than 10 % of all intracranial arachnoid cysts. There is no obvious sex ratio. ACs are described in the literature at all ages, but intrasellar ACs are reported more in adulthood while suprasellar ACs appear to be more frequent in the pediatric population.

Two different types of AC can be distinguished in the sellar region: intrasellar ACs, which often demonstrate a suprasellar extension that displaces the pituitary stalk posteriorly (Fig. 48.1), and suprasellar ACs, also called suprasellar-prepontine ACs, which push the floor of the third ventricle upward and the pituitary stalk forward (Fig. 48.2). The exact origin of intrasellar AC remains controversial, because there is no arachnoid membrane in the sella turcica. Therefore, an accepted hypothesis is an extension of a suprasellar AC cyst through the diaphragm aperture (Fig. 48.3).

If common intracranial ACs are typically clinically silent and almost never require surgical intervention, headache, visual disturbances, endocrine dysfunction, and hydrocephalus are frequent problems associated with AC in the sellar region. Thus, ACs may behave like any other

nonsecreting sellar lesion but may also be asymptomatic and depicted randomly on brain CT or MRI.

Preoperative diagnosis of sellar AC looks simple on CT or MRI because such cysts are only filled with CSF-like fluid whose density and signal intensities are similar to those of CSF. They appear as well-defined, sharply marginated intra-/suprasellar or suprasellar purely cystic lesions with homogeneous T1-low signal intensity and T2-high signal intensity, with no solid portion, no contrast enhancement, and no calcification. However, on T1WI, intrasellar ACs have been reported to be slightly more intense than the CSF because of stagnation of the fluid or elevated protein concentration. FLAIR sequence is of great additional value by demonstrating signal suppression of the cyst content in ACs (Figs. 48.1, 48.2, and 48.4), while this content returns high signal intensity in other cystic lesions such as cystic pituitary adenoma, Rathke cleft cyst, craniopharyngioma, or neurenteric cyst.

Other MR features of sellar ACs are due to their mass effect. The sella turcica may be markedly increased in cases of large intrasellar component, with ballooning of its floor. Visual pathways, pituitary stalk, and gland may be displaced, while brain parenchyma and third ventricle can be compressed by large cysts. As mentioned earlier, the AC wall shows no enhancement after intravenous gadolinium

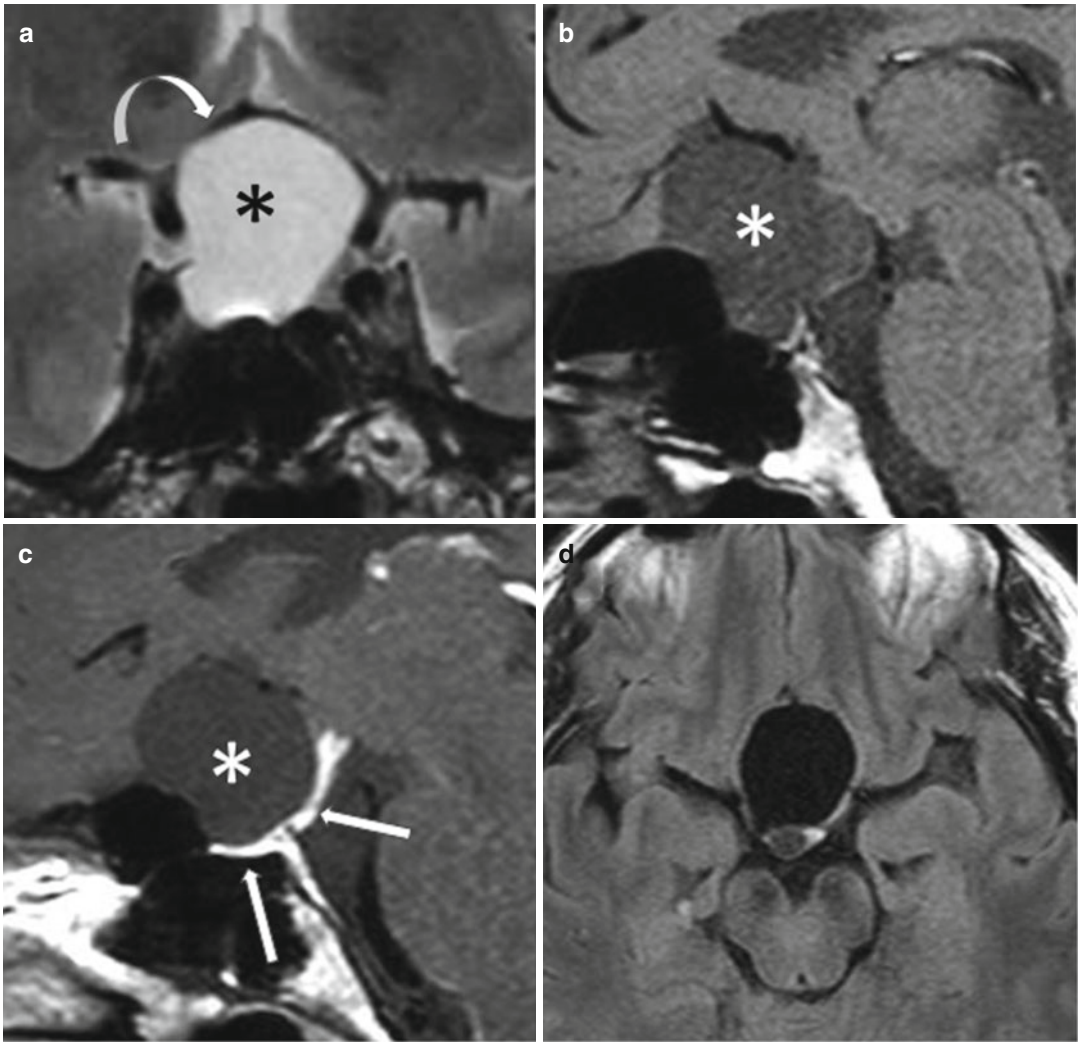


Fig. 48.1 Intra- and suprasellar AC. (a) Coronal T2WI shows a homogeneous intra-/suprasellar hyperintense cyst (*asterisk*) displacing the optic chiasm and anterior cerebral arteries (*curved arrow*) upward. (b, c) Sagittal T1WIs before and after gadolinium administration demonstrate no contrast uptake of the cyst walls. Only the normal ade-

nohypophysis and pituitary stalk pushed posteriorly are enhancing (*straight arrows*). Note that cyst signal intensities are similar to those of CSF on all sequences, especially on (d) FLAIR image, on which the signal intensity is completely attenuated

administration. Peripheral and discontinuous gadolinium uptake may, however, be depicted at the edge of the cysts and is related to normal laminated adenohypophysis or pituitary stalk (Figs. 48.1 and 48.4).

Finally, the differential diagnosis between an AC and an empty sella may not be as easy as

thought, because both entities have signal intensities similar to that of CSF. However, ACs have no communication with the surrounding subarachnoid space, which distinguishes them from an empty sella. In addition, high-resolution heavily T2W MRI sequences such as CISS, FIESTA, or DRIVE are able to

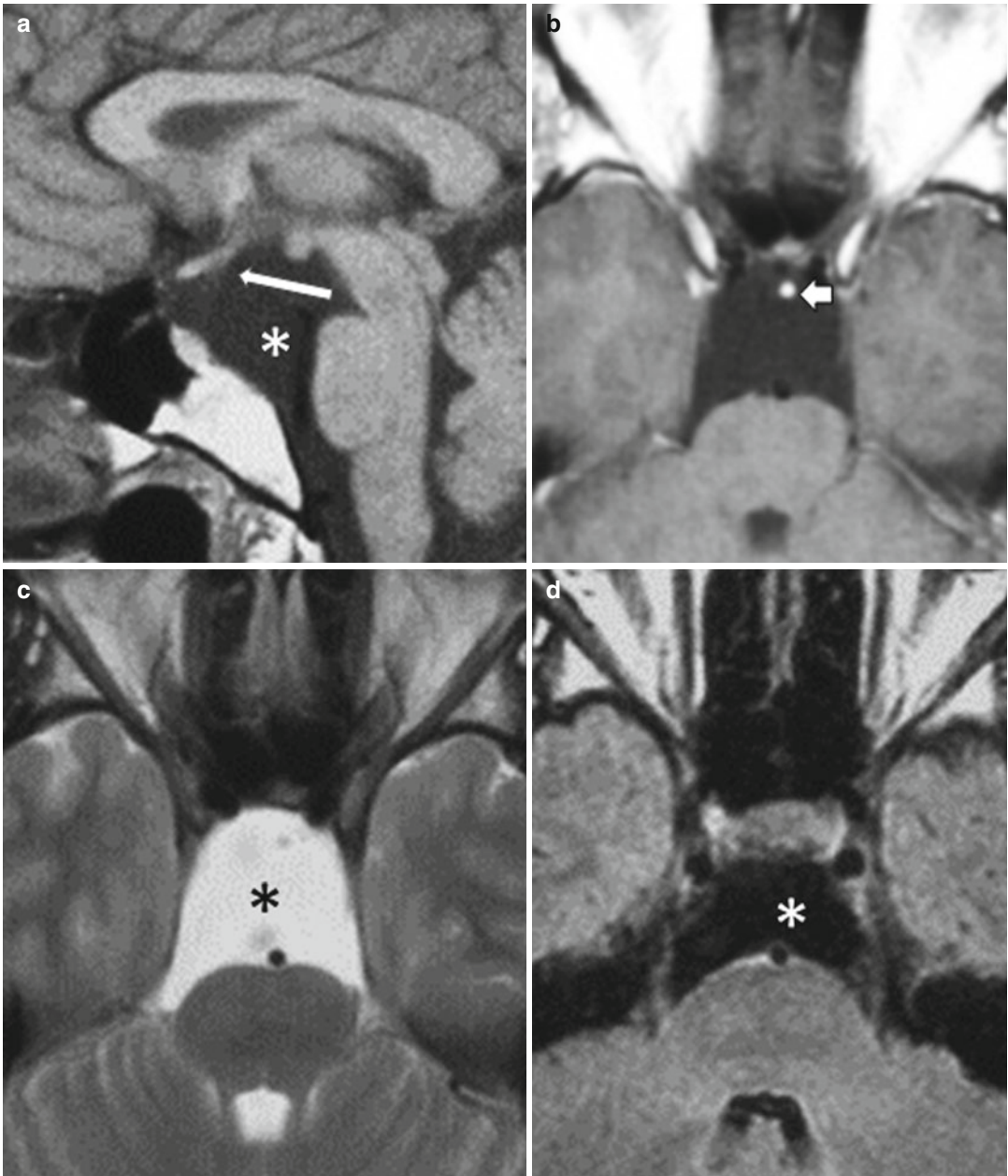


Fig. 48.2 Suprasellar AC. (a) Sagittal T1WI image illustrates the mass effect of a suprasellar-prepontine AC (asterisk) on pituitary stalk and floor of the third ventricle (arrow). The dorsum sellae is eroded. (b) Axial CE T1WI demonstrates no gadolinium uptake of the cyst wall. Only

the pituitary stalk is normally enhancing (small arrow). (c, d) Axial T2WI and FLAIR images confirm the purely CSF-like nature of the cyst content, thus confirming the diagnosis of AC

delineate the membranous borders of the AC, which do not exist in empty sella. Moreover, the diagnosis of empty sella appears less likely in cases of mass effect by the cystic lesion and

subsequent outer displacement of the parasellar structures, such as the optic nerve (Fig. 48.3), pituitary stalk, and floor of the third ventricle.

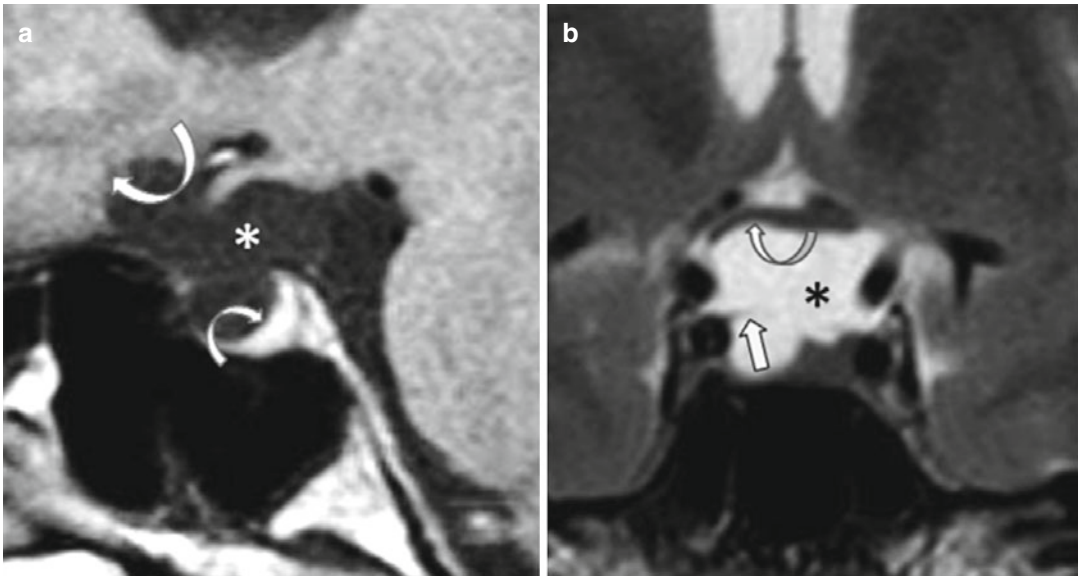


Fig. 48.3 Intra- and suprasellar AC (asterisk). (a) Sagittal T1WI shows a widening of the optochiasmatic cistern with mass effect on the superior aspect of the pituitary gland (small curved arrow) and orbitofrontal brain parenchyma (long curved arrow). (b) Coronal T2WI dem-

onstrates the focal impingement of the inferior right aspect of the optic chiasm (curved arrow) and the intrasellar extension through a large aperture of the sellar diaphragm (straight arrow)

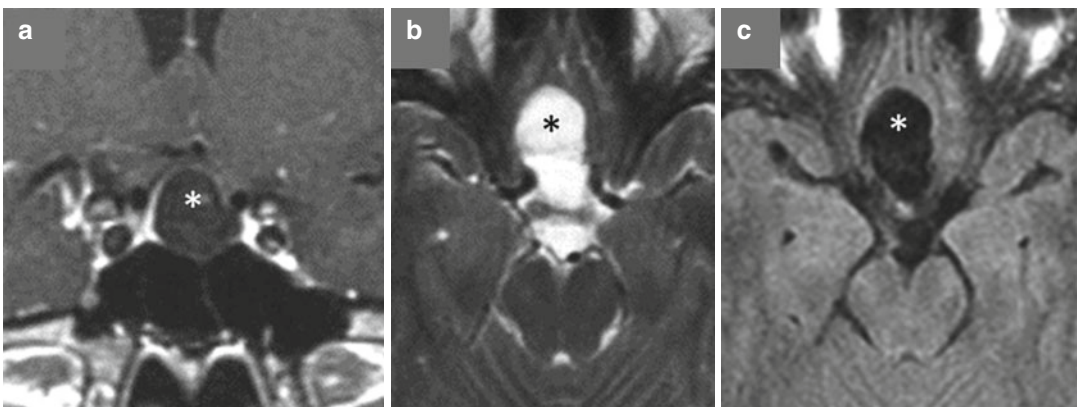


Fig. 48.4 Intra- and suprasellar AC. (a) Coronal CE T1WI shows a cystic lesion (asterisk) surrounded by a thin rim of normally enhancing pituitary tissue (arrow).

(b) Axial T2WI image depicts the suprasellar component of this AC, only filled with CSF-like fluid completely attenuated on (c) FLAIR image

Further Reading

Dubuisson AS, Stevenaert A, Martin DH et al (2007) Intrasellar arachnoid cysts. *Neurosurgery* 61:505–513

Ozek MM, Urgan K (2013) Neuroendoscopic management of suprasellar arachnoid cysts. *World Neurosurg* 79(2 Suppl):S19.e13-8
 Shim KW, Park EK, Lee YH et al (2013) Transventricular endoscopic fenestration of intrasellar arachnoid cyst. *Neurosurgery* 72:520–528