1 Developmental Origins of the Cerebral Ventricular System and Colloid Cysts

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Abstract

Colloid cysts are rare, benign brain tumors, predominantly located in the third ventricle and constitute less than 3% of all benign intracranial tumors. Although they can occur in various brain regions, the vast majority are found within the third ventricle. The genetic basis of colloid cysts remains largely undefined, although there have been reports of occasional familial instances. The etiology of colloid cysts has been a subject of debate, with theories suggesting both neuroectodermal and enterogenous origins. This chapter focuses on the embryological origins of colloid cysts within the context of nervous system development, emphasizing the anatomy around the third ventricle to understand their primary embryological sources. The development of the human ventricular system, from the closure of the neural tube to the formation of the ventricular system, underpins the understanding of colloid cyst pathophysiology. The chapter explores the differentiation of the choroid plexus and ependymal cells, shedding light on the complexities of the ventricular system and its association with colloid cyst formation. An anatomical and histopathological examination attributes the origins of colloid cysts to abnormal embryonic foldings that trap endodermal elements, suggesting that these cysts likely originate from detached embryonic diencephalic vesicular recesses or remnants of the paraphysis cerebri. This insight provides a comprehensive understanding of colloid cyst development and its clinical implications, highlighting the significance of embryological perspectives in diagnosing and managing these tumors.

Keywords: development, embryology, paraphysis, colloid cyst, cerebral ventricle, ependyma, neural tube

1.1 Background

Colloid cysts are slow-growing developmental lesions that constitute approximately 0.2 to 3% of all benign brain tumors.[1,2,3,4,5,6] Although they have been identified in many regions of the brain including the fourth ventricle,[7,8] parenchyma,[9] sella,[10] and within the leaflets of the septum pellucidum,[6,11,12,13,14] approximately 99% of identified colloid cysts are located within the third ventricle[15] (▶ Fig. 1.1). The role of genetics in colloid cysts development has not been characterized; however, rare familial clusters have been documented.[16,17,18,19,20] Since colloid cysts were first reported in 1858,[21] there has been considerable debate on their pathophysiology and embryological origins.[22] Indeed, several studies have implicated structures within the vicinity of the third ventricle, including the paraphysis cerebri,[23,24] velum interpositum,[11,22] ependyma, and choroid plexus neuroepithelium,[23,25] as potential embryological origins. Other studies have suggested an enterogenous origin (e.g., respiratory epithelium[26,27,28]).

The objective of this chapter is to describe the current thinking regarding the embryological basis of colloid cysts, particularly in the context of the development of the nervous system. A close examination of the anatomical structures within the vicinity of the third ventricle will provide the background to understand the embryological origins of the vast majority of colloid cysts.

1.2 Neurodevelopment and the Cerebral Ventricles

Human development originates from three primitive embryonic layers, namely, ectoderm, mesoderm, and endoderm, which are formed through purposeful cell movement and organization following fertilization.[29] During neurulation, around 2 weeks of embryonic development, the neural plate thickens and invaginates into a neural groove along its midline, which then fuses into a rostro-caudal neural tube.[29,30] The neural tube momentarily remains in communication with the surrounding environment via its rostral (anterior) and caudal (posterior) neuropores, until they both eventually close off to form the closed ventricular system[29] (▶ Fig. 1.2). The closed anterior neuropore can later be identified as the *lamina terminalis* in the developed brain.

During neurulation, the anterior and posterior neuropores close to isolate the ventricular system from its embryonic environment. Formation of the five-vesicle complex structure and differential expansion of regions around the neural tube flexures along with the neural canal create the cerebral ventricles.

Around week 3, the rostral end of the neural tube undergoes rapid morphogenetic movement and expansions to develop three symmetric primary swellings: the prosencephalon (forebrain), mesencephalon (midbrain), and rhombencephalon (hindbrain). The three-vesicle stage is facilitated by symmetric folding of the neural tube rostrally along its lateral axis to form two flexures: the cephalic flexure which develops between the prosencephalon and mesencephalon, and cervical flexure which divides the rhombencephalon and the spinal cord.[29] Due to significantly restricted morphogenesis at the cephalic flexure in relation to the rest of the prosencephalon, two distinct structures develop: the telencephalon rostrally and the diencephalon caudally. At approximately 6 weeks, the pontine flexure develops to demarcate the rhombencephalon into myelencephalon and metencephalon. Thus, by week 7, the three primary vesicles have developed into five secondary vesicles,[29] which are (from rostral to caudal): telencephalon (*future cerebral hemispheres*), diencephalon (*future thalami, hypothalamic and optic cups*) mesencephalon (*future midbrain*), metencephalon (*future pons and cerebellum*), and myelencephalon (*future medulla oblongata*)[29] (▶ Fig. 1.2).

Development of the ventricular system is a fascinating and clinically important topic in its own right and is integral to understanding the complexities of ventricular-associated pathologies such as colloid cysts. In the developed human brain, the ventricular system comprises of two lateral (telencephalic) ventricles that connect through the foramen of Monro to the third (diencephalic) ventricle, which in turn connects to the fourth

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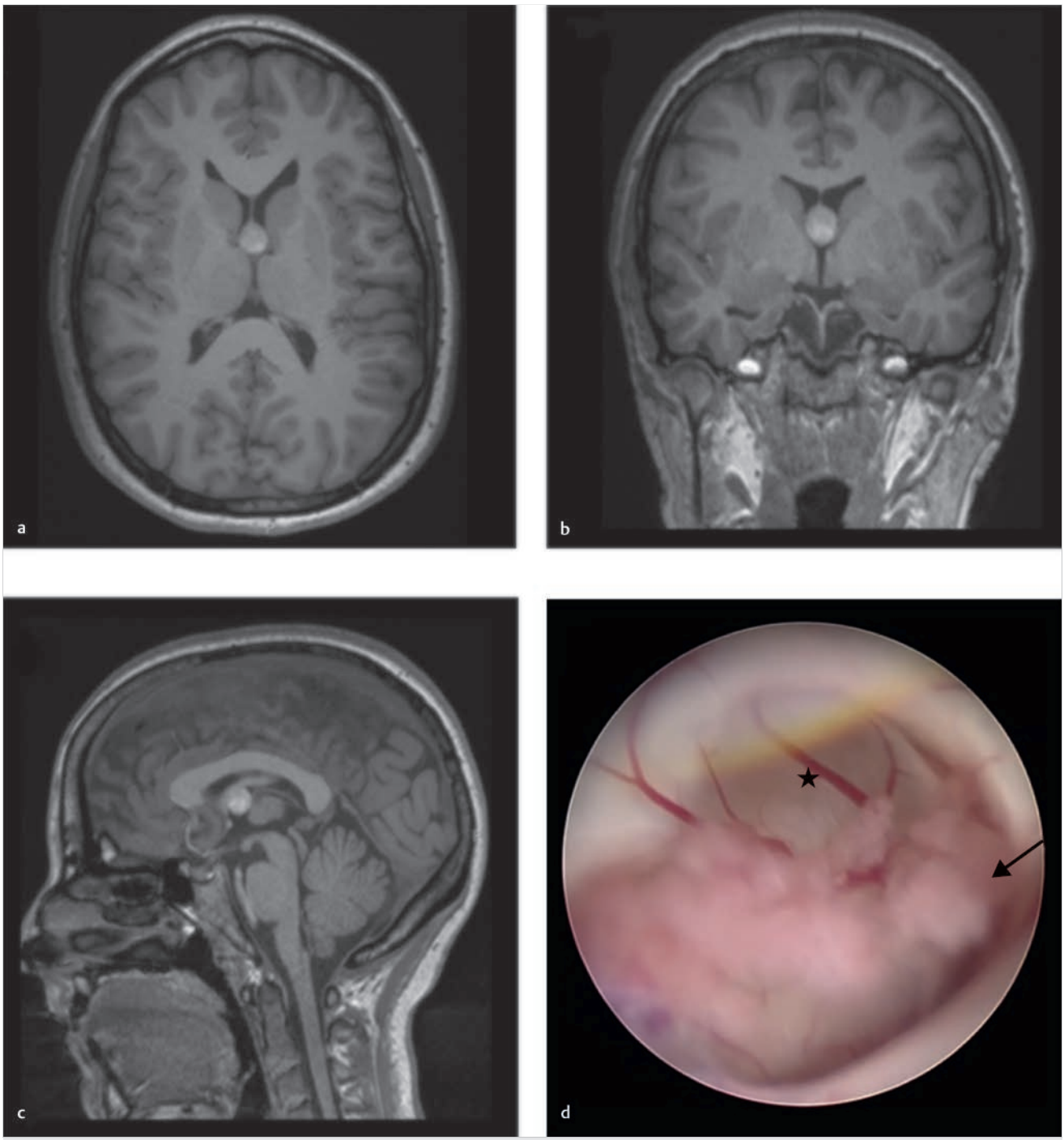

Fig. 1.1 Case presentation. Representative axial **(a)**, coronal **(b)**, and sagittal **(c)** T1-weighted (T1W) magnetic resonance imaging (MRI) images of a 21-year-old female demonstrating a colloid cyst located at the roof of the third ventricle that appears to obstruct the foramina of Monro, but without features of hydrocephalus. An endoscopic view **(d)** showing the cyst (★) with a vascularized smooth glistening wall draped by choroid plexus (*arrow*) as in the foramen of Monro.

(rhombencephalic) ventricle via the cerebral aqueduct. Although the ventricular system is continuous with the central canal of the spinal cord during development, and potentially thereafter, it communicates with the subarachnoid space through the foramen of Magendie and foramina of Lushka at the level of the pons.

Embryonic ventricular development is heralded by closure of the anterior and posterior neuropores, which are connected midline by a cerebrospinal fluid (CSF)-filled lumen lined by ependymal cells—the neural canal (▶ Fig. 1.2). Development of the three-dimensional ventricular structure from the primitive neural tube configuration is mediated by complex mechanisms of morphogenesis, which include both physical forces and interactions that occur concurrently with neural tube expansion. Physical forces are created through osmotic absorption of water

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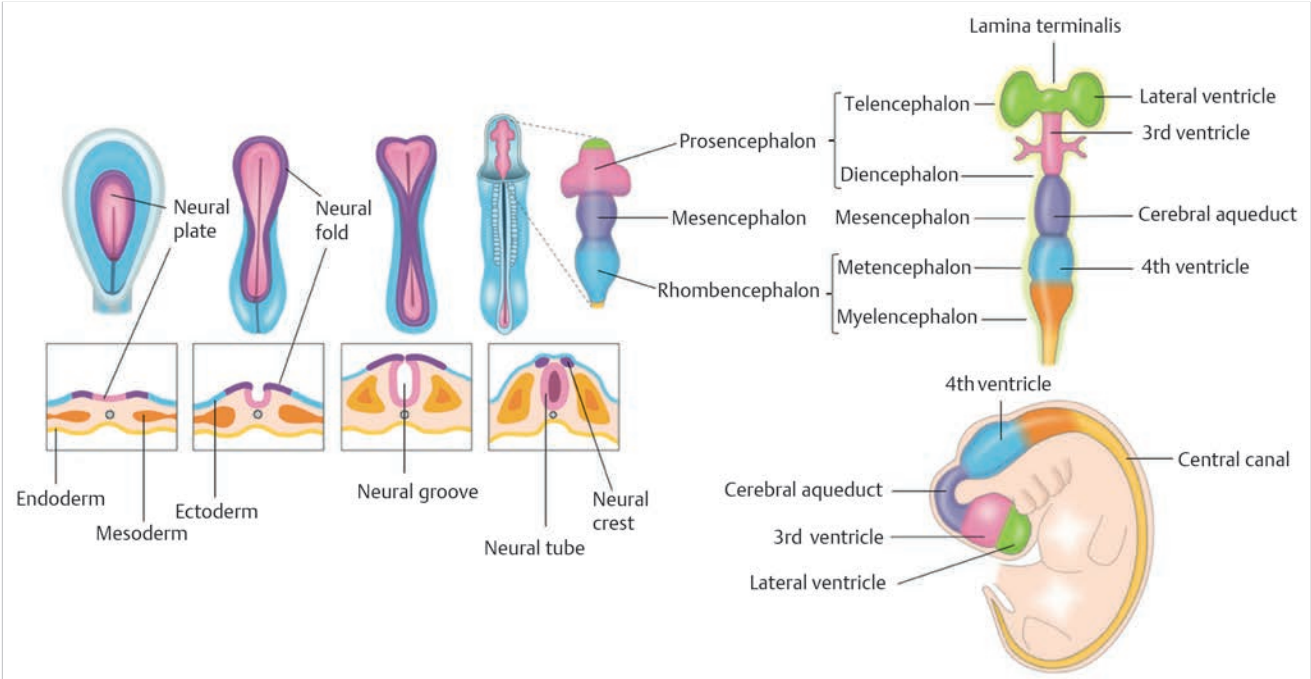

Fig. 1.2 Embryological origins of ventricular development.

by cells lining the primitive ventricles and by secretion of fluid into the ventricular cavity by the choroid plexus (see below for details of choroid plexus development). The segment of the neural canal that spans the telencephalon develops into the *lateral ventricles* (▶ Fig. 1.2). The lateral ventricles maintain a large, inflated shape due to massive CSF production by the choroid plexus in those regions. The C-shaped configuration of the lateral ventricles occurs as a result of ventromedial expansion of the ganglionic eminence bilaterally.[29] The lateral ventricles and the fornices are separated later around weeks 10 to 12[31] into left and right by a thin membranous bridge, the *septum pellucidum*. The septum pellucidum develops inextricably with the corpus callosum and anterior and hippocampal commissures from the basal medial portion of the *lamina terminalis* known as the commissural plate[31,32,33] (▶ Fig. 1.3). During week 6 of gestation, the *foramen of Monro* is formed from the anterior enlargement of the initial slit-like communication between the diencephalon and bilateral telencephalon vesicles (▶ Fig. 1.2). The *velum interpositum* on the other hand derives from duplication of the pia–arachnoid layer that overlies the roof of the diencephalon and superior walls of the telencephalon, as they undergo a U-shaped infolding (▶ Fig. 1.3). While the cavity in the diencephalon forms the *third ventricle*, the *cerebral aqueduct* forms from the remnant of the neural canal at the level of the mesencephalon (▶ Fig. 1.2). The fourth ventricle develops from splaying of the pontine flexure (▶ Fig. 1.2). Unlike the fourth ventricle which enlarges during formation of the rostral neural tube structures, the cerebral aqueduct is relatively large during early embryological stages, but later narrows. The remainder of the neural canal stretches inferiorly as the central canal of the spinal cord.

1.2.1 Ependyma Development

During neurodevelopment, progenitor stem cell proliferation occurs in the ventricular zone, an area immediately *adjacent* to the ventricles. The stem cells either migrate via radial glial cells to differentiate into neurons of the cerebral cortex or supporting glial structures, which include ependymal cells that form the ventricular CSF–brain barrier.[34] During neurulation, the ventricular zone spans the entire neural tube, but as the brain matures it is replaced by the ependymal layer and limited portions of the stem cells are largely limited to specific areas of the subventricular zone. These stem cells retain restorative and repair capabilities, for example, regenerating neurons of the olfactory bulb and dentate gyrus via the rostral migratory stream.[35,36] Histologically, the ependyma contains multiciliated cuboidal cells with cilia that, among other purposes, may facilitate the movement of CSF through the ventricular system.

1.2.2 Choroid Plexus Development

As development of the neural tube progresses, a shallow sulcus limitans at the caudal end demarcates the roof and alar plates from the floor and basal plates. Further differentiation of non-neural tissues at the roof plate forms the highly vascularized choroid plexus that are found within each cerebral ventricle.[37] After neural tube closure, the choroid plexus in the fourth ventricle appear first, which is followed by those of the lateral ventricles synchronously, and then lastly in the third ventricle.[38] Despite their distinct appearance, the choroid plexuses in the lateral ventricles from the choroid fissures merge at the exit levels of foramina of Monro, and fuse with the third ventricle plexus to form a continuous choroid plexus structure.[38,39,40] The

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many functions of the choroid plexus have been extensively described in the literature and include CSF secretion,[38,41] solute transport,[42] and immune system regulation.[43] Histologically, the choroid plexus comprises of a network of connective tissues and fenestrated capillaries, surrounded by cuboidal epithelial cells that are held together by tight and adherens junctions to form the blood–CSF barrier.

1.2.3 Development of Colloid Cysts

Given the intimate anatomical proximity of the sellar region to the third ventricle, the differential diagnoses of colloid cysts may include other cystic lesions that originate in the vicinity, such as Rathke's cleft cysts and cystic craniopharyngiomas.[44] Rathke's cleft cysts develop between the lobes of the pituitary as cystic remnants of the craniopharyngeal duct, while craniopharyngiomas are considered neoplastic transformation of ectodermal-derived epithelial cell remnants of the craniopharyngeal duct and Rathke's pouch.[45,46] Although the embryological origins of Rathke's cleft cysts and craniopharyngiomas are considered by many to be distinct from colloid cysts, it has been hypothesized that the presence of some endodermal features that are common to colloid cysts and Rathke's cleft cysts suggests they may be embryologically related.[47]

Third Ventricle Anatomy

Majority of colloid cysts originate from structures around the roof of the third ventricle. The third ventricle is defined by the foramen of Monro, lamina terminalis, columns of the fornix, and the anterior commissure, while the posterior border is defined by the cerebral aqueduct and the suprapineal recess[48] (▶ Fig. 1.3). The two lateral walls are defined by the left and right diencephalon, which comprises of the thalami, interthalamic adhesion, hypothalami, paraventricular nuclei, and subthalami. The floor extends from the optic chiasm anteriorly to the orifice of the cerebral aqueduct posteriorly with intermediary structures such as the infundibular recess, tuber cinereum, mamillary bodies, posterior perforated substance, and tegmentum[48] (▶ Fig. 1.3).

The roof of the third ventricle, where majority of colloid cysts are located, is defined superiorly to inferiorly by five layers. The first layer, the superior layer, is formed by the fornix. The next two layers, the tela choroidea layers, are characterized by a bundle of choroid plexus around the choroidal fissures that attaches to the choroid plexus of the third ventricle. The fourth layer, velum interpositum, is a vascular layer that contains the internal cerebral and medial posterior choroidal arteries. The fifth layer is the choroid plexus layer (▶ Fig. 1.3).

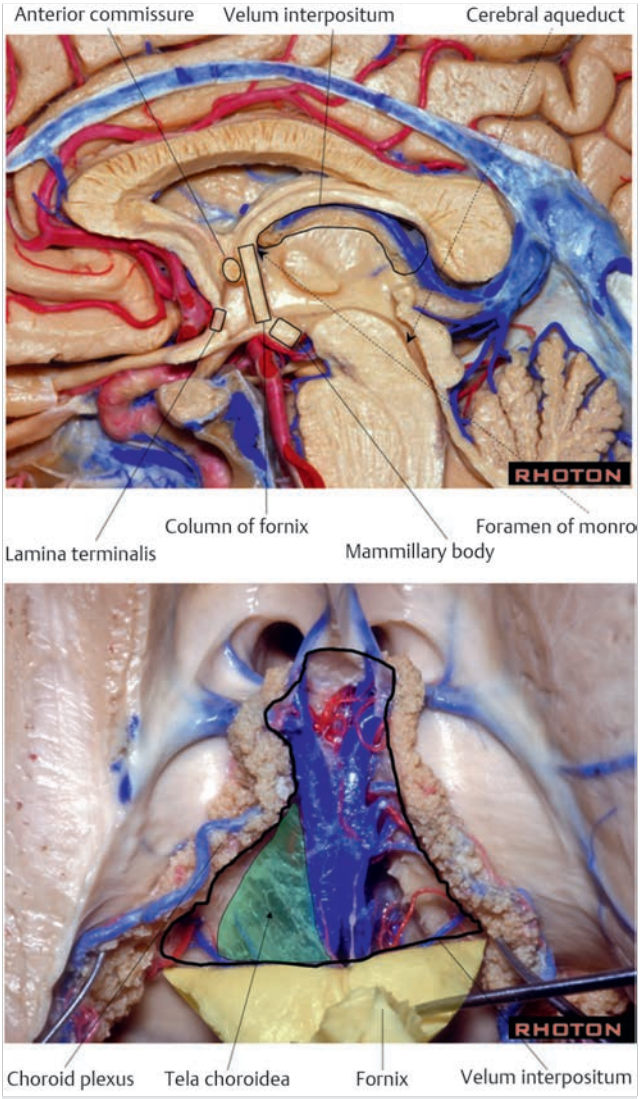

Fig. 1.3 Borders and roof of the third ventricle. *Top:* Sagittal view of third ventricular anatomy demonstrating the anterior border demarcated by the lamina terminalis, anterior commissure, and column of fornix, and posterior border demarcated by the cerebral aqueduct and suprapineal recess. The velum interpositum is seen at the roof. *Bottom:* A superior view of the roof of the third ventricle demonstrating structures that form its five layers; layer 1 (fornix) has been reflected to reveal layers 2 and 3 (telea) and the demarcated layer 4 (velum interpositum). The choroid plexus is labelled to depict layer 5 (choroid plexus layer). (Used with permission from the Rhoton Collection, American Association of Neurological Surgeons [AANS]/Neurosurgical Research and Education Foundation [NREF].)

Histopathology of Colloid Cysts

On gross pathology, colloid cysts are typically spherical to ovoid in shape with a smooth, partially translucent cyst wall (▶ Fig. 1.1). Their contents are highly variable and can range from clear, semi-viscous fluid to gelatinous or even rock solid[49,50,51] (▶ Fig. 1.4). Histological analyses of explanted colloid cysts demonstrate a fibrous collagenous outer layer.[52] Piloid gliosis with Rosenthal material may also be seen in the cyst wall bed, as can sometimes adhered choroid plexus tissue (▶ Fig. 1.4). The lining epithelium is variable, albeit often well-differentiated and mature, and may contain columnar to cuboidal epithelium with or without a pseudostratified appearance with mucin secretory organelles[52,53] (▶ Fig. 1.4). It is possible that the secretory activity of mucin organelles is partly responsible for the slow enlargement of colloid cysts,[54] as symptomatic colloid cysts, which tend be larger than nonsymptomatic ones, and often demonstrate a T2-weighted (T2W) signal indicative of relatively higher mucinous content. However, the osmotic effects of intracystic mucin-laden electrolytes[55] such as sodium, calcium, and magnesium that may draw fluid into the cyst cannot be discounted. Majority

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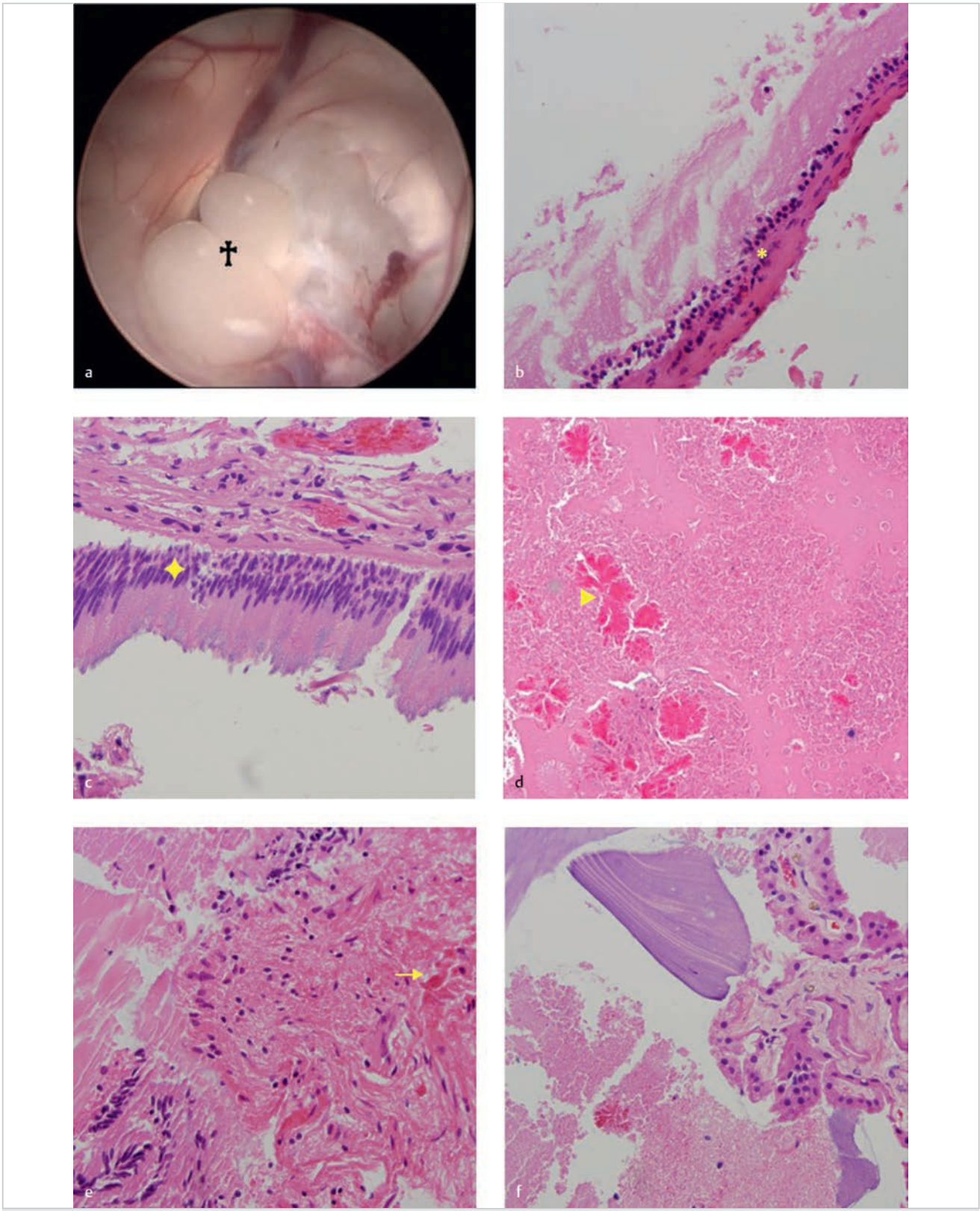

Fig. 1.4 Representative pathological and histological features a colloid cyst. Intraoperative endoscopic image of a colloid cyst **(a)** showing extruded gelatinous cyst contents following surgical opening of the cyst wall. The H&E stained images of an explanted colloid cyst demonstrating a thin fibrous layer and simple cuboidal to columnar epithelium (**b**, ✻) to pseudostratified mucinous epithelium (**c**, ✦) from two different examples. Often the predominant pathology material is composed of acellular proteinaceous material, that is, cyst contents **(d)**, which can sometimes form small aggregates resembling crystalloids (*arrowhead*). Piloid gliosis with Rosenthal material (*arrow*) may also be seen in the cyst wall bed **(e)**, and sometimes adhered choroid plexus tissue also **(f)**.

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of colloid cysts also contain cilia and basal bodies or mucin (resembling bronchogenic epithelium)[26,28,49,52,56]; however, variants that lack cilia have been reported.[26,28] Commonly, fragments of normal choroid plexus may be found attached to the cyst (▶ Fig. 1.4). Rarely, intralesional hemorrhage due to colloid cyst apoplexy[57,58] may be identified.

To correlate colloid cysts with potential structures of their origin, several stains and antibodies have been employed on explanted colloid cysts to better characterize their microstructure, some of which have been outlined in ▶ Table 1.1.

Table 1.1 Histological stains and antibodies that have been used to characterize the cellular microstructure of colloid cysts

Stain/Antibody	Result
Epithelial membrane antigen	Positive[52,59,60]
Keratin	Positive[52,59]
PAS	Positive[6,52,61]
S100	Positive[52]
Trichrome	Positive[52,62]
Cytokeratin (CAM 5.2)	Positive[63]
GFAP	Negative[52]
Monoclonal antibody HNK-1	Negative[59]
Leukocyte antigen	Negative[52,59]
Neurofilament	Negative[52]
Silver	Negative[52]
Enolase	Inconsistent[52,59]
Vimentin	Inconsistent[52]

Abbreviations: GFAP, glial fibrillary acidic protein; PAS, periodic acid-Schiff stain.

Paraphyseal Origin of Colloid Cysts

Sjövall first described the etiology of colloid cysts as remnants of nondegenerated paraphysis,[24] an embryonic structure that develops from the neuroectodermal paraphyseal arch,[23] which is anatomically located at the border of the diencephalon and the telencephalon in the developing embryo[23,24] (▶ Fig. 1.2). In later development, the paraphyseal arch defines the roof of the third ventricle.[23,24] The paraphysis starts around week 7 of embryonic development, at approximately 15- to 17-mm embryonic crown–rump (CR) length, as a solid bud that projects from the paraphyseal arch.[23] The bud then forms one or more lumens, which subsequently develop into vesicles (▶ Fig. 1.5). Typically, there is significant variation in the rates and patterns of differentiation of the vesicles. However, as the embryo develops further, the principal vesicular anlagen, the most developed midline paraphyseal vesicle, persists near the roof of the third ventricle, while the remaining vesicles regress.[23] Complete degeneration of all vesicular rudiments is normally observed by 3.5 months of gestation at approximately 100-mm CR length.[23] However, occasionally one or more vesicular remnants fail to regress, which may later develop into a colloid cyst. McLean postulated that it is the weight of the paraphyseal cysts that projects it downward into the third ventricle from the velum interpositum.[64] It remains unclear whether one or a coalescing of two or more persistent vesicular rudiments develop into a colloid cyst. Nevertheless, given their anatomical location, embryological development, and histological features that are for the most part similar to that of colloid cysts, the paraphysis has been hypothesized as the precursor to colloid cysts for many decades.[8,13,23,24,52,65,66,67,68]

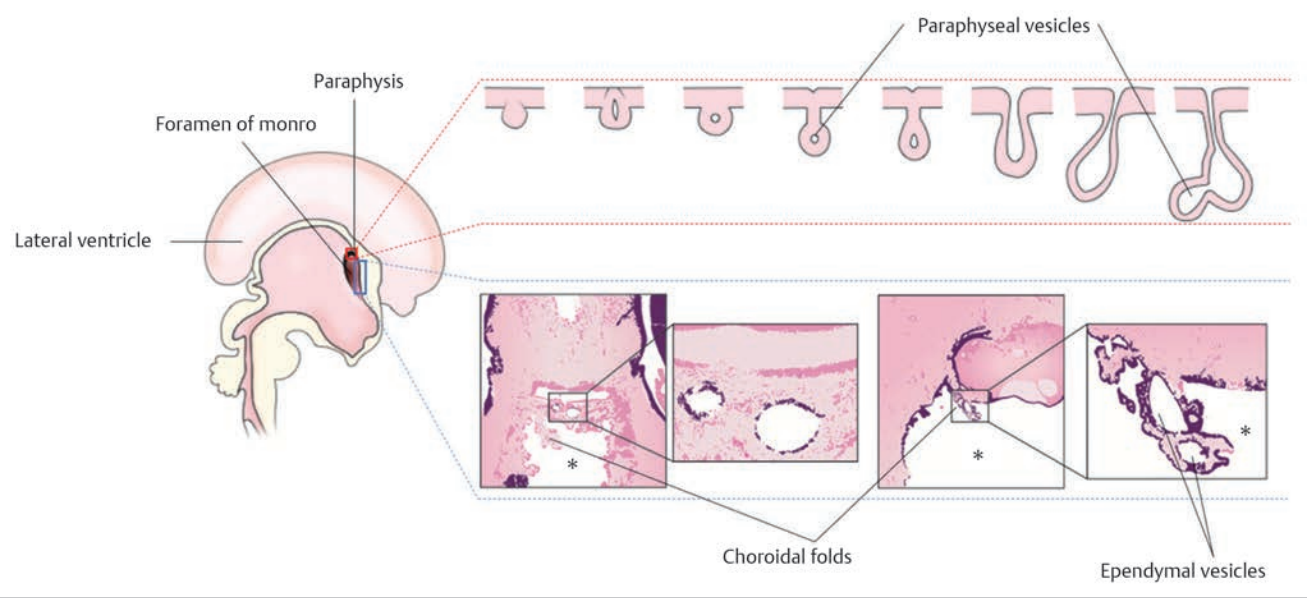

Fig. 1.5 Schematic representation of colloid cyst development demonstrating paraphyseal and ependymal origins of a third ventricle colloid cyst, as described by Kappers.[23] A solid paraphyseal bud develops from the paraphyseal bud around embryonic week 7. The bud then develops a lumen, and several vesicles may form. A vesicular anlage that fails to regress gets incorporated into a choroidal fold. An ependymal recess may also detach as a closed vesicle and get incorporated into a choroidal fold. A choroidal fold containing paraphyseal or ependymal vesicular anlage may hang from the superior aspect of the third ventricle (*) as a colloid cyst.

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Ependymal Origin of Colloid Cysts

Although the paraphysis is arguably one of the most likely referenced etiologies for third ventricle colloid cysts,[8,13,23,24,52,65,66,67,68] it is important to note that there are several findings that do not fit this narrative. For example, a paraphyseal origin does not explain posteriorly located third ventricle colloid cysts,[52,69] let alone those that are found in the fourth ventricle[7,8] or at extraventricular sites.[6,9,10,11,12,13,14] In addition, majority of explanted colloid cysts demonstrate histological features of cilia while the paraphysis is noncilliated,[22,23] although Ho and Garcia[26] and Macaulay et al[28] both reported a mixed population of ciliated and nonciliated cells in their respective cohorts of explanted colloid cysts.[26,28]

Considering the shortfalls of the hypothesis of paraphysis-based pathophysiology, Kappers[23] suggested that colloid cysts arise from recesses that develop in the ependyma of the diencephalic postvelar arch, which then detach as closed vesicles surrounded by connective tissues of the velum interpositum[24] (▶ Fig. 1.5). However, based on reports of paraphyseal rudiments found to be buried within choroidal folds of patients with colloid cysts,[25] Kappers suggested the paraphysis is plausible, yet an uncommon, precursor of colloid cysts.[23] However, a distinction can be made in that cysts with cilia are more likely to be of ependymal origin, whereas those that lack cilia may be of paraphyseal origin.[23] Kappers's proposed mechanisms of third ventricle colloid cyst development[23] is perhaps the most comprehensive pathophysiological perspective to date.[22] For one, it considers the intricate anatomic relationships between diencephalic–telencephalic junctional structures such as the third ventricle, foramina of Monro, ependymal layer, and choroid plexus to propose the development of colloid cysts.[23] Second, in addition to recognizing the paraphysis as a potential origin of colloid cysts,[24] Kappers's ependymal origin helps reconcile presence of cilia in colloid cysts, which the paraphysis theory was unable to address.[22,23] From an anatomical perspective, both of the paraphyseal and ependymal derived colloid cysts[23] appear to originate from Zone I of Beaumont et al's three-zone stratification of the third ventricle into colloid cyst risk zones.[48] Zone I, defined as the region of the third ventricle anterior to a line tangential to the mamillary body and the massa intermedia, is also associated with nearly all colloid cysts that present with pre-aqueductal hydrocephalus.[48]

1.3 Conclusion

Majority of colloid cysts are consistently located in the anterior roof of the third ventricle, near foramen of Monroe, and are thought to originate from abnormal folding of the primitive neuroectoderm resulting in trapping of endodermal elements within velum interpositum in the course of the later stages of primary neurulation. Although there have been several theories on the embryological basis of colloid cysts, a unifying hypothesis is that majority of the lesions arise from detached and degenerated embryonic diencephalic vesicular recesses, while some may develop from remnants of the paraphysis cerebri.

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