Commentary

The authors have reported an extremely rare case of an unusual association between a suprasellar dermoid cyst and a colloid cyst of the third ventricle in a 22-year-old man.^[1] To the best of the authors' knowledge, only one similar case has been reported previously.^[2]

Both dermoid cysts and colloid cysts belong to the class of benign brain tumors caused by disordered embryogenesis.^[3] The incidences of dermoid cysts and colloid cysts among all brain tumors are reported to be 0.3% and 0.4%, respectively.^[3,4] Although dermoid cysts are known to be derived from the ectoderm, the pathogenesis of colloid cysts is debatable – these cysts are derived from either an endodermal or ectodermal source. Hirano *et al.* suggested that colloid cysts arise from an endodermal source because the secretory cells of a colloid cyst are covered by an electron-dense surface coat that is not found in the neuroepithelium at any stage of development.^[5] On the other hand, Nagaraju *et al.* recently reported a case of a colloid cyst of the third ventricle, which was likely of neuroepithelial origin.^[6] The authors supported the latter case, and hence, they have focused on the “anterior neuropore corridor defects” theory proposed by Cheng *et al.* in 1999.^[2] As dermal sinus in the lumbosacral area is well known to be caused by caudal neural tube defects, this theory is based on a continuous spectrum of abnormalities that involves an entire range of midline defects in a corridor extending from the nasal dorsum and plate through the anterior midline skull base to the anterior portion of the third ventricle.^[2] When the embryonic epithelial remnants (ectodermal origin) migrate through the anterior neuropore corridor, a dermoid cyst may develop in the sella, and a colloid cyst might develop simultaneously in the anterior third ventricle.

However, there are several drawbacks of this report. First, no histopathological evidence is provided to support the epithelial origin of the colloid cyst in this patient. Second, the types of malformations caused by the anterior neuropore corridor defect in the patient remain unclear. Third, the presence of the colloid cyst during previous examinations also remains unclear. Considering the slow growing nature of colloid cysts, the cyst might have been developing in the third ventricle during fetal life or relatively soon after birth. Finally, very few studies support the “anterior neuropore corridor defects” theory; therefore, the authors and I were unable to find a similar case in the literature. Further research and documentation of such cases along with detailed histopathological examinations of colloid cysts of the third ventricle are needed.

Besides the theory of “anterior neuropore corridor defects,” this case also highlights the fact that clinicians should not rule out a possibility of synchronous tumors in a patient. The proverb that “why only one and not two?” should be kept in mind.

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