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Pituitary Colloid Cyst

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Abstract: Colloid cysts appear most commonly in the third ventricle, their occurrence in the sellar region is uncommon. The authors report a female patient with a pituitary colloid cyst. She was diagnosed incidentally with a sellar lesion by a routine paranasal computed tomography examination performed for planning of a dental implant surgery. Radiologic examinations revealed a pituitary lesion that was removed by transnasal transsphenoidal route. Her pathologic examination revealed that the lesion was a colloid cyst. Although rare, colloid cysts should be considered in the differential diagnosis of pituitary lesions

Key Words: Colloid cyst, pituitary colloid cyst, pituitary cysts, sellar cysts

Colloid cysts are rare intracranial neoplasms accounting for 0.2% to 2% of all intracranial tumors. They usually arise from the roof of the anterior third ventricle, constituting 15% to 20% of all intraventricular masses. Extraventricular locations have rarely been reported including cerebellum, olfactory groove, optic chiasm, cerebral hemisphere, fourth ventricle, brainstem, pituitary gland, and velum interpositum in addition to suprasellar involvement.[1]

Embryologically they were thought to have a neuroepithelial origin, immunohistochemical studies of colloid cyst epithelium demonstrated endodermal rather than neuroectodermal characteristics.[2]

Although histopathologically they are benign lesions, unexpected and potentially lethal complications may develop in accordance with the localization.[1]

In the current report, we present the first well-documented pituitary colloid cyst radiologically and histopathologically.

CLINICAL REPORT

History

A 27-year-old previously healthy, having a 2-year-old daughter woman was referred to our clinic for a pituitary cystic lesion diagnosed incidentally on paranasal computed tomography (CT) during work-up for the dental implantation treatment. The patient did not have a history of galactorrhea or irregular menstrual cycles. The results of neurological and ophthalmological examination including visual acuity, fundoscopy, and visual field studies were unremarkable with no focal sign. The complete blood count, routine biochemical tests, urinalysis were all within normal limits. Pituitary hormone profiles yielded a slightly decreased prolactin (3.88, range 4.79–23.3 ng/mL) and elevated thyroid stimulating hormone (5.03, range 0.27–4.2 uIU/mL) levels.

Imaging

Anteroposterior/lateral craniographies and sella spot graphy were normal. Dynamic pituitary–magnetic resonance imaging (MRI) study revealed a well-defined, round, $14 \times 8 \times 10$ mm. Cystic lesion located between the anterior and posterior lobes of the pituitary gland displacing the stalk to the left and superiorly and compressing the optic chiasm. The lesion was uniformly iso- to hyperintense on T1- and profoundly hypointense in T2-weighted sequences. Contrast enhancement was not seen throughout the lesion including the cyst wall (Fig. 1).

Based on the lesion's cystic nature, homogeneity and enhancement pattern pituitary adenoma, craniopharyngioma, and Rathke cleft cyst (RCC) were considered in the differential diagnosis.

Operation

The patient underwent microsurgical resection of the lesion through a transnasal transsphenoidal approach (Fig. 2). Intraoperatively, a white cystic lesion having a thin, smooth capsule, filled with a white mucoid content was noted. Some parts of the cyst wall were harder and white-yellow in color. The cyst content was white, viscous, and oozed easily when we opened the cyst wall. The lesion was easily dissected from the normal hypophysis. Postoperative 24-hour control MRI verified that the lesion was totally removed (Fig. 3).

Histopathology

Pathologically, the cyst wall was made of mono-/multilayered ciliated cuboidal epithelium with focal pseudostratifications. There were randomly distributed goblet cells that were full of glycoprotein in periodic acid Schiff pretreated with diastase (PAS/PASD), and showing cytoplasmic acid mucopolysaccharides collection in PAS-AB (Fig. 4A and B). Gomori stain showed dense reticulum fibers in the cyst wall, and hyalinized connective tissue was present on the outer side of the cyst wall. The cyst content was consistent with colloid cyst (Fig. 4C) Immunohistochemical examination was performed using glial fibrillary acidic protein (GFAP), epithelial membrane antigen, ki-67, synaptophysin, p53, CK7, CK-20, and S-100 antibodies of which only epithelial membrane antigen was found to be positive (Fig. 4D). The presence of normal adenohypophysis was present.

Postoperative Course

Patient was started on oral desmopressin treatment due to diabetes insipidus developed on the postoperative third day and continued to use for 2 months until the resolution of the complication. A control MRI obtained 5 months after the operation demonstrated neither recurrence nor residue of the colloid cyst (Fig. 3).

DISCUSSION

Colloid cysts are rarely seen intracranial lesions usually located within the ventricular system, particularly the third ventricle.

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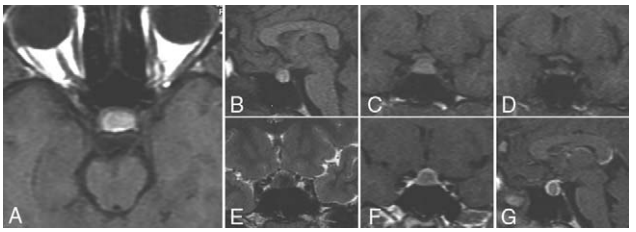

FIGURE 1. (A–C) Preoperative axial sagittal and coronal T1-weighted MRI studies showing an iso- to hyperintense round lesion located in the pituitary causing compression of the optic chiasm. (D) Pituitary stalk was displaced laterally to the left. (E) The lesion was homogenous and hypointense compared with the normal pituitary in T2-weighted images. (F, G) The lesion was not enhancing while the normal pituitary, surrounding the lesion was enhancing.

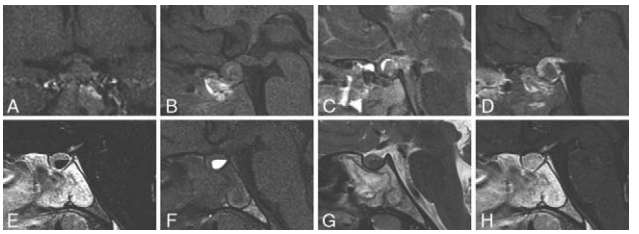

FIGURE 3. (A–D) Postoperative 24 hours. MRI study of the patient, the stalk is seen and there is no compression on the optic chiasm, in sagittal T2-weighted images there is hyperintense signal in the place of the removed cyst, in enhanced T1-weighted sagittal image the normal pituitary is seen enhancing. (E–H) Postoperative 5 months control MRI. (E) Subtracted image show the enhancing normal pituitary gland and the space of the removed cyst in between the anterior and posterior pituitary is seen. (F, G) The removed cyst space and the normal pituitary is seen and in (H) diffuse enhancement of the normal pituitary gland is shown.

Extraventricular involvement is uncommon and few patients for a specific localization were reported in the literature.[1] Although they were included in patient series involving the sellar region in the literature, the histologic criteria were not defined to establish the diagnosis as was the only clinical report by Bladowska.[3,4]

Although pathological definition is not simple, the histopathologic examination remains the standard diagnostic method. Colloid cysts have an outer fibrous capsule and an inner lining of a single layer of squamous, cuboidal, or columnar ciliated, or nonciliated epithelium. The cyst consists of gelatinous material that reacts positively to PAS staining.[2]

Computed tomography and MRI can be used in diagnosis of colloid cysts. On CT images the lesion is usually seen as hyperdense or rarely as hypodense or isodense. The MRI findings depend on the composition of the cyst content, that is, cerebrospinal fluid like or mucous cyst content. Approximately half of the cysts are hyperintense on T1-weighted images, whereas T2-weighted images are variable. Colloid cysts do not show enhancement even in the cyst wall.[3] In our patient, the lesion was uniformly isointense on T1- and profoundly hypointense in T2-weighted sequences. Contrast enhancement was not seen throughout the lesion including the cyst wall.

In contrast to ventricular localization where most of the colloid cysts originate from, the differential diagnosis from other cystic lesions of the sellar region such as RCC, arachnoid cyst, cystic pituitary adenoma, craniopharyngioma, empty sella, pituitary necrosis, or hypophysitis is challenging.[1,3]

Among these lesions RCCs deserve a special attention since they imitate colloid cysts both histopathologically and radiologically. Rathke cleft cysts sporadically show squamous metaplasia, a finding never observed in colloid cysts and stain positively for GFAP and skin type keratin,[5] whereas colloid cysts do not. Our patient did not show a positive staining pattern with GFAP, keratin 7, and keratin 20. Another discriminating feature is that capsule of the RCCs tends to be thicker than colloid cysts.[1,5] Finally, RCCs containing mucoid fluid are indistinguishable from colloid cysts whereas serous RCCs exhibit cerebrospinal fluid signal intensity on all MR sequences.[3]

Bender et al[3] reported a series of 38 patients with sellar cystic lesions to discriminate sellar colloid cysts from RCCs and found hyperintense T1 signal or mixed T2 signal with focal areas of hypointensity (dot sign) in majority of colloid cysts consistent with previous reports. All RCCs showed wall enhancement compared with colloid cysts with a sensitivity of 100% and specificity of 93%. They also demonstrated that RCCs had more prominent suprasellar extension.[3]

Radiologic images of the colloid cysts can be helpful in treatment planning. The hyperdense appearance on CT or a hypointense appearance on T2-weighted MRI is a result of viscous ingredient and would suggest that stereotactic aspiration would be ineffective.[3]

The management of these cysts includes cyst aspiration, microsurgical, and more recently endoscopic resection. Simple decompression of the cyst content without the removal of the cyst wall often results in recurrence in third ventricular colloid cysts.

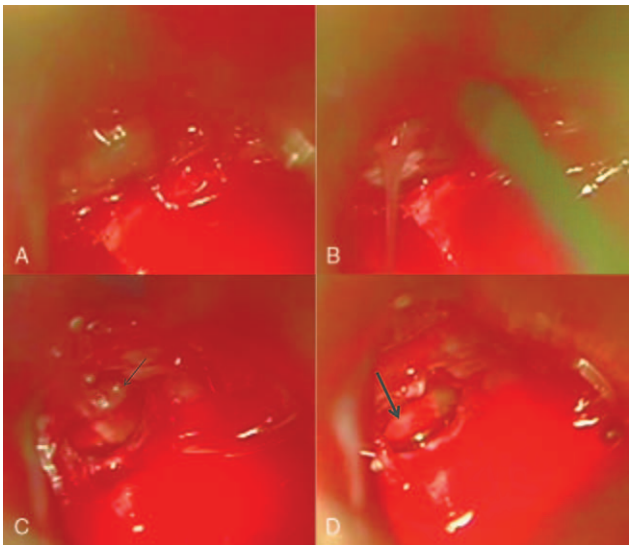

FIGURE 2. (A) Intraoperative images of the white colored, thin, smooth cyst wall. (B) The white, viscous cyst content is seen oozing. (C) The cyst wall was thicker in some parts (arrow). (D) The normal hypophysis is seen (thick arrow).

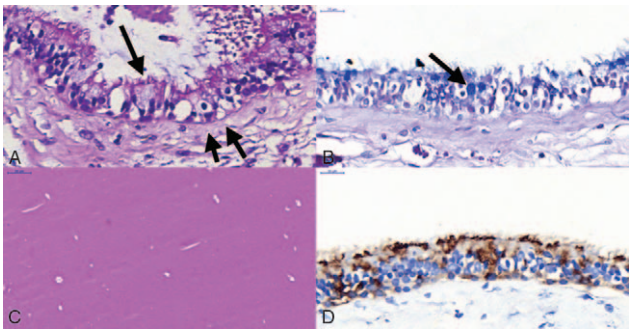

FIGURE 4. (A) Ciliated pseudostratified columnar epithelium with scattered goblet cells (arrow) resting upon thick basement membrane (double arrow) (hematoxylin and eosin, ×63.8). (B) Goblet cells (arrow) interspersed in epithelial lining (Alcian Blue-PAS, ×63.8). (C) Viscid and homogeneous cyst content consistent with colloid cyst (PAS, ×63.8). (D) Membranous epithelial membrane antigen (EMA) reactivity of epithelial lining of cyst (Biotinylated Streptavidin Complement, EMA, ×63.8).

Recently, there has been increased interest in endoscopic approaches to minimize postoperative complications.[1] We resected the lesion microsurgically through a transnasal transsphenoidal approach in our patient with no recurrence in follow-up.

We report a patient of pituitary colloid cyst successfully removed through the transnasal transsphenoidal approach. Although rarely found in this location colloid cysts should be considered in the differential diagnosis in patients who present with a pituitary cystic lesion.

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Neurocutaneous Melanosis Presenting as Cavernous Hemangioma Persistent Abdominal Pain

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Abstract: Neurocutaneous melanosis (NCM) is a rare congenital syndrome characterized by the presence of multiple congenital melanocytic nevi and the proliferation of melanocytes in the central nervous system. The authors present a 9-year-old Chinese boy whose clinical manifestations are intermittent headache for 2 months and persistent abdominal pain for 10 days. 3D-reconstruction computed tomography angiography image, digital subtraction angiography, and magnetic resonance imaging plus angiography (MRI+MRA) examinations results suggested that cavernoma at left frontal lobe potentially associated with hemorrhage. In addition, miliary abnormal signals were widely scattered on MRA image so that other malignant metastatic diseases cannot be ruled out. GI physical examination had not any abnormal findings, antispasmodic drugs were ineffective but antiepilepsy drugs were effective to abdominal pain. In surgery, no cavernoma was noticed but an accumulation of densely melanocytic mass located at the lesion on radiology images. The lesions spread along with perivascular of sylvian veins and leptomeningeal. Pathology investigation demonstrated brain metastatic malignant melanoma associated with hemosiderosis. The lesion of brain parenchyma was totally removed but the spread lesions could not be treated with surgery. Adjuvant radiotherapy was performed but failed to control the malignant development, still the patient died in 3 months postinitial operation. The authors conclude that abdominal pain was a manifestation of epilepsy related to the frontal lobe lesion. Neurocutaneous melanosis is a rare disease, brain metastases result in abdominal pain is rare even more, and it is worth the attention of clinicians.

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KL and SQ shared first authorship.

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Neurocutaneous melanosis is a rare congenital syndrome characterized by the presence of large or multiple congenital melanocytic nevi and benign or malignant pigment cell tumors of the leptomeninges. The syndrome is thought to represent an error in the morphogenesis of the embryonal neuroectoderm.[1,2] Most patients with neurocutaneous melanosis (NCM) will show neurological manifestations within 2 years after birth, which includes increased intracranial pressure, mass lesions, seizure, or spinal cord compression.[1] We present a 9-year-old Chinese boy, whose major clinical manifestations were headache and persistent abdominal pain; radiology examinations results suggest similar to cavernous hemangioma associated with hemorrhage. Diagnosis of NCM brain metastases is achieved by surgery and pathology.

CLINICAL REPORT

A 9-year-old Chinese boy suffered from intermittent headache for 2 months. Pain drugs were ineffective. CTA, 3D reconstruction, DSA, MRI+MRA examinations were performed. 3.0 T magnetic resonance imaging result suggested that the lesion located at left frontal lobe, and communicating hydrocephalus. The main body of the lesion is hypointense in T2-weighted image and moderately enhanced on the T1-weighted gadolinium-enhancement image. The upper part of the lesion is obviously hyperintense on T2-weighted image and hypointense on T1-weighted gadolinium-enhancement image, indicating the occurrence of cystolization (Fig. 1A and B). However, an unusually result of miliary abnormal signals were widely scattered on the horizontal, sagittal and coronal images of MRA (Fig. 1C–E). CTA and 3D reconstruction showed an enhancing mass was closely beside the left middle cerebral artery (Fig. 1F and G). DSA was performed but no obvious abnormality could be seen (data not show). Post DSA procedure, the patient showed signs of muscle weakness in his right limb, activity limitation, muscle strength III level, and aphasia. Before surgery, the first diagnosis was cavernous hemangioma associated with spontaneous hemorrhage, but other malignant metastatic diseases cannot be ruled out. While hospitalized, the patient suffered persistent abdominal pain for 10 days but showed no symptoms in the digestive tract or abdominal tenderness. GI physical examination