Turkish Neurosurgery 2007, Vol: 17, No: 2, 138-141

Pituitary Adenoma Coexisting With a Suprasellar Arachnoid Cyst

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ABSTRACT:

OBJECTIVE: Biochemical and radiological properties of sellar and parasellar lesions are quite similar in some instances. This leads to a difficulty in preoperative diagnosis. Here, a pituitary adenoma and a suprasellar arachnoid cyst in the same patient is presented, and possible etiopathogenetic mechanisms and surgical treatment are discussed.

CASE: A 56-year-old male patient was admitted to the hospital with a history of seizures, urinary incontinence and visual disturbances. Preoperative MRI revealed a mass lesion in the sella turcica with suprasellar extension and a coexisting large supra- and parasellar cyst.

DISCUSSION and CONCLUSION: A slow-growing mass lesion beneath the defective mesencephalic leaf of the Liliequist membrane may lead to a one-way valve system on its surface. It might be speculated that CSF will become trapped in the cyst during tumor growth. The other possible mechanism to explain the coexistence is discussed. In light of these comments and intraoperative observations, we suggest a third type of suprasellar arachnoid cyst. a semi-communicative type.

KEY WORDS: Membrane of Liliequist, Parasellar mass, Pituitary adenoma, Sellar mass, Suprasellar arachnoid cyst.

Received : 07.03.2007
Accepted : 15.05.2007

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INTRODUCTION

The sella turcica and juxtasellar regions host various solid and cystic lesions (8,9). The most common mass lesions located in these sites are pituitary adenomas. Arachnoid cysts, Rathke cleft cysts and craniopharyngiomas are the most common sellar and suprasellar cystic lesions (4,9). These different pathologies can exhibit similar radiological and biochemical properties (1,3). Preoperative diagnosis of the wide variety of neoplastic and non-neoplastic space-occupying lesions is very difficult despite modern imaging techniques (3,9). These lesions also mimic each other biochemically (1). Preoperative differentiation of these lesions is extremely important in planning treatment regiments (1). The coexistence of two distinct pathologies of a different nature in the same location makes preoperative diagnosis complicated. The preoperative evaluation of this dilemma would provide appropriate intervention in such cases. We therefore present a case harboring two different lesions in the sellar region.

CASE REPORT

A 56x-year-old male patient was admitted to the hospital with a one-week history of seizures, urinary incontinence and visual disturbances. He had noticed visual loss in his left eye after the first seizure attack and this symptom was persistent. The seizure activity increased and he experienced urinary incontinence unrelated to the seizures.

Neurological examination on admission revealed papilledema in his left fundus, mild concentric narrowing of the left visual field and loss of visual acuity in his left eye. Preoperative MRI revealed a mass lesion in the sella turcica with suprasellar extension and the coexistence of a large supra- and parasellar cyst. The lesion that entirely occupied the sella was seen as homogenously enhanced hyperintense mass, extended to both cavernous sinuses and compressed the optic chiasm on T1-W postcontrast sequences (Figure 1). The cystic lesion was observed in the supra- and left parasellar regions in similar intensity and homogenity. The left lateral ventricle configuration was changed by the mass effect. Routine laboratory investigations showed no abnormality. All basal serum pituitary-hypothalamic hormonal levels were within normal limits.

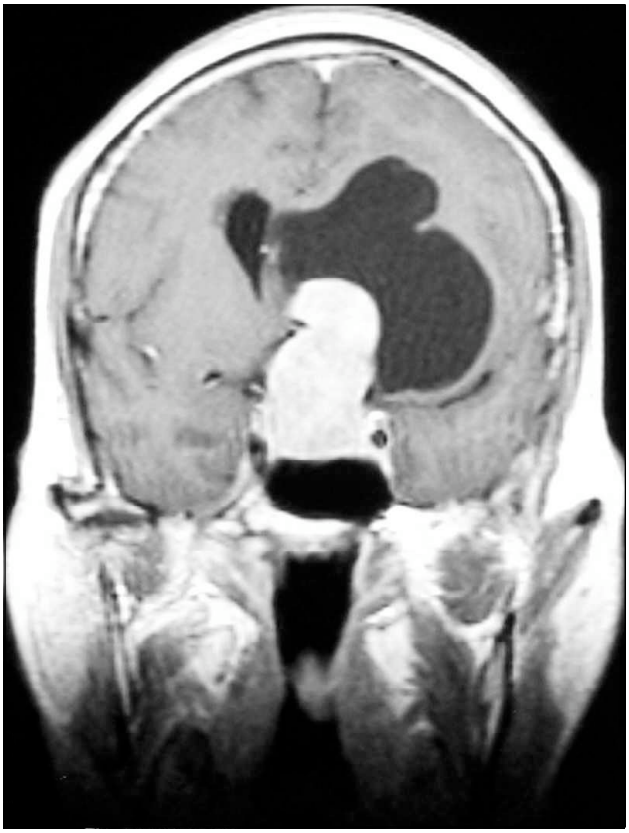

Figure 1: A solid sellar mass lesion with suprasellar extensions and a large supra-parasellar cystic cavity are seen on the T1-W postcontrast coronal MRI. Hyperintensely-enhanced pituitary adenoma and an arachnoid cyst compress left lateral ventricle. It is noticed that the cyst is slightly hypointense compared to the lateral ventricles.

The patient underwent surgery. A left-sided pterional craniotomy was performed. The thin cyst wall gave an impression of a separate lesion. The clear cyst content was aspirated and partial cyst wall was removed for histopathological examination. After evacuation of the cyst, the solid lesion was exposed. The mass compressed the left optic nerve and chiasm; however, there was clear cleavage from the surrounding neural tissues. The mass lesion was grayish purple in color, soft and mildly vascular. The lesion was removed totally.

The pathological diagnosis was pituitary adenoma (Figure 2). Histopathological examination of the cyst wall revealed normal arachnoid membrane.

The patient had an uneventful postoperative period and was discharged without any additional treatment. Postoperative findings 4 months later were normal and the follow-up MRI showed no residual or recurrent lesion (Figure 3).

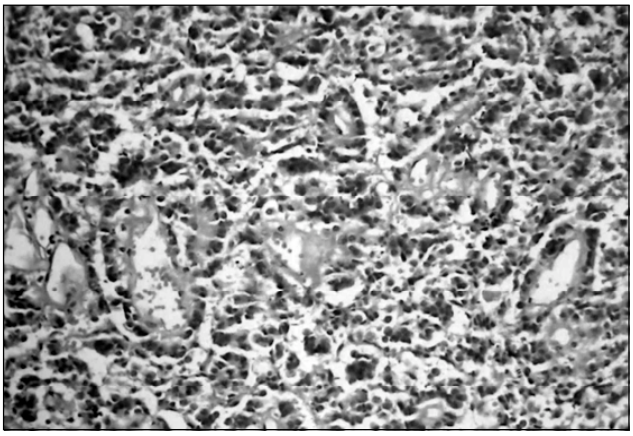

Figure 2: The microphotograph of the pathological specimen of the mass lesion shows tumor cells with large cytoplasm and round, uniform nuclei making adenoid structures. The solid growth pattern is seen (HE X 200).

DISCUSSION

Arachnoid cysts account for approximately 1% of all intracranial mass lesions. The suprasellar region hosts approximately 9-11% of those lesions (5,6,7). The majority of suprasellar arachnoid cysts are congenital lesions, secondary to imperforation of the Liliequist membrane. A defective sellar diaphragm and subarachnoid space extending through this gap were found at a rate of 5.5% in a necropsy study (2). This imperforation leads to blockage of the CSF flow at the level of the suprasellar cistern (6) and a

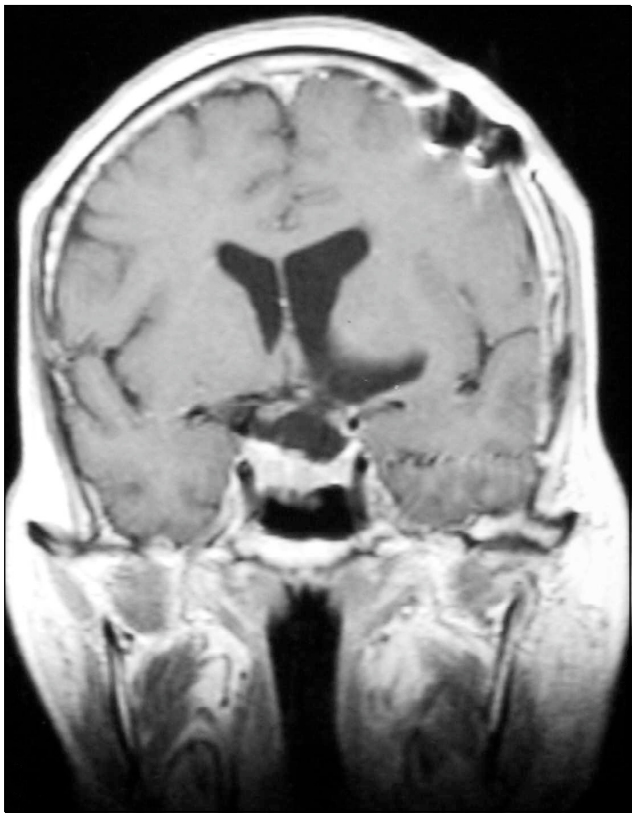

Figure 3: Postoperative coronal T1-W MRI shows no residual or recurrent lesion. The left lateral ventricle has regained its normal size and place.

noncommunicating arachnoid cyst develops. The cystic dilation of the interpeduncular cystern is another type of arachnoid cyst that allows CSF flow partly, so-called communicated type. Liliequist membrane splits into two layers as two separate arachnoid sheets. The upper sheet attaches to the diencephalon and separates the chiasmatic and interpeduncular cysterns and is called a diencephalic membrane. The lower sheet, known as the mesencephalic membrane, attaches to the junction of the midbrain and medulla, separating the prepontine and interpeduncular cysterns. The mesencephalic membrane frequently has an opening (6). A slow-growing mass lesion under the mesencephalic membrane, such as a pituitary adenoma, may lead to a one-way valve system on the surface of the defective mesencephalic membrane (5). Consequently, a true arachnoid cyst may develop with a dome from the diencephalic membrane and a base from the mesencephalic membrane. However, the cyst in our case does not appear to be the complete communicating type as CSF flows one way, and CSF will become trapped in the cyst as the tumor grows. On the other hand, the cyst is not also the complete noncommunicative type, because the one-way CSF flow continues until the late stages of the tumor growth. Since the Basilar Artery (BA) bifurcation was not inside the cyst, our case cannot be considered as the communicating type. The signal intensity of the cyst on all MRI sequences is slightly hypointense compared to the CSF in surrounding arachnoid cysterns, indicating entrapment of the CSF inside the cyst as the intensity of the cyst should be the same to that of the peripheral subarachnoid space on all the MRI sequences. A high level of protein or hormones in the content should not affect the intensity (5). It might be speculated that the possibility of unverified slow flow may lead to this difference in intensity.

The other possible mechanism to explain the coexistence is the growing mass lesion leading to the development of a diverticulum on the thin mesencephalic membrane. This diverticulum may cause CSF trapping and result in a noncommunicative arachnoid cyst. This noncommunicative cyst may have a two-layer capsule with a diencephalic membrane and a diverticular mesencephalic membrane. These layers might be detected intraoperatively. In light of these comments and intraoperative observations, our proposal for the development in a sellar solid mass lesion is the third type of suprasellar arachnoid cyst, which is the semi-communicative type. Thus, the occurrence of this third type of suprasellar arachnoid cyst is dependent of the existence of both a slow growing sellar mass lesion and a defective mesencephalic membrane.

CONCLUSION

The origin of suprasellar arachnoid cysts with a sellar mass lesion remains unclear. Theories of the development of suprasellar cysts in the literature seem incapable of explaining the occurrence of arachnoid cyst in the existence of the sellar mass lesions. Neurosurgical observations can enable us to clarify this rare coexistence.

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