Neurologia medico-chirurgica (1997.03) 37巻3号:284～287.

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Neurol Med Chir (Tokyo) 37, 284~287, 1997

Glial Cyst in the Thalamus with Intracystic Hemorrhage

—Case Report—

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Abstract

A 19-year-old female presented with an unusual glial cyst of the thalamus that caused development of acute hydrocephalus due to hemorrhage and manifested as headache and fainting attacks. Computed tomography showed a large cystic mass lesion in the left thalamus with intracystic hemorrhage. The cyst was subtotally removed. Microscopic examination revealed mild gliosis with marked hemosiderin deposits. The inner surface of the cystic wall lacked an epithelial lining. The diagnosis was glial cyst. Magnetic resonance (MR) imaging 2 months after surgery showed a residual cyst in the left thalamus. However, after 12 months she was asymptomatic, neurologically intact, and MR imaging showed no regrowth of the cyst. Treatment of glial cyst must provide sufficient communication between the cyst and the cerebral ventricles rather than attempt total removal of the cyst, which may present a considerable challenge.

Key words: glial cyst, benign intracranial cyst, intracystic hemorrhage

Introduction

Magnetic resonance (MR) imaging is identifying an increasing number of cases of intracranial benign cysts. Asymptomatic pineal cysts, in particular, occur at a frequency of 1–4%.[4,14] Glial cysts that develop in the cerebral hemisphere and become symptomatic are still rare.[13,19] We recently encountered a case of glial cyst originating in the left thalamus that resulted in an intracystic hemorrhage and was discovered because of headache and disturbance of consciousness.

Case Report

A 19-year-old female experienced headache and general malaise on the morning of September 16, 1994. The symptoms persisted on the next day. She suffered a fainting spell in the bathroom that lasted for 1 minute on September 18. Later, her headache increased and was complicated by nausea. She then visited a physician. Computed tomography (CT) revealed a cystic lesion of the third ventricle and marked enlargement of the bilateral lateral ventricles. She was transferred to our department on September 19. Her medical history was unremarkable.

On admission, she was fully conscious. There was moderate papilledema in both fundi. No abnormalities were detected in the cranial nerves, motor functions of the extremities, or sensory perceptions. She exhibited a slightly ataxic ambulatory pattern.

Skull radiography revealed marked digital markings and abnormal calcification in the corresponding area to the CT finding as follows. CT indicated a cystic lesion measuring 55 mm across the greatest diameter that extended from the left thalamic region to the lateral ventricle. The cyst wall appeared as slightly high density. Administration of contrast medium caused no obvious enhancement. Part of the cyst wall was calcified. The intracystic area was characterized by a slightly higher density than that of the cerebrospinal fluid and there was a niveau that was interpreted to be an intracystic hemorrhage. Both lateral ventricles were markedly enlarged (Fig. 1). MR imaging showed the cyst as a slightly high-intensity signal on the T_1-weighted im-

Received July 17, 1996; Accepted November 20, 1996

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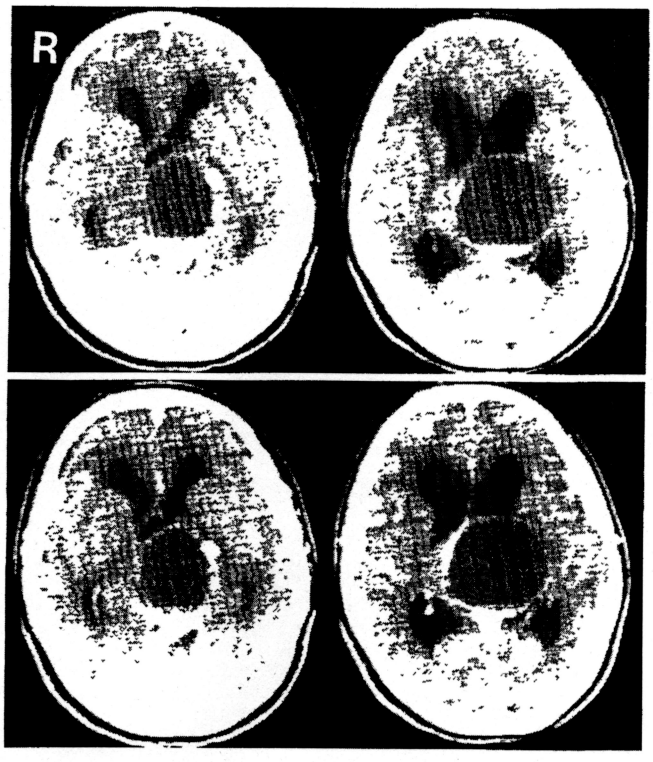

Fig. 1 Preoperative computed tomography scans, showing a cystic lesion with a high density water level and moderate hydrocephalus, with calcification in part of cyst wall (*upper row*), and absence of enhancement after injection of contrast medium (*lower row*).

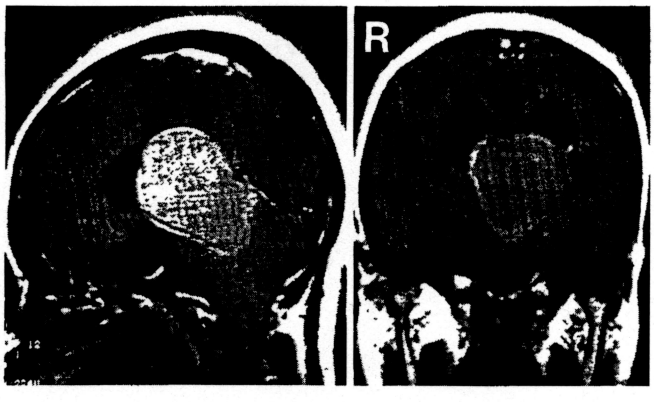

Fig. 2 Preoperative T_1-weighted magnetic resonance images with gadolinium-diethylenetriaminepenta-acetic acid, sagittal (*left*) and coronal views (*right*), showing a slightly high-intensity mass with moderately enhanced capsule.

age and a high-intensity signal on the T_2-weighted image. The cyst wall appeared as an iso-intensity signal on the T_1-weighted image and a low-intensity signal on the T_2-weighted image, with slight enhancement by gadolinium-diethylenetriaminepenta-acetic acid. The coronal MR image showed an ambiguous border of the cyst and the left thalamus, indicating extrusion of the third ventricle to the right and the left ventricle upward. The sagittal MR image clearly indicated that the lower border of the cyst had reached the mesencephalic level (Fig. 2). Left internal carotid angiography showed an avascular mass with no tumor staining. The arterial phase indicated lateral deviation of the lenticulostriate artery and the left medial cerebral artery and unrolling of the pericallosal artery in the upper direction. The venous phase showed displacement of the internal cerebral vein and the septal vein toward the right. The arterial phase of the left vertebral angiography showed that the left lateral posterior choroidal artery was stretched in the upper direction along the cystic wall, whereas the right medial posterior choroidal artery had shifted laterally along the right lateral wall of the cyst.

Under a preoperative diagnosis of a low grade astrocytoma originating at the left thalamus, she underwent cyst removal through a left transcortical and transventricular approach. The cyst was covered by ependyma and projected into the lateral ventricle. Excision of the ependyma exposed the relatively soft cyst wall, which was stained yellow with hemosiderin. The cystic fluid was clear and yellow with low viscosity. Old hematoma was seen in part. The presence of the left posterior choroidal artery and thalamostriate vein were confirmed along the upper surface of the cystic wall. The medial side of the border was clearly identified, enabling ready dissection. However, the lateral side bordering the thalamus was extremely ambiguous, and the part in the quadrigeminal cistern adhered firmly to the vein and dissection was difficult. An intraoperative frozen section examination of the cyst wall indicated the lesion as a glial cyst or low grade astrocytoma. Total removal, which appeared extremely difficult, was avoided and the cyst was only subtotally removed.

Microscopic examination of the tumor revealed mild gliosis with marked hemosiderin deposits. The inner surface of the cystic wall lacked an epithelial lining. No atypical cell or abnormal cellular proliferation was found (Fig. 3). Based on these findings, the diagnosis was glial cyst originating in the left thalamus with subsequent hemorrhage.

Following surgery, she developed mild hemiparesis on the right, mainly in the upper extremity, but her condition gradually improved. On October 24, she was free of neurological symptoms and was discharged. MR imaging 2 months after surgery

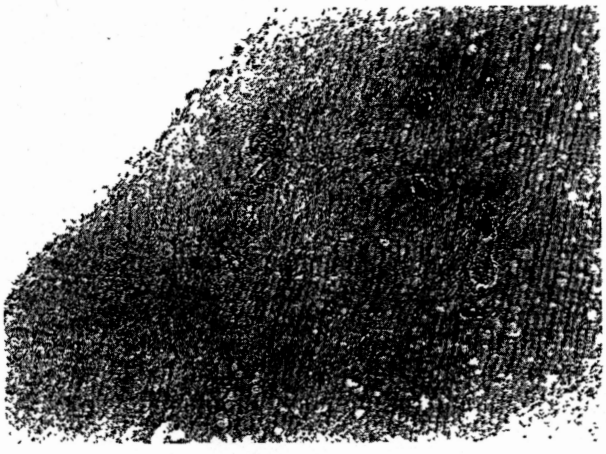

Fig. 3 Photomicrograph of the cyst specimen showing glial tissue lining the lumen of the cyst, hemosiderin deposits, and vasculature. HE stain, original magnification ×100.

Fig. 4 Postoperative T_1-weighted magnetic resonance image with gadolinium-diethylene-triaminepenta-acetic acid, sagittal (*left*) and coronal views (*right*), showing a residual cyst in the left thalamus extending to the midbrain and the third ventricle.

showed a residual cyst in the left thalamus and the third ventricle but improvement in hydrocephalus (Fig. 4). She was asymptomatic and neurologically intact, and MR imaging showed no regrowth of the cyst at the 12-month follow-up examination.

Discussion

Benign intracranial cystic lesions without neoplastic changes are divided into two types: with and without epithelial lining.[1,2,5–7,10,11,15,16] The former include ependymal cysts, choroidal cysts, colloid cysts, and neuroectodermal cysts,[3,5,8] and the latter include arachnoid cysts,[9] cysts formed secondary to inflammation (such as abscess and cysticercosis), those due to degeneration (such as hemorrhage and infarction), and glial cysts[12,13,17,19] as seen in the present case.

The etiological mechanism of cysts with an epithelial lining may involve heterotopic growth of the epithelial cells during formation of the cerebral ventricles in the embryonic stage. Cysts of the pineal body may originate from a vestige of the cavum pineale during pineal body formation in the embryonic stage. However, there is no established theory to explain the development of glial or gliotic cysts in the cerebral hemisphere. Such cysts may originally occur in the ventricular system but their continuity is disrupted by stretching of the ependymal cells due to a pressure effect exerted by the intracystic fluid.[19] In our case, hemosiderin deposition was evident inside the cystic wall and the possibility of degeneration following a cerebral hemorrhage that culminated in the formation of the cyst cannot be denied. Her medical history included no abnormalities during early childhood and she had experienced no episode suggestive of cerebral hemorrhage. Therefore, it is highly likely that repeated hemorrhages within the original cyst resulted in a gradual increase in cystic dimensions and the development of associated symptoms.

The initial symptoms in most clinical cases were associated with the development of hydrocephalus, which was caused by cystic enlargement.[8,12,13,17–19] The prognosis is satisfactory if the hydrocephalic condition is improved. Therefore, the treatment must provide sufficient communication between the cyst and the cerebral ventricles rather than attempt total removal of the cyst, which may present a considerable challenge.

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