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症　　例

Communicating Hydrocephalus Occurred in the Postoperative Course of Glioblastoma Multiforme

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Summary

We experienced a case of glioblastoma multiforme which exhibited dementia, gait disturbance, headache, and uninary incontinence six months after subtotal removal of the tumor. These symptoms were not due to tumor recurrence, but to communicating hydrocephalus. Communicating hydrocephalus in cases of malignant brain tumors has not often been reported. We discuss the development of this abnormality.

Introduction

In 1954 GARDNER et al reported a case of communicating hydrocephalus in a patient with a small acoustic neurinoma[5]. Since then, a considerable number of authors have reported that communicating hydrocephalus can arise as a complication of tumors in the central nervous system (CNS)[1,6,7,11]. Most of the reports were, however, those associated with benign brain or spinal tumors. Communicating hydrocephalus in malignant brain tumors, especially in cases without meningeal dissemination of tumor cells, has rarely been reported[8,9,12]. In the present paper, we present a case of communicating hydrocephalus which developed postoperatively following subtotal removal of a glioblastoma multiforme.

Case Report

A 57-year-old woman underwent subtotal removal of glioblastoma multiforme in the right temporal lobe on June 23, 1986 (Fig. 1 A, B). The only postoperative symptom was left homonymous hemianopsia. Postoperative irradiation and chemotherapy reduced the size of the residual tumor (Fig. 2 A, B, C).

At the beginning of January, 1987, mental symptoms such as disorientation, acalculia, or apathetic countenance appeared. Uninary incontinence, headache, and gait disturbance

Key words: Communicating hydrocephalus, Cerebrospinal fluid, Malignant brain tumor.
索引語：交通性水頭症，脳脊髄液，悪性脳腫瘍.
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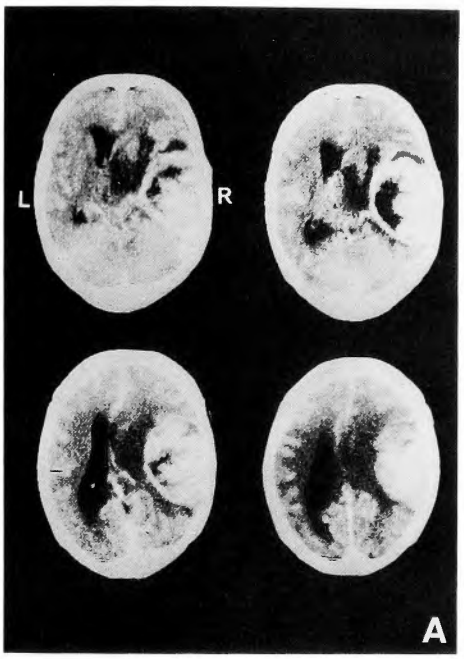

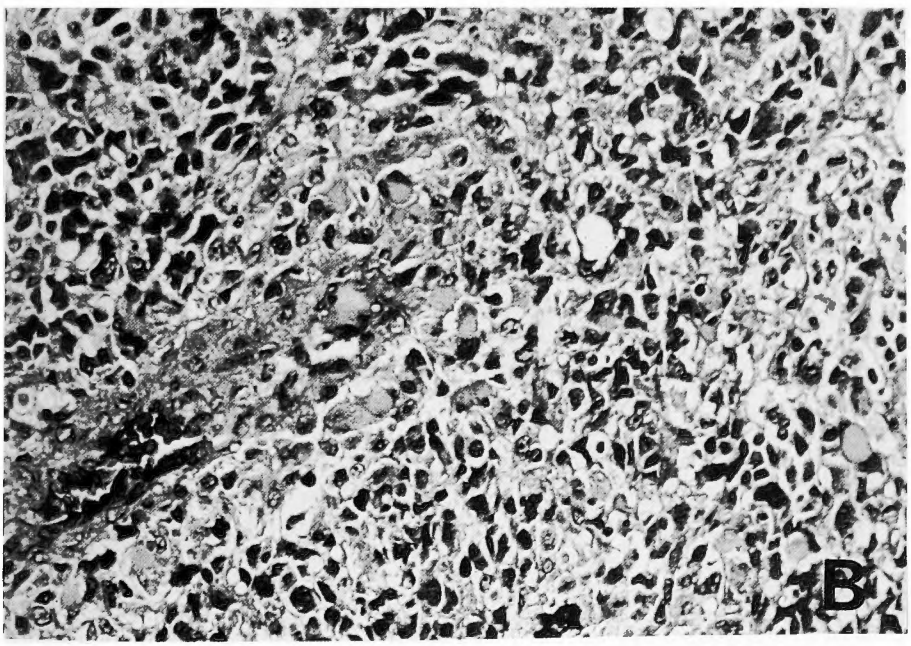

Fig. 1.

A, Precperative CT. A large enhanced mass was situated in the right temporal lobe.
B, Microscopical appearance of the removed tumor. Typical findings of glioblastoma multiforme were observed (hematoxylin and eosin ×200).

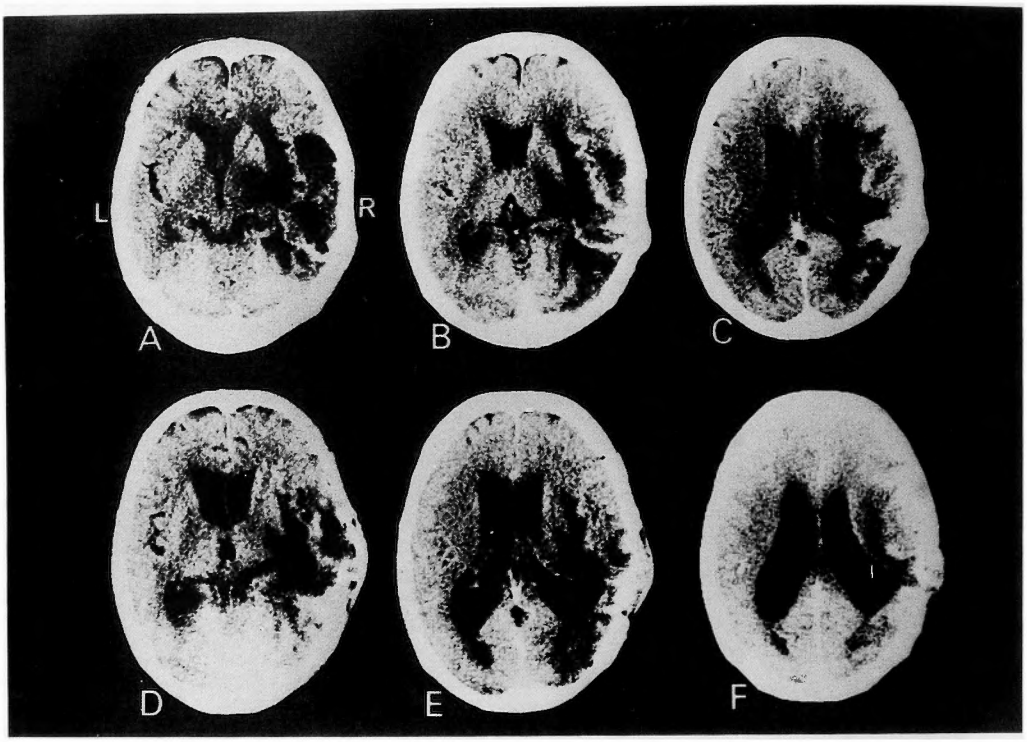

Fig. 2. Enhanced CT.

A, B, C, four months after svbtotal removal of the tumor. The size of the tumor became remarkably smaller than that of the preoperative one. D, E, F, six months after subtotal removal of the tumor. The local tumor did not increase in size, but enlargement of all ventricles was shown.

subsequently occurred. Choked disc was then found. Enhanced computed tomography (CT) revealed enlargement of all ventricles (F g. 2 C, D, F). The reisdual tumor did not increase in size. The symptoms described above were considered not to be due to recurrence of the tumor but due to hydrocephalus. Ventriculo-peritoneal (VP) shunt was performed on January 26, 1987. Several days after the shunt operation, the patient became asymptomatic except for left homonymous hemianopsia. Metrizamide CT which was performed postoperatively revealed that all ventricles, the basal cisterns including the cisterns around the brain stem and the left sylvian fissure were communicating with each other. However, the right sylvian fissure and the subarachnoid space over the bilateral cerebral convexities were not filled with metrizamide. The size of the ventricles decreased (Fig. 3). On the basis of these findings, we concluded that communicating hydrocephalus had existed before the shunt operation was performed, due to the blockade of the pathway of the cerebrospinal fluid (CSF) in the subarachnoid space over the cerebral convexities.

On February 22, 1987 sombhlency appeared, and enhanced CT showed regrowth of the tumor. It was observed on CT that the ventricular system did not become enlarged. Dementia, urinary disturbance, geographical and dressing apraxia, left hemiparesis and consciousness

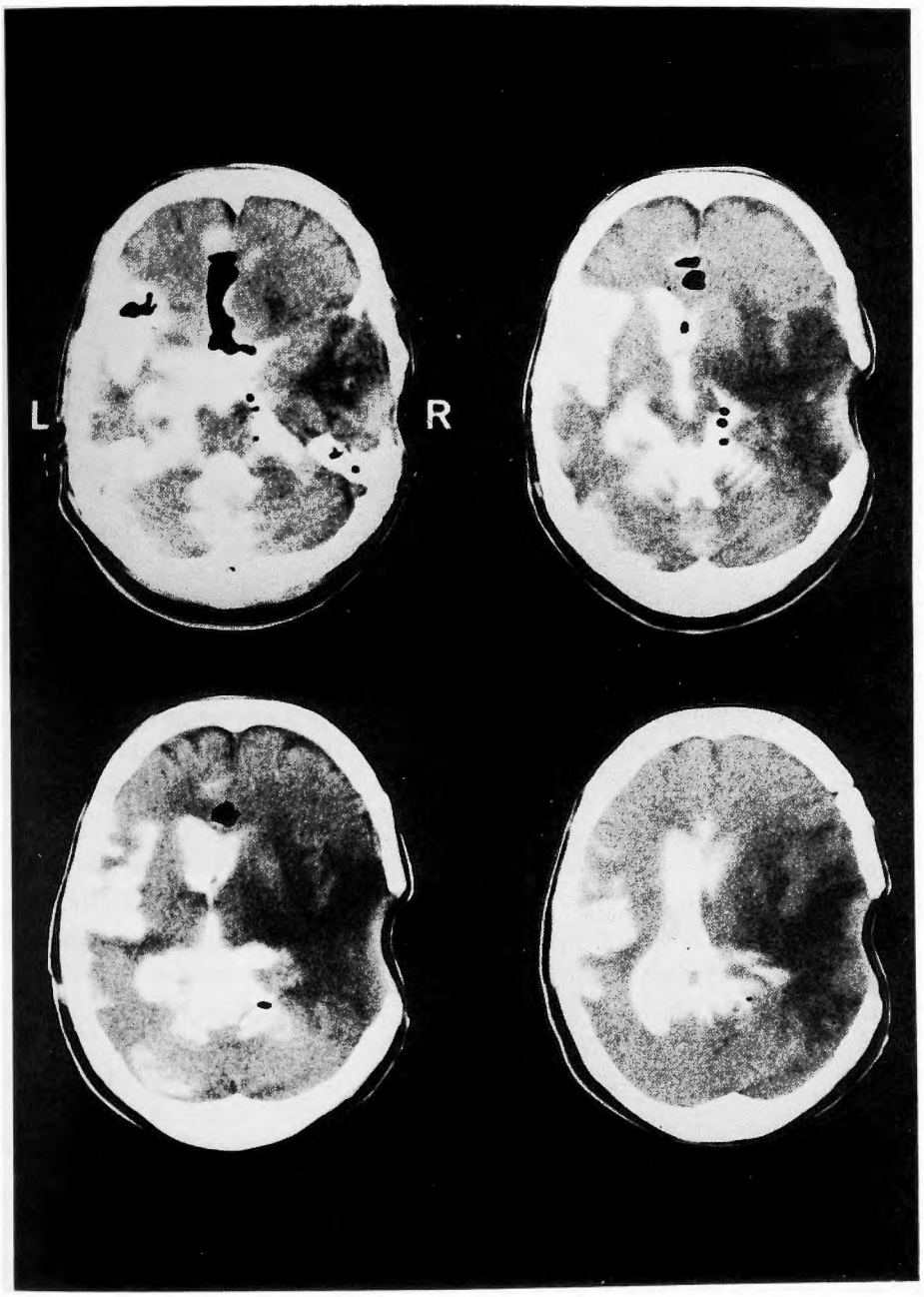

Fig. 3. Metrizamide CT after VP shunt. All ventricles, the basal cisterns, and the left sylvian fissure were communicating with each other. The right sylvian fissure and the subarachnoid space over bilateral convexities were not filled with metrizamide. The size of ventricles decreased.

Table 1. The Findings of the Cerebrospinal Fluid.

date	Jan. 21 '87	Jan. 24 '87	Jan. 26 '87 (shunt op.)	Feb. 16 '87	Feb. 24 '87
site of CSF removal	lumbar S.S.	lumbar S.S.	right Lat. V.	shunt valve	shunt valve
initial P. (mm H^2O)	110	190			
cell count (μl)	37/3	21/3	2/3	1/3	
L : N	1 : 0		1 : 0	1 : 0	
protein (mg/dl)	213	134	124	186	104
					Alb 76.4%
					α_1-G 4.4
					α_2-G 5.2
					β-G 7.3
					γ-G 6.7
					fibrinogen 0.5 (mg/dl)

Abbreveations : op, operation; CSF, cerebrospinal fluid; S.S., subarachnoid space; Lat. V., lateral ventricle; P., pressure; L : N, lymphocytes : neutrophils.

disturbance progressively appeared, in relation to the rapid increase in the tumor size on CT. The patient died on April 15, 1987 due to respiratory arrest.

The protein concentration in the CSF, the mean value of five measurements being 152.4 mg/dl, was maintained at a high level before and after the VP shunt. The ratio of each protein fraction in the CSF, measured postoperatively once, was within a normal range. Fibrinogen (0.5 mg/dl) was detected once (Table 1).

Autopsy revealed bilateral uncal and tonsillar herniation, regrowth of the tumor, and generalized brain edema. Adhesion between the arachnoid membrane and the brain surface was not remarkable. No tumor dissemination or no other organic changes were microscopically demonstrated in the subarachnoid space (Fig. 4).

Notable pathological findings in the pacchionian granules were not obtained.

Discussion

Dementia, gait disturbance, headache and uninary incontinence appeared six months after subtotal removal of glioblastoma multiforme in the right temporal lobe. These symptoms were not due to tumor recurrence but were caused by communicating hydrocephalus. VP shunt improved these symptoms remarkably, although for only one month.

Communicating hydrocephalus associated with malignant brain tumors is thought to have two different predisposing factors. One factor is a high concentration of proteins in the CSF[1,5,6,7,11]. The other is mechanical obstruction of the CSF pathway with tumor cells disseminated in the subarachnoid space[2,3].

The protein concentration in the CSF is usually high in cases of benign or malignant tumors

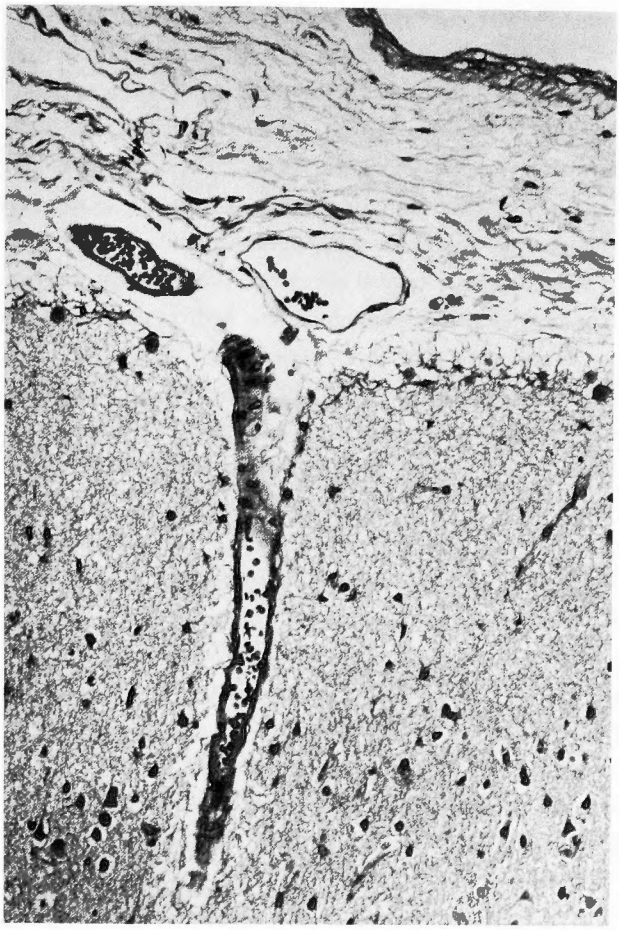

Fig. 4. Arachnoid membrane and the subarachnoid space, which had not been filled with a contrast medium on metrizamide CT, in the right parietal lobe. No dissemination of tumor cells or no other notable organic changes were found (hematoxylin and eosin ×200).

of the CNS[4,5,6,10,11]. Leakage of serum proteins from the tumoral or peritumoral vessels[4,5], production of immunological proteins by the peritumoral lymphoid cells[6,10,11], or subarachnoid hemorrhage from the tumor[11] are thought to be responsible for the high protein concentration in the CSF. A high protein concentration is known to induce both functional and organic disturbances of CSF dynamics. Functionally, some authors have speculated that the CSF circulation is slowed or halted due to increased viscosity of the CSF[1,6,11]. Organically, adhesive arachnoiditis is known as a complication of a high protein concentration[1,6,11]. Furthermore, the passing or absorptive routes of the CSF might be obstructed by plugging of protein molecules or by cellular and fibrous exudate[5,6].

The protein content was maintained to be at a high level in our case (Table 1). Judging from the normal ratio of protein fractions in the CSF, proteins in the CSF of our case are thought to have derived from serum proteins which had leaked from damaged vessels. The existence of

fibrinogen, which cannot be detected from the normal CSF, in the CSF might confirm this supposition (Table 1)[4]. Meningeal dissemination of tumor cells or other organic changes in the subarachnoid space were not observed on CT and autopsy. The communicating hydrocephalus in our case is, therefore, not due to mechanical obstruction of the CSF pathway by tumor cells, but due to the high protein concentration in the CSF. From the pathological point of view, it is concluded that a functional rather than an organic disturbance of the CSF circulation played the major role in developing the communicating hydrocephalus in our case.

Aside from cases caused by mechanical obstruction of the CSF pathway with disseminated tumor cells in the subarachnoid space, communicating hydrocephalus associated with malignant brain tumors has been reported in only three papers since 1945[8,9,12]. There are two reasons for the rare occurrence and reporting of communicating hydrocephalus in cases of malignant brain tumors. The first is that a functional or an organic disturbance of CSF circulation is known to occur only after a considerably long period for which the protein concentration of the CSF has been maintained at a high level[5,7,11]. Malignant brain tumors usually grow too rapidly to have communicating hydrocephalus as a complication. The second reason is that the existence of communicating hydrocephalus will often be overlooked because of severe neurological symptoms due to the tumors themselves in cases of malignant brain tumors. In our case, it was not difficult to find symptoms due to communicating hydrocephalus because the glioblastoma multiforme situated in the right temporal lobe had exhibited only left homonymous hemianopsia. The relatively slow regrowth of the residual tumor in our case might be a factor predisposing to the development of communicating hydrocephalus.

The possible influence of surgery, irradiation or chemotherapy in the occurrence of communicating hydrocephalus has not been drawn the attention of previous authors, but demands consideration in the future research.

The indication of shunting procedures for communicating hydrocephalus in malignant brain tumors must be carefully determined on the basis of the size of the primary tumor, the presence or absence of meningeal dissemination, and the general and neurological symptoms of the patient.

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和文抄録

多形膠芽腫の術後経過中に発生した交通性水頭症の1例

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右側頭葉の多形膠芽腫を亜全摘した．術後の放射線および化学療法により残存腫瘍は縮少し，左同名性半盲のみを残していた．術後7か月目に，痴呆，歩行障害，頭痛さらには尿失禁が出現した．この時点で腫瘍の再発所見はなく，これらは交通性水頭症による症状であった．当症例では，髄液腔への腫瘍播種は生前のCTその他の諸検査でも，また剖検においても認められなかった．

悪性脳腫瘍において，腫瘍の髄液腔への播種による交通性水頭症や，腫瘍それ自体の増大による機械的な髄液腔の閉塞を基盤とする交通性水頭症は実地臨床上しばしば経験する．しかし当症例のように，腫瘍の増大がなく，しかも髄液腔への腫瘍播種も伴わない交通性水頭症の報告は稀である．

われわれの症例における交通性水頭症は髄液中の高蛋白をその原因とするものと思われた．