Intraparenchymal Metastatic Tumor with Periventricular Dissemination

—Case Report—

Liu RONG-YAO, Kengo MATSUMOTO, Yusuke YOSHIMOTO, and Takashi OHMOTO

Department of Neurological Surgery, Okayama University Medical School, Okayama

Abstract

A 50-year-old male presented with a rare intraparenchymal metastatic tumor spreading through the periventricular tissue. Magnetic resonance (MR) imaging demonstrated the tumor as a heterogeneous low-intensity area on T\textsubscript{1}-weighted images with enhancement by gadolinium-diethylenetriaminepenta-acetic acid, and as a heterogeneous high- or isointensity area on T\textsubscript{2}-weighted images. Histological examination of a biopsy sample showed adenocarcinoma. This MR imaging appearance is typical of malignant glioma. The differential diagnosis of tumor in the cerebral parenchyma with ventricular dissemination should include both primary and secondary intracranial malignant tumors. MR imaging is useful in the diagnosis of such tumors, but the final diagnosis should be based on either tissue biopsy or cytological examination of the cerebrospinal fluid.

Key words: metastasis, paraventricular tumor, periventricular spread, magnetic resonance imaging, adenocarcinoma

Introduction

Tumor cell dissemination to the cerebral ventricles or subarachnoid space occasionally occurs in cases of primary intracranial tumors, such as glioblastoma,\textsuperscript{6} malignant lymphoma and germinoma,\textsuperscript{1} and metastatic disease.\textsuperscript{11} Metastatic tumor with dissemination into the cerebral ventricle and subarachnoid space is rare, with only four cases diagnosed by computed tomography (CT) or at autopsy.\textsuperscript{4,10} We present a case of paraventricular metastatic tumor from lung cancer which disseminated through the periventricular wall of the lateral ventricles.

Case Report

A 50-year-old male was admitted to a local hospital because of severe headache following a traffic accident on May 27, 1995. Neurological examination revealed no abnormal signs. CT revealed a round mass 2.3 × 2.0 cm in the left paraventricular region. He was transferred to our hospital for further examination and treatment on July 26, 1995. He was indifferent and apathetic with fluctuating drowsiness. Laboratory studies revealed a carcinoembryonic antigen concentration of 9.43 ng/ml (normal value < 5.0 ng/ml) and an alpha-fetoprotein concentration of 1.1 IU/ml (normal value < 5.0 IU/ml).

Magnetic resonance (MR) imaging showed a round mass 3.2 × 3.0 cm enhanced with gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA) with infiltration of the tumor into the subependyma of the lateral ventricle and septum pel-lucidum. The mass appeared as a heterogeneous low intensity area on T\textsubscript{1}-weighted images, and an irregular heterogeneous high- or isointensity area on T\textsubscript{2}-weighted images. Peritumoral edema was moderate (Fig. 1). Dissemination of a primary intracranial malignant tumor was strongly suspected because the ventricular wall was also enhanced. Involvement of the choroid plexus and leptomeninges seemed to be absent.

The mass was deeply located and he had a past history of myocardial infarction, so surgical resection was considered too risky. CT-guided stereotactic biopsy was performed to guide further treatment on August 3. Histological examination revealed that...
the tumor was a metastatic adenocarcinoma (Fig. 2). Carcinoma cells were also found in the cerebrospinal fluid (CSF). Subsequent chest CT demonstrated the carcinoma, 2 cm in diameter, in the left lung with bilateral pleural effusion on August 14. Diabetes insipidus appeared on the 3rd day after biopsy. Repeat MR imaging on August 17 demonstrated an enhanced infiltrative mass in the wall of the third ventricle, pituitary stalk, and pituitary gland (Fig. 3) that was not detected on admission. His diabetes insipidus seemed to be caused by invasion of the hypothalamus and pituitary stalk. Diabetes insipidus was hard to control, and his fluid balance was difficult to regulate. He was complicated with severe pneumonia and died on September 24, 2 months after the biopsy. Autopsy permission was refused.

Discussion

Periventricular dissemination of intracranial metastatic disease has been described in only four previous cases in the past 20 years (Table 1). However, the true incidence may be higher as patients have usually died before a diagnosis has been made. Three other cases were diagnosed by autopsy among 50 patients with leptomeningeal invasion due to systemic cancer, but the clinical data were not reported. Three previous cases of intracranial metastatic dissemination to the ventricles were confirmed at autopsy,2 and a single case was diagnosed by CT.3 Contrast-enhanced CT was reported to be the best method to detect the periventricular spread of tumor. Periventricular tumor infiltration on unenhanced CT scans has also been described.4 In our case, MR imaging demonstrated an intraparenchymal solitary metastatic tumor spread through the periventricular tissue. Based on our experience, this entity is more clearly depicted by MR imaging than by CT. CT can also lead to a false negative diagnosis.7

Diagnosis is rarely made before death. The lack of localizing signs and symptoms make the clinical diagnosis difficult and the patients were in a critical
condition when the ventricular metastases occurred and died before the diagnosis could be made.\textsuperscript{3,5,10} The prognosis for diffuse subependymal periventricular metastases is poor since the delay between the onset of the first neurological symptoms and death never exceeded 2 months.\textsuperscript{10} Clinically, patients with diffuse subependymal periventricular metastases presented with indifference and increasing, often fluctuating, drowsiness. Besides these non-specific symptoms, focal neurological signs in patients could be related to associated hematoma, infarct, or intraparenchymatous metastasis.

The histological diagnosis in previous cases was small cell carcinomas from the lung, and malignant melanoma (Table 1). Small cell carcinoma of the lung tends to metastasize to other organs including the brain, and produces a hormone which has affinity for other tissues. However, tumors causing metastatic infiltration of the leptomeninges, including intraventricular dissemination, are most frequently carcinoma of the breast (36\%) and lymphomas (28\%), and only 16\% were caused by lung tumors.\textsuperscript{5,10}

Three possible mechanisms for dissemination into the ventricular wall have been proposed. First, tumor cells may infiltrate directly into the subependyma forming a diffuse sheet on the wall of the ventricle. Second, tumor cells may metastasize to the cerebral parenchyma heterogeneously, enlarge, and then break with dissemination into the ventricular wall or subarachnoid space resulting in plaque-like depositions. Thirdly, both of these mechanisms may occur simultaneously.\textsuperscript{2,4,6,7} In the previously reported cases, the tumors most likely infiltrated directly to the subependyma as there was no solid tumor in the parenchyma. In our case, we suspected that the tumor broke directly and then disseminated throughout the wall of the lateral ventricle because the solid metastatic tumor was close to the ventricle and the choroid plexus was not markedly enhanced as the tumor involved. The subependymal spread of the tumor and the absence of moderate involvement of the choroid plexus and leptomeningeal spaces suggests that the latter is a consequence of periventricular tumoral infiltration rather than its cause. Therefore, retrograde seeding of the periventricular areas by malignant cells in the CSF appeared less likely. Periventricular seeding of tumor cells manifests as

![Image](https://via.placeholder.com/150)

**Fig. 3** Repeat axial (left), coronal (center), and sagittal (right) \( T_1 \)-weighted MR images with Gd-DTPA obtained a month after admission, showing enhancement of the wall of the third ventricle, pituitary stalk, and pituitary gland (arrow).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Summary of patients with metastatic tumor dissemination to the ventricular wall</th>
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<tbody>
<tr>
<td>Case No.</td>
<td>Author (Year)</td>
</tr>
<tr>
<td>1</td>
<td>McGeachie et al. (1977)\textsuperscript{11}</td>
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<tr>
<td>2</td>
<td>Vannier et al. (1986)\textsuperscript{10}</td>
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<td>3</td>
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<td>5</td>
<td>Present case</td>
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enhancement of the ependyma-subependyma in either a diffuse or nodular pattern.

The MR imaging appearance of metastatic brain tumors is generally identical with that of glioma: iso- or low intensity signals on the T1-weighted images and iso- or high-intensity signals on the T2-weighted images. The relationship between the MR signal intensity and the possible histology of the primary malignancy is unclear. Therefore, differentiating between primary intracranial malignant tumors and metastases associated with ventricular dissemination is not easy. In our case, glioblastoma was first suspected. Retrospectively, some features were consistent with metastases. Most metastatic tumors are round and have regular enhancement, and mass effect may be more apparent with a glioblastoma than with a metastasis.

The differential diagnosis for a tumor in the cerebral parenchyma with ventricular dissemination includes both primary intracranial malignant tumor such as glioblastomas, malignant lymphomas, and germinomas, but also secondary tumor. MR imaging is useful for the diagnosis of such tumors, but the final diagnosis should be based on either tissue biopsy or cytological examination of the CSF.

References


Address reprint requests to: K. Matsumoto, M.D., Department of Neurological Surgery, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama 700, Japan.