

Case Report

Intracranial Dissemination from Spinal Cord Anaplastic Astrocytoma

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We report a case of intracranial dissemination developing approximately 4 months after partial removal of a spinal cord anaplastic astrocytoma in a 22-year-old male. He presented with paraplegia on initial admission at a local hospital. Spinal magnetic resonance (MR) images disclosed multiple intramedullary lesions at the T3-11. The tumor was partially removed. The final histologic diagnosis was anaplastic astrocytoma. Four months after the operation, he was admitted with the symptoms of headache and deterioration of consciousness. MR images showed enhanced lesions in the anterior horn of the left lateral ventricle, and septum pellucidum. He underwent computed tomography-guided stereotactic biopsy and histological appearance was consistent with anaplastic astrocytoma. The clinical course indicates that the tumor originated in the spinal cord and extended into the subarachnoid space, first the spinal canal and later intracranial.

KEY WORDS : Anaplastic astrocytoma · Spinal cord · Intracranial dissemination.

INTRODUCTION

Spinal cord gliomas are rare, accounting for only 1% of all primary neoplasms in the central nervous system. Intracranial dissemination from spinal cord are particularly rare^{2,7-9}. We describe a case of spinal cord anaplastic astrocytoma with intracranial dissemination which onset with paraplegia. The mechanism of dissemination of spinal cord gliomas to intracranial space is still unclear. It is considered to have metastasized intracranially through the spinal subarachnoid space and ventricles after partial removal of the primary lesion.

CASE REPORT

A 22-year-old male was admitted to a local hospital complaining of paraplegia. He was diagnosed as an acute transverse myelitis. Initial brain magnetic resonance (MR) images showed no abnormalities (Fig. 1). He was treated

with high dose steroid intravenously. However, 7 months later, he was admitted to our hospital with progressive left upper limb numbness. Spinal MR images disclosed a mass lesion at the T3-11 levels appearing as a hyperintense area on both T1- and T2-weighted images (Fig. 2A). The tumor was partially removed through a laminectomy at T8-10. The histological diagnosis was anaplastic astrocytoma, immunochemical stain showing GFAP(+), P53(+), EGFR(+). Whole spine irradiation (total 50 Gy) was given. Four months after the operation, he was admitted with headache, and deterioration of consciousness. MR images showed enhanced lesions in the anterior horn of the left lateral ventricle, and septum pellucidum (Fig. 2B). He underwent CT-guided stereotactic biopsy and histological appearance was consistent with anaplastic astrocytoma (Fig. 3A, B). Immunochemical staining showed GFAP(+), P53(+), EGFR(+). Diagnosis was same anaplastic astrocytoma confirmed by immunocytochemistry. Despite the conservative treatment in the intensive care unit, his general condition and neurological status gradually deteriorated. He died from the tumor progression.

DISCUSSION

Growth of a spinal intramedullary glioma into the cranial cavity is a rare event, especially in cases of anaplastic

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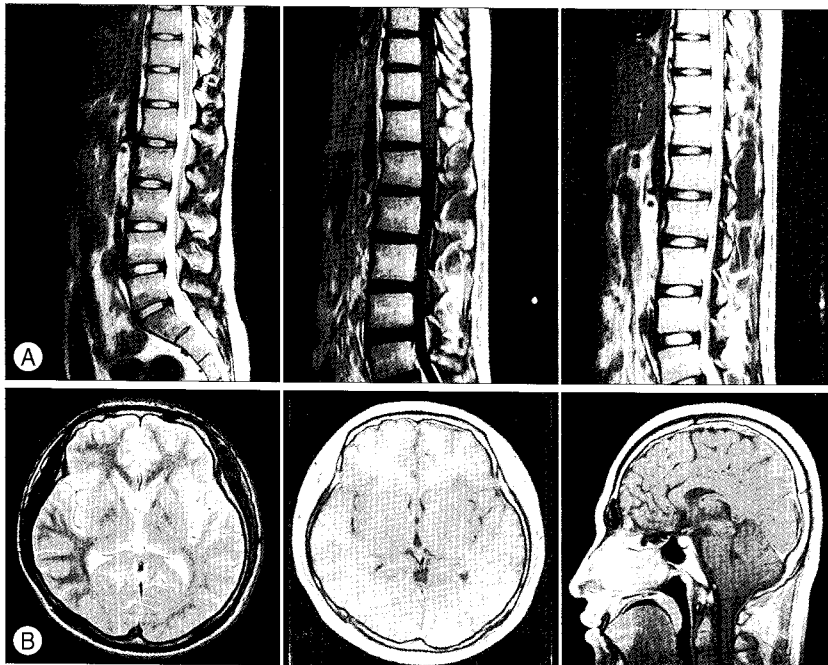


Fig. 1. A : Spinal T2-weighted magnetic resonance (MR) image (left), T1-weighted image (middle), and T1-weighted enhanced image (right) showing an intramedullary spinal tumor at T8-11 levels. B : Brain T2-weighted MR image (left) and T1-weighted enhanced images (middle and right) showing no evidence intracranial tumor

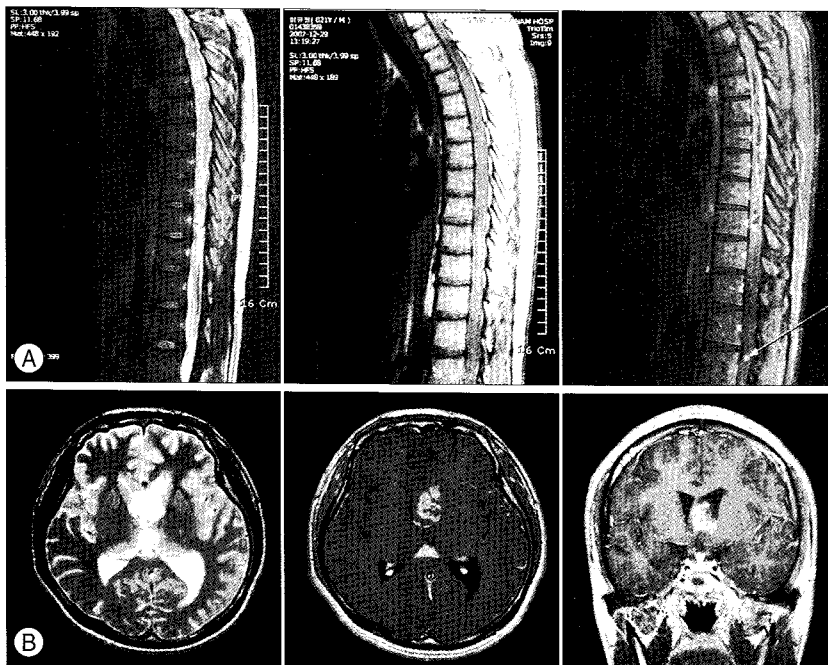


Fig. 2. A : Spinal T2-weighted magnetic resonance (MR) image (left), T1-weighted image (middle), and T1-weighted enhanced image (right) showing an intramedullary spinal tumor at T3-11 levels and distant seeding to L2 level. B : Brain T2-weighted MR image (left) and T1-weighted enhanced images (middle and right) showing enhanced lesions in the anterior horn of the left lateral ventricle, septum pellucidum.

astrocytoma. Intracranial seeding of spinal cord glioma may occur due to dissemination of the tumor into the spinal subarachnoid space and subsequent intracranial spread into the cerebral subarachnoid space or brain parenchyma^{1,2,7-9}. In addition, there is a possibility that a spinal intramedullary

tumor may grow directly into the cranial cavity in an exophytic manner. However, exophytic growth of a spinal intramedullary glioma is extremely uncommon. Intracranial dissemination is more common in patients with malignant glioma after surgical manipulation^{7,9}. This possible mechanism can be also applicable to our case. Surgical manipulation of a spinal cord anaplastic astrocytoma may facilitate tumor cell seeding to the subarachnoid space, resulting in intracranial dissemination^{1,2,7-9}.

Also, previous studies show increasing values of Ki-67/MIB-1 labeling index correlates with increasing grade of malignancy and proliferation^{4,5}. Although there is not anaplastic astrocytoma, a report present that central neurocytomas with MIB-1 labeling index over 10% showing rapid tumor growth and dissemination⁶. The spinal tumor immunohistochemistry present Ki-67/MIB-1 labeling index 10-20% in this case (Fig. 3C). Therefore, in spinal cord anaplastic astrocytoma patient, increased Ki-67/MIB-1 labeling index is very important factor that causes intracranial dissemination and poor prognosis.

The median survival from surgery to death is generally 6 months to 1 year in spinal cord anaplastic astrocytoma patient³. Intracranial dissemination is an ominous feature characterizing the terminal stage of disease. In our patient, intracranial dissemination appeared suddenly, followed by rapid neurological deterioration and death. Intracranial dissemination is a major indicator of a poor prognosis and makes it difficult to treat local recurrence. Therefore, the first priority is the prevention of dissemination when initially treating spinal cord anaplastic astrocytoma. We conclude that an aggressive approach using radical surgery, entire neuraxis irradiation, and adjuvant chemotherapy should be considered as the initial treatment for high-risk young subjects with Ki-67/MIB-1 labeling index over 10%.

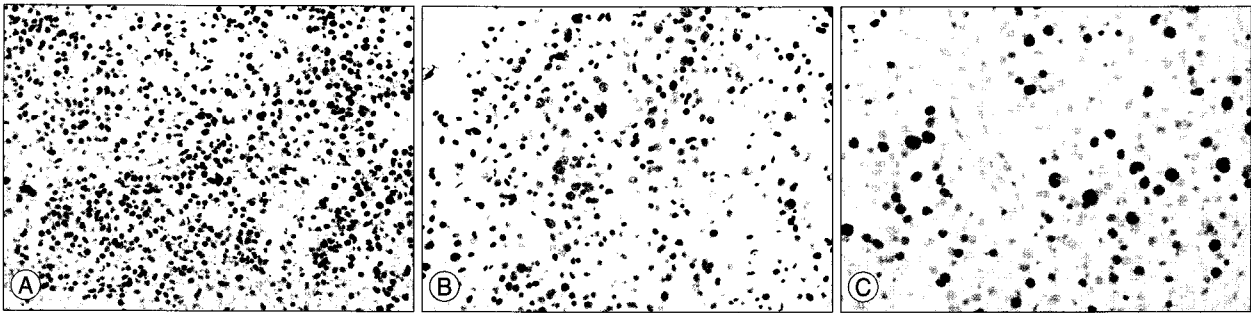


Fig. 3. A : Photomicrographs of a specimen from brain tumor. The photograph reveals anaplastic astrocytoma with moderate cellularity and pleomorphic nuclei (H & E, $\times 400$). B : Photomicrograph of a specimen from spinal tumor. The photograph reveals same findings of brain specimen (H & E, $\times 400$). C : Photomicrograph of a specimen from spinal tumor. The photograph reveals Ki-67/MIB-1 labeling index 10-20%.

CONCLUSION

Growth of a spinal intramedullary glioma into the cranial cavity is rare event, especially in cases of anaplastic astrocytoma. Intracranial dissemination from a thoracic spinal cord anaplastic astrocytoma is associated with tumor cell proliferative state. Ki-67/MIB-1 labeling index presents tumor cell proliferation and prognosis. Ki-67/MIB-1 labeling index over 10% are considered with understanding the cause of rapidly dissemination of anaplastic astrocytoma.

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