Intracranial Metastasis of a Spinal Cord Astrocytoma

—Case Report—

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Abstract

The authors report a case of cerebral metastasis from a spinal cord astrocytoma. The first metastatic tumor was controlled by radiation therapy. A second metastatic brain tumor was detected 2 years later, but attempts to control it through subtotal removal and chemotherapy failed. Metastasis from a spinal cord tumor to the brain is a rare event.

Key words: astrocytoma, spinal glioma, metastatic brain tumor

Introduction

Primary brain tumors often disseminate into the spinal cord, and for this reason, postoperative prophylactic radiation therapy to the whole neuraxis has been recommended in cases of ependymoma, germinoma, and medulloblastoma. However, intracranial metastasis from primary spinal cord tumors is very rare; only 24 cases have been reported in the literature. In most these cases, the tumor metastasized to the subarachnoid space or ventricular system. Metastasis to the brain parenchyma is thought to be extremely rare.

We encountered a case of cerebral metastases from a spinal cord astrocytoma. Among the reported cases of metastatic brain tumors originating from spinal cord astrocytomas, our patient had the longest survival.

Case Report

A 44-year-old male was hospitalized in April, 1979 with a 7-month history of low back pain and progressive weakness of the right leg. Neurological examination revealed paraparesis with sensory loss at the L4 level on the right side and the L5 level on the left. Urinary and fecal incontinence was also noted. Surgery was performed on May 25, 1979, under a diagnosis of intramedullary spinal cord tumor. The

tumor, which extended from the conus medullaris, was subtotally removed. The upper portion of the tumor was in the spinous process at the Th12 vertebra. Histological examination disclosed a low-grade astrocytoma. Postoperatively, he reported relief from back pain, but flaccid paraparesis persisted unchanged.

During the 3 years after the first operation, the lumbago and paraparesis gradually worsened. A neurological examination performed in December, 1982 demonstrated anesthesia of the right foot and the lateral aspect of the right leg. The upper limit of hypesthesia extended to the L1 level. Regrowth of the spinal cord tumor was suspected, and radiation therapy was administered at a total dose of 36.5 Gy delivered to the Th11-L2 area. However, the motor weakness of the lower extremities did not improve.

He was referred to our hospital in November of 1983, complaining of severe lumbago and memory disturbance. A computed tomographic (CT) scan of the brain showed moderate hydrocephalus and a homogeneously enhanced mass extending from the anterior half of the third ventricle to the corpus callosum (Fig. 1). A ventriculoatrial (VA) shunt was inserted on December 29, 1983, and this procedure was followed by radiation therapy to the brain at a total dose of 60 Gy. Cytological examination of the cerebrospinal fluid (CSF) was negative both before and after shunting. Signs of increased intracranial pressure were alleviated, and CT scans showed gradual reduction in the size of the enhanced mass.

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Fig. 1 Postcontrast CT scan taken on December 12, 1983, showing symmetrical dilatation of the lateral ventricle. Part of the corpus callosum and the anterior half of the third ventricle are homogeneously enhanced.

He exhibited confabulation and disorientation in July of 1985, and a CT scan demonstrated a heterogeneously enhanced mass in the right frontal lobe. At this time, the sensory loss extended to the Th5 segment. Magnetic resonance (MR) imaging disclosed swelling of the spinal cord from the Th11 to the L2 level (Fig. 2). A second operation was carried out on November 11, 1985, and a reddish-brown tumor was subtotally removed from the spinal cord. The tumor was hemorrhagic and fragile, and its margin was poorly defined. The histological diagnosis was astrocytoma, grade III (Fig. 3).

He soon developed headaches and vomiting, which gradually became more frequent. Enlargement of the frontal lobe tumor was demonstrated by both postcontrast CT (Fig. 4) and MR imaging. A reddish-brown tumor around the right frontal horn was partially removed on February 19, 1986. The tumor was histologically diagnosed as a grade III astrocytoma (Fig. 5). Postoperatively, his condition worsened despite intracarotid injection of ACNU and systemic administration of beta-interferon. Intratumoral hemorrhage occurred on May 16, 1986, and he died on May 30, at the age of 50 years. Autopsy was not permitted.

Discussion

There have been 24 reported cases of metastatic brain tumor originating from spinal cord gliomas^{1-7,9,21)} (Table 1). The patients' ages ranged from 9 to 54 years, with an average of 26 years. Most patients were relatively young; only four were over 40 years of age. Malignant glioma of the spinal cord

Fig. 2 MR images of the spinal cord, sagittal view. The spinal cord is swollen from the Th11 to the L2 level. *left*: Saturation recovery, repetition time (TR) 600 msec, echo time (TE) 45 msec (T_1 -weighted image). *right*: Saturation recovery, TR 1000 msec, TE 45 msec.

was more common in the older patients, the oldest of whom was 54 years old and had a grade III astrocytoma.

At the time of his death, our patient was the third oldest of all those reported. Seven years passed between the first operation and death, and the total clinical course was 7 years and 8 months. This was much longer than the average of 36.9 months among the previously reported cases ($n = 18$). The longest clinical course, that of a patient with an ependymoma, was 29 years.¹³⁾ In our patient, the period from the first spinal surgery to the appearance of the first brain metastasis was 4.5 years, which is also much longer than the reported average of 11.2 months ($n = 11$). The outcome for these patients was very poor; death usually occurred within

Fig. 3 Photomicrograph of the intramedullary spinal cord tumor (grade III astrocytoma). The tumor cells contain thin fibrillary cytoplasmic processes and the nuclei vary in size. Glial fibrillary acidic protein (GFAP) staining was positive, but no necrosis was found. HE stain, $\times 100$.

Fig. 5 Photomicrograph of the right frontal lobe tumor (grade III astrocytoma). Many tumor cells contain thin cytoplasm. The nuclei are irregular in shape and vary in size. GFAP staining was positive. HE stain, $\times 100$.

Fig. 4 Postcontrast CT scan obtained on February 17, 1986. Irregularly enhanced masses are seen in the right frontal lobe and right basal ganglia. The tumor invades the left frontal lobe.

1 year. High-grade astrocytomas and glioblastomas had especially short clinical courses, and there have been no reports of survival.

In our patient, VA shunting and radiation therapy to the brain were temporarily effective in relieving the symptoms of the metastatic brain tumor, and useful life was sustained for a short time. Bleeding from the metastatic brain tumor (the ultimate cause of death in our case) had occurred in only two of the previously reported cases.^{2,18)} Although we were unable to obtain the specimen from the first operation, which was performed at another hospital, the primary tumor was diagnosed as a low-grade astro-

cytoma. Therefore, malignant changes apparently took place during the 6 years following the first operation.

In our patient, clinical evidence of extension of sensory loss to the Th5 segment was not in accord with the findings of MR imaging. This may have been due to the poor imaging quality of the apparatus that we used (resistive type, 0.2 Tesla) and failure to obtain MR images at the appropriate thoracic level. The extension of the anesthetic area together with the gradual increase in the size of the metastatic brain tumor supports our hypothesis that the tumor spread *via* the CSF pathway. Furthermore, whereas the first metastatic brain tumor was homogeneously enhanced on CT scans, the second was heterogeneously enhanced. Therefore, there may have been histological changes between the first and the second brain tumors.

Since direct surgery was performed on the spinal cord tumors in almost all the reported cases,^{12,18)} it is possible that surgical manipulation facilitated seeding of the intracranial cavity. In fact, signs of brain metastasis usually appeared soon after surgery for the original spinal cord tumor, and the symptoms of metastatic brain tumor rarely preceded those of the spinal cord tumor.^{7,14)}

Spinal cord gliomas can metastasize to the brain *via* the CSF pathway or the bloodstream, or they can directly invade the brain. Although metastasis occurred *via* the bloodstream in a case of ependymoma reported by Rubinstein and Logan,¹³⁾ the tumors metastasized to the basal cisterns in almost all the reported cases. In these cases, metastasis to the basal

Table 1 Reported cases of spinal cord glioma with intracranial metastases

Author (Year)	Age, Sex	Site of primary tumor	Histological grade*	Surgery	Interval from surgery to metastasis	Interval from onset to death	Radiation therapy	Hydrocephalus	Site of metastasis
Mallory (1908) ¹⁰⁾	child	lumbar	III-IV	NR	NR	NR	NR	NR	SS, cervical cord, pons, cerebellum
Greenfield (1934) ⁴⁾	48, M	cauda equina	medullo-epithelioma	+	NR	6 yrs	-	NR	SS, V
Eden (1938) ³⁾	19, M	lumbosacral	III (mixed glioma)	+	1 wk	7 mos	-	+	SS
O'Connell (1946) ¹¹⁾	16, M	thoraco-lumbosacral	IV	+	NR	16 mos	+	+	SS, V
Ressell (1949) ¹⁴⁾	37, M	cervical	oligodendro-glioma	-	NA	5 mos	-	+	SS, V
Perese <i>et al.</i> (1959) ¹²⁾	39, M	conus medullaris	II	+	11 mos	2 yrs 4 mos	+	+	SS, V, cerebellum
Russell and Rubinstein (1959) ¹⁵⁾	11, F	cervical	IV	-	NA	6 mos	-	+	SS, V
Rubinstein and Logan (1970) ¹³⁾	17, F	filum terminale	ependymoma	+	6 yrs	29 yrs	+	+	V, lungs, pleura, para-aortic lymph nodes
Eade and Urich (1971) ²⁾	21, F	thoraco-lumbosacral	II (mixed glioma)	+	4 mos	8 mos	+	NR	SS, V
	21, F	thoracic	II (mixed glioma)	+	3 mos	11 mos	+	+	SS, V
	19, M	thoraco-lumbosacral	II (mixed glioma)	+	NR	6 mos	+	+	SS, V
Russell and Rubinstein (1971) ¹⁶⁾	16, F	conus medullaris	III-IV	NR	NR	NR	NR	+	SS
Salazar and Rubin (1976) ¹⁷⁾	NR	NR	IV	NR	NR	NR	+	NR	NR
	NR	thoracic	IV	NR	NR	NR	+	NR	brainstem, cerebellum, thalamus
Tashiro <i>et al.</i> (1976) ²¹⁾	12, F	thoraco-lumbosacral	IV	+	3 wks	11 mos	-	+	SS, V, CC, hypothalamus, thalamus, brainstem, cerebellum
Andrews <i>et al.</i> (1978) ¹⁾	45, M	thoraco-lumbosacral	IV	+	11 mos	1 yr 1 mo	+	+	SS, V, fornix, septum pellucidum
Simonati <i>et al.</i> (1981) ¹⁹⁾	19, F	thoracic	II	+	NR	5 yrs	+	+	SS, V
Kopelson and Linggood (1982) ²⁰⁾	32, F	NR	II (mixed glioma)	+	NR	NR	+	NR	midbrain
Hely <i>et al.</i> (1985) ⁵⁾	38, F	thoracic	III-IV	-	NA	9 mos	+	+	SS, V, hypothalamus, thalamus, midbrain, pineal region
	19, F	conus medullaris	II	+	9 mos	2 yrs 4 mos	+	+	SS, V, cerebellum, CC, basal ganglia, frontal lobe
Takara <i>et al.</i> (1985) ²⁰⁾	20, M	thoracic	IV	+	2 mos	6 mos	+	+	SS, V, cerebellum, brainstem
Sarabia <i>et al.</i> (1986) ¹⁸⁾	54, M	thoracolumbar	III	+	6 mos	1 yr 1 mo	+	+	SS, V, CC, chiasm
Johnson and Schwarz (1987) ⁶⁾	9, F	thoraco-lumbosacral	III	+	4 mos	17 mos	+	+	SS
Kendrick <i>et al.</i> (1987) ⁷⁾	41, F	thoracic	IV	+	NA	NR	-	+	SS
Present case	44, M	conus medullaris	II (1st), III (2nd)	+	4 yrs 6 mos	7 yrs 8 mos	+	+	SS, V, CC, frontal lobe, basal ganglia

*After Kernohan and Sayre.⁸⁾ NR: not reported, NA: not applicable, SS: subarachnoid space, V: ventricles, CC: corpus callosum.

cisterns and ventricular system may have occurred through the CSF space, after which ventricular dilatation and signs of increased intracranial pressure became manifest.^{6,12)} In our case, the fact that the metastatic mass was near the ventricular system suggests metastasis *via* the CSF pathway.

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