

Intramedullary thoracic spinal cord metastases of cranial glioblastoma

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Abstract: Intramedullary thoracic spinal cord metastases of cranial glioblastoma are very rare, but are increasing as patients live longer. Choucair and Schuster, et al estimated the probability of such metastases at 0.4 - 1.2%. We report a case of 54-year-old woman who presented paraparesis with bladder dysfunction 16 months after undergoing craniotomy for glioblastoma multiforme. The spinal magnetic resonance images showed a thoracic intramedullary mass which was confirmed as glioblastoma metastasis and the patient died 2 months later. We discuss this case with review of the literature focusing on clinical, paraclinical, therapeutical data and prognosis of spinal cord metastases. (p108-111)

Key words: Spinal cord metastases and cerebral gliomas

Introduction

Symptomatic intra and extraspinal metastases in patients with primary intracranial gliomas are rarely described, but the longer the patients survive postoperatively, the higher the chance for developing additional tumours including spinal metastases. Choucair and Schuster, et al estimated the probability of such metastases at 0.4 - 1.2%.⁴

To date, the cause of the metastases spread still remains under discussion. In the following report, we present our own case focusing on the diagnostic difficulties, the origin of this metastases and the therapeutic options.

Case Report

A 54-year-old woman had recurrent generalised seizures without any neurological deficits or other medical problems. Computed tomography (CT) scan of the head confirmed right frontal glioblastoma (Fig. 1), which was removed completely via a right frontal craniotomy.

Histopathological examination corroborated a multiforme glioblastoma (Fig. 2). Immunohistochemical study was

positive with NSE and PS100, and negative with anti-LC, anti-Pan T, anti-Pan B, anti-cytokeratin, desmin and vimentin.

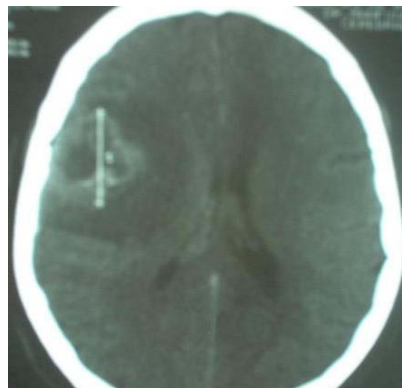


Figure 1 – CT scan: A right parietal mass with heterogeneous enhancement after contrast: Glioblastoma multiforme

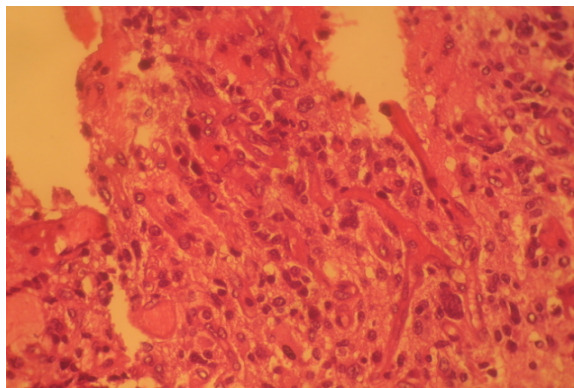


Figure 2 – A right frontal glioblastoma multiforme HE x 400 proliferating malignant astrocytes cell tumour

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The postoperative course was uneventful. Control head CT scan showed no residual tumour (Fig. 3). Postoperative radiation therapy with 55 Gy in the tumoural location and 35 Gy for the head was performed and well tolerated by the patient. No further seizures appeared during the follow-up and the patient returned to normal daily activities.



Figure 3 – Postoperative control: Total resection

Sixteen months after the operation, the patient complained of back pain radiating to the liver region which was diagnosed as vesicular lithiasis and patient underwent surgical cholecystectomy, but the back pain increased and we diagnosed heavy paraparesis with sensory loss in lower limbs and urinary retention. Magnetic resonance imaging (MRI) of the spine revealed a thoracic intra-dural lesion between the levels T4 and T5 (Figs. 4 and 5). The preoperative



Figure 4 – Sagittal MRI on T1 after gadolinium shows intramedullary mass on T4 - T5 level with homogeneous enhancement



Figure 5 – Sagittal MRI on T2: Hyperintense lesion in the same level (T2 - T5)

diagnosis was meningioma or neurinoma (context of neuro-fibromatosis) or primitive intramedullary tumour, or less probably metastases of the intracranial glioblastoma. Cranial CT scan showed a recurrent tumour with diameter of 1.5 cm in the right frontal lobe (Fig. 6). The patient

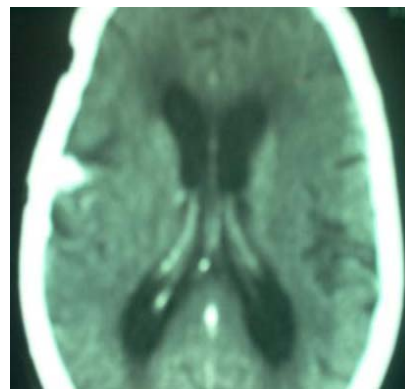


Figure 6 – CT scan with contrast 16 months after the first intervention (craniotomy): A small mass with enhancement (recurrence of glioblastoma) is shown

underwent T3 - T6 laminotomy. After opening the dura, the tumour was intramedullary and necrotico-haemorrhagic. Histopathology of tumour biopsy confirmed metastases of multiforme glioblastoma (Fig. 7). The postoperative course was marked by decrease of pain with complete paraplegia. The patient could not support spinal radiation therapy and died two months after the spinal operation with pulmonary emboli.

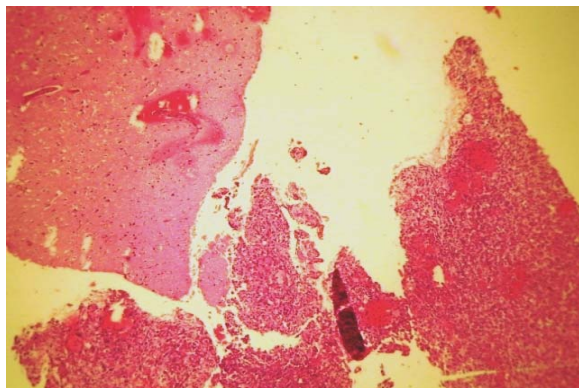


Figure 7 – HE x 100 showing intramedullary metastase of glioblastoma multiforme

Discussion

Symptomatic spinal metastases in patients with multiforme glioblastoma are very rare. Choucair, et al found extraneural metastases in 0.1% of patients with malignant cranial gliomas and described 1.38% of patients with multiforme glioblastomas, who had multiple CNS lesions involving the spinal cord.⁴ Based on the present findings of autopsies, 20 - 40% of supratentorial gliomas tend to metastasize into the spinal canal, whereas infratentorial gliomas do so in up to 60%. The number does not differ substantially from earlier reports of Cairns and Eden.^{3,5} On the other hand, the number of symptomatic metastases seems to increase. It is thought two main reasons might be responsible for this increase in frequency.⁸ Firstly, diagnostic tools have improved now CT and MRI are readily available. They certainly assist in the early diagnosis of spinal metastasis in the absence of neurological deficit/s, but not in our case in which a diagnostic mistake was made before the patient deteriorated with paraplegia. The prolonged survival time due to improved therapy regimes may be the second cause.

The mean interval time between diagnosis of intracranial disease and diagnosis of metastases is 14.1 months, as in our case (16 months). Hamilton reported a case which was a single intramedullary thoracic spinal cord glioblastoma diagnosed 10 months after treatment for supratentorial glioblastoma.⁷

As risk factors for this complication, Seidel, et al thought the young age of the patient, the anaplastic parts of the tumour and the midline location of the primary tumour might be contributory to metastases.¹⁰

Firsching, et al suggested that leptomeningeal dissemination in patients with gliomas may occur when the primary tumour reaches the circulation of the cerebrospinal fluid (CSF), as in the first case of Buhl, et al in which the close

location of the primary tumour to the lateral ventricle and the vicinity of the ventricular system could be an explanation for the CSF spread of tumour cells, and leptomeningeal craniospinal involvement could be the result of metastases through subarachnoid space.^{2,6}

Generally, metastases are mainly the result of intraoperative manipulations, thus releasing tumour cells and scattering them via the blood, CSF or lymph fluids. Rare cases of glioblastomas have been described in which invasion of the dura veins or sinuses led to spontaneous extracranial metastases in the absence of a previous craniotomy.¹

The spread of glioblastoma multiforme is also an important factor for further treatment. Kochi, et al reported that the ventriculolumbar perfusion of 3-(4-amino, 2-methyl, 5-pyrimidinyl, methyl)-1-(2-chloroethyl)-1-nitro-sourea hydrochloride is a safe and feasible treatment against subarachnoid dissemination of primary nervous system tumours.⁹

However, once symptomatic metastases were diagnosed, not one of the treatment modalities (surgery, irradiation and chemotherapy) was able to stop the fast and finally fatal progression of the disease.⁴ In fact, after diagnosis of tumour spread, subsequent mean survival time is 2.8 months.¹¹ In our case it was two months.

Conclusion

Symptomatic spinal metastases of cranial multiforme glioblastoma are very rare. The follow-up of patients operated for cranial multiforme glioblastomas must be clinical and radiological to detect any possible spinal metastases. The clinical examination has to be vigilant to ensure there is no false diagnosis. Radiological follow-up should include spinal MRI every 6 months after the cranial removal. The prognosis is still very poor although prolongation of survival can be obtained with combined therapy.

References

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GENTLE REMINDER

Localisation of tumours of the nervous system

Brain		Spinal Cord
Supratentorial	Infratentorial	
<p>Hemispheres Glioblastoma multiforme Astrocytoma Oligodendroglioma Meningioma Metastases</p> <p>Midline Pituitary tumours Pineal tumours Craniopharyngioma</p>	<p>Adult Acoustic neuroma Metastases Meningioma Angioblastoma (von Hippel-Lindau)</p> <p>Children Cerebellar astrocytoma Medulloblastoma Ependymoma Brain stem glioma</p>	<p>Extradural Metastases Dermoid</p> <p>Intradural extramedullary Meningioma Neuroma Angioma</p> <p>Intradural intramedullary Ependymoma Astrocytoma Metastases</p>