Surgical Science, 2011, 2, 140-143
doi:10.4236/ss.2011.23029 Published Online May 2011 (http://www.scirp.org/journal/ss)

Clinical and Pathological Studies of Meningioma-Glioma Mixed Tumor

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Received February 20, 2011; *revised April* 2, 2011; *accepted April* 8, 2011

Abstract

Meningioma-glioma mixed tumor is rare central nervous system tumor. It is necessary to study its clinical and pathological characteristics as well as its possible genesis. This case was a 54-year-old man who was readmitted for recurrent glioma. Magnetic resonance imaging showed a big mass in right temporal lobe which was confirmed as meningioma-glioma by immunohistochemical analysis. Specific immunohistochemical staining is significant in tumor differential diagnosis, and helps to confirm tumor histic origin. By pathological studies, we found that glioma could stimulate adjacent normal meninges into neoplastic proliferation.

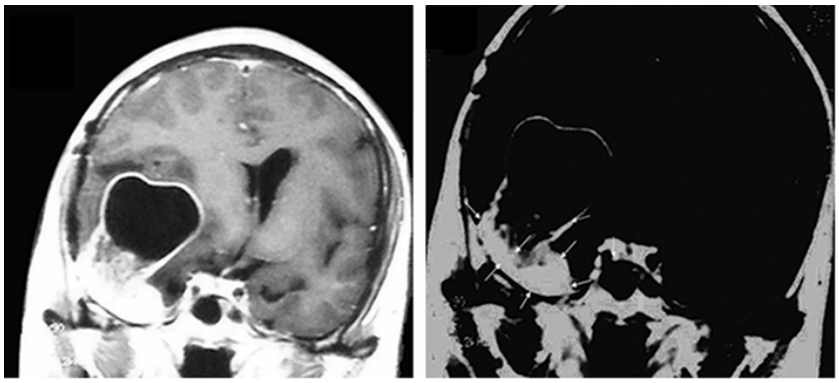
Keywords: Glioma; Immunohistochemical Staining; Meningioma; Mixed Tumor; Neoplastic Proliferation

1. Introduction

The term “meningeal gliomatosis” was firstly adopted to designate involvement of the leptomeninges by tumors of neuroectodermal origin by Polmeteer [1]. The authors primarily did studies about metastatic implantations from gliomas to the leptomeninges of the brain and the spinal cord and the ependyma of the ventricular system. These gliomas established themselves in multiple sites via cerebrospinal fluid (CSF) system and were responsible for a generalized involvement of the leptomeninges. Usually, there are no differences in tumor cell morphous between the meningeal gliomas and intracerebral gliomas. We presently report a special case of meningioma-glioma which possessed meningioma pathological morphous, but its immunohistochemical characteristics belong to glioma.

2. Case Report

This 54-year-old man was readmitted for “recurrent glioma in the right temporal lobe” on October 27, 2008. In August, 2007, he was admitted with onset of iterative headache and abduction limitation of the left eyeball. MRI showed a mass (size 3.0 × 3.0 cm), surrounded by digitatus type of cerebral edema in the right temporal lobe. The ventriculus dexter cerebri was severely extruded. The entire mass was resected after the first operation. Pathological diagnosis was glioma (Grade IV) with glial fibrillary acidic protein (GFAP) positive of immunohistochemical staining. The patient refused radiotherapy and chemotherapy after the first operation because of financial limitation. In October 2008, he felt headache again, accompanied by limbs’ acratia, function loss of left eyeball abduction, myodynamia decrescence of limbs and malfunction of equilibrium. MRI showed a mass (size 5.0 × 6.8 cm) with a bursa located in the right temporal lobe, and the ventriculus dexter cerebri was extruded. Enhanced MR images showed the capsule wall in the superior part and the inferior part of the mass was significantly enhanced (**Figures 1(a)** and **(b)**). The patient’s second operation was performed on October 29, 2008. About 20 ml intraluminal fluid was drawn from the cavum inside the mass. After we resected the soft and fish-flesh-like tissue of its superior part, an elliptic and smooth hemisphere mass appeared at the bottom of the middle cranial fossa with a clear and smooth boundary, which involved the anterior part of the tentorium of cerebellum. In this one solid tumor, there were no interlaced tissues found between the two parts, in spite of the fact that they were closely proximate in his MRI and during operation. Pathological examination showed the spherical or elliptic cells with mild heteromorphosis and plentiful cytoplasm of the superior part tissue. The cells



(a) (b)

Figure 1. MRI for the patient before his second surgery. (a) A mass (size 5.0 × 6.8 cm) with a bursa located in right temporal lobe, the ventriculus dexter cerebri was extruded. (b) After image contrast processing, the inferior part tumor was markedly illustrated, which was indicated by arrows.

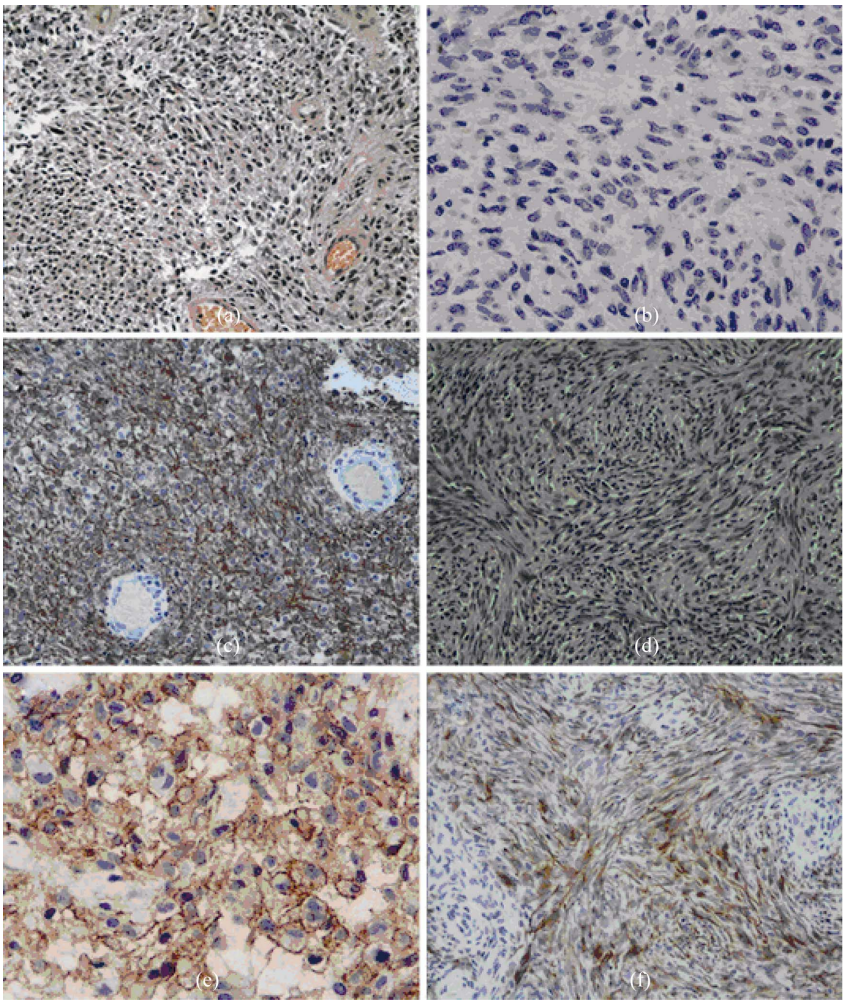


Figure 2. Histopathology of specimens at the time of diagnosis of the tumor which was resected in second surgery for the patient. (a) HE staining shows spherical or elliptic cells of superior part tumor, with mild heteromorphosis and plentiful cytoplasm (× 200). (b) EMA staining for superior part tumor cells is negative (× 400). (c) GFAP staining for superior part tumor cells is positive (× 200). (d) HE staining shows cells of inferior part tumor, composed of spindle cells arranging in whorl patterns (× 200). (e) EMA staining for inferior part tumor cells is positive (× 400). (f) GFAP staining for inferior part tumor cells is positive (× 200).

were active in growth and accompanied with abundant vascular proliferation and extensive tissue necrosis. All of these indicated glioma (**Figure 2(a)**). The immunohistochemical staining showed epithelial membrane antigen (EMA) negative, GFAP positive (**Figures 2(b)** and **(c)**). However, the inferior part of the tumor was outside the cerebrum and based at the bottom of the middle fossa. Microscopically, this part composed of spindle cells arranged in whorl patterns. The cells grew slowly and mildly with no karyokinesis. These were characteristics of meningioma (**Figure 2(d)**) with EMA positive (**Figure 2(e)**) and GFAP positive (**Figure 2(f)**) of immunohistochemical staining. The patient recovered well after operation, under advice of radiotherapy and chemotherapy.

3. Discussion

In this case, we found the inferior part, extracerebral smooth, tough, and firm tumor, which located in the bottom of the middle cranial fossa, had defined and clear border to the intracerebral upper part tumor. Pathological findings indicated that the upper part tumor exhibits morphological characteristics of glioma, as well as GFAP positive and EMA negative. Meanwhile, inferior part tumor reflect the morphing characteristic of meninges, but its immunohistochemical staining shows GFAP positive, and EMA positive appeared in cells arranging in whorl patterns. EMA is specific marker for normal epithelium or epithelial origin tumor which expressed in most meningioma, but usually negative in glioma cells [2]. As a kind of intermediate filaments in gliocyte, GFAP expressed in most astrocytoma and was considered as the major evidence for diagnosing glioma [2,3]. In our case, the inferior part tumor exhibits the basement of meninges and there was a clear boundary with the upper part tumor even though they were in one solid mass in MRI. Positive of EMA indicated that tumor cells of the inferior part originated from meningothelium, and positive of GFAP indicated its features of glioma. We think the term "meningioma-glioma" was more suitable than "meningeal glioma" for our case.

Our meningioma-glioma case is different from previously reported cases of meningeal gliomatosis. The latter concerned with tumor-like lesions in meninges of the brain or the spinal cord or the ependyma of the ventricular system caused by metastatic implantations from intracerebral glioma. Those metastatic glioma cells established themselves in multiple sites via CSF system, caused clinical manifestation like meningitis, and usually, there're no differences in cell characteristics between meningeal gliomatosis and intracerebral gliomas [4,5]. Moreover, previous researches showed that glioma cells which were cultured in normal leptomeningeal extracellular matrix proteins still kept the features of gliocytes [6]. This suggested that if glioma cells invaded meninges directly and proliferated, their features won't be changed by extracellular environment of meningocytes. To our meningioma-glioma case, it is not only there was a boundary between the superior and inferior parts but also the cells of the meningeal glioma possess features like meningioma cell rather than glioma cell, and immunohistochemical staining showed EMA positive. These indicated that this inferior part tumor was not an invaded or implanted tumor of intracerebral glioma cells but was a tumor originated from the meningocytes.

Cooper and Kernohan had suggested that primary leptomeningeal glioma originated from dedifferentiation of heterotopic glial nests [4]. This suggestion seemed unsuitable for our case, since the tumor divided into two parts (**Figure 1**) in one solid mass, the inferior meningeal glioma tight conjuncted the superior glioma. And there's no neoplasm in meninges in first surgery for glioma. We presumed that the recurrence malignant glioma stimulated the adjacent meninges into neoplastic proliferation, although there is no more knowledge about the biomechanism or material basis relate to this irritant action. For our case, this hypothesis seemed more reasonable than "leptomeningeal gliomas originated from dedifferentiation of heterotopic glial nests" [4]. Davis had given similar hypothesis after their research of concurrent meningioma and astrocytoma growth [7]. They considered that meningioma or glioma can stimulate the adjacent brain parenchyma or arachnoid cells into neoplastic proliferation. To our case, we consider that the irritant action from the malignant glioma not only caused neoplastic proliferation of meninges but also changed their phenotypes into expressing both EMA and GFAP. Further more works are needed to demonstrate the biomechanism and material basis relate to this kind of irritant action.

4. Conclusions

Pathological examinations are necessary for meningioma-glioma mixed tumor diagnosis. Specific immunohistochemical staining should be an important determination in differential diagnosis and could help confirm tumor histic origins. This rare tumor which we reported was recurrent and had meningeal neoplastic proliferation to primary tumor lesions and was considered as single and unitary tumor in MRI scans and intraoperative findings. Further pathological studies were necessary to reveal the tumor's real characteristics. Our case demonstrated that glioma may stimulate adjacent meninges into neoplastic proliferation, but related biomechanism and

material basis warrants further investigation.

5. References

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