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CASE REPORT

Regional skin invasion by glioblastoma multiforme

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Abstract Glioblastoma multiforme is the most common and aggressive primary malignant brain tumor and usually invades the intracranial space. Extracranial invasion is uncommon, and skin invasions are even less common. Here, we present an extremely rare case of aggressive skin invasion by glioblastoma multiforme. Once skin invasion occurs, its spread is too rapid to be resectable in only a few weeks and is often accompanied by deterioration of the primary lesion and short-term survival. Recognizing small cutaneous tumors adjacent to a previous surgical site is vital at the initial stage of skin invasion. Early recognition of cutaneous lesions, resection, and pathological confirmation, followed by chemotherapy and radiotherapy, may be beneficial for these patients.

1. Introduction

Glioblastoma multiforme (GBM) is the most common primary brain tumor and the most malignant of the astrocytomas, representing ~12—15% of all intracranial tumors and 50—75% of astrocytomas. GBM is an aggressive malignant astrocytoma classified as a World Health Organization Grade IV astrocytoma and has poor differentiation. The mean survival time of affected patients is usually < 2 years.[1] GBMs have various spread patterns, including white-matter metastases, which is the most common route,

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cerebrospinal fluid (CSF) dissemination, ependymal and subependymal spread, skull–dura invasion, and extracentral nervous system (CNS) metastases.[2] Cases of extracranial lesions are < 2%, with the lung and pleura, regional lymph nodes, spine, bone marrow, and liver being common sites.[3] In this study, in addition to reviewing relevant literature, we present an extremely rare and devastating case of regional skin invasion by GBM.

2. Case Report

In December 2012, a 50-year-old woman presented with right hemiparesis with a muscle power of 3–4 and Broca's aphasia. Imaging studies revealed the presence of a brain tumor on the left frontal–parietal area with peritumoral edema, for which she underwent immediate surgery. The tumor was pathologically confirmed to be GBM and after surgery, her clinical symptoms improved. Two weeks after the operation, she was administered concurrent radiotherapy with 6000 cGy for 30 courses and chemotherapy with temozolomide at 75 mg/m^2/d for 42 days. Additionally, she received a 5-day course of adjuvant chemotherapy with temozolomide at 200 mg/m^2/d every 28 days.

In May 2013, a small, firm, round, and immobile scalp tumor measuring 1 cm × 2 cm, with no inflammatory change or active bleeding, was noted adjacent to the previous craniotomy site. The tumor showed rapid progression in 1.5 months, with contiguous spread to the scalp, left preauricular area, and left neck (Figure 1). The lesions were characterized by firm, solid, multiple, and immobile nodules with no pus discharge, but easy bleeding. The patient also presented with worsening right hemiparesis and dysarthria. Magnetic resonance imaging (MRI) revealed cutaneous tumors in the subgaleal space, subcutaneous layer of the preauricular space, and left neck and recurrence of intracranial GBM with peritumoral edema (Figure 2). Resection of the tumor, including cutaneous and intracranial lesions, was planned. During surgery, no gross invasion of the dura matter and the bone flap by the tumors was observed (Figure 3). However, the patient died because of rapid neurological deterioration and respiratory failure 3 months after the scalp tumor was first recognized.

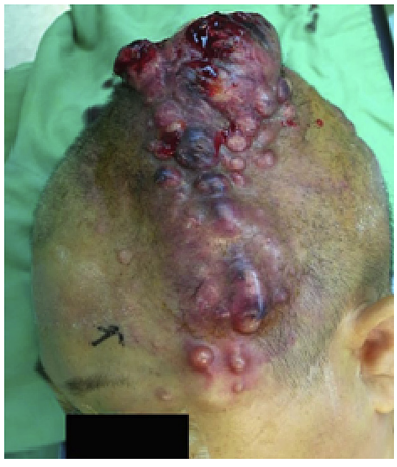

Figure 1 Glioblastoma multiforme with extracranial metastasis in a 50-year-old woman. Skin invasion progressed rapidly from a small scalp nodule to fulminant multiple lesions.

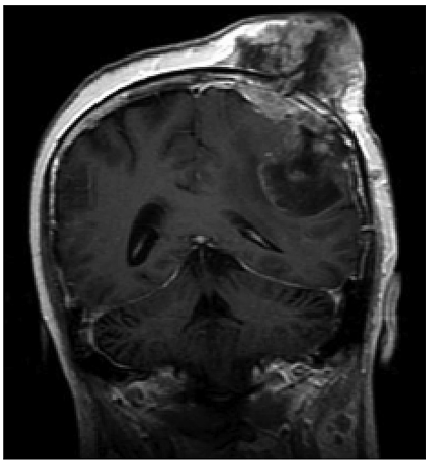

Figure 2 MRI (T2 + contrast) findings in a 50-year-old woman with glioblastoma multiforme. An intracranial tumor is present on the left frontal–parietal area and a large mass with subdural and scalp invasion along the previous surgical site is observed. MRI = magnetic resonance imaging.

Specimens from the subgaleal and subcutaneous lesions showed necrosis with dermal and subdermal infiltration (Figure 4). The tumors contained spindle-shaped, highly pleomorphic cells with abundant mitoses, necrosis, and microvascular proliferation and marked nuclear atypia. Immunostaining was positive for glial fibrillary acidic protein (GFAP). Immunohistochemistry of both primary parenchymal and cutaneous tumors showed similar features of focal GFAP positivity. These histological features confirmed skin invasion by GBM.

3. Discussion

GBM is the most common and aggressive primary brain tumor in adults and is characterized by a high local recurrence rate. It is the most common primary CNS tumor that causes "brain to brain" invasion. Dissemination to the pons, cerebellum, medulla, and spinal cord can also occur along the compact white tracts, including the corpus callosum, anterior commissure, fornices, and corticospinal tract. Other routes of intracranial spread include CSF dissemination, ependymal and subependymal spread, and skull–dura invasion. However, extracranial spread is rare, with an incidence rate of < 0.4–2%.[4] The first case of lung metastasis was reported in 1928 by Davis,[5] and to date, only 200–300 cases have been reported in the literature.[3,6,7] Although the mechanisms underlying extracranial spread remain unknown, some hypotheses have been proposed[8] to explain such low metastatic rates. First, patients do not survive long enough to have detectable extracranial invasion because they often have rapid enlargement of the brain tumor, which causes brain herniation and death. Second, the brain is physically protected by the thick basement membrane of the blood–brain barrier. Third, the absence of a lymphatic system in the brain appears to prevent distant spread.