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Giant Cell Glioblastoma Manifesting as Traumatic Intracerebral Hemorrhage

—Case Report—

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Abstract

A 33-year-old male presented with intracerebral hemorrhage in the left temporoparietal region after a traffic accident. Ten months later, the traumatic hemorrhage was found to originate in an underlying giant cell glioblastoma. Our case indicates that non-traumatic underlying pathologies, such as vasculopathies, coagulopathies, or tumors, should be considered in the differential diagnoses of intracerebral hemorrhage occurring in unusual locations after traumatic accidents.

Key words: glioblastoma, head trauma, intracerebral hemorrhage

Introduction

Intracerebral hemorrhagic lesions caused by head injury range from surface contusions to deep hemispheric hematomas. A patient with a bleeding tendency of any cause may develop large intracerebral hematomas after a minor trauma.[5] Glioblastoma is the most frequent cause of spontaneous intratumoral hemorrhage among the primary brain tumors.[2,3,7] The histological structure of glioblastoma includes abnormal fragile vessels with leaky walls which may shear and bleed during head trauma. We present a case of intracerebral hematoma in the temporoparietal region after trauma which originated from a preexisting glioblastoma.

Case Report

A 33-year-old male was referred to our clinic from a state hospital 200 km away on June 29, 1997. He had been a passenger in a traffic accident the day before. Computed tomography (CT) showed ethmoidal fracture and a heterogeneous hyperdense area with moderate perifocal edema in the left temporoparietal region, which was thought to be intracerebral hemorrhage (Fig. 1). His Glasgow Coma Scale score was 6 (E1M3V2). Both pupils were isocoric and reactive, and the other cranial nerve functions were intact. There was no sign of motor dysfunction and no pathological reflexes. Physical examination found bilateral epistaxis and swelling of the right hand due to multiple phalangeal fractures. His level of consciousness improved in 5 days, and repeat CT taken on the 4th day after the accident clearly showed no increase in the dimensions of the hemorrhage. On the 6th day he had a body temperature of 38°C, and signs of meningeal irritation. Analysis of the cerebrospinal fluid taken by lumbar puncture revealed meningitis, which was treated with ceftriaxone and vancomycin. Neurological examination found no abnormalities on the 18th day. He was discharged and was advised to present for magnetic resonance (MR) imaging after a month. He was lost to follow up for about 9 months.

He was brought to our clinic because of deterioration in his mental status within a few weeks on April 30, 1998. MR imaging had been performed in another university clinic because of headache and marked depression in December 1997. The T_1-weighted images showed a well-circumscribed hypointense area, and T_2-weighted images showed a hyperintense area, with homogeneous enhancement 2 cm in diameter in the left posterior parietal region (Fig. 2). The diagnosis was a brain abscess or a sequela of contusion. Neurological examination re-

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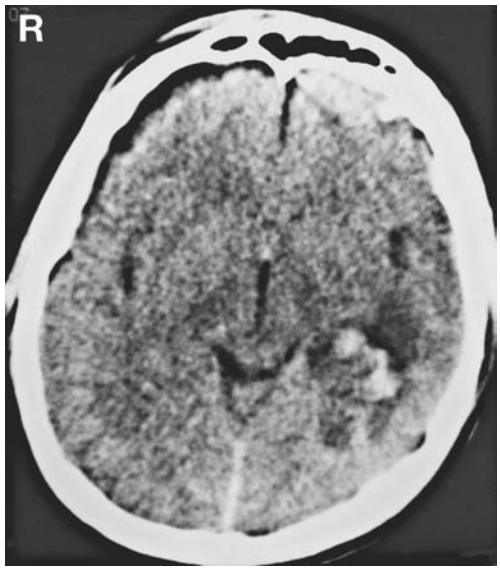

Fig. 1 Computed tomography scan taken soon after the trauma showing a heterogeneous hyperdense area in the left temporoparietal region indicating posttraumatic intracerebral hemorrhage.

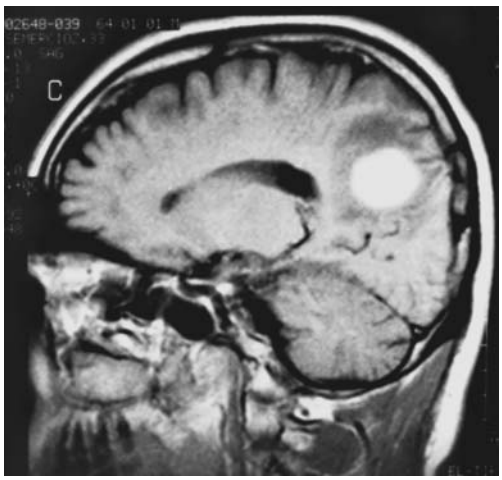

Fig. 2 Sagittal T_1-weighted magnetic resonance image with contrast medium performed 24 weeks after the trauma showing a well-circumscribed homogeneously enhanced lesion in the left posterior parietal region.

vealed nominal aphasia, alexia, acalculia, emotional and affective disturbances, right lower quadrantanopsia, right-sided astereognosis, and slight hemiparesis. We thought that the lesion shown by the MR images taken in December could be a space-occupying lesion. MR imaging taken on admission demonstrated a nonhomogeneously enhanced mass with a

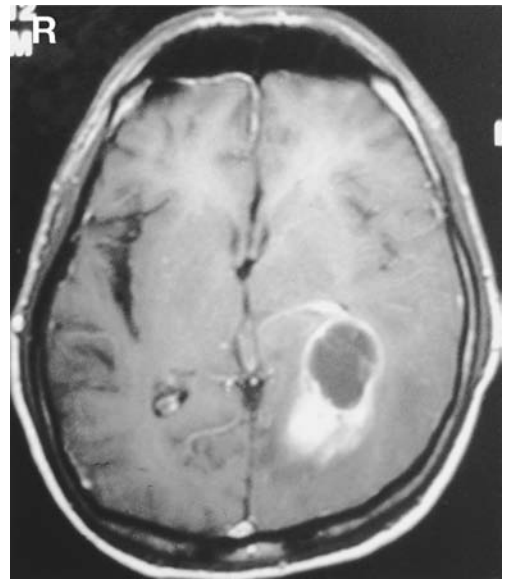

Fig. 3 Axial T_1-weighted magnetic resonance image with contrast medium showing a nonhomogeneously enhanced mass with a well-delineated cyst In the left parietal lobe.

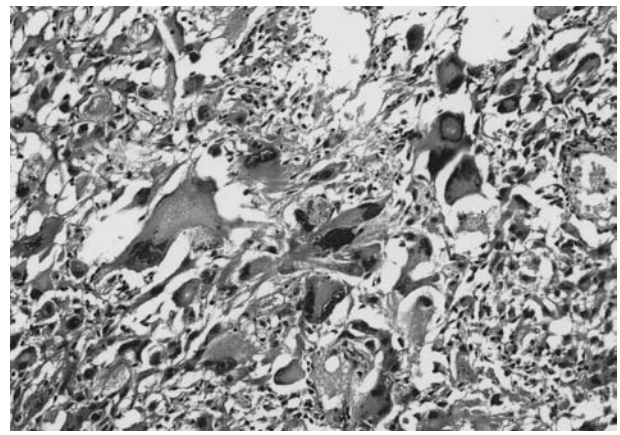

Fig. 4 Photomicrograph of the specimen. HE stain, ×400.

well-delineated cyst in the left parietal lobe (Fig. 3). He underwent surgical removal. The histological diagnosis was giant cell glioblastoma (Fig. 4). He did well postoperatively and was discharged to receive radiation therapy.

Discussion

Glioblastoma multiforme is the most common primary brain tumor in adults, and constitutes 50% to 55% of all gliomas and 25% of all intracranial

tumors.[5] Histological examination demonstrates marked hypercellularity of heterogeneous glial cells, nuclear and/or cytoplasmic pleomorphism, necrosis with pseudopalisading, vascular endothelial proliferation forming coiled masses of sinusoidal vascular channels, and high mitotic activity. Glioblastomas composed of bizarre, multinucleated giant cells with granular cytoplasm containing glial filaments are referred as giant cell glioblastomas. Cyst formation and a well-circumscribed border are commonly seen in this type of glioblastoma. The prognosis for this type may be slightly better than for other types. The interval between the onset of symptoms and the diagnosis is usually less than 6 months. Symptoms reflect either increased intracranial pressure or focal neurological dysfunction. Patients may rarely suffer sudden deterioration caused by large intratumoral bleeding.[5]

Massive hemorrhage into a brain tumor is uncommon, accounting for 6% to 10% of all intracerebral hematomas in clinical and radiological series, and only 1% in an autopsy study.[2] The great majority of underlying neoplasms are malignant and may be primary or metastatic.[1,2,4,7] Glioblastoma multiforme predominates among the primary brain tumors causing intracerebral hemorrhages, whereas metastatic tumors are mostly melanoma, choriocarcinoma, bronchogenic carcinoma, and renal cell carcinoma.[1,2,7] The hemorrhage is often limited to the peripheral zone of the tumoral tissue at the sites of penetrating nutrient vessels, so CT shows ring-like enhancement. Hemorrhages originating in glioblastoma are frequently located deep in the cerebral hemispheres, basal ganglia, or corpus callosum, whereas those from metastatic tumors usually occur at the corticomedullary junctions. Intratumoral hemorrhage is thought to originate from the vessels that traverse the necrotic areas or from the tumoral invasion of large cerebral vessels, which leads to thinning and rupture of the vessel wall, distortion, compression, and aneurysm formation, or from the abnormally dilated, ectatic, thin-walled, fistulous vessels at the proliferating margin.[2] The gliovascular relationship is different in tumor vessels. Tumor vessels are not well invested with glial meshwork, which may contribute to reduced resistance to shearing forces of the brain.

Mechanical forces causing movement of the hemispheres within the skull may result in traumatic intracerebral hemorrhage. Complex distortive motions of the cerebral hemispheres towards the prominences of the sphenoid or other basal bones can cause contusion of cortical surfaces. Torsion and shearing forces and especially imbalanced cortical versus white matter movements may result in diencephalic and other deep cerebral hemorrhages. Large single hematomas after minor traumas occur in patients with coagulopathies or in the elderly due to cerebrovascular amyloid angiopathies.[4] Traumatic intratumoral bleeding is a very rare event.[3,8] The present case is an extremely unusual case of glioblastoma causing intracerebral hemorrhage after head trauma.

The relationship between traumatic brain injury and the development of a brain tumor is well documented.[6,9] The proposed criteria include a sufficient time interval between the injury and the development of the tumor.[9] In our case, the short interval (5 months) suggests that the hemorrhage originated from a preexisting tumor.

Intracerebral hemorrhage after minor trauma may prompt a search for non-traumatic causes of cerebral hemorrhage. In our case, the major trauma causing a basal fracture was initially considered as the only reason for the entire clinical picture. The patient had no clinical history or findings compatible with tumor presentation, such as previous seizure or progressive neurological impairment, so the hemorrhage detected by the CT soon after the accident was thought to be a purely traumatic event. However, the unusual site of the bleeding provoked consideration of other possible causes, so further MR imaging was advised. Later, the laboratory investigations showed no evidence of coagulopathy.

We propose that traumatic intracerebral hemorrhage occurring in an uncommon site should indicate a search for underlying non-traumatic causative factors such as brain tumor, even in the absence of history and clinical findings suggestive of tumoral growth preceding the onset of hemorrhage.

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