Supratentorial Glioma Manifesting as Acute Onset of Pure Motor Hemiparesis

—Case Report—

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Abstract

A 68-year-old male presented with an anaplastic astrocytoma deep in the sensorimotor cortex manifesting as acute pure motor hemiparesis suggestive of a vascular mechanism rather than tumor mass effect. Perfusion-weighted magnetic resonance (MR) imaging showed a significant decrease of blood flow in the sensorimotor area, where fluid-attenuated inversion recovery imaging demonstrated a prominently edematous area. Angiography also suggested ischemia with poor visualization of the precentral and central arteries. Diffusion-weighted MR imaging failed to identify the edema as cytotoxic or vasogenic due to technical problems. Brain tumors may manifest through impairment of peritumoral blood supply, which can be clarified by recent MR methods.

Key words: apoplectoid, brain tumor, perfusion, diffusion, magnetic resonance imaging

Introduction

Brain tumors regardless of type or location may manifest as acute onset or deterioration of neurological symptoms.^{2,4,5,7-9,12)} The most obvious cases of such presentation are hemorrhage or epilepsy. Vascular mechanisms are also likely causes, although difficult to characterize precisely by routine radiological studies.⁴⁾ Brain tumors can mimic the symptoms of a vascular accident at onset, and such apoplectoid brain tumors often resulted in a misleading diagnosis.⁸⁾ The initial misdiagnosis of acute stroke syndromes was approximately 3% before the introduction of computed tomography (CT).¹²⁾ CT has shown that about 10% of patients with an initial diagnosis of cerebrovascular syndromes have brain tumor.¹²⁾ Perfusion- or diffusion-weighted magnetic resonance (MR) imaging can provide more useful information concerning the vascular pathophysiology of brain tumors.^{3,11)} We report a case of frontal glioma which mimicked pure motor hemiparesis due to lacunar infarction at onset.

Case Report

A 68-year-old male awoke with left hemiparesis and dysarthria on February 20, 1996. He had previously been free from neurological symptoms. He stayed at home without improvement of his symptoms, which became aggravated 2 days later in his upper extremities. Medical history was unremarkable except for mild hyperlipidemia. Neurological examination revealed left hemiparesis grade 4 out of 5 without sensory deficit. He was ambulatory but was hemiparetic. Deep tendon reflexes were accelerated in the left extremities, but no pathological reflexes were noted. Speech was markedly dysarthric. Blood pressure was 130/80 mmHg with regular pulse, and no neck bruit was audible.

Immediate CT because of the suspicion of pure motor hemiparesis from lacunar infarction unexpectedly revealed a right frontoparietal mass with marked perifocal edema (not shown). The tumor was strongly enhanced by contrast medium with a central necrotic area. There was no sign of intratumoral hemorrhage. The tumor was located at the subcortical area of the sensorimotor cortex without direct invasion into the internal capsule. Since the symptoms were suggestive of a vascular accident, diffusion- and perfusion-weighted MR as well as routine T₁-

Fig. 1 upper left: T_1 -weighted image showing a low intensity mass located in the white matter beneath the sensorimotor cortex. upper right: Gadolinium-enhanced T_1 -weighted image showing the tumor as homogeneous with strong enhancement except for a small portion within the tumor. Perifocal edema, which became strong toward the sensorimotor cortex, contrasted sharply with the tumor. lower left: T_2 -weighted image showing the tumor and the surrounding edema as a high intense lesion. lower right: Fluid-attenuated inversion recovery image showing the tumor central area as low intensity indicating the presence of highly mobile fluid in that area.

and T_2 -weighted and fluid-attenuated inversion recovery (FLAIR) images were obtained on the same day (Fig. 1). The T_2 -perfusion-weighted image indicated poor perfusion of the peritumoral area, particularly in the subcortical sensorimotor area rather than the tumor itself (Fig. 2). Cerebral angiography revealed poorly perfused precentral and central arteries, which supported the results of perfusion MR images (Fig. 3). Diffusion-weighted MR imaging could not differentiate cytotoxic from vasogenic edema because of technical problems (not shown), as the diffusion sensitivity factor used was not high enough (400 sec/mm²) and the apparent diffusion coefficient (ADC)⁹ could not be computed because of inadequate data acquisition.

Fig. 2 upper: T_2 -perfusion-weighted image with regions of interest indicated as circles. lower: Time-intensity curves of T_2 -perfusion-weighted images. The area B had less sensitivity to the influx of gadolinium-enhancement agent indicating reduced blood flow.

Fig. 3 Right carotid angiogram, lateral projection, showing irregular, bead-like stenosis of the precentral artery (arrows), and poorly visualized central artery.

The tumor was resected subtotally. Histological examination of the specimen found edematous tissue with less prominent cellularity, moderate cellular atypia, occasional micronecrosis, and vascular

hyperplasia without recognizable mitotic cells. The histological diagnosis was fibrillary astrocytoma with anaplastic component (grade II-III).

Discussion

Several pathological processes may result in acute manifestation of brain tumors. Nearly 50% of brain tumor patients who present with sudden neurological deterioration have hemorrhage.³⁾ Other potential etiologies for rapid deterioration are sudden increase in edema adjacent to the mass lesion, sudden depression of cortical function due to metabolic impairment, and interference with the blood supply by the tumor mass effect.

The acute pure motor hemiparesis occurring in our patient is a clinical syndrome usually related to lacunar infarction. Three previous patients presented with abrupt onset of pure motor hemiparesis as the initial manifestation of primary and metastatic brain tumors.⁴⁾ Early CT of these and our patient revealed nonhemorrhagic, right frontal, enhanced lesion. The mechanism by which such tumors presented as simulated stroke remains uncertain although vascular impairment was the most likely cause.

We tried to detect the vascular mechanism causing apoplectic onset in our patient using MR imaging, CT, and angiography. The gadolinium-enhanced T₁-weighted coronal images showed that the internal capsule was spared from direct involvement by the tumor, although the peritumoral edema had spread to the capsule. We thought it unlikely that the spread of peritumoral edema was the cause of the sudden manifestation of the clinical syndrome. T₂-weighted images showed marked swelling of the sensorimotor cortex and subcortical white matter. Tumor central and peritumoral edematous areas, as well as the cerebrospinal fluid in the ventricles, were suppressed in the FLAIR images, indicating the presence of highly mobile fluid in these areas. T₂-perfusion images revealed significant reduction of blood flow at the sensorimotor areas, suggesting ischemia of the functional area which could have been responsible for the hemiparesis. Angiography confirmed the reduced vascular supply to this area by showing an irregularly narrowed precentral artery and a poorly visualized central artery.

Diffusion-weighted MR imaging is the most sensitive technique for visualizing acute ischemic lesions as an area of elevated ADC within the first 4 hours of onset.¹³⁾ The ADC values of the imaging are significantly correlated with the time elapsed after the onset of cerebral infarction for the first 2 months.^{1,13)} High grade enhanced gliomas have a lower ADC than peritumoral edema,¹⁰⁾ so the sudden onset of

ischemia in the peritumoral area should result in a higher ADC indicating cytotoxic edema. We tried to confirm that the ischemia in the sensorimotor area was in the acute phase by demonstrating the presence of cytotoxic edema, but the diffusion sensitivity factor (b-value) was not high enough to differentiate cytotoxic from vasogenic edema. Diffusion-weighted MR imaging and other MR modalities such as perfusion imaging or MR spectroscopy may be useful in evaluating acute ischemic changes including similar cases of brain tumor with apoplectic onset.

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