

Case Reports

Brain Tumor Presenting as an Acute Pure Motor Hemiparesis

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Acute pure motor hemiparesis is a clinical syndrome of isolated hemiparesis usually related to lacunar infarction, although other etiologies have been described. We recently encountered three patients with the abrupt onset of pure motor hemiparesis as the initial manifestation of primary or metastatic brain tumor. In each patient, early computed tomography demonstrated a nonhemorrhagic, right frontal, enhancing mass lesion. While the mechanism whereby brain tumor may present abruptly and simulate a stroke remains uncertain, these cases illustrate that pure motor hemiparesis can be the initial symptom of intracranial tumor. Early computed tomography or magnetic resonance imaging is suggested for all patients who present acutely with pure motor hemiparesis. (*Stroke* 1989;20:288–291)

The acute development of pure motor hemiparesis (PMH) typically represents a clinical stroke syndrome of hemiparesis without associated sensory deficit, homonymous hemianopia, aphasia, or other higher cortical function deficits.¹ PMH is usually caused by lacunar infarction in the posterior limb of the contralateral internal capsule or basis pontis, although small intracerebral hemorrhages in these anatomic locations can also manifest similar clinical symptoms.² PMH has also been described in association with ischemic cortical infarction, subdural hematoma, nocardial brain abscess, and demyelinating disease.^{3,4} Brain tumors, either primary or metastatic, occasionally present with the abrupt development of focal neurologic deficits mimicking stroke.⁵ PMH has rarely been associated with metastatic brain tumor, but the clinical details have not been described.^{3,6} We recently encountered three patients who presented with acute PMH symptoms as the initial manifestation of a primary or metastatic brain tumor.

Case Reports

Patient 1. A 77-year-old woman awoke with left-sided weakness. There were no visual or sensory symptoms. For the month prior to admission, she had felt generally fatigued and had sustained several falling episodes associated with knee buckling. Prolonged electrocardiographic monitoring as an outpatient had demonstrated supraventricular

tachycardia and sinus pauses of 1–3 seconds. Her medical history was otherwise unremarkable. The general physical examination on admission was remarkable only for the presence of a soft systolic ejection murmur. The neurologic examination demonstrated a moderate (Medical Research Council [MRC] Grade 3⁷) left hemiparesis that involved her face, arm, and leg equally. The left plantar response was extensor. Extensive examination of higher cortical function, language, sensation including pain, temperature, joint position, vibration, graphesthesia, and stereognosis, cerebellar function, and deep tendon reflexes revealed no abnormalities. A head computed tomogram (CT scan) with contrast on the day after admission revealed a large enhancing mass in the right frontal-parietal lesion with associated edema and shift of the midline structures (Figure 1). A cerebral angiogram was consistent with a large hypovascular mass in the right frontal-parietal region with minimal extracranial atherosclerotic vascular disease. One week after admission, a craniotomy was performed and the biopsy specimen demonstrated the mass lesion to be a malignant glioma. The patient received radiation therapy postoperatively, but there was little change in her left hemiparesis.

Patient 2. A 63-year-old woman presented with the abrupt onset of left facial droop, left hand clumsiness, and weakness. Walking was unsteady because of slight left leg weakness. Her medical history was unremarkable. General physical examination on admission was unremarkable aside from a firm nodular right breast mass, which at biopsy was found to consist of fibrous fatty tissue. Neurologic examination demonstrated higher cortical functions to be intact. The cranial nerves were unre-

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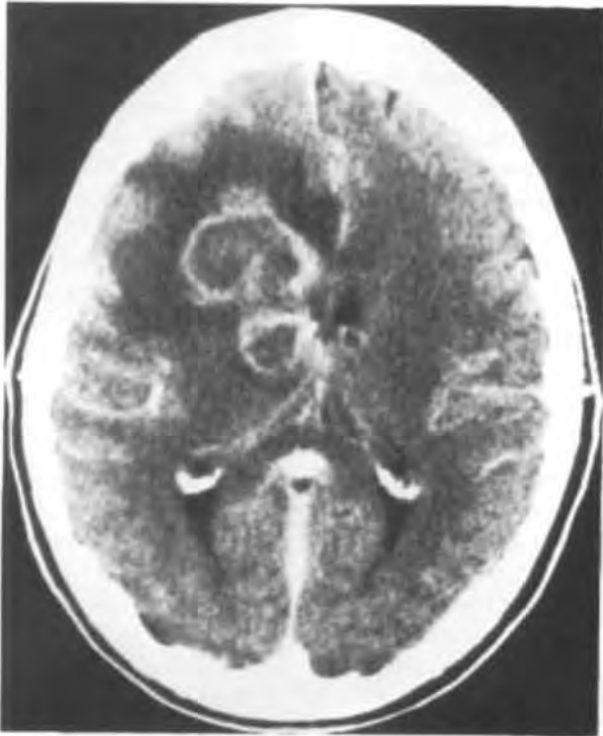


FIGURE 1. Cranial computed tomogram of Patient 1 demonstrating large enhancing mass in right frontal-parietal region with associated edema and mild shift of midline structures.

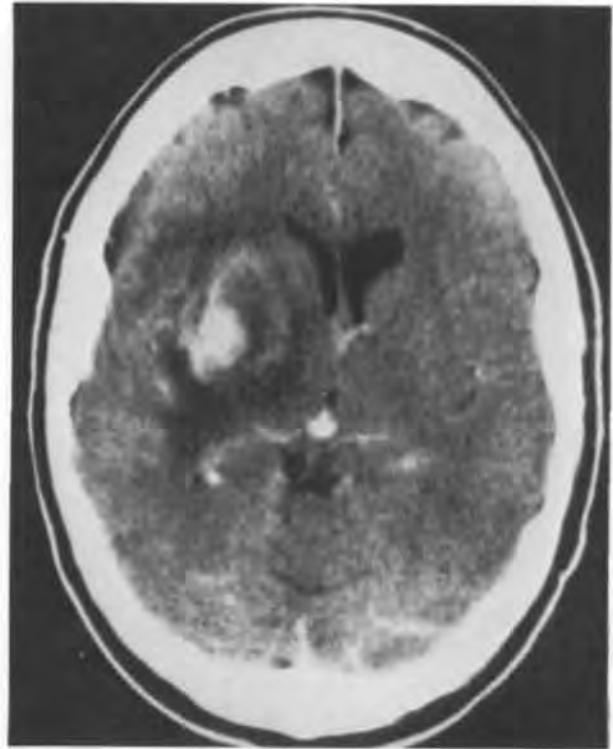


FIGURE 2. Computed tomogram of Patient 2 demonstrating large enhancing mass in right frontal lobe with associated edema.

markable aside from a severe left central facial weakness. A left pronator drift was noted, but left arm and leg strength was otherwise intact. Rapid alternating movements were diminished in the hand. There was diminished pain sensation confined to only the left hand. Sensory examination was otherwise intact to temperature, joint position, vibration, graphesthesia, and stereognosis. The deep tendon reflexes were slightly more active in the left upper extremity than the right but were symmetrical in the lower extremities. Both plantar responses were flexor. CT performed the day after admission demonstrated a large enhancing mass lesion in the right frontal lobe with associated edema (Figure 2). A large avascular mass lesion in the right frontal lobe was noted on cerebral angiography without evidence of extracranial or intracranial vascular abnormalities. The lesion responded to dexamethasone but relapsed when the dosage was tapered. A diagnosis of probable central nervous system lymphoma was made.

Patient 3. A 59-year-old woman awoke with left-sided weakness. She denied any other neurologic symptoms and had been asymptomatic the night before. Her medical history was entirely unremarkable. The general physical examination on admission was unremarkable except for the presence of a pelvic mass. Neurologic examination revealed a mild (MRC Grade 4) left hemiparesis with facial sparing and left-sided hyperreflexia. The

left plantar response was extensor and the right was flexor. Extensive examination of higher cortical function, cranial nerves, sensation including pain, temperature, joint position, vibration, graphesthesia, and stereognosis as well as cerebellar function was otherwise unremarkable. CT with contrast on the day of admission demonstrated a large enhancing mass lesion with associated edema in the right frontal lobe (Figure 3). Carotid noninvasive studies were performed and were unremarkable. A pelvic CT scan revealed the presence of a large mass, which was biopsied; a diagnosis of small cell undifferentiated carcinoma was made. Three days after admission, the left hemiparesis abruptly worsened. A repeat head CT scan demonstrated a hemorrhage into the previously observed mass lesion. Patient 3 was treated with dexamethasone and radiation therapy, but she did not improve clinically.

Discussion

These three patients acutely developed PMH, and in each case a brain tumor in an appropriate anatomic location was diagnosed by early CT scanning. PMH associated with lacunar infarction usually involves the face, arm, and leg equally, but considerable variation has been described.⁸ In Patient 2 the motor deficits were primarily facial, and in Patient 3 facial sparing was present. Both variants have been observed in lacunar PMH cases.^{9,10} In Patient 2 a mild sensory disturbance was also noted, and this has also been commonly

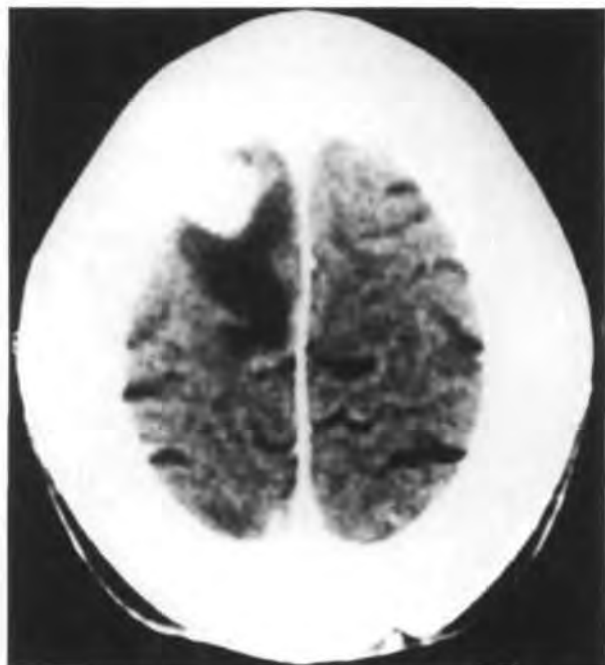


FIGURE 3. Computed tomogram of Patient 3 demonstrating area of contrast enhancement in right frontal lobe with associated edema consistent with presence of tumor.

observed in lacunar PMH cases.¹¹ No patient had underlying risk factors for lacunar infarction such as hypertension or diabetes mellitus, but these disorders are not present in all PMH lacunar infarction cases.¹² No patient had a recognizable proximal carotid arterial lesion, which might have served as a source of intracranial emboli, another possible etiology of lacunar PMH.¹³ In Patient 1, sick sinus syndrome may have served as a potential nidus for cardiogenic brain embolus, but this is an unusual cause of PMH.⁸

A number of previous reports from the pre-CT era have mentioned the occurrence of brain tumors misdiagnosed as cerebrovascular disease.¹⁴⁻¹⁸ McLaurin and Helmer,¹⁵ for example, noted that almost 2% of their autopsy-proven brain tumor cases had been misdiagnosed as cerebrovascular disease during life and that this error was most likely to occur in elderly patients. Since the advent of CT, it has become possible to readily diagnose these patients, and such errors should not occur if early CT scanning is performed in stroke patients.⁵

How a relatively slow-growing lesion such as brain tumor produces sudden neurologic deterioration remains unclear. There are a number of potential mechanisms whereby brain tumors may produce the sudden onset of focal neurologic deficits in the absence of seizure. Sudden hemorrhage into a tumor is one such mechanism that is often cited, but it is relatively uncommon.^{17,19,20} A recent study noted that <50% of brain tumor patients who present with sudden neurologic deterioration prove to have a hemorrhage.²⁰ This observation is supported

by our three patients, none of whom initially had a macroscopic hemorrhage (although Patient 3 developed one 3 days after initial presentation). Other mechanisms must, therefore, be invoked. Daly et al,²¹ commenting on the sudden onset of symptoms in patients with meningiomas, cited three additional potential etiologies for sudden deterioration: sudden increased edema adjacent to the mass lesion, a spreading depression of cortical function secondary to mechanical irritation, and lastly, interference with blood supply by direct vascular compression; the latter mechanism was thought to be most likely. It is interesting that over a half-century ago, Cushing (cited by Oldberg¹⁷) also hypothesized a vascular cause for the sudden deterioration in brain tumor patients without apparent hemorrhage. Others have also favored this vascular hypothesis, although convincing proof is lacking.¹⁵ To explain how brain tumor patients acutely develop PMH symptoms, one might speculate that a tumor-associated thrombosis or embolism occurs involving one of the deep penetrating arteries, simulating lacunar PMH. CT scans did not demonstrate a small-vessel territory infarct, but the extensive mass lesions and edema in each case could have obscured such a subtle abnormality.

All three of our patients had nondominant hemisphere, predominantly frontal lobe tumors. This location in a relatively silent brain area presumably accounts for the paucity of neurologic signs before the acute symptoms developed. By contrast, in dominant-hemisphere tumors motor symptoms would probably be associated with, or preceded by, language disturbance, a frequent early manifestation of dominant-hemisphere brain tumor in elderly patients.²²

Whatever the mechanism, these cases emphasize that acute PMH has a variety of potential etiologies, although lacunar infarction related to small-vessel intracranial vascular abnormalities is certainly the most common cause.⁸ The clinical features observed with brain tumor-associated PMH cases are not distinctive, and patients may not manifest previous symptoms to suggest malignancy. Therefore, early CT or magnetic resonance imaging is suggested in patients with acute PMH to exclude the presence of a brain tumor.²³

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