Pure motor hemiplegia secondary to brain-stem tumour

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SYNOPSIS 'Pure motor hemiplegia' is a common stroke syndrome defined by Fisher as paralysis of face, arm, and leg on one side, unaccompanied by sensory signs, visual field defect, aphasia, or apractognosia. It occurs almost exclusively in hypertensive patients and carries a good prognosis. We report a case of a normotensive patient in whom pure motor hemiplegia was the presenting feature, not of a cerebrovascular syndrome, but of a pontine glioblastoma. We note that brain-stem tumours may masquerade as brain-stem strokes.

'Pure motor hemiplegia' is a common stroke syndrome defined by Fisher and Curry (1965) as paralysis of face, arm, and leg on one side, unaccompanied by sensory signs, visual field defect, aphasia, or apractognosia. It occurs almost exclusively in hypertensive patients, and carries a good prognosis. It is usually secondary to an infarct in the pons or internal capsule of the brain.

CASE REPORT

R.M., a 60 year old married woman with no preceding history of hypertension, was admitted to Sacred Heart Hospital because of the sudden onset, over a one-hour period, of weakness of her left arm and left leg. During the preceding month she had complained of sore throat and fever, and was treated with oral tetracycline for pharyngitis by her local physician. Two weeks before admission, she had shaking chills, myalgias, and headaches. The antibiotic was changed from tetracycline to cephalosporin, but the chills, fever, and headache persisted.

On admission, blood pressure was 160/80 mmHg; pulse 90 per minute; respirations 22 per minute, and temperature 38.3°C. General physical examination was normal except for a basal systolic ejection murmur and pain on neck flexion. Neurological examination showed a normal mental status without denial or impersistence. Language function, memory, judgement, and calculation ability were intact.

Visual fields were full. There was left facial weakness, left hemiparesis, left hyperreflexia, and a left Babinski sign. Sensory examination was normal to pin, touch, position, and stereognosis.

Complete blood count, urinalysis, and electrolytes were normal except for 10,800 leucocytes/mm³. Lumbar puncture showed opening pressure of 300 mm water, closing pressure of 180 mm with 1,952 leucocytes/mm³ (58% polymorphonuclear cells, 42% mononuclear cells), protein 0.74 g/l, and glucose 4.11 mmol/l. Gram stain and India ink preparations were negative; bacterial, fungal, and tuberculosis cultures showed no growth. EEG, skull radiograph, and brain isotope scan were normal.

The patient was treated with intravenous ampicillin, and what had been assumed to be a meningitis cleared. Repeat lumbar puncture one week later showed only 9 lymphocytes/mm³; protein and sugar were normal. She was discharged and remained stable for the next two months, when she had a sudden onset of diplopia and was found to have a left sixth nerve palsy. Over the next two months she developed progressive ataxia of gait, dysarthria, and further weakness of her left limbs. Vertical nystagmus appeared and a diminished gag reflex and dysphagia. A four-vessel cerebral angiogram was performed and was normal. She continued to deteriorate and died six months after her initial admission.

NECROPSY Findings at necropsy included a large pulmonary embolus which was considered to be the immediate cause of death. The basis pontis, surrounding structures, and meninges were replaced and infiltrated by a diffuse, focally necrotic mass

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FIG. 1 *Glioblastoma multiforme of the basis pontis with area of old necrosis on the right side (arrow). The basilar artery (B) is surrounded but not occluded by tumour.*

FIG. 2 *High power view of section taken from the right basis pontis showing tumour cells (above) and necrosis and fibrosis within the glioblastoma (below). Haematoxylin and eosin, $\times 130$.*

which compressed the fourth ventricle and encased the basilar artery (Fig. 1).

Microscopic sections showed pseudo-palisading, hypercellularity, pleomorphism of giant cells, and endothelial proliferation which was diagnostic of glioblastoma multiforme (Fig. 2). Regions of necrosis and fibrosis were most prominent in the right basis pontis, within the tumour mass (Figs 1 and 2). The basilar artery, though surrounded by tumour, was not occluded. In the medulla, the right pyramidal tract showed a loss of myelin and also some infiltration of tumour. Examination of the cerebral and cerebellar hemispheres and remaining brain-stem showed no infarct or tumour. The meninges over the cerebral hemispheres were thin without inflammation. Slight atherosclerosis was seen.

DISCUSSION

Fisher and Curry (1965) noted that pure motor hemiplegia, involving face, arm, and leg, is the result of contralateral infarction of either the posterior limb of the internal capsule or the basis pontis. In both places, the motor fibres to these body parts are closely grouped together and amenable to interruption without other clinical signs. Fisher and Curry (1965) indicated that thrombosis is probably the responsible vascular process. The present case illustrates that a patient with glioblastoma of the pons with necrosis predominantly on one side can present as a 'pure motor hemiplegia' without a history of hypertension. It seems reasonable to assume in our case that the necrosis in the pons was secondary to the glioblastoma, especially since neither infarcts nor lacunes were found in the cerebral or cerebellar hemispheres.

The similarity of our patient's presentation to that of the vascular pure motor syndrome led to an error in initial diagnosis. It was not until the progression of cranial nerve signs that it was realized that the process was neoplastic. Confusion of brain tumours with cerebrovascular disease has been described and cautioned against in the past. Elsberg and Globus (1929) reported 37 cases of 'acute brain tumor' in which patients experienced a sudden neurological deficit, usually a hemiparesis or aphasia. Eighty per cent of their patients had glioblastoma multiforme at necropsy. Moersch *et al.* (1941), noted that one-third of their patients with necropsy proved glioblastoma were diagnosed clinically as strokes.

McLaurin and Helmer (1962) reported 22 cases of brain tumours misdiagnosed during life, including two primary brain-stem gliomas clinically diagnosed as basilar artery occlusions. The authors described several cases in which cerebral thromboses were incorrectly diagnosed and the patients were given anticoagulants in the absence of arteriography with resultant fatal complications.

In an angiographic series reported by Silverstein (1965), the clinical histories of 280 patients with presumed occlusive cerebrovascular disease were carefully analysed before angiography and any cases felt to have signs of subacutely progressive or non-vascular disease were removed from the series. Nonetheless, 5% of the patients were discovered at angiography to have mass lesions as the explanation of their symptoms. Progression of signs after 'stroke' is usually the clue to the mistaken diagnosis. Failure of the process to respect vascular territory may be another clue to the presence of brain tumour rather than stroke.

Intramedullary tumours of the brain-stem are most commonly encountered in childhood but are well known in adults as noted by Barnett and Hyland (1952) and White (1963). Involvement of the pons with glioblastoma multiforme in adults, though rare, has been previously reported by Masucci *et al.* (1966). The occurrence of a brain-stem neoplasm simulating one of the lacunar stroke syndromes has not been, to our knowledge, reported previously. The importance of this potential diagnostic error is heightened by the good prognosis usually given for patients with lacunar strokes, and the tendency not to perform angiography in these cases. The development of computerized transaxial tomography may provide a safe, non-invasive method to assist in correctly diagnosing similar cases. With this case, we note that 'pure motor' hemiplegia need not be a 'pure' vascular syndrome and that brain-stem tumours may masquerade as brain-stem strokes.

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