LETTERS TO THE EDITOR

Ipsilateral hemi-Parkinsonism secondary to an astrocytoma

Parkinsonism has rarely been described in association with intracranial tumours. In a previous review by Polyzoidis et al in 1985, 49 verified cases with intracranial tumours were collected from the literature, the majority of which were meningiomas.1-3 Further review of the current literature adds another four cases.4-7 Only six of these 55 cases directly involved the thalamus and or the basal ganglia.8 All six were infiltrating intra-axial gliomas.9 When symptoms of Parkinsonism occur in a patient with intracranial tumour, it is not uncommon to make an incorrect diagnosis of Parkinson's disease.10

We report an additional case of an intra-axial glioma located in the left thalamic region with ipsilateral hemi-Parkinsonian features, but without any clinical signs of increased intracranial pressure.

A 62 year old man presented with a one year history of increasing tremor of the left limbs, impairment of memory, slowness in his actions and unusual fatigue. His complaints failed to respond to anti-Parkinsonian drug therapy. There was no other previous medical history of note.

At the time of admission on 28 August 1989, physical and neurological examination revealed facial impalpability, a marked resting tremor involving the left limbs and head in combination with mild cog-wheel rigidity. There was no sign of pyramidal involvement and no sensory deficits. Plantar responses were flexor. There was no other evidence of an intracranial involvement except for a right homonymous hemianopia. There was no papilloedema. His attention span was brief. He had great difficulty buttoning clothes and his walking was slow with a tendency to fall frequently.

A cranial enhanced CT scan showed an extensive area of mixed attenuation in the left thalamic region with some mass effect (fig 1) which turned out to be an astrocytoma Grade II after the biopsy on 20 September 1989 was taken. He was then transferred to receive a course of radiation therapy. At the time of transfer the clinical picture was unchanged.

Structural lesions of the basal ganglia are well recognised and sometimes also produce hemidystonic syndromes.8 Most of these cases have been due to haemorrhages, infarction or tumours. Tumours may produce Parkinsonism either by pressure on the basal ganglia11-12 or more rarely by direct infiltration13 as in our case. Extra-axial masses impinging on the basal ganglia appeared to be more common than infiltrating lesions.4 Several authors have shown the reversibility of clinical findings after resection of the tumour which caused compression and displacement of the basal ganglia structures rather than infiltration or destruction.14 Direct destruction of the basal ganglia may result in a clinical response to anti-Parkinsonian drugs in our patient and also in other patients described in the literature. Garcia de Yebenes J, et al reported a significant decrease of caudate postsynaptic dopamine receptors which was accounted for their patient’s Parkinsonism secondary to a craniopharyngioma.2

Intracranial tumour can mimic many neurological conditions including Parkinsonism. When Parkinsonian signs have been present in the intracranial tumour patient, resting tremor and rigidity were reported to be mostly contralateral11 in contrast with our patient who presented with an ipsilateral hemi-Parkinsonism. Oliver3 and Polyzoidis et al4 reported two other patients who presented with an ipsilateral resting tremor and rigidity due to a parieto-occipital and a frontoparietal glioma respectively. They both argued that the signs in these cases were not due to direct pressure on or invasion of the basal ganglia but were due to midbrain compression from upward or downward herniations displacing the midbrain against the tentorial edge. We prefer this explanation because in our case the Parkinsonism was ipsilateral to the lesion.

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7 Pall HS, 74 year old lady who developed bilateral parkinsonism secondary to an intracerebral tumour. J Neurol Neurosurg Psychiatry 1987;50:1360.

Spontaneous intraneural haematoma of the optic nerve

Acute unilateral optic neuropathy is most commonly caused by retrobulbar neuritis. We describe a patient with visual loss caused by a spontaneous intraneural haematomata of the optic nerve. This has not previously been described.

A healthy 37 year old Greek housewife presented with a three week history of decreased visual acuity in the right eye. She awoke with poor vision in association with a generalised headache lasting 24 hours. A diagnosis of probable retrobulbar neuritis was made by the referring hospital and she was given oral prednisolone for five days. Her vision did not improve. Vision in the left eye was unaffected. There was no previous visual or neurological history of note.

On examination she could read J16 with the right eye and J1 with the left. There was a central scotoma with colour desaturation. Fundoscopy showed right optic atrophy. General examination was unremarkable and she was normotensive. A CT brain scan showed a minimally enhancing lesion in the region of the right optic nerve which could have been either a meningioma or an optic nerve glioma (fig). Angiography was normal. In view of the possibility of vision in the left eye becoming affected, surgery was recommended.

At craniotomy the right optic nerve was found to be haemorrhagic and measured 3 cm in diameter compared with the normal left nerve measuring 0.5 cm. Careful dissection revealed no evidence of a vascular abnormality. An incision was made in the swollen nerve and the haematoma evacuated. Again no evidence of abnormal vessels was identified. Histology showed blood clot, haemosiderin, fibrin, and disrupted optic.