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. Further review of the current literature adds another four cases. If symptoms of Parkinsonism occur in a patient with intracranial tumour, it is not uncommon to make an incorrect diagnosis of Parkinson's disease. We report an additional case of an intracranial glioma located in the left thalamic area 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 head and 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 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) 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 pyramidal syndromes. Most of these cases have been due to haemorrhages, infarction or tumours. Tumours may produce Parkinsonism either by pressure on the basal ganglia or more rarely by direct infiltration as in our case. Extra-axial masses impinging on the basal ganglia appeared to be more common than infiltrating lesions. 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. Direct destruction of the basal ganglia may even then present a clinical response to anti-Parkinsonian drugs in our patient and also in other patients described in the literature. Garcia de Yébenes J, et al reported a significant decrease of caudate postsynaptic dopamine receptors which accounted for their patient's Parkinsonism secondary to a craniopharyngioma.

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 contralateral in contrast with our patient who presented with an ipsilateral hemi-Parkinsonism. Oliver and Polyzoidis et al 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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