

Case report

Total recovery from Parkinson syndrome after surgical removal of a meningioma: A clinical case

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Signs of parkinsonism, such as resting tremors, rigidity, bradykinesia, and gait disturbance, typically have a unilateral onset and result from a malfunction in the extrapyramidal system involving the basal ganglia. Parkinson's disease is mostly idiopathic and is caused by progressive and irreversible loss of dopaminergic neurons in the substantia nigra. However, in some cases, parkinsonism may be secondary to various conditions such as the following:

- dopamine-antagonist action of neuroleptic drugs;
- pathologies of cerebral vessels;
- post-encephalitis damage;
- exposure to toxic substances, such as CO, manganese, etc.;
- idiopathic normopressure hydrocephalus;
- neurometabolic disorders;
- post-traumatic damage (as occurs in boxers); and
- intracranial tumours.

The latter case is quite rare, occurring in only 0.3% of supratentorial tumour cases [1], and appears to result from the infiltration/compression of the basal ganglia. Most studies in the literature describe secondary parkinsonism with unilateral onset,

often contralateral to the cerebral lesion. Here, we describe a patient who developed a pure bilateral parkinsonism that completely regressed after neurosurgical removal of the patient's tumour.

2. Case report

A 57-year-old woman had previously undergone surgery to resect a colorectal tumour. Ten years before her current admission, the patient developed behavioural problems consisting of disinhibition and inappropriate conduct. For this reason, she was followed in a psychiatric centre and treated with mood stabilisers (Depakin CR 600 mg/day) for a mild increase in impulsive behaviours, but without apparent benefit. To our knowledge, she was not administered any neuroleptic or antiparkinsonian drugs. Approximately 3 months before the patient's current admission, her symptomatology was complicated by the appearance of anosmia and a marked bilateral and symmetrical resting tremor localised to the jaw and all four limbs. In addition, the tremor was accompanied by bradykinesia and difficulty walking. After an accidental fall, the patient went to the emergency department, where a brain computed tomography scan was performed. Neuroimaging showed a large lesion occupying both of the frontal lobes and a midline shift to the left. She was then sent to our department, where brain magnetic resonance imaging (MRI) with mdc and intracranial angio-MRI was performed under sedation. These tests confirmed the presence of a massive median frontal meningioma, likely originating from the ethmoidal planum and olfactory groove, with a maximum diameter of 7.5 cm. The tumour

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was responsible for the compressive phenomena on the adjacent parenchyma (Fig. 1). The lesion compressed and displaced the corpus callosum posteriorly and the head of the caudate nucleus bilaterally. The tumour volume occupied the anterior cranial fossa up to the sylvian fissure bilaterally, and displaced the anterior and middle cerebral arteries. A neurological examination confirmed the presence of a resting tremor with a frequency of 4–6 Hz. The tremor increased in intensity as a result of emotional stimuli, and decreased during the execution of voluntary movements. On walking, the patient's arm swing was decreased. Mild bradykinesia was observed when the patient touched her finger tips, and when she closed and opened her hands. Hypomimia and marked rigidity in all four limbs, with cogwheel rigidity at the elbows, were observed. Deficits in strength and sensitivity were not found. The cranial nerves were free. The patient's plantar responses were flexor.

The pre-neurosurgery UPDRS value (Unified Parkinson's Disease Rating Scale, Movement Disorder Society Task Force on Rating Scales for Parkinson's Disease [2]) was 71. The blood test, thyroid function, and ECG results were without abnormalities. The tumour was removed through a bilateral subfrontal approach, with release of the anterior cerebral arteries and their branches, which were markedly stretched (Fig. 1). The histological examination confirmed the presence of a meningotheial meningioma (World Health Organization grade I). A control MRI showed total resection of the lesion and re-expansion of the frontal lobes (Fig. 2). The clinical condition of the patient after neurosurgery rapidly improved. In particular, there was a marked reduction in the patient's tremor in all four limbs on the first post-operative day, and a nearly complete resolution of the parkinsonism' symptoms 30 days later (Fig. 3). Administration of valproate was uninterrupted as antiepileptic coverage. At the 18-month follow-up visit, the patient was still in good health and

had no neurological symptoms. She was walking independently and had not experienced a recurrence of the tumour. Her UPDRS was 3. Her behavioural issues were also reduced, but her anosmia remained unchanged.

3. Discussion

The first case of secondary parkinsonism was described by Blocq and Marinesco in 1893. During an autopsy, they observed the presence of a tuberculoma of the cerebral peduncle involving the substantia nigra (for historical reference, see Blocq R, Marinesco G. *Sur un cas de tremblement Parkinsonien hémiplégique, symptomatique d'une tumeur de pédoncule cerebral. C.R. Soc. Biol. Paris 5:105–111, 1893*). Since then, other cases have been described, and they are usually characterised by the onset of unilateral tremors, bradykinesia, and difficulty walking as the consequence of the presence of a brain tumour. These symptoms occur in 0.3% of patients with supratentorial brain tumours [1] and could result from the following:

- mechanical compression of the basal ganglia exerted by a tumour or by the development of hydrocephalus;
- torsion or compression of the midbrain and tentorial herniation which causes the symptoms indirectly;
- neoplastic infiltration of the basal ganglia;
- tumour-induced vascular and metabolic abnormalities.

Various clinical reports have shown that, in these cases, removal of the tumour may induce an almost complete regression of the patient's symptoms. This finding is very important because it builds a causal link between brain neoplasms and parkinsonism, and excludes the possibility of an idiopathic Parkinson pathology

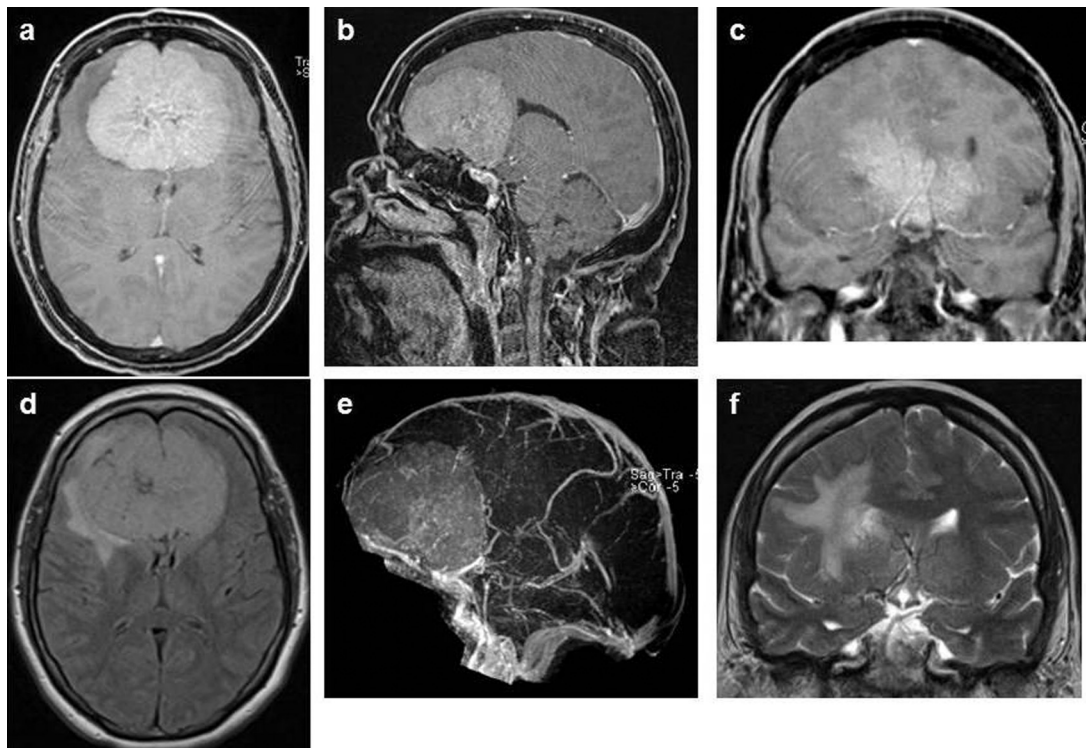


Fig. 1. Presurgical MRI. (a–c) Representing contrast enhanced T1 images on axial, sagittal and coronal planes. (d) The T2 flair axial plain, (e) the MRI angiography reconstruction and (f) a coronal T2 section. It appears noticeable the intimate relationship of the lesion with the head of caudate nuclei bilaterally, as visible on axial post contrast T1 and particularly on T2 Flair images. The coronal sections are particularly useful to show the spatial relationship between the lesion and the basal ganglia (c) and the pushing effect on the aforementioned structures (f). The sagittal plane (b) enables us to show the distortion of brain tissue just adjacent to the thalamus. (e) Summarises the entire lesion and its complex vascular relationships.

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