Radiation-induced parkinsonism in a patient with glioma

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Objectives: Here we report the case of a patient developing partially Levodopa-responsive parkinsonism about 9 months after radiotherapy for an anaplastic oligodendroglioma.

Case report: A 61 year-old man underwent a subtotal surgical removal for a right fronto-mesial tumor in 2009; histological diagnosis was oligodendroglioma gr II WHO and chemotherapy with temozolomide and procarbazine-lomustine was administered. At the time of progression (2013) a second surgery was performed, followed by conformal radiotherapy (59,4 Gy in 30 fractions) as the tumor was a gr III. Neurological examination showed moderate hemiparesis in the left limbs with autonomy in gait and in most of the daily living activities.

About 6 months after radiotherapy the patient developed depression, apathy and retropulsion tendency with several falls. Successively, a sub-acute development of left limbs rigidity appeared, leading to a relevant worsening of gait and impairment in the use of the left arm, with severe loss of autonomy. Neurological examination of April 2015 showed camptocormia, hypomimia, wheelcharing for retropulsion and freezing of gait, incoercible plastic-spastic rigidity of the left limbs, with assumption of plastic postures, and severe bradykinesia. Right limbs were normal. Brain MRI (Fig. 1) showed a widespread alteration of white-matter around the surgical-lesion site, involving the fronto-parietal-temporal white-matter of the right hemisphere, as result of post radiation damage. No signs of lesions in the basal ganglia were revealed. DaT-Scan scintigraphy was normal and the neuropsychological assessment showed exclusively impairment in the attention domain. Low levodopa doses (300 mg/daily) lead to moderate improvement in the rigidity of the left limbs, mild improvement in bradykinesia and no effectiveness on gait.

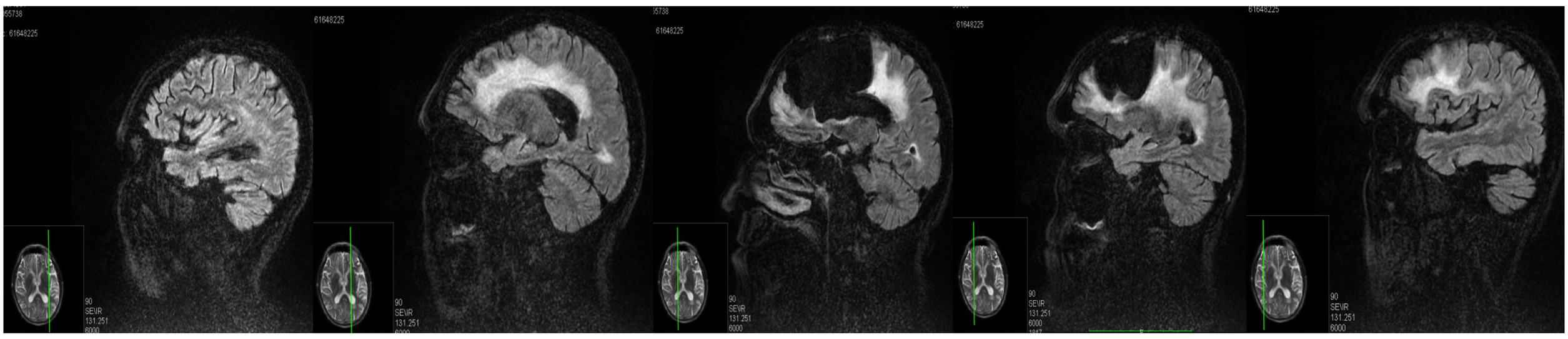

Figure 1: brain MRI performed in 2015, 1 year after radiotherapy

Discussion: Parkinsonism is related to functional abnormalities in the motor cortico-striato-pallido-thalamo-cortical loop and related neural pathways. Secondary parkinsonism is usually associated with lesions, cerebrovascular damage and/or metabolic causes, while radiation-induced parkinsonism does not represent a well-established entity.

Conclusions: This case demonstrates that white-matter diffuse damage related to radiotherapy may be cause of parkinsonism with partial response to levodopa, even without evidence of a direct involvement of basal ganglia.