

Diffuse astrocytoma presenting with parkinsonism and gliomatosis-like infiltration

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In 2007, a 51-year-old woman presented with progressive left-sided bradykinesia, rigidity, micrographia and gait disturbance without a resting tremor. An initial brain magnetic resonance imaging (MRI) performed within weeks of symptom onset was unremarkable. The initial diagnosis of Parkinson's disease was made by a clinical neurologist, and dopaminergic therapy provided partial benefit. Although a causal link between the tumour and the parkinsonian syndrome is possible, the coexistence of two unrelated pathologies—such as an atypical parkinsonian disorder preceding tumour development—cannot be excluded. Over the ensuing 6 years, the patient's parkinsonian symptoms persisted until a generalised tonic-clonic seizure in 2013 prompted repeat imaging and diagnosis of a diffuse infiltrative glioma (Figure 1).

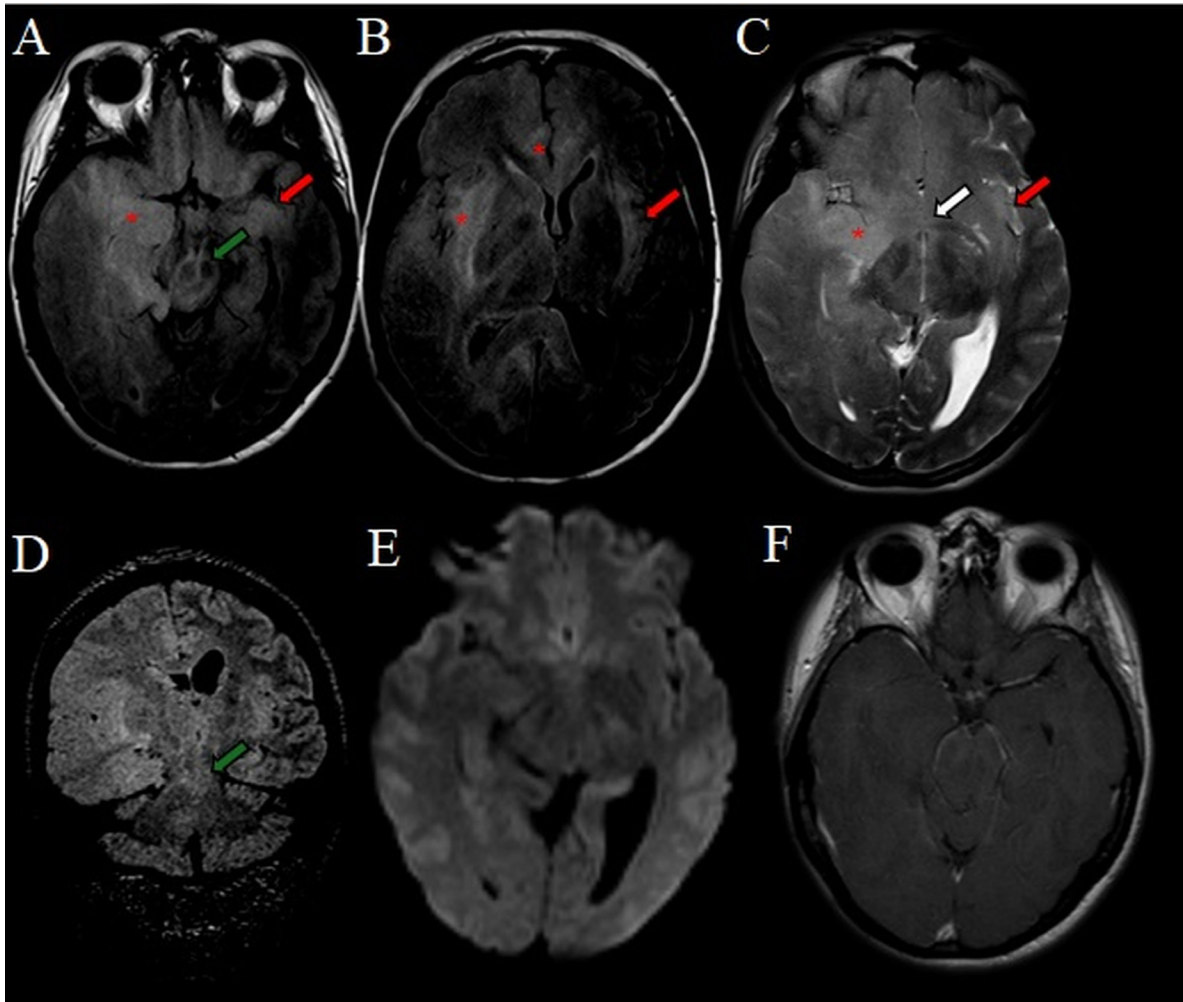
Gliomatosis cerebri—no longer a distinct entity in the World Health Organization (WHO) classification—describes diffuse infiltration of three or more cerebral lobes, typically with preserved architecture but extensive white matter spread.¹ Early imaging may be normal, delaying diagnosis,

as in this case.² Advanced imaging and MR spectroscopy can aid recognition when parkinsonian symptoms are atypical or refractory to dopaminergic therapy.³

Treatment is often palliative, as complete resection is rarely feasible. While median survival for gliomatosis-like diffuse astrocytoma is typically reported as 12–30 months,⁴ our patient survived 9 years after tumour diagnosis and 15 years from symptom onset. This prolonged course likely reflects the tumour's lower histological grade (WHO grade 2) and the influence of individualised supportive care.

This case highlights the importance of periodic reappraisal of parkinsonism diagnoses, particularly when clinical evolution is atypical, regardless of whether gliomatosis-like infiltration is suspected. While the temporal association in our case raises the possibility of a causal link, we cannot exclude two unrelated pathologies. The unusually long survival of 15 years from symptom onset likely reflects the tumour's lower histological grade (WHO grade 2) and the benefit of individualised care.

Figure 1: Magnetic resonance imaging (MRI) sequences in axial FLAIR (A and B), axial T2-weighted (C), coronal FLAIR (D), axial diffusion-weighted (E) and axial post-contrast T1-weighted (F) images. The cortico-subcortical infiltrative lesion shows mass effect and T2/FLAIR hyperintensity, without diffusion restriction or enhancement after intravenous contrast administration. The lesion involves the right temporal lobe, mesial temporal region, amygdala, insula and cingulate gyrus (*). Extension through the anterior commissure (white arrow) is observed, with additional involvement of the anterior aspect of the mesial temporal region and insula on the left (red arrow). The midbrain and pons are also affected (green arrow).



COMPETING INTERESTS

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