Temporal lobe epilepsy mimicking a hippocampal amnestic syndrome suggestive of Alzheimer disease: the value of biological diagnosis

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Background and purposes

Detection and management of early onset mild neurocognitive disorders requires a thorough diagnostic evaluation, screening for potentially reversible causes. In the era of biomarkers-based etiological diagnosis, careful and strict clinical follow up often yields a more accurate framing of the underlying pathology as indicative symptoms and manifestations become evident. We describe a case of amnestic mild cognitive impairment with late evidence of temporal lobe epilepsy.

Case report

A 48 years old man sought medical attention for progressive memory impairment, new onset anxiety disorder and insomnia impacting on his work abilities. Past medical history was only significant for a single tonic-clonic seizure during infancy. Neurological examination was unremarkable; as for neuropsychological testing, he scored **24/30 MoCA**, with main memory deficits. Brain MRI was unremarkable. A brain PET-FDG/MRI showed posterior **parietal** and **occipital** hypometabolism bilaterally with partial sparing of the calcarine cortex and mild global atrophy (GCA score of 1 and Koedam score of 1 on the right) (*Figure 1*). CSF analysis showed negative ATN status, and PET with 18F-Florbetaben was negative for tracer retention with early frames suggestive of hypoperfusion of the posterior parietal cortex. CSF and blood testing for common antibodies associated with limbic encephalitis as well as extensive genetic testing for early onset dementia were negative. During follow up visits the patient reported multiple daily episodes of mental confusion, altered awareness and disorientation. An urgent EEG (*Figure 2*) showed abundant **epileptiform discharges** in both temporal lobes, and he was started on antiepileptic therapy. The patient reported improvement in both sleep and mood disorder and a reduced frequency of the episodes but no effect on memory deficits after 20 days of treatment.

Neuroimaging

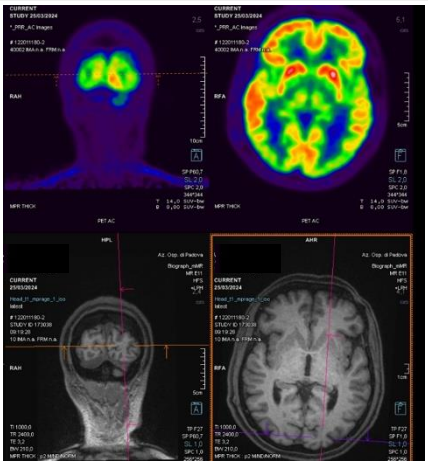

Figure 1. Brain PET-FDG/MRI. Note posterior bilateral parieto-occipital hypometabolism.

Neurophysiology

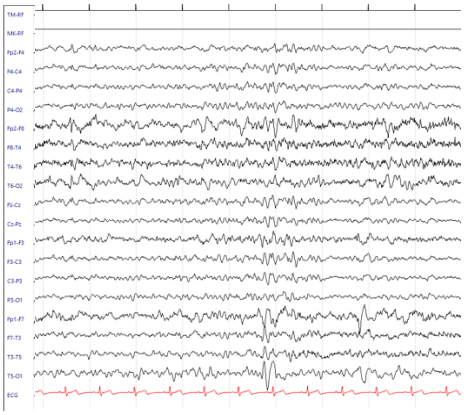

Figure 2. Electroencephalogram showing bilateral temporal epileptiform discharges

Conclusions

Temporal lobe epilepsy is closely associated with memory deficits, particularly affecting episodic memory due to involvement of the hippocampus and surrounding mesiotemporal structures. Although MRI was normal, we can't disprove cortical microstructural gradients reorganization correlating with cognitive network reorganization and episodic memory dysfunction or default mode network disturbances resulting in cognitive dysfunction. Follow-up visits will clarify whether memory deficits will be improved by a course of effective antiepileptic treatment.