Interesting images

Brain metabolic changes in limbic encephalitis evidenced by $^{18}$FDG PET. Correlation with symptomatology

Cambios metabólicos cerebrales en una encefalitis límbica evidenciada mediante $^{18}$FDG PET. Correlación con la sintomatología


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**A R T I C L E  I N F O**

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Limbic encephalitis (LE) is a common etiology of subacute dementia, clinically characterized by the association of temporal lobe seizures, episodic memory impairment and psychiatric symptoms. This process is usually associated with malignant tumors, although has been described in association with autoimmune disorders such as Sydenham chorea, lupus or antiphospholipid syndrome.1

We report a 59 years old patient with complex neurological syndrome with behavioral disturbances visual hallucinations, paroxysts faciobrachial dystonic seizures, “déjà vu” and

![Figure 1](image-url)

**Figure 1.** The upper row shows a set of three transverse slices of $^{18}$FDG PET, two performed at striatal level (A) and one at parahippocampal and temporal cortex (B) acquired at the initial diagnosis. Intense and symmetric glycemic metabolism in caudate nucleus and putamen with respect to the brain cortex and a focal area of increased metabolism in the right anterior temporomedial cortex (arrow) is observed. The lower row shows $^{18}$FDG PET slices acquired three months after the diagnosis, showing striatal system (C) and parahippocampal temporal cortex (D). With respect to the findings of A and B the visual ratio between the metabolism of basal ganglia and the temporal cortex had significantly diminished and the right temporomedial focal increased glycemic metabolism had disappeared suggesting a good evolution of the process.

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short-term memory loss episodes. A limbic encephalitis (LE) was suspected and a brain MR was performed that was reported as normal. A wholebody and brain PET-CT with $^{18}$FDG was requested in order to exclude a paraneoplastic syndrome. Sixty minutes after a 3.7 MBq/kg of $^{18}$FDG dose, low-dose CT (120–140 kV, 80 mA) and 3D wholebody PET were obtained (2 min/bed), followed by brain dedicated images (5 min/bed) with a Discovery © PET-CT system. Wholebody images did not show any pathological uptake or lesions that could suggest the presence of a neoplastic process. However, brain images showed pathological intense and symmetric glycidic metabolism in caudate nucleus and putamen and small focus in the right anterior parahippocampal cortex (Fig. 1A and B). The patient showed intense and characteristic neurological symptomatology of LE and hippocampal or medial temporal lobe dysfunction²: somnolent, partially disoriented with frequent faciobrachial dystonic seizures (one each 5 min), visual hallucinations and daily “déjà vu” episodes. The dystonic seizures affected the left facial side, but also the right upper and lower limbs. The patient was diagnosed of LE with faciobrachial dystonic seizures secondary to autoimmune disorder with IgG antibodies. The patient followed three months steroid therapy and the neurological symptomatology improved: the patient remained oriented with diminishing of the faciobrachial dystonic seizures in length and frequency (one each 15 min and with less intensity), knowing previously the patient when the seizures were going to start. The visual hallucinations and “déjà vu” episodes had disappeared. A new $^{18}$FDG PET-CT was performed with the same dose and acquisition parameters showing that the metabolism of nigroestriatal system had significantly diminished and the right temporomedial focus had disappeared (Fig. 1C and D). In the following months, the patient continued with slow favorable evolution.

LE may show a great variety in its clinical presentation that leads to a wide differential diagnosis including, viral infections, autoimmune disorders and paraneoplastic syndromes.² Functional neuroimaging could be useful to identify brain dysfunction associated with psychiatric symptoms, but few precise data are available up to now.² The dystonic symptomatology might be related with the nigroestriatal system alterations, meanwhile the visual hallucinations and “déjà vu” episodes were referred to the limbic and hippocampal dysfunction.³

Due to the possible etiology of LE, we should perform a whole body PET-CT including brain images. We enhance the role of brain images due to that finding of symmetric striatal hypermetabolism could be highly indicative of autoimmune LE.¹ This finding might help in the diagnosis of this disease, and provide a useful marker of outcome.

References