Interesting images

Role of $^{18}$F-FDG PET in the diagnosis of Rasmussen’s disease

Papel de la $^{18}$F-FDG PET en el diagnóstico de la Enfermedad de Rasmussen

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Rasmussen’s disease is a chronic cerebral, inflammatory disease which affects only one hemisphere of the brain and is characterized by its progressive course, with convulsive episodes resistant to antiepileptic treatment.¹

The etiology of this disease is uncertain, being related to infection by different neurotropic viruses, the presence of cerebral glutamate receptor-3 antibodies (anti-GluR3), glutamic acid antidecarboxylase antibodies (anti-GAD) or T-cell mediation in the cerebral inflammatory process.¹

This disease is included among the catastrophic epilepsies and, at present, the only effective treatment is functional hemispherectomy, which is usually performed in early stages of the disease.¹

The use of positron emission tomography (PET) with 2-$(^{18}$F)-fluoro-2-deoxy-D-glucose (FDG) in pediatric neurology is limited.² Some authors have demonstrated that $^{18}$F-FDG PET is better than concomitant magnetic resonance (MR) for detecting the lesions and their extension early in Rasmussen’s disease.³

We present the case of a 5-year-old girl who suddenly developed right hemiparesia with myoclonic and clonic seizures lateralized to the right side, predominantly in the buccal commissure and the upper right extremity in 2010. Posteriorly, generalized tonic–clonic seizures and convulsions were also presented. The patient was admitted to the pediatric ICU showing poor response to antiepileptic medication. Thereafter, partial continuous epilepsy, cognitive deterioration and progressive hemiparesia developed. The EEG showed continuous delta activity with an anterior acute-spike discharge with clear left predominance. Several MR tests performed until September 2010 were unspecific. However, on evolution of the clinical picture, an area of a high signal in the T2 sequence was observed involving the left periventricular white substance associated with slight ventricular and homolateral subarachnoid hemispheric space enlargement.

In June 2011, $^{18}$F-FDG PET-CT study was performed, demonstrating moderate hypometabolism throughout the left cerebral hemisphere with greater involvement of the frontal and temporal cortex as well as moderate–severe hypometabolism in the anterior and mesial region of the left temporal lobe. No significant alterations in glycidic metabolism were observed in the subcortical structures, the lymph nodes of the base or cerebellum. In addition, left cortical atrophy was observed with a smaller size of the left cerebral hemisphere (Figs. 1 and 2).

In July 2011, left hemispherectomy was performed due to disease progression as well as a lack of response to the anticonvulsive medication and immunotherapy. The histological diagnosis was Rasmussen’s disease.

Although some authors² have described metabolic changes using $^{18}$F-FDG PET in early stages of the disease, few studies have reported advantages of $^{18}$F-FDG PET over MR in the study of these patients.

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Figs. 1 and 2. Axial and coronal slices of $^{18}$F-FDG PET showing moderate hypometabolism of all the left cerebral hemisphere with greater involvement of the frontal and temporal cortex as well as moderate–severe hypometabolism in the anterior and mesial region of the left temporal lobe. No significant alterations were observed in glycidic metabolism in the subcortical structures, basal ganglia and cerebellum. Moreover, left cortical atrophy was detected, with a smaller size of the left cerebral hemisphere.

References

