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18F-FDG PET/CT as A Diagnostic Tool in A Pediatric Patient with Rasmussen's Encephalitis. Case Report

Nathalie Hernández-Hidalgo[1*], Alejandra Gómez Marting[2], Clarena Zuluaga-Arbeláez[1], Claudia Gutiérrez-Villamil[1], Víctor Marin-Oyaga[1]

[1]Department of Nuclear Medicine, Fundación Cardioinfantil- Instituto de Cardiología, Bogotá, Colombia.
[2]Faculty of Medicine, Specialization in Nuclear Medicine, Fundación universitaria Sanitas. Bogotá, Colombia.

[*]**Corresponding author:** Nathalie Hernández-Hidalgo, E-mail: nathaliehh2809@gmail.com; Tel: +57 3508919905

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Abstract

Rasmussen encephalitis (RE) is a rare condition characterized by a chronic inflammatory disorder due to unilateral inflammation of the cerebral cortex. Typically, it affects one cerebral hemisphere and manifests itself through intractable epileptic seizures. It occurs most frequently in children and its occurrence in adults is rare. We present a case of a 2-year-old male patient, presenting focal seizures with altered consciousness, progressive left-sided hemiparesis and ipsilateral hyperreflexia. Despite a normal brain magnetic resonance imaging (MRI), 18F-FDG PET/CT revealed marked hypometabolism in the right cerebral hemisphere, predominantly involving the parietal and temporal lobes. Based on these findings, the patient underwent therapeutic plasmapheresis, which resulted in a significant reduction in seizure frequency and clinical improvement of the neurological deficit. This case highlights the diagnostic value of 18F-FDG PET/CT in Rasmussen encephalitis, particularly in early stages where structural imaging may be inconclusive. Early detection through functional imaging enabled prompt initiation of immunomodulatory therapy, with a favorable impact on the patient's neurological outcome.

Keywords: *Rasmussen's encephalitis, focal epilepsy, brain PET/CT, molecular imaging, pediatric patient.*

Introduction

Rasmussen's encephalitis is a rare chronic neurological disorder, characterized by unilateral inflammation of the cerebral cortex, drug-resistant focal epilepsy, and progressive neurological and cognitive deterioration, with unihemispheric brain atrophy (1). Affects mostly children or young adults (2). This entity was first described in 1958 by neurosurgeon Theodore Rasmussen and colleagues (1,2).

Fluorine-18 deoxyglucose (FDG), a glucose analogue taken up by metabolically active cells, is a radiopharmaceutical commonly used in PET studies. It has shown a good correlation between changes in structural images and hypometabolism in the affected hemisphere, in relation to the parenchymal changes generated by the RE (3). Some authors mention that the metabolic changes evidenced in the PET study may precede the structural changes evidenced in MRI (1), however, there are few studies that justify the use of brain PET/CT as the study of choice in RE. We present the case of a patient with a suspected diagnosis of Rasmussen's encephalitis in whom 18F-FDG PET-CT was used as a diagnostic approach.

Clinical Case

We present the case of a 2-year-old male infant who presented with focal seizures with altered consciousness. Two video telemetries were performed with abnormal results, compatible with focal epilepsy in the right posterior quadrant, discharges characterized by spikes and sharp waves between 30 and 50 microvolts, with maximum P4 negativity in 30% of the tracing. The value of immunoglobulins was normal. Anticonvulsant therapy with oxcarbazepine and topiramate was initiated, without achieving crisis, with an increase in ictal frequency and neurological targeting, with the appearance of left hemiparesis and ipsilateral hyperreflexia. Subsequently started methylprednisolone pulses, for suspicion of immunomedized epilepsy type encephalitis of Rasmussen.

Initially, an MRI was performed, without showing any abnormal findings (Figure 1). Then they perform 18F-FDG PET/CT finding marked generalized decrease in metabolism in the right cerebral hemisphere (Figure 2).

With the results of the PET/CT they decide to take the patient to 5 plasmapheresis sessions, performed at the pediatric ICU, obtaining as a result decrease in the number of epileptic crises and improvement of left hemiparesis, thus

confirmed the autoimmune etiology. Currently the patient has improved clinically, seizures are less frequent and immunomodulatory management continues.

Eight months later, a PET/CT was performed, which showed mild generalized hypometabolism in the cortex of the right cerebral hemisphere, predominantly parietal, but with significant improvement compared to the previous study (Figure 3).

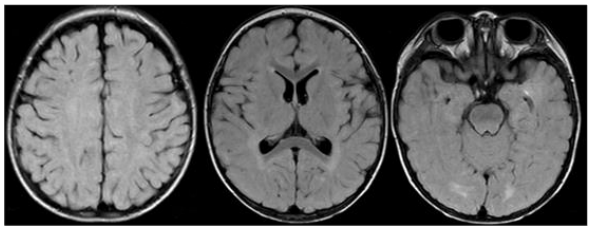

Figure 1. MRI. Diffusion-weighted, T2-weighted gradient echo, TSE, and FLAIR sequences were performed in the sagittal, axial, and coronal planes, obtaining T1- and T2-weighted images (epilepsy protocol). The signal intensity of the visualized brain and cerebellar parenchyma was normal, with no focal or diffuse abnormalities identified.

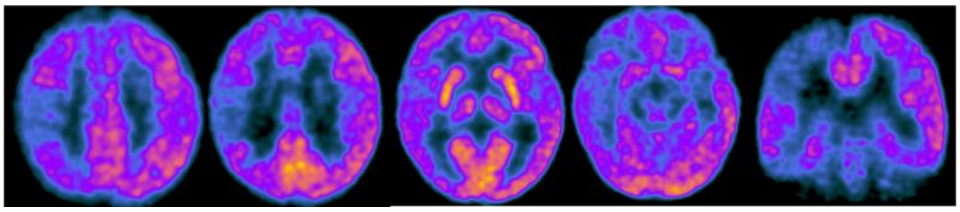

Figure 2. ^{18}F-FDG PET/CT Marked decrease in metabolism in the right cerebral hemisphere compared to the contralateral hemisphere, predominantly in the right parietal and temporal lobe.

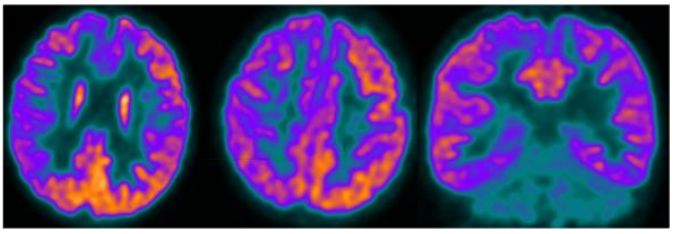

Figure 3. 18F-FDG PET/CT. Eight months after the PET/CT scan was performed, which showed mild generalized hypometabolism in the cortex of the right cerebral hemisphere, predominantly parietal, but with significant improvement compared to the previous study.

Discussion

Rasmussen's encephalitis is a rare pathology; the median age of onset is 6 years, with a range from infancy to adulthood. The natural clinical course of RE is characterized by prodromal stage with mild hemiparesis or infrequent seizures, might precede the onset of the acute stage by up to several years (1). The acute stage is marked by continuous seizures arising from one cerebral hemisphere, about 50% of patients have epilepsia partialis continua (EPC). Untreated, children will develop hemiparesis, hemianopia, and cognitive decline within a year of epilepsy onset, and if the language-dominant hemisphere is affected, dysphasia. Residual stage with a severe fixed neurological deficit, motor and cognitive problems, and with persisting difficult-to-treat relapsing epilepsy (1,3).

Its etiology is uncertain, although theories related to infection by various neurotropic viruses, the presence of antibodies against brain glutamate receptor 3 (anti-GluR3), anti-glutamic acid decarboxylase (anti-GAD) antibodies (4) and other studies support the notion that Rasmussen's encephalitis is probably driven by a T-cell response to one or more antigenic epitopes, with potential additional contribution by autoantibodies. Careful analysis of the association between histopathology and clinical presentation suggests that initial damage to the brain is mediated by T cells and microglia, suggesting a window for treatment if Rasmussen's encephalitis can be diagnosed early (3).

According to the 2005 European Consensus, three cardinal criteria are required for RE diagnosis: 1. focal seizures with unilateral cortical deficits, 2. unilateral EEG abnormalities, and 3. mono-hemispheric MRI focal cortical atrophy with grey and/or white matter hyperintense signals or atrophy of the ipsilateral head of the caudate nucleus. Alternatively, a diagnosis may be made if two of the following criteria are met: 1. EPC or progressive unilateral cortical deficits, 2. progressive mono-hemispheric cortical atrophy, or 3. histopathological features of RE on brain biopsy (1).

The brain magnetic resonance is used as the image of choice, presenting some characteristic findings such as contralateral cerebral hemi atrophy to the hemicuerpus that is presented the neurological deficit, diffuse brain atrophy, injuries with hyperintense signal on T2W/FLAIR alterations in the hippocampus, caudate nucleus atrophy, ex vacuo dilatation of the ventricular system and sulci (5). However, when the resonance is normal or the findings are not conclusive, other image methods must be chosen to guide the diagnosis, so the 18F-FDG PET/CT charges special relevance (6).

18F-FDG PET/CT has shown better performance than MRI in early detection of lesions and determining the extent of involvement in Rasmussen's encephalitis (7). Some of the characteristic findings of RE on PET/CT include diffuse interictal hypometabolism in one cerebral hemisphere, hypometabolism in the contralateral cerebellar hemisphere (crossed cerebellar diaschisis), and hypermetabolic zones secondary to ictal foci (4,7,8). These changes are evident

from the earliest stages of the disease and may even precede the anatomical changes seen on MRI (9).

Treatment of RE includes antiepileptic drugs which has a limited effect on seizures and disease progression, immunosuppressive or immunomodulatory treatments are being assessed, some publications have shown the most positive experience with long-term corticosteroids, intravenous immunoglobulins, plasmapheresis or protein A immuno-absorption, and the T-cell inactivating drugs tacrolimus and azathioprine, and finally surgery still remains the only cure for the seizures caused by Rasmussen's encephalitis. This has functional consequences because the only effective surgery remains complete disconnection of the affected hemisphere (hemidisconnection), either as (functional) hemispherectomy or hemispherotomy (1,10,11).

Conclusions

Rasmussen's encephalitis is a rare and poorly understood disease. An early diagnosis can change a patient's prognosis by starting therapies early, thus preventing accelerated neurological deterioration. Therefore, pediatricians, neurologists, and diagnostic imaging specialists should be familiar with this pathology in order to properly identify it.

It is also important to highlight the usefulness of 18F-FDG PET/CT of the brain as a first-line imaging technique for this pathology, since the limited evidence from clinical case reports and case series demonstrates the diagnostic efficacy of molecular imaging in this context. In the case presented, this technique was the fundamental pillar for the clinician to make decisions regarding the patient's management, improving the neurological prognosis.

The patient's mother signed the informed consent form where she agrees to the publication of the clinical case.

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