

Interesting images

Changes in cerebral metabolism detected by ^{18}F -FDG PET-CT in a case of anti-NMDA receptor encephalitis[☆]Cambios en el metabolismo cerebral detectados mediante ^{18}F -FDG PET-TC en un caso de encefalitis antirreceptor de NMDAM.A. Ochoa-Figueroa^{a,*}, C. Cárdenas-Negro^b, A. Allende-Riera^b, J. Uña-Gorospe^b, D. Cabello García^b, M. DeSequera-Rahola^b^a Servicio de Medicina Nuclear, Hospital Universitario Insular de Gran Canaria, Las Palmas de Gran Canaria, Spain^b Servicio de Medicina Nuclear, Hospital Universitario de Nuestra Señora de La Candelaria, Santa Cruz de Tenerife, Spain

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Anti-NMDA receptor encephalitis (antibodies against the N-methyl-D-aspartate receptor) has only recently been described. It was initially identified in young women with paraneoplastic syndrome mainly associated with ovarian teratoma and with testicular teratoma and small cell lung carcinoma in men, however, a proportion of patients without neoplasm has been identified. The clinical manifestations are characterized by alterations in consciousness, hallucinations, convulsions, complex involuntary movements and respiratory failure. The discovery of these autoantibodies has widened the spectrum of differential diagnoses in the context of young patients with encephalitis with no established cause and in whom infectious and inflammatory causes have been excluded. The MR may be normal or show temporal hyperintensities or atrophy in different areas. Diagnosis is confirmed by the detection of antibodies against the NR1 subunit of the NMDA receptor in the CSF.^{1,2} On the other hand, PET provides non invasive information on the functioning of the human brain *in vivo* at a biochemical-molecular metabolic level and constitutes an excellent tool for clinical evaluation.³ It is therefore of great interest to study this type of patient by PET.

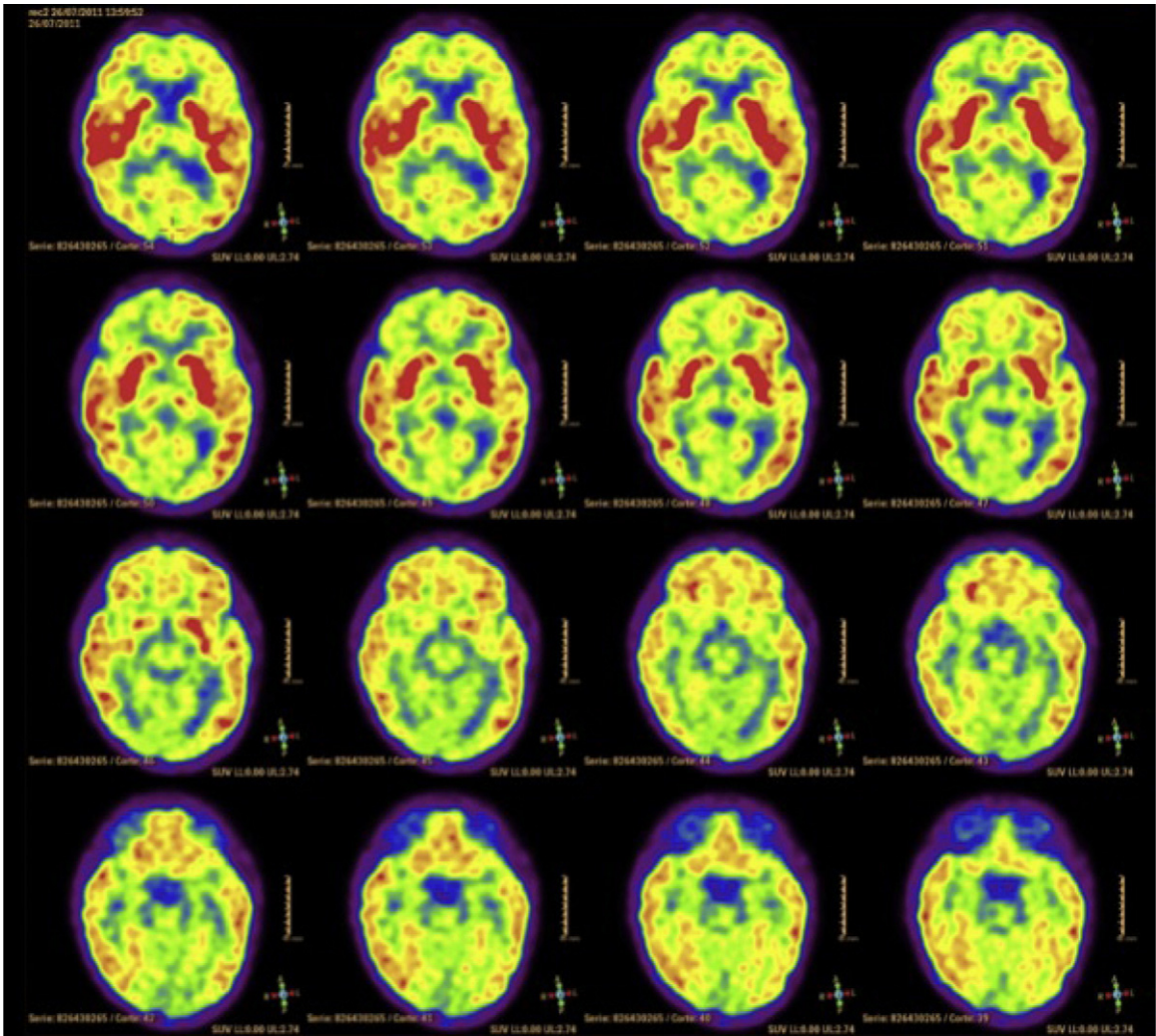
We present the case of a 3-year-old girl who presented psychomotor agitation and dystonic movements of the upper extremities as well as orolingual movements with occasional protrusion of the tongue and intermittent horizontal nystagmus. The patient also presented fever and alterations in the CSF and in the EEG compatible with acute encephalitis. Antibiotic therapy was begun, albeit with no apparent clinical response.

At 48 h after admission a cerebral MR was requested which was normal. The patient continued with a torpid evolution, demonstrating EEG findings compatible with severe encephalopathy. With the clinical EEG worsening the patient was admitted to the pediatric ICU for better monitoring and adjustment of treatment, beginning with methylprednisolone on suspicion of encephalomyelitis. At 14 days after admission the cerebral MR was repeated and was unspecific, thereby ruling out acute disseminated encephalomyelitis and a descending schedule of corticoids was initiated. The dystonias were treated with L-DOPA with no apparent response. Lumbar puncture was repeated and the serologic and metabolic studies were widened since all the results, until then, had been normal. Moreover, with the probability of an anti-NMDA receptor encephalitis the patient was also treated with immunoglobulins. Treatment with sedation was continued (midazolam, clonazepam, carbamazepin and propofol bolus). Thirty days after admission an ^{18}F -FDG PET-CT study was requested showing global cortical hypometabolism which may have been due to the medication administered, although it was of note that this was more marked in the right basal frontal cortical region and bilateral temporal-occipital region of right predominance with uptake of the normal tracer in the left basal ganglia and asymmetry of uptake in the caudate, showing less glycidic uptake on the right. No other findings of interest were detected. Two days after the PET-CT study the CSF examination confirmed the diagnosis of anti-NMDA receptor encephalitis (Figs. 1–3).

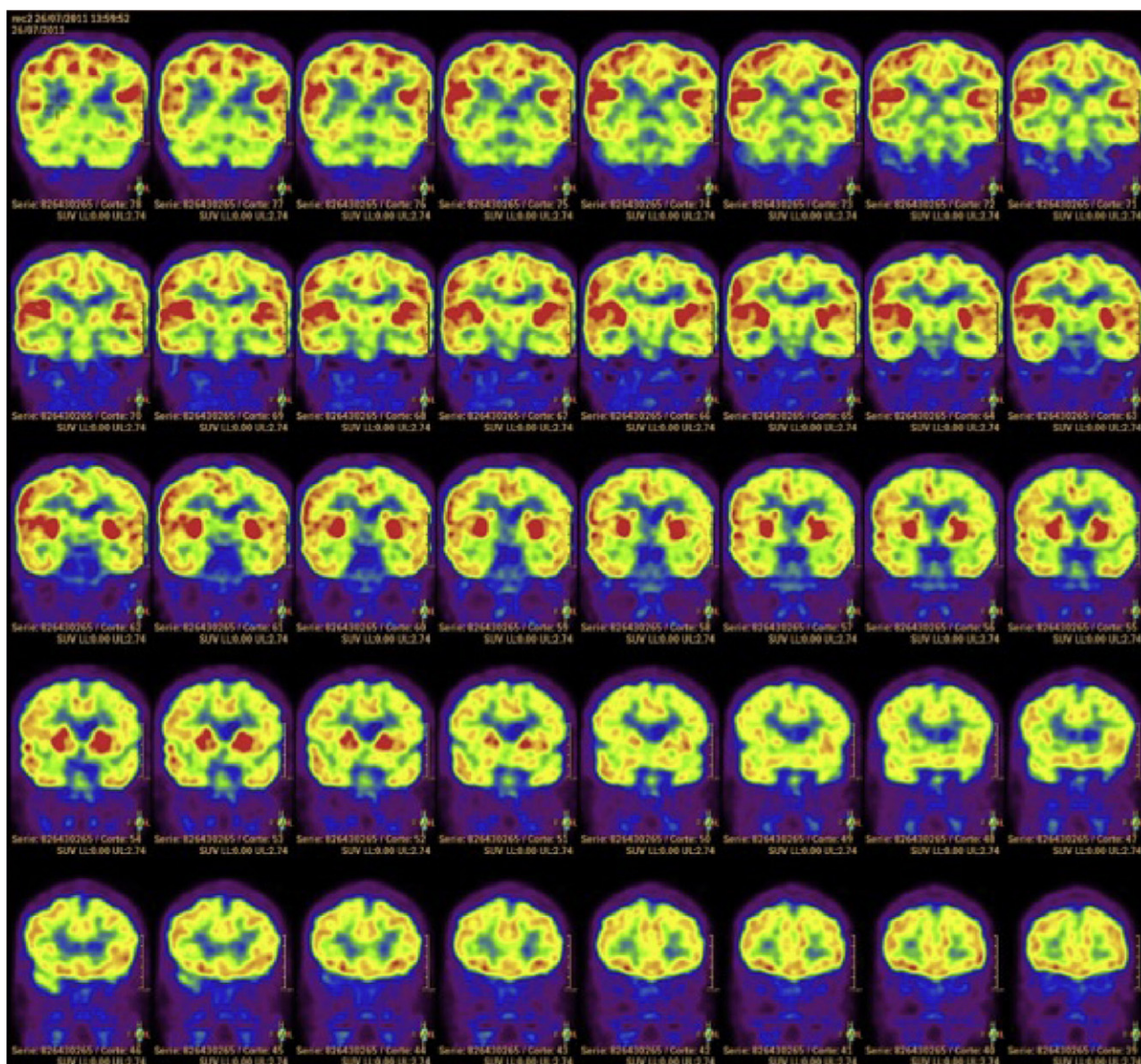
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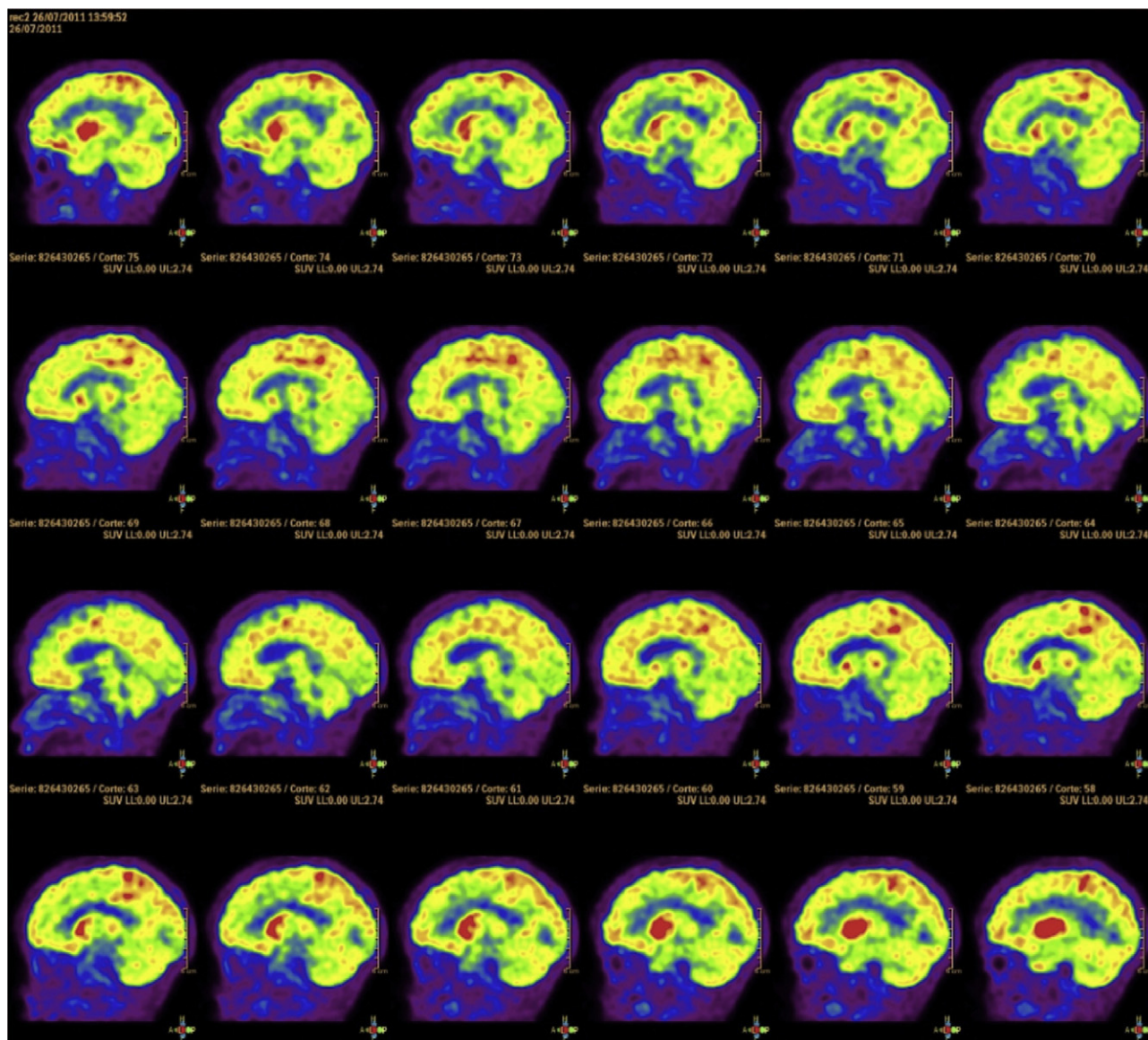
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Figs. 1–3. Axial, coronal and sagittal slices of ¹⁸F-FDG PET, showing a global cortical hypometabolism which was more marked in the right basal frontal cortical region and bilateral temporal-occipital of right predominance with uptake of the normal tracer in the left basal ganglia and asymmetry of uptake in the caudate, showing less glycidic uptake on the right.



Figs. 1–3. (Continued)



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References

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