

Interesting image

¹⁸F-FDG PET/MR in herpes simplex virus encephalitis: A case study



¹⁸F-FDG PET/RM en la encefalitis por virus herpes simple: un caso clínico

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Encephalitis usually presents different patterns of 2-deoxy-^{2-(¹⁸F)} fluoro-D-glucose (¹⁸F-FDG) distribution¹ and generally manifests itself on ¹⁸F-FDG areas of hypermetabolism but large areas of hypometabolism can be detected as well.¹ To date, the findings of one of the main studies performed on patients with encephalitis suggest that, in the acute phase, the ¹⁸F-FDG hypermetabolism can be the result of active inflammation, but it is necessarily to exclude ictal seizure activity as a possible cause of hypermetabolism for a correct interpretation of the PET findings.¹ On the other hand, ¹⁸F-FDG hypometabolism may represent one of the chronic sequelae of encephalitis as a consequence of neuron loss due to the inflammatory process.¹ In agreement with the evidences on ¹⁸F-FDG PET in seizure disorders, Lee et al. concluded that when the electroencephalogram is negative the hypermetabolism is most likely associated with the inflammatory process characteristic of encephalitis regardless of exact etiology.¹

A 72-years-old man with a 3-year history of mild memory loss and cognitive impairment as reported by relatives, was referred to our center for a further worsening of a neurologic symptomatology characterized by spatial-temporal disorientation, anomic and mixed aphasia, apraxia and cognitive impairment in absence epileptic seizures and a 2 months history of fever (temperature at admission 37.9 °C). The diagnosis of herpes simplex virus (HSV) encephalitis was confirmed by laboratory testing and cerebrospinal fluid PCR. The patient underwent a brain contrast enhanced Magnetic Resonance (ceMR) and an ¹⁸F-FDG PET/CT after one day from the ceMR scan.

In agreement with a previous similar report,² the brain PET/MR images (Fig. 1) obtained by means of Mimvista software (MIMvista Corp., Cleveland, OH) showed a wide hypometabolism of the left temporal, parietal and occipital lobe(a, b) in agreement with the

neurological symptoms of the patient. This metabolic pattern is widely recognized as a typical finding in progressive dementia (which symptoms are similar to those shown by our patient and, generally, in patients with dementia associated with infectious diseases)¹ and corresponded to a white matter-cortical wide hyperintensity areas in the T2-weighted MR images and hyperintense areas in T1-weighted MR images, likely necrotic-hemorrhagic (arrowhead in c and d) that were overlapped to a markedly reduced ¹⁸F-FDG uptake (c,d,e,f).³

PET images showed a reduced left thalamic and striatal ¹⁸F-FDG uptake(arrowhead in a) that did not correspond to any pathological ceMR finding (c) and probably determined by a large ipsilateral cortical necrosis resulting in a synaptic loss.

A focal area of increased glucose uptake in the inferior left temporal gyrus (arrowhead in b) corresponded with a focal gyral enhancement in post-contrast-T1w ceMR images (arrowhead in d, f); this finding has been interpreted as a focal inflammation with local meningeal vasodilatation (as in the case of active viral replication) that could explain the mild fever onset in our patient.^{1,3}

As in the case of our patient, viral encephalitides are sometimes insidious and, although typically presenting as acute encephalitis, do present with more gradual behavioral and mental status changes with frequent exacerbations.¹

Together with clinical data (i.e. the presence of seizure activity and other conditions) (1) the combination of ¹⁸F FDG PET and MR examination may add important data for a better overall clinical evaluation (identification of active foci), disease prognosis (functional involvement of other cortical and sub cortical structures without morphological abnormalities), treatment and follow-up of patients affected by herpetic encephalitis.

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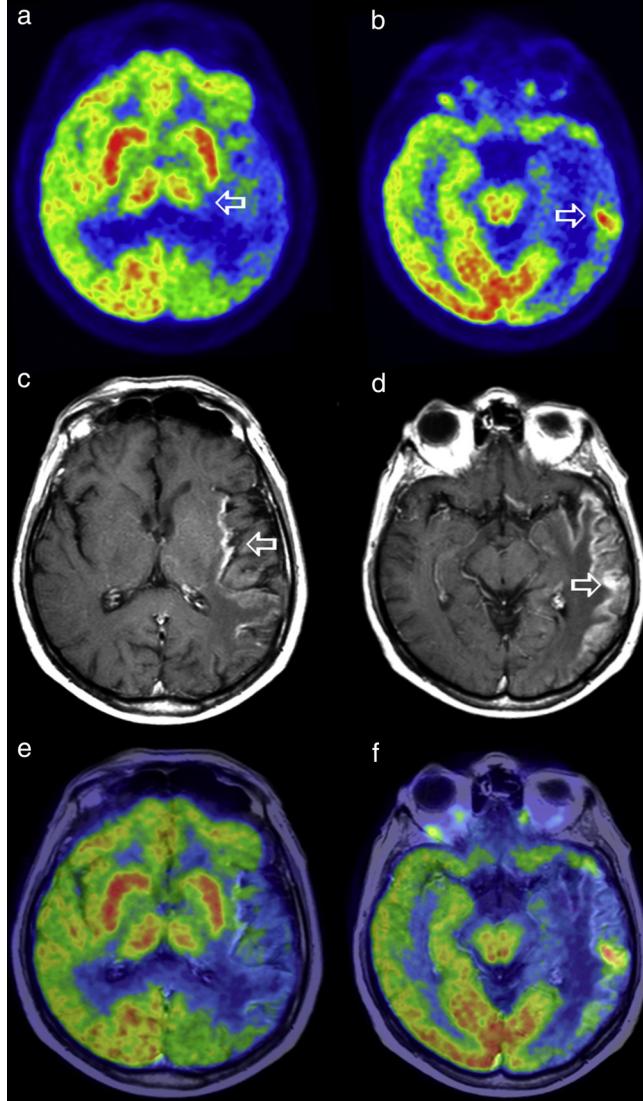


Fig. 1. Axial ^{18}F -FDG PET slices showing a wide reduction of glucose metabolism at a cortical level in the left hemisphere (a, b). A reduced tracer uptake is detectable in left thalamus and basal ganglia (arrow in a). A focal area of ^{18}F -FDG increased uptake is detectable in the left temporal lobe (arrow in b). T1-weighted MR axial slices (c, d) showing the morphological correlate of the PET findings (arrows in c and d, see text). Co registered PET/MR axial views are shown in e and f.

References

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