Cerebral glucose hypometabolism in tick-borne encephalitis, a pilot study in 10 patients

ABSTRACT

Background: Tick borne encephalitis (TBE) is an acute meningoencephalitis with or without myelitis caused by an RNA virus from the flavivirus family transmitted by Ixodes spp ticks. The neurotropic TBE virus infects preferentially large neurons in basal ganglia, anterior horns, medulla oblongata, Purkinje cells and thalamus. Brain metabolic changes related to radiologic and clinical findings have not been described so far. Methods: Here we describe the clinical course of 10 consecutive TBE patients with outcome assessment at discharge and after 12 month using a modified Rankin Scale. Patients underwent cerebral MRI after confirmation of diagnosis and before discharge. ^{18}F-FDG PET/CT scans were performed within day 5 to day 14 after TBE diagnosis. Extended analysis of coagulation parameters by thrombelastometry (ROTEM® InTEM, ExTEM, FibTEM) was performed every other day after confirmation of TBE diagnosis up to day 10 after hospital admission or discharge. Results: All patients presented with a meningoencephalitic course of disease. Cerebral MRI scans showed unspecific findings at predilection areas in 3 patients. ^{18}F-FDG PET/CT showed increased glucose utilization in one patient and decreased ^{18}F-FDG uptake in seven patients. Changes in coagulation measured by standard parameters and thrombelastometry were not found in any of the patients. Discussion: Glucose hypometabolism was present in 7 out of 10 TBE patients reflecting neuronal dysfunction in predilection areas of TBE virus infiltration responsible for development of clinical signs and symptoms.

Keyword: FDG-PET; Glucose metabolism; Neuroinfection; Tick borne encephalitis