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CLINICAL AND RADIOLOGICAL FEATURES OF TICK-BORNE ENCEPHALITIS IN AN IMMUNOSUPPRESSED PATIENT

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Introduction. Tick-borne encephalitis (TBE) virus remains one of the most frequent causes of central nervous system infections in Europe. The disease caused by European TBE virus (TBEV) subtype is typically characterized by a biphasic course. In the first phase, main symptoms include fever, fatigue, headache, and arthralgia. Neurological manifestations are characteristic for the second phase, with a clinical spectrum ranging from mild meningitis to severe encephalitis, which may be accompanied by myelitis and acute flaccid paralysis. Older age and immunosuppression are among risk factors for severe disease course and neurological complications. TBE is fatal in about 1-2% of patients.

Aim. The aim of this report is to present clinical and radiological features of tick-borne encephalitis in an immunocompromised patient.

Material and methods. A retrospective analysis of a medical history and radiological images of a patient who had been hospitalized in the Department of Infectious Diseases and Neuroinfections has been conducted.

Results and discussion. A 36-year-old female with a history of corneal transplant was admitted to the hospital with symptoms of encephalitis. The patient has been on post-transplant immunosuppressive therapy for the past six months and has not been vaccinated against TBE. The neurological examination at admission revealed drowsiness, mutism, weakness of left-side limbs, positive Babinski's sign, and nystagmus. Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis, however serological assays were negative for TBEV. Initial magnetic resonance imaging (MRI) revealed changes that apart from flaviviral infection could have also suggested Creutzfeldt-Jacob's disease, nontheless CSF analysis ruled out this possibility The patient received acyclovir, ceftriaxone and dexamethasone. During hospitalization the patient developed seizures and began showing symptoms of pulmonary edema. She was transferred to the intensive care unit where she was intubated and mechanically ventilated. Due to deteriorating state, she was placed into a drug-induced coma. After a month from the first TBE serology, another serology testing of serum and CSF was performed which finally confirmed the diagnosis of TBE. During the next months, neurological deficits were increasing. The patient had anisocoria, flaccid tetraparesis and showed no reaction to stimuli. Due to lack of therapeutic possibilities intravenous immunoglobulins were administered. However, the outcome was fatal in spite of symptomatic therapy.

Conclusions. Delayed production of anti-TBE antibodies can be observed in some patients with immunosuppression, which can hinder the correct diagnosis. Although MRI abnormalities are not a characteristic feature in patients with TBE in our patient they were located in brain areas which could have corresponded to flavivirus infection and were useful in the diagnostic process. Clinical course of TBE is unpredictable and in immunocompromised patients it tends to be more severe. There is no causative treatment and only symptomatic therapy is available, which may be not sufficient in some of the cases.