Vaccine breakthrough tick-borne encephalitis: a case report from an endemic country

Anja Šterbenc1 | Matej Furlan2 | Mateja Jelovšek1 | Matjaž Jereb2 | Tatjana Avšič-Županc1
1 Institute of Microbiology and Immunology, Faculty of Medicine, University of Ljubljana, Ljubljana Slovenia
2 Department of Infectious Diseases, University Medical Centre, Ljubljana, Slovenia

BACKGROUND

Slovenia is a small, highly forested country located in Central Europe. It is endemic for tick-borne encephalitis (TBE), an important vector-borne infection of the central nervous system. TBE virus (TBEV) is predominantly transmitted to humans through Ixodes spp. tick bites. Unfortunately, despite having high incidence rates of TBE (yearly incidence ranged between 3 and 15 cases/100,000 inhabitants during the last 5 years), TBEV vaccine uptake in Slovenia remains low (<15%).

CASE REPORT

We report the case of a 65-year old previously healthy male that was admitted to ICU due to rapidly progressing proximal muscle weakness. He complained of fever, chills and weakness that started 3 days before admission but denied headache, nausea or muscle pain. He had a history of tick bites and had a complete TBEV vaccination 7 years ago but failed to receive timely booster vaccination. Laboratory results revealed increased CRP (43 mg/L) and SR (32 mm/h) and cerebrospinal fluid (CSF) analysis showed pleocytosis. TBE was suspected; however, TBEV IgM and IgG in CSF were initially negative, whereas IgG (202 U/ml) exhibiting high avidity (91.2%) were detected in serum (Figure 1,2). Concurrent infections, including Borrelia spp., Toxoplasma spp., HSV1/2, EBV, CMV, VZV, HHV-6, enteroviruses and West Nile virus were excluded. His condition deteriorated two days later as he developed diplopia, confusion, and spasticity and had to be intubated, sedated, and mechanically ventilated. Brain MRI showed no clear signs of encephalitis. TBEV IgG increased in serum (243 U/ml) and CSF (34 U/ml), whereas IgM remained negative in both samples. On the fifth day of hospitalization the patient developed a grand-mal epileptic seizure that resolved after antiepileptic treatment. On the sixth day of hospitalisation, serum and CSF TBEV IgG further increased (319 U/ml and 304 U/ml, respectively) and IgM (0.214) was detected in CSF (Figure 1,2). Follow-up MRI showed meningoencephalitic changes, whereas serum and CSF IgM and IgG levels continued to increase. Patient’s condition gradually improved although significant neurological sequelae (partial flaccid tetraparesis and dysphagia) still remain 3 months after first presentation.

CONCLUSIONS

In countries endemic for TBE, the diagnosis should be suspected regardless of the vaccination status and even in the absence of intrathecal TBE Ig production at first presentation. Moreover, patients experiencing TBE vaccine breakthrough were shown to have a more severe course of the disease and should be thus carefully monitored.