Tick-borne-encephalitis-vaccine

S

Vaccination failure: case report

A 22-year-old man experienced vaccination failure following vaccination with tick-borne-encephalitis-vaccine [*route and dose not stated*].

The man, who was previously healthy, was transferred to the ICU with severe meningoencephalitis of unknown origin. Three days prior to the admission, he was admitted in a lower-level facility with high fever, malaise and headache. His history revealed a tick bite 2–3 weeks prior to the onset of symptoms. However, he had a positive history of vaccination against tick-borne encephalitis. The first two vaccinations were performed according to accelerated immunisation schedule and he had received tick-borne-encephalitis-vaccine [FSME Immune] first dose at day 0, second dose after 1–3 months and the third dose after 3.5 years. The subsequent booster doses were administered every 5 years with the latest booster dose 1 year prior to the admission. Because he had received a full course of vaccination against tick-borne encephalitis, West Nile virus infection was suspected. A diagnostic lumbar puncture was performed twice, revealing moderately elevated CSF protein, lymphocytic pleocytosis and normal lactate and glucose, consistent with viral meningoencephalitis. Tick-borne encephalitis-specific serum IgG, but not IgM antibodies, were positive, which was interpreted as immunization against tick-borne encephalitis (ELA IgG positive 6480 Vienna Units). Empirical antimicrobial therapy with aciclovir [acyclovir] and ceftriaxone and symptomatic treatment [*details not stated*] were initiated. On the second day of inpatient care, he developed left hemiparesis. Initial MRI showed ill-defined FLAIR-hyperintensity in the right thalamus and both corticospinal tracts with no diffusion restriction und no enhancement on post-contrast T1-WI. On a follow-up MRI two days later, swelling and FLAIR-signal intensity abnormalities were detected in the thalami, left caudate nucleus and corticospinal tract on both sides without enhancement, haemorrhage or diffusion restriction. The clinical course was complicated by temporal lobe epilepsy-like seizures, warranting symptomatic treatment with benzodiazepines and levetiracetam. Over the course of the next day, he became unresponsive and required endotracheal intubation. He showed no signs of organ failure, was undergoing mechanical ventilation for airway protection and was under sedation. An electroencephalogram (EEG) showed bursts of focal epileptiform discharges and generalised severe brain dysfunction. A cranial CT scan revealed moderate, diffuse cerebral oedema but excluded increased intracranial pressure. In the absence of papilloedema, a third diagnostic spinal tap and extensive testing were performed. Biochemical and cytological CSF tests were largely unchanged, with a moderately elevated protein level of 76.4 mg/dL, lymphocytic pleocytosis (120 cells/microL) with scarce monocytes and granulocytes and normal glucose and lactate. Based on the clinical presentation, the radiological findings and the history of complete vaccination against tick-borne encephalitis, West Nile virus infection was suspected.

Due to excellent safety profile reported by previous studies, poor health condition of the patient and lack of alternatives, the man initiated on off-label treatment with favipiravir 1600mg twice a day followed by 600mg twice daily for 7 days. At day 4 of favipiravir treatment, microbiological analysis revealed tick-borne-encephalitis virus-specific IgM and IgG in CSF, confirming the presence of tick-borne encephalitis virus infection and excluding West Nile virus infection. Meanwhile, his general status improved significantly and the follow-up EEG did not show focal waveform abnormality or epileptiform discharges. Therefore, therapy with favipiravir was continued. Further serological and PCR assays for influenza, respiratory syncytial virus, West Nile virus, Japanese encephalitis virus, rickettsia, babesia, cytomegalovirus, Epstein-Barr virus, Herpes simplex virus and Parechovirus were negative. He was successfully weaned from the ventilator and at the end of the 7-day antiviral treatment course almost complete resolution of tick-borne encephalitis virus infection was observed. By discharge, 14 days after the symptom onset, he had completely recovered.

Bologheanu R, et al. Unexpected complete recovery of a patient with severe tick-borne encephalitis treated with favipiravir. Antiviral Research 184: Dec 2020. Available from: URL: http://doi.org/10.1016/j.antiviral.2020.104952

803523111