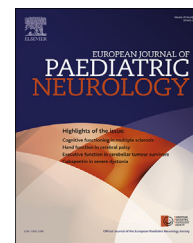




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Original Article

MRI-imaging and clinical findings of eleven children with tick-borne encephalitis and review of the literature



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ABSTRACT

Objectives: The incidence of tick-borne encephalitis (TBE) is increasing in many countries. Magnetic resonance imaging (MRI) in the course of TBE is not regularly performed in children. The aim of our study was evaluating MRI-findings of children and adolescents with TBE.

Patients and methods: Retrospective evaluation of the charts and MRIs of patients who had been treated for TBE in the four participating hospitals in the last twenty years.

Results: 11 patients (5 male; age at TBE 3 weeks–15 9/12 years; mean 104.9 months) were included. MRI (within the first week after admission) revealed symmetric or asymmetric T2-hyperintensities in both thalami in 7/11 patients with additional bilateral lesions in putamen and/or caudate nucleus in 3 patients, and additional cortical lesions in 2 patients. Our youngest patient presented with T2-hyperintensities affecting the whole left cerebral

Abbreviations: TBE, tick-borne encephalitis; CSF, cerebrospinal fluid; IVIG, intravenous immunoglobulines; FLAIR, fluid-attenuated inversion recovery.

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hemisphere including white and grey matter and both cerebellar hemispheres. One patient had a minimal reversible T2-hyperintensity in the splenium of the corpus callosum (RHSCC). 3/11 patients had a normal MRI. 4/11 patients showed complete neurological recovery (2/4 with a normal MRI, RHSCC patient). 6/11 children survived with significant sequelae: hemiparesis (n = 4); cognitive deficits (n = 4); pharmacoresistant epilepsy (n = 2). One patient died of a malignant brain edema.

Discussion: A spectrum of MRI findings can be found in children with TBE, often showing involvement of the subcortical deep grey matter structures. In children presenting with a meningoencephalitis and bilateral thalamic involvement TBE should be included in the differential diagnosis.

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1. Introduction

Tick-borne encephalitis (TBE) is caused by a member of the flaviviridae family and transmitted to humans mainly by tick bites. In the last years, the number of infected persons has increased in most countries¹ and is believed to be underestimated in many parts of Europe.² Other flavivirus infections, transmitted by insect bites, include diseases like dengue fever, yellow fever and Japanese encephalitis.³ Among diseases caused by neurotropic flaviviruses, only Japanese encephalitis is more frequent than TBE.⁴ There are three subtypes of TBE viruses: the European, the Siberian and the Far Eastern.⁴ For the European subtype the vector is *Ixodes ricinus*.⁵ After a median incubation time of eight days (range 4–28 days), the infection presents with a biphasic course, beginning with flu-like symptoms, which after a symptom-free interval, is followed by neurologic symptoms ranging from mild meningitis to severe encephalitis, cranial nerve involvement, radiculitis or myelitis.⁶ The entry of the TBE virus into the brain is supposed to be blood-borne.⁷ A severe course of TBE and neurological sequelae are mostly reported in adolescents beyond the age of 16 years and in adults⁸ and occur ten times less frequently in children.⁹ However, severe courses have been described in children as well.^{10,11}

Cerebrospinal fluid (CSF) analysis typically shows pleocytosis (>60 cells/ μ l). In serum TBE-specific IgM antibodies are found in the acute phase and conversion to virus-specific IgG in a follow-up sample at least two weeks later.^{12,13} EEG is often non-specific, with slowing of background activity. MRI shows preferential involvement of the thalamic nuclei, cerebellum, brainstem, caudate and the anterior horn of the cervical spinal cord in adolescents.^{14,15} In children MRI investigations in the course of TBE are only rarely reported.^{16,17} In a retrospective study of 19 children by Schmolck et al., 13 were diagnosed with a clinical picture of meningoencephalitis, while the other 6 patients had the clinical picture of meningitis. Out of four MRI investigations in the patients with meningoencephalitis, two revealed involvement of both thalami, and one of the cerebellum.¹⁶ In the prospective study by Kaiser, 14 of 44 children with TBE presenting as meningoencephalitis had MRI. In five children, the thalami showed hyperintense signals on T2-weighted images.¹⁷ Marjelund

et al.¹⁵ reported the MRI findings in TBE in 4 patients, one below the age of 18 years and described in three of their patients thalamic hyperintensities in T2-weighted images. In addition to these studies there are three case reports of children which also report detailed MRI findings. Jones et al. reported in the MRI of their 17-day old baby diffuse edematous changes in the cortex and subcortical areas of the left occipitoparietal region with marked meningeal contrast enhancement over left temporal lobe.¹⁸ Iff et al. described the MRI results of a 6-week-old patient as findings suggestive of a meningoencephalitis because of the cortico-subcortical hyperintensity with contrast enhancement.¹⁹ In the 5-year-old patient from Waldvogel et al. the MRI showed bilateral high signal in the thalamus, lentiform and caudate nuclei as well as contrast enhancement in the posterior right thalamus.²⁰ Apart from these case reports and the above mentioned studies by Schmolck, Kaiser and Marjelund^{15–17} has been to our knowledge no systematic study on MRI findings of children with TBE.

We therefore reviewed the clinical course of eleven patients with well-documented TBE with a particular focus on the MRI findings.

2. Patients and methods

In the authors' hospitals the patients' databanks were screened for patients who had been treated for TBE in the last 20 years. In a second step the charts of these patients and their MRI of the acute phase and in the subsequent course were reevaluated (CS and PW). The magnetic field strength of the MR was 1.5 T in all patients.

Ethical committee approval was obtained.

3. Results

Eleven patients were included (5 male; age at TBE 3 weeks–15/12 years; mean 104.9 months); three of these patients who had been treated for TBE have been previously reported.^{18,21} Two patients had received passive and two active immunizations. Seven patients recalled a tick bite. 4 patients showed the classical two-peak course, while headache and vertigo,

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