Signature: © Pol J Radiol, 2017; 82: 742-747
DOI: 10.12659/PJR.903940

Polish Journal of Radiology
www.PolRadiol.com
REVIEW ARTICLE

Received: 2017.02.21
Accepted: 2017.03.21
Published: 2017.12.15

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Evaluation of Imaging Methods in Tick-Borne Encephalitis

Radosław Zawadzki[1ABEFG], Adam Garkowski[1ABF], Bożena Kubas[2D], Joanna Zajkowska[3AF], Marcin Hładuński[2B], Dorota Jurgilewicz[2D], Urszula Łebkowska[1ADFG]

[1] Department of Radiology, Medical University of Białystok, Białystok, Poland
[2] Laboratory of Molecular Imaging, Medical University of Białystok, Białystok, Poland
[3] Department of Infectious Diseases and Neuroinfections, Medical University of Białystok, Białystok, Poland

Author's address: Radosław Zawadzki, Department of Radiology, Medical University of Białystok, M. Skłodowskiej-Curie 24A Str., 15-276, Białystok, Poland, e-mail: zaw.radoslaw@gmail.com

Summary

Tick-borne encephalitis (TBE) is caused by a virus that belongs to the *Flaviviridae* family and is transmitted by tick bites. The disease has a biphasic course. Diagnosis is based on laboratory examinations because of non-specific clinical features, which usually entails the detection of specific IgM antibodies in either blood or cerebrospinal fluid that appear in the second phase of the disease. Neurological symptoms, time course of the disease, and imaging findings are multifaceted. During the second phase of the disease, after the onset of neurological symptoms, magnetic resonance imaging (MRI) abnormalities are observed in a limited number of cases. However, imaging features may aid in predicting the prognosis of the disease.

MeSH Keywords: Encephalitis, Tick-Borne • Magnetic Resonance Imaging • Neuroimaging • Tomography, X-Ray Computed

PDF file: http://www.polradiol.com/abstract/index/idArt/903940

Overview

Tick-borne encephalitis (TBE) is a viral infection that affects primarily the central nervous system (CNS), and is caused by the TBE virus (TBEV) of the *Flaviviridae* family. The vectors of TBE are infected ticks; in Europe, mainly *Ixodes ricinus* [1,2]. The disease has two distinct stages. The first phase manifests itself after an incubation period of 2-28 days from the tick bite with non-specific, flu-like symptoms, and usually lasts 1–9 days [3,4]. Subsequently, after an asymptomatic period, in approximately 1/3 of patients, the second phase is observed, and is characterized by meningitis, encephalitis, meningoencephalitis, myelitis, and/or polyradiculitis. Most patients with TBE have mild symptoms and experience complete remission, but 1–2% of cases can have a fatal outcome [5]. Additionally, some authors describe persistent post-encephalitic symptoms, such as neuropsychiatric complaints (e.g. reduced stress tolerance, impaired memory), balance disorders, headache, dysphasia, hearing defects, and spinal paralysis [6–10]. Over the last two decades, there has been an increase in the incidence of TBE, which can be connected to the increase in TBE incidence in endemic regions as well as spreading of the virus into new areas (Austria, Switzerland, Slovakia, Finland, Russia) [11–13]. In Poland, the region with the highest occurrence is the Podlaskie Voivodeship, where 46% of all TBE cases are reported [6].

The method of choice for diagnosing TBE infection is by detecting TBEV-specific IgM and IgG antibodies in serum by enzyme-linked immunosorbent assay (ELISA). Additionally, cerebrospinal fluid (CSF) can be examined for the presence of TBEV-specific antibodies [14]. On post-mortem examinations (RT-PCR) of brain tissue samples from fatal TBE cases, we can observe accumulation of virus particles predominantly in the cerebral and cerebellar cortex, basal ganglia, thalamus, substantia nigra, pons, medulla oblongata, and the spinal cord [15]. The changes can be also detected with routine diagnostic methods such as computed tomography (CT) and magnetic resonance imaging (MRI). Advanced techniques have been increasingly used to study TBE. MR spectroscopy with proton spectroscopic MRI shows promising results in typical areas that are not characterized by any morphological changes. Importantly,

there is no causal treatment for TBE, and only symptomatic treatment of life-threatening symptoms, such as cerebral edema, is available.

Computed Tomography

CT has a limited utility in the diagnosis of TBE with a lower sensitivity than MRI, which is mainly due to improper delineation of involved areas, leading to underestimation of the extent of encephalitis [16]. Typical TBE changes include hypodense regions mainly in the basal ganglia and the thalamus. There are only few articles that document CT characteristics of pathological lesions in TBE. Horger et al. described a case of a 66-year-old man after a tick bite. On axial unenhanced CT images, low density regions were seen in the left putaminal and pallidal nuclei, and internal and external capsules[16]. In a case report of a 21-year-old male with confirmed TBE (Marjelund et al.), CT scans revealed a slight hypodense line in the left thalamus [17]. Waldvogel et al. reported a case of a 5-year-old girl, not vaccinated against TBEV and with a history of a tick bite, in whom CT, performed 4 days after the onset of symptoms, showed a hypodense, non-enhancing lesion within the right thalamus [18].

Magnetic Resonance Imaging

MRI shows only 20% of pathological lesions in the acute phase of the disease [19]. MRI is the preferred imaging method and supports the diagnosis of TBE. Maximal inflammatory changes around the tenth day correlate with optimal detectability of IgM and IgG antibodies in CSF [14], which suggests an optimal time for MRI examinations. As mentioned earlier, the virus shows a particular predilection to the basal ganglia and the thalamus [20]. Marjelund et al. reported MRI findings in 3 patients with severe TBE. In the aforementioned article, in one patient, T2-weighted and fluid-attenuated inversion recovery (FLAIR) images revealed increased signal intensity on the left side within the thalamus, putamen, and internal capsule. In another patient, it showed bilateral changes in the hypothalamus, cerebral peduncles, and pons. The same lesions were found in the last patient in both thalami. There was only one patient in whom T1-weighted gadolinium-enhanced images showed pathological lesions around the cerebral peduncles (on follow-up MRI, signal abnormalities were not seen in any patient) [17]. In the study by Kaiser, which involved 102 patients with a diagnosis of TBE, 18 patients had pathological changes on MRI, of whom 15 had pathological changes limited to the thalamus, and in the remaining 3 patients, lesions where found in the cerebellum, brainstem, and caudate nucleus [7]. Lorenzl et al., presented a case of a 38-year-old woman with bilateral lesions in the thalamus, cerebral peduncles, and the left caudate nucleus without contrast enhancement [21]. Valdueza et al., reported of a TBE case with hyperintense bilateral lesions in the thalamus, left stratium, insular cortex, tegmental mesencephalic area, pons, left inferior olive; these lesions showed a discrete gadolinium enhancement on follow-up MRI after 12 weeks [22]. Waldvogel et al. observed hyperintense lesions bilaterally in the thalamus, lentiform nucleus, and caudate nucleus on T2-weighted images. In that report, T1-weighted images also showed enhancement within the lateral posterior nucleus of the right thalamus following gadolinium administration, indicating disruption of the blood-brain barrier in this region; follow-up MRI performed after 3 months showed full recovery [18]. Pfister et al. described a case of a 55-year-old patient with severe TBE. In that case, MRI revealed regions of hyperintense signal bilateral in the thalami and in the left putamen [23]. Alkadhi et al. reported a case of TBE in a 52-year-old woman, who had pronounced bilateral hyperintense lesions in the thalamus, putamen, pallidum, and caudate nucleus on T2-weighted images, with no gadolinium enhancement on T1-weighted images; CT scans showed no pathological changes [24]. A patient described by Vollman et al. had a single hyperintense lesion on T2-weighted and diffusion weighted imaging (DWI) in the splenium of the corpus callosum, with a signal reduction of the apparent diffusion coefficient (ADC); on follow-up MRI, no pathological changes were observed [25]. Horgel et al. described 12 cases of TBE with pathological lesions. Contrast-enhanced T1-weighted images showed meningeal enhancement in the left precentral gyrus, geniculate ganglion, and tympanic segment of the left facial nerve. In other cases, contrast enhancement was seen in the cerebellar folia and peduncles. FLAIR images showed hyperintense lesions in the parietal gray-white matter junction, peritrigonal white matter in the thalami, basal ganglia, hippocampi, and vermian folia; T2-weighted images of the spinal cord showed hyperintense lesions in the anterior and posterior horns of the spinal cord. Horgel et al. also described a case of a 9-month-old boy with TBE. In that case, T2-weighted images showed a hyperintense lesion in the left putamen, and single-voxel ^{1}H-MR spectroscopy revealed altered metabolite spectral peaks at 0.5–1.5 ppm, which represent the superposition of lactate and alanine doublets; according to the authors, this could represent end products of microorganisms [16]. Grimm et al. described a TBE case in a 46-year-old patient with pathological hyperintense lesions in the splenium of the corpus callosum on T1-contrast-enhanced and T2/FLAIR images, with distinct diffusion-restriction and low ADC values on DWI; follow-up MRI, performed 10 days after the initial scan, showed full regression of the changes [26]. Figures 1 and 2 demonstrate typical morphological changes in the CNS during TBEV infection.

The TBEV has a special affinity to the gray matter including the anterior horn cells of the spinal cord, and in about 10% of TBE cases, patients develop myeloradiculitic symptoms with progressive flaccid weakness of the limbs and trunk [7,27]. However, only a small number of articles document lesions in the spinal cord. Stich et al. described a case of 43-year-old male with progressive tetraparesis without any sensory deficits. On initial MRI, T2-weighted images showed a hyperintense line in the anterior part of the cervical cord, involving the anterior horns from C3 to T1; no gadolinium-enhancement was detected. Follow-up MRI of the spinal cord, performed 2 weeks later, showed no pathological changes in the same area, suggesting a history of previous inflammation [28]. These findings correspond with other reports. Beer et al. also documented hyperintense, isolated anterior horn lesions on T2-weighted images from C3 to T1 that resolved on follow-up imaging after 6 weeks [29]. A rare case of TBE involving the brainstem and

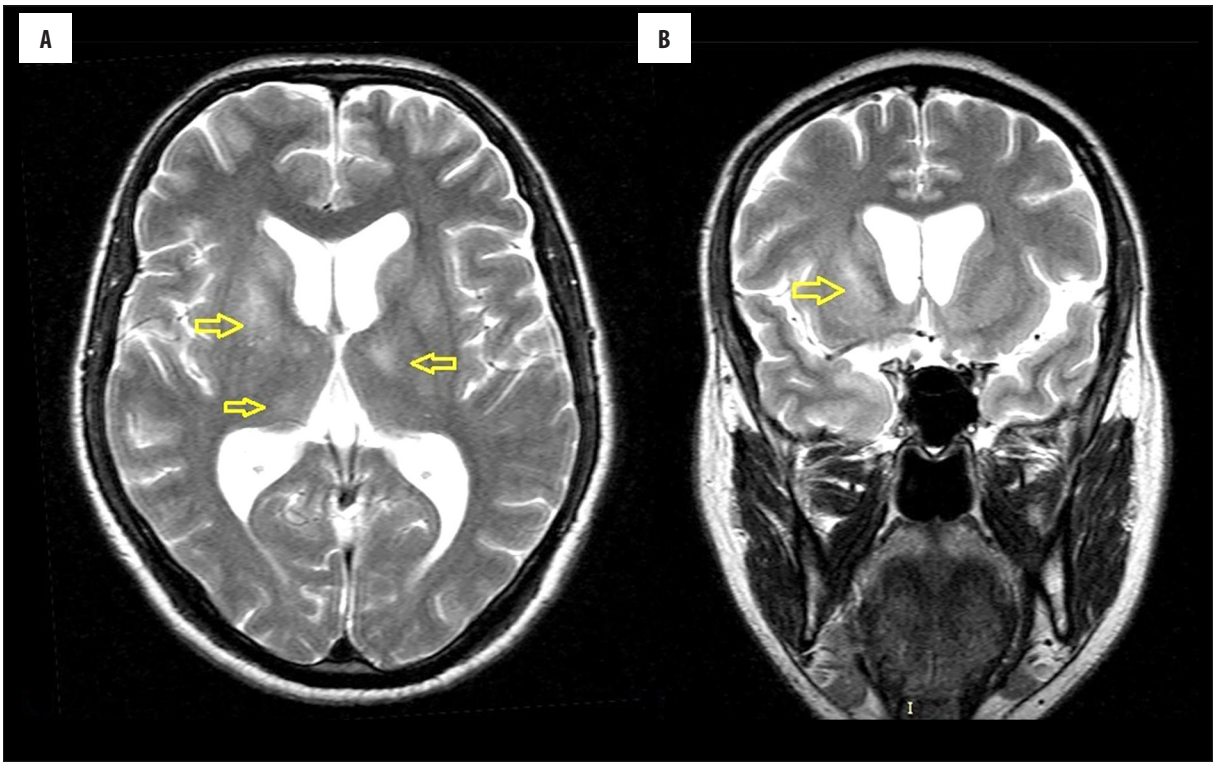

Figure 1. Axial (**A**) and coronal (**B**) T2-weighted MRI images demonstrate bilateral hyperintense lesions within the lenticular nucleus, internal capsule, and thalamus (arrows). MRI examination was performed in TMS Diagnostyka, Medical University of Białystok Clinical Hospital.

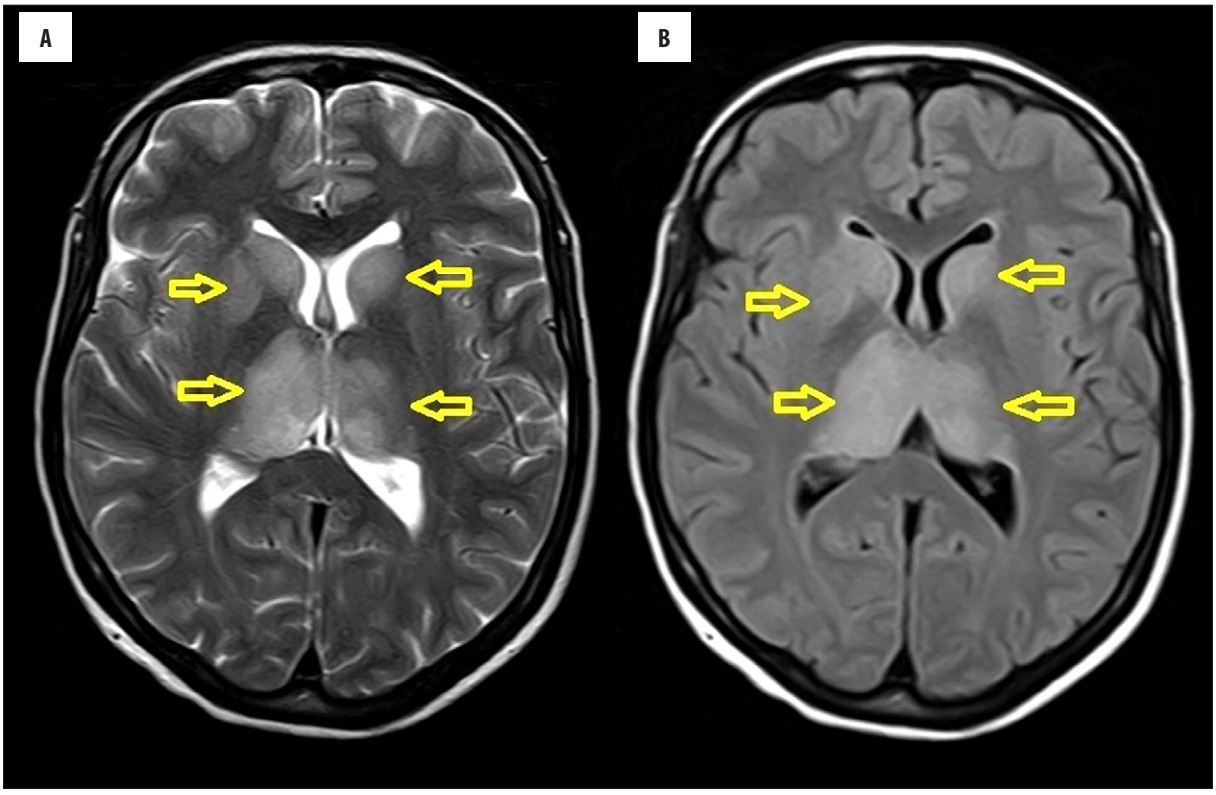

Figure 2. Axial T2-weighted MRI image (**A**) and FLAIR image (**B**) demonstrate bilateral hyperintense lesions within the basal ganglia and the thalamus (arrows). MRI examination was performed in TMS Diagnostyka, Medical University of Białystok Clinical Hospital.

Table 1. Typical topographical mapping of pathological MRI and CT findings in patients with confirmed TBE.

References	Number of cases	Sex/Age	Thalamus	Lesion location															Contrast enhancement
				Lentiform nucleus	Caudate nucleus	Internal capsule	Corpus callosum	Putamen	Frontal lobe	Parietal lobe	Temporal lobe	Occipital lobe	Brainstem	Peduncles	Cerebellum	Spinal cord	Other		
Zajkowska et al. [3]	1	M/38	–	+	+	+	–	–	–	–	–	–	–	–	–	–	–	–	–
Kaiser [7]	18	N/A	+	–	+	–	–	–	–	–	–	–	+	–	+	–	–	–	–
Horger et al. [16]	13	F/22	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	+	
		M/21	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	+	
		M/66	–	–	–	–	–	–	–	+	–	–	–	–	–	–	–	–	
		M/27	–	–	–	–	–	–	–	–	–	–	–	–	–	+	+	–	
		M/48	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	
		M/46	+	–	+	+	–	–	–	–	–	–	–	–	–	–	–	–	
		M/66	–	+	–	–	–	–	–	–			+	–	–	–	+	–	
		M/15	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
		M/9-month-old	–	–	–	–	–	+	–	–	–	–	–	–	–	–	–	–	
		M/13	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	
		M/16	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	
		M/34	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	
		M/66	–	–	–	+	–	+	–	–	–	–	–	–	–	–	+	–	
Marjelund et al. [17]	3	M/21	+	–	–	+	–	+	–	–	–	–	–	–	–	–	–	–	
		F/12	+	–	–	–	–	–	–	–	–	–	+	+	–	–	–	+	
		F/24	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
Waldvogel et al. [18]	1	F/5	+	–	–	–	–	–	–	–	–	–	–	–	–	–	+	+	
Lorenzl et al. [21]	1	F/38	+	–	+	–	–	–	–	–	–	–	–	+	–	–	–	–	
Valdueza et al. [22]	1	M/32	+	–	+	–	–	+	–	–	–	–	+	–	–	–	+	+	
Pfister et al. [23]	1	NA/55	+	–	–	–	–	+	–	–	–	–	–	–	–	–	–	–	
Alkadhi and Kollias [24]	1	F/52	+	–	+	–	–	+	–	–	–	–	–	–	–	–	+	+	
Vollmann et al. [25]	1	M/42	–	–	–	–	+	–	–	–	–	–	–	–	–	–	–	–	
Grimm et al. [26]	1	M/46	–	–	–	–	+	–	–	–	–	–	–	–	–	–	–	–	
Stich et al. [28]	1	M/43	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	
Beer et al. [29]	1	M/44	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	

Table 1. Typical topographical mapping of pathological MRI and CT findings in patients with confirmed TBE.

References	Number of cases	Sex/Age	Thalamus	Lesion location															Contrast enhancement
				Lentiform nucleus	Caudate nucleus	Internal capsule	Corpus callosum	Putamen	Frontal lobe	Parietal lobe	Temporal lobe	Occipital lobe	Brainstem	Peduncles	Cerebellum	Spinal cord	Other		
Bender et al. [30]	1	M/29	+	–	–	–	–	–	–	–	–	–	+	–	–	+	–		+
Marjelund et al. [31]	1	M/48	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–		+
Pfefferkorn et al. [32]	1	M/50	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–		+
Schmolck et al. [33]	3	N/A	+	–	–	–	–	–	–	–	–	–	–	+	–	–	–		–
Iff et al. [34]	1	F/6-week-old	–	–	–	–	–	–	+	+	+	–	–	–	–	–	–		+
Jones et al. [35]	1	M/17-day-old	–	–	–	–	–	–	–	+	+	+	–	–	–	–	–		+

NA – not available.

spinal cord was documented by Bender et al. The authors described a case of a 29-year-old male after a tick bite. Initial head MRI, performed on day 6 since admission, showed typical T2-hyperintense abnormalities bilaterally in the thalamus and basal ganglia. On day 14, the patient developed flaccid tetraparesis, and spinal MRI on day 21 showed increased signal on T2-weighted and T1-weighted contrast-enhanced images within the anterior part of the cervical cord [30]. In another report, *Marjelund* et al. described a case of a patient with TBE, in whom the initial brain and spine MRI was normal, but two weeks after admission, repeat MRI showed an enhancement within the anterior nerve roots at the level of conus medullaris on T1-weighted images after gadolinium administration [31]. Pfefferkorn et al. described a case of TBE with polyradiculitis, in whom T1-weighted gadolinium contrast-enhanced MRI of the lumbar spine, performed 2 weeks after admission, showed marked contrast enhancement of all lumbar roots [32].

Few reports document MRI findings in children with TBE. Schmolck et al. described 19 pediatric patients with TBE, of whom 4 underwent MRI in the acute phase of the disease. One child had a hyperintense unilateral lesion in the right cerebral peduncle on T2-weighted images. Another child had a similar lesion in the right thalamus. Yet another child had bilateral lesions in the thalami, extending anteriorly to the right head of the caudate nucleus and involving the left cerebral peduncle. In that case, follow-up MRI, performed after 5 months, revealed a persistent lesion in the left thalamus. The remaining child had normal MRI [33]. In another report, a 6-week-old infant with tick-borne meningoencephalitis had cortico-subcortical hyperintensities on T2-weigted images and contrast enhancement in both frontal lobes as well as discrete leptomeningeal enhancement [34]. Jones et al., documented a TBE case with edematous cortical and subcortical changes in the left occipitoparietal lobe on T2-weighted images and with contrast enhancement over the left temporal lobe [35].

Of all the TBE cases mentioned in Table 1, the most commonly affected brain region is the thalamus. The most common sequences that revealed pathological lesions are T2-weighted and FLAIR images, although some authors mention that contrast-enhanced T1-weighted images may reveal more morphological changes in the CNS.

Nuclear Medicine Imaging

Günther et al. examined regional cerebral blood flow (rCBF) in 73 patients with aseptic meningoencephalitis due to TBE. rCBF scintigraphy with technetium-99m-hexamethyl propyleneamine oxime (HMPAO) was performed at week 6 with a 1-year follow-up after the onset of the disease. At week 6, 36 of 73 patients with TBE showed disturbed cerebral perfusion, with a significantly decreased rCBF in patients with encephalitis in comparison to patients with meningitis. The authors stated that the reduced rCBF did not appear to depend on the severity of disease in any group. After 1 year, there was no association between remaining symptoms and worsening of rCBF [36]

Differential Diagnosis

Although MRI findings in TBE are similar to other diseases of the CNS, involvement of the thalami, basal ganglia, cerebellum, and the anterior horns of the spinal cord can suggest TBE in an appropriate clinical setting. Differential diagnosis of MRI changes should include toxic brain damage due to carbon monoxide, methanol, and cyanide poisoning, hypoxic ischemic encephalopathy,

Creutzfeldt-Jakob disease, Leigh disease, Wilson disease, osmotic myelinolysis, Wernicke encephalopathy, neurodegeneration with brain iron accumulation, deep cerebral venous thrombosis, arterial occlusion, neuro-Behçet disease, other flavivirus infections (e.g. Japanese encephalitis, West Nile fever, and Murray Valley encephalitis), cerebral toxoplasmosis, primary CNS lymphoma, primary bilateral thalamic glioma, and neurofibromatosis type 1 [37].

Conclusions

The diagnosis of TBE is based mostly on clinical symptoms and specific antibody tests, although imaging methods such as CT and MRI can play an important role in elucidating the pathological basis of symptoms. Many patients show long-lasting symptoms that often affect quality of life, despite successful treatment and absence of macroscopic changes on CT/MRI. Patients with TBE should be considered for advanced MRI techniques like spectroscopy using proton spectroscopic MRI. This technique can show abnormalities in typical areas that seen unaffected by any morphological changes, even after hospitalization.

References:

1. Sumilo D, Bormane A, Asokliene L et al: Tick-borne encephalitis in the Baltic States: Identifying risk factors in space and time. Int J Med Microbiol, 2006; 296: 76–79
2. Bender A, Schulte-Altedorneburg G, Walther EU, Pfister HW: Severe tick borne encephalitis with simultaneous brain stem, bithalamic, and spinal cord involvement documented by MRI. J Neurol Neurosurg Psychiatry, 2005; 76(1): 135–37
3. Zajkowska J, Moniuszko A, Czupryna P et al: Chorea and tick-borne encephalitis, Poland. Emerg Infect Dis, 2013; 19(9): 1544–45
4. Valarcher JF, Hägglund S, Juremalm M et al. Tick-borne encephalitis. Rev Sci Tech, 2015; 34(2): 453–66
5. Zajkowska J, Czupryna P, Pancewicz S et al: Fatal outcome of tick-borne encephalitis – a case series. Neurol Neurochir Pol, 2011; 45: 402–6
6. Czupryna P, Moniuszko A, Pancewicz SA et al: Tick-borne encephalitis in Poland in years 1993–2008 – epidemiology and clinical presentation. A retrospective study of 687 patients. Eur J Neurol, 2011; 18(5): 673–79
7. Kaiser R: The clinical and epidemiological profile of tickborne encephalitis in southern Germany 1994–98. A prospective study of 656 patients. Brain, 1999; 122: 2067–78
8. Haglund M, Gunter G: Tick-borne encephalitis – pathogenesis, clinical course and long term follow up. Vaccine, 2003; 21: 1–18
9. Kaiser R: Tick-borne encephalitis. Infect Dis Clin North Am, 2008; 22(3): 561–75
10. Mickiene A, Laiskonis A, Günther G et al: Tick-borne encephalitis in an area of high endemicity in Lithuania: Disease severity and long-term prognosis. Clin Infect Dis, 2002; 35(6): 650–58
11. Suss J: Tick-borne encephalitis in Europe and beyond – the epidemiological situation as of 2007. Euro Surveill, 2008; 13(26): pii: 18916
12. Gray JS: Ixodes ricinus seasonal activity: Implications of global warming indicated by revisiting tick and weather data. Int J Med Microbiol, 2008; 298(1): 19–24
13. Broker M, Gniel D: New foci of tick-borne encephalitis virus in Europe: Consequences for travelers from abroad. Travel Med Infect Dis, 2003; 1(3): 181–84
14. Holzmann H: Diagnosis of tick-borne encephalitis. Vaccine, 2003; 1(21): 36–40
15. Gelpi E, Preusser M, Garzuly F et al: Visualization of Central European tick-borne encephalitis infection in fatal human cases. J Neuropathol Exp Neurol, 2005; 64(6): 506–12
16. Horger M, Beck R, Fenchel M et al: Imaging findings in tick-borne encephalitis with differential diagnostic considerations. Am J Roentgenol, 2012; 199(2): 420–27
17. Marjelund S, Tikkakoski T, Tuisku S, Räisänen S: Magnetic resonance imaging findings and outcome in severe tick-borne encephalitis. Report of four cases and review of the literature. Acta Radiologica, 2004; 45(1): 88–94
18. Waldvogel K, Bossart W, Huisman T et al: Severe tick-borne encephalitis following passive immunization. Eur J Pediatr, 1996; 155(9): 775–79
19. Gritsun TS, Nuttall PA, Gould EA: Tick-borne flaviviruses. Adv Virus Res, 2003; 61: 317–71
20. Gelpi E, Preusser M, Laggner U et al: Inflammatory response in human tick-borne encephalitis: Analysis of postmortem brain tissue. J Neurovirol, 2006; 12(4): 322–27
21. Lorenzl S, Pfister HW, Padovan C, Yousry T: MRI abnormalities in tick-borne encephalitis. Lancet, 1996; 347(9002): 698-99
22. Valdueza JM, Weber JR, Harms L Bock A: Severe tick borne encephalomyelitis after tick bite and passive immunisation. J Neurol Neurosurg Psychiatry, 1996; 60(5): 593–94
23. Pfister HW, Lorenzl S, Yousry T: Neuroradiographic manifestations of encephalitis. N Engl J Med, 1997; 337(19): 1393–94
24. Alkadhi H, Kollias SS: MRI in tick-borne encephalitis. Neuroradiology, 2000; 42(10): 753–55
25. Vollmann H, Hagemann G, Hans-Joachim Mentzel HJ et al: Isolated reversible splenial lesion in tick-borne encephalitis: A case report and literature review. Clin Neurol Neurosurg, 2011; 113(5): 430–33
26. Grimm T, Barinka F, Uhl M et al: Reversible splenial lesion syndrome (Resles) in a patient with clinically mild tick-borne encephalitis and hyponatremia. J Neuroinfect Dis, 2015;6: 4
27. Schellinger PD, Schmutzhard E et al: Poliomyelitic-like illness in central European encephalitis. Neurology, 2000; 55(2): 299–302
28. Stich O, Reinhard M, Rauer S: MRI scans of cervical cord provide evidence of anterior horn lesion in a patient with tick-borne encephalomyeloradiculitis. Eur J Neurol, 2007; 14(6): 5–6
29. Beer S, Brune N, Kesselring J: Detection of anterior horn lesions by MRI in central European tick-borne encephalomyelitis. J Neurol, 1999; 246(12): 1169–71
30. Bender A, Schulte-Altedorneburg G, Walther EU, Pfister HW: Severe tick borne encephalitis with simultaneous brain stem, bithalamic, and spinal cord involvement documented by MRI. J Neurol Neurosurg Psychiatry, 2005; 76(1): 135–37
31. Marjelund S, Jaaskelainen A, Tikkakoski T et al: Gadolinium enhancement of cauda equina: A new MR imaging finding in the radiculitic form of tick-borne encephalitis. Am J Neuroradiol, 2006; 27(5): 995–97
32. Pfefferkorn T, Feddersen B, Schulte-Altedorneburg G et al: Tick-borne encephalitis with polyradiculitis documented by MRI. Neurology, 2007; 68(15): 1232–33
33. Schmolck H, Maritz E, Kletzin I, Korinthenberg R: Neurologic, neuropsychologic, and electroencephalographic findings after European tick-borne encephalitis in children. J Child Neurol, 2005; 20(6): 500–8
34. Iff T, Meier R, Olah E et al: Tick-borne meningo-encephalitis in a 6-week-old infant. Eur J Pediatr, 2005; 164(12): 787–88
35. Jones N, Sperl W, Koch J et al: Tick-borne encephalitis in a 17-day-old newborn resulting in severe neurologic impairment. Pediatr Infect Dis J, 2007; 26(2): 185–86
36. Gunther G, Haglund M, Mesko L: Regional cerebral blood flow scintigraphy in tickborne encephalitis and other aseptic meningoencephalitis. Journal of Nuclear Medicine, 1998; 39(12): 2055–61
37. Hegde AN, Mohan S, Lath N, Tchoyoson Lim CC: Differential diagnosis for bilateral abnormalities of the basal ganglia and thalamus. Radiographics, 2011; 31(1): 5–31