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LYME NEUROBORRELIOSIS

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Key-word: Lyme disease

Background: A 24-year-old woman presented to the emergency department with headache, apathy, aphasia and mild paresis of the right leg. Routine laboratory results revealed slightly raised infectious parameters.
CT scan of the brain was requested. Non-enhanced CT scan of the brain demonstrated a large low density lesion with irregular borders in the left thalamus. There was no enhancement of the lesion after iodine contrast administration (not shown). CT findings prompted immediate MRI.Repeated anamnesis revealed that the patient had been in contact with ticks three years previously. There was no recollection of skin manifestations such as erythema chronicum migrans at that time.

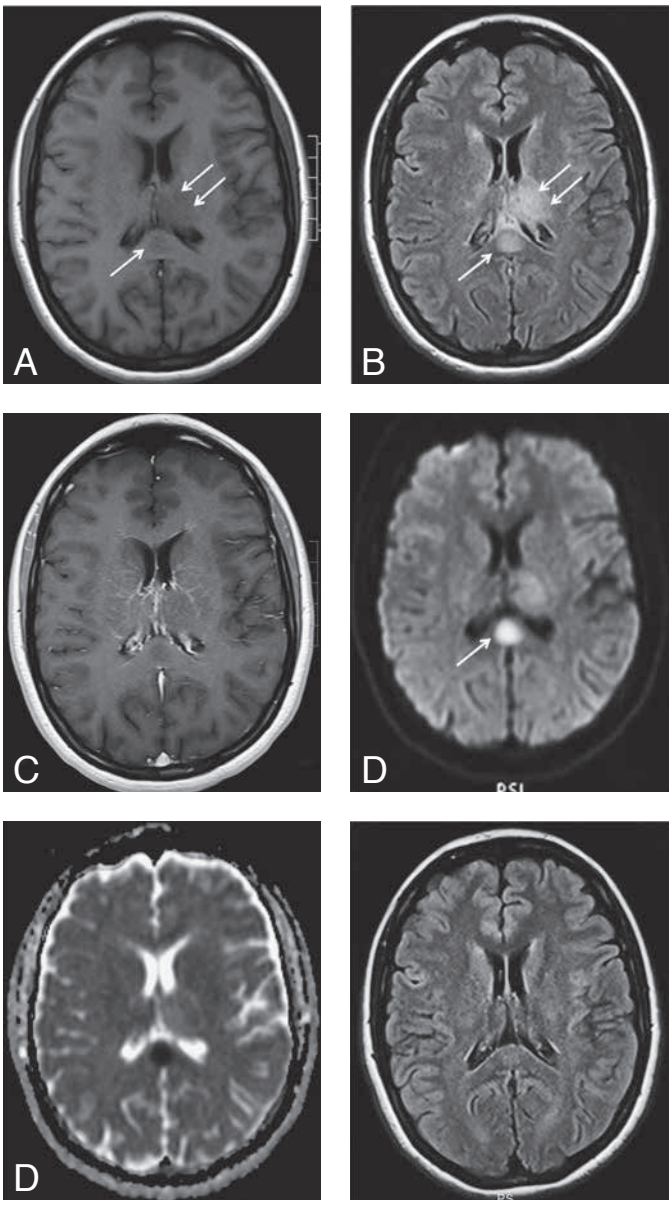

Fig. 1A 1B / 1C 1D / 1E 2

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Work-up

On MRI of the brain on admission (Fig. 1), axial T1-weighted image (A), a slightly hypointense lesion compared to the white matter is seen within the left thalamus (double arrows). A second hypointense lesion in the splenium of the corpus callosum is also demonstrated (arrow).

On axial FLAIR image (B), both lesions are hyperintense compared to the surrounding perilesional white matter.On Gd-enhanced axial T1-weighted image (C), none of the lesions show enhancement. On diffusion weighted image (DWI) (D) and corresponding apparent diffusion coefficient (ADC) map (E), the lesion within the splenic portion of the corpus callosum shows diffusion restriction (arrow) but the thalamic lesion does not.

MRI of the brain, axial FLAIR image after treatment (Fig. 2), resolution of both thalamic and corpus callosum lesions is noted.

Radiological diagnosis

Because of the anamnestic finding of contact with ticks three years ago, analysis of serum and cerebrospinal fluid (CSF) was performed and revealed raised IgM and IgG Borrelia-antibodies. Subsequent enzyme linked immunosorbent assay (ELISA) and Western blot analysis were also positive.

Based on the combination of clinical, laboratory and imaging findings, the diagnosis of *Lyme neuroborreliosis* was made and the patient was treated with intravenous antibiotics. Follow-up MRI six weeks later after antibiotic treatment showed complete resolution of the lesions (Fig. 2).

Discussion

Lyme borreliosis is an insidious multisystem inflammatory disease caused by the spirochete Borrelia garinii and Borrelia afzelli in Europe transmitted to humans through the bite of infected ticks. There are three different clinical stages which are classically described as *early localized, early disseminated* and *late*. In the early stage fever, myalgias, a petechial rash and neurological symptoms such as headache are the main features. The *early disseminated* stage comprises erythema chronicum migrans, arthralgias and adenopathies. Arthritis, carditis, scleroderma-like lesions, central and peripheral nervous system abnormalities including cranial nerve palsies, white matter and spinal cord involvement are typical for the *late stage*.

Typical CT findings of Lyme neuroborreliosis are small hypodense periventricular lesions. There is no or only faint enhancement after intravenous iodine administration. On MRI, these lesions are typically less than 3 mm, hypointense on T1-WI and hyperintense on T2-WI and FLAIR. T1-WI after intravenous gadolinium contrast administration variably shows faint enhancement of white matter lesions or the meninges. Third, fifth and seventh cranial nerve enhancement has been reported but the seventh cranial nerve is affected most frequently. Less frequent imaging characteristics are the presence of larger lesions, corpus callosum involvement and diffusion restriction of some lesions, as demonstrated in our case. The diffusion restriction in the corpus callosum lesions can be explained by the presence of intramyelinic and/or cytotoxic edema. Follow-up MRI is essential for therapy monitoring. If the correct treatment is implemented, resolution of the lesions on T2-WI, FLAIR and DWI can be demonstrated.

The main differential diagnoses of intra-axial hyperintense lesions on T2-WI and FLAIR include a tumoral process, multiple sclerosis and neurosarcoidosis. A solitary peripheral enhancing white matter mass with central necrosis, mass effect and edema would be more typical for a tumor. Multiple sclerosis shows predilection of the lesions for the callososeptal region and enhancement after contrast administration in case of active inflammation. In neurosarcoidosis the lesions are typically located in the perivascular space and show more prominent enhancement.

Excluding tumoral pathology is essential to avoid unnecessary biopsy procedures. The combination of initial and follow-up imaging studies, clinical data together with laboratory findings is essential and guides therapy.

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