

Asymptomatic brainstem hypertensive encephalopathy (HBE) in a multiple sclerosis patient

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Case description: a 47 years old woman affected with multiple sclerosis (MS) since 2007, treated with interferon beta 1 a 44 mcg tiw, on a routine MRI showed bilateral diffuse, non-enhancing, T2 wheighted/FLAIR MRI hyperintensities in the brainstem, putamen and cerebellar folia with swelling of the pons and diffuse micro bleeds in the brainstem, cerebellar hemispheres and lenticular nuclei. The patient was asymptomatic. MS course was stable since onset.

Measures taken: i.v. steroids ex-juvantibus, in order to assess any subsequent MRI variation. Infusion was prematurely interrupted due to intolerance. But...

Differential diagnosis: inflammatory-(lymphoproliferative infiltrative disease disease, Wilson's disease, celiac disease, diabetes, metabolic encephalopathy)

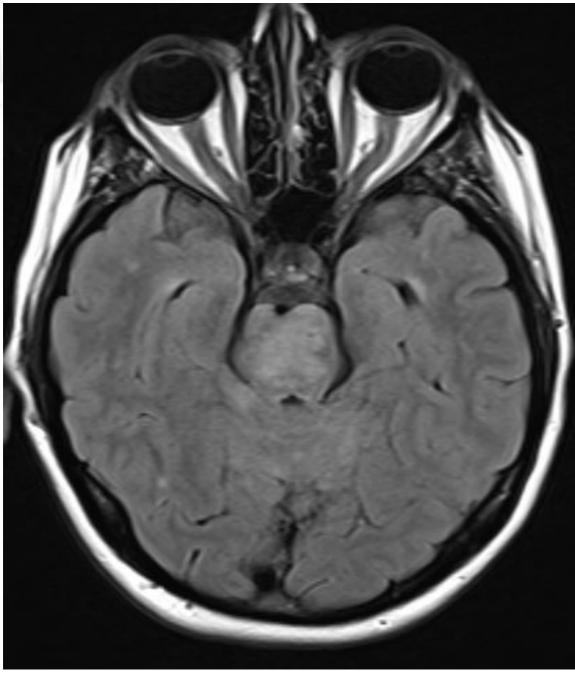
Investigations:

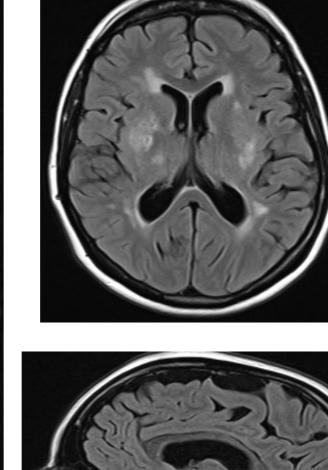
- celiac disease, thyroid function, complete blood count, immunophenotyping, serum copper, ceruloplasmin level, blood serum glucose, glycosylated haemoglobin level, erythrocyte sedimentation rate, clotting tests, kidney and liver function: normal.
- Cerebrospinal fluid: Albumin index 11.4, Link index 1.1, positive oligoclonal bands.
- Contrast-CT-scan of thorax, abdomen and pelvis:



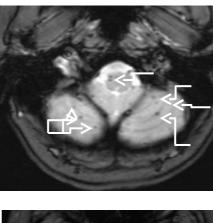
Severe hypertension was incidentally found

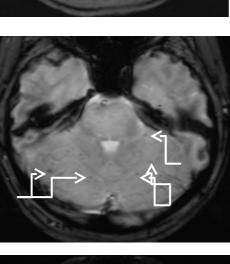


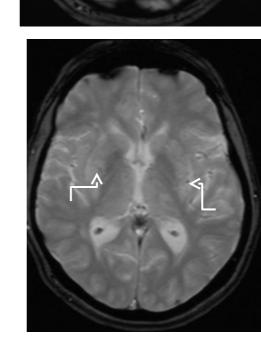




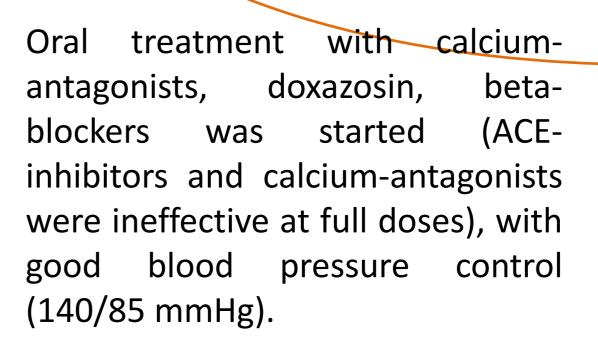
FLAIR





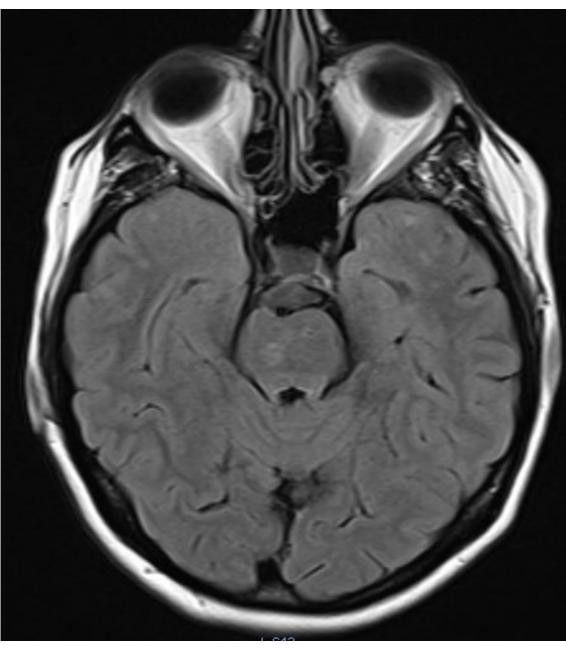


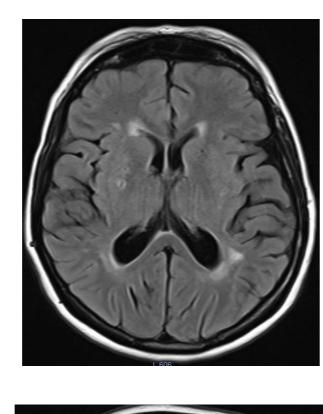
T2 Gradient-Echo Hemo (microbleeds-arrows)





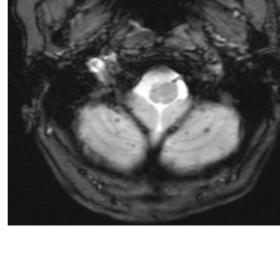


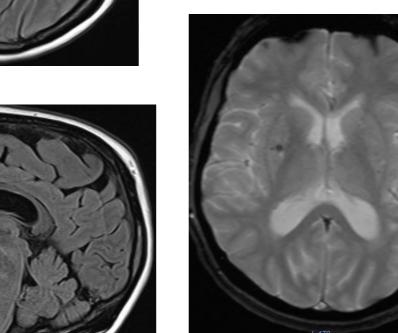




FLAIR

FLAIR



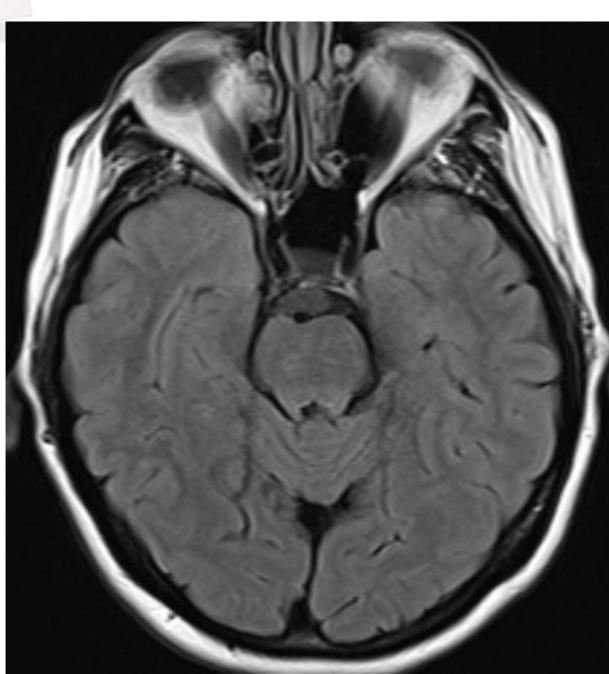


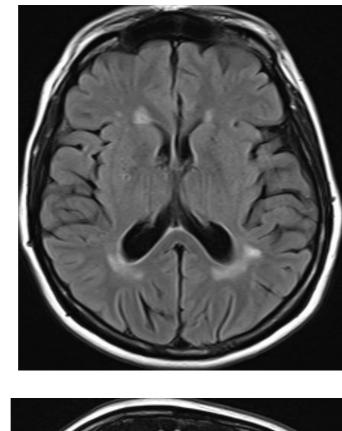
T2 GE Hemo

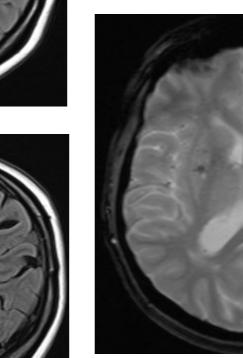
MRI ameliorated: picture progressive shrinkage of the hyperintensity areas, disappearance of pons swelling but persistence of micro bleeds. Anti hypertensive treatment continued. Secondary hypertension was ruled out.

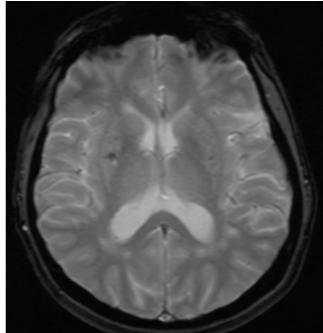












T2 GE Hemo

The exclusion of inflammatory, infiltrative and metabolic diseases and the remarkable improvement of imaging during anti hypertensive treatment suggest the diagnostic hypothesis of reversible brainstem hypertensive encephalopathy. To date only symptomatic cases without other neurological diseases have been published. The pathophysiological hypothesis are related to endothelium over-relaxation, local production of antifibrinolytic and inflammatory molecules, fluid extravasation and cytotoxic oedema