CASE 2013-3

Submitted by:

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Clinical History:

The patient is a 60-year-old man who presented to an outside hospital after one day of worsening confusion and memory deficits. Past medical history was significant for coronary artery disease, myocardial infarction, and hypertension. On physical exam he appeared drowsy and was somewhat uncooperative, but was oriented to person, year, and location. Neurological exam was significant for saccadic intrusions of ocular pursuits, a right pronator drift, and mild bradykinesia. Computed tomography of the head showed thalamic abnormalities; the differential diagnosis included Wernicke’s encephalopathy and carbon monoxide poisoning. The patient acknowledged drinking 8 or more drinks per day, but denied risks for exposure to carbon monoxide. Whether he received corticosteroids or thiamine at the outside hospital was unclear. After three days without mental status improvement, he was transferred to our institution, where thiamine was administered. Magnetic resonance imaging (MRI) showed compression of the third ventricle, bilateral thalamic lesions with T2 hyperintensity and homogenous enhancement, and scattered nonspecific T2 hyperintensities throughout the cortex; radiological differential diagnosis included lymphoma, glioma, encephalitis, Wernicke’s encephalopathy, and carbon monoxide poisoning. Magnetic resonance angiogram (MRA) and venogram were both read as negative. Positron emission tomography (PET) showed mild asymmetric hypermetabolism, considered to be consistent with lymphoma or high grade glioma. EEG showed left frontal lobe sharply contoured slowing. CBC showed mild leukocytosis. CSF showed elevated protein, very few cells and no evidence of malignancy; evaluation for organisms (viral, fungal, and bacterial) and autoimmune and paraneoplastic antibodies were negative. Stereotactic needle biopsy (of the right thalamus) was performed for diagnosis.

Material submitted:
One H&E scanned slide

Points for discussion:
1. Diagnosis
2. Pathogenesis