

CASE 1994-3

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A 40 year old male presented to another hospital with disorientation, difficulty speaking and fevers. He was found to be HIV seropositive 2 months earlier when evaluated for shingles in a T6 dermatome distribution. He denied any risk factors for HIV infection. Additional clinical history included hepatitis B, peripheral neuropathy and legionella pneumonia. Admitting neurological examination was unremarkable. On admission he was treated for pneumonia. Sputum cultures grew *Pseudomonas aeruginosa*, but were negative for legionella. Liver function tests were found to be abnormally high. His T4 count was 4 (normal range 370-1482). He suffered a seizure and was placed on phenobarbital. His serum and CSF toxoplasma titers were negative. CSF VDRL was negative and no cryptococcal organisms were identified. MRI scan showed a focal lesion in the left basal ganglia consistent with infarction. He was discharged after 9 days, however, his neurological symptoms progressed and he developed fever and lower extremity weakness. He was unable to stand on his own and was admitted to the Medical Center. Admitting physical examination showed dysphasia, but was otherwise unremarkable and there were no focal neurological signs. CT scan of the head was unremarkable (the MRI scan from his initial admission was not available for comparison). Phenobarbital levels were not elevated. His respiratory function worsened and he expired that evening.

Systemic autopsy revealed severe pulmonary edema, chronic severe adrenalitis and splenomegaly. The fresh brain weighed 1500 gm and showed no external abnormalities. Coronal sections showed a lesion in the left putamen and globus pallidus. Microscopic examination of sections of the brain showed subacute lesions in the left basal ganglia, thalamus and mamillary bodies with mild perivascular lymphocytic infiltrate. Similar microscopic lesions were present in the right basal ganglia. There were scattered microglial nodules in the basal ganglia and medulla.

Enclosed is an H&E stained glass slide of one of the lesions and a kodachrome slide of a Bielschowsky stain of a similar lesion.

Points for discussion:

1. What is the pathogenesis of these lesions?
2. What further studies might be done to prove the diagnosis?