This 50-year-old African American woman had a past medical history of hypertension and Type II diabetes mellitus with retinopathy, neuropathy and a neurogenic bladder. She presented with difficulty ambulating in April 2004 and was admitted to the hospital. She also described tremor, occasional falls, intermittent slurred speech and periodic posterior neck pain with radiation to the temporal regions. Imaging studies at that time revealed diffuse leptomeningeal enhancement of the central nervous system, including the spinal cord. Lumbar puncture did not reveal an infectious etiology for this enhancement. She was treated with a course of steroids, and a follow-up MRI showed some improvement in the enhancement.

She was readmitted in July 2004 secondary to urinary retention and progressive lower extremity weakness. Imaging studies revealed continued abnormal leptomeningeal enhancement affecting the lumbosacral nerve roots, as well as a lacunar infarction in the left basal ganglia. Abdominal and chest CT scans were negative and ACE (serum and CSF) levels were normal. Electromyography showed bilateral lumbosacral polyradiculopathy as well as sensory motor neuropathy. Biopsy of the brain was suggested, but the patient refused the procedure. Her ambulation was improved with a course of intravenous methylprednisolone.

The patient’s final admission was in December 2004 due to a two day history of lethargy, speech difficulty and confusion. She also complained of headache and dysphagia. MRI scans revealed new lacunar infarctions in the subcortical white matter but was otherwise unchanged. CSF Gram stains and cultures were negative and a urinary tract infection was treated with ciprofloxacin. She had a slowly declining neurological course with progressive lethargy and obtundation in the weeks prior to her death at home. An unlimited autopsy was requested by the family.

General autopsy revealed the immediate cause of death to be bilateral acute pulmonary thromboembolism. The fixed brain weighed 1410 grams and the leptomeninges were thickened and cloudy. The lacunar infarcts were confirmed, in the left globus pallidus, the left putamen and the left corona radiata.

Material submitted: 1 H&E-stained section of neocortex or hippocampus or midbrain
1 unstained section of neocortex or hippocampus or midbrain

Point for discussion: Significance of biopsy for diagnosis