Weakness in a Young Man

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A 39 year-old African-American male with past medical history significant for uncontrolled hypertension presents with a sudden onset of weakness and numbness on the left side of his face, left arm, left leg, and right leg while watching television twenty hours prior to presentation. At that time, the patient did have a mild headache. The patient initially thought that it was positional, but soon realized that he could not move the affected extremities at all. He also began to have some difficulty swallowing and minor difficulties with speech as well.

Two hours after the initial onset of symptoms, the patient was able to ambulate without difficulty. Shortly afterwards, he regained function and sensation in his left upper extremity. His dysarthria and dysphagia also resolved but he had a persistent drooping of his left lip with numbness on the left side of his face.

On review of systems, he admitted that he had run out of medications 3 days prior to presentation. He had also noted a rash on the lateral portion of his left upper extremity but he did not recall when it started. The rash was pruritic for 2 days and then resolved without further treatment. The patient denied fevers, chills, neck pain, visual changes, diplopia, tinnitus, dizziness, and lightheadness. The patient had no urinary or bowel incontinence.

His past medical history is significant for uncontrolled hypertension with multiple visits to the emergency department; atypical chest pain, with a normal stress test 2 years prior to admission; gastroesophageal reflux and hiatal hernia; hepatitis A; a positive PPD, treated with a full course of prophylactic isoniazid; superficial perivascular lymphocytic dermatitis diagnosed in 2000, currently stable; and condyloma acuminatum, excised by urology five months prior.

His medications included atenolol 100 mg twice daily, clonidine 0.1 mg twice daily, nifedipine (SA) 90 mg daily, enalapril 20 mg daily, rabeprazole 20 mg daily, sertraline 50 mg daily, and hydrocortisone cream twice daily as needed. He had a syncopal reaction to loratadine. The patient does not drink alcohol. He has a positive smoking history but he quit in 1999. He has never used illicit drugs. He was a former prison guard, served in the Navy in the 1991 Persian Gulf War and is currently active in the US Army. His father died in his 50s of prostate cancer.

Physical examination revealed a 39 year-old male in no acute distress with an initial blood pressure of 204/106. Vital signs were otherwise stable. The patient had decreased sensation to pinprick and dull pressure on his left face. The patient also had weakened muscles of mastication on his left side and flattened left nasolabial folds. His strength, sensation, and reflexes in his extremities were all within normal limits. He had normal Babinski reflexes bilaterally with normal gait. He also had a hyperpigmented rash noted on his left upper extremity and scars on the lateral aspect of the left neck and on his right thigh from old stab injuries. His cardiovascular, pulmonary, abdominal, and extremity examinations were benign. Fundoscopic examination revealed changes attributable to hypertension but no papilledema. His main facial findings are shown in Figure 1. (Permission was granted by the patient prior to the picture being taken; this is documented with the paper chart from this admission).

Laboratory data was significant for a slight hypokalemia (3.3mmol/L), elevated glucose (120mg/dL), an elevated ALT (205U/L), and a normal complete blood count. A CT of the head without contrast was obtained showing no acute bleed or pathology. Initial ECG (Figure 2) shows sinus rhythm with left ventricular hypertrophy and left-sided/lateral strain.

He was initially admitted to the intensive care unit with a presumptive diagnosis of transient ischemic attack secondary to the presence of significant hypertension. Appropriate measures were taken to reduce systolic pressure. A neurology consult was obtained the next morning that discovered that much of his recent Army training had been in wooded areas. Although the possibility of hypertensive stroke/TIA could not be ignored, Lyme neuropathy and neurosarcoidosis were now on the differential due to this new information. Serum Lyme titers, as well as a lumbar puncture with CSF Lyme titers and angiotensin-converting enzyme, were recommended.

Figure 1

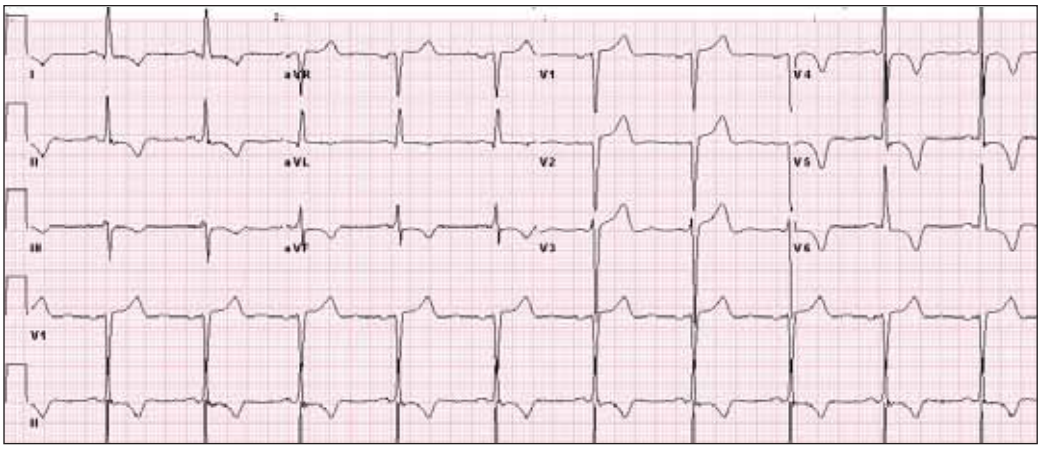

Figure 2

Serum Lyme titers were negative. A lumbar puncture showed colorless, clear fluid with 6 RBCs, 2 WBCs (both neutrophils), glucose 63mmol/L, and protein 5mg/L. Gram stain and culture were negative for cells or organisms. Titers for VDRL and HSV were both negative. An ACE-I level from the cerebrospinal fluid was within normal limits. However, Lyme titers from the cerebrospinal fluid returned positive at 1:52 (nml 1:10). An MRI was performed which demonstrated mild enhancement near the location of the left seventh nerve.

The patient had been started on ceftriaxone because it took several days for the Lyme titers from the cerebrospinal fluid to return. His facial signs slowly improved and eventually resolved over the next few weeks.

Neuroborreliosis is only seen in approximately 10 percent of untreated Lyme disease. Clinical presentations are variable but there are distinct time periods during which they arise. Aseptic meningitis, cranial nerve palsy (usually involving the facial nerve), and radiculoneuritis (severe localized radicular pain or motor weakness, with variable sensory deficits) usually occur during early disseminated disease (1-3 months). Aseptic meningitis and peripheral neuropathies may also occur with reactivated disease. Headache and neck stiffness are usually mild. Asymmetric dermatomal and myotomal abnormalities, also known as lymphocytic meningoradiculitis or Bannwarth's syndrome, can be seen. These cases are usually seen in Europe, although rare cases can be seen in North America. Encephalopathy and chronic encephalomyelitis are usually manifestations of late persistent infection (> 3 months). Neuroborreliosis is treated effectively with one month of intravenous ceftriaxone. Oral doxycycline is an option in isolated facial nerve palsy, but due to the severity of cerebrospinal fluid inflammation, intravenous antibiotics are usually preferred.

References

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