

## Cerebral Syphilitic Gummata in an HIV-infected Patient

Learning Objectives: 1) Recognize syphilitic gummata as a cause of central nervous system mass lesions, even in patients infected with HIV; 2) recognize that, as with neurosyphilis, syphilitic cerebral gummata are treated with intravenous penicillin; and 3) know that cerebrospinal fluid analysis can be normal in patients with syphilitic cerebral gummata.

Case: A 39-year-old male with newly diagnosed HIV-infection presented to the emergency department after three weeks of progressive right arm and leg weakness and 2 days of right vision loss. Prior to his presentation at the emergency department, he had recent neuroimaging at an outside facility for these complaints which demonstrated multiple “brain masses” per the patient. His initial physical exam was notable for 4/5 right upper extremity strength, 3/5 right lower extremity strength, normal left-sided strength, and decreased right eye visual acuity. Initial laboratory analysis demonstrated a positive HIV antibody with CD4 count of 236/mm<sup>3</sup> and HIV viral load of 2,520 copies/mL; his rapid plasma reagin and *Treponema pallidum* antibody were also positive. Cerebrospinal fluid (CSF) analysis showed normal glucose and protein, no white blood cells, and negative venereal disease research laboratory test (VDRL), *Toxoplasma gondii* IgG antibody, and John Cunningham (JC) virus. Magnetic resonance imaging of the brain showed heterogeneous ring-enhancing lesions with restricted diffusion within both cerebral hemispheres and suggested a demyelinating process affecting the right optic nerve. He was initially treated for toxoplasmosis but continued to worsen on this therapy, and empiric dexamethasone was started. A brain biopsy was performed with final pathology showing a demyelinating process, perivascular infiltration of lymphocytes and plasma cells, and structures appearing to be spirochetes, suggesting a diagnosis of cerebral syphilitic gummata. He was started on intravenous penicillin G and continued on dexamethasone, and therapy for toxoplasmosis was discontinued. With treatment for neurosyphilis and cerebral gummata, his prior strength deficits improved drastically, and a repeat MRI after therapy showed improvement in his brain lesions.

Discussion: Cerebral syphilitic gummata are a unique presentation of neurosyphilis, and a rare cause of brain masses in HIV-infected patients. The differential diagnosis for patients with HIV infection and brain masses is broad and includes a number of infectious and neoplastic entities such as toxoplasmosis, primary central nervous system (CNS) lymphoma, progressive multifocal leukoencephalopathy (PML), bacterial abscesses, and infrequently, syphilitic gummata. Distinct from neurosyphilis, CNS gummata can cause a variety of focal neurologic symptoms such as seizures, weakness, altered mental status, and headaches. This patient’s CSF studies were unremarkable and CSF VDRL for syphilis was negative. The patient’s brain biopsy was highly suggestive of the final diagnosis. We excluded all other infectious and neoplastic etiologies and our patient improved with targeted therapy for syphilis. Current recommendations are to treat both neurosyphilis and cerebral syphilitic gummata intravenous penicillin G for 14 days, regardless of HIV-positivity. In an immunocompromised patient with characteristic brain masses, cerebral gummata should be considered, and a negative CSF VDRL in HIV patients does not always exclude syphilitic infection.