of communication during clinical care; diversification of the clinical staff; optimization of the receipt of care and HAART; and enhancement of adherence to treatment. All of the preceding strategies are good ways to reduce the negative impact of HIV infection and AIDS on minorities and to “improve the likelihood that minority patients will engage in care, be satisfied with care, and have positive HIV/AIDS-related outcomes” [1, p. 403].

Stone did not mention another potentially culturally sensitive mechanism for improving the care of minorities with HIV infection and AIDS, which is utilization of routine testing for HIV instead of risk-based testing. Routine testing for HIV in the primary care setting should help to decrease the social stigma associated with screening for HIV and AIDS by the equitable offering of the test to all sexually active patients. This form of testing still requires the patient’s consent but does not require the health care provider to conduct an intrusive inquiry into the patient’s social or sexual history prior to testing. Often, patients, health care providers, or both feel uncomfortable when probing or being probed for sexual and social histories [2, 3]. A recent study demonstrated that patients from underserved minority populations and from the majority population have indicated their willingness to undergo routine testing [4]. Several studies have found that those at risk from heterosexual contact underestimate their risk for HIV-infection and frequently present to a clinic later in the course of the infection [5, 6]. Routine testing has helped to identify HIV infections earlier in the course of the infection, has helped to identify those who are unaware of their HIV infection status [7, 8], and may increase overall awareness of people in both the heterosexual and the same-sex risk communities who underestimate their risk for the disease. Routine testing has been shown to be acceptable to prenatal patients and their health care providers. More importantly, this form of testing may reduce the stigma currently associated with HIV testing, which contributes to the late diagnosis of HIV infection. In one study, >40% of patients received a diagnosis of AIDS within 1 year after diagnosis of HIV infection [9].

HIV/AIDS is not the same disease that it was at the beginning of the epidemic. The epidemiology of this disease has changed; the disease is now a chronic instead of a fatal illness. The medical profession needs to respond appropriately to this evolved epidemic. Routine testing may represent one of the most culturally sensitive ways for providers both to communicate to patients the importance of screening for this preventable disease and to destigmatize HIV testing. We hope this method will increase the practice and acceptability of HIV testing, the timeliness of diagnosis, and the number of opportunities for all patients, including underserved minorities, to receive care in a timely but nonjudgmental manner.

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The Diagnostic Usefulness of Cerebrospinal Fluid Lactic Acid Levels in Central Nervous System Infections

Sir—I read with interest the recent article by Dr. Davis and colleagues [1] regarding decreased CSF glucose levels in patients with herpes simplex virus (HSV) encephalitis. The authors’ patients, who had HSV-1 encephalitis, also had decreased CSF glucose levels. In my experience, decreased CSF glucose levels in HSV-1 encephalitis are neither uncommon nor universal. The authors state that low CSF glucose levels are predictive of CNS bacterial infections because CNS viral infections are associated with normal CSF glucose levels [1].

Of course, other nonbacterial CNS infections are associated with low CSF glucose levels: for example, fungal infections, tuberculosis, amebic infections, brain abscess with ventricular leak, and parameningeal infections. The report by Davis et al. [1] should prompt clinicians to review the differential diagnostic possibilities of decreased CSF glucose levels in CNS viral infections. Nonviral infections that may present with mental confusion—for example, mycoplasma meningoencephalitis, neuroborreliosis, legionnaires’ disease, rickettsial infection, and encephalopathy
due to noninfectious disorders, such as hepatic encephalopathy and drug-induced meningitis/encephalitis, among others—
are not associated with decreased CSF glucose levels [2–4].

Among the viral infections that present as aseptic meningitis, meningoencephalitis, or encephalitis, a decreased CSF glucose level may occur in CNS infections due to mumps virus, enteroviruses, and lymphocytic choriomeningitis virus, in addition to HSV-1 and viral infections presenting as aseptic meningitis, meningoencephalitis, or encephalitis. A decreased CSF glucose level is not a feature of arboviral encephalitis, including West Nile encephalitis [3].

CSF lactic acid levels are a useful way to determine the etiology of decreased CSF glucose levels by differentiating bacterial versus viral CNS infections [4–8]. In patients with meningitis/encephalitis, CSF lactic acid levels are an underused modality with diagnostic significance. CSF lactic acid concentrations can be determined within minutes, whereas results of PCR for HSV-1 usually take several days to be reported. Because early empiric treatment for bacterial infections and HSV-1 encephalitis is different, an early presumptive diagnosis is crucial, pending definitive HSV-1 PCR test results. The main diagnostic use of CSF lactic acid levels is rapid differentiation of viral meningitis/encephalitis (characterized by levels of ≤3 mmol/L) and partially treated viral meningitis (with levels of 3–6 mmol/L) from bacterial CNS infections (with levels of ≥6 mmol/L). For this purpose, determination of CSF lactic acid levels is without equal for rapid and inexpensive differentiation between these clinical entities [2, 3].

If CSF glucose levels are decreased, and fungal and tubercular meningitis are excluded as possibilities, the differential diagnosis is often between HSV-1 encephalitis and bacterial CNS infection. RBCs in the CSF not due to trauma and/or hemorrhage should suggest the possibility of HSV-1 encephalitis. RBCs in the CSF are not part of the clinical picture of bacterial CNS infection. Obtaining an electroencephalogram (EEG) is useful early in the diagnosis of HSV-1 encephalitis. In HSV-1 encephalitis, the EEG characteristically shows a localized temporo-parietal focus, in contrast to other causes of viral encephalitis that demonstrate bilateral diffuse hemispheric EEG abnormalities.

Levels of CSF lactic acid may be used to differentiate CNS infections with decreased CSF glucose levels from HSV-1 encephalitis. CSF lactic acid levels are highly elevated in bacterial meningitis (>6 mmol/L) but are not elevated in HSV-1 encephalitis (<3 mmol/L) or other CNS viral infections.

The number of RBCs in the CSF in HSV-1 encephalitis is directly proportional to the degree of parenchymal damage/hemorrhage. Metabolism of RBCs in the CSF produces lactic acid, which may increase lactic acid levels slightly. However, CSF lactic acid levels in bacterial CNS infections are highly elevated (6–20 mmol/L), even in the presence of RBCs in the CSF, and should not be confused with the minimal elevations seen in HSV-1 encephalitis with RBCs in the CSF (~2–4 mmol/L) [2, 3, 9].

Viral CNS infections that may present with low CSF glucose levels include mumps, enteroviral infections, lymphocytic choriomeningitis infection, and HSV infection [9, 10]. Low CSF glucose levels may be present with a variety of noninfectious disorders, including, for example, sarcoid meningitis, carcinomatous meningitis, and posterior fossa syndrome [3]. It has been said that CSF lactic acid levels do not offer a diagnostic advantage over CSF glucose levels for the differential diagnosis of CNS disorders [10]. In patients with CNS infection and decreased CSF glucose levels, if the differential diagnosis is between viral (e.g., HSV-1) and bacterial infection, CSF lactic acid levels will clearly differentiate between them, whereas CSF glucose levels will not [9].

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References


Successful Discontinuation of High-Dose Fluconazole for Histoplasma capsulatum Meningitis in an AIDS Patient after Sustained Immune Reconstitution

Sr—The observations made by Goldman et al. [1] demonstrate the safety of withdrawing antifungal therapy (i.e., that being used as secondary prophylaxis) from AIDS patients who experience successful immune reconstitution after prior dissemi-