complicated gonococcal infection until further information is available. In the rare instances in which tetracycline therapy is used because of serious allergy to penicillins, careful follow-up with test-of-cure cultures is mandatory. Tetracycline monotherapy has often been used because it was inexpensive and it treated both gonococcal and chlamydial disease in nonspecific urethritis. Recent studies of the tetracycline-resistant strain (or strains), as well as the 1985 treatment guidelines, would discourage that practice.[2]

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LOW SENSITIVITY OF ELISA TESTING IN EARLY HIV INFECTION

To the Editor: During seroconversion to human immunodeficiency virus (HIV, or HTLV-III/LAV), we have noted a "window period" of low sensitivity with the enzyme-linked immunosorbent assay (ELISA) as compared with confirmatory serologic assays. The following two cases illustrate the clinical relevance.

A patient at high risk for exposure to HIV presented with mononucleosis-like symptoms and a roseola-type rash. Since acute HIV infection and subsequent seroconversion have been reported to present at times with these signs or symptoms,[1,2] suspicions were high that the patient's symptoms were due to HIV infection. A previous serum sample was available, and serum samples were drawn weekly thereafter. The results of the serologic assays performed on these samples, using two different commercially available ELISAs, Western blotting, and radioimmunoprecipitation, are shown in Table 1. The patient's serum was consistently negative for antibodies to HIV on ELISA, yet positive on confirmatory tests, until three weeks after presentation.

Recently, another high-risk patient presented with progressive dementia and no reactivity for antibodies to HIV on ELISA.[3] When we tested the initial serum sample, it was negative by two different ELISAs yet markedly positive by membrane immunofluorescence, Western blotting, and radioimmunoprecipitation. An ELISA performed on a serum sample obtained two weeks later, however, did show reactivity (Bach M: personal communication).

Both cases, therefore, demonstrate that during seroconversion, there is a period of decreased sensitivity of the available ELISA systems as compared with other confirmatory assays, even when early clinical sequelae of HIV infection may be present. If a positive serologic status for HIV is to be used in the diagnostic criteria for acute HIV symptomatology, as recently recommended,[4] this window period of low sensitivity should be considered.

We estimate this period to last only two to three weeks, but we have also found certain clinical situations that may prolong it. Several patients with underlying cancer (two described by Anderson et al.[5]) have had weeks to months of ELISA negativity while other confirmatory assays were reactive. In addition, patients on intensive or long-term immunosuppressive regimens or patients previously infected with HIV who undergo bone marrow transplantation for other reasons may occasionally lose their ELISA reactivity while remaining positive on Western blotting and radioimmunoprecipitation (unpublished data).

Table 1. Results of Assays for HIV Performed with Two ELISAs, Western Blotting, and Radioimmunoprecipitation (RIP).

TIME OF TESTING	ELISA 1	ELISA 2	WESTERN BLOT	RIP
Before illness	–	–	–	–
During illness	–	–	+	+
1 Wk after illness	–	–	+	+
2 Wk after illness	–	–	+	+
3 Wk after illness	+	+	+	+

The performance characteristics of the ELISA in relation to its sensitivity have usually been studied with serum from patients with established cases of acquired immunodeficiency syndrome (AIDS).[6,7] The vast majority of persons infected with HIV, however, do not have AIDS. The patients mentioned here fall outside the Centers for Disease Control surveillance definition of AIDS but do have certain HIV-related signs or symptoms. In these specific clinical situations involving early HIV infection, the available ELISA quantitative systems may show a window period of poor sensitivity as compared with other serologic assay systems.

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VACUOLAR ENCEPHALOPATHY OF AIDS

To the Editor: Vacuolar myelopathy, characterized by vacuolation and intramyelinic edema of white matter in the spinal cord, occurs in up to 22 percent of patients with AIDS.[1] Its resemblance to subacute combined degeneration suggested that vacuolar myelopathy might be due to a deficiency of vitamin B_{12} or folate.[1] We have observed similar vacuolar degeneration in the brain of a patient with AIDS; the histopathological features are distinguishable from subacute combined degeneration.

The patient was a 45-year-old male homosexual with a two-month course of AIDS, *Pneumocystis carinii* pneumonia, anemia, thrombocytopenia, and symptoms of a decreased ability to concentrate, paranoid delusions, and visual hallucinations. Postmortem examination disclosed mild subacute encephalitis of AIDS,[2] and mild vacuolar myelopathy confined to the periphery of the spinal cord. In addition, the patient had vacuolation of the pyramidal tracts in the medulla and gray-matter structures of the left medial temporal lobe, including the cortex and amygdala (Fig. 1). He had no opportunistic infections in the central nervous system.

On the basis of these and other observations, there are three arguments against vacuolar encephalomyelopathy of AIDS having the same pathogenesis as subacute combined degeneration: first, although vitamin B_{12} deficiency may cause vacuolar degeneration of cerebral white matter, it does not involve gray-matter structures as described here[3]; second, in the present case, the vacuolar myelop-