Detection of HHV-8 DNA in PBMCs was independent of the patient’s HIV RNA level, CD4⁺ cell count, or levels of antibodies to HHV-8 (figure 1). In contrast, levels of antibodies to HHV-8 correlated with both the HIV viral load and the inverse of the CD4⁺ cell count from months 84 to 133. At month 133, as the HIV RNA level peaked and the CD4⁺ cell count was the lowest, all 3 antibodies to HHV-8 reached their highest levels (figure 1).

The patient had a low titer of antibody to HHV-8 lytic antigen (<1:40), which is generally considered unreliable for diagnosis for 7 years before seroconversion to HHV-8 ORF65 and K8.1 peptides. However, because of the HHV-8 DNA positivity for multiple HHV-8 gene segments at multiple times during this 7-year period, we believe that the patient was infected with HHV-8 at study entry, either before or at a very early stage of HIV infection. The lack of DNA sequence variation over time suggested that this individual was infected by a stable, single predominant HHV-8 strain.

This study provides clear evidence that an individual infected by HHV-8 before or in the early stage of HIV-1 infection can be either intermittently HHV-8 DNA-positive or seropositive for HHV-8 for an extended period before progression to KS. Because HHV-8 viremia can precede the development of significant levels of antibodies to HHV-8, both PCR and serological tests may be useful for the early detection of HHV-8 infection in individuals at high risk for KS.

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**Treatment of Hepatic Abscesses in Chronic Granulomatous Disease with Granulocyte-Macrophage Colony-Stimulating Factor**

Chronic granulomatous disease (CGD) is a rare inherited disorder characterized by high susceptibility to severe infections. In 60% of cases, the genetic defect is in the X-linked gene encoding gp91phox, whereas in the remaining cases, transmission is autosomal recessive (p22phox, p47phox, and p67phox). The expression of these abnormal genes determines a deficiency in a membrane-associated NADPH oxidase. This renders phagocytes unable to produce superoxide and other potent oxidants via the respiratory burst, and so unable to kill ingested catalase-positive bacteria and fungi. Continuous antibiotic and antifungal prophylaxis and aggressive treatment of infectious episodes have greatly improved the prognosis for patients with CGD [1]. Stimulation of the immune system with IFN-γ has also been shown to be effective in preventing or resolving acute infectious processes [1–4]. However, life-threatening infections may develop, sometimes with fatal outcomes.

A 21-year-old patient with X-linked CGD was hospitalized because of fever and deep asthenia that had lasted for 10 days. Two hepatic abscesses were found during abdominal ultrasound scanning. Blood cultures and serological tests were negative. Administration of iv vancomycin plus netilmicin and oral itraconazole did not lead to any improvement. After 9 days, subcutaneous IFN-γ (0.05 mg/m² 3 times a week) was added to the treatment. One week later, defervescence and a reduction in inflammatory parameters (C-reactive protein level decreased from 150 to 40 mg/L) were observed. IFN-γ therapy was continued, whereas vancomycin and netilmicin were replaced with rifampin and then azithromycin.

After 1 month, fever reappeared (temperature, 39°C) in association with hepatosplenomegaly and anemia. A new hepatic abscess was found, and the previous lesions were unchanged. *Staphylococcus aureus* was isolated from a tissue specimen ob-
tained during echography-driven liver biopsy. The patient was given different combinations of in vitro active antibiotics at high doses (vancomycin, rifampin, netilmicin, ciprofloxacin, teicoplanin, trimethoprim, doxycycline), IFN-γ 3 days a week, and a 5-day cycle of infusions of $6.7 \times 10^8$ to $11 \times 10^8$ polymorphonuclear leukocytes obtained by leukapheresis of blood from normal donors. Nevertheless, over the following 2 months, fever persisted, hepatic abscesses slightly increased, and the patient’s general condition worsened with significant weight loss.

Because several studies have shown that purified human granulocyte-macrophage colony-stimulating factor (GM-CSF) not only stimulates proliferation of immature progenitors but also enhances functions of mature effector cells [5], IFN-γ therapy was stopped, and subcutaneous GM-CSF (0.005 mg/kg/d) was administered, whereas the ongoing therapy with teicoplanin, ciprofloxacin, and itraconazole remained unmodified. Within 8 days, the patient became apyretic, and the C-reactive protein level decreased from 150 to 50 mg/L. In the following 4 months, GM-CSF administration was continued 3 times a week, and antibiotic treatment was reduced. During this period, the patient’s condition improved substantially, although fever occasionally occurred. GM-CSF gave rise to no side effects, but the number of peripheral polymorphonuclear leukocytes increased (peak neutrophil count, $18,600/\text{mm}^3$; peak eosinophil count, $2300/\text{mm}^3$). At the end of GM-CSF administration, MRI showed quiescent hepatic lesions, which gradually disappeared during the following year.

Therefore, for this patient, conventional therapy, including IFN-γ, was only transiently effective in controlling the severe hepatic infection, whereas the addition of GM-CSF to antibiotic treatment resulted in a persistent recovery. This case suggests that GM-CSF may be a valid alternative to IFN-γ to stimulate bacterial killing by phagocytes in patients with functional impairments such as CGD. An additional therapeutic advantage of GM-CSF may derive from the increased production of mature granulocytes and monocytes, which may be recruited into the site of infection.

**Rhodococcus equi Nosocomial Meningitis Cured by Levofloxacin and Shunt Removal**

*Rhodococcus equi* is increasingly being recognized as a pathogen in immunocompromised patients, especially those infected with HIV. The overwhelming majority of reported *R. equi* infections in humans involve the lung; CNS infections are rare, occurring as brain abscesses, and meningitis in a healthy host has never been reported [1].

Infections caused by non-*equi* *Rhodococcus* have largely been associated with procedures involving medical devices, but *R. equi* has not been reported as a nosocomial pathogen [2]. We report a case of meningitis due to *R. equi* in a patient with an external ventricular shunt, which we successfully treated with levofloxacin.

A 51-year-old woman was admitted to the neurosurgical department at our facility with a head trauma due to an accidental fall. She had a history of occasional alcohol consumption. Physical examination findings were remarkable for left hemiparesis. A CT of the head revealed a linear left-occipital fracture and a left-cerebellum hemATOMA that slightly compressed the brain stem. Chest radiographic findings were normal.

The next day, because of worsening coma, an external ventricular shunt was placed and ventilation assistance was started. A right traumatic pneumothorax was due to a central venous catheter and was treated by pleural drainage. After 2 weeks of improvement, a septic fever occurred with worsening of the neurological condition. A CT of the head revealed triventricular hydrocephalus. The external ventricular shunt was replaced, and laboratory studies of CSF showed the following values: glucose, 65 mg/dL (serum glucose, 226 mg/dL); protein, 90 ml/dL; and WBCs, $82/\text{mm}^3$ (all lymphocytes).

Routine cultures of the CSF and of the external ventricular shunt tip yielded *R. equi*. Susceptibility to antimicrobial agents was determined by the Kirby-Bauer disk-diffusion technique; and the organism was susceptible to ampicillin, ciprofloxacin,

References