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Use of Viral Blood Cultures in Diagnosing Herpes Simplex Virus Infection in Immunocompromised Patients. S.F. Reising and L.R. Stanberry, Children's Hospital Research Foundation, University of Cincinnati, Cincinnati, OH, USA.

Viral blood cultures are seldom obtained as part of the routine laboratory evaluation of suspected herpes simplex virus (HSV) infection because viremia due to HSV is rare in the normal host. Viremia, however, does occur in immunodeficient patients and has been detected in cultures of the buffy coat. Unfortunately, preparation of the buffy coat is tedious and leukopenic patients may have insufficient white blood cells to form a buffy coat layer. We have developed a simple technique for culturing whole blood. This method has permitted detection of herpes simplex viremia in 8 immunologically compromised pediatric patients: four neonates, three cancer patients and one renal transplant recipient. Only one patient initially exhibited evidence of mucocutaneous HSV infection. The indications for obtaining the viral blood cultures included routine surveillance, evaluation of fever, esophagitis, and perinatal exposure. In two patients HSV was only recovered from blood and in three other patients the viral blood cultures were the first positive cultures. Time to detection of HSV by viral blood culture ranged from 1 to 11 days. All patients were treated with intravenous acyclovir including three in whom therapy was initiated because of the positive blood culture. In addition to viral cultures of suspected lesions, mucosal surfaces and biopsy specimens, viral blood cultures should be obtained from neonates and immunocompromised patients with fever. The detection of HSV in blood may provide important prognostic information as well as facilitate early initiation of antiviral therapy.

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Antiviral Therapy of Acute Retinal Necrosis and Cerebral Vasculitis due to Varicella Zoster Virus in HIV Infected Patients. J.L. VILDE, F. ROUSSEAU, C. PERRONNE, P. LISOVOSKI, P. LONGUET, C. LEPORT. Claude Bernard-Bichat Hospital. Paris. France.

Drugs active against the varicella zoster virus (VZV) can usually control VZV infections in patients infected with HIV. We reviewed the cases of three HIV pts with acute retinal necrosis (ARN) and cerebral vasculitis related to chronic or recurrent VZV infection where antiVZV drugs were unable to avoid an unfavorable outcome. All three pts had ARC, mean CD4 cell count, 60/mm^3, at the beginning of the neurophtalmic complications. They developed an ARN on the first eye, 8 days, 30 days and 3 years after an episode of cutaneous infection due to VZV. Treatment of this first complication with I.V. acyclovir, 30 mg/kg/d in one pt, combined with I.V. ganciclovir 10 mg/kg/d in another pt, did not prevent the complete loss of vision of the eye which occurred within three weeks. All three pts developed ARN in the fellow eye, 3 months, 3 weeks and 2 years thereafter, while they were receiving low dose foscarnet, high dose acyclovir plus ganciclovir, and no treatment respectively. Evolution to blindness occurred within 3 to 6 weeks despite reinstitution of a high dose treatment with I.V. foscarnet 180 mg/kg/d in the three pts, combined with acyclovir 30 mg/kg/d in one pt. Severe neurological manifestations due to cerebral vasculitis developed 8 days to 5 months after blindness while the pts were receiving low dose foscarnet, high dose acyclovir, and high dose acyclovir plus foscarnet repectively. It lead to death in one pt who did not receive any antiVZV drug. In the two other pts improvement occurred without any treatment change, and a maintenance treatment with oral acyclovir 3-4 g/d was started. One pt died 14 months later of acute encephalitis, and the other pt is alive 18 months later. During this period they have had no relapse of VZV infection and related vasculitis. These observations suggest that high doses of antiVZV drugs, acyclovir, ganciclovir or foscarnet were unable to control the acute vasculitis process probably related to VZV, once it has started. However when the vasculitis is in remission, high oral doses of acyclovir may be sufficient to prevent reactivaion of the virus replication and thus relapse of the vasculitis.