Detection of WU Polyomavirus in Cerebrospinal Fluid Specimen from a Patient with AIDS and Suspected Progressive Multifocal Leukoencephalopathy

To the Editor—We read with interest the article recently published in the Journal of Infectious Diseases by Sharp et al. [1], who investigated whether the newly discovered KI, WU, and Merkel cell polyomaviruses could persist and reactivate in immunodeficient patients. To this aim, the authors screened for the presence of genomic sequences of KI, WU, and Merkel cell polyomaviruses, in addition to BK and JC polyomaviruses, in a series of lymphoid tissue autopsy samples from immunosuppressed and immunocompetent patients with AIDS but without signs or symptoms suggestive of polyomavirus-associated diseases, such as polyomavirus-associated nephropathy or progressive multifocal leukoencephalopathy (PML), and from control subjects without AIDS. The authors demonstrated that, overall, polyomaviruses were significantly more frequently detected in immunosuppressed patients than in non-immunosuppressed persons. In particular, with regard to the new polyomaviruses, KI polyomavirus was detected in 3 (7.1%) of 42 patients with AIDS and in 1 (1.8%) of 55 control subjects, WU polyomavirus was detected in 4 (9.5%) patients with AIDS, and Merkel cell polyomavirus was identified in only a human immunodeficiency virus (HIV)–infected injection drug user in the control group. Moreover, a higher viral load and a higher rate of mutations in the transcription control region of the viral genome were observed in immunosuppressed patients than in nonimmunosuppressed individuals with KI or WU polyomavirus infection.

We would like to support the findings of Sharp et al. [1] of a higher risk of reactivation of KI and WU polyomaviruses in immunosuppressed patients with AIDS and their hypothesis that these viruses might be responsible for disease in such patients [1], by reporting a case of WU polyomavirus DNA detection in a cerebrospinal fluid (CSF) specimen from a patient with AIDS who showed signs of PML but did not have JC polyomavirus infection. The case was identified among a series of CSF samples from patients with suspected viral encephalitis that were retrospectively analyzed for the presence of WU, KI, and Merkel cell polyomavirus genome sequences.

In particular, the study was performed using 60 CSF samples collected from January through June 2008 at the Microbiology and Virology Unit of Padova University Hospital (Padova, Italy) from patients (26 female and 34 male patients; median age, 44 years [range, 4–88 years]) with neurological signs and symptoms suggestive of acute or chronic viral encephalitis; results of polymerase chain reaction (PCR) testing for genome sequences of herpes simplex virus types 1 and 2, varicella zoster virus, and enteroviruses were negative. Six patients were HIV infected. Total nucleic acids were purified from CSF samples and were tested by quantitative real-time PCR for the presence of KI, WU, and Merkel cell polyomaviruses, as well as JC polyomavirus and BK polyomavirus DNA, as reported elsewhere [2–4].

All CSF samples were negative for KI and Merkel cell polyomaviruses and BK polyomavirus DNA, and only 1 of the 60 analyzed CSF samples, from a 41-year-old man, was positive for WU polyomavirus DNA (2500 genome copies/mL); however, this patient’s sample was negative for JC and other polyomavirus DNA. To exclude false-negative results [5], 4 different validated real-time PCR methods were used for JC polyomavirus testing, as reported [2]. The identity of the detected WU polyomavirus sequence was confirmed by sequencing, which demonstrated that it was identical to the published EU711058 sequence. The WU polyomavirus–positive patient was an injection drug user with depressive syndrome; he had HIV infection and AIDS, had been receiving highly active antiretroviral therapy since 1997, and was hepatitis B virus and hepatitis C virus positive with cirrhosis. He was admitted to the division of infectious diseases because of worsening asthenia, tremors, anxiety, and cognitive impairment for 1 month. His CD4+ T cell count was 400 cells/mL, and his HIV RNA load was <40 copies/mL. CSF examination findings were negative for bacteria and HIV RNA. Cerebral computed tomography findings were negative, electroencephalography showed diffuse anomalies, and cerebral magnetic resonance imaging identified several T2-hyperintense areas in the white subcortical matter of the frontal and temporal regions, without contrast enhancement, which were suggestive of PML.

To our knowledge, this is the first investigation of WU, KI, and Merkel cell polyomaviruses in CSF from patients with suspected viral encephalitis. Although detection of WU polyomavirus in a CSF specimen from a patient with AIDS could represent an occasional finding, its association with clinical and radiological signs suggestive of PML but with undetectable JC polyomavirus DNA in CSF is intriguing and warrants further investigation. Therefore, we suggest that, in addition to lymphoid tissues, cerebral samples from both immunosuppressed and immunocompetent individuals be analyzed to obtain ad-
ditional information on tissue distribution, tropism, and the potential diseases that might be caused by the new polyomaviruses, whose pathogenic roles have not yet been clearly demonstrated [6–8].

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