Vaccinia Complications and Methisazone Therapy

To the Editor—Nacla et al. [1] commented adversely on the toxicity of methisazone (Marboran; Burroughs Wellcome) during treatment of vaccinia virus infections. From 1965 to 1975, I treated 7 patients (5 children and 2 adults) with complications due to vaccinia virus infection using a 1965 non-nauseating suspension of methisazone dispensed in sealed plastic sachets [2]. Patients received 40 mg/kg per day orally in an aqueous vehicle in equally divided doses every 8 h for a total of 72 h. Vesicles from implantation vaccinia, eczema vaccinatum, or generalized vaccinia in 6 patients became dry within 1–2 days, and defervescence occurred 1 day after commencement of methisazone therapy. No toxicity was encountered in any patient. Remission of vaccinia necrosum without toxicity was noted after 4 days in an additional adult patient with leukemia. However, in 1964, when methisazone was dispensed as a tablet only, toxicity manifested as vomiting was encountered in a child with perineal vaccinia infection; her lesions remitted within 3 days.

Therapeutic response to complications due to vaccinia virus infection (without toxicity) using the 1965 methisazone suspension occurred equally promptly as did response after intravenous administration of vaccinia immune globulin in ocular vaccinia infection [3]. Therapeutic potency of methisazone in sachets was retained for at least 10 years when stored at 4°C.

Acknowledgments

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References

Acute Schizophrenic Manifestation as the Initial Manifestation of HIV Infection That Respond to Highly Active Antiretroviral Therapy

To the Editor—We read with a great interest an article in your journal by Shah et al. [1] that showed successful treatment of a case of HIV encephalopathy. Here, we report a case of a patient who had schizophrenic symptoms as the initial manifestation of HIV infection and had a regression of mental abnormalities following the initiation of HAART.

A previously healthy 25-year-old Japanese woman presented to Hokkaido University Hospital with psychotic symptoms, such as hallucinations and persecutory and somatic delusions. She had no history of abusing drugs or alcohol. Her Mini-Mental State Examination score was 27/30. The initial diagnosis was schizophrenia, defined according to International Classification of Diseases and Related Health Problems, 10th edition [2]. Although she was treated with quetiapine fumarate (300 mg daily), her psychotic symptoms did not show any improvement. Then, recurrent fever of unknown origin was observed and systemic examinations were performed. Radiography of the chest had no findings and tests for collagen diseases, vasculitic syndromes, and herpes groups viruses had negative results, but HIV-1 antibodies were present on ELISAs and Western blots. Her CD4+ T lymphocyte count was 2 cells/mm³. The plasma HIV-1 RNA load (Amplicor HIV-1 Monitor assay; Roche Molecular Systems) was 140,000 copies/mL. Enhanced MRI of the brain revealed no abnormalities. A lumbar puncture showed 3 lymphocytes/mm³ without atypia, a normal protein concentration of 25 mg/dL (normal concentration, <45 mg/dL), and a glucose concentration of 57 mg/dL. CSF cultures for bacteria, mycobacteria, and fungi were negative. Specific PCRs of CSF, including HIV, JC virus, cytomegalovirus, and varicella-zoster virus, were not done. Neurologically, coordination and all sensory and motor modalities were preserved. However, 18F-fluoro-2-deoxy-D-glucose positron emission tomography (FDG-PET) at rest revealed diffuse diminished metabolism in the patient’s brain (figure 1). Because treatment with quetiapine fumarate did not improve the patient’s symptoms, the diagnosis of HIV-associated psychosis presenting as a schizophrenic form was suspected.

HAART with lamivudine (300 mg